GUIDE
TO HEALTH AND HYGIENE IN AGRICULTURAL WORK
The International Programme for the Improvement of Working Conditions and Environment (PIACT) was launched by the International Labour Organisation in 1976 at the request of the International Labour Conference and after extensive consultations with member States.

PIACT is designed to promote or support action by member States to set and attain definite objectives aiming at "making work more human". The Programme is thus concerned with improving the quality of working life in all its aspects: for example, the prevention of occupational accidents and diseases, a wider application of the principles of ergonomics, the improvement of the content and organisation of work and of conditions of work in general, a greater concern for the human element in the transfer of technology. To achieve these aims, PIACT makes use of and co-ordinates the traditional means of ILO action, including:

— the preparation and revision of international labour standards;

— tripartite meetings between representatives of governments, employers and workers, including industrial committees to study the problems facing major industries, regional meetings and meetings of experts;

— action-oriented studies and research;

— clearing-house activities, especially through the International Occupational Safety and Health Information Centre (CIS); and

— operational activities, including the despatch of multidisciplinary teams to assist member States on request.

This publication is the outcome of a PIACT project.
Guide to health and hygiene in agricultural work
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International Labour Office Geneva
Preface

The International Labour Organisation’s concern with the safety, health and welfare of agricultural workers derives from its Constitution, which requires it to further programmes to achieve adequate protection for the life and health of workers in all occupations. Following the Second World War the International Labour Office embarked on a series of activities at the international level to improve the occupational safety and health of the numerous persons in the agricultural sector, at that time considered a neglected branch of economic activity, and in 1965 it published *Safety and health in agricultural work*, a code of practice on the subject. The recommendations in the code, which were expressed in the form of concise rules with a minimum of explanation and detail, were intended for the guidance of authorities, professional groups and all those with responsibilities in the promotion of occupational safety and health in agriculture, including the self-employed farmer. Agriculture was taken to mean farming, including cattle and fruit farming, but excluding forestry and the manufacture of food products in factories.

The code was followed in 1969 by a related *Guide to safety in agriculture*. The guide concentrated on the same aspects of safety as the code, but in more detail and with additional explanations and illustrations; it dealt only in summary fashion with matters of health and hygiene. It was mainly intended for economically advanced countries, but was also expected to be useful to those countries where agricultural mechanisation was spreading rapidly.

The present guide, on health and hygiene, completes the triad of ILO publications intended for the protection of the agricultural worker from occupational accidents and diseases. With its predecessors, it provides a wide spectrum of information on agricultural health hazards and their prevention.

It is hoped that this guide, which focuses on the problems of developing countries but deals also with issues of concern to industrialised countries, will be a useful source of reference for
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all persons and bodies with responsibility for health and hygiene in agricultural work, including teachers, persons in charge of agricultural undertakings, employers' and workers' organisations, farmers' co-operatives and self-employed farmers, wherever they might be.

It is realised that it is not easy to deal in one volume with all aspects of health and hygiene in all kinds of farming settings, but the Office feels that the guide will help to improve occupational health in a branch of economic activity which in many countries is still rather neglected.

The following specialists collaborated in the preparation of this guide: Dr. Mostafa A. El Batawi, World Health Organization, Geneva (Switzerland); Professor L. J. Medved, USSR; Dr. Donald P. Morgan, United States; Professor G. Preuschen, Federal Republic of Germany; Professor M. N. Rao, India; Professor Jean Vacher, France; Professor Marcus Wassermann and Dr. Dora Wassermann, Israel. Thanks are due for the observations made on the preliminary drafts by the members of the ILO Panel of Consultants on Safety and Health in Agriculture, representing government, employer and worker circles; and particular appreciation must be expressed to Professor L. W. Knapp, Jr., University of Iowa, United States, for his extremely helpful review of the final text of the guide.

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1. Living conditions and environmental hygiene

1.1. Introduction

Agriculture has been defined as the science or practice of cultivating the soil and rearing animals. It was one of the major activities of primitive man, and it is still of great economic importance today. During recent years, particularly in the developed countries, agriculture has been undergoing a technological revolution, as a result of which activities in this sector now range from primitive hand-ploughing in underdeveloped regions still untouched by the revolution to highly mechanised farming in many of the industrialised nations. Agricultural medicine, a science concerned with the health of the rural worker and his family, is an acknowledged occupational health specialisation in some countries, particularly in those where it has been recognised that there are features peculiar to the rural environment, workplace and home that affect the health of the agricultural worker.

Rural areas may be defined as areas where agriculture is the chief or even the sole industry and where there are often few skills and/or organised community services (such as facilities for water supply and excreta disposal, control of vectors of diseases, and similar services). In such areas, where dwellings are scattered or gathered together in remote small groups, it is difficult to provide organised community services at a cost commensurate with the economic resources of the persons concerned. Some of the larger agricultural labour forces are to be found in the developing countries, and their members often have to contend not only with the lack of community services and poor communications systems but also with the numerous environmental problems peculiar to agricultural life.

The patterns of life in rural areas are far from uniform, and agricultural medical and health problems are at least as varied
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as those encountered in industry: in fact, the range of health problems may be greater in agriculture. At one extreme, in isolated communities composed of people leading a primitive, nomadic life, the most urgent need is for basic health services, concentrating on matters of elementary hygiene and sanitation. At the other extreme, on large industrialised farms, health services have to cope with more sophisticated problems—chemicals, noise, vibration, and so on. Agricultural health problems on modern highly automated farms may be more complicated than industrial health problems in the organised urban sector.

In this introductory chapter dealing with general aspects of environmental hygiene in agricultural life, only certain basic principles will be discussed. This exposition is intended in particular for small farmers all over the world, but those who operate larger enterprises in developing countries may also draw benefit from it.

1.2. Housing

Strictly speaking, rural housing includes housing in the countryside used for occasional or seasonal residence by urban dwellers. However, for the purposes of this guide it will be taken to mean only housing used for agricultural activity, lived in by the working farmer, the agricultural labourer or the rural craftsman while he works a small agricultural holding or practises his craft.

Rural housing differs in some respects from housing for industrial workers in an urban community:

(1) An agricultural worker’s occupation and home life are not so distinct as those of an industrial worker, and his working conditions are blended with his living conditions.

(2) Except in rural slum areas, such as exist on the fringe of some industrial communities, the chief problem of the agricultural worker is usually not overcrowding but isolation. He may suffer from a lack of communications, transportation and amenities such as gas and electricity. Although the radio for
many and the helicopter for a few may bring the modern world nearer, the agricultural worker often has little access to the advantages of modern science and has to be self-reliant.

(3) Unlike the highly specialised worker living in an urban area, the agricultural worker has to live closer to nature, with his livestock and crops, so that the provision of outbuildings has to be taken into account in agricultural housing programmes.

(4) Many simple housing problems assume exaggerated proportions for the farmer, and as a rule he will require more technical advice on housing than his urban counterpart. In the absence of advice, he is likely to fall back on traditional practices which may not always be in his best interest. He must therefore be given a clear explanation of the best methods of construction to follow, of the kind of materials (including artificial materials) that he needs and of how to use them. Modern sanitary knowledge and services have to be brought to his doorstep.

1.2.1. Construction

Planning

The opportunities for sanitary amenities are an important consideration in the choice of a new construction site. Among the principal health criteria are: orientation (which should take account of the sun and prevailing winds); good drainage; a good household water supply; and facilities for sewage disposal.

In many countries, those responsible for regional planning should envisage the construction of new rural dwellings in groups, from which the workers could travel each day to their worksite. This would help to overcome a sense of isolation and at the same time make it easier to provide better sanitation, water supplies, educational services, transportation and communications.

For reasons of safety and health, the farmer's living quarters should be set apart from outbuildings which house livestock, crops and implements. Back-to-back construction, as is the practice in some agricultural communities, is to be discouraged.
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Certain features and practices are common to farmhouses in many parts of the world, but some of them should be discouraged. Combining the kitchen and the living-room is not a healthy practice, and the use of beaten earth for outbuildings and sometimes for living-room floors is not conducive to good housekeeping. The use of cellars, especially in cold countries, can pose some particular health problems, and crowding the family into a common living-room in the cold winter months is also unhealthy.

Problems of this kind, like many others encountered in agricultural communities, imply a need for scientific and technical advice, health education and follow-up control arrangements. There is clearly a need for persons who can supply these services with the help of simple reference material suited to local requirements.

Construction material

If the materials used and the methods followed are in line with the provisions of local building and health codes, the risk of accidents and fire is likely to be small. However, in isolated communities it may not be possible to ensure that the provisions of such codes are applied or that inspections are carried out.

While the choice of roofing material usually depends on what is available locally, whatever kind is used should be a good insulator against external temperature changes. Straw or thatched roofs afford effective protection against solar heat or intense cold, but they offer a serious fire risk and are not to be recommended.

The problem of damp flooring and walls should not arise if suitable construction materials or simple damp-proofing techniques are used.

In many agricultural communities, termite infestation is common. These destructive insects can weaken the structure of wooden buildings, thereby creating a risk of accidents. Wooden foundations should consequently not be used in areas where termite infestation is known to be severe, and the construction material chosen should be such as to provide protection against termites.
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Accidents

Common household accidents can easily be avoided if adequate preventive measures are taken at the proper stage. Thus, staircase accidents may be prevented by providing for uniform steps about 25 cm deep (run) and 18 cm high (rise), with a non-slip surface. The risk of falls in general is likely to be minimised if adequate lighting and handrails are provided. Railings around porticos and balconies are a necessary protection for both children and adults.

Fire hazards should always be borne in mind. Electric wiring and equipment should conform to the provisions of safety codes; stoves and other sources of heat should be mounted clear of combustible material; smoke pipes and chimneys should be adequately constructed; and there should be proper fire escapes.


1.2.2. Physiological principles

In a rural environment there are normally not so many recreation facilities as in an urban environment, so that a villager may spend more time at home than his urban counterpart. This implies that rather more than minimum requirements should be taken into account in the construction of rural housing. The house becomes a "home" when psychological as well as physiological needs are met.

Satisfying physiological needs means ensuring that environmental conditions such as heat, light and air movement are maintained at a level compatible with human health and comfort, irrespective of climatic conditions outside.

Heating

In cold climatic conditions housing must be constructed in such a way as to permit the conservation of heat. Stoves, radiators or fireplaces act as direct heat sources and are usually located in the room being warmed. With indirect heating the heat source
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is located elsewhere and heated air or water is circulated to the room to be warmed through a pipe or duct system. In the case of modern “radiant” heating, for example, hot water is circulated through a piped system of coils within the floors or the walls or by electric wires imbedded in the ceiling. It is desirable to maintain a comfortable environmental temperature and to keep temperature fluctuation to a minimum between ground level and eye level (1.5 m) in living-rooms.

Carbon monoxide

Accidental carbon monoxide intoxication occurs more often in cold countries when homes are heated by gas. Carbon monoxide is a colourless and odourless gas (see subsection 3.6.5) which can be highly dangerous to the occupants of a closed room since they are given no warning of its presence. It is toxic even in concentrations of as little as 0.02 per cent (“natural” gas, on the other hand, is non-toxic).

Simple household heating appliances of the kind widely used on primitive farms have been known to produce a high concentration of carbon monoxide through inefficient combustion of fuel. The open burning of charcoal in a closed room is particularly dangerous. Normally, even if pockets of high carbon monoxide concentrations are produced in a house, the constant circulation of fresh air will minimise this risk to life. Other precautions will also reduce the hazard. In the case of ovens in kitchens, water heaters in basements or bathrooms and heaters in living-rooms, pipes or vents designed to carry the products of combustion to flues or chimneys and permitting a free flow of air to the flues are the solution to the problem. However, they must be inspected regularly for holes or obstructions.

Cooling

In hot countries, where solar radiation is excessive, there is a physiological necessity to cool the living environment. A difference of up to 30°C between shade temperature and solar radiation temperature can be expected in summer in regions
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with only a mild tropical climate, and sometimes even with a subtropical climate. Recommended countermeasures include a verandah all round the house, adequate roof construction to minimise the re-radiation of heat into living-rooms, and the planting of shady trees. In semi-desert conditions, where nocturnal temperatures are low and diurnal fluctuations are great, a flat roof construction may be called for. Those who like to sleep in the open may opt for terraces or porches, in which case these should be screened in such a way as to minimise exposure to insects. Evaporative or mechanical refrigeration may also be considered as a means of reducing the ambient temperature of housing. As a rule, a high roof is conducive to a cooler room.

Illumination and ventilation

Adequate window space, especially in tropical and subtropical countries, contributes to both illumination and ventilation.

If the window space provided amounts to between 10 and 15 per cent of the floor area, sufficient natural daytime illumination can be obtained. Windows (suitably oriented for cross-ventilation) are best placed rather high in the walls so that convection currents may be created.

To secure adequate ventilation, the provision of a minimum of 50 m³ of fresh air per person per hour is desirable. Such air movement also permits the removal of body odours and minimal evaporative cooling of the body.

1.2.3. Insect pests

Cockroaches

Cockroaches are not only a nuisance but also a health hazard. They are household pests consuming any accessible foodstuff and multiplying in dark corners and sewers. They not only carry infection from sewers on the outside of their bodies but also harbour in their intestines bacteria associated with food poisoning in human beings. Chemicals such as powdered sodium fluoride
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are effective against cockroaches, but some of them are poisonous to man also. A 2.5 per cent oil solution or emulsion of chlordane is less toxic and will bring them out of their hiding places, as will any irritant. They can be killed by 5 per cent chlordane dust or 10 per cent DDT powder. When any insecticide is used, food dishes and utensils must be covered. Primary prevention is based on good housekeeping and food storage practices denying them access to a food supply.

Bugs

Another insect in the house which plays an uncertain part in the transmission of disease, but which is a definite nuisance, is the blood-sucking bedbug. It is nocturnal and hides in inaccessible crevices. Bedbugs can be dealt with in a few minutes by heat (about 46°C) and in a few days by DDT (5 per cent solution). It may be necessary to repeat the treatment after a week or ten days for the second generation of bugs, which may have been in the resistant egg stage and which therefore may have escaped extermination the first time that eradication measures were taken.

Lice

Lice on the human body, although they are not directly related to housing conditions, are indicative of poor personal hygiene. In addition to being a personal nuisance, they can be responsible for the spreading of typhus. Body hairs are their home, and adequate skin care is therefore the only preventive. They can be dealt with by dusting the hairy parts of the body and the underwear with 10 per cent DDT powder or with certain other insecticides, such as lindane, malathion and carbaryl dust. As with other insects, the eggs are not affected by DDT, and hence a second application a week or ten days later (the gestation interval for the next generation) is recommended.

Other insects, such as ticks and fleas, may be brought into agricultural workers’ homes by animals. They can usually be dealt with effectively by measures similar to those described above.
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1.2.4. Rodents

Rodents can be a problem, particularly where agricultural workers’ dwellings are close to the quarters of domestic animals. The foundations of houses should be constructed of concrete or other rat-proof material. Where this is not practicable, rodent-proof cupboards or food stores should be provided. All food that attracts rodents should be kept in metal containers.

1.3. Farm buildings

The agricultural worker lives and works in the same environment, and for him occupational health and general health are more closely related than in the case of the factory worker. During the planting and harvesting seasons he may have to work from dawn to dusk and attend to farm animals at all hours. It is understandable that he may prefer to live close to his workplace. However, it is not desirable, from the health point of view, that his home should be a part of his working environment, and farm animals, including pet dogs and cats which are often kept in large numbers on farms, should be housed separately. Kennels may be inside the compound if required, but definitely not inside the farmer’s house. Farm buildings, including those where farm implements are kept, or which serve as a workshop (tools can be carriers of diseases), should be located as far away as possible as a precaution against the risk of fire and for health reasons.

1.3.1. Grain stores

Agricultural families the world over store grain for use throughout the year, and a grain store-room is usually part of the farm buildings. Grain stores attract vermin, so measures to prevent their infiltration must be taken. A well designed grain store is rat-proof, well ventilated and adequately screened. Contrary to what has long been believed in some cultures, a silo-like structure
with thick mud walls is *not* rat-proof. However, where there is no alternative, the floor, if it is to be impervious, should be of stone or concrete or brick (10 cm thick) with 1.5 cm of cement plaster. Filling the basement with sand to a depth of 60–90 cm helps to prevent rats from burrowing their way into a grain store. The basement should be 60–90 cm above ground level, preferably with no steps leading to it. A step-ladder can be used to get to it. All entrances to the store should be 90 cm or more above ground level. A ledge or other horizontal projection at least 25 cm wide round the building, 45–60 cm above the ground, will also prevent rats from crawling up the walls. These should be thick, with a 1.5 cm brick or cement plaster lining going up to a minimum height of 90 cm. There should be no plumbing pipes outside the walls, and no windows are necessary. Ventilators should be rat-proofed by means of expanded metal shields. All doors and windows should be self-closing and any openings fitted with wire gauze. If there are any timber doors, the bottom 25 cm should be finished with metal to prevent the rats from gnawing through the wood. Windows and doors should be as air-tight as possible so as to permit fumigation whenever necessary.

The space between the wall and the roof should be covered with masonry blocks. The roofing must be such that rats cannot find their way through in any circumstances. If it is terraced or tiled, there should be no opportunity for them to do so. The possibility that rats may reach the roof directly without having to crawl up the walls or drain pipes must be borne in mind; rats can climb trees and can drop easily on to a roof from overhanging branches.

1.3.2. *Farm incinerators*

A practical method of disposing of refuse is to make it into compost on the farm, where a piece of land can usually be found for the purpose. The most hygienic method, however, especially when the refuse is of little value or is offensive or dangerous, is incineration. Incinerators range from the simple drum type that
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may be used on a small farm to highly sophisticated, mechanised containers serving whole communities. Those used on farms should be located downwind from the dwelling so that the occupants are not exposed to the smoke or foul odours.

Incombustible refuse (glass, tin, etc.) should be put aside before combustible refuse is fed into the incinerator. The ash should be collected from the ashpit and buried. Proper care must, of course, be taken to prevent the escape of open flames or the dispersal of burning refuse.

1.3.3. Buildings for livestock

Dairy livestock are found on many farms. The quality of the milk reaching the consumer is largely determined at the farm. This means that the milk must be stored there in sanitary conditions until it is either used on the spot or sent away for consumption elsewhere. It is therefore an advantage to have a milk-house exclusively for the handling and storage of milk. This should always be separate from the cattle-shed. In many cases the two units—the cattle-shed and the milk-house—are combined into one, an arrangement which may not be satisfactory if the building is poorly designed. In a combined unit the cattle should be provided with adequate light and protection against extremes of temperature. There should be separate approaches for the cattle themselves, for the delivery of fodder, and for sending away milk.

Cattle-sheds

Cattle are usually housed in sheds. These should have a smooth, compact floor paved with concrete or other impermeable material, good drainage and if possible partitioned stalls for each animal, with provision for a manger. They should be properly ventilated and lit. Manure must be removed frequently and dumped in a pit away from the sheds or fed into a water-tight chamber for the recovery of methane and fertiliser for domestic use.
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Farm milking units

The farm milking unit should be separate from the cattle-shed. It should be as sanitary as possible, with screened windows and self-closing screened doors. There should be adequate washing sinks with hot and cold water. Paved flooring with drains to lead away waste water is essential. Well designed, good-quality equipment for straining, cooling and storing the milk must be provided.

1.3.4. Piggeries

Garbage on the farm is a potentially valuable substance, and household garbage can, if cooked, be used for feeding hogs. Hog-keeping is common in many parts of the world. However, hogs that are fed with garbage collected from cities and industries are more prone to a Trichinella spiralis infestation than grain-fed hogs, and more often suffer from vesicular exanthema. A recommended sanitary practice is to heat the garbage to boiling point for an hour before feeding the hogs.

Foul odours, fly-breeding and rodent nuisance are examples of other problems encountered on hog farms. These problems can be reduced by constructing an impermeable platform (for instance, with reinforced concrete) which prevents the hogs from trampling feed into the ground. Feeding places should be equipped with facilities for adequate draining and daily cleaning, so that manure and uneaten feed may be collected for sanitary disposal or recycling.

The principles concerning the ventilation and illumination of cattle-sheds outlined above apply also to large piggeries. Moreover, piggeries must be designed in such a way that a hungry pig's access to dirty water and excreta is reduced to the minimum. Separate dunging and feeding can be encouraged by arranging the pen suitably for a diagonal traverse by the pig. Swine are clean animals if they are allowed to be so. The floor should be well paved and should slope down to inaccessible narrow channels along the walls, inside or outside, preferably leading to a manure
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sump. Liquid waste from piggeries has a high nitrogen content and is valuable as a farm fertiliser.

The roof should preferably slope away from the pig-pen to avoid flooding of the yard.

1.3.5. Poultry sheds

Sound poultry farming begins with adequate plans for housing the poultry. An ill ventilated, insanitary and uncomfortable poultry house is likely not only to result in low egg production and poor fertility but also to affect the health of the poultry and lead to a high mortality rate. The poultry in the house must be protected against natural enemies, such as dogs, cats, foxes and crows, and special measures must be taken to protect them against snakes and insects such as ticks, lice and mites.

Poultry houses can be planned in a variety of ways. A house with wire-netting on all sides, a clean floor set above dry ground, and a good large roof will suffice in the majority of instances where temperatures are moderate. (Moist ground and damp floors are known to increase morbidity among poultry.) Sandy loam soil affording good drainage and suitable for growing grass is to be recommended.

There should be as few corners and crevices as possible, to permit easy cleaning and spraying. There should be as little woodwork as possible, and what there is should be treated with insecticides such as coal tar.

Farm hands working with poultry should be made aware of the need for cleanliness and should be advised to take prompt care of scratches or abrasions.

1.4. Water supply

1.4.1. Importance

Water-borne diseases are the major cause of death in economically underdeveloped and basically agricultural countries. Plain
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diarrhoea and enteric infections, bacillary dysentery and cholera can usually be traced to the drinking of unsanitary water. In some cases, water has an indirect relationship to disease, as in the transmission of the mosquito-borne diseases of malaria and filariasis, and it is sometimes responsible for the transmission of brucellosis, tularaemia, haemorrhagic jaundice and several other protozoal and viral infections. The control of these diseases through a clean water supply programme is a matter of top priority in the field of agricultural health.

It is important that a thorough field investigation be carried out before a water supply programme for agricultural communities is developed. The adequacy of physical and chemical standards, the bacteriological quality of water, and—of particular relevance but rarely recognised as such—the potential of the environment for contamination must all be examined. The risk of chemical pollution, especially in the form of chlorides from barns and manure pits located too close to a water source, or of bacterial pollution by human excreta from nearby latrines, must always be borne in mind. As long ago as 1958, a WHO report\textsuperscript{1} summarised world opinion on the subject of water sources and human pollution as follows:

Regarding the location of latrines with respect to sources of water supply, the following conclusions may be drawn from up-to-date information:

1. There can be no arbitrary rule governing the distance that is necessary for safety between a privy and a source of water supply. Many factors, such as slope and level of ground water and soil permeability, affect the removal of bacteria in ground water. It is of the greatest importance to locate the privy or cesspool downhill, or at least on some level piece of land, and to avoid, if possible, placing it directly uphill from a well. Where uphill locations cannot be avoided,

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a distance of 15 m will prevent bacterial pollution of the well. Setting the privy off to either the right or the left would considerably lessen the possibility of contaminating the ground water reaching the well. In sandy soil a privy may be located as close as 7.5 m from a properly constructed household well if it is impossible to place it at a greater distance. In the case of a higher yielding well, not less than 15 m should separate the well from a latrine.

2. In homogeneous soils the chance of ground water pollution is virtually nil if the bottom of a latrine is more than 1.5 m above the ground water table. The same may be said if the bottom of a cesspool is more than 3 m above the level of the ground water.

3. A careful investigation should be made before building pit privies, bored-hole latrines, cesspools and seepage pits in areas containing fissured rocks or limestone formations, since pollution may be carried directly through solution channels and without natural filtration to distant wells or other sources of drinking-water supplies.

1.4.2. Sources

Common sources

In villages where dwellings are scattered, families draw their drinking water from separate sources. Thus, in cases of waterborne disease, the source of contamination can be pinpointed from epidemiological data. This is not so in cities, where there is a common source and where the spread of such diseases can be dramatic.

Agricultural workers tend to draw water from the nearest source. The main reason why civilisation spread along the banks of rivers and canals is that running water was close at hand. It is also common practice in rural areas to draw water from natural lakes or from tanks or wells. Lake water undergoes self-purification by sedimentation, which eliminates suspended matter and reduces turbidity. The bacterial count is reduced in the self-purification process through an environment which is hostile to bacterial growth. But one cannot entirely depend on this method of self-purification under rural conditions because of the difficulty of stopping the continuous contamination of the water by human
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beings and animals. Where the level of subsoil water is very high, as in delta regions, every agricultural family may have its own tanks or each group of houses may share a tank. Most surface water in rural areas is exposed to gross contamination and frequently does not conform to sanitary standards. Family tanks are invariably used for all purposes, including drinking, and tanks serving as water sources in the village are always liable to misuse. Provided that proper precautions are taken, a water supply from dug wells may therefore be more sanitary than one from natural accumulations of water in tanks. Open shallow wells are common in agricultural communities, and here the main risk of pollution comes from the surface itself, for example from the use of common buckets and ropes or because the lining of open wells is not always pollution-proof; even pottery rings are known to retain contamination from the surface for a long time. Sanitary improvement of existing dug wells may be the only solution in many cases. For example, it may be possible to provide a satisfactory parapet, an apron and a drain, and then to close the mouth of the well and to replace the rope and bucket by a pump for drawing water (see figure 1).

When new wells are being sunk, it is advisable to give each well a brick lining extending from the top of the well to a point 3 m below the surface, with a layer of cement plaster at least 2.5 cm thick. A concrete apron surrounding the well, at least 45 cm wide and preferably 90 cm wide, is also to be recommended; this apron should have a 1:24 slope. In the case of a closed water-tight well, the pump should be properly fixed to the cover, and surface pollution through seepage of surface water must be avoided. Waste water must be disposed of in a satisfactory manner. Whether this is done through drainage into tanks or soakage pits or by means of surface drains will depend on local facilities.

Tapping subterranean water below an impermeable layer by means of bored wells offers a satisfactory solution to the problem of obtaining drinking water of optimum sanitary standard on a farm. Bored wells may be of different kinds, depending on the type of soil, the level and extent of the first impermeable layer,
Figure 1. Dug well

- Manhole cover
- Cement concrete
- 1.20 m
- 90 cm
- Slope 1:24
- Plaster with cement mortar
- 2.5 cm thick
- Clay
- Cylinder
- Water-bearing stratum
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the level at which the subterranean water is available, and the techniques in general use. Three kinds of well are commonly found. For the first kind, holes a few centimetres in diameter can be bored in loose soil, by a hand-operated auger, with machine drills being used for larger-diameter bores; however, the water supply will be limited by the superficiality of the water-bearing strata. For the second kind, either hand sunk for small diameters or machine sunk for larger diameters, a pipe is driven, usually with 1–2 m of screen at the end of the pipe near the driving point. For the third kind, a hole, small or large, is drilled either hydraulically or pneumatically into the impermeable rock layers. This kind, though more costly, yields a greater and more continuous supply of water of a sanitary quality than do other bored wells.

Where there are alluvial soils and the water-bearing strata are not very deep (not more than 10 m below the surface), it is more economical to have tube wells, since a tube well is the most sanitary kind for both human beings and animals on a farm. A tube well of small diameter—usually 4 cm—with 2–4 m of strainer, depending on the number of users, and a hand pump are recommended as a drinking-water source for the agricultural population in delta areas of developing countries (see figure 2). Where a large quantity of water is wanted on a farm, a larger-diameter tube well with a longer strainer and power pumps may be required. Though initially costly in hard soils and occasionally in need of repair, tube wells are a steady source of hygienic water supply in any agricultural community where the water-bearing subsoil is not very deep and where an efficient maintenance service is available. Whenever there is reason to fear contamination, chlorination is advisable. Coliform organisms have been known to linger in leather buckets; after each repair, the tube well and the pump should be disinfected.

Springs as sources of drinking water

It is a common belief that spring water is safe to drink. This is not always true, because of the way that accumulations of subsoil water are formed.
Figure 2. Tube well

Clay, fine sand and impermeable stratum

= 4 cm

2–4 m

Coarse sand
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Unlike surface water accumulations (for example tanks, where the water is stagnant), subterranean water is always in motion. The subsoil water level is not horizontal, but slopes according to the geological formation of the strata in which it lies.

If the local health agency, after making a laboratory study, can certify natural springs as safe, they can be an excellent source of drinking water for agricultural communities.

Sanitary standards

Bacteriological standards. The usual public health yardstick for measuring the safety of water sources is the extent of contamination by faecal organisms. Coliform organisms, of which Escherichia coli is one, are found in abundance in the large intestines of man and animals, and the degree of contamination from faecal sources depends on the number of coliform bacteria in the drinking water. The higher the coliform count, the greater the possibility of faecal contamination. In the case of open shallow wells, the coliform count may run up to thousands of bacteria per 100 cm$^3$ of water. If the open top of a shallow well is completely covered, the bacterial count will be somewhat less; and if, instead of the water being drawn manually, it is pumped up by mechanical means, this count will be very much less. In a majority of cases, open shallow wells on a farm can be made fit for drinking purposes merely by providing the well with a pump and a concrete cover. Experiments have shown that by such simple methods the bacterial count can be reduced to fewer than 100 coliform organisms per 100 cm$^3$ of water sample. A number of samples should, however, be tested over a period of time before a water source is finally accepted as sanitary.

Chemical standards. An important consideration in relation to ground water is its degree of hardness or salinity. It may contain calcium, magnesium, potassium and sodium salts, carbonates, bicarbonates, sulphates or chlorides. Excessive amounts of some of these may be harmful to man and vegetation. For example, in certain areas where there is a high content of fluoride and water
in the soil (i.e. more than the permissible concentration of 1.5 ppm), farm animals and human beings have been found to suffer from fluorosis, a disease of the bones, teeth and ligaments. Nitrate, indicating surface pollution in shallow wells, may turn into nitrite and lead to methaemoglobinaemia in infants.

1.4.3. Treatment

Treatment on a community basis

In planning the layout and construction of water treatment plants, allowance should be made for future extensions, taking account of the likely rate of growth of the rural community.

There are four main treatment processes: sedimentation, coagulation, filtration and chlorination. The use made of each one, separately or in combination, must be supervised by a competent authority, preferably a sanitary engineer.

Most pathogenic micro-organisms find water an unsuitable medium for multiplication, and hence plain sedimentation resulting from holding water in a selected storage space or reservoir reduces the bacterial content significantly. The possibility that algae may be produced in exposed reservoirs should, however, be borne in mind, and competent technical guidance is needed to prevent this from happening or to find a remedy if the problem has already arisen. The turbidity of water also decreases during storing processes unless the particulate matter is fine clay or of a colloidal nature. Coagulation through the addition of suitable chemicals is necessary to remove colloidal and suspended matter and some dissolved solids. Alum used in appropriate amounts, usually 1–5 g per 75 l of water, will reduce turbidity and help filtration. Slow sand filtration is recommended to reduce moderate turbidity and bacterial counts. Rapid sand filters, commonly used in urban areas, are not generally recommended for agricultural communities because of the scarcity of skilled operators.

Chlorination—and not treatment with potassium permanganate—is recommended for the destruction of organisms usually associated with water-borne diseases. Well preserved chemicals
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have to be used and allowance must be made for a rapid reduction in the chlorine content of the solution. Whether the content is sufficient can be determined by simple orthotolidine reagent tests for residual chlorine. A solution of the desired amount of disinfecting bleaching powder is added and after 30 minutes a sample is taken. The chlorine colour comparator contains sealed tubes whose standard colours represent various levels of residual chlorine. Drops of orthotolidine solution are added to the sample, and the developed colour is compared with the standard one. This simple test is recommended for use on farms.

In agricultural communities whose members enjoy good relations with one another, a community water supply programme providing for frequent inspection services and for orthotolidine testing for chlorination can be introduced.

Treatment on a family unit basis

Because of the microscopic size of bacteria, they may be present in water whose crystal-clear appearance may induce a false sense of security in both children and adults. Although a majority of bacteria may not be pathogenic, there is no simple way of knowing what proportion of pathogenic bacteria is present in each instance. The most practical method of disinfection or destruction of bacteria is to boil the water, though sometimes this may be expensive. Boiling is also helpful for removing temporary hardness. All pathogenic organisms are killed if water is brought to a “rolling boil” and not merely a “bubbling boil”. To be sure of killing the spores, if any, the method of alternate heating and cooling three times to encourage them to vegetate enough to become heat-susceptible can be recommended; it is rarely practised, however.

Simple disinfection procedures may be adopted for use at home when agricultural communities are located in very isolated situations, or for use during emergencies or when travelling. For routine purposes, fresh bleaching powder in doses of 4–6 ppm is generally adequate. Iodine disinfecting tablets have been known to be effective against amoebic cysts, cercaria, leptospirosis and some viruses. A few drops of tincture of iodine in a bucketful of water giving a
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concentration of 6–8 ppm can be recommended for use in emergencies (for example, if a water-borne disease breaks out on a farm) as a suitable alternative to destroying the bacteria by boiling. Diatomaceous earth filters of indigenous origin have been widely used on farms in many countries (see figure 3). In many cases they are the most suitable filter, but users should be taught to look for sources of faulty filtration, such as choking off, cracks in the filter units or porous "candles".

1.4.4. Distribution

A piped water supply to each household is undoubtedly the best method of water distribution, but is not generally feasible in rural communities. In areas where communities are dense, a piped water supply is always to be recommended, but the use of indigenous material such as split bamboo should be discouraged.

A WHO report\(^1\) recommends the following order of priorities when the choice of a water-supply programme is being considered:

(a) natural water of adequate standard delivered to the consumer by a gravity system;

(b) natural water of adequate standard delivered to the consumer by pumping (for instance, water from wells);

(c) water requiring simple treatment (for instance, storage, chlorination or sand filtration) delivered to the consumer by a gravity system;

(d) water requiring simple treatment delivered to the consumer by pumping.

Whichever of these is adopted, the natural sources must in any case be adequately protected because of the lack of technical resources, both human and material, in the homes of agricultural workers.

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Figure 3. Domestic filter

A  Candle
B  Cylinder
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1.5. Manure and sewage

1.5.1. Importance

A public sewage system, whether in urban or in rural areas, is theoretically the most hygienic and aesthetic method of disposing of night soil (i.e. human excreta). Although at first sight a public sewage system might seem to be an expensive proposition, one such system for the entire population of a village would perhaps cost no more than a series of individual household sewage systems such as septic tank installations. Moreover, to organise and maintain individual systems requires trained personnel, their construction calls for considerable financial outlay, and expert advice is needed for their installation. Nevertheless, in areas with scattered dwellings, individual sewage systems must be relied on, despite the difficulties of operation and maintenance that they present.

It has been said that 90 per cent of the health problems of agricultural populations centre on the two ends of the human alimentary canal. The active participation of the family, as the most important social unit in the agricultural community, is desirable in any practical sanitation programme of excreta disposal. Health education is one of the most important areas where a public health agency must co-operate with the community. The necessity of a health education programme cannot be overemphasised, nor can the importance of the role of the peripheral multipurpose health worker, who is a link between the agriculturalist and the public health agency. A good sanitary latrine is an excellent advertisement for an effective health education programme. Subsidies from the community or the State should perhaps be encouraged through the state health agency for improving sanitation programmes in developing countries.

1.5.2. The latrine

The personal habits of people away from urban civilisation are varied and often primitive. The most primitive habit, that of de-
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Faecation in open fields, still persists in many parts of the world. In some countries the privies are sometimes placed outside the dwellings, next to the manure platform, and the mixture of human and animal excreta is used directly on the farm as a fertiliser. In other areas, dry human excreta are mixed with peat and, along with sanitary paper, removed to the manure heap.

The spreading of untreated night soil directly on fields or gardens in this way should be discouraged. The high incidence of hookworm disease—ancylostomiasis—in some Asian countries can be directly attributed to the practice of defaecation in open fields where villagers walk barefoot. It is obvious too that vegetables grown in gardens which have been manured with untreated night soil or sewage will almost inevitably be contaminated.

Location

The most important factors affecting the location of latrines are the risk of ground-water contamination and the possibility of faecal pollution. In all instances, technical advice on their location and construction should be obtained from a competent health agency.

In countries where excretion in the open is still practised, the provision of a suitable enclosure to ensure privacy is the primary consideration. In a community which uses water rather than sanitary paper for cleansing purposes, one way of encouraging the inhabitants to use a latrine is to provide water next to the latrine itself.

Types of latrine\footnote{See Wagner and Lanoix: \textit{Excreta disposal} . . ., op. cit., for a more detailed treatment.}

The three basic necessities in the construction of a sanitary latrine are the superstructure to ensure privacy, the receptacle for the excreta and the satisfactory disposal of the waste.

A sanitary latrine is one which will not expose human excreta to flies, and will not lead to soil pollution and water contamination.

\footnote{See Wagner and Lanoix: \textit{Excreta disposal} . . ., op. cit., for a more detailed treatment.}
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In early types of latrine such as the pit privy and the bore-hole latrine, the contents are allowed to remain where they are deposited. The night soil and the cleansing water seep into the ground, and when the pit or bore-hole fills up, usually after a few years, a fresh latrine is constructed. Health hazards to users arise only if the superstructure is neglected, leading to foul odours and fly nuisance. Following recent developments in the construction of latrines, these now have a longer life and the use of sludge or digested solids from night soil as manure has been facilitated.

Figures 4 to 9 illustrate various methods of excreta disposal.

*Pit privy.* Any type of latrine with a pit dug as a receptacle can be called a pit latrine or pit privy. If it is a round and long pit, it is sometimes called a dug-well latrine. In its simplest form, the pit for the use of a small family is usually about 1 m in diameter or about 1 m square, and 2–2.5 m deep; it has a base and a plate for squatting, with a longitudinal slit for droppings; and it has a suitable superstructure for privacy.

*Bore-hole latrine.* The bore-hole latrine, of which various kinds are in use in different parts of the world, is itself a modification of the pit latrine. The hole, about 40 cm in diameter, is bored to a depth of about 6 m by some standard manual or mechanical means. However, bore-hole latrines do present sanitary and health hazards, and expert advice should be sought before they are constructed.

It is not practicable to construct these two types of latrine in waterlogged areas and areas with a high water table because of the risk of flooding of the latrines, difficulties in leaching, and general flooding during monsoon months. In such cases it may be necessary to line the latrines with concrete and cement, even though this can be costly.

*Aqua privy.* In an aqua privy, urine and faeces are dropped into a water-tight tank which stores and decomposes the excreta in the absence of oxygen (i.e. anaerobically) as in a septic tank. It differs from the septic tank as regards the method of entry of the contents,
Figure 4. Pit privy

A Superstructure for privacy
B Squatting plate
C Base
D Surrounding raised surface
E Pit
Figure 5. Water-seal latrine

A Water-seal bowl with S trap
B Water tank filled by hand and provided with plug cock and overflow pipe
C Water pipe leading from tank to bowl for flushing purposes
D Drain pipe leading to seepage pit
E Seepage pit
F Ventilation pipe for pit

A Roof
B Door
C Split bamboo or suitable cover
D Dug well
E 1.5 cm cement plaster over 5 cm thick concrete
F 7.5 cm clay pipe
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Figure 7. Type design for aqua privy

A Mosquito-proof wire-netting cover
B Manhole
C Cement-plastered digestion chambers
D Effluent pipe
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Figure 8. Seepage pit

A Variable depth of soil
B Cement joints
C Open joints
D Rock fill 15 cm or more

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Figure 9. Septic tank and dispersion trench

- Cover
- Vent pipe
- Inlet from privy
- Outlet to distribution box
- Sludge store
- Sludge withdrawal by hydrostatic pressure
- Septic tank

From septic tank

- Open-jointed tiles in dispersion trench

Distribution box and dispersion trench

- Ground
- Hay or straw
- Tar paper
- Gravel
- Burnt clay pipe to a slope of between 1:200 and 1:400

Dispersion trench
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and from the pit latrine in that the sludge is easier to remove. The aqua privy may be located above ground level or partly above and partly below. The contents of the tank have no contact with the ground. Unlike the septic tank, the aqua privy does not require much water; but each day a quantity equivalent to that of the added cleansing water has to be evacuated from it, and therefore provision must be made for a means of draining off effluent. This effluent should not be allowed to run into open fields or gardens. Aqua privies may be recommended whenever the supply of water is limited, although they do not always work satisfactorily. Mosquitoes (for instance *Culex* species, which is responsible for the transmission of filariasis) have been known to breed in the vicinity of this type of latrine.

*Cesspool and seepage pit.* Cesspools are either water-tight or leaching, and receive only raw sewage. They may be found in some agricultural areas. The seepage pit receives the effluent from aqua privies, cesspools and septic tanks, and allows it to percolate into the surrounding ground, prepared for the purpose by embedding large stones or rocks in porous soil. It is a pit made with a cement-jointed lining at the top. It is loosely jointed throughout so that the liquid wastes may seep through. There is always a danger of ground water pollution with these seepage pits.

*Septic tank.* The septic tank may be recommended not only for excreta but also for liquid wastes from isolated dwellings in rural areas. The amount of available water determines the efficiency of the septic tank. The tank is actually a settling tank and a digestion tank combined. The sewage solids settle down, and the settled sludge undergoes anaerobic digestion with the help of bacteria and fungi. The effluent from the septic tank may be disposed of in numerous ways, depending on the proximity of the tank to drinking-water sources which may be contaminated, the porosity of the soil and the subsoil water level in relation to the ground level. Competent scientific advice should be sought before any effluent disposal system is constructed.
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The sludge from the septic tank must be removed periodically. The contents may be mixed with refuse and straw and the material allowed to become compost under earth cover. Such compost, after maturation, contains adequate amounts of nitrogen, phosphorus and organic matter for use as fertiliser.

1.5.3. Disposal of solid wastes: composting

The decomposition of organic matter is a natural phenomenon. When a dead animal is buried deep without access to atmospheric oxygen, it decomposes under anaerobic conditions, and foul-smelling gases (e.g. carbon disulphide and hydrogen sulphide) are generated. However, when animal excreta are deposited on the surface with access to adequate oxygen, relatively stable humus is evolved aerobically through decomposition. When man helps nature to carry out this biological decomposition—either anaerobic or aerobic—he is said to be "composting". Composting destroys disease-producing organisms and the resulting compost can be used on the fields with few health hazards. Zoonotic organisms excreted in animal faeces may include those causing undulant fever (Brucella) or Q fever (Coxiella). There are many in human night soil (e.g. bacteria causing bacillary dysentery, cholera or the typhoid group of diseases; protozoa causing amoebic dysentery or balantidial diarrhoea, ova of numerous intestinal worms such as the roundworm and the threadworm).

Aerobic methods

Anaerobic composting techniques do not produce as much heat as aerobic composting techniques, which generate sufficiently high temperatures to kill parasites and pathogenic micro-organisms.

Night soil, animal manure, sewage sludge and garbage are readily putrescible. Less putrescible and relatively more stable materials such as leaves and straw are also available on the farm. Alternate layers of these two varieties of refuse, piled up and turned over periodically according to a schedule, should yield a compost which is practically innocuous and may be used as a fertiliser (see figure 10).
Two undesirable health hazards are associated with composting operations: flies, and hazards to the operator. Flies find excellent feeding in manure, human excreta and garbage, and compost pits will nearly always add to the fly nuisance unless good practices are followed.

The high temperature reached in aerobic digestion is the keystone of the composting process. A period of two to three weeks is required for the completion of the process, depending on the com-
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position of the ingredients, the efficiency with which high temperatures are maintained and the possible intensification of aerobic decomposition by mixing the compost daily.

Rapid composting techniques have been developed in recent years. For example, in many countries, quantities of waste organic substances (hay, straw, etc.) can be converted into high-grade organic fertiliser in as little as a week by enclosing the waste materials in a special “digester”.

Anaerobic digestion

It is possible to produce fuel gas (bio-gas) through the biological decomposition of organic waste material such as farm manure, night soil, litter and garbage. Since the Second World War, many countries have developed anaerobic digesters for such materials. A gas-holder may be added to the digester to retain the digested gas mixture. The gas can be used for heating, cooking, lighting and refrigeration, and sometimes for providing power for small engines on the farm (see figure 11).

Other anaerobic methods of decomposition of farm wastes are known; however, as they are less feasible than aerobic methods or digestion for methane recovery, they are not usually recommended.

1.6. Environmental health

“Environment” is a general term which covers many factors known to affect the health of man, and perhaps many more of which our knowledge is inadequate. Our scientific knowledge of the factors promoting health is not as advanced as our understanding of those contributing to disease. The scientist theorises, conducts experiments based on specific epidemiological clues, seeks to understand the causative agents in the environment and studies the relationship between health or disease in man and other animals and the environment.

Environmental factors are numerous and all-encompassing: the climate we live in, the air we breathe, the food and water we con-
sume, the plants and animals around us, the vast unseen world of microscopic life. Practitioners of the science of environmental sanitation, which promotes environmental health, try to influence as many of these factors as possible, but in many cases can only make a few practical suggestions to enable man to live more comfortably and with less disease. Adverse elements in the environment
1. Living conditions and environmental hygiene

should be minimised in quantity and potency so that man is less likely to succumb to disease.

Environmental factors may be chemical, physical or biological. Before what has been called the "green revolution", chemical factors were not an important problem in rural areas. However, recent developments in agricultural technology, and specifically the increasing use of pesticides and artificial fertilisers, are bringing in their wake new hazards which are all the more serious because they are unfamiliar to those living in the less developed regions of the world.

Physical factors are mainly influenced by man's geographical habitat. He attempts, through housing, to protect himself against adverse climatic conditions (for example, excess sunshine and rain) and thereby creates an artificial climate of his own. At home, he screens himself as far as possible from rodents, insects and other pests which are both a nuisance and contribute to the transmission of disease. The ecology of his environment is very complicated, and man can influence life around him only to the extent of his resources.

In a rural environment man must perforce live close to nature, and the nature of his occupation exposes the agricultural worker to the biological hazards in the environment. He may contract diseases caused by viruses, rickettsiae, bacteria, fungi or parasitic agents. The Joint ILO/WHO Committee on Occupational Health\(^1\) lists a total of 45 diseases contracted through work in agriculture, including viral encephalitis, Q fever, anthrax, brucellosis, erysipelas, glanders, melioidosis, leptospirosis, tetanus, bovine tuberculosis, tularaemia and ancylostomiasis. Among the 45 diseases are a number that are occasionally contracted through agricultural activities, for example ornithosis from birds and contagious ecthyma from sheep, and others that may possibly be contracted

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through them, such as cowpox from cows and actinomycosis from swine and horses. Further information on these diseases is given in Chapter 5.

1.6.1. Man-made problems

As man strives to improve his condition, the normal balance in nature between life and environment may be disturbed and new problems created. Man-made malaria and the rise of bilharziasis following the establishment of irrigation farming are good examples. Malaria already appears to be a disease of the past, thanks to world-wide control programmes; however, schistosomiasis seems likely to become a future scourge mainly affecting agricultural populations, and can be taken as a good example of a man-made problem.

1.6.2. Plants

Airborne pollens are perhaps one of the commonest causes of allergy in village populations. For example, experience has shown that ragweed is an important factor in the incidence of allergy and hay fever. It is, however, difficult for a farmer to estimate the ragweed pollen content in the air. Agricultural workers in general should be taught how to identify and eliminate both this plant and some of the poisonous plants (for example, poison ivy) or fungi (for example, poisonous mushrooms). Many other plants or weeds, as well as wood or hay, may be skin irritants. On repeated contact they may sensitise the skin, or prolonged aerosol inhalation may lead to respiratory insufficiencies.

1.6.3. Animal life

Insects

The fly. Although the common house-fly does not bite, it is annoying and obnoxious. Because of its habit of feeding indiscriminately at both the dining table and the sewage pit, there is every
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chance that it will carry filth on or within its hairy body and transfer disease-producing germs (of which *Vibrio cholerae* and *Salmonella typhi* are examples) to food and water. Some bacteria can even thrive in the intestines of the fly. When it feeds at the dining table, the fly can discharge through either end of its alimentary canal matter ("fly specks" and "vomit spots") which may include live tubercle bacilli (*Mycobacterium tuberculosis*). Some common species of fly may feed on live flesh, either of farm animals or through skin wounds or ulcerations of the subcutaneous tissues of human beings, which become infested by larvae (myasis). The tsetse fly, with its association with African sleeping sickness, is a good example.

*The mosquito.* There are a number of mosquitoes carrying a variety of diseases, but it takes a trained public health team to identify, locate and eliminate or control either the mosquito or the disease. The farmer, however, can help himself and his community to stamp out both the diseases and the mosquitoes by cooperating with the health agency. After the eggs are laid, about a week elapses before the young mosquito can use its wings. Once it starts flying, it is difficult to control. Usually the mosquito cannot fly for more than a kilometre or so, but strong winds may carry it much further. Eradication is far easier, therefore, if the farmer tackles the pest during that week of early development. Depending on the species, the female mosquito lays its eggs sometimes in a mass, sometimes singly, but always near or on water. The *Aëdes* mosquito, a vector of dengue and yellow fever, lays its eggs on wet surfaces near bodies of water. The *Anopheles* mosquito, a vector of malaria, lays its eggs in comparatively unpolluted water in ponds, lakes and swamps with growing vegetation. On the other hand the *Culex* mosquito, a vector of filariasis, lays its eggs in artificial containers and ground pools and prefers places contaminated by sewage. Eggs develop into larvae in a couple of days. Only in extremely cold conditions and only in the case of marsh mosquitoes can mosquito eggs stay alive for much longer periods. Larvae and pupae of all types of mosquito are aquatic, and hence
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the farmer can always help to control the mosquito population by examining bodies of water around the farm and by initiating a weekly inspection system for breaking the life cycle. The female mosquito has an instinct for laying eggs in, for example, sagging roof gutters, flower vases, discarded tins and motor car tyres, chicken or animal water troughs and, very commonly, open shallow wells and rainwater barrels. Such breeding spots can easily be done away with or mended.

The poor maintenance of irrigation ditches is an important cause of the increased incidence of mosquito-borne diseases. Water-logged soil with excess water in low areas, standing pools formed by overflows from irrigated lands and leaky ditches are the most common faults seen in irrigated areas.

Mosquito breeding may also be controlled through the use of chemical insecticides, and the usefulness of pisciculture should not be overlooked. The farmer can easily select the local indigenous larvicidal fish and introduce them in the water around the farm. Gambusia fish are very effective in that they breed fast and have a selective appetite for the mosquito larvae. This method alone, however, should not be relied on in areas where the mosquito population is dense or where mosquito-borne diseases are endemic. In such areas, and where control measures in the field are inadequate, the farmer must think in terms of adult mosquito control in his own home. He may, for instance, screen the houses with metal or plastic material with an appropriate size of mesh. If this is not possible, he should at least consider fitting mosquito netting over the beds in a house to reduce the period of contact with the mosquito. In tropical countries, however, the net may be thought to hinder ventilation.

Worms

Worm infestation is common in agricultural populations. Hookworm is an example of a worm affecting adults and children alike. This is a world-wide disease, especially in warm and subtropical climates. Though a person harbouring a few worms (i.e. fewer than 100) does not necessarily become ill himself, he may become
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a carrier, spreading the disease to the community. At one time the disease was thought to be spread only by the infective larvae, which can remain alive up to three months in moist soil and bore their way at the earliest opportunity through the soft skin between the toes of barefoot persons. It is now known, however, that larvae may also penetrate through the skin of other parts of the body—for example, the hands and the arms or perhaps even through the mouth—in order to reach their ultimate destination: the human intestines. Control measures include treatment of the infected individual and sanitary disposal of faecal waste. As a supplementary measure, agricultural workers may be recommended always to wear footwear. Reinfection is possible unless a continuing health education programme is in operation.

Rodents

Rodents are troublesome, and moreover constitute a danger to the health of agricultural workers. For centuries, villages were regularly decimated by plague conveyed by the rat flea. In recent years, as a result of the increase in the rat population, rat-bite fever has assumed endemic proportions. While different species of rodents are found in different parts of the world, they share the common characteristic of rapid breeding. They produce a litter every two to three months, and thrive on garbage, vegetables, meat and stored grain. Their omnivorous habits are a menace to the farmer and of great economic importance in all countries; reports indicate that upwards of 40 per cent of the harvest may be lost to these rodents. This is catastrophic in a developing country which is already suffering from a shortage of food.

Attempts to keep rodents in check by using their natural enemies, such as cats and dogs, are generally not successful, because domestic pets are usually too well fed to fight rats aggressively.

The use of poisons (phosphorus and thallium compounds) to control rodents also involves a degree of risk to human beings, since these poisons are toxic to man as well. Modern technology has produced chemicals such as warfarin and pindone, which are liked by the rats and which, being anticoagulants, kill them by
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causing internal bleeding. The use of anticoagulant poisons is now discouraged in some developed countries, in favour of the so-called “humane” poisons. In some countries, rodents have begun to display resistance to warfarin. The elimination of rats by trapping, either independently or soon after a poisoning campaign, is also recommended on the farm. The simultaneous use of a large number of “snap” or “guillotine” traps in a methodical campaign can be very effective indeed.

Dead animals

The disposal of farm animal carcasses, small or large, is a matter in which the farmer has to be educated. It is recommended that an isolated village plot be reserved as a burial ground for the use of all farmers, as is done in some Asian countries. In some communities where human bodies are buried and not cremated, a site adjoining the human burial ground may be specifically assigned for the burial of dead animals in the village. Burial at a minimum depth of 1 m would reduce the risks to health. However, if the farmer has to use his own property and if it is extensive enough, the burial of accumulated ashes and non-combustible material in a distant corner can be combined with the occasional burial of farm animals which cannot be disposed of otherwise.

Animals which die of certain infectious diseases, such as anthrax, pose a special problem. They should be buried where they are found.

1.7. Food sanitation

The sanitary treatment of food and water is one of the most essential measures in maintaining the health of the agricultural community. Unlike the situation in water sanitation, the influencing factors in food sanitation are mostly related to the home, and can therefore be controlled with greater effectiveness. Generally, food is handled by housewives, and in some instances by farm servants.
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An effective health education programme for sanitary food handling is therefore warranted.

1.7.1. Sources of infection

There are many possible sources of infection when food is prepared or served. Food sanitation can be examined under two headings: prevention of disease due to infection, and prevention of disease due to chemical contamination. Modern food preservatives, insecticides and pesticides used on agricultural produce, chemical fertilisers and radioactive fall-out have all been known to contaminate foodstuffs, to the detriment of human health and welfare. Chemical contamination is dealt with elsewhere in this guide; food infection will be discussed in some detail here.

Environmental factors

Infection is transmitted through physical agents or, less commonly, directly from person to person. Insects, chiefly flies, also play a major part in carrying disease from person to person. Flies are known to transmit infection either mechanically through the hairy parts of their body or occasionally through their alimentary tract. They make a common practice not only of defaecating at the place of feeding but also of regurgitating their last meal over the next one. The evidence as regards other insects, such as cockroaches, is not so clear, but they can be regarded as being more than a mere nuisance.

Rodents (rats, mice, bandicoots, and so on) abound on the farm, where there is plenty of food for them, and are a common nuisance. They themselves can be reservoirs of infection—for example, epidemics of bubonic plague have been traced to infected rat fleas. Rat-bite fever is an equally severe infection that can break out in epidemic form. The rat transmits a microbial infection of Spirillum minus through its saliva.

As with most household pests, feeding is accompanied by defaecation and urination, with the consequent risk of salmonella or leptospiiral infection. Salmonella infection leads to outbreaks of
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severe intestinal upsets. Leptospiral infection causes severe damage to the liver, leading to jaundice with fever. Leptospiroa are among those microbes that can enter the human body through unbroken mucous membranes or skin. Not only do leptospiroa spread infection through drinking water or food, so causing leptospiroal jaundice, but skin contact with water contaminated with rodent excreta has also been known to cause outbreaks of epedemics of leptospirosis, for example among sewage workers in cities and among sugar-cane plantation workers.

The quality of water used for cleansing purposes determines the standard of food sanitation. Even if the quality of the water is satisfactory, if too little water is used for cleansing purposes food is left on the utensils, and bacteria multiply rapidly. In fact, in modern public health practice the sanitary quality of a public eating-place is determined by the bacterial counts taken from the utensils used in these places. The materials of which eating utensils are made is also an important factor, particularly with respect to the storage of acid foods in utensils made of copper or lead and their alloys.

A less common path of infection is through visitors to agricultural homes. If the cutlery or crockery they use is not cleansed and disinfected properly, it may harbour residual infections from their mouths or respiratory tracts, with the risk that infection may be transmitted to the next users.

The places where food is prepared and served should invariably be kept clean. Dust can be considered to be synonymous with germs, and germs carry disease. The concentration of dust is usually very high at floor level, and decreases with height. It is therefore commonly recommended that food preparation or service should take place not at ground level but on a raised platform. In Asia, however, a common practice in the kitchen as well as in the dining-room is to prepare and serve food at floor level. This is to be discouraged. Moreover, a kitchen stove or a cooking fire at ground level represents a fire hazard to the housewife and others moving around the kitchen.

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Personal factors

An important aspect of food sanitation, and one which requires constant attention, is the health and personal habits of persons engaged in the preparation and service of food. These people may spread infection very innocently through the upper respiratory tract, by speaking loudly or coughing or sneezing near the food-stuffs. Handlers who are not necessarily suffering from any respiratory disease may still be harbouring pathogenic germs to which they may be immune, but which may lead to the spread of infection to others who may be less immune to the same germs. This immunity in food handlers, if due to a previous attack of illness, can be a reason for not re-employing them during their convalescence from the disease. It should be borne in mind, however, that even without an acute attack of illness, food handlers may be carrying germs in their upper respiratory tract or mouth after a subacute or abortive attack of asymptomatic illness. The food handlers themselves will not be aware of this illness, and at times they will remain infectious for prolonged periods.

Bacterial and parasitic infections of the gastro-intestinal tract can also be transmitted through the fingers, more commonly by food handlers and less commonly by visitors. This is due to dirty habits (biting nails, picking noses, poor personal hygiene after a visit to toilets, and so on). Poor cleansing after defaecation is probably the cause of transmission of gastro-intestinal diseases. Personal habits differ in different parts of the world. The cleanest procedure, some claim, is cleansing with plenty of water; but it has to be remembered that in such a case infection is only diluted through water. Therefore, washing the hands a second time, preferably with soap and water, is recommended.

1.7.2. The dairy farm

The proper handling of milk on the farm can be taken as an example to illustrate the application of food sanitation.

Infection may be transmitted in numerous ways. The cow may
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be suffering from contagious abortion, and the causative organism, *Brucella abortus*, passes through its milk and causes undulant fever in human beings. Buffaloes in the tropics are in the habit of cooling themselves in muddy waters. These waters are likely to be contaminated with sewage, and infectious material from such waters might lodge on the teats and udders. Tubercular ulcers may be found on the teats and udders of cattle. Tubercle bacilli may also be found in the faeces of infected cows and, under insanitary conditions, may find their way into the milk. Unclean water, if used to wash the udders and teats before milking, as well as the milk receptacles, is yet another source of infection.

The infection may be passed on to the milk by the farm employees themselves. If the milker or dairy employee handling the milk has poor habits of personal hygiene, he may contaminate milk with faecal organisms. Even apparently healthy looking individuals milking cattle or handling milk might inadvertently spread droplet infection by spitting, sneezing, coughing or shouting. Streptococci and staphylococci of human origin have been known to grow on the teats of udders.

Clearly, therefore, a good health education programme aimed at the rural community is an important task for the local public health agency to undertake. The programme should deal with the care of cattle and their surroundings and the sanitary collection, storage and distribution of milk.

1.7.3. Milk sanitation

In milk sanitation, the important factors are healthy workmen; utensils and equipment of sanitary design for routine and effective cleansing and periodic sterilisation; and suitable processing and handling units for cooling, pasteurisation and bottling. Progressive farmers look upon milking machines as a means of eliminating many of the usual problems, but it must also be remembered that in order to maintain these machines in a sanitary state the recommended procedures for cleaning must be strictly followed. In their education programmes, local health agencies can put to good use
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the care and intelligence traditionally attributed to the farmer, in order to help to maintain high standards of milk sanitation.

In countries where co-operative systems or communes exist, the health consciousness of the individual farmer and processor is of greater importance, since infectious agents in a distribution system would be a potential threat to a large population. In dairy cooperatives, not only milk but also many milk products are processed as a home or cottage industry. Hard or soft cheese, cream or butter, condensed or powdered milk, milk concentrates or milk beverages, curds or buttermilk—these are some common examples of milk products the sanitary standards of which depend ultimately on health consciousness at the dairy farm and at the health agency which is responsible for the safety of food products.

All food handlers, like restaurant attendants in towns or cities or dairymen in rural areas, must be educated in personal hygiene. Their health care must be ensured and, as potential carriers of disease, they should be periodically inspected by local health agencies. But inspection services on the farm can never be as wide-ranging as those in urban areas, and hence greater emphasis must be placed on health education. Constant re-education is necessary, and no opportunity should be given for back-sliding.

Dairy workers should take care to keep their hands and clothing clean while at work, and most of all when they come into direct or indirect contact with milk. A reliable disinfectant should be kept ready to disinfect workers’ hands, the teats and udders of cattle and also the inside surfaces of utensils.

Unless the farmer is using primitive methods and the milk produced is for use at the farm only, some kind of milking equipment is generally in use. This should always be cleaned before it is used again, and preferably immediately after use. The maximum reduction in bacterial counts can be obtained only if the dairy worker devotes time and effort to keeping the equipment clean.
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1.8. Other problems of agricultural life

1.8.1. Some special problems

At one time, agricultural life in all parts of the world was equally primitive and remote from the influence of civilisation. With today's rapidly changing social structure, however, new problems arise—not only inside the village but also outside it. Townships may spring up close to villages, social communication may increase between the agricultural and urban populations, or some rural populations may themselves become urbanised. The reclamation and settlement of arid and humid regions may create favourable environments for new vectors of disease. Social changes of this kind have always had an impact on the health of the agricultural population. The rapid spread of infectious illness such as pulmonary tuberculosis and venereal diseases in previously disease-free rural communities soon after the introduction of social changes is a phenomenon of epidemiological importance.

The mental health problems that arise from the social impact of industrialisation on agricultural communities are also of far-reaching importance and call for special study.

The general improvement in environmental sanitation and conditions of hygiene around the farm leads directly to a decrease in specific and general morbidity. There are very important indirect results as well.

A state of well-being changes the farmer's attitude to life and is conducive to better social development. More modern practices and time-saving devices help to improve the agricultural economy. The control of disease leads to longer life and hence to problems of old age. In promoting the total health and welfare of the agricultural population, therefore, all these aspects have to be borne in mind: suitable additional occupations have to be introduced to make use of the extra man-hours at the disposal of the able-bodied, and rehabilitation programmes have to be organised for the growing numbers of old people.
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Some agricultural processes

It should be clear by now that agricultural operations are as varied as industrial operations and are equally difficult to classify. Different practices exist in different countries, and the stresses and strains produced by a process on one agricultural worker may differ from those affecting another. The use of agricultural machinery brings new problems in its train. Similarly, the use of pesticides and fertilisers creates various toxicological problems.

A factor which perhaps needs to be stressed here is that the performance of some agricultural operations requires increased food intake. In rice cultivation, for example, it has been shown that the energy demands of certain operations are more severe than in some heavy industrial operations. Indeed, energy expenditures as high as 300 kcal/h per square metre of body surface have been encountered.

1.8.2. Health legislation

Agriculture is literally "big business", by reason of the large amount of human "capital" that it employs. It is estimated that some 44 per cent of the world’s economically active population is employed on farms. The farms on which these people work vary widely in size, from small fragmented farms in highly populous countries to vast sheep farms extending over many hundreds of square kilometres. They all have certain features in common which have a bearing on the health of the population.

In many ways, farmers stand in marked contrast to townsfolk and factory workers. They do not live in compact homogeneous societies but are scattered far and wide. The results of social progress reach the remote villager much later than his urban counterpart, if at all. Similarly, the benefits of social administration are much less easily available to him. The International Labour Organisation has, since its foundation, realised the importance of the farmer in matters of international health, and a dozen international labour Conventions and even more Recommendations, aiming at
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improving the welfare of the agricultural population in a variety of ways, have so far been adopted. They deal not only with the head of the household (usually a male member) and with male agricultural workers but also with women and children employed on the farm, whether for remuneration or not; and they cover topics such as maternity protection and the age of admission of children to agricultural employment. Many countries have ratified these Conventions but only a few have been able to implement them satisfactorily. The main reason for this is that it has been found difficult to apply government administrative procedures, including legislative control, implementation and inspection services, to the rural population.

Legislation has therefore only a limited application to rural communities. Consequently, considerable reliance must be placed upon disseminating up-to-date knowledge on the scientific aspects of personal and community health. There are two commonly accepted approaches here: first, providing continuous (recurrent) education through whatever channels of communication may be available in rural areas, where educational levels are comparatively low; and second, encouraging the farmer himself to participate actively in the definition and solution of his own health problems.

The universal medium of communication—the spoken word—can be suitably adapted to have its maximum effect in the rural community. All the known aids to the spoken word can be used: demonstrations and models; still pictures and filmstrips; mobile pictures and flannelgraph techniques; films and television; drama and story-telling. After a preliminary interview with the village elders, suitable talks or lectures may be arranged, followed by group discussion. Successful group discussion can be a most effective way of changing, when necessary, the customary pattern of life of the villager, bound as he is by deep-rooted traditions, beliefs and cultural patterns.
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1.8.3. Examples of effective health education through community participation

An example of the effectiveness of community participation in rural areas is given in a report by the WHO Expert Committee on Health Education of the Public:

Beginning with the demand of the people for treatment of their skin diseases, a cleanliness campaign was carried out by the villagers, and then a welfare centre established. A villager was then trained for visiting the homes and looking after the welfare of the children. Later, gardens were started, to produce vegetables needed to enrich the diet. A desire for soap led to the setting up of a co-operative store, and trainees were taught to weigh goods, to serve, and to count, the village leaders themselves running the shop. A fire in the village caused great co-operative effort and led to plans for building new homes, a co-operative cattle-shed, a craftwork centre, and a village crèche.¹

A more specific example can be given from Indian experience with the disease dracontiasis, or guinea-worm disease. This illustrates how a change in the simple, customary (though insanitary) practice of drawing water from a “step-well”—a shallow-surface water source with steps leading into it—has virtually resulted in the local eradication of an endemic, centuries-old menace. Once the villagers had been convinced of the cause-and-effect relationship of an unhealthy practice and the symptomatology of a disease that they were accustomed to seeing daily, they took action of their own and helped the public health agency effectively to control the disease.

A number of rural areas in Asia and South America have localised, highly endemic foci of infection by this worm. The adult worm is harboured in the host tissues, and its larvae escape from the human host when the viviparous adult female migrates to deposit her larvae through a small and usually insignificant blister on the skin of the host. When a person walks through the water in

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open wells, step-wells, ponds or the like, the deposited larvae from the ruptured blister reach the confined water. The larvae are ingested by small water insects (*Cyclops* species) and develop in them to the infective form. Human infection follows the drinking of water containing infected *Cyclops*, the larvae being liberated and activated when the *Cyclops* are killed by gastric hydrochloric acid. In severe cases of this disease, signs such as urticaria, vomiting or diarrhoea may develop at about the time of blister formation, and if by any chance a worm is injured in the subcutaneous tissues, severe cellulitis develops. Environmental control of this worm includes treatment of the water with copper sulphate or bleaching powder to kill the intermediary host, or biological control through pisciculture of small fish which feed on *Cyclops*. But even without these environmental sanitation measures, action by the villagers through simple health education programmes has eliminated the disease from some remote villages. On becoming convinced that drinking the water from the age-old step-well was dangerous, the village community took self-help action by merely changing their method of water collection and boiling all drinking water after simple cloth filtration.

This example indicates that the local health agencies must study the cultural patterns of rural communities and their age-old habits in detail before launching a health education programme aimed at controlling specific communicable diseases. Furthermore, farmers will become more health-conscious when they see for themselves such dramatic improvements in community health.

In previous sections, various ways have been outlined in which the farmer and his family can improve their environment and state of health through education and action. Depending on the local needs and the socio-economic living standards of the communities, the local health agency has the responsibility of defining the priorities in health programmes and using suitable methods of health education to create community interest (and ultimately community participation) through voluntary leaders and socially minded members of the community.
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1.8.4. Health services

The organisation of general health measures in agricultural populations is beset with difficulties. Whether they deal with medical examination or health counselling, the maintenance of statistical records or emergency programmes, the supervision of working environments or other measures for health promotion, all local health services are more effective when they are combined with private agencies. The protection and promotion of the agricultural worker's health can be achieved efficiently and economically only when all the plans, including special services for occupational health, are integrated into the existing health programmes in the community.

In the organisation of such services, rural health units have proved successful in many countries. The health unit is an organised body under the direct supervision of a doctor or a competent paramedical worker at the health centre. The rural health centre is a conveniently located place where the community has access to all basic services, such as environmental sanitation and communicable disease control, medical care and nursing, health education, and care of the mother and child.
2. Problems of occupational physiology and ergonomics

2.1. General principles of occupational physiology

Before we attempt to assess the effect of work on the health of man, it is expedient to summarise briefly our knowledge of the human body and the working of its organs. Of course, all the organs of the body take part in the life, and hence in the work, of man. However, certain parts of the body that determine working capacity are more particularly involved; they can therefore be considered as representative, and only they need be studied.

2.1.1. Muscular work

Muscles

All physical work is done by muscles, in which the necessary energy is created. The first task of the muscles is to maintain the body in the requisite posture (upright, seated, kneeling, and so on) and then to effect the various movements. It is through them that useful work is finally done.

Muscles work by alternating contraction and relaxation of the component fibres, resulting from chemical action. Muscle fibres, which are mostly arranged in groups or bundles in different parts of the body, cause various movements by acting on the bones. Muscles also cause movements in internal organs.

The energy required to contract the muscle fibres is provided by the oxidisation of glucides. The combustion residues include lactic acid, water and carbon dioxide. Since the chemical reactions take place within the fibres themselves, it becomes necessary for the oxygen and the fuel to be brought to these fibres and for the waste products of combustion to be removed, either to be discharged outside or to be reintroduced in a regeneration cycle. The working capacity of muscle therefore depends on the number
of fibres (musculature), the capacity of the transport routes (arteries and veins), the speed of the transport (blood flow), the functioning of the regulatory system which has to harmonise the physiological phenomena with the effort exerted, and the pulmonary function which ensures the renewal of the oxygen in the blood and the elimination of gaseous waste.

Bones and joints

To effect movements of the body, muscles require a firm anchorage; bones may therefore be considered as being practically rigid. To a certain extent they are also elastic, especially in young persons. This elasticity, however, does not play any part in work: it is needed to take the strain of heavy loads. If the elasticity is insufficient, as is often the case in accidents, a bone will break. Most bones in the body are connected by joints (such as the knee, the hip and the elbow), or they are semi-rigidly connected by ligaments or cartilage (as the ribs are to the upper part of the spinal column), or they are fastened together like the bones of the skull, whose purpose is to protect the brain. The spinal column has quite a special structure. The vertebrae are so shaped that the upper part of the body can assume the most widely differing positions in relation to the lower part, and it can also rotate independently. There is a special reason for this structure, in that the spinal column protects the abdominal organs. Since it can only move by arching and cannot bend like the knee, these organs always have enough room. It does happen that in certain positions of the torso some organs are slightly compressed, but their functions are only very slightly impaired on this account. In order to leave the organs with the space that they require and to maintain the torso in a suitable position, the vertebrae are connected by joints that are only slightly mobile and by ligaments called meniscuses or intervertebral discs. Because of their inelasticity, the meniscuses are very sensitive to repeated jolts, such as those caused by the bumping of a vehicle that has neither springs nor shock absorbers.

The spinal column protects only the hinder part of the abdomen;
the rest is protected by a wall of ligaments and muscles. The muscles are superimposed and the fibres are criss-crossed so as to constitute an envelope that is both elastic and strong. This enables the body to bend forwards and sideways, and hence allows it to work in a bent position. When the abdominal wall is overloaded, especially when heavy weights are lifted, the weakest points may give way and this may lead to hernias.

The thorax, which can move only a little, protects not only the vital organs—the heart and the lungs—but also the top of the stomach, the liver, the gall bladder and the spleen. The vertebrae are connected by muscles and ligaments, and the shoulder-bones and collar-bones are connected to the chest by the same means. The back muscles play an important part in maintaining the position of the body. The less they are developed, the more the vertebrae are pressed together, and consequently the greater the risk of deformation of the spinal column. The back muscles are also needed to compensate for the efforts made when the arms are working.

The strength of bones is invariable over a good part of a person’s life span, and it is wrong to suppose, as often happens, that because old people are particularly subject to bone fractures they have soft and weak bones. In fact, a predisposition to bone fractures is the result of poor musculature that is no longer able to hold the bones together adequately, coupled with vagueness and lack of co-ordination of movements.

2.1.2. Circulation of the blood and respiration during work

One of the principal determinants of the power of muscles is the amount of blood flowing through them. The total amount of blood may be considered as a personal constant because normally it is subject to only slight variations. The amount of red pigment (haemoglobin) determines the amount of oxygen that can be fixed in the blood. The velocity of the bloodstream and the volume of the vessels (arteries and veins) govern the quantity of oxygen available in muscles. The blood is moved by the heart, first through
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the lungs where it fixes the oxygen, and then through the muscles and the organs where part of the oxygen is consumed. From these it returns to the heart and lungs. It is not only the size of the heart but also, and directly, the rate at which it beats (pulse) that determines the blood flow. Hence, measurement of the rate of beating or pulse is of great importance in assessing the strength required to perform a given job. Naturally, the amount of oxygen consumed is directly proportionate to the muscular energy produced. It is, however, more difficult to measure this than the heart rate. All that is usually done is to compare the oxygen content of the inhaled and exhaled air, whereas at the same time it is necessary to measure the respiration, which is proportionate to the effort expended.

During inhalation the lungs fill with fresh air, rich in oxygen; this air passes through the membranes of the alveoli of the lungs, enters the blood stream and is fixed by the haemoglobin. Each cell can fix and transport only a certain amount of oxygen. Muscular work depends on the amount of oxygen that the blood can convey to the muscles; similarly, the rate of elimination of carbon dioxide depends on the blood flow through the body. Consequently, good circulation and respiration are of essential importance to working capacity.

The heavier the demands made on muscle power, the faster the blood must flow and the faster the man must breathe, because the concentrations of energising substances and cells transporting oxygen are almost constant. When the composition of the blood is normal, the amount of oxygen that it transports is sufficient for combustion. However, if it is too poor in haemoglobin the oxygen flow is insufficient and the muscle cannot do as much work; if, in such a case, it is desired to intensify muscular work, the circulation of the blood must be accelerated and the shortage of haemoglobin must be compensated by an acceleration of the rate of oxygen exchange in each muscle. In a person whose blood is poor in haemoglobin, the amount of muscular work will be less than in a person whose blood contains a normal amount of haemoglobin. The requirements of a muscle considered in isolation naturally depend on the work it has to do.
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During periods of rest, at each heart beat the pumped blood is distributed among the different organs in accordance with a fixed pattern. During work, an additional flow of blood must irrigate the regions producing the energy so as to feed the muscles and remove the waste products. The regulatory mechanism of the circulatory system works with extraordinary precision and sensitivity. Its reaction to change must be almost instantaneous, because the movements of the body alter very quickly. One needs only to think, for example, how quickly the different muscles of the legs act in turn when one is walking; and the same, of course, applies to the muscles of the arms and legs, or the hands and arms, in other movements. The blood flow per minute is regulated by the rate at which the heart pumps; this can be measured by the pulse, and is only slightly influenced by variations in the size of the heart. The quantity of blood flowing towards any particular part of the body depends on the cross-section of the blood vessels concerned. The blood arrives through the arteries and leaves through the veins. When the blood supply to a certain region has to increase, the arteries carrying it have to dilate in order to increase their carrying capacity, as do the veins when the blood is returning to the heart.

When certain regions of the body need intense irrigation, the arteries and veins contract in other regions, in which irrigation is thereby reduced so that the circulation can meet the increased needs of the first-mentioned regions. It is true that the vital organs continue to receive just enough blood to enable them to function, but in these conditions they are not particularly active. It is, however, very important to maintain the irrigation of the brain. Of course, the amount of blood required there is small in comparison with that required by the large muscles, such as those of the thighs when they are working at full capacity. But the brain needs blood that is rich in oxygen, and its activity declines when the demand for blood becomes too high in other parts of the body. Naturally, other organs, such as those of the digestive system, may compete with the muscles for blood supply. After meals the digestive system needs a large quantity of blood, not only to make its own muscles
work but also to transport and distribute the products of digestion. Man should therefore stop working during and immediately after meals so as not to hinder the working of the digestive tract. This is also the reason why the zeal for work declines even before the meal break. This changing blood distribution between organs and muscles is also due to the regulation of circulation. The system is so responsive to the variations with which it has to deal that it can work for several decades without breaking down. Thus, working capacity depends also on the proper working of the regulatory mechanism of the circulation.

2.1.3. Basal metabolism

A minimum expenditure of energy is always required, independently of any activity, and even during sleep. This is "basal metabolism". It is the minimum energy exchange that is essential for the maintenance of life. Basal metabolism (measured in calories) depends on the weight of the body and its surface area (temperature regulation) and varies slightly with sex and age. Such determinations of basal metabolism as may be necessary for medical reasons or for work study must be carried out in a specially equipped laboratory. For routine purposes it is considered sufficient to use the values given in tables.

2.1.4. Static work

So far we have spoken only of dynamic muscular work—that is, work done by movements of the body.

There is, however, another kind of work: static work, or the work of maintaining a position. Such work entails constant effort by the muscles that maintain certain parts of the body in particular positions (crouching, kneeling, sitting, and so on). Carrying loads on outstretched arms or on the head are examples of static work. If the body is to maintain a certain posture, the first requirement is that the head is in such a position that the functioning of the brain is not hampered. Second, the posture should be such that
the reactions of dynamic work (for instance, when walking or making tractive effort) can be absorbed without loss of balance.

As we saw above, muscles work by alternating contraction and relaxation of their component fibres. However, the work of maintaining a position cannot be accomplished by continuous contraction, since muscular relaxation is indispensable to irrigation by the blood and to the removal of the waste products of oxidation. In static work, the contraction phase of each fibre is much longer than the relaxation phase, and there are therefore always more contracted than relaxed muscles. Consequently, the time available for removing waste products is much shorter than in dynamic work, and static work causes fatigue much more quickly —indeed, a given group of muscles produces 15 per cent less effort in static than in dynamic work. Carrying an object with outstretched arms soon causes fatigue, and standing still for a long time may cause fainting due to imbalance of the circulation. Work done by the hand in static contraction, on a tool, a workpiece, a pen or other object often causes pain, indicating an accumulation of waste products in the muscles.

Posture during work and the manner of working therefore have a considerable effect on output.

2.1.5. Thermal regulation

If all the vital functions of the human body are to remain unimpaired, the body’s internal temperature must be maintained at or about 37°C. If a man lives in a cool or cold environment, he is constantly dissipating a certain amount of heat. This leads to the intensification of basic combustion in order to make up for the constant loss of calories. In adults, the basal metabolism needed for the functions of the various organs represents between 1,200 and 1,600 kcal per day, or between 0.85 and 1.1 kcal per minute. But this amount of heat is not enough to compensate for the losses unless the environmental temperature is at least 20°C. In agricultural work it is practically impossible to regulate the environmental temperature, and the clothing must therefore be adapted to the working conditions.
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The additional heat produced by physical work is sometimes very great and may amount to several times that of basal metabolism. For an eight-hour day, depending on the effect required, the expenditure of energy ranges from 2,000 to 3,000 kcal. The average is therefore 4–6 kcal/min, with peaks which may reach 12 kcal/min. This great amount of heat has to be eliminated as quickly as possible. The body dissipates heat by radiation, convection or evaporation (sweat). Radiation and convection, by which the body can dissipate only 2–2.5 kcal/min, are restricted by clothes. The heat losses by radiation and convection depend primarily on the difference in temperature between the skin and the environment, and this is regulated to a certain extent by the circulation of the blood. The greater the amount of excess heat to be removed, the more the circulation increases at the level of the skin and the faster the heat exchange with the environment becomes. The thermal conductivity of the skin is different in the two sexes, being lower in women than in men. This is why women can generally bear to be more lightly clothed than men. The dissipation of body heat increases in a draught, which is constantly bringing cooler air into the vicinity of the skin.

If the work generates more heat than can be dissipated by radiation and convection, sweat is produced, which evaporates on the skin. The phenomenon of sweat evaporation enables large quantities of heat to be dissipated in the environment. The larger the sweating area of the skin and the drier the environmental air, the greater the dissipation of heat by sweat. While for light work the humidity of the air is of no great significance, intensive work can be done only if the air is not saturated with humidity (that is, if it is comparatively dry), as otherwise the sweat cannot evaporate. Sweat can remove excess calories only by evaporating, and this is why streaming sweat represents a useless waste of energy.

As evaporation depends on the environmental temperature and air movements, clothing is an important factor here too. Since sweating is not uniform over the whole surface of the body, underclothes may facilitate the dissipation of heat if they are completely soaked with sweat. Thus underclothes should rapidly
absorb sweat, distribute it and ensure uniform and regular evaporation. The larger the surface of the garments, the more effectively will they fulfil these tasks. Natural fibres such as wool and cotton are impregnated more slowly than synthetic fibres. Closely woven materials are less effective than loose materials, such as knitted garments.

Equatorial and tropical countries are normally regarded as "hot countries". They may nevertheless have temperate seasons and cool upland regions (for instance, the east central African plateau, which is at an average altitude of 1,500 m and has intensively cultivated areas up to an altitude of about 3,000 m near the equator). As a rule, workers in hot climates cannot be expected to have the same output as those in temperate countries.

2.1.6. Co-ordination of physiological functions

The foregoing very brief description of some physiological functions suggests the existence of a very precise regulatory system for the necessary harmonisation of these functions. This regulation is controlled by the nerves, which receive their impulses from widely differing centres, most of them in the brain. It is almost entirely unconscious and involuntary, and depends on the physiological automation that keeps the body alive. The over-all co-ordination of the maintenance of body balance, the adaptation of respiration and circulation and the dissipation of heat are automatic reflex functions that do not require any voluntary interference. Automatic regulation is surer and more precise than conscious regulation and also seems to need a smaller expenditure of energy. This is in fact the reason why, whenever possible, man tends to replace certain processes, movements and actions by reflexes. Advantage is taken of this fact in training and working. However, this natural tendency, imposed on man as it were by his physiology, has as its counterpart an attitude, varying in degree, of inertia towards changes in working habits. In fact every new process has first to be controlled by the will; only later does it come within the province of reflexes, which, if necessary,

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will succeed and replace reflexes controlling the processes adopted hitherto.

The precision of regulatory functions varies with the importance of each in the maintenance of vital equilibrium, health and welfare. While internal temperature is regulated very precisely, the oxygen content of the blood is less so, and the water content of the body still less. Some types of regulation, such as that of blood supply to muscle, are almost instantaneous; others, such as the reconstitution of energy reserves, sometimes take several hours. Some have a daily rhythm—for instance, the alternation of activity (day) and rest (night)—and others have a periodicity of a year or more (duration of sleep in summer and winter, variation of activity with age). The organs can retain their vitality only by functioning regularly; they have no need of prolonged rest. On the contrary, inaction may atrophy them and put an end to the corresponding regulatory function. The proper working of the regulatory system depends on healthy development in childhood and adolescence, suitable training during growth and continuous exercise later.

2.1.7. Adaptation to environment

The co-ordination of the different functions of the body is not the sole task of physiological regulation; it must also ensure the correct adaptation of the individual to his environment. This is of capital importance for the maintenance of maximum working capacity in face of the enormous variations that may occur in the nature and place of work. For example, the muscular energy must correspond to the effort demanded, and the dissipation of heat to the environmental temperature. This adaptation can be easily observed: if the intensity of the effort increases, the pulse and breathing rates will steadily increase too. The eye adapts itself automatically to the luminosity and distance of objects. These are only a few of the countless regulatory activities induced by stimulation from the environment.
2.1.8. Working capacity

State of health and working capacity

Man's working capacity depends on the sum total of his physiological functions. It is based to some extent on a certain natural predisposition, but more on the development and training of the body, muscles and regulatory organs and centres. A person's working capacity is thus closely bound up with his state of health.

Physical work calls for certain qualities that man, if he enjoys good health, can develop fully by training. It requires well developed muscles, a robust skeleton, sound organs (circulatory, respiratory, renal, digestive, etc.) and a good neuro-endocrinian regulatory system.

Diet and work

An adequate and balanced diet is one of the indispensable conditions of satisfactory working capacity.

The more muscular work a man does, the greater must be his consumption of the substances required for chemical combustion. Energy reserves must therefore be replenished by a diet rich in carbohydrates. Most of the carbohydrates in a diet come from cereals: wheat in Europe and North America, rice in Asia and maize in Latin America. Many other plants are rich in carbohydrates, such as sorghum, manioc and potato. In making bread and paste, cereals must be treated to make them more digestible; they also undergo transformation in the body. On the other hand, sugar can be absorbed without any preparation and quickly passes into the blood, so that it is a very important food in intensive work.

When a man does less strenuous work, his diet should contain correspondingly fewer carbohydrates. It is a problem peculiar to modern nutrition in industrial countries (in which muscular work is steadily declining and consequently the consumption of carbohydrates should decline to the same extent) that because of habit or appetite people still consume large quantities of carbohydrates. This leads to obesity, which is not only inimical to work but is also at the origin of many diseases. In addition to carbohydrates, food
should contain proteins and fats, the latter contributing to the energy balance, more especially in the internal organs. Protein is needed in the formation of cell tissue, which is constantly being renewed; this is why muscle too needs a supply of protein. It is obvious that an adolescent whose muscular growth is not completed will need more protein than an adult; but the adult must have a certain minimum amount to maintain his energy balance. The body needs various proteins, and if a diet is to be balanced it must be adequate in quantity and quality. If it is not, there will be a food deficiency. Above all, there must be a minimum proportion (about 30 per cent) of animal protein for persons doing heavy, difficult or intellectual work.

All food is transformed in the digestive tract before being conveyed to the organs for which it is intended. Digestion is a cyclic and not a continuous process, but since requirements in muscular energy are either continuous (in the heart muscle, for example) or spread evenly over the hours of the day (as in the muscles of locomotion), reserves have to be constituted. Thus food can be absorbed and digested at the intervals fixed by meals. In healthy persons the body has sufficient reserves to enable it to burn, over a period of several days, more substance than is supplied by the food consumed during those days. In the long run, however, the food intake must restore the balance or exhaustion will ensue. The more energy the work demands, the richer and more frequent the meals should be; but meals should be spaced out if the work falls off. Thus a man doing heavy work needs five meals a day, while a tractor driver, for example, if he is comfortably seated, is so little affected by eight hours of work that he should easily be able to manage with three meals a day. The proper working of the digestive system is just as important for working capacity as are the soundness of the skeleton and the development of muscles.

Training

Working capacity is determined by muscular development as well as by food and by the adaptation of the circulatory and
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respiratory systems and their regulatory mechanisms. It is possible to develop individual predispositions by training, up to an advanced age, and to maintain them at a high level.

In physical training stimulations are produced by muscular work, the maximum stimulation corresponding to overwork up to the limit of fatigue. A short spell of overwork—from two to five minutes, for example—is the best form of physical training. Naturally, persons suffering from pathological changes or disorders should avoid such efforts.

The stimulation produced by training may be deliberate, as in sport, and be intended to develop the muscles of a particular part of the body, but it occurs automatically in all work. The greater the amount of work required of the different parts of the body, the better will be the physical condition. Thus varied muscular work, as encountered in many agricultural activities, is one of the best means of achieving a harmonious development of the body. The physical development of adolescents should therefore be promoted systematically, by means of work suited to their body strength and their age. Muscular stimulation that is due to training and causes muscles to develop also strengthens certain organs that participate indirectly in muscular work. Both the heart muscle and the regulatory mechanism of circulation benefit from the training constituted by steady work. The functions of each are decisive for the maintenance of a person’s working capacity. The regulation of the circulation is a very good example of the organisation of an aggregate of physiological reflexes designed to produce the requisite effect at any point and moment.

Physiological regulation operates at two different levels. One is independent of the will and is essentially concerned with the maintenance of life. It comprises the regulation of the heart, the circulation and the respiration, the regulation of the digestive system, the co-ordination of circulation and respiration, and so on. The other consists of regulatory mechanisms that depend on the will and govern processes bound up with voluntary action. But as a result of exercise and training, the control of work very soon passes into the domain of automatic reflexes. It must be supposed
that unconscious regulation is more economical, and at the same
time quicker and more precise, than conscious regulation. The
performance of any job requires a rapid and precise system of
regulation. This system also is subject to the laws of training:
the more it is used, the better it is trained and the more serviceable
it becomes.

There are limits in both directions to these biological processes.
The science of work has long concerned itself with discovering
man’s optimum working capacity. The permanent optimum rate
is attained when the energy supply just balances the loss.

There is also a lower limit to physical work. We all know that
after a long spell in bed the body has lost strength and must be
laboriously retrained for work. This is because the stimulation of
training has been absent for too long, and consequently the
muscles, muscular movements and organic regulation have all
become too weak. The optimum working capacity lies between
insufficient work and excessive work; however, frequent alterna-
tion of working intensities—light work, normal work, heavy
work—is, within certain limits, probably more beneficial to the
body than working at uniform intensity for a very long spell.

If training is to be effective, the same exercises must be repeated
often and correctly. In simple work the effects of training are
felt very soon, generally after a few hours. It is not necessary to
repeat the exercises without a break; they can be performed on
alternate days. This is very important in agricultural operations,
some of which cannot be carried on for long without a break.
However, in all the repetitions the course of the process must be
identical. Operations calling for very close co-ordination of various
movements or perceptions, and those involving analogous but
very varied actions, need longer training periods (up to 50 or 60
hours). Here too, however, one may count on a sufficient degree
of assimilation, which means that the work is performed correctly
under the control of the unconscious. This sufficiency of assimila-
tion of operations is particularly important for the agricultural
worker, who can rarely concentrate on the actual work, being
frequently obliged to watch the results so as to control quality.
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The farmer, acting both as the head of an undertaking and as a worker, is even more bound to watch the result of the work, so that he has no time to see how the work is actually being carried on. Thus, for the farmer, the introduction of new processes means a heavy psychological strain, and attempts should be made to lighten it by all possible means.

Age and aptitude for work

If the individual limits for these various factors are not exceeded, working capacity can be maintained over the whole span of active life. With age, it is true, many functional capacities decrease (see figure 12), as well as aptitude for training, but this does not matter much if the work done remains fairly constant. Moreover, the elderly worker often replaces failing strength by greater skill. Retraining elderly persons for new and arduous operations is difficult, but a man of 60 can do most field work just as well as a man of 30, except for those operations wherein human reaction time may be critical. If ability to work is to be measured by age, it may be said that it can equally well begin relatively early and continue well beyond 65 years. The quality declines only slowly with age if activity is regularly maintained. There is practically no wear on muscles and organs, as was once thought. However, the effects of illness increase with the years, because the aptitude of the body and its functions for training continuously decline. This is why convalescence in old people is more prolonged than in the young.

Curve of physiological work and biological rhythm

The working capacity of an individual varies in the course of the day, and does so in a rhythm that is independent of the actual work. It increases in the morning from 6.30 to 8 a.m., reaches its maximum about 10 a.m. and declines towards 11 a.m. In the middle of the day, between noon and 1 p.m., it is low, whatever the meal taken, and then it rises again. The afternoon maximum, between 2 and 3 p.m., is a little lower than the morning maximum. After 4 p.m. working capacity falls rapidly. At night it is always
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Figure 12. Decrease in functional capacity with age

MB  Basal metabolic rate
P   Working capacity
Q   Cardiac index
CV  Vital capacity
V_{max}  Maximal breathing capacity

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lower than in the daytime. No training can alter this natural rhythm. It persists even among persons who have worked only at night for several years; their working capacity remains greater during the day than during the night.

2.1.9. Fatigue

Whatever the tempo of work, man tires in the course of the day. Fatigue is a complex physiological condition entailing a reversible lowering of working capacity.

In addition to muscle fatigue due to work, which is the more acute the more the work is concentrated in a few muscle groups, man usually experiences general fatigue.

There are days when a person feels very tired after work; there are others when after the same work he is less tired. Fatigue is thus a subjective phenomenon that depends on both physiological and psychological factors. Normal fatigue occurring at the end of the day is usually overcome by sleep, so that when a man wakes up he is ready to resume work. However, matters are not always so simple; after the night’s rest some fatigue may remain, but this may be eliminated one or two days later by a good night’s sleep. On the average, daily fatigue due to work should not exceed the maximum that can be overcome by a night’s sleep. If overwork persists, fatigue accumulates and may cause serious trouble, or at least reduce working capacity. Even purely muscular fatigue is overcome by rest, chiefly by nightly rest. However, local phenomena may also occur in muscles; they are mostly due to insufficient elimination of waste products, which is often the result of the manner of working as much as of the intensity of the work.

Static effort is always particularly arduous and tiring, and this is why attempts should always be made to eliminate it from methods of working. If this is impossible, spells of static work must be shortened and interrupted by spells of dynamic work. If static work lasts too long, the minimum results will be local cramp (for example, tractor drivers suffer from cramp in the right calf when the accelerator pedal is badly placed and requires excessive effort by the foot).
2. Problems of occupational physiology and ergonomics

Intense muscular fatigue and stresses on the brain and the sense organs (eye, ear and so on) lead to considerable strain on the central nervous system and consequently to general fatigue, which in turn is characterised by a general lowering of working capacity. It will even affect body organs that have taken scarcely any part in the effort.

The working environment can also affect the functioning of the central nervous system and contribute to the development of fatigue. This is more particularly so in workplaces that are dark, noisy and hot to an unhealthy extent.

Monotonous work causes drowsiness and can lead to extremely serious problems in many kinds of work.

2.1.10. Measurement of physical work

Oxygen consumption

Since the degree of fatigue is not always directly proportionate to the work done, and since even today it cannot be measured, other criteria have been sought for measuring human work. One that has been applied for a long time, and is very suitable for measuring dynamic work, is the amount of oxygen consumed. This amount is in fact directly related to the energy consumed, so that the amount of oxygen consumed is a direct indication of the intensity of the work (see table 1). Naturally, it can be used only to measure dynamic muscular work, to the exclusion of static work and intellectual work which consume comparatively little oxygen.

The consumption of oxygen is measured as follows: the subject, whose nose is pinched, has in his mouth a valve that allows him to inhale fresh air. All the exhaled air passes through a volumetric counter which gives a reading of the amount of air breathed. Part of the exhaled air is collected in a vessel and then analysed in the laboratory. The carbon dioxide content and the oxygen content are determined and compared with the content of the inhaled air. This latter content need not be specially determined unless the air has been contaminated by exhaust or other gases, but can be
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Table 1. Work classification according to oxygen uptake and calorie expenditure

<table>
<thead>
<tr>
<th>Physiological variables</th>
<th>Work intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Very light</td>
</tr>
<tr>
<td>Oxygen uptake (l/min)</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Cal/min</td>
<td>&lt;2.5</td>
</tr>
</tbody>
</table>


taken from tables. The difference between the oxygen content multiplied by the volume of air inhaled gives the oxygen consumption, from which, with the aid of tables, the calorie production can be found. To find the number of calories produced by the work, the number due to basal metabolism must be deducted from this result. Although the apparatus used is much less cumbersome than formerly, the valve for breathing does inconvenience the subject and he needs a certain time to get used to it before exact measures can be taken.

Heart rate

As the intensity of physical work gradually diminished, it became necessary to find another method of measurement independent of oxygen consumption. A good indicator of work is the pulse. Since the amount of blood delivered by each heart beat is almost constant for an individual, within certain limits, the pulse rate is a direct indication of the amount of blood demanded by a particular part of the body. Measuring instruments are used for pulse counting during work. In simple investigations integrating instruments count the pulsations during a minute or a number of minutes. For more precise investigations instruments that record
each pulsation are used. Some have a small lamp which is placed under the lobe of the ear and the light of which is momentarily dimmed as each surge of blood passes. The variation in the light is converted into electric impulses that are shown on the recording instrument. Other instruments pick up the nervous impulses directly by means of electrodes. Heartbeats can be recorded electrically on magnetic tape or mechanically on paper reels. It is essential to obtain recordings that are as accurate as possible, for it is not at all easy to interpret them. In fact, the pulse rate does not depend solely on the oxygen consumption, and hence on the amount of dynamic work. Static effort will accelerate the pulse, and hence measuring the heart rate usefully supplements the measurement of oxygen consumption. Mental and intellectual activities also affect the heart rate, as do many other factors, especially psychological factors such as anguish, bad temper, joy and mental effort. Their influence is seen in the circulation. Moreover, regulation of the heart rate is subject to quite precise laws that are related to work. For example, the rate increases before work begins; during work it decreases to correspond with the intensity of the work. When the work is finished, the rate decreases slowly until it corresponds to the rest conditions prevailing before the work began. If this does not occur, it must be supposed that some fatigue remains after the work. To measure this, the method just described can be used. When it is desired to measure the pulse rate during work, the rest rate must be subtracted from the rate measured. The rest rate can be measured when the subject has rested for a sufficient time lying down. It varies greatly from one person to another, and so must be determined not only for each person separately but also several times for the same person, before and after work. In many measurements it is better to count only the pulsations above the number corresponding to the period immediately preceding the work; for example, for work done in a sitting position the pulse rest rate in that position would be subtracted, for work done in a standing position the rest rate would be subtracted, and so on. This “starting rate” is useful in most effort tests. The “effort” pulse rate is the difference be-
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Table 2. Classification of physical work by heart beat

<table>
<thead>
<tr>
<th>Degree of effort</th>
<th>Heart rate (pulse/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very light</td>
<td>&lt; 75</td>
</tr>
<tr>
<td>Light</td>
<td>75–100</td>
</tr>
<tr>
<td>Moderately heavy</td>
<td>100–125</td>
</tr>
<tr>
<td>Heavy</td>
<td>125–150</td>
</tr>
<tr>
<td>Very heavy</td>
<td>150–175</td>
</tr>
<tr>
<td>Extremely heavy</td>
<td>&gt; 175</td>
</tr>
</tbody>
</table>

Source: Adapted from Christensen.

...between the aggregate rate during work and the rest rate or starting rate.

Measurement of the heart rate, rather than measurement of the oxygen consumption, makes it possible to determine the limit of continuous work (see table 2). If the pulse remains constant during the work, this is within normal limits; but if the pulse rate continuously rises while the work remains constant, the work is exceeding the limits of normal effort. This test often brings to light organic defects or functional troubles that render a person unfit for heavy work.

2.2. Principles of ergonomics in agriculture

2.2.1. Definition and purpose

Ergonomics may be defined as the joint application of certain biological sciences and techniques so as to ensure the optimum mutual adaptation of man and work, to improve the worker’s output and to enhance his welfare.¹

¹ The basic principles of ergonomics are set out in W. T. Singleton: Introduction to ergonomics (Geneva, World Health Organization, 1972).
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Ergonomics is essentially characterised by multidisciplinary collaboration that brings together in a single team the physician, the physiologist, the psychologist, the sociologist and the engineer, whose aim is to organise and arrange the work and working equipment in such a way that the man-machine combination is made safer and sounder, and at the same time more productive.

2.2.2. Arrangement of the workplace

It is generally harder to improve a worksite or a workplace in agriculture than in industry. Agricultural operations are carried on in extensive open spaces, so that the workplace is constantly changing; the operations are influenced by the nature of the soil, the vegetation and the climate, or else they are linked to farm animals. These considerations fundamentally affect the working conditions. Thus in agriculture the workplace is in most cases determined by the work, and the work is generally performed with the help of hand-tools, implements or machines. Few operations are performed with the hands alone.

Posture of the body

However wide the variety of jobs, certain fundamental principles should never be lost sight of in the organisation of work. The posture of the body should not cause strain but should allow some changes of position during the work, unless a particular position is necessary for a specific job, such as sitting for milking. Moreover, the greatest efforts can be accomplished when all the muscles are brought into play. Hence all work calling for great muscular effort should be done when standing, for this position allows all the muscles to work, from the legs to the arms by way of the trunk. It is this fact that has determined the postures to be adopted in heavy work such as tree felling, pickaxing and digging. Heavy work is less tiring when it is rhythmical. Rhythmical movements of the body induce a wave-like activity in the muscles, characterised by a regular succession of contracting and relaxing phases which facilitates the regular irrigation of the blood and the evacuation

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of oxidisation toxins. Rhythmical work also avoids useless accelerations and decelerations of tools or certain parts of the body. Cutting with a scythe and beating with a flail are typical rhythmical operations. In team work, rhythmical operations can be accompanied by music, singing or rhythmical sounds, but their stimulating effect has certain drawbacks if the members of the team are not all about the same size. This is because the rhythm of work should be suited to the size of the workers and the length of their limbs: the amplitude of the movements should increase with the size, whereas the frequency of the rhythm, on the contrary, should decrease.

*Tempo of work and body size*

The tempo of work thus depends on body size. It also depends on the movements required by the job: the shorter they are, the faster the tempo; the longer they are, the slower the tempo. Very short movements, such as those of the hand and the forearm when picking potatoes or fruit off a conveyor belt, can be repeated 130 times a minute, movements with a scythe 50 to 60 times a minute, and movements when loading sheaves on to vehicles only from 14 to 18 times a minute. It is dangerous to force the tempo of work beyond the normal body rhythm. In the first place, this would require a considerable effort of will; second, in most cases movements would then become less precise, leading to a risk of misses or even accidents. In any case, the worker tires sooner and in the end his health suffers. This is why it would be a mistake to have a team for chain work composed of men of widely differing sizes. If each worker cannot be given a job that is suited to him, teams should at least be physiologically homogeneous, so that the optimum tempo of work can always be attained.

*Alternation of heavy and light work*

Light work brings only a small number of muscles into play. However, if in such work the body has to be kept in the same position for some time (as when milking in a sitting position), local muscular overload may occur notwithstanding the small
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expenditure of energy. In this kind of work, the working posture should therefore be abandoned every 10 or 20 minutes, and all the muscles in the body should be exercised to release tension and hasten the removal of waste products in the muscles bearing the brunt of the work, by activating the general circulation. Thus there are many advantages in rising after milking a cow and taking a few steps to empty the pail of milk. Output would decrease if the worker were to remain continuously in his sitting position. The minute spent in emptying the pail is thus not wasted: ultimately it stimulates the output. This also applies to all light or moderately heavy work that is done in a squatting position, on the knees or bent forward: for instance, thinning beetroots, planting out, weeding, and collecting potatoes. In all these forced postures some muscles are overworked and become prematurely tired through static work. It is therefore necessary to make some movements so as to facilitate muscular relaxation. Since it is often very difficult to make a foreman or even a worker understand the purpose of these breaks, it is simpler to organise the work in such a way that each worker is obliged to change his posture frequently. For example, muscular relaxation is obtained automatically if the worker himself has to go and fetch the plants that he is to plant out. If, on the contrary, other persons have to fetch the plants, those planting them out will have no opportunity for muscular relaxation and the team’s output will decline.

**Precision work and visual effort**

The posture of the body when working is often also determined by visual necessities. Theoretically, planting out could be done in a standing position, but in practice this is not possible because the operations of making the hole, inserting the roots and filling up the hole could not be performed with sufficient precision. The smaller the workpiece and the more precise the work has to be, the closer the eyes must be to the workpiece. The gardener lays planks on the bed so that he can be directly over the plants for planting out. For thinning very small beetroot plants the eyes have to be within 60 or 70 cm of the workpieces, and so the work has

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to be done kneeling or in a bent position. Sorting fruit or roots on a conveyor belt, for which the eyes have to be within 40 or 50 cm of the workpieces, is usually done in a sitting position so that the belt passes close to the worker’s eyes.

*Work done while walking*

Many agricultural operations are performed while the worker is walking. This is simply because the work covers a large area. If machines are not used, the worker has to cover the area on foot. But walking is itself fairly heavy work and absorbs large parts of a man’s working capacity, even if he is carrying a light load. On uneven or muddy ground, or ground covered with clods, the walker soon comes to the limit of heavy work. As far as possible, therefore, walking should be avoided, or in any event made easier. Workers can often be carried on farm machines. However, in the case of machines hauled by draught animals, to avoid overworking the animals the machine should at least have a handle which the man could hold on to and be pulled along. The faster the machine moves, the more desirable this is. Wherever possible, seats should be provided on hauled machines so that all walking is done away with. Seats are particularly necessary in mountainous country, where the effort of climbing is in itself a physical overload.

*Adaptation of tools to man*

Lastly, the posture of the body may be determined by the tool. The tool, invented and made by man, should be adapted not only to the work for which it was designed but also to the worker using it. He should not be compelled to make up for the shortcomings of the tool by the suppleness of his body. Many old tools are well adapted to man, but often tools that were originally designed for a particular job are later used for quite different ones and thus force the worker into bad postures. For instance, there are spades with short handles (80 cm or less) that are quite suitable for digging irrigation channels or shifting watercourses but not at all suitable for digging or lifting potatoes. For such work this implement imposes a posture that is so bent that abnormal efforts are
needed for digging. The same spade blade, set at a different angle and mounted on a longer handle, would do more work for a lower expenditure of energy.

The extent of muscle fatigue in the hands depends on the shape and diameter of the tool handle. Handles with too small a diameter require an excessively firm grip, with the hand closed too tightly, while handles that are too thick tire some finger muscles because only some of the joints and muscles can be used for gripping. Consequently, tools for men should have thicker handles than tools for women, and tools for small persons should have thinner handles than those for large ones because the extremities are generally in proportion to the rest of the body. When the hand has to slide along the handle, as in raking, the handle should be thin, smooth and circular in cross-section. If, on the other hand, the tool has to be guided in a particular direction (spade, axe, hammer), the handle should be oval in cross-section, since this is essential for controlling the direction of the blow. The bigger the tool, the more oval the handle. Tools that are swung (pickaxes, hatchets, axes) should have a handle with a bulge at the end to prevent them from slipping, without at the same time requiring extra hand effort for retention when used.

*Adaptation of machines to physiological capacities*

The controls of a machine should always be of fairly simple design, so that they are easy to use. All a worker’s movements should be in harmony with the natural reactions of his body. There should be a means of controlling every regulatory mechanism. Direct visible control, such as that of the forward movement of a tractor by turning the steering wheel, should be preferred to control by instruments. If instruments are used for control, however, they should be placed where the driver can easily see them, and their shape and appearance should be adapted to natural mental reactions. The greater the responsibility entailed in operating the machine, the more necessary it is to respect these conditions. The control layout of machines that are very
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powerful, or very fast, or have a wide range of action, should be arranged with particular care.

The effort required to operate the different controls should be adapted to physiological capacities. If considerable effort is necessary and can be avoided only by technical measures, pedal controls, which in a favourable position of the body permit effort of from 50 to 70 kg, are to be preferred. Effort exerted by the foot should be centrifugal; the greatest effort of the hands should be centripetal because the arm can pull more powerfully than it can push. The effort required to keep steering wheels and levers steady should be weak. If pedals are intended to be maintained in certain positions, about 80 per cent of the weight of the feet on the pedals should be compensated by means of springs. Steering wheels and lever handles should be so designed that the hand can grip them firmly without excessive pressure. All control instruments should be so placed in relation to the driver when seated that, without straining to change his position, he can both operate the controls and drive the machine directly or by means of the instruments. It is generally thought that a bad arrangement of controls intensifies physical strain only, but in fact the intensification of the mental strain it causes is the more dangerous. Many accidents can be attributed to excessive mental strain due to the illogical arrangement of control posts on machines.

A properly arranged control layout should not have too many controls, as this might lead to confusion. On the other hand, too much automation causes monotony, and this again adds to the operator’s mental strain.

Arrangement of seats

In agriculture it is rare to find jobs that carry on for a long time at the same place and allow the worker to remain seated (milking is one of the few examples). It is therefore rare to find fixed seats for use while working. Seats that are provided have to be so designed that the worker can adopt widely differing positions in order to avoid local fatigue. The seat proper should be padded so that the pressure on the skin is reduced, but the supporting
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surface should be kept rather small so as to allow a good flow of blood in different parts of the buttocks in changes of position. If the work involves much movement by the torso, a back is inadvisable. If there is a back, it should be padded. If the work is done in a sitting position but the worker has to get up frequently to remove or fetch material, his movements should not be hampered by table legs or other obstacles.

Operators of agricultural machines, whether hauled or self-propelled, should as a rule be seated, for it is easy to fix a seat on any machine. Recommended dimensions for tractor seats and controls are shown in figure 13. In a few jobs people have to go to and fro while a machine is working. This is necessary, for example, to maintain the regularity of working of very wide seed drills, and here it is advisable to replace the seat by a gangway. Often the operation and servicing of machines and their parts entail considerable effort (working the steering wheel, pedals and levers), which is transmitted to the seat through the pelvis. If the machine is moving on a hillside the torso also has to counteract the effect of the slope. This is why machine seats should satisfy certain conditions. They should be padded in the form of a basin (convex or flat cushions are unsatisfactory) and the edges of the basin should be sufficiently high. If in the operation of the machine the legs have to work (operating the brake and accelerator pedals, for instance), the seat should have a padded back to absorb the reaction from the forces applied to the pedals. All things considered, most machines should preferably be fitted with a deep seat. The bottom of the seat should be so shaped that the torso can be kept upright or bent without the need for support. On tractors and similar machines the seat should not be placed too far away from the steering wheel (see figure 14).
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Figure 13. Tractor driver's work station (measurements in millimetres)
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Figure 14. Too great a distance between the seat and the steering wheel leads to local strain and limited elasticity of the intervertebral discs

2.2.3. Conditions of mechanised work

Physiological aspects of mechanised work

The effects of mechanised work on man are different from those of work done with hauled machines or with hand-tools. As a rule the machine operator must perform two very different functions simultaneously: driving the machine at a suitable speed in a given direction, and doing a particular job (ploughing, sowing, harvesting, and so on). Hence he must give his attention to performing the operations required to control the machine, and to performing those required for the job. The results of the latter operations have to be verified frequently, and if necessary the machine has to be

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1 See also ILO: Protection of workers against noise and vibration in the working environment (Geneva, 1977); idem: Safe construction and operation of tractors (Geneva, 1976.)

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adjusted to improve them. The machine also has an indirect influence on the operator. Mechanical work is faster and hence requires greater visual activity; reflexes and actions have to be rapid and hence there is increased fatigue of the nervous system. Lastly, mechanical work increases responsibility because the power and speed given to man may be destructive.

Further, the operator is subjected to mechanical influences. The motive equipment and the movements themselves cause vibrations. Internal combustion engines, gears, hoods and metal wheels make noises. Some environmental conditions (sunshine, rain, wind, dust, and so on) are much the same as in manual work but they may prove more disagreeable for the machine operator, as when he has to drive for a long time against the setting sun. In such cases one can always turn slightly to one side so as to obtain suitable lighting for the working zone. Wind and rain are more disagreeable because the operator is seated and cannot easily protect himself against bad weather. Machines which are used a great deal in bad weather, such as tractors, should be equipped with a hood and windscreen as part of a crush-resistant roll-over protective cabin. Mechanical work also produces more dust, if only because of the greater speed and the greater working surfaces of machines. The need to keep moving in the same direction over long distances may aggravate the unwelcome effects of dust. These may be avoided by closed cabs (ventilated in temperate climates and air-conditioned in hot climates).

Vibration

A particularly important source of danger to which man is exposed is the mechanical vibration of moving machines. The human body has no organ for damping this vibration, and cannot absorb it when sitting. The sources of mechanical vibration differ widely. High frequency vibration is usually generated by the engine or the gearbox. It is usually of low amplitude, and even if it causes an unpleasant sensation in persons who are in contact with vibrating parts, it is not very dangerous. In fact, high frequency, low amplitude vibration is absorbed and damped by the layers of muscle
that envelop the skeleton. A typical example is the vibration of the steering wheel of a tractor or a combine harvester. Such vibration is transferred to the body only when the hands grip the steering wheel hard, and even then is usually propagated only as far as the forearm. But if the steering wheel is held loosely, the vibration is almost completely damped by the layers of muscle. It does cause disagreeable local muscular fatigue and possibly numbness and pins and needles, and for this reason controls should be protected against vibration.

High frequency, high amplitude vibration such as that produced by the handle of a motor plough, a pneumatic pick or a portable chain saw may be very dangerous. These machines can produce vibration in the hand and the forearm that is clearly visible and may cause inflammation of the joints. When it is impossible to suppress dangerous vibration (and it is always very difficult to do so), the machine should be operated by the same person for fairly short periods only. Low frequency, high amplitude vibration is dangerous for the skeleton and the internal organs. Such vibration occurs on all vehicles, but chiefly on those without springs (tractors, combine harvesters, and so on) and on machines drawn by tractors. These may produce frequencies of 1 to 20 Hz with amplitudes of a few centimetres. On bad tracks shocks of several kiloponds\(^1\) may be experienced, which may injure the spinal column. The greater the capacity of mechanical vibrations for setting up synchronous vibrations in the body or parts of the body, the more dangerous they are. Each part of the body has its own vibration frequency, and the smaller the part and the less it is associated with the rest of the body, the higher the frequency. The particular frequency of the torso of a sitting man is about 4 Hz, that of the internal organs varies from 2 to 8 Hz, that of the head is about 20 Hz, that of the hand is higher still, and that of the body as a whole is less than 4 Hz. All these specific frequencies are within the range of vibrations most commonly caused by agricultural vehicles and machines.

\(^1\) Kilopond = kilogram-force (kgf). This is the force which, when applied to a body having a mass of 1 kg, gives it an acceleration of 9.80665 m/s\(^2\).
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The effects of vibrations are very varied. The slow peristalsis of the stomach causes nausea. The specific vibrations of the head often cause headaches. Synchronous vibration of the torso may injure the vertebral menisci and aggravate deformations of the spinal column. Injuries of the vertebrae and their menisci are painful. In industrialised countries many injuries of the spinal column are attributable to motor transport (motor buses and light vehicles), for here too the frequency of the vibrations is between 1 and 6 Hz.

When a man is standing he can attenuate the effect of vibration on his body. The metatarsal, ankle and knee joints are elastic. When these joints are not fully extended, they absorb vibrations and shocks. However, absorption by the foot and leg muscles requires much effort and is very tiring. When working conditions are extremely bad, the machine operator should be able to stand on the machine so that he can more easily absorb excessively violent vibration and shocks. Normally, however, a machine operator is seated, and a sitting person is quite unable to absorb vibration. It is often difficult to avoid machine vibration that is directly due to the unevenness of the ground; a spring suspension, which is not always practicable, would only reduce their intensity. The undesirable effects of vibration on the torso can be reduced considerably only if the seat support is properly designed. Today it is possible, even with simple means (metal springs, rubber, hydraulic shock absorbers), to make seat supports that absorb up to 65 per cent of the vibration. It should be possible to adjust the suspension in accordance with the operator’s weight, and also to move the seat backwards or forwards. In any case, no person with a defective spinal column (scoliosis, vertebral deformations) should be allowed to operate a machine.

Noise

Mechanisation has not only introduced vibration; it has also increased noise. Man is not greatly troubled by noises up to 50 or 60 dB(A). This level is in fact often found in natural phenomena. However, most technical noises (measured at the level of the oper-
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ator’s or worker’s ear) are between 80 and 100 dB(A). This level can continue to be tolerated only at the cost of some loss of hearing. Noise strikes the ear with full force and weakens the nervous system. Shrill sounds are more disagreeable than low ones, and this is why work with small fast rotating machines, such as portable grinders or power-driven saws, is particularly trying. The high noise level of these machines soon causes fatigue and even bodily injury (damage to the internal ear), and as they are also sources of vibration a person should only use them for short periods. It is suspected that the dulling of hearing increases the risk of accidents. Ear protectors in the form of wadding are disagreeable, and moreover they are not as efficient in absorbing noise as ear muffs or devices specially designed for insertion into the ear canal.

Dust

Dust is often unavoidable in agricultural work. Dust from earth, hay and straw is not in itself very dangerous for the lungs because it is not so siliceous as mine or quarry dust. It may, however, hamper breathing, may also hamper perspiration by clogging the pores of the skin, may be dangerous for the eyes, and may carry other contaminants (fungi, bacteria, and so on) which cause illness.

It is not always easy to provide protection against dust. Very often workers will not wear a dust mask because working in a mask is extremely uncomfortable, especially in hot climates. Mention has already been made of tractor cabs. If the driver’s seat can be raised high enough (and this will depend on the angle of observation of the track), he will be less exposed to dust. A fan may prove helpful in keeping the dust away from him.

Special attention should be paid to the dangers threatening persons exposed to toxic powders. These powders can be blown on the worker by the wind, in the same way as dust, or by the jet from the sprayer, and so be injurious to his health. Hence, on all sprayers for toxic powders, the nozzle should be so arranged that even in an unfavourable wind the powder cannot reach the operator.
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**Exhaust gases**

Exhaust gases are much more dangerous than dust. All internal combustion engines give off toxic gases. It is a mistake to think that, in work in the open, exhaust gases mix quickly enough with the air to prevent the concentration from ever being harmful. Most exhaust pipes have not been well enough designed to prevent all exhaust gas from blowing back on the driver, irrespective of the direction of travel or the wind. Workers are also often bothered by having to work in the exhaust stream. Closed and well ventilated cabs reduce the risk of poisoning. When it is feasible, the best arrangement is to discharge the exhaust upwards and well above the driver's head. It would, of course, be very beneficial if the toxicity of exhaust gases could be reduced.

**Lighting**

Lighting is an important factor affecting the performance and tempo of work and the worker's safety. For agricultural work in the open there is always sufficient light in normal working hours. When the sun is dazzling, eye fatigue can be prevented by light-limiting goggles, but persons who normally wear glasses should have corrective lenses fitted. In regions with strong sunlight, pale tints and shiny metal surfaces (aluminium, chrome) should not be allowed on machines because of their high reflective level. Workplaces should be adequately lighted for night work so as to ensure clear vision, and the machine itself should be sufficiently illuminated to enable the worker to investigate irregularities or breakdowns without endangering himself.

The lighting of farm premises is often poor. Work is not made any easier when windows are too small and, as often happens, are fitted with wooden shutters, with a single weak lamp to light a whole stable. On every farm with electricity, sufficient and suitable lighting of stables is an economic necessity. High productivity in cattle can be obtained only by effective supervision of the animals and perfect cleanliness. In cowsheds the lamps should be arranged so that the milker's shadow is not in his way, for that would
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hamper him in checking the cleanliness of the udders and in milking properly. The lighting of stables can be improved if they are white-washed at frequent intervals.

2.2.4. Vocational training and health protection

Work and productivity in agriculture

Agricultural production, unlike industrial production, is essentially conditioned by nature: the soil, plants and animals furnish food and raw materials, and human labour intervenes only to allow them to do so and make them as productive as possible in the service of man.

The life of the soil, plants and animals depends less on the amount of work done than on its precision and suitability. The operations should be adapted to the life cycle, and above all to the climatic conditions.

In agriculture it is better to act at the right place and at the right time than let the work mount up. The essential condition of agricultural productivity, always and everywhere, is knowing the best method and applying it properly.

Similarly, the productivity of labour will not always be improved by replacing horses with tractors. It is more important to know how to direct operations.

The tractor will be useless if the farmer does not know when to plough the field so as to get the best results that the soil and the climate can give, or how to set the ploughshare so as to cover the seed well, or how to make a furrow that will drain off the water satisfactorily. Consequently, to improve productivity the farmer must be well trained.

Apprenticeship methods

For many centuries the same age-old methods of work were handed down from father to son, and the slightest change needed more than a generation to establish itself. It was thought (and in many countries it still is thought) that if a young man took part
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in the work of a farm—usually his father’s—that was quite enough to enable him to acquire all the knowledge he needed about working methods and techniques. Today this kind of apprenticeship is no longer adequate.

In fact, methods of work are now changing more quickly and more radically in agriculture than in many other occupations. The industrial worker has always worked with machines, even if their purpose and shape have changed (and will continue to change); but the farmer has passed suddenly out of the hand-tool age into the machine age. In many countries the farmer has covered in a single bound stages of development that elsewhere have been spread over centuries. This too rapid transition, coupled with inadequate apprenticeship, has led to many difficulties. Errors in the use of machines and in the application of technical methods can lead frequently to injuries to health or accidents. This is why the farmer should abandon family customs and opt for a systematic apprenticeship.

Systematic apprenticeship and health protection

Young persons should not only be enabled to acquire technical knowledge; they should also be shown how to apply it in practice, their interest in work should be aroused by a judicious selection of certain operations, they should be made to understand what is meant by the quality of work and to realise the skill that they can acquire by systematic training of the body and its movements. The second stage of training is the acquisition of good working methods, permitting economies in energy. It is only when the third stage is reached that one can hope to achieve a large amount of work in a given time—that is, high productivity. If a very rapid tempo of work is imposed too soon, it often happens that the natural ambition of the young to do more work than adults causes them to force the pace and make wrong movements, leading to one-sided muscular fatigue and, in the course of time, to bad working habits that are injurious to health. Nearly all alleged typical illnesses of old agricultural workers and old farmers (scolioses, muscular troubles, and so forth) may be largely attributed to bad
working habits acquired in their youth. The more apprenticeship is deliberately directed towards control of the body and the effective use of all the muscles, the more a man’s work will eventually benefit his health and the less it will be a source of accidents. The development of this apprenticeship by stages should be well understood by the learner, so that later on he will be able to train himself by the same method in new types of operation. Adult workers can be trained in new methods much more rapidly if they have received training in their youth. This is another reason for attaching great importance to a systematic apprenticeship.

Well planned and well directed apprenticeships are essential not only for economic reasons but also for promoting the health of adolescents, maintaining the health of adults and preventing work accidents.
3. Prevention and management of occupational pesticide poisonings

3.1. Introduction and statement of the problem

Pesticidal chemicals represent increasingly important instruments for the improvement and protection of world supplies of food and fibre. In common with other industrial chemicals, they all have potential for causing injury or death to workers who must have some contact with them in the process of manufacture, formulation, packaging, transport and application, or in the course of harvesting treated crops. Some materials are extremely toxic (hazardous in small absorbed doses), while others cause harmful effects only when very large amounts have been absorbed. Many cause irritation of the skin, eyes and mucous membranes of the respiratory tract. A few are "sensitisers", producing reactions of increasing severity on repeated contact. When possible, the more humane poisons and pesticides that are less toxic for human beings should be used. The circumstances of exposure (duration, formulation, task performed, weather conditions) all influence the absorption of toxic ingredients and the actions of these materials on vital tissues. Well nourished, comfortably housed workers, enjoying adequate rest and hygiene, are less vulnerable to toxic chemicals than persons who are burdened with malnutrition, disease and fatigue. Note that the indiscriminate use of pesticides can have catastrophic effects on wildlife.

Despite the difficulty of providing safe working conditions for persons handling pesticides, 30 years of occupational experience have demonstrated that these chemicals can be used safely if elementary safety precautions are observed. None the less, some countries have banned the use of certain pesticides owing to their resistance to degradation. Delay in achieving safe working conditions has occurred mainly because the workforce requiring education and protection is geographically dispersed and technologically uninitiated. But as the main causes of poisonings have come to
3. Occupational pesticide poisonings

light, the cardinal safeguards needed to avoid poisonings have become evident. This experience in the workplace has generated the well accepted rules for safe handling of pesticides which follow in sections 3.2. and 3.3. Section 3.4. provides some basic information on the physiology of chemical absorption, metabolism and excretion of toxic substances, together with a number of practical definitions that are important to persons working with these materials. Section 3.5. describes some of the salient characteristics of the major classes of pesticides commonly used today. Section 3.6. provides information on other chemical substances used in agriculture.

3.2. General precautions for the prevention of pesticide poisonings

3.2.1. Storage of pesticide containers

(1) Pesticides must be stored in locked enclosures, secure from animals, children and incompetent adults.

(2) Containers must be inspected carefully for leakage before they are stored. Many pesticides are volatile, so that leakage from a single container can permeate the air of storage premises, making the discovery and removal of the offending package difficult and dangerous.

3.2.2. Sanitation in space used for formulation, packaging and loading of application equipment

(1) Adequate ventilation is essential in spaces where pesticides are poured, mixed, bagged or otherwise transferred from one container to another. In some circumstances, natural air movement may be sufficient through wall-less shelters. If the odour of pesticides lingers more than a few seconds under such structures, large fans must be used to establish a strong draught over the worksite to carry the toxic material into areas where it will not endanger man, animals or desirable vegetation.
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If pesticides are handled in enclosed areas, ample powered ventilation must be provided by overhead exhaust fans. Dust mills must be maintained free of leaks. Special means of personal protection must be provided to persons assigned to maintain such equipment (see subsection 3.2.6. below).

(2) Spilled pesticides must be promptly removed. Floor drains over disposal tanks should be readily accessible to the worksite so that spilled material can be promptly flushed away. Spills remote from floor drains should be neutralised (usually with caustic soda), taken up with sawdust or adsorbent clay (bentonite), and buried. Workers must have adequate personal protection during clean-up operations (see subsection 3.2.6. below).

(3) An ample supply of clean water must be close at hand for washing away pesticide spilled or splashed on to operators.

(4) Personnel involved in pesticide application frequently have the most prolonged contact with quantities of chemicals, and are therefore the most likely to suffer poisonings. Poisoning of applicator personnel may be caused by spillage of liquids, drift of dust or spray formulations, rupture of hoses carrying liquid mixtures under high pressure, and back-drainage of material from loading hoses. Pesticide-laden dust propelled by aircraft prop wash must be directed away from the worksite. The maintenance of application equipment (cleaning of nozzles and conduits, washing of hoppers, repairing of hoses and valves, welding and sealing of tanks) requires special protective measures and intelligent work habits.

(5) Because of the extraordinary performance standard required of aerial applicators, they must take no part in mixing or loading operations that might subject them to the risk of even slight poisoning.

3.2.3. Worker education

(1) Workers likely to come into contact with pesticides must be thoroughly instructed as to the hazardous properties of each.
3. Occupational pesticide poisonings

They must be taught safe techniques for carrying out assigned tasks.

(2) They should be advised of the warning symptoms that occur early in many poisonings (headache, dizziness, nausea, weakness) and also of the particular symptoms peculiar to poisonings by specific chemicals. They should be encouraged to report promptly to a supervisor any symptoms possibly caused by excessive absorption of chemicals.

3.2.4. Disposal of used containers

The same provisions must be made for safe storage of used (not quite empty) containers as are established for full containers (locked enclosure) until they are either decontaminated (triple-rinsed by appropriately protected personnel) or disposed of by burial or fire. No container (rinsed or not) should be used for purposes other than those legally allowed by the pesticide manufacturer (sometimes recycling and refilling). Workers must be made to realise that any pesticide remaining in “used” containers is a serious hazard to children, domestic and wild animals, and unwitting adults who may venture to clean and re-use the containers without taking safety precautions.

3.2.5. Re-entry of pesticide-treated fields

Agricultural workers and their supervisors must be made aware that it is sometimes hazardous to re-enter fields which have been treated recently with particular pesticides. The actual risks involved depend on: (a) toxicity of the applied pesticide; (b) dosage and formulation applied to the crop; (c) rate of degradation of pesticide; (d) weather conditions (temperature, humidity, wind); and (e) degree of contact of workers with treated foliage. Supervisors must be guided by regulations designed to protect workers from excessive exposure to foliage residues of the toxic pesticides. In several countries safe re-entry intervals have been promulgated for many chemicals.
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3.2.6. Cleaning and repair of equipment

Cleanliness and good performance of equipment are as essential to workers' safety as they are to good agricultural practice. It is significant, however, that a large proportion of pesticide poisonings have occurred in persons assigned to these tasks. These workers should therefore be: (a) carefully selected as competent and careful workers; (b) thoroughly advised of the hazards represented by the contaminating chemicals; (c) instructed in safe techniques for execution of their respective tasks; and (d) required to work in pairs at maintenance tasks involving substantial toxic hazards.

3.3. Specific strategies to minimise worker injury by pesticides

3.3.1. Sanitary standards and protective gear

(1) Workers regularly exposed to pesticides (particularly the more toxic ones) must adhere to rigid sanitary standards and use protective gear consistently to avoid poisoning, as follows:

(a) workers handling pesticides should shower and shampoo at the end of each work day, and put on a freshly laundered coverall at the beginning of each work day;

(b) food and drink should never be consumed at the worksite. Hands must be washed scrupulously before meals to avoid contamination of food, drink and smoking materials; and

(c) workers in close proximity to concentrates or dusts containing very toxic pesticides must wear adequate respiratory protective gear. The equipment must be carefully selected to ensure that the system of air purification is suitable for the tasks at hand (whether dusts, aerosols, vapours or gases). Face masks must fit snugly to be effective, and the inner surfaces must be cleansed frequently with clean water (or rubbing alcohol) to ensure freedom from pesticide contamination. Air filter cartridges must be changed regularly (the interval will depend on the level of air
3. Occupational pesticide poisonings

contamination) to be effective. Air supplied to helmets must be free of toxic substances (including carbon monoxide and oil vapour from compressors).

(2) Outer protective gear (suits, aprons, boots, goggles, hats, gloves) must also be cleaned and maintained. When cracks, holes and worn areas appear, equipment must be replaced. Gloves and boots must be cleaned inside and out periodically to remove contaminating material.

(3) The selection and use of protective gear must take intelligent account of the safety requirements of the task at hand and the work environment. Chemically impervious protective gear (such as rubber suits) is also heat impervious, and cannot be worn for indefinite periods in hot environments. Work with highly toxic pesticides must therefore be interrupted regularly to allow relief from heat stress and fatigue. The provision of ample clean drinking water and electrolytes and adequate rest periods are essential to safety in these circumstances. Many pesticides having low toxic and irritative potential, or present in low concentration, can be handled safely without the use of elaborate protective gear, provided that commonsense standards of hygiene and cleanliness are observed.

3.3.2. Medical surveillance

(1) For some tasks involving contact with dangerous pesticides, pre-employment examinations should be required. Persons subject to severe asthmatic attacks or convulsive disorders should not be employed at tasks involving significant exposure to neurotoxic or irritant chemicals. Persons with chronic skin disease (psoriasis, eczema) are very likely to suffer exacerbations of these conditions when they come in contact with pesticides, or even the solvent vehicles. Workers assigned to prepare formulations must be sufficiently literate and intelligent to read and understand container labels and instructions, including those important to their own safety and that of their associates.
Health and hygiene in agriculture

Individuals with advanced liver and kidney disease should not be exposed to pesticidal chemicals or solvents.

(2) Periodic physical or biochemical examinations should be carried out during the employment of some workers regularly exposed to chemicals that pose either acute or chronic threats to health. In some circumstances premonitory symptoms and signs and laboratory tests can be used to forewarn of poisoning from excessive cumulative absorption of toxicants. These may be valuable, not only directly in protecting workers but also indirectly in monitoring the adequacy of hygienic practices at the worksite.

(3) While it is not possible to monitor the health status of all workers exposed to pesticides, vigilant observation of the most regularly and intensively exposed persons can contribute to the development of standards and safety practices appropriate to this sector of industrial hygiene. Previously unsuspected adverse effects may be detected and lead to development of better protective methods.

3.3.3. Management of poisonings

(1) Those responsible for medical treatment centres (hospital emergency rooms, clinics) that will be utilised in the event of poisoning should be kept informed of the pesticides being handled locally, and urged to prepare for the provision of appropriate treatment.

(2) Plans should be made for the prompt removal of poisoned workers to the treatment centre. Supervisors must never allow a person who may be poisoned to leave work unattended. Poisoning by some pesticides worsens rapidly, leaving the victim disabled. A container label describing the probable offending chemical should be taken to the treatment centre with the victim to assist emergency personnel to cope with the poisoning.

(3) If the worksite is very remote from a treatment centre, it may be necessary to instruct supervisory personnel in the administration of antidotes and in emergency resuscitation.
3. Occupational pesticide poisonings

(4) It is neither practical nor safe to administer antidotes to workers prophylactically.

3.4. Physiology of pesticide absorption and action in man:

general measures for management of poisonings

Pesticides are “absorbed by” (taken into) the body by three routes: inhalation (through the lungs); ingestion (through the stomach and intestines); dermal absorption (through the skin).

Absorption by way of the lungs is very rapid (a few seconds); by the gut intermediate in rate (minutes to hours); and across the skin usually slower still (hours to days). Chemicals absorbed by the lungs and skin enter the systemic circulation directly; those absorbed from the gut are conveyed mainly by the portal circulation to the liver, where biotransformations may occur, before they enter the systemic circulation.

Only vapours, fine aerosols and very fine dust particles (less than 5 μm in diameter) are absorbed directly by the lung. But inhaled materials of larger dimensions do not escape from the body: they are trapped in the mucous lining of the respiratory tract, delivered into the pharynx by ciliary transport of mucous, and swallowed. They are then absorbed by the gut, just as though they had been taken by mouth. Pesticide inhalation is a particular problem in formulation and application activities.

Ingestion of pesticide may occur as a result of manual contamination of food, drink and smoking material. Dermal absorption is of particular importance in workers who have extensive body surface exposure to foliage residues. This commonly occurs during the harvesting of orchard crops previously treated with pesticides.

Some pesticides (such as organochlorines) remain essentially intact long after absorption, finally undergoing chemical change and slow excretion through the liver and gut or the kidneys. Other chemicals are degraded promptly in the body to substances much less acutely toxic than the parent pesticides. Degradation products are then excreted by the liver or kidney. The urinary degradation
products of some pesticides can be used to evaluate prior absorption of the parent chemical.

The intermediate formation products of some pesticides (-oxons of organophosphates) are much more toxic than the absorbed chemicals. This is an important feature of the toxicology of organophosphates.

Pesticides are said to have “adverse effects” when they are in any way detrimental to well-being. This expression includes superficial injury, such as skin or mucous membrane irritation, as well as “toxic” effects, which are usually understood to mean biochemical disorders involving internal organs and tissues. The word “poisoning” (or intoxication) is properly reserved for states of chemical injury manifest as symptoms and signs—that is, actual sickness. A “poison” (toxicant, toxic substance) is a chemical capable of causing injury to internal organs and tissues even when the quantity absorbed into the body is small.

Some absorbed poisons cause “acute” (immediate and brief) effects. Others in moderate dosage cause little immediate sickness, but produce instead “chronic” (protracted) poisoning when absorption is long continued.

Certain pesticides are “lipotropic” (fat-seeking). Once they are absorbed by the gut, they are distributed to all the fatty tissues of the body. Because they undergo chemical change so slowly, and because they are so inefficiently excreted, these pesticides are “stored” in the fatty tissues for periods of days to years. The organochlorines (typically DDT, dieldrin and BHC (benzene hexachloride)) exhibit this behaviour. Of itself, the storage of a chemical in fat does not necessarily represent a hazard to health. Absorbable lead, arsenic and mercury, on the other hand, remain for some time in the vital non-fatty tissues, where they exert toxic effects as long as they are present.

The effects of some pesticides on the body are limited to particular organs or organ systems (nervous system, lung, liver and others). Other pesticidal poisons exert their effects by damaging critical chemicals in the body (“enzymes”) which are necessary for the normal functioning of many organs and tissues. The organo-
3. Occupational pesticide poisonings

chlorines (DDT, BHC and others) act principally by impairing the function of the brain. Toxic metals commonly combine with sulph-hydryl groups essential to the function of critical enzymes in the liver, kidney and nervous tissue. The organophosphate and carbamate inhibitors of the enzyme cholinesterase act wherever this enzyme transmits nerve impulses to nerve or effector cells in the body—for example, in the brain, the eye, skeletal muscles, bladder, intestines, salivary glands. The warfarin rodenticides act by impairing liver enzymes essential to the production of prothrombin (a substance required for normal blood clotting). For humane reasons, anti-coagulant poisons are discouraged in some countries. Thallium and lead damage the functions of multiple enzymes throughout the body, including those needed for normal production of the oxygen-carrying pigment of blood, haemoglobin. Still other chemicals cause “sensitisation” of persons experiencing contact with them. This means that tissue reactions (frequently dermal or upper respiratory) are more severe as contact occurs repeatedly. In some cases, individuals may suffer life-threatening or disabling reactions to substances to which they have become sensitive.

By whatever mechanisms various poisons act, their approximate relative capacities for killing (lethality) are conventionally evaluated by administration to laboratory animals. The lower the dose of a poison required to kill, the greater its toxicity. The dose needed to kill 50 per cent of a group of test animals is known as the LD₅₀ value. Male or female rats, mice, guinea pigs and other animals may be used. Lethality may be measured within 48 hours of a single dose (acute LD₅₀) or it may be estimated over an extended period of lower level dosing (chronic LD₅₀). The toxicant may be given by mouth (oral), by injection (parenteral) or by application to the skin (dermal). LD₅₀ measurements by these various criteria are, of course, numerically different. The most commonly quoted LD₅₀ values are based on acute effects in the orally dosed adult male rat. Estimates for several pesticides are shown in comparative fashion in figures 15 to 17. It is important to realise that LD₅₀ measurements reflect only potential for killing,
Heath and hygiene in agriculture

and that this characteristic has no necessary relation to a chemical’s potential for causing symptoms, chronic illness or other sublethal effects.

For a limited number of pesticidal toxicants, there are counteracting medications which, when introduced into the body directly, block or nullify the actions of the poisons. These counteracting drugs are known as antidotes. Some act by accelerating the destruction and excretion of the toxicant; others act by various mechanisms of interference with toxicant action. Vitamin K is a specific antidote for warfarin poisoning; atropine is a specific antidote for poisonings by organophosphates and carbamates.

For a majority of pesticides, unfortunately, no specific antidotes exist. Even so, effective methods for saving victims of poisoning do exist, as indicated below. Skilful application of these “supportive measures” can frequently make all the difference between life and death in cases of poisoning for which no antidote is available:

(1) Continuing absorption of pesticide can be stopped by cleaning it from the skin surface, or washing or purging it out of the gut. Adsorbent materials (such as activated charcoal) can be used to minimise gastro-intestinal absorption of some poisons by binding them within the gut.

(2) The movement of air into and out of the lungs can be assisted mechanically to sustain life even when chest and diaphragm muscles are paralysed. External cardiac massage may also be required.

(3) Convulsions can be controlled by appropriate medications.

(4) Oxygen can be added to the air breathed, to compensate for various toxic actions causing poor oxygenation of tissues.

(5) Increases or decreases in body temperature can be limited by the use of appropriate blankets or baths.

(6) Excretory and metabolic processes can be supported by the infusion of intravenous solutions containing sugar and electrolytes.

(7) Within limits, medications can be used to control extreme changes in blood pressure.


Figure 15. Relative acute toxicities of 37 organophosphate and carbamate pesticides

<table>
<thead>
<tr>
<th>Carbamates</th>
<th>Organophosphates</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEMIK</td>
<td>THIMET, PHORATE</td>
</tr>
<tr>
<td></td>
<td>TEPP and DASANIT</td>
</tr>
<tr>
<td></td>
<td>SYSTOX, DEMETON</td>
</tr>
<tr>
<td></td>
<td>DISYSTON</td>
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<tr>
<td></td>
<td>E PARATHION</td>
</tr>
<tr>
<td></td>
<td>PHOSDRIN</td>
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<tr>
<td></td>
<td>M PARATHION</td>
</tr>
<tr>
<td>FURADAN</td>
<td>GUTHION</td>
</tr>
<tr>
<td></td>
<td>CO-RAL</td>
</tr>
<tr>
<td>LANNATE</td>
<td>MONITOR</td>
</tr>
<tr>
<td>CARZOL</td>
<td>AZODRIN</td>
</tr>
<tr>
<td></td>
<td>BIDRIN</td>
</tr>
<tr>
<td></td>
<td>PHOSPHAMIDON</td>
</tr>
<tr>
<td></td>
<td>TRITHION</td>
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<tr>
<td></td>
<td>EPN</td>
</tr>
<tr>
<td></td>
<td>DELNAV</td>
</tr>
<tr>
<td></td>
<td>VAPONA, DDVP</td>
</tr>
<tr>
<td></td>
<td>ETHION</td>
</tr>
<tr>
<td></td>
<td>META SYSTOX R</td>
</tr>
<tr>
<td>BAYGON</td>
<td>DIAZINON</td>
</tr>
<tr>
<td></td>
<td>DURSBAN</td>
</tr>
<tr>
<td></td>
<td>BAYTEX, ENTEX</td>
</tr>
<tr>
<td></td>
<td>DEF</td>
</tr>
<tr>
<td></td>
<td>CYGON, DIMETHOATE</td>
</tr>
<tr>
<td></td>
<td>IMIDAN</td>
</tr>
<tr>
<td>VAPAM</td>
<td>DIBROM, NALED</td>
</tr>
<tr>
<td></td>
<td>DYLOX, DIPTEREX</td>
</tr>
<tr>
<td>SEVIN</td>
<td>MALATHION</td>
</tr>
<tr>
<td></td>
<td>RONNEL, KORLAN</td>
</tr>
<tr>
<td></td>
<td>GARDONA</td>
</tr>
</tbody>
</table>

Acute oral LD₅₀ values (mg/kg) in rats

105
Of the measures mentioned above, some can be applied immediately at the worksite when poisoning symptoms appear, thereby improving the victim’s chances of survival:

1. Remove him from the contaminated environment.
2. Remove all contaminated clothing, and wash any contaminating toxicant from the body surface and hair with soap and water.
3. If pesticide has been swallowed, induce emesis (mechanically or with mustard water) only if the victim is fully conscious.
4. If the victim is unconscious, establish a clear airway and sustain respiration by mouth-to-mouth or mouth-to-nose resuscitative methods. External cardiac massage may be necessary if the heart action has ceased. Continue the resuscitative efforts while the victim is being transported to a centre where treatment can be administered.
3. Occupational pesticide poisonings

Figure 17. Relative acute toxicities of five chlorophenoxy pesticides commonly used in Arizona

Chlorophenoxy

PENTACHLOROPHENOL
2,4-D
2,4,5-T
ERBON
DACTHAL

MOST TOXIC
DECREASING TOXICITY

Acute oral LD₅₀ values (mg/kg) in rats

3.5. Toxic properties of specific classes of pesticide, and special precautions to avoid poisoning

3.5.1. The large molecular organochlorine insecticides

Chemical structures of common agents

Lindane

Chlordane

Dieldrin

Endosulphan

Heptachlor
Health and hygiene in agriculture

Commonly used chemicals of this class are (approximately in order of toxicity):

Highly toxic: endrin (Hexadrin), a stereo-isomer of dieldrin.
Moderately toxic: aldrin (Aldrite, Drinox), endosulphan (Thiodan), dieldrin (Dieldrite), toxaphene (Toxakil, Strobane-T), lindane (Isotox, Gammexane), benzene hexachloride (BHC, HCH), DDT\(^1\) (Chlorophenothane), heptachlor, terpene-polychlorinates (Strobane), chlordane (Chlordan), dicophol (Keltbane), chlorobenzilate (Acaraben), mirex, methoxychlor (Marlate).

Toxicology

In adequate dosage, these chemicals interfere with axonic transmission of nerve impulses, and therefore disrupt the function of the nervous system, principally that of the brain. This results in behavioural changes, sensory and equilibrium disturbances, involuntary muscle activity and depression of vital centres, particularly respiration. Adequate doses increase the irritability of the myocardium, causing arrhythmis, and produce degenerative changes in the liver.

Most frequent presenting symptoms and signs of poisoning

These include: apprehension, excitability, dizziness, headache, disorientation, weakness, paresthesiae, muscle twitching, tremor,

\(^{1}\) The World Health Organization recommends that the outdoor use of DDT should be avoided as far as possible.
3. Occupational pesticide poisonings

tonic and clonic convulsions (often epileptiform), coma. Soon after ingestion, nausea and vomiting are often prominent. When chemicals are absorbed by parenteral routes, apprehension, twitching, tremors and convulsions may be the first symptoms. Respiratory depression is caused by the petroleum solvents in which these pesticides are usually dissolved. Pallor occurs in moderate to severe poisoning. Cyanosis may result as convulsive activity interferes with respiration.

Confirmation of diagnosis

The pesticide and/or metabolites can usually be identified in the blood or urine by gas-liquid chromatographic examination of samples taken within 72 hours of poisoning. Some chlorinated hydrocarbon pesticides persist in the serum for weeks or months after absorption. Treatment of poisonings cannot be delayed pending confirmatory analysis of the blood.

Avoidance and emergency management of poisoning

(1) Workers handling concentrated formulations must wear masks (with charcoal filters) or helmets supplied with purified air, aprons, gloves and boots impervious to hydrocarbon-soluble materials, such as thick rubber protective gear.

(2) Workers exposed to the more dilute spray and dust formulations should minimise their personal exposure by standing upwind during handling or application operations. If the wearing of rubber protective gear is impossible (for instance, because of heat or the need for mobility), clean coveralls and caps provided daily usually afford adequate protection against dermal exposure to dilute pesticide formulations.

(3) Soap and water showers and shampoos are extremely important hygiene measures for limiting the absorption of chlorinated hydrocarbon pesticides after occupational exposure. Unless removed by adequate cleansing, the toxicants will continue to penetrate contaminated body surfaces long after contact. The removal of toxicant from the skin and hair is also essential to proper first-aid treatment of poisonings by these agents.
(4) Because convulsive seizures are a common manifestation of acute poisoning, supervisors should be prepared to deal with this condition. A clear airway through the nosopharynx must be maintained by forward traction on the tongue and mandible. Mouth-to-mouth resuscitation may be required in event of severe respiratory depression, and external cardiac massage may be needed to maintain circulation if the heart is arrested or fibrillating.

(5) There are no specific antidotes for poisoning by chlorinated hydrocarbons, although the skilful use of anticonvulsant medication can often save a life. This treatment is itself hazardous and must be administered by qualified medical personnel.

3.5.2. The cholinesterase-inhibiting organophosphate pesticides

General chemical structure

Commonly used pesticides of this class are (approximately in order of decreasing toxicity):

Highly toxic: TEPP, phorate (Thimet), mevinphos (Phosdrin), phensulphothion (Dasanit), demeton (Systox), disulphoton (Dicyost), sulphotepp (Bladafume, Dithione), ethyl parathion (Parathion, Thiophos), phonophos (Dyfonate), EPN, azinphosmethyl (Guthion), methyl parathion (Dalf), monocrotophos (Azodrin), dicrotophos (Bidrin), methamidophos (Monitor), carbophenothion (Trithion), phosphamidon (Dimecron).

Moderately toxic: phamphur (Warbex, Bo-Ana, Famfos), ethoprop (Mocap), coumaphos (Co-Ral), demeton-methyl (Metasystox), dichlorvos (DDVP, Vapona, No-Pest), dioxathion (Delnav), crotoxyphos (Ciodrin), chlorpyrifos (Dursban), ethion, fenthion (Baytex, Entex), diazinon (Spectracide), dimethoate (Cygon), naled (Dibrom), trichlorphon (Dylox, Dipterex, Neguvon), cruphosphate (Ruelene), ronnel (Korlan), malathion (Cythion).
Toxicology

Toxicants of this class phosphorylate almost irreversibly varying amounts of the acetylcholinesterase enzyme of tissues, allowing the accumulation of acetylcholine at cholinergic neuro-effector junctions (muscarinic effects), and at skeletal muscle myoneuronal junctions and in autonomic ganglia (nicotinic effects). Poison also impairs the functioning of the central nervous system. Toxicants can be absorbed in inhalation, ingestion and skin penetration. Some are converted to a more toxic intermediate before they are metabolised. All undergo hydrolytic degradation in liver and other tissues, usually within hours of absorption. Degradation products are of low toxicity, and are excreted in urine and faeces.

Most frequent presenting symptoms and signs of poisoning

Symptoms of acute poisoning develop during organophosphate exposure or within 12 hours of contact. They include: headache, dizziness, extreme weakness, ataxia, tiny pupils, blurred or “dark” vision, muscle twitching, tremor, sometimes convulsions, mental confusion, incontinence, unconsciousness; nausea, vomiting, abdominal cramps, diarrhoea; tightness in chest, slow heartbeat, wheezing, productive cough, sometimes pulmonary oedema (up to 12 hours after poisoning); sweating, rhinorrhoea, tearing, salivation. Severe poisoning may cause sudden unconsciousness, or produce a toxic psychosis resembling acute alcoholism. Extreme bradycardia and heart block have been observed. Respiratory depression is caused by the toxicant and also by hydrocarbon solvent. Continuing absorption at intermediate dosage may cause a “flu-like” illness characterised by weakness, anorexia and malaise.

Confirmation of diagnosis

Depression of plasma and/or RBC cholinesterase activity is the most satisfactory and generally available evidence of excessive absorption of this class of toxicants. In response to organophosphates, depression of plasma cholinesterase often persists for one to three weeks, while depression of RBC acetylcholinesterase persists for up to 12 weeks. Organophosphates yield metabolites that
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Table 3. Approximate lower limits of normal plasma and red cell cholinesterase activities in man

<table>
<thead>
<tr>
<th>Method</th>
<th>Plasma</th>
<th>RBC</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔpH (Michel)</td>
<td>0.4</td>
<td>0.8</td>
<td>ΔpH per ml per hour</td>
</tr>
<tr>
<td>pH STAT (Nabb-Whitfield)</td>
<td>2.3</td>
<td>8.0</td>
<td>μM per ml per hour</td>
</tr>
<tr>
<td>ChE-tel (Pfizer)</td>
<td>40</td>
<td></td>
<td>ChE-tel units</td>
</tr>
<tr>
<td>A ChE-tel (Pfizer)</td>
<td>210</td>
<td></td>
<td>A ChE-tel units</td>
</tr>
<tr>
<td>1-Test Cholinesterase (EM Diagnostics)</td>
<td>3.6</td>
<td></td>
<td>Units per ml</td>
</tr>
<tr>
<td>Acholest Test Paper</td>
<td>&gt;20</td>
<td></td>
<td>Minutes</td>
</tr>
<tr>
<td>Dupont ACA</td>
<td>&lt;8</td>
<td></td>
<td>Units per ml</td>
</tr>
<tr>
<td>Garry-Routh (Micro)</td>
<td>7.8 (male)</td>
<td>5.8 (female)</td>
<td>μM –SH per ml per 3 min</td>
</tr>
<tr>
<td>Merckotest</td>
<td>3.0</td>
<td></td>
<td>Units per ml</td>
</tr>
</tbody>
</table>

1 Because measurement techniques vary from laboratory to laboratory, more accurate estimates of "minimum normal" values are usually provided by individual laboratories.

are commonly detectable in the urine of poisoning victims from 12 to 48 hours after the absorption of significant quantities.

The approximate lower limits of normal plasma and red cell cholinesterase activities of human blood, measured by generally available methods, are listed in table 3. Test values below these levels usually indicate excessive absorption of a cholinesterase-inhibiting chemical. (About 3 per cent of individuals have a genetically determined low plasma cholinesterase activity due to generation of an atypical enzyme by the liver.) Whenever possible, a comparison of the "test" sample with a "pre-exposure" value offers the best confirmation of organophosphate absorption: a depression of 25 per cent or more is strong evidence of excessive organophosphate absorption.

Caution: If diagnosis is probable, do not delay treatment until diagnosis is confirmed by blood tests.
3. Occupational pesticide poisonings

Avoidance and emergency management of poisoning

(1) Although organophosphate pesticides vary in toxicologic potential from extreme to low, most of the chemicals of this class used in agriculture are highly toxic. It is important to determine the actual hazard presented by each specific material, and to reinforce protective measures when dealing with chemicals presenting extreme hazards.

(2) The most stringent sanitary and protective measures must be observed when handling the more toxic of these chemicals, either in concentrated or in dilute form. The inhalation of volatile material or of a dust formulation presents an extreme hazard to workers, the prevention of which requires assiduous respiratory protection. Workers must not eat or smoke at the worksite, and must wash their hands thoroughly before touching food or cigarettes.

(3) Contact with foliage residues must be allowed only in accordance with official re-entry safety standards. Daily changes of clean lightweight coveralls offer protection against dermal absorption of crop residues remaining after legal re-entry times.

(4) Workers handling these chemicals must be prepared to recognise the more common early symptoms of poisoning (headache, nausea, weakness, twitches) and the more definitive signs (pinpoint pupils, slow heartbeat). Supervisors must be alert to the occurrence of "flu-like" illness and to disturbances in the mental and emotional state of persons affected by prolonged low level absorption of these chemicals. Workers in whom any degree of poisoning is suspected must be referred for medical examination in the company of an associate or relative.

(5) Blood cholinesterase tests must be carried out as a differential diagnostic test on workers in whom poisoning is suspected. In some circumstances, it is useful to perform the test routinely (every one to three weeks) on regularly exposed workers. Depression of blood cholinesterase activity may be a warning sign of cumulative poisoning from continuing exposure, and
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all personnel should be alerted to unsafe handling practices at the worksite.

(6) First-aid measures in the event of poisoning include: (a) the removal of the victim from further contact with the toxicant (fresh air, removal of contaminated clothing, bath, shampoo); (b) the maintenance of vital functions (open oropharyngeal airway, artificial respiration and intermittent cardiac compression, if needed); and (c) prompt transportation to a treatment centre for the administration of antidotes, while vital functions are maintained.

(7) Excellent antidotes exist for the treatment of poisonings by this class of pesticides: atropine and pralidoxime. (Certain other oxime compounds are also useful, but have not been so extensively tested as pralidoxime.) Both antidotes are absorbed by the gastro-intestinal tract. However, for several good reasons, they are not recommended for use at the worksite except when the plant is very remote from a regular treatment centre. Even then, the antidotes are best given by injection, after which the victim must be transported as promptly as possible to a regular clinic or hospital.

3.5.3. The cholinesterase-inhibiting carbamate insecticides

General chemical structure

![Chemical structure](image)

Commonly used pesticides of this class are (approximately in order of toxicity):

*Highly toxic*: aldicarb (Temik), oxamyl (Vydate), carbophuran (Furadan), methomyl (Lannate, Nudrin), Zectran, methiocarb (Mesurol), dimetilan (Snip Fly Bands).
3. Occupational pesticide poisonings

*Moderately toxic*: propoxur (Baygon), Landrin, carbaryl (Sevin), metalkamate (Bux).

Chemicals of this class are commonly "systemic", that is, to some degree they are taken up by the plant and translocated into the foliage and sometimes into the fruit, where they are toxic to sucking insects.

*Toxicology*

Toxicants of this class cause reversible carbamylation of the acetylcholinesterase enzyme of tissues, allowing the accumulation of acetylcholine at cholinergic neuro-effector junctions (muscarinic effects), and at skeletal muscle myoneural junctions and in autonomic ganglia (nicotinic effects). Poison also impairs the functioning of the central nervous system. The carbamyl-enzyme combination dissociates more readily than the phosphorylated enzyme produced by organophosphate insecticides. The lability tends to mitigate the toxicity of carbamates, but also limits the usefulness of blood enzyme measurements in the diagnosis of poisoning. Carbamates are absorbed by inhalation, ingestion and dermal penetration and are actively metabolised by the liver, and the degradation products are excreted by the liver and kidney.

A few of the carbamate insecticides are formulated in methyl (wood) alcohol. In cases of ingestion of these formulations, the toxicology of the methanol must be taken fully into consideration: severe gastro-enteric irritation, metabolic acidosis and injury to the central nervous system.

*Most frequent presenting symptoms and signs of poisoning*

Symptoms of acute poisoning develop during carbamate exposure or within 12 hours of contact. They include: *headache, dizziness, extreme weakness, ataxia, tiny pupils*, blurred or "dark" vision, muscle *twitching, tremor*, sometimes convulsions, mental confusion, incontinence, unconsciousness; *nausea, vomiting, abdominal cramps, diarrhoea; tightness in chest, slow heartbeat, wheezing, productive cough, sometimes pulmonary oedema; sweating, rhinorrhoea, tearing, salivation*. Severe poisoning may cause sudden unconsciousness
or produce a toxic psychosis. *Respiratory depression* may result from actions of the toxicant and solvent. Continuing absorption at intermediate dosage may cause protracted weakness, anorexia and malaise.

**Confirmation of diagnosis**

Depression of plasma and/or RBC cholinesterase activity is sometimes useful in detecting excessive absorption of toxicants of this class. However, enzyme activities commonly revert to normal within a few hours. They are therefore not reliable detectors of carbamate poisoning, since intoxication may exist when blood cholinesterase activities are normal. The rapid methods for cholinesterase estimation (Acholest, ChE-tel, Merckotest) are more likely to detect depressions. Some carbamates yield metabolites that are measurable in the urine of poisoning victims up to 48 hours after absorption of significant quantities.

The approximate lower limits of normal plasma and red cell cholinesterase activities of human blood, measured by generally available methods, were listed in table 3 above. When test values are below these levels, excessive absorption of a cholinesterase-inhibiting carbamate may be suspected. (About 3 per cent of individuals have a genetically determined low plasma cholinesterase activity, due to generation of an atypical enzyme by the liver.) Whenever possible, a comparison of the “test” sample with a “pre-exposure” value offers the best confirmation of excessive carbamate absorption: a depression of 25 per cent or more is strong evidence of excessive exposure.

**Caution**: If diagnosis is probable, do not delay treatment until diagnosis is confirmed by blood tests.

**Avoidance and emergency management of poisoning**

(1) Because of the range of toxicants exhibited by this group of chemicals, it is important for workers to be aware of the degree of hazard involved in handling each compound.
3. Occupational pesticide poisonings

(2) Stringent sanitary and protective measures (masks, chemically impervious garments) must be enforced during occupational exposure to either concentrated or dilute forms of the more toxic carbamates. Dilute formulations of those having only moderate or low toxicity can be applied without the use of impervious rubber clothing, gloves, boots, and so on. The use of a mask is still advisable if the application is carried out in an enclosed space or in other circumstances where air concentrations may become excessive.

(3) Fields treated with very toxic carbamate insecticides must not be re-entered until foliage residues have degraded to safe levels. Official safe re-entry intervals have been published for toxic carbamates used in agriculture. Daily changes of clean coveralls offer substantial protection to workers whose body surfaces are regularly exposed to foliage residues.

(4) Workers handling these chemicals must be prepared to recognise the more common early symptoms of poisoning (headache, nausea, weakness, twitching, nervousness) and the more definitive signs (pinpoint pupils, slow heartbeat). Supervisors should be aware that workers complaining of extreme fatigue, nervousness, malaise, depression or other ill defined symptomatology may, in fact, be suffering the effects of prolonged absorption of carbamates, without exhibiting the distinctive indications of acute poisonings.

(5) Blood cholinesterase tests are of limited value in monitoring workers for excessive absorption of carbamates. The enzyme reverts so readily to the uninhibited form that depressions of activity can be detected only by using the more rapidly executed tests: ChE-tel, Acholest, Garry-Routh. Even when these are used, negative results do not preclude the occurrence of excessive carbamate absorption. More reliance must be placed, therefore, on symptoms and signs of intoxication.

(6) In the event of poisoning, first-aid measures must include: (a) the removal of the victim from further contact with the toxicant (fresh air, removal of contaminated clothing, bath,
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shampoo); (b) the maintenance of vital functions (open oro-
pharyngeal airway, artificial respiration and intermittent car-
diac compression, if needed); (c) prompt transportation to a
treatment centre for the administration of antidotes, while
vital functions are maintained.

(7) Atropine is an excellent antidote for carbamate poisoning.
It must be given in large doses. Under conditions of intoxica-
tion, it is much more effective when administered by injection
than when given orally (Pralidoxime, the more definitive anti-
dote used in organophosphate poisonings, is contra-indicated
in carbamate intoxications.) Atropine should not be given to
workers as a prophylactic measure. If the worksite is very re-
 mote from the treatment centre, it may be necessary to train
supervisory personnel to inject atropine under emergency con-
ditions.

3.5.4. The nitrophenolic herbicides

General chemical structure

![Chemical structure of nitrophenolic herbicides]

Commonly used chemicals of this class are: dinitrophenol (Chemox
PE), dinitro-orthocresol (DNOC, DNC, Sinox), Dinoseb (DNBP,
DN-289), dinosam (DNAP), DN-111 (DNOCHP), dinoprop, dinot-
erbon, dinoterb, dinosulphon, binapacryl (Morocide, Endosan,
Ambox, Mildex), dinobuton, dinopenton.

Toxicology

These materials should be regarded as highly toxic to man. Most
nitrophenols and nitrocresols are well absorbed from the gastro-
intestinal tract, across the skin, and by the lung, when very fine
droplets are inhaled. Except in a few individuals, they are only
3. Occupational pesticide poisonings

moderately irritating to the skin. They usually produce a yellow stain wherever contact occurs. In common with other phenols, they are toxic to the liver, kidneys and nervous system. The basic mechanism of toxicity is a stimulation of oxidative metabolism in cell mitochondria, by interference with the normal coupling of carbohydrate oxidation to phosphorylation reactions. Increased oxidative metabolism depletes body carbohydrates and fat stores and leads to pyrexia, tachycardia and dehydration. Most severe poisonings from absorption of these compounds have occurred in workers who were concurrently exposed to hot environments. Direct action on the brain causes cerebral oedema, manifest clinically as a toxic psychosis and sometimes convulsions. Liver parenchyma and renal tabules show degenerative changes. Albuminuria, pyuria, haematuria and increased BUN are often prominent signs of renal injury. Agranulocytosis has occurred following large doses of dinitrophenol.

Cataracts have occurred in some chronically poisoned laboratory species. This is a possible, but as yet unconfirmed, hazard in man.

Death by nitrophenol poisoning is followed promptly by intense rigor mortis.

**Most frequent presenting symptoms and signs of poisoning**

*Yellow staining* of skin and hair signifies contact with a chemical of this class. Staining of the sclerae and urine indicate absorption of potentially toxic amounts. *Profuse sweating, headache, thirst, malaise* and *lassitude* are the common early signs of poisoning. *Warm, flushed skin, tachycardia* and *fever* characterise a serious degree of poisoning. *Apprehension, restlessness, anxiety, manic behaviour or unconsciousness* reflect severe cerebral injury. Cyanosis, tachypnoea and dyspnoea occur as a consequence of extreme stimulation of metabolism, pyrexia and tissue anoxia. Weight loss occurs in persons chronically poisoned at lower dosage levels.

**Confirmation of diagnosis**

Unmetabolised nitrophenols and nitrocresols can be identified spectrophotometrically in the serum and urine at concentrations
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well below those necessary to cause poisoning. In addition, many laboratories can analyse for these compounds by gas-liquid chromatography. If poisoning is probable, do not await confirmation before commencing treatment.

Avoidance and emergency management of poisoning

(1) The toxicity of these compounds has been underestimated by workers engaged in vegetation control, leading to avoidable illnesses and fatalities. Stringent protective measures should be provided to personnel exposed to nitrophenols, and the regular use of that equipment must be enforced. Because these compounds impart a deep yellow stain to tissues with which they come in contact, it is possible for workers to recognise direct contact of the chemical with body surfaces. Handling and application techniques should be executed without staining of the workers’ skin. In cases of excessive nitrophenol absorption, the sclerae and urine take on an intense yellow colour. This should be regarded as a sign of incipient poisoning.

(2) Most serious and fatal poisonings have occurred in workers exposed to the hot environments that usually prevail in weed control operations. Adequate protective clothing is likely to exaggerate the heat stress aspect of such labour as it mitigates the threat of poisoning. It is important, therefore, to allow ample periods of rest and relief for workers so engaged, and to supply them with adequate drinking water and electrolytes to support high rates of perspiration and insensible water loss.

(3) By the proper selection of application equipment and techniques (upwind position, long wands, large droplets), it is often possible to minimise the opportunity for personal contact with the herbicide.

(4) Workers experiencing the symptoms and signs of nitrophenol poisoning should be removed from the job immediately and taken to a cool environment. The control of body temperature by sponge baths and cool liquids by mouth greatly enhances the likelihood that the victim will survive the poisoning. If the
3. Occupational pesticide poisonings

victim’s consciousness level is depressed, give nothing by mouth.

(5) There are no chemical antidotes for poisoning by these chemicals. Aspirin and other antipyretic medications should not be used to control fever. Atropine is contra-indicated. Glucose and electrolyte solutions infused intravenously serve to support heat loss mechanisms, to enhance excretion of the toxicant and to protect the liver from toxic action.

(6) Persons poisoned by nitrophenols should stay away from all possible contact with similar chemicals for at least three weeks, to allow the complete recovery of nervous, hepatic and renal tissues. Stringent protective measures should be enforced to prevent subsequent contact.

3.5.5. Pentachlorophenol

General chemical structure

![Chemical structure of pentachlorophenol]

Commonly used commercial products are: PCP, Dowicide-7, Pentachlorol, Pentacon, Penwar, Weedone, Veg-I-Kill, Wood Preserver, Wood Tox 140, Purina Insect Oil Concentrate, Gordon Termi Tox, Usol Cabin Oil, Certified Kiltrol-74 Weed Killer, Ciba-Geigy Ontrack OS 3, 4 or 5, Ortho Triox Liquid Vegetation Killer, Black Leaf Grass, Weed and Vegetation Killer Spray.

Pentachlorophenol has many uses as a weed killer, defoliant, wood preserver, germicide, fungicide and molluscicide. It is an ingredient of many other formulated mixtures sold for one or more of these purposes.
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Toxicology

Pentachlorophenol is irritating to the skin, eyes and upper respiratory mucous membranes. It is efficiently absorbed across the skin, the lung and the gastro-intestinal lining. Like the nitrophenolic compounds, it stimulates oxidative metabolism of tissue cells by uncoupling oxidative processes from the normal stepwise phosphorylation reactions. In common with other phenols it is toxic to the liver, kidney and central nervous system. Impurities in the technical formulation may well be responsible for chloracne.

The majority of severe poisonings have occurred in workers exposed to hot environments. However, a major epidemic of poisoning occurred in newborn infants who absorbed PCP from treated napkins (diapers). Dehydration and metabolic acidosis are important features of poisoning in children.

Albuminuria, glycosuria and elevated BUN reflect renal injury. Liver enlargement has been observed in some cases. Anaemia and leucopaenia have been found in chronically exposed workers, but leucocytosis is more commonly found in acute poisonings.

Most frequent presenting symptoms and signs of poisoning

Irritation of nose, throat, eyes and skin is the most common symptom of exposure to PCP. Severe or protracted exposure may result in a contact dermatitis. Intensive occupational exposure has resulted in chloracne.

Profuse sweating, headache, weakness and nausea are the most consistent presenting symptoms of systemic poisoning by absorbed PCP. Fever is usually present, but may be minimal or absent. Tachycardia, tachypnoea and pain in the chest and abdomen are often prominent. Thirst is usually intense, but may be masked by nausea and vomiting. Declining mental alertness may progress to stupor and/or convulsions. Protracted exposure results in weight loss from increased basal metabolic rate.

Confirmation of diagnosis

PCP can be measured in blood, urine and adipose tissue by gas-liquid chromatography. A few parts per thousand million can
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usually be found in the blood and urine of persons having no
known exposure. Based on studies of persons occupationally ex-
posed to PCP, symptoms of systemic toxicity probably do not
appear in adults until blood and urine concentrations reach at least
one part per million (0.1 mg per cent, or 1,000 parts per thousand
million).

If poisoning is strongly suspected on the grounds of exposure
history, symptoms and signs, do not postpone treatment until
diagnosis is confirmed.

Avoidance and emergency management of poisoning

(1) Avoidable deaths and serious illnesses have occurred because
the toxic hazard of PCP was underestimated. By reason of the
chemicals' volatility, substantial amounts can be absorbed by
inhalation of vapour from seemingly insignificant environmen-
tal sources. Exposed workers must use respirators fitted with
activated charcoal cartridges, or they must wear helmets sup-
plied with pure air from an outside source or from an air-
purifying device.

(2) Irritant effects on the eyes, nose and throat are at least a
nuisance and at worst a cause of disabling injury. If regular
heavy exposure of workers is unavoidable, they should be pro-
vided with chemically impervious suits and helmets supplied
with clean air from an outside source or from a backpack air
purifier. Frequent showers and relief from the contaminated en-
vironment may be the most practical means of protecting work-
ers who have intermittent or accidental exposures. Masks and
goggles offer some protection, if properly used, although great
care must be taken to keep the inner surfaces of these articles
free of PCP. The chemical is readily absorbed across the skin
occluded by rubber, and its irritant effects are magnified thereby.

(3) Copious flushing with clean water should be used to relieve
eyes irritated by PCP. Water and mild soap should be used
to clean contaminated skin.
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(4) A worker experiencing early symptoms of systemic PCP poisoning should be removed immediately to a cool environment. Cool liquids should be given by mouth, as tolerated, unless the victim’s consciousness level is depressed. If he is obtunded, give nothing by mouth. Sources of toxicant available for continuing absorption (such as contaminated clothing, boots and hat) must be removed, and if PCP is present on the body surfaces, bathing and shampooing should be used to remove it.

(5) Do not give aspirin or other antipyretic medication to reduce temperature. Atropine is contra-indicated.

(6) If any degree of poisoning is suspected, the victim should be taken promptly to a treatment centre for medical management. The condition of the victim may deteriorate rapidly after the appearance of the initial symptoms.

(7) Intravenous infusions are frequently useful in controlling body temperature and in accelerating excretion of the toxicant. These measures can often save a life, even though they are not specifically antidotal.

3.5.6. Chlorophenoxy compounds

Approximate general chemical structure

Commonly used pesticides of this class are: 2,4-D (Weedone), 2,4,5-T, 2,4,5-TP, Silvex (Kuron), 2,4-DB (Butyrac, Butoxone), Erbon, Fenac, 2,4-DEP, MCPA, MCPB, MCPP (Mecoprop), Weedestron, Esteron, Estone, Dacamine, Weed-B-Gon, Weed-No-More, Weed-Out, Ded-Weed, Weed or Brush-Rhap, Broadleaf Weed Killer, Dandelion Killer, Vegetation Killer, Chickweed and
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Clover Killer. There are several hundred commercial herbicide preparations which include one or more chlorophenoxy compounds. They can usually be identified in the active ingredient description on the product label.

Toxicology

The chlorophenoxy acids, salts and esters thereof are mildly to severely irritating to the skin, eyes and respiratory and gastrointestinal linings. They are absorbed across the gut wall, the lung and the skin. They are not significantly fat storable: excretion occurs within hours, or at the most within days, primarily in the urine.

These compounds apparently have very low toxic potential for most individuals. Human subjects have tolerated 0.5 g ingested doses daily for two to four weeks without adverse effects. Paradoxically, several cases of peripheral neuropathy have been reported in workers after seemingly minor exposures to 2,4-D. It is not known whether these individuals were peculiarly predisposed, or were exposed concurrently to other unidentified neurotoxic materials. In a few individuals, local depigmentation has apparently resulted from prolonged and repeated dermal contact with chlorophenoxy materials.

Given in large doses to experimental animals, 2,4-D causes vomiting, diarrhoea, anorexia, weight loss, ulcers of the mouth and pharynx, and toxic injury to the liver, kidneys and central nervous system. Myotonia (stiffness and incoordination of hind extremities) develops in some species and is apparently due to damage to the central nervous system; demyelination has been observed in the dorsal columns of the cord, and EEG changes have indicated functional disturbances in the brains of heavily dosed experimental animals. A single victim of accidental ingestion of over 7 g of 2,4-D exhibited direct toxic damage to skeletal muscle, manifest as myoglobinuria and creatininuria. Other chemicals in the ingested formulation may have contributed to the unusual pathology in this case. In another isolated instance of extreme dosage, convulsions apparently occurred before death.
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Most frequent presenting symptoms and signs of poisoning

Irritation of the skin follows excessive contact with many of these compounds. Protracted inhalation of spray is likely to irritate the nose, eyes, throat and bronchi, causing disagreeable local burning sensations and cough. Prolonged inhalation has also caused dizziness and ataxia, usually of a transient nature. When ingested, these compounds irritate the mouth and throat, and usually cause enough gastro-intestinal irritation to induce prompt emesis. Chest pain from oesophagitis is common. Abdominal pain and tenderness and diarrhoea usually ensue. Absorption of large quantities of chlorophenoxy herbicide may produce fibrillary muscle twitching, skeletal muscle tenderness and myotonia (stiffness of muscles of extremities). Respiratory insufficiency from muscle weakness occurred in one victim of accidental poisoning (which may have involved additional toxicants).

Confirmation of diagnosis

Gas-liquid chromatographic methods are available for detecting and measuring many of the chlorophenoxy compounds in urine. These analyses are useful in confirming and assessing the magnitude of toxicant absorption. Urine samples should be collected as soon as possible after exposure because these materials may be almost completely excreted in 24 to 72 hours (depending on the dose). The analyses can be done at special laboratories operated by, inter alia, agricultural research centres, commercial chemical companies and environmental protection agencies.

Avoidance and emergency management of poisoning

(1) The majority of adverse effects from compounds of this class of chlorophenoxy acids, salts and esters are due to irritative properties. Certain esters are particularly irritating to skin, eyes and respiratory mucous membranes; other products cause little trouble. In any case, measures to protect workers from dermal, facial and inhalation contact should be implemented, particularly when the concentrated materials have been handled.
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(2) In general, chlorophenoxy compounds have low systemic toxicity. However, workers inhaling drift from application operations have sometimes complained of dizziness, headache and nausea. These symptoms usually resolve in a short time, if the worker is simply removed from the contaminated worksite.

(3) A few cases of peripheral neuropathy have been reported following minor occupational contact with chlorophenoxy compounds. It cannot be stated definitely whether these cases occurred in peculiarly predisposed individuals or were actually due in part to other neurotoxic substances. In any case, the remote possibility of this complication in exposed workers should be recognised.

(4) Eyes contaminated with these herbicides should be flushed with clean water. Vigorous scrubbing with soap and water removes dermal contamination. Persons developing illness during spray operations should be removed to fresh air and allowed several hours to recover. Thereafter, they should either be provided protection from drift inhalation or discouraged from continuing work that involves exposure to chlorophenoxy compounds.

(5) In rare cases, the chlorophenoxy compounds (like certain chlorophenolic germicides) have caused depigmentation of the skin after very prolonged contact.

3.5.7. Dipyridyl compounds (paraquat, diquat, morfamquat)

General chemical structures

![Paraquat and Diquat structures](image-url)
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\[
\text{Morfamquat}
\]

Commercial formulations. The highly polar dipyridyl compounds are available commercially as halide and dimethyl sulphate salt solutions. Both are used as contact herbicides, diquat being particularly effective against water weeds. Both absorb strongly to plant tissues and soil particles. Concentrates are more likely to cause poisoning than the more dilute agents sold over the counter.

Paraquat products: Paraquat Cl, Dual Paraquat, EM-7217, Gramoxone S, Weedol and Dextrone X are all concentrates containing 20 per cent paraquat ion. Preeglone extra and Gramonol are concentrate mixtures with other herbicides. Ortho Spot Weed and Grass Killer contains 0.2 per cent paraquat ion.

Diquat products: Aquakill, Aquacide, Heavy Duty Weed Control, Aquatat, Aquatic Weed Killer, Reglone, Vegetrole, Watrol and Di-Kill Vegetation Killer are all packaged as concentrates. Preeglone extra is a concentrate mixture with paraquat.

Morfamquat products: Morfoxone, PP-745.

Toxicology

The dipyridyl compounds bind to, and injure, the epithelial tissues of the skin, nails, eyes, nose, mouth and respiratory and gastro-intestinal tracts. Concentrated solutions cause inflammation and sometimes necrosis and ulceration of mucosal linings.

The toxicology of paraquat has been much more thoroughly investigated than that of other dipyridyls. Diquat appears to be
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substantially less toxic than paraquat. Little is known of the effects of morfamquat.

The consequences of ingestion of paraquat concentrate (which accounts for nearly all of the mortality and serious morbidity from these compounds) are unique. Because the dosages necessary to produce poisoning in men have varied over a wide range, all cases of ingestion should be treated vigorously, irrespective of the estimated intake.

For 24 to 72 hours after ingestion, there is often very little indication of systemic toxicity, evidence of poisoning being limited to pain, vomiting and diarrhoea from irritation of the gastrointestinal linings. Between 48 and 72 hours, kidney damage may be apparent from proteinuria, haematuria and rising BUN and creatinine levels, while liver damage is reflected in hyperbilirubinaemia, and in increased serum GOT, GPT, alkaline phosphatase and LDH enzyme activities. Between 72 and 96 hours after ingestion, indications of a diffuse toxic pneumonitis often appear.

The histopathology of this process is complex, consisting in part of intra-alveolar oedema and haemorrhage, in part of a rapid proliferation of bronchiolar epithelium and in part of focal atelectatic changes, possibly secondary to impaired gas exchange, due to patchy consolidation, oedema and alveolar collapse, together with increased airway resistance. Pulmonary fibrosis may follow the acute pneumonitis (from one to two weeks after ingestion). For this reason, surviving patients should be examined for permanent lung injury as long as three to six months after poisoning. The injuries to the kidney and liver are commonly reversible, ameliorating even as the pulmonary lesion worsens. The lung injury is usually, but not always, fatal.

Electrocardiographic evidence of toxic myocarditis is commonly observed, and cranial nerve palsies have been reported as toxic manifestations.

Lens cataracts are reported to occur in laboratory animals dosed with diquat.
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Most frequent presenting symptoms and signs of poisoning

Skin irritation, drying and cracking follow untreated skin contact with dipyrindyl concentrates. Discoloration and irregularity of fingernails commonly occur in workers regularly exposed to paraquat concentrates. Delayed conjunctivitis and keratitis develop between 12 and 48 hours after contact of the chemical with the eye. The inhalation of spray droplets irritates the nose and throat and sometimes caused nosebleed.

Following ingestion of paraquat concentrate, the earliest symptoms and signs are due to mucosal irritation and ulceration of the gastro-intestinal tract. Pain (oral, substernal, abdominal), vomiting and diarrhoea (sometimes melaena) occur. Generalised muscle aching is reported. Early symptoms are sometimes so mild that vigorous treatment is improperly delayed.

Between 48 and 72 hours after ingestion, indications of renal and hepatic insult appear. Albuminuria, haematuria, pyuria, elevated BUN and creatinine occur. Oliguria may develop and this signals severe poisoning. Jaundice and elevations of serum GOT, GPT, alkaline phosphatase and LDH reflect hepatocellular injury. The effects on the liver and kidney are generally reversible.

Usually between 72 and 96 hours after ingestion, indications of lung injury appear. Cough, dyspnoea and tachypnoea often progress in the manner of a diffuse pneumonitis. In some cases, severe pulmonary oedema occurs and persists for several days. The pulmonary lesion is usually, but not invariably, fatal.

Confirmation of diagnosis

Qualitative and quantitative methods for paraquat and diquat in urine are available at some toxicology laboratories.

Management of occupational injury

(1) Although over 100 deaths have occurred as a result of ingestion of paraquat (intentional or accidental), occupational poisonings by these compounds have been extremely rare. Injuries to handlers and applicators have consisted essentially of nose-
3. Occupational pesticide poisonings

bleeds (from irritation of the nasal mucous membranes) and fingernail discoloration and atrophy (occurring after direct contact with concentrated solutions). These effects can be avoided by proper application techniques (long wands, upwind position), relatively simple respiratory protection (adequate to trap aerosol droplets) and by the use of impervious gloves when solutions are being transferred, diluted or mixed.

(2) Ingestion of paraquat is likely to cause death. An important feature of the systemic poisoning is the long delay (two to five days) between ingestion and the appearance of pneumonic symptoms that progress almost always to death. If measures are taken during this time to eliminate the toxicant from the gut (emesis, lavage and catharsis) and to bind the residue with charcoal, the victim's chances of survival are greatly increased.

(3) Although the dipyridyls are apparently absorbed very inefficiently by the skin and respiratory tract, the inherent toxicity of the compound dictates the necessity for sound hygienic and sanitary practices in occupational exposure situations. Clean coveralls should be worn daily; impervious gloves should always be worn in formulation and transfer operations; and showers and hand-washing facilities should be used frequently to minimise dermal absorption.

(4) Extremely stringent precautions should be taken to prevent the storage of these chemicals in unlabelled containers and to prevent access to them by children and unauthorised adults.

(5) Little occupational experience is available for the evaluation of hazards from diquat and morfamquat. The little that is available indicates that these dipyridyls are less hazardous than paraquat.

3.5.8. Dimethyldithiocarbamate fungicides

General chemical structure

\[
\text{(CH}_3\text{)}_2\text{N-C-S} \quad \text{Metal} \quad \text{S-C-N-(CH}_3\text{)}_2
\]
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Commonly used chemicals of this type are:

*Tetramethyl thiuram disulphide:* Thiram (Arasan, Thiramad, Thirasan, Thylate, Tirampa, Pomasol forte, TMTDS, Thiotex, Fernasan, Nomersan, Tersan, TUADS).

*Metallodimethylthiocarbamates:* Ziram, Pomasol Z forte (zinc), Ferbam (iron).

Toxicology

Many compounds of this class are irritants and sensitisers. They may exacerbate allergic skin and respiratory disease and sensitise otherwise normal individuals to subsequent contact with similar chemicals.

The two types of fungicide listed above are metabolised in a manner similar to disulfiram (tetraethyl thiuram disulphide), used to condition individuals against beverage alcohol. The molecule is first cleaved to yield two of alkyl dithiocarbamate, then further degraded to dialkyl amine and carbon disulphide. The metabolites are powerful inhibitors of multiple key sulph-hydryl enzymes in the liver and central nervous system. Carbon disulphide is neurotoxic in its own right. To this extent, the toxicology of these fungicides can be expected to bear some resemblance to that of disulfiram, whose effects have been most thoroughly explored. Experiments on animals indicate that thiram is more toxic than the medicinal disulfiram. Even so, systemic reactions (excluding irritation and sensitisation) to the fungicides themselves have been rare.

The systemic effects that occur in response to these compounds must be considered in two categories: (a) those following absorption of the toxicant alone; and (b) those resulting from the ingestion of alcohol following the absorption of a dithiocarbamate compound.

Given to laboratory animals in extreme doses, disulfiram itself has caused gastro-intestinal irritation, demyelinisation of the central nervous system and necrosis of the liver, spleen and kidney parenchyma. Functional and anatomical damage to the central nervous system has been demonstrated in rats on high chronic
dietary intakes of the iron and zinc dimethyldithiocarbamates. Peripheral neuropathy and psychotic reactions have occurred in alcohol-abstinent individuals on disulfiram regimens (ingestion of several hundred mg daily). A possible role of the metabolite carbon disulphide has been suspected in these neurotoxic reactions.

Illness following the combined intake of disulfiram and alcohol is due primarily to inhibition of the liver enzymes necessary for the oxidation of acetaldehyde to acetic acid. Peripheral vasodilatation is the main pathophysiological feature of the disulfiram–alcohol reaction, presumably due to high tissue levels of acetaldehyde. This may occasionally lead to shock, and even more rarely to myocardial ischaemia, cardia arrhythmias, failure and death. Animal experimentation has supported certain other biochemical mechanisms of toxicity involving reaction products of ethanol and disulfiram.

Most frequent presenting symptoms and signs of poisoning by dialkyldithiocarbamates

*Itching, redness* and *eczematoid dermatitis* have resulted when sensitive or predisposed individuals come into contact with these compounds. Persons excessively exposed to air-borne fungicides have suffered *upper respiratory congestion, hoarseness, cough* and even *pneumonitis*. When large amounts have been ingested, *nausea, emesis* and *diarrhoea* ensue. *Hypothermia* and *ataxia* are characteristic. *Muscle weakness*, progressing to a condition of *ascending paralysis* and finally *respiratory paralysis*, can be anticipated from animal toxicological studies based on extreme dosage.

The *reaction to ethanol* which follows absorption of disulfiram is characterised by *flushing, sweating, pounding headache, sensation of warmth, weakness, congestion of upper respiratory and conjunctival membranes, dyspnoea, hyperpnoea, chest pain, tachycardia, palpitation* and *hypotension*.

Respiratory distress may resemble *asthma*, and, in some instances, *respiratory depression* has been life-threatening. *Emesis* commonly occurs. Severe reactions may result in *shock, unconsciousness* and/or *convulsions*, and therefore threaten coronary
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insufficiency in predisposed individuals. It must be emphasised that the absorption of sufficient fungicidal compounds to cause a severe reaction to ethanol would occur only under exceptional circumstances.

Confirmation of poisoning

The native pesticides are too rapidly metabolised in the body to permit their detection in the blood or urine. There are biochemical methods for measuring blood acetaldehyde to confirm an ethanol–dithiocarbamate reaction.

Management of excessive exposures

(1) Even though these compounds have rarely caused serious injury or illness, their inherent capacities for sensitisation and inhibition of critical enzymes are such that workers must be provided with effective means for limiting contact with them. Skin surfaces should be protected with coveralls and impervious gloves. Masks should be used in work environments (mainly enclosures) wherein exposure by inhalation is likely to be significant. When these compounds are applied in the open air, avoidance techniques (such as upwind position and long wands) may offer the most satisfactory and practical protection.

(2) Individuals having chronic skin diseases (especially eczema), asthma, severe hay fever or simply a tendency to react severely to irritants should not be employed in work involving contact with these chemicals. This restriction applies to workers contacting treated foliage as well as formulators and applicators of these fungicides.

(3) Workers suffering from hypersensitivity reactions of the skin or respiratory system must be treated by established medical procedures. These individuals must thereafter be removed permanently from work environments contaminated with thiocarbamate chemicals, as no available protective techniques are efficient enough to prevent the minimal contact that triggers off hypersensitivity responses.
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(4) Personal protection from the thiocarbamate fungicides should always be adequate to prevent absorption of the amounts that are necessary to provide an ethanol–thiocarbamate reaction. However, conditions at the worksite may occasionally, through accident, allow such an excessive uptake. For this reason, workers should be made aware of the possibility of disagreeable symptoms after consuming alcoholic beverages. The occurrence of such a reaction in a worker should provoke an intensive effort to reduce the exposure of the workforce.

3.5.9. Urea-, uracil- and triazine-based herbicides

Approximate general chemical structures

\[ \text{Urea derivatives} \]

\[ \text{Uracil derivatives} \]

\[ \text{s-Triazine derivatives} \]

\( X = \text{Halogen} \quad R = \text{Alkyl} \)

Common commercial products of this type are:

**Urea derivatives**: monuron (Monurex, Telvar), diuron (Di-on, Diurex, Karmex, Vonduron), linuron (HOE 2810; Afalon, Lorox, Sarclex).

**Uracil derivatives**: bromacil (Borea, Hyvar X, Hyvar X-L, Borocil IV, Urox HX or B, Isocil), terbacil (Sinbar).

**Triazine derivatives**: atrazine (Aatrex, Atranex, Gesaprin, Primatol A), simazine (Princep, Primatol S, Simanex, Gesatop), propazine (Milogard, Gesamil, Primatol P), prometone (Pramitol, Gesafram, Prometon), atraton (Atratone), prometryn (Caparol, Gesagard,
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Primatol Q, Prometrex), ametryn (Evik, Ametrex, Gesapax), desmetryne (Semon), terbutryn (Igran, Shortstop E), cyanazine (Bladex, Scogal), cyprazine (Outfox).

Toxicology

Adverse effects have occurred only rarely as a result of human contact with these herbicides. Most injuries reported have been cases of skin irritation after prolonged contact. The improbability of severe systemic poisoning is suggested by the high acute oral LD$_{50}$ values listed in the following table:

<table>
<thead>
<tr>
<th>Herbicide</th>
<th>LD$_{50}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>monuron</td>
<td>3,600</td>
</tr>
<tr>
<td>diuron</td>
<td>3,400</td>
</tr>
<tr>
<td>linuron</td>
<td>1,500</td>
</tr>
<tr>
<td>bromacil</td>
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<td>terbacil</td>
<td>5,000</td>
</tr>
<tr>
<td>cyanazine</td>
<td>182</td>
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<table>
<thead>
<tr>
<th>Herbicide</th>
<th>LD$_{50}$</th>
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<tbody>
<tr>
<td>atrazine</td>
<td>3,080</td>
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<tr>
<td>simazine</td>
<td>5,000</td>
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<tr>
<td>propazine</td>
<td>5,000</td>
</tr>
<tr>
<td>prometone</td>
<td>2,980</td>
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<tr>
<td>atraton</td>
<td>1,465</td>
</tr>
<tr>
<td>ametryn</td>
<td>1,110</td>
</tr>
<tr>
<td>desmetryne</td>
<td>1,390</td>
</tr>
<tr>
<td>cyprazine</td>
<td>1,200</td>
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</table>

When administered at very high dosage levels to laboratory animals and to sheep and cattle, some of these chemicals have been shown to injure the nervous system, liver and kidney, and to cause increased permeability of capillaries. Anaemia and altered adrenal function have also been detected in animals to which extreme doses of certain triazine compounds have been given. These effects have not been observed in persons exposed occupationally or by reason of accidental ingestion. In varying degrees, these herbicides are likely to produce irritation of the gut if ingested in substantial quantities. They are efficiently absorbed from the intestine. (Absorption across the skin and lung has not been investigated.) Following absorption, they are partially metabolised; the native chemicals and metabolites are promptly excreted by the kidney and liver. In cases of accidental ingestion in man, the hazards of petroleum distillate solvents may equal or exceed hazards presented by the active herbicidal ingredients.
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Possible presenting symptoms and signs of injury

Some compounds of these classes cause irritation of the eyes and mucous membranes, particularly if direct contact is protracted. Nausea, vomiting and diarrhoea can be expected to result from ingestion of large quantities.

Confirmation of absorption

Some industrial, university and government laboratories can detect these compounds or their metabolites in the urine of persons who have absorbed significant amounts. These laboratories can be reached through health departments or poison control centres.

Avoidance of excessive exposure

(1) The hands and other potentially contaminated body surfaces of workers handling the herbicides should be adequately protected. The actual need for protection depends on the formulation: liquid concentrates are more likely to cause dermal injury than granules. Care must be taken to avoid saturation of clothing, gloves and boots with spilled chemical. Skin irritation is much more likely to occur when the contaminated skin surface is “occluded” by fabric or rubber.

(2) Masks and goggles are required during the application of distinctly irritant chemicals. Spray formulations of the uracil derivatives in particular have this property.

(3) Illness which apparently results from excessive exposure to these chemicals should be managed by removing the worker from the contaminated worksite until he has fully recovered. No specific antidote or method of medical management is available. If symptoms persist for more than a few minutes, the victim should be taken to a doctor for clinical evaluation.
3.5.10. Liquid and gas fumigants

Structures of common fumigants

(1) Enzyme poisons (extremely toxic)

| PH₃ | HCN | CS₂ | CH₂ = CH—C ≡ N |
| Phosphine | Hydrogen | Carbon | Acrylonitrile |
(From metal cyanide disulphide phosphide)

(2) Small-molecular halogenated hydrocarbons (highly toxic)

| CH₃Br | BrCH₂—CH₂Br | ClCH₂—CH₂Cl |
| Methyl bromide | Ethylene dibromide | Ethylene dichloride |

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<td>Cl</td>
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<th>HC≡C—C—H</th>
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Dichloropropene | Carbon tetrachloride | Chloropicrin

(3) Miscellaneous (moderately toxic)

<table>
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<tr>
<th>F</th>
<th>SO₂</th>
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Sulphuryl fluoride (Vikane) | Paradichlorobenzene | Naphthalene

Toxicology

The fumigant gases hydrogen cyanide, carbon disulphide, phosphine and acrylonitrile are absorbed rapidly by the lung. They move readily into tissue cells where they attach to critical binding sites of vital respiratory enzymes. Their high toxicity,
3. Occupational pesticide poisonings

rapid absorbability and relatively inoffensive non-irritating properties make them particularly hazardous poisons.

The small molecular halogenated hydrocarbons are central nervous system depressants, myocardial irritants and hepatotoxins. Some (methyl bromide and chloropicrin, especially) are irritants. The liquids are readily absorbed across the skin. Liver injury may occur as a result of protracted absorption of some toxicants at rates insufficient to cause acute poisoning.

Naphthalene causes injury when ingested, causing haemolysis and a consequent acute tubular necrosis. Paradichlorobenzene is less toxic, but occasionally produces sensitisation. Vikane causes central nervous system (including respiratory) depression and other manifestations of acute injury to the central nervous system after prolonged inhalation.

Confirmation of diagnosis

Various gas-chromatographic techniques are applicable to samples of air (including exhaled air) for the identification and measurement of the halogenated hydrocarbons. Somewhat less sensitive and specific methods are available for the other fumigant gases.

Avoidance of poisoning

Extraordinarily efficient respiratory protection must be worn by workers exposed to fumigant air concentrations of these chemicals. If ordinary respirators are worn, the masks must fit very tightly over the face, and the air-cleansing charcoal must be of optimal quality and fineness. Activated charcoal canisters are preferable to filters attached to masks, and still better protection is afforded by helmets supplied with compressed air from back pack units (Scott air packs).

The concentrated liquid fumigants must be handled with impervious rubber protective equipment (gloves, aprons, boots) to avoid dermal contamination from spillage.

Ample time must be allowed after fumigant operations to allow the dissipation of the gases. In some circumstances, analytical air
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monitoring may be required. Although the odour of some fumi-
gants may provide a useful warning of persistent gas, it must be
remembered that this sensory modality fatigues rapidly on con-
tinuing stimulation by odorous chemicals, leaving the exposed
worker unaware of noxious concentrations.

Emergency management

(1) Remove the poisoned individual from the fumigated atmo-
sphere.
(2) Sustain respiration and circulation by mouth-to-mouth re-
suscitation and cardiac resuscitative manipulation, as needed.
(3) Administer oxygen by mask, if available.
(4) Sustain the vital functions until the victim can be delivered
to a treatment centre.

3.5.11. Arsenicals

Structures of common arsical compounds

(1) Extremely toxic (trivalent inorganic arsicals)

\[
\begin{align*}
O & = \text{As} - O - \text{As} = O \\
\text{Arsenic trioxide} & \\
O & \\
\parallel & \\
\text{Cu} - (O - C - \text{CH}_3)_2 & \\
\text{Copper aceto-arsenite} & \\
(\text{Paris green}) & \\
K & = O - \text{As} = O \\
\text{Potassium arsenite} & \\
\text{H} & \\
\text{As} & \rightarrow \\
\text{H} & \\
\text{Arsine (gas)} &
\end{align*}
\]
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(2) Moderately to highly toxic (pentavalent organic arsenicals)

\[ \begin{align*}
\text{CH}_3 & \quad \text{OH} \\
\text{As} & \quad \text{O} \\
\text{O} & \quad \text{ONa}
\end{align*} \]

Mono sodium methyl arsonate

\[ \begin{align*}
\text{CH}_3 & \quad \text{ONa} \\
\text{As} & \quad \text{O} \\
\text{O} & \quad \text{ONa}
\end{align*} \]

Di sodium methyl arsonate

\[ \begin{align*}
\text{H}_2\text{N} & \quad \text{As} \\
\text{O} & \quad \text{OH} \\
\text{O} & \quad \text{OH}
\end{align*} \]

Arsanilic acid

\[ \begin{align*}
\text{CH}_3 & \quad \text{CH}_3 \\
\text{As} & \quad \text{O} \\
\text{O} & \quad \text{OH}
\end{align*} \]

cacodylic acid

(3) Moderately to highly toxic (pentavalent inorganic arsenical)

\[ \begin{align*}
\text{OH} & \\
\text{HO—As═O} & \\
\text{OH}
\end{align*} \]

Arsenic acid

Common commercial products of this type are:

Inorganic arsenicals: arsenic trioxide (white arsenic), sodium arsenite (Acme Weed Killer, Atlas A, Penite, As-655 Weed Killer, Kill All), copper arsenite (Paris green), copper ammonium arsenite (Chemonite), arsenic acid (Zotox Crabgrass Killer, Dessicant L-10, Lincks Liquid Di-met, Pax Total, Purina Top Grass and Weed Killer).

Organic arsenicals: MSMA (Ansar 170, Bueno, Weed-E-Rad, Ansar 529, Broadside, Crabgrass Dallis Grass Killer, Daconate, Fertilome Nutgrass Killer), DSMA (ANSAR 8100, Biochecks, Burpee Crabgrass Killer, Chipco Crab Kleen, Clout, D Krab
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R+Prills, DMS, E Krab R, Greenfield Crabgrass and Dandelion Killer, Sears Liquid Crabgrass Killer, Lawn Weed Killer, Proturf Monocot Weed Control), cacodylic acid (Silvisar 510), sodium cacodylate (sodium dimethyl arsinite, Phytar 560, Acme Weed Killer), ammonium methane arsenate (AMA, Ansar 157, Super Crab E-Rad, C-4000, Antrol Crabgrass Killer, Crabgrass Broadleaf Killer, Systemic Crabgrass and Broadleaf Killer), methane arsenic acid (MAA, Ortho Crabgrass Killer).

Toxicology

Although the pentavalent arsenicals are generally less toxic than the inorganic trivalent chemicals, all poisonings by arsenic-containing substances should be regarded as serious threats to life and health. To some degree, the pentavalent compounds undergo reduction in the gut and/or body tissues to trivalent forms. Some absorption of solid arsenical compounds may occur by dermal or pulmonary routes, but the great majority of poisonings occur as a result of ingestion. Intestinal absorption is generally efficient. Most absorbed arsenic is excreted by way of the kidneys, a lesser proportion by the liver and gut. Arsine gas is absorbed rapidly by the lung; an arsenic metabolite is excreted in the urine.

The toxicology of arsine gas (absorbed by inhalation) is unique, in that it causes haemolysis and secondary acute renal tubular necrosis. (Arsine is not used as a pesticide, but is involved in the manufacture of organic arsenicals.)

Trivalent arsenicals bind critical sulph-hydryl-containing enzymes in tissues. When taken up from the gut, they injure the splanchnic vasculature, causing colic and diarrhoea. Once absorbed, they produce toxic injury to the liver, kidney, bone marrow, brain and peripheral nerves. Liver injury is manifest as hepatomegaly, jaundice and an increase in circulating hepatocellular enzymes LDH and GOT. Renal damage is reflected in albuminuria, haematuria, pyuria, cylindruria, then azotaemia. Acute tubular necrosis may occur in severe poisoning. Injury to blood-forming tissues can take the form of agranulocytosis, aplastic anaemia, thrombocytopenia

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or pancytopenia. Toxic encephalopathy may be manifest as speech and behavioural disturbances. Peripheral neuropathy occurs in both acute and chronic forms. The inhalation of arsenic dusts may cause bronchitis or pneumonitis.

Sequelea of arsenic poisoning include cirrhosis, hypoplastic bone marrow, renal insufficiency and peripheral neuropathy. Excessive exposures to arsenicals have caused cancers of the skin and various epithelial tissues.

*Most frequent presenting symptoms and signs*

**Acute arsenic poisoning, solid compounds.** Colic, burning abdominal pain, vomiting and watery or bloody diarrhoea are the primary manifestations of ingestion of arsenical poisons. More severe symptoms follow the ingestion of inorganic arsenicals than result from the pentavalent organics. Headache, dizziness, muscle spasms, delirium and sometimes convulsions reflect direct injury to the central nervous system plus extracellular electrolyte disturbances and shock. An odour of garlic on the breath and faeces helps to identify the responsible toxicant. Shock, toxic nephrosis and hepatitis (hepatomegaly and jaundice) and neurological injury (delirium, paralysis, respiratory depression) may progress to a fatal outcome.

**Subacute arsenic poisoning, solid compounds.** Dosages less than those necessary to produce severe acute symptoms are known to cause chronic headache, abdominal distress, salivation, low grade fever and persistent symptoms of upper respiratory irritation. Stomatitis and garlicky breath are characteristics.

**Chronic arsenic poisoning, solid compounds.** Prolonged low intakes of arsenic cause peripheral neuropathy (paresthesiae, pain, anaesthesia, paresis, ataxia), encephalopathy (apathy), varied dermatological disorders (keratosis, pigmentation, eczemas, brittle nails, loss of hair) and toxic hepatitis (hepatomegaly, sometimes progressing to cirrhosis with ascites). Weakness and vulnerability to infections may result from bone marrow depression. Local oedema (frequently eyelids) is characteristic of some chronic poisoning cases.
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_Acute arsine poisoning, gas_. The gas causes _haemolysis_ of red blood cells in addition to inhibiton of sulph-hydryl respiratory enzymes. Haemolysis causes _haemoglobinemia_ and _haemoglobinuria_. This, in turn, causes _acute tubular necrosis_. Early symptoms of poisoning (chills, weakness, burning sensation) are followed by abdominal cramps, vomiting and prostration, as renal function deteriorates to _anuria_.

**Confirmation of diagnosis**

The measurement of 24-hour urinary excretion of arsenic probably represents the most satisfactory means of confirming excessive arsenic absorption, although methods for blood arsenic concentration are available. Persons on ordinary diets usually excrete less than 20 μg per day, but diets rich in seafood may generate as much as 200 μg per day. Excretions in excess of 100 μg per day should be viewed with suspicion, and the measurement should be repeated. Excretions in excess of 200 μg per day should be regarded as indicative of absorption at a potentially toxic dosage.

The qualitative Gutzeit test for arsenic in the urine is available at most hospital laboratories, and is useful in identifying acute poisonings promptly.

Chronic storage of arsenic can be detected by analysis of the hair or fingernails.

The haemoglobinuria caused by arsine is identified by the presence of the pigment in fresh urine from which intact red cells are absent.

3.5.12. _Acetanilide-, acetamide-, carbanilate- and anilide-based herbicides_

_Approximate general chemical structures_

![Chemical structures](image-url)
3. Occupational pesticide poisonings

\[
\begin{align*}
&\text{Alachlor} \\
&\text{Propanil} \\
&\text{Chlorpropham}
\end{align*}
\]

Common commercial products are: propachlor (Ramrod), allidochlor (Randox, CDAA), alachlor (Lasso), chlorpropham (Chloro IPC, CIPC, Furloe), propanil (DPA, Stam, Propanex).

Most technical formulations of these materials are in petroleum distillates.

Toxicology

These newer herbicides exhibit low systemic toxicity in laboratory rats (lowest oral LD\(_{50}\) of those listed is 700 mg per kg). They are, however, irritating to the skin, eyes and mucous membranes. Propachlor and alachlor appear to have sensitising properties. Severe skin reactions have occurred in sensitised individuals.

Whatever systemic toxicity these compounds have appears only at high dosage levels. Adverse effects from accidental ingestion by man have not been reported. In all likelihood, substantial doses would cause gastro-intestinal irritation.

Most common presenting symptoms and signs of undue exposure

Irritation of the skin and membranes of the upper respiratory tract is the principal adverse effect of contact with these compounds.
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Exaggerated reactions on repeated contact (sensitisation) occur in some individuals. Propachlor has been the principal offender. Sensitisation can result in acute and chronic skin injury, and in protracted irritation of the nose, eyes and throat.

Nausea, abdominal distress and diarrhoea might be expected to result from ingestion of these compounds.

3.5.13. Anticoagulant rodenticides

Approximate chemical structures of principal classes

Commonly used commercial rodenticides are:

_**Coumarin type**: warfarin (Kypfarin, Warf-42, D-Con, Warficide, Prolin), coumafuryl (Fumarin), Dethmor, Rax.

_1,3-Indandione type_: diphacinone, or diphenadione (Ramik), chlorophacinone (Drat, Caid, Liphadione, Microzul, Ramucide, Rotomet, Raviac, Topitox), pindone (Pivalyn, Pivacin, Tri-ban, Pival), valone (PMP).

These materials are commonly added to baits or dissolved in small amounts of water for pest rodents to drink; 100 g of the prepared commercial baits must be ingested to yield 25 mg of anticoagulant. Rodenticide “drinks” are made by adding dry concentrate (0.54 g of active ingredient per 100 g of powder) to specified volumes of water. The poison in the concentrate is coated on sugar or sand to facilitate measurement and handling.

In view of the intense distress which they cause to animals, the use of anticoagulent rodenticides is discouraged in some countries.
**Toxicology**

Gastro-intestinal absorption of these toxicants is efficient, beginning within minutes of ingestion, and continuing for two to three days afterwards. Apparently, warfarin can also be absorbed across the skin, although the circumstances under which this has occurred in the past are extraordinary.

Both types of anticoagulant depress the hepatic synthesis of substances essential to normal blood clotting: prothrombin (factor II) and factors VII, IX and X. The antiprothrombin effect is best known, and provides the basis for the detection and assessment of clinical poisoning. Direct damage to capillary permeability occurs concurrently. In rare instances, coumarin-type anticoagulants have caused ecchymosis and extensive skin necrosis in man for reasons not related to excessive dosage.

Unlike the coumarin anticoagulants, the indandiones cause symptoms and signs of neurological and cardiopulmonary injury in laboratory rats; these often lead to death before haemorrhage occurs. These actions may account for the somewhat greater toxicity of this class of anticoagulant. Cardiopulmonary and neurological symptoms and signs have not been reported in human poisonings.

Lengthened prothrombin time for a toxic dose can be expected to appear within 24 hours of toxicant ingestion and reach a maximum 36 to 72 hours afterwards. Without intervention, hypoprothrombinaemia may persist for 10 to 15 days, depending on the agent and dosage. Prothrombin depression will occur in response to doses that are much lower than those necessary to cause haemorrhage.

**Most frequent presenting symptoms and signs**

In most instances of accidental ingestion of anticoagulant baits, victims have remained asymptomatic due to the small dosage taken. Even in cases involving the ingestion of substantial doses, hypoprothrombinaemia occurs without symptoms of poisoning. Haemorrhage appears only when extraordinary amounts have been
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absorbed. In these cases, the anticoagulants were either taken deliberately, were absorbed over long periods through neglect of elementary standards of hygiene or were ingested by starving indigents who used quantities of rodent bait for food.

Victims of large doses exhibit haematuria, nosebleed, haematoma, bleeding gums and melaena. Abdominal pain and back pain probably reflect haemorrhage in the abdominal and retroperitoneal tissues. Weakness occurs as a result of anaemia. Renal colic often complicates severe haematuria. Nasal and gastro-intestinal haemorrhages have caused death from exsanguination.

Methods for confirming diagnosis of poisoning

An increase of the prothrombin time (Quick) reflects a reduction in serum prothrombin concentration, and occurs in response to a physiologically significant absorption of these toxicants. This widely available clinical test offers a sensitive and reliable diagnostic method for the detection of a toxic effect of these compounds. Readily detectable change in prothrombin time appears within 24 to 48 hours of ingestion.

Methods are available in a few laboratories for the measurement of warfarin and its metabolites in human urine.

3.6. Other chemical substances in agriculture

3.6.1. Mineral fertilisers

The normal growth and development of plants requires the presence in the soil of a large number of chemical elements. Very often the soil is deficient in nitrogen, phosphorus and potassium. Because of this, the commonest mineral fertilisers contain one, two or all three of these elements. Some substances are used to improve the physico-chemical properties of the soil: limestone and dolomite, for instance, neutralise excess acidity, which is harmful to plants; and gypsum is used to improve the properties of salty soils.
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Mineral fertilisers which contain nutritive ingredients in small amounts are called micro-fertilisers. These fertilisers contain boron, copper, manganese, zinc and other trace elements.

Nitrogenous fertilisers

Ammonia. A colourless gas with a pungent odour (threshold of perception 0.037 mg/l); coefficient of solubility in water 762.6 (20°C), 655.8 (28°C).

Ammonia is used for liquid fertilisers (ammoniates) and in an aqueous solution. The aqueous solution easily emits ammonia. Free ammonia in air quickly turns into ammonium carbonate or is absorbed by water. The aqueous solution has alkaline properties due to the formation of ammonium hydroxide.

Ammonia irritates especially the upper respiratory tract. In large concentrations it affects the central nervous system, causing spasms. The changes in the nervous system are probably caused by a deficiency of blood oxygen.

In man, high concentrations cause profuse lachrymation and pain in the eyes, choking, violent fits of coughing, giddiness, abdominal pains, vomiting and retention of urine. After exposure to high concentrations, patients are sometimes violently stimulated to the point of raging delirium and are unable to stand up. Severe respiratory and circulatory disorders are observed. A very few hours (sometimes only a few minutes) after poisoning, the patient may die of cardio-asthaenia. More often, death occurs a few hours or days after the accident, due to laryngeal or pulmonary oedema. There may be chemical burning of the eyes and upper respiratory tract. Acute poisoning may result in the dimming and even perforation of the cornea and the loss of an eye, hoarseness or total loss of voice, chronic bronchitis and expectoration of blood. Cases involving paralysis and deafness have also been reported.

In small concentrations, ammonia causes milder irritation of the eyes and of the mucous membrane of the nose, sneezing, salivation, slight nausea and headache, reddening of the face, tendency to perspire, pain in the chest and an urge to urinate.
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In man, a concentration as small as 0.1 mg/l causes irritation. The smallest concentration immediately causing irritation of the throat is 0.28 mg/l, that causing irritation of the eyes is 0.49 mg/l, and that causing a cough is 1.2 mg/l. It is possible (but difficult) to endure a concentration of 0.25 mg/l for one hour without after-effects. According to other data, work with ammonia is possible at concentrations of 0.07 to 0.14 mg/l, difficult at concentrations of 0.14 to 0.21 mg/l and impossible at concentrations of 0.35 to 0.7 mg/l. The latter concentrations may endanger life.

If ammonia water is splashed into the eyes, these must immediately be washed (while wide open) with copious amounts of clean water. If the patient experiences sharp pains, one or two drops of a 1 per cent solution of novocaine or one drop of a 0.5 per cent solution of dicaine with adrenaline (1 : 1,000) could be used. Protective spectacles should be worn.

If the skin is affected, it should be washed with clean water and a compress consisting of a 5 per cent solution of acetic, citric, tartaric or hydrochloric acid should be applied.

In cases of ammonia poisoning through the respiratory tract, the patient must breathe fresh air and should inhale warm water vapour (preferably with vinegar or a few crystals of citric acid added) and a 10 per cent solution of menthol in chloroform. He should drink warm milk and sodium bicarbonate. Sodeine (0.015 g) or dionine (0.1 g) should be given. In the event of asphyxia, the patient should be given oxygen, which is inhaled until the dyspnoea or cyanosis is reduced. In the case of a spasm of the glottis, the neck area should be kept warm, warm water vapour should be inhaled and a subcutaneous injection of atropine should be given (1 ml of a 0.1 per cent solution). If respiration is interrupted or stops, artificial respiration should be given (without pressure on the thorax).

Sodium nitrate (sodium or Chilean saltpetre). Colourless crystals; solubility 88 g/100 ml water (20°C). Decomposes at 380°C into NaNO₂ and O₂.
After a year of work, workers employed in the extraction of nitrate in northern Chile were found to be suffering from swellings of the skin, chiefly on the palms and on the soles of the feet. These swellings attained 1.3 cm in diameter and were painful when pressed. In a number of cases, the swellings developed into cancerous tumours in 12 to 15 years, even in workers who had left the job long before.

The fact that nitrates can be reduced in the body into nitrites often leads to the formation of methaemoglobin. In a group consuming water containing 50 to 100 mg of nitrates per litre, the number of persons with an increased methaemoglobin content in the blood rises sharply, compared with a group consuming pure drinking water.

*Potassium nitrate (or potassium saltpetre).* Colourless crystals; solubility 3.15 g/100 ml water (20°C).

After only a few months of work, workers in contact with potassium nitrate have been found to be suffering from ulceration of the mucous membrane of the nose and even perforation of the nasal septum.

Preventive measures consist in the protection of the respiratory organs from dust (by the wearing of respirators) and in protecting the skin from direct contact.

*Calcium nitrate (Norwegian or calcium saltpetre).* Colourless crystals; solubility in water (anhydrous) 54.8 g/100 ml water (18°C).

The commercial product is in the form of fine granular powder and contains 75 to 80 per cent calcium nitrate, plus a small amount of lime.

Only the irritant and caustic effects of the commercial product are significant. These take the form of reddening of the skin, itching and ulcers, which are sometimes deep and extensive and which heal slowly and leave large scars. Parts of the skin showing even the slightest wound, scratch or other damage are primarily affected.

Preventive measures consist in washing carefully after work, and
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in applying barrier creams and fatty greases or in wearing protective clothing during work.

Ammonium nitrate (ammonium saltpetre). Colourless crystals, easily diffused in air; solubility 122 g/l water (0°C).

Ammonium nitrate is used as a fertiliser. It has irritant effects on the skin, which undergoes violent itching, reddening around the follicles, herpes-like swellings and reddening on the back of the hands and forearms. Contact with small wounds or fissures causes a burning pain.

The preventive measures are the same as for potassium nitrate.

Phosphatic fertilisers

This class of fertiliser contains phosphorus pentoxide in a form that can be assimilated by plants.

Phosphorite meal is produced by crushing and grinding natural phosphorites. It is used as a fertiliser and as a neutralising additive to superphosphate.

Simple (or single) superphosphate is obtained through the reaction of natural phosphates with sulphuric acid. It is a mixture of calcium, magnesium, aluminium and iron phosphates and contains phosphoric acid, calcium sulphate, silicon dioxide, etc. It has a strong acid reaction. Concentrated phosphoric fertilisers obtained through the reaction of natural phosphates with phosphoric acid are known as double (triple) superphosphate. This contains two or three times as much assimilable phosphorus pentoxide as simple superphosphate. Sulphates are present only as contaminations.

Enriched superphosphate is obtained by processing phosphates with a mixture of sulphuric and phosphoric acids. Through its composition and properties it occupies an intermediate position between simple and double superphosphate.

Precipitate is the name given to secondary calcium phosphate, which is used as a fodder additive and a phosphoric fertiliser. It is obtained by the neutralisation of phosphoric acid by lime.

Ammophos is a mixture of primary and secondary ammonium phosphate. It is used as a concentrated mixed fertiliser and is
3. Occupational pesticide poisonings

obtained by the neutralisation of phosphoric acid by ammonia. A mixture of ammophos with ammonium sulphate (sulpho-ammophos) is also used.

The complex fertilisers also include the nitrophosphorus mixtures of ammonium nitrate with potassium compounds and with phosphates. The thermal phosphates are products of the processing at high temperatures of the natural phosphates in a mixture with sand, limestone, alkali compounds, etc., and include defluorinated fused magnesium and thermo-alkali phosphates. They are used as fertilisers. The defluorinated phosphates are also used as auxiliary feeds to provide mineral intake for animals.

3.6.2. Toxic properties of the salts of phosphoric acid and of commercial products containing them

The salts of phosphoric acid can give rise to a general poisoning only if taken in very large doses. A fair number of observations have been made of the irritant and caustic effects of acid salts (for example, simple superphosphate, partly due to free phosphorus dioxide) on the mucous membranes and on the skin, especially in contact with fissures and wounds in the skin. If superphosphate dust comes into contact with the eyes it causes violent irritation of the conjunctiva, oedema of the eyelids, dimming and sometimes even perforation of the cornea and prolapse of the iris. The neutral salts (at least as regards tricalcium phosphate) apparently have no significant noxious effects on the skin.

Toxic effects of apatites and superphosphate depend mainly on the admixture of fluorine. When apatite dust was fed to animals, changes developed in the dental and osseous tissue; superphosphate dust also caused paresis and paralysis. The following symptoms are observed: lowering of the bactericide capacity of the blood, adiposis of the heart muscle, catarrhal gastritis, dystrophic-necrotic processes in the liver and kidneys, and manifestations of interstitial lymphocytic nephritis.

When animals were made to inhale the dust of an apatite concentrate, nodular changes were also found in the pulmonary tissue.
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The development of pneumoconiosis (apatitosis) was also estab-
lished among workers who had inhaled apatite dust. Apatitosis is
characterised by its early appearance (two to five years after a
person starts work) and its slow development. The disease takes a
benign course; it does not reach stage III.

Individual protective equipment to be provided includes anti-
dust respirators. Preventive inhalations include a 0.5 to 1 per cent
solution of sodium bisulphite and a 0.8 to 1 per cent solution of
sodium chloride.

Basic slag

Basic slag is used as a phosphoric fertiliser. The cold slag is
crushed and ground into meal, which takes the form of a dark
grey powder. More than 55 per cent of the dust particles are less
than 5 µm in size. Ground basic slag contains 45 to 50 per cent
calcium oxide, 12 to 22.5 per cent phosphorus pentoxide, 12 to
16 per cent ferrous and ferric oxide, 5 to 10 per cent silicon dioxide,
1 to 2 per cent aluminium oxide and 2 per cent sulphur and other
elements.

The dust of ground basic slag causes severe inflammation of the
lungs, mostly lobular (basic slag pneumonia). It begins suddenly
with shivering, a rise in temperature and a stitch in the side.
Sometimes illness is preceded by fatigue, headache, lack of appetite,
bronchitis, fits of coughing, and so on. At the height of the illness
the patient’s condition is serious and he is often partly unconscious,
dyspnoea is observed, the sputum is viscous (at first dark grey in
colour and later resembling thick honey, but unlike the “rusty
sputum” of membranous pneumonia). There is blood in the sputum.
Sometimes the patient suffers from violent pulmonary bleeding
which may even result in death. In 50 per cent of cases there is
albumen in the urine. Recovery is slow. There are indications that
the greater the capacity of the mucous membrane of the nose to
stop the dust, the longer the inflammation takes to develop and the
milder it is.

Prevention consists in wearing respirators and protective goggles
to protect the upper respiratory tract and the mucous membrane of the eyes from dust.

3.6.3. *Calcium cyanamide*

A white crystalline preparation; solubility 2.5 g/100 ml water (20°C).

The commercial product contains 50 per cent calcium cyanamide, up to 25 per cent calcium oxide, 13 per cent carbon, etc. It is a fine dust-like powder, dark grey in colour, with an odour of acetylene. Calcium cyanamide decomposes in water. Cyanamide, dicyanamide and other products form when calcium cyanamide is exposed in storage to moisture and carbon dioxide in the air. Admixtures may be present and these may emit toxic gases such as phosphine.

The oral lethal dose in adults is 40 to 50 g. Sensitivity to calcium cyanamide increases sharply when alcohol is consumed. The effects of calcium cyanamide are characterised by the violent irritant action of lime and the influence of cyanamide, which strikes at the respiratory and vasomotor centres.

Calcium cyanamide, when inhaled via the respiratory organs, causes reddening (with a bluish tinge) of the skin of the face, the neck and the upper part of the breast and back, and of the mucous membrane of the eyes and pharynx, with shivering and an accelerated pulse rate. Tachycardia and a feeling of anxiety develop. Cyanhaemoglobin may appear in the blood.

On the skin, calcium cyanamide causes dermatitis and sometimes eczema.

The symptoms of chronic poisoning are giddiness, headache, irritability, rapid weariness and loss of appetite.

As a protection against calcium cyanamide, workers should wear special clothing of impervious material, gloves, protective goggles and anti-dust respirators. The skin is protected by an application of neutral fatty greases. The consumption of alcoholic drinks during work must be strictly prohibited.
3.6.4. Fuels, lubricating oils and products of the incomplete combustion of fuel

Various products of petroleum processing are used as fuels and lubricating oils in machines used in agriculture (tractors, self-propelled combine harvesters, motor trucks, stationary engines).

Petroleum is a complex mixture consisting primarily of hydrocarbons but also containing organic compounds. A distinction is made between methane, methano-naphthene, naphthene, naphthenoaromatic and aromatic petroleum, according to the class of hydrocarbons which predominates. Different petroleums also contain different amounts of oxygen, nitrogen and sulphur compounds.

Crude oil is distilled into petroleum fractions or "cuts" which have specific boiling temperature ranges—for example, liquefied petroleum gas, white spirits, kerosene, fuel oil.

Skin diseases induced by petroleum products are treated by conventional methods. Above all, the patient must be removed from contact with irritants.

Chronic poisonings in agricultural workers have not been widely reported and their occurrence is very unlikely; however, in mild cases of acute poisoning, the patient must be removed from the contaminated atmosphere. If there is nervous excitement, the treatment should consist of bromides, valerian drops and rest.

The best method of preventing injuries to health resulting from the use of various petroleum products in agriculture is to ensure that these products do not come into contact with clothing and with uncovered parts of the body. If petroleum products contaminate the clothing, it must be cleaned; if they come into contact with exposed parts of the body, the products must be removed and the affected parts of the body washed.

3.6.5. Products of the incomplete combustion of fuel in motor engines

Exhaust gases (carbon monoxide, nitrogen oxides, aldehydes, carbon dioxide, vapours of unburnt fuel, tars (containing 3,4-benzpyrene in certain cases), and so on) may contaminate the air inhaled by persons working on tractors and other machines. Many
of the substances detected in exhaust gases have toxic properties. Carbon monoxide is harmful to the central nervous system. Formaldehyde, acrolein and others affect the central nervous system and irritate the mucous membranes. The vapours of nitrogen dioxide and nitrogen oxide irritate the mucous membranes of the eyes and the respiratory tract.

Unburnt hydrocarbons give exhaust gases an unpleasant odour and also irritate the mucous membrane.

Tests of the air in tractor cabs and in the breathing zone of drivers on cableless tractors have revealed the presence of carbon monoxide, hydrocarbons, sulphur gas, nitrogen oxides, formaldehyde and carbon dioxide. In rare cases, carcinogen 3,4-benzpyrene was found in very small amounts. Most of the substances were detected in insignificant amounts and in a minority of the tests. Carbon monoxide was found most frequently in concentrations two to three times higher than the permissible maximum. In such cases the tractor drivers complained of headache, fatigue, and so forth.

*Carbon monoxide*

Boiling point –192°C; burns with a bluish flame.

Carbon monoxide is formed by the incomplete combustion of substances containing carbon.

It is a tasteless, colourless gas with a very faint odour (usually imperceptible), slightly reminiscent of the smell of garlic. A mixture of two volumes of carbon monoxide and one volume of oxygen explodes when ignited. At high temperatures carbon monoxide has reducing properties.

In the cabs of motor vehicles, the concentration of carbon monoxide may reach 0.05 mg/l or more, increasing as long as the vehicle remains in operation. Carbon monoxide entering the passenger compartment of a bus together with exhaust gases sometimes causes very severe poisoning.

In kerosene-burning tractors, concentrations of 0.2 to 0.5 mg/l of carbon monoxide have been found in the driver's breathing zone. In petrol-driven tractors and on self-propelled combines, concentrations of 0.02 to 0.7 mg/l have been observed.
The danger of carbon monoxide poisoning in garages is very great unless adequate preventive measures are taken. A powerful engine can emit 28 l of carbon monoxide in one minute, which is sufficient to build up a lethal concentration in five minutes in a single-vehicle garage with the doors closed.

**General nature of poisoning.** Carbon monoxide acts by displacing oxygen from the oxyhaemoglobin in the blood, forming carboxyhaemoglobin. Thus the blood becomes unable to carry enough oxygen from the lungs to the tissues. The reduced content of blood oxygen (anoxaemia) leads to asphyxiation. When this happens, the oxyhaemoglobin content may fall from 18–20 per cent to 8 per cent and the difference between the oxyhaemoglobin content in the arterial and venous blood may be reduced from 6–7 per cent to 2–4 per cent. The ability of carbon monoxide to displace oxygen from a compound with haemoglobin is explained by its close affinity for the latter.

**Acute poisoning.** The chief symptoms are loss of consciousness, convulsions, dyspnoea and asphyxia, but often the forms of poisoning vary greatly. The initial symptoms are particularly important. The inhalation of small concentrations (up to approximately 1 mg/l) causes well known subjective symptoms to appear (often immediately), which may serve as a warning of the danger.

These symptoms may vary in their intensity and nature and in the sequence of their appearance. The head becomes heavy, there is a feeling of constriction in the forehead and this is followed by violent pain in the forehead and temples. Flashing and mist appear in the eyes and there is throbbing in the temples. Often the most pronounced symptoms are giddiness, a buzzing noise in the ears, peculiar sensations in the skin, trembling, a feeling of weakness, accelerated pulse rate and vomiting.

Carbon monoxide poisoning chiefly affects the central nervous system. As anoxaemia develops, a person gradually loses his powers of reasoning. The co-ordination of movements is disturbed. A person who has been poisoned cannot walk straight or write evenly; gradually he loses control of his legs and then of his hands.
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The functioning of the sensory organs is disturbed (hearing being affected last). The sense of pain disappears at an early stage (while still conscious, persons poisoned by carbon monoxide are insensitive to burns). Memory, especially logical and visual memory, deteriorates, sometimes to such an extent that the patient ceases to recognise persons nearby. Very often the patient cannot recollect the circumstances of the poisoning.

Serious sensory disorders have been described, especially visual disorders such as diplopia, reduction of the field of vision, colour blindness, temporary loss of sight and, in some cases, total blindness. Disorders of pupil reaction, paralysis of the eye muscles and, in certain cases, nystagmus have been recorded.

**Chronic poisoning.** The first symptoms usually appear two to three months after the start of work involving contact with carbon monoxide. The workers complain of noise in the head and headache (especially during work and in the morning), giddiness (especially when looking up), a feeling of intoxication, a tendency to fatigue, a weakening of memory and attention, apathy and irritability, emaciation, lack of appetite, insomnia at night and drowsiness during the day, pallor, greyness of skin, obsessive fears, deep depression, dyspnoea, tachycardia, pains around the heart, in the chest and in the sides, neuralgic pains, a disposition to perspire, an increased urge to urinate, and sometimes a fainting condition after work.

**First-aid treatment.** In the case of acute poisoning, the very first measure is the immediate and longest possible inhalation of oxygen, in order to displace carbon monoxide from the haemoglobin and restore blood oxygenation. The patient should be removed from the contaminated environment at once. If respiration stops or is seriously depressed, artificial respiration must begin as soon as possible and oxygen should be administered. If there is no pulse and/or if the heart stops, the cardiac region should be massaged rhythmically at the same time.

All first-aid measures should continue until normal respiration and blood circulation are restored.
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4.1. Diseases due to climate

Agricultural operations are generally carried on in the open air, and most of them are closely related to climatic conditions.

Although technology in agriculture has made remarkable progress during the past few decades, the specific characteristics of agriculture which determine safety and health conditions have remained almost unchanged. Ploughing, sowing, harrowing, hoeing, harvesting, and so on, are definitely seasonal operations, so that agricultural workers are sometimes exposed to unfavourable weather (excessive heat or cold, rain, snow, and so forth). The meteorological conditions that characterise the climate and the seasons (temperature, humidity, wind, sunshine, and above all their sudden changes) may affect the workers' health unless suitable precautions are taken. Meteorological conditions may also considerably reduce working capacity if the physiological limits of thermal comfort are much exceeded. Climatic conditions may vary widely from season to season and from one geographical region to another.

Of all the climatic factors that may exert an influence on the workers' organism, two are of special practical importance: excessive heat, and excessive cold.

4.1.1. Hot climates

The ill effects of work in a hot climate are associated with the deficiency or exhaustion of the thermoregulatory mechanisms.

In a temperate climate, when the environmental temperature is lower than that of the body, the organism gets rid of the surplus heat produced by metabolic processes and muscular activity by radiation and convection. In a hot climate, besides the heat resulting from metabolic processes, the organism has to get rid of heat from the environment also. A hot climate thus imposes extra work
on the organism. If to this is added the heat from muscular metabolism during work (and the more arduous the work is, the greater the heat from muscular metabolism), it will be easy to understand that this combination of heat sources imposes a very great strain on the organism.

The characteristics of a hot climate differ from region to region and even within the same region. Thus the tropical zone includes regions with a hot, dry climate and regions with a hot, humid climate; again, temperatures may differ by day and by night or in the different seasons, or may show no appreciable variation. The hot climate is also characterised by intense light and electrostatic conditions that are very different from those in temperate regions. The most trying conditions occur in hot regions where the temperature differences between day and night and between season and season are small, and in very humid regions with little air movement.

In a hot climate, a man’s heat balance is maintained essentially by the evaporation of sweat. The requisite conditions for this physical regulation of temperature are, first, abundant production of sweat, and second, the possibility of evaporating it.

Exposure to a hot climate brings about an increased capacity for secreting sweat. This process is effected by a reflex stimulation of the sudoriferous glands to secrete, and a redistribution of blood in the circulatory system. The vessels of the skin dilate and the blood of the interior circulatory system is diverted to the periphery, thus helping to lower the internal temperature. The congestion in the skin encourages the activity of the sweat glands. The diversion of the blood to the skin is set off by a reflex starting in the nerve endings of the skin, which are stimulated by the rise in temperature of the blood irrigating it.

Sweating eliminates large quantities of salt and water. The balanced consumption of liquids and salt helps the organism to maintain its heat balance.

The heart output, which is at first reduced by the peripheral vasodilatation, increases following the diversion of interstitial and tissue liquids into the circulatory system and an acceleration of the heartbeat.
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The evaporation of sweat depends on the relative humidity of the air, convection and the clothing worn.

Inurement to heat reduces the thermoregulatory effort that the organism has to make. The heart rate is lower and the sweat poorer in salt. In unacclimatised persons, the concentration of sodium chloride in sweat is one-third, or even one-half, of its concentration in blood. After acclimatisation it is only one-tenth.

The diversion of water from the kidneys causes oliguria which they do not easily support. Persons whose cardiovascular system is not in a satisfactory condition are not able to transfer internal heat quickly enough to the skin, and at the same time carry on the oxygenation needed to satisfy the requirements of intensified metabolism.

Persons suffering from skin diseases such as ichthyosis and eczema have deficient perspiration.

The physical effort combined with the thermoregulatory effort demanded by the climate makes demands on the same organs, that is, the sudoriferous and the cardiovascular systems. Formulae exist for calculating the thermal stresses on manual workers. It is recommended that the rectal temperature be taken to determine the worker's ability to continue in his job. A temperature of 39°C is said to be the maximum beyond which work should not be continued. The insufficient supply of blood and oxygen to the muscles following the diversion of the circulation to the periphery reduces working capacity. The insufficient supply of blood to the brain causes fatigue and inattention and leads to mistakes in working and to accidents.

Thus the pathology created by the hot environment results from the hydroelectrolytic imbalance in the organism, the insufficiency or the inefficacy of perspiration, the exhaustion of the heart and a rise in body temperature above physiological limits.

Heat cramps

Heat cramps may appear some hours after physical work in a hot environment is begun. The external temperature need not exceed that of the body, and even direct exposure to the sun is not
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an essential condition for provoking these cramps. They have, in fact, been observed among persons working in a cold environment who were too heavily clothed.

Symptoms. The symptoms are painful muscle contractions. The pain ceases when the muscular spasm ends.

Aetiology. The aetiology of the disorders resides in the loss of sodium chloride from the body liquids as a result of excessive sweating. In the muscle cells a relative or an absolute loss of the salt is observed. This is probably not the only cause of the syndrome, for it is rarely observed in patients in whom the amount of chlorides has been reduced by a diet poor in salt, by a diuretic treatment or by hyperhydration.

The symptoms can be produced by a massive ingestion of water after profuse sweating. This massive intake reduces the concentration of salt in the blood.

Diagnosis. Diagnosis is confirmed by painful muscular contractions, especially at the extremities and in the abdomen, without a large rise in internal temperature and without other general symptoms exhibited by persons working in a hot environment.

Prophylaxis. Gradual training for physical work in a hot environment and the addition of 0.1 per cent sodium chloride to water or effervescent drinks, or additional salt in the diet, will prevent the occurrence of cramps.

Treatment. This consists in administering saline water containing from 2 to 4 g of salt per litre (the concentration is higher than in prophylaxis). In acute cases in which the pains are very sharp, the treatment must begin with an intravenous injection of physiological serum. If the pains persist, morphine hydrochloride may also be slowly injected intravenously.

Anhydrotic exhaustion due to heat

Symptoms. The onset of the disease is foreshadowed by certain prodromes: giddiness, difficulty in breathing, loss of appetite and
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frontal headaches. Patients complain of fatigue and palpitations during work. The skin of the trunk and limbs is dry. Perspiration is normal on the face, the neck, the palms of the hands and the soles of the feet and in the armpits and groin. The salt content of the sweat is high—a proof that the sweat glands are exhausted. The skin is usually affected by a miliary dermatosis. Diuresis is normal, the density of the urine is low and, in spite of a certain lack of salt, there is no dehydration.

Aetiology. The clinical picture suggests a breakdown of the body’s defences against heat as a result of failure of the sudoriferous system. The dermatoses caused by heat may be responsible for this syndrome because the blockage of the sweat glands leads to ineffectual perspiration when the sweat fails to reach the skin.

Prophylaxis. Maceration of the skin by sweat and irritation by clothes must be avoided. Good-quality soap should be used. The effects of the disease could be lessened if patients spent a few days in a cooler climate after a stay of two months in a hot region.

After inurement to heat, the severity of recurrent lesions diminishes.

Treatment. The patient should spend two months in a cooler place or stay there until sweating returns to normal.

Dermatoses caused by heat

Miliary dermatosis. Miliary dermatosis (punctiform dermatosis; also called prickly heat, miliaria ruba, miliaria pustulosa or tropical lichen) is a skin eruption that is very common in hot and humid tropical regions where sweating is a permanent process. It appears especially at places that are rubbed, beginning with small pimples that change into blisters. Each pimple is surrounded by a zone of erythema. These lesions are pruriginous and produce a stinging sensation. A secondary infection may appear on scratches and purulent lesions. There are also areas of peeling.

In chronic cases, the skin is thickened and erythematous, and
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on this background papulous lesions (pimples) appear and disappear.

Heat dermatosis results from a blockage of the sudoriferous canals; the sweat secreted cannot reach the surface of the skin and the canals burst under the pressure of the liquid, which then spreads through the thickness of the skin, causing an irritation.

The purpose of treatment is to allow the sudoriferous system to rest. The patient should stay in a cool place where he no longer has to sweat. In these conditions the blockage symptoms disappear in a week or two. The application of anhydrous lanoline may help. Astringents (such as a 1:2,000 solution of mercury chloride in 95 per cent alcohol) often give good results also. The skin can then be dusted with zinc oxide, or a calamine lotion may be applied several times a day.

_Tropical lichen._ This dermatosis appears more especially after the miliary form. The skin of the trunk and limbs is covered with small excrescences, 1 mm in diameter, on which there are blisters containing a clear liquid. The lesions become more apparent after exposure to heat. Perspiration is reduced and the concentration of salt in sweat is increased.

The patient should rest in a cool place.

_Heat exhaustion_

This is a syndrome characterised by a rapid fall in blood pressure (vasomotor collapse) in persons doing arduous physical work in a high temperature.

_Symptoms._ The disease may be foreshadowed by prodromes. Some days before it appears, the patient complains of headaches, weakness, loss of appetite, nausea, giddiness, disturbed vision and anxiety. Severe vomiting may occur. The patient also suffers from muscular cramps, accelerated pulse, dilated pupils and a pale, cold and damp skin. The blood pressure is lower. Collapse may ensue. Chloraemia is reduced. There is a haemoconcentration with oliguria. The quantity of urine excreted falls and its density rises. It may contain albumen and cylinders; however, urinary chlorides

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are less than 2 g/l. In serious cases there may be shock with anuria and acute uraemia. The disease may begin with a collapse.

Aetiology. The considerable loss of salt and water is at the root of this syndrome. In persons who do much manual work in a hot climate and who do not consume enough liquid and salt to replace the losses caused by profuse sweating, the circulatory system is unable to meet the simultaneous demands of the skin, muscles and brain.

Diagnosis. Differential diagnosis for heat stroke is based on the persistence of perspiration and on the usual absence of a definite rise in body temperature.

Prophylaxis. Subjects who grow thin or who have a high urinary concentration with low chloruria should consume more salt and several litres of liquid.

Treatment. With mild forms of the disease, the patient is laid down in a cool place with his clothes unbuttoned, and given cool saline drinks. In serious cases the patient is put to bed and given salt and liquids to restore his hydroelectrolytic balance. In the event of shock, plasma and physiological serum are given every four hours. A close watch should be kept on the hydroelectrolytic balance; repeated analyses of the chloruria will reveal the moment when urinary chlorides begin to increase. The intravenous treatment can then be interrupted and liquids can be given by mouth. After the clinical signs have disappeared, the patient’s circulatory system can be carefully examined.

Heat stroke

This disease may occur independently or be a sequel to heat exhaustion. It may appear during work in a high temperature, but also in the rest period following such work or after prolonged exposure to heat. It may also be the result of overproduction of body heat after eating too much meat, or be associated with hyperthyroidism. Acute febrile affections may cause heat stroke by inhibiting
perspiration. Thus, in a bout of malaria, heat stroke may be considered to be a complication.

The symptoms appear some minutes after the cessation of sweating or evaporation of sweat. There is very considerable rise in body temperature.

**Symptoms.** The subject experiences a feeling of heat with headaches, dizziness, weakness, vomiting, diarrhoea, and sometimes abdominal pains, vasomotor collapse with hypotension, tachycardia and dyspnoea. In the severest cases there may be spells of mental confusion, uncertainty of gait, delirium and coma. Sphincteric incontinence has been observed. Death may ensue because of shock or the rise in temperature. A rectal temperature of over 41°C is a serious sign. When the rectal temperature is between 40.5°C and 41.5°C, the patient may reach 43°C or 44°C. The skin is dry and hot, the face is red, the pupils are dilated and there is no sweating. A leucocytosis up to 20,000 m³ may be observed. In serious cases the number of platelets falls. Albumen, cylinders and sometimes ketonic bodies are found in the urine. There may be a rise in blood urea. The venous pressure is high and the right heart is dilated. The electrocardiogram shows a tachycardia, a sinusoidal irregularity, a flattening or even an inversion of the T wave and a diminished deflection of the ST segment.

**Aetiology.** The cause of the disease is the insufficient removal of heat, resulting from the exhaustion of the perspiration system on which demands have been made for too long. This system cannot function continuously at high pressure without rest.

The cold nights of some tropical regions (or air-conditioning installations) enable the sweat glands to recover their functional capacity. In addition, the relative humidity of the air is of great importance because, even if the sudoriferous system is intact and perspiration is sufficient, the latter is effective only if adequate evaporation is possible.

The incidence of heat stroke in hot climates increases with the duration of the heat.
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Pathology. The lesions found in autopsies are cerebral oedema, with destruction of nerve cells (especially in the cortex), necrosis of liver and kidney cells, oedema, congestion and haemorrhages in various organs (brain, heart, kidneys, liver).

Diagnosis. The diagnosis is confirmed by high temperature in a patient who is comatose or in a pre-comatose state, with dry and hot skin as a result of exposure to heat. Differential diagnosis should be practised for cerebral haemorrhage and other diseases with which there is a marked rise in temperature (first stages), infectious diseases, malaria and tubercular meningitis.

Prognosis. This is unfavourable in alcoholics and people who are old, obese or in a bad state of health. The fatality rate may be very high if treatment is not given immediately. Death may ensue in less than an hour. If the subject can be kept alive for 24 to 48 hours, he may be saved. The lesions of the nervous system consequent upon either high temperature or haemorrhages may leave sequelae. In a few cases hemiplegia, disturbed memory, listlessness, childish behaviour and sphincteric incontinence may be found.

Prophylaxis. Dwellings should be so designed that they do not retain heat. Rest should be adequate. Clothing should be light, and alcohol, violent physical exercise and overeating avoided. Workers who have to work in a very hot environment should be carefully chosen: they should be young and their physical and mental health should be good. The principal contra-indications are febrile states, gastro-intestinal troubles, cardiovascular complaints, dermatoses, insomnia and alcoholism.

Recurrences are serious; it is therefore advisable to move victims of heat stroke to cooler regions and to keep them there for a long enough period.

Treatment. Urgent treatment is called for. The temperature must be lowered by all possible means and as quickly as possible to prevent brain injury. The patient should immediately be put into a cold bath. His temperature should be taken; when it falls
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to 39°C he should be taken out of the bath and placed in a cool room. Whenever there is a rise in temperature (which may fluctuate for some days), the subject should be cooled. However, if he is not taken out of the bath at the right moment, the temperature may fall considerably and continue to fall even after he has come out of the bath. After a certain time, natural perspiration may be restored. If the patient cannot be given a bath, he should be placed in a cool room and wrapped in wet sheets on which a fan directs cool, dry air to evaporate the water and so to cool the patient. Ice may be put round the head and neck, and the extremities may be massaged to promote the circulation of blood and hasten the fall of the body temperature. Cold enemas may also be given. It is sometimes advisable to give liquids, but this should be done only under medical control, because untimely hyperhydration may bring about cardiac deficiency with acute pulmonary oedema. Whether liquids are indicated will depend on the degree of dehydration (characterised by the number of red cells and electrolytic balance) and the heart condition.

If breathing is deficient, oxygen and assisted respiration may be necessary.

Sunburn

Aetiology. This is a burn caused by ultraviolet light after exposure to the sun for a certain time. Some parts of the body are particularly sensitive (trunk, neck, popliteal areas). Darker skin is more resistant.

Symptoms. These are erythema and itching. In severe cases the skin swells. Blisters and sacs of clear liquid may appear. The area of the skin affected by the dermatitis is very painful. During healing, pruritus is exacerbated.

The reaction disappears in a few days, but the skin may remain marked with pigmented and depigmented zones. A secondary infection may complicate healing and leave scars. However, recovery is usual.
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Prophylaxis. This consists in avoiding exposure to the sun and in wearing suitable clothing. The skin may be protected by creams containing substances that absorb ultraviolet light (such as tannic acid at 5 per cent, para-aminobenzoic acid and salol at 5 per cent). They should be applied every three or four hours. The disadvantage of using these creams is that perspiration may be impaired, and this could lead to thermoregulatory troubles.

Treatment. The affected area is cleaned with liquid paraffin, the phlyctenas are opened and the skin is pulled back. The skin may be bathed with calamine lotion containing 5 per cent crystal violet. The more serious lesions can be covered with calamine compresses. Subsequently an ointment of zinc oxide and castor oil can be applied. Antihistamines given by mouth may assuage the burning sensation and the itching, while soporifics may be given to treat insomnia and anxiety. Antibiotics are used in cases of secondary infection.

4.1.2. Cold climates

In cold climates, which are essentially characterised by low air temperatures, the heat losses of the organism are increased.

The cooling power of the environment varies not only with the thermal gradient but also with other factors such as air humidity and movement and the temperature of neighbouring surfaces and objects. Air humidity and movement increase this cooling power considerably without any change of temperature. It may increase by a factor of from 10 to 30, depending on the relative humidity and the wind velocity.

In cold environments, man succeeds in maintaining his normal temperature to a certain extent through the thermal insulation of his clothing and the functioning of the physiological mechanism of thermoregulation. This mechanism functions through a considerable increase in metabolic combustion (especially in the muscles), which results in a greater production of heat. At the same time there is a reduction in the heat loss to the environment because the
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skin temperature falls as the direct consequence of diminished circulation of blood in the skin.

When the heat losses exceed a certain amount, the mechanism producing metabolic heat comes into action and sets off involuntary muscular contractions (shivering).

Voluntary exercise during work steps up the metabolism but also increases the heat loss by convection as a result of the movements made.

According to some authors, the subcutaneous adipose tissue plays a highly important part in the thermal insulation of the organism that serves to maintain the normal temperature. In man the value of the layer of fat as insulation may be less than that of an extra pullover; however, the wearing of bulky clothing reduces work efficiency.

To a large extent, clothing insulates man from cold. It should be thick, consist of a number of layers, be light, permeable to vapour but impermeable to wind and permit ventilation of the skin.

The degree of adaptation to cold can sometimes be measured by the amount of clothing required to ensure thermal comfort in a given environment. Adaptation to cold seems to be easier than adaption to tropical heat. Adequate clothing will create a comfortable climate round the body. In addition, the intensification of metabolic processes during work will result in an increased production of heat and stimulate the organism’s defences against cold. However, working capacity in a cold environment is less than in a temperate environment because the increased metabolic activity hastens the onset of fatigue. Clothing that is adequate for rest is found excessive during work. It should be so made that it can easily be opened at the neck and wrists.

Perspiration during intense physical work in a low temperature may be dangerous when the physical activity stops, because it may continue for time, exposing the organism to severe cooling.

Excessive physical effort in a low temperature may expose the organism to lung diseases (bronchitis, bronchopneumonia), influenza and neuralgia, the onset of which, it is often claimed, is favoured by cold.
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Agricultural work carried out in a cold environment may give rise to pathological states characterised by local or general clinical troubles that vary with the different aetiological factors or circumstances: weather, intensity of work, duration of exposure, part of body most exposed, individual particularities (age, sex, state of acclimatisation, and so on).

General disorders due to cold

In cases of prolonged exposure to intense cold, body temperature may fall below the normal. This condition is called hypothermia.

Up to a certain point, a fall in body temperature may be compensated by reactions set off by the physiological thermoregulatory mechanism. There is thus a first stage in active defence against the danger of cooling the body. If the internal temperature of the body falls below 24°C, the vital functions diminish, the defence reactions are paralysed and death ensues.

The state of health, individual particularities, age and especially adaptation are some of the important factors that determine the general resistance of the organism and in practice have a great influence on the clinical signs. Among these signs there are two of special interest from the standpoint of pathology in agricultural environments: hypothermia in adults; and hypothermia in the aged.

Hypothermia in adults. This begins with general shivering and agitation; the pulse is rapid, blood pressure increases and breathing is intensified. A powerful constriction of the peripheral blood vessels restricts the dispersal of heat into the environment and serves to maintain the normal body temperature of the organism.

If the exposure to cold is prolonged, there will be a second period when the body temperature falls. There will then be muscular rigidity accompanied by repeated jerks in certain muscular groups and muscular pains in the limbs, and especially in the nape of the neck. The pulse slows and becomes irregular. Breathing also becomes irregular. The blood pressure collapses. Lastly, the subject loses consciousness and his condition evolves slowly towards death.
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_Hypothermia in the aged._ Cases of accidental hypothermia of occupational origin are rather frequent in elderly agricultural workers. The response of their organism to the prolonged effects of cold is less marked. The clinical symptoms develop insidiously and painlessly, without muscular contractions and without shivering. Numbness sets in gradually, the pulse and breathing slow down and the blood pressure and the temperature collapse. All the vital functions gradually diminish as a result of the exhaustion of the defence mechanism. Other symptoms are difficulty in speaking, somnolence, obnubilation and even coma.

At first there is a sensation of cold, but later this sensation becomes very vague. The face is cold and pale and sometimes puffy. If suitable treatment is given in time, the evolution of these conditions can be changed; however, complications such as broncho-pneumonia, cardiac infarction and peripheral circulation troubles may follow.

The only effective treatment of hypothermia is to warm the subject; this should not be done too quickly, however, as there may be deficient circulation because too much blood is going to the surface of the body.

After the patient has been warmed, possibly through immersion in a hot bath, it is essential that he should rest completely for several days so as to avoid acute heart failure due to effort.

_Local disorders_

As a rule, the extremities are not so well protected by clothing as the rest of the body. Furthermore, they cool down more quickly because their surface-to-volume ratio is higher and the thermo-regulatory reactions in the local circulation are weak. This explains the frequency of frostbitten fingers, feet (especially big toes and heels), cheeks, nose and ears.

According to the circumstances of exposure to cold, in particular its intensity and the duration of exposure, several forms of clinical sign are distinguished, the most important being chilblains and frostbite.
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Chilblains

The chilblain is an affection caused by irritation of the skin through exposure to relatively moderate cold. In agricultural occupations, chilblains are a common cause of suffering. They are often responsible for considerable increases in the frequency and duration of incapacity for work.

Aetiology. The special significance of damp cold and the immobility of the subject must be emphasised. A long period of immobility in a cold place, with little or no heating, when the hands are bare, is the optimal condition for the development of chilblains. Contact with cold water or objects such as metal tools has the same effect.

Cold, aggravated by humidity and immobility, is undoubtedly the decisive cause of these lesions. The repeated pressure of a cold object on a part of the skin, and all circumstances hindering the circulation, are factors conducive to the development of chilblains. Among general factors, stress has been laid on the subject’s state of health, his age (old people are more often affected than young), pre-existing circulatory troubles, disturbances in the autonomic nervous system, and above all conditions due to a poor diet.

The mechanism in the development of chilblains consists essentially of localised vasomotor phenomena. In the first stage, there is intense local vasoconstriction; this is confined to the peripheral vessels of the bare skin exposed to the cold, and serves to reduce the heat loss and maintain body temperature.

The principal phenomenon, however, is the reactional dilation that follows the vasoconstriction of the first stage. This is intense and lasting. It is this secondary vasodilatation that causes the redness, swelling and sensory disorders.

Symptoms. Chilblains are characterised by local red and violet patches on the extremities, accompanied by more or less serious sensory disorders.

The hands are the preferred site, the parts most often affected being the back and the sides. On the feet, chilblains are most often
found on the big toes and the heels, and more rarely on the instep and the bottom of the leg.

Usually two stages of chilblain are distinguished:

(1) The first or erythematous stage is constituted by lesions that are sometimes limited, round or oval but are more often diffuse with vague outlines. The eruption is accompanied by sensory phenomena: numbness, cramps, itching and pains in the limbs. When no longer exposed to cold, the chilblain disappears slowly in a few weeks. If exposure to cold continues, there will be fresh outbreaks which will continue until the weather is warmer. Some forms persist all the year in a more or less attenuated state.

(2) The second or ulcerative stage appears if the exposure is long or if the chilblain has not been treated. The lesions become violet; the epidermis cracks or splits, especially at creases where a seropurulent liquid oozes. There may also be phlyctenas containing a serous or serosanguine liquid which, after bursting, leaves an ulceration which may heal fairly quickly or alternatively deepen, become torpid and form a crust. The evolution ends in a persistent scar. Infectious complications may ensue and lead to suppuration of the blisters and ulcerations. In serious cases the ulcerations turn into mutilations of the fingers, nose and ears. The nails may also fall off.

Prophylaxis. Preventive measures may be based on the following recommendations, which agricultural workers should be advised to follow:

(1) Provide general protection against cold by activating the circulation and doing gymnastic exercises (it has been observed that persons who are accustomed to effort hardly ever have chilblains).

(2) Avoid immobility, which is one of the chief causes of the slowing down of the blood circulation.

(3) Ensure local protection against cold by avoiding all compression and constriction of the local circulation. Gloves should prefer-
ably be woollen and rather large and should not obstruct capillary circulation. To protect the feet, footwear should be large and should not compress the foot. It is advisable to wear woollen stockings, and particularly advisable to wear several pair of stockings with the finest next to the skin.

(4) Avoid warming the extremities too quickly after long exposure to cold.

(5) Consult a doctor as soon as the first signs of chilblains appear. Prompt treatment will prevent the development of complications and ulcerations.

(6) Do not over-indulge in alcoholic drinks which, after causing temporary excitation, weaken the defences against cold.

*Treatment.* The treatment is designed in the first place to improve the peripheral circulation. Antibiotics should be used for open or infected chilblains.

*Frostbite*

*Aetiology.* Frostbite is a lesion of the extremities caused by exposure to very low temperatures. Its seriousness depends on the intensity of the cold and the duration of exposure.

If the temperature is below freezing, the superficial tissues will begin to freeze, thus disturbing the cellular chemistry. These disorders are sometimes irreversible, in which case cellular necrosis will occur. In the underlying area there will be vasoconstriction, often aggravated by conditions of the organism that reduce its capacity for resistance (such as fatigue, hunger, haemorrhage).

When the temperature is not low enough to freeze the tissues, there is still vasoconstriction, which in turn slows down local circulation. Sometimes the circulation will stop completely in the affected area, which may continue to cool until the environmental temperature is reached.

Wind, humidity and high altitudes aggravate the effects of cold. Immobility of posture and tight-fitting footwear that compresses the feet are also conducive to frostbite.
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The lesions are more or less extensive and deep. They evolve towards tissue necroses and dry gangrene. If there is secondary infection, this evolves towards moist gangrene.

According to the degree of congelation, frostbite may be superficial or deep.

*Superficial frostbite.* Superficial frostbite is soft to the touch. It affects only the skin and the subcutaneous cellular tissue. The skin is pale and waxy. After being warmed, it is numb, mottled, blue or purple. It then swells with a sensation of paraesthesia, throbbing pains and burning, which may last for some days or weeks according to the severity of the exposure. In more serious cases phlyctenae will appear after 24 to 36 hours; they will dry up in about two weeks. The local swelling will subside sooner if the subject rests completely. The skin peels and remains very sensitive even to slight cold.

*Deep frostbite.* Deep frostbite feels hard and woody and affects the skin and the underlying tissues (subcutaneous tissue, muscles and even bones). Usually giant blisters form; they appear later than in superficial frostbite. The limb remains inflamed for a number of weeks and is coloured blue, violet or grey (grey is a bad sign). Violent throbbing pains continue for several weeks. The big blisters dry up, leaving a thin red skin which is very sensitive to cold and looks more or less normal only after some weeks. Sometimes itching and considerable perspiration persist for several months, and increased sensitivity to cold may remain. With deep frostbite, recovery is accompanied by loss of dead flesh, especially when the victim has not been warmed soon enough. The area of dead flesh forms very slowly, usually beginning after two weeks and possibly continuing for several months. In such cases, the skin has a grey colour from the outset and big blisters mark off the healthy area. If there is a secondary infection, the necrotic lesion turns into moist gangrene and the limb is swollen and painful, for the infection penetrates beyond the tissues affected by the frostbite.
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Clinical picture. Generally speaking, there are three degrees of frostbite. The first degree is characterised by redness, corresponding to mild frostbite. The second degree is characterised by the appearance of big phlyctenas which, on opening, evolve towards ulcerations with only a very slow scar formation. The third degree, corresponding to death of the tissue, is dry or moist gangrene.

Prophylaxis. Sufficient clothing should be worn to conserve the heat of the body.

Footwear should be large enough not to injure the feet. Stockings in contact with the skin should be fine, soft and clean, so as to avoid secondary infection in the event of lesions. Over these stockings one or two pairs of coarser stockings should be worn.

Gloves should permit the performance of work and at the same time afford suitable thermal protection.

Periods of exposure to cold should be reduced as its intensity increases. Periods of exposure can be alternated with breaks for warming up and consuming hot drinks. Alcohol is prohibited because, in spite of the passing sensation of warmth that it gives, it actually weakens the organism’s defences against cold.

Persons who have circulatory disorders in the extremities, very old persons and diabetics should not be exposed to cold. Diet should be adequate.

Persons occupationally exposed to cold should be given antitetanus prophylactic treatment, because serious lesions due to cold may be considered to be exposed to the risk of tetanus.

Treatment. The patient should be immediately taken to a hospital. If this is not possible, he should be made to rest completely in a well heated room and be given hot drinks.

The frozen limb should be warmed to 42–44°C to avoid irreversible lesions of the tissues. As the extremity warms, steadily increasing pains may be experienced; these are usually more severe in elderly persons suffering from circulatory disorders.

Alcohol should be allowed only if the patient remains in the heated room to which he was taken.
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Massage is contra-indicated because it may induce lesions and lead to secondary infection. For the same reason, such traditional practices as rubbing with snow and dipping in cold water must be avoided.

Antibiotics may be given to prevent secondary infections, or to treat them if they occur.

Since prolonged complete immobility may considerably reduce the functional capacity of the affected limb, it is advisable to move the joints during convalescence.

Recovery is facilitated by a balanced diet rich in proteins and vitamins.

No ointment should be applied locally, for this might predispose to infection and moist gangrene.

Surgery is the last resort, especially when there are secondary infections that cannot be controlled by medical treatment.

4.2. Commonest lung diseases due to vegetable dusts

4.2.1. Byssinosis

Byssinosis is an occupational lung disease found in workers exposed to cotton, flax or hemp dust.

In order to place the disease in its occupational setting, it will be useful to outline briefly the various processes through which raw cotton passes from picking to spinning.

Cotton is picked either by hand or by machine. Picking is carried out in the open air and is practically free from risks. The seeds and most of the dirt and plant debris are separated out by blowing the cotton into the air. The cotton is then put into a gin which removes the seeds. This is a dust-raising operation, but it is a seasonal one, taking place only in autumn and winter. Next, the cotton is conveyed to factories, where it is pressed and conditioned in bales wrapped in gunny (coarse sacking) for onward transport to the mills. The opening and loosening of bales are dusty operations. In the carding process that follows, the fibres are combed and dirt
and short fibres are removed. The fibres then pass through a series of machines that prepare them for spinning.

*Aetiology.* Byssinosis appears after more or less protracted exposure to vegetable dust. From one to ten years may elapse before the clinical signs appear: the more intermittent the work, the longer the interval, especially among seasonal workers. Among women, precisely because of the intermittent nature of their work, the frequency of the disease is lower than among men.

The frequency of the disease is proportional to the amount of dust at the workplace. Thus, in operations in which raw cotton is handled, the frequency is higher than in operations with processed cotton. In flax-handling operations, byssinosis appears uniformly in all the stages, for all are equally dusty. With flax, however, the disease is less severe and no significant weakening of the ventilatory function has been found, but clinical cases have been found only after a few months of exposure.

The replacement of hand picking by machine picking has increased the quantity of dirt in the cotton, and this has aggravated the symptoms in the workers affected. Measures designed to reduce dustiness at workplaces have produced some results, especially among young workers, whose frequency rate for the disease has been reduced.

Dust in the cotton industry contains short fibres (between 0.2 and 1.5 mm), plant debris, minerals, bacteria and fungi. Cotton is a cellulose that is insoluble in water and biologically inert. Cellulose is not pathogenic in wood or rayon dust. The dirt containing the plant debris is a mixture of organic substances, some of which are soluble in water.

*Pathogeny.* The theory that the dust of textile plants irritates the respiratory tract fails to explain why the effects are not always observed.

The amount of silica in the dust is too small to cause silicosis, and the subjective manifestations observed are absent in silicosis. In industries with a silicosis risk, the concentration of silica in the atmosphere is 100 times higher. Moreover, the results of radio-
logical examinations are different. However, the role of other mineral fractions in the dust has still to be assessed.

However this may be, and whatever the fraction of vegetable dust in question, the process that it sets off seems to be the liberation by an unknown substance of an active substance (histamine) in the respiratory tract, resulting in oedema and bronchoconstriction without previous sensitisation. The fact that previous sensitisation is not necessary shows that this is a case where allergic reactions do not follow the ordinary course. Moreover, skin tests have not revealed the existence of a proteic antigen of a vegetable, bacterial or fungoid nature.

It appears that, in order to liberate a fresh quantity of histamine, the organism needs a certain time in which to accumulate it. This may explain why the symptoms appear regularly after the weekend and why it happens that the longer the rest is, the more pronounced they are.

The appearance of clinical signs after several years of exposure can be explained by the fact that continuous contact with the dust enables a larger quantity of histamine to be liberated at a given moment. We are thus in the presence of a phenomenon that has been in existence for a long time and, when it reaches a certain threshold, gives rise to subjective phenomena.

Some authors consider that bacterial toxins liberate histamine and that other substances are responsible for the fever, but other authors maintain that histamine is liberated by a substance produced by bacteria and fungi.

The hypothesis that a substance related to quinine is liberated in the organism by cotton dust might explain the slow appearance of the symptoms and their persistence until the evening, some hours after the cessation of work.

**Symptoms.** In 10 to 50 per cent of cases, subjects newly exposed to cotton dust catch a cold the first day, or rather in the evening of the first day. The temperature fluctuates between 38°C and 39.5°C. The subject suffers from headaches, nausea and even
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vomiting. The next day his general condition is good. Sometimes the symptoms are repeated several evenings running. The fever is usually moderate, but the subject feels an intense fatigue. After a week, or at the most a month, these symptoms disappear, even if the subject continues to work in the same undertaking. If in the course of the first year he stops working for two weeks, when he resumes working he will exhibit the same immunisation phenomena but in a much milder form. After some years at his occupation, it is only at the end of a break of two months that on resuming work he will exhibit the vague symptoms that recall the fever of the new worker.

The symptoms of byssinosis proper do not become clinical until many years of exposure to vegetable dust have elapsed. The effect of exposure to the dusts is progressive. The establishment of the symptoms and their evolution depend both on the degree of pollution of the atmosphere and on individual susceptibility. Three stages of the disease have been described, each lasting one, two or more decades.

As has been seen, from the first day after the weekend or a break in the work, the worker suffers from tightness in the chest with dyspnoea and coughing. The symptoms establish themselves progressively and reach a peak in the afternoon of the first day. Coughing is troublesome for smokers, who usually refrain from smoking on Mondays. The next day the symptoms have disappeared and the worker feels well. In some workers who work longer hours, the symptoms may persist for two or three days but their intensity diminishes towards the middle of the week. The longer the absence from work, the more pronounced are the phenomena. There is thus a reversible bronchial constriction with oedema of the mucous membranes and bronchial secretion. These symptoms disappear altogether if the subject abandons this kind of occupation.

In the second stage of byssinosis, the symptoms may persist for over a week, and will not disappear unless the work is interrupted during the weekend or holidays. The workers have become bronchitic, and absenteeism increases. If they change their occupation,
their condition improves and, as a rule, the pulmonary symptoms disappear, leaving a good working capacity.

There is a diminution of the ventilatory capacity of the lungs during work, and an increase in the resistance of the respiratory tract that is particularly pronounced on Mondays. The maximum ventilatory capacity, measured on any day except Monday during the first and second stages of byssinosis, shows a significant decline from the levels found in normal subjects. In second-stage byssinosis, the levels found are below those found in the first stage. The maximum levels of ventilatory capacity measured on Mondays are below those measured on Thursdays. Investigations among normal subjects not working in vegetable-fibre industries show that this test gives lower values even in persons exposed to the dusts for the first time. The changes in the respiratory function are found even in the absence of any clinical sign in workers exposed to risk.

In the third stage, which is exceptional because the workers have usually changed their occupation before it appears, the respiratory troubles are permanent, even if the work has been abandoned. The worker suffers from chronic bronchitis with emphysema, a cough with mucous or mucopurulent expectoration, and dyspnoea when making an effort. The evolution is towards the chronic pulmonary heart and cardiac deficiency. If the worker leaves the undertaking, the evolution of the disease may be arrested, and sometimes the symptoms will regress. At this stage, radiological examination shows a certain degree of fibrosis without any specific aspect and analogous to ordinary chronic bronchitis.

Other pathological manifestations, such as weaver's fever, have also been described. The clinical aspects of this disease are characterised by tightness of the chest and difficult breathing of the asthmatic type, with coughing, malaises, fever and headaches. The disease is thought to be due to a fungus in contaminated threads. A microbial agent (*Aerobacter cloacea*) found in dirty cotton is also thought to be responsible for the disease. In experiments it has been shown that the causal agents are endotoxins of this micro-organism inhaled with cotton dust; some days after starting work, the subject suffers from generalised pains accom-
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panied by fatigue, anorexia, nausea, vomiting, and sometimes shivering and fever.

Differential diagnosis.
(1) Chronic bronchitis: the symptoms, although similar, appear at times unrelated to hours of work.
(2) Bronchial asthma: the progressive diminution of the maximum ventilatory capacity during the first working day after the weekend is not the same as with asthma. Moreover, an asthma crisis develops immediately after contact with the specific allergen and not some hours later. In byssinosis, breathing disorders affect both inhalation and exhalation, whereas in asthma exhalation only is affected.

Prophylaxis. Since the harmful agent is found in the debris of plants, and especially of leaves, the most effective preventive measure would be to separate the fibres from the rest of the plant at the moment of picking. However, this is not practicable at present. Care must therefore be taken to ensure that the amount of dust per mm³ of air does not exceed 1 mg. The finest dust, which is the most dangerous because it most easily penetrates into the respiratory tract, is the most difficult to eliminate. The worker should be removed from the dust as completely as possible.

Mechanisation should replace manual work in the most exposed operations.

Periodical medical inspections make it possible to discover susceptible persons and direct them to other occupations so as to prevent them from becoming incapacitated.

Treatment. The inhalation of bronchodilator substances during work seems to be the most suitable treatment; it makes breathing easier, and the subject can continue working.

4.2.2. Bagassosis

Bagassosis is a vegetable pneumoconiosis caused by inhaling the dust of bagasse that has been stored outside for long periods.
Bagasse is the fibre of sugar-cane stalks which remains after the juice has been extracted. It is collected, baled and kept in heaps around the mill. It contains 4 per cent of ash, 2 per cent of protein and a large percentage of cellulose and complex hydrocarbons. There is also a small quantity of silica.

Fungi grow on bagasse that has been baled in a warm, moist state. When dry, bagasse is rapidly broken down into dust. Bagasse dust consists of particles of the pith and the cortex fibres of the sugar-cane together with fungi spores.

Bagasse is used as a fuel or as a raw material in the manufacture of plastics, pasteboard, fertilisers and refractory bricks. The bagasse bales are sent to different factories. The workers transporting them, and more so those opening the bales, are exposed to heavy dust concentrations.

Besides tractor drivers and workers handling bagasse bales while they are being opened, other workers are exposed to the risk of bagassosis—for example, watchmen and electricians—but their exposure is incidental and usually slight.

The duration of exposure required to cause the disease normally varies from three weeks to a few months, but in some cases it has appeared after only four days of exposure.

The risk of being infected with bagassosis is fairly high; 50 per cent of the workers exposed to the dust are liable to contract the disease.

Aetiology. Although bagassosis is known to be caused by the inhalation of airborne dust from bagasse fibres, the specific aetiological element has not been discovered. At present the data are insufficient to determine whether the symptoms at the start of the disease are allergic and due to an antigen in the bagasse dust, or inflammatory and due to a bacterial mycotic infection, or indicate a simple irritation reaction. It is also possible that the symptoms represent combined action by two or more agents: for instance, the irritant action could stimulate bronchial reactions to a fungoid or a bacterial antigen.
Symptoms. The disease appears after exposure to airborne bagasse dust for a period that may vary from a few weeks to a few months. It may take a mild, moderate or severe form. Every fresh exposure leads to an aggravation of the symptoms.

A worker who is affected feels chest pains behind the sternum, is fatigued, loses his appetite and sometimes loses weight. In some cases a very considerable loss of weight is the first symptom.

The patient experiences difficulty in breathing, sometimes accompanied by a state of anxiety and a morning cough that is usually dry but may be accompanied by sputum spotted with blood. The temperature rises slightly in the evening, and the rise is accompanied by shivering and sweating which persist for some days at the beginning of the disease. In the severest cases, the temperature may reach 40°C. The pulse accelerates as the temperature rises. Sometimes there are fronto-occipital headaches, nausea, vomiting, giddiness and, in severe cases, a cyanotic condition. Breathing difficulties persist and may last for months. They are the most characteristic sign of the disease and lead to incapacity for work.

The majority of the patients cure themselves by ceasing to expose themselves to the dust. Most of the symptoms disappear in a few weeks and complete recovery ensues after a few months. Exceptionally, recovery may take a year or even longer.

In the event of recurrence, breathing is progressively affected.

Radiology. The radiological picture shows a diffuse punctiform infiltration of the two lungs, resembling typical miliary (punctiform) tuberculosis. The bronchopneumonic infiltrations are extensive, and bands of fibrosis radiating in the hilar zones have been observed, sometimes together with an accentuated bronchovascular pattern (see figure 18).

In cases of prolonged exposure, a condition of pulmonary fibrosis has also been described. Such conditions can be found a year after the acute stage.

Diagnosis. Diagnosis is confirmed by clinical symptoms and by radiological pictures of a person occupationally exposed to bagasse dust.
Figure 18. Bagassosis, showing miliary infiltration of the two lungs. Reproduced by courtesy of Howard A. Buechner, M.D., Chief, Medical Service, Veterans Administration Hospital, and Associate Professor of Clinical Medicine, Tulane University School of Medicine, New Orleans (United States).
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*Prophylaxis.* Workers exposed to bagasse dust should wear respirators, of which the filters should be changed at suitable intervals. Bagasse bales should be dampened to prevent the dust from dispersing in the air. Pollution can be reduced by a good exhaust ventilation system.

It is very important that the diagnosis be made at an early stage, so that the affected worker can be put out of reach of the causal agent. If he remains in the dusty environment, recurrences of the disease will impair the respiratory function.

A good degree of prevention may be obtained through mechanisation at places where exposure is most serious.

*Treatment.* There is no specific treatment. Symptomatic treatment aims at calming the cough and reducing the respiratory difficulties. The disease regresses if the patient is removed from the harmful environment.

4.2.3. *Farmer’s lung*

“Farmer’s lung” is a pneumoconiosis caused by vegetable dust. The disease is also known by other names such as “thresher’s disease”, “harvester’s lung” or *bronchomycosis fenisectorum*, depending on the prevailing aetiological conceptions and the categories of worker in which it has been described.

*Aetiology.* Most cases are found in males aged between 30 and 40 and as a rule without any allergic antecedents. The disease is caused by inhaling the dust of mouldy hay and straw. Hay and straw become mouldy if they are damp when they are stacked, a common occurrence in wet summers. When mouldy, they contain a large quantity of dust composed of fungal spores and vegetable particles.

The clinical picture can be produced in persons who have been sensitised by means of a bronchial test with an aqueous extract of mouldy hay. The pathogenic agent has not been identified, owing to the complexity of the harmful dust.
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Some authors consider farmer's lung to be an allergy caused by sensitisation of complex substances present in the dust of mouldy vegetable products.

Extracts of mouldy hay contain a considerable number of antigens produced by the fungi and additional antigens that are specific to the disease. These antigens have been found in hay that is becoming mouldy.

The appearance of the clinical manifestations is said to depend on the dimensions of the dust and the amount of antigen.

The disease should be distinguished from bronchial asthma, from which it differs fundamentally.

The two kinds of work in which the highest concentrations of hay and straw dust are found are work in cattle sheds and work in teams of threshers in barns. The cowherd who prepares the hay for the animals in the shed before they come back for the night often raises clouds of dust. He remains in this atmosphere while milking, exposed to a high concentration of dust. This exposure lasts for about an hour a day throughout the winter and at the beginning of spring. The thresher is exposed for eight to ten hours a day, but for a shorter period. The amount of dust is considerable, especially if the hay comes from mouldy ricks. The disease is commoner in wet years.

**Symptoms.** Three phases of the clinical evolution of the disease can be distinguished: the initial phase; the phase of bouts with remission (reversible); and the chronic phase (irreversible).

1. The initial phase: some hours after exposure to the harmful dust, the worker suffers from shivering, fever, headache, profuse sweating and tightness in the chest; general malaise and coughing are frequent, and dyspnoea may also occur. Sometimes it is only after the acute phase, while the farmer is returning from work, that the least effort causes difficulty in breathing. Anorexia and loss of weight (from 4 to 9 kg) are common. The anorexia and headaches persist for some days after the disappearance of the chest symptoms. The temperature usually rises to 37°C or 38°C, but in many patients a rise to 40°C or
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even 41°C has been observed. The bout is characterised by fever, which lasts two or three days only.

The cough which accompanies the acute phase lasts for several days, even if the patient is no longer in contact with mouldy material. The expectoration is minimal; it is yellow, greenish or colourless, sometimes spotted with blood. Haemoptysis and epistaxis are rare.

(2) The second phase: if the subject continues to work in the harmful atmosphere, he will be liable to undergo numerous crises more or less similar to the first bout. After repeated bouts the chest becomes emphysematous and the subject becomes cyanotic.

(3) The third phase: this is the chronic phase in which the emphysema and pulmonary fibrosis establish themselves as the result of repeated exposures over several years.

Radiological aspect. The radiological picture in the first phase of the disease may be normal. However, there is usually a diffuse interstitial pneumonia and a certain degree of compensatory emphysema. The pulmonary consolidations disappear one or two weeks after the end of a bout, but the pneumonia lasts for some months.

The disappearance of the radiological symptoms is not accompanied by complete recovery. Dyspnoea accompanying effort also persists after the radiological symptoms have disappeared.

Each repeated exposure causes an interstitial pneumonia which in the long run merges into the chronic and fibrous picture of pneumoconiosis, which is irreversible.

Diagnosis. Questioning will lead the diagnosis towards an occupational disease associated with the performance of certain work on the farm. Radiology will stress the pneumoconiotic character of the complaint.

Differential diagnosis.

(1) Silo filler’s disease: the acute stage of farmer’s lung may resemble, both radiologically and clinically, silo filler’s disease.
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However, in the latter disease, close questioning will disclose that the patient was exposed at the workplace to a gas with a particular odour. This gas is nitrous oxide, given off during the fermentation of the nitrates in the fodder stored in the silo.

(2) Influenza: many cases of farmer's lung have been diagnosed as influenza. The clinical picture and the rapid disappearance of the acute symptoms favour this diagnosis.

(3) Pneumonia and bronchitis: the initial stage of the disease also often resembles pneumonia or bronchitis, but the persistence of dyspnoea with effort after the disappearance of the other clinical symptoms should suggest another diagnosis. Close questioning and an exact knowledge of the disease should lead to the correct diagnosis.

Prognosis. The prognosis depends on the phase in which the disease was diagnosed. During the first phase, when the lesions are reversible, the prognosis is good; hence the importance of correct prophylaxis.

If the disease is diagnosed in the irreversible phase of pulmonary fibrosis, the prognosis will be uncertain, and a loss of working capacity may be feared. The farmer may not be able to do any more manual work, even away from the environment that caused the disease. The prognosis will be pulmonary deficiency leading ultimately to cardiac deficiency.

Prophylaxis. The purpose of preventive measures is to avoid sensitisation and the subsequent occurrence of bouts of the disease. Farmers should be warned of the danger of wet hay and straw and should take care not to wet vegetables. If, however, the vegetables become mouldy, agricultural workers should wear a mask when handling them.

The mechanisation of harvesting is a means of prophylaxis for harvesters.

If sensitisation has already occurred and the disease appears and recurs, the patient should change his job on the farm. This is the most effective form of prophylaxis.
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Treatment. In the majority of cases withdrawal from the harmful environment is followed by regression of the disease. The anti-allergic and anti-inflammatory action of corticotherapy greatly helps to reduce the acute symptoms.

4.3. Dermatosis due to contact with plants

Some plants cause mechanical lesions by their thorns or bristles, others cause both chemical and mechanical lesions, and others again cause irritative or eczematous dermatitis through an allergic mechanism. A person may be allergic to a well defined species of plant, or his sensitivity may extend to several species of the same kind or of different kinds (because the substance responsible may exist in plants that are more or less related).

The amount of allergenic substances in a plant at any particular moment depends on its stage of development, the conditions in which it is grown and the season, for the plant may be more harmful at certain times.

The sensitising or irritant substances are sometimes uniformly distributed over the whole plant and sometimes concentrated in certain parts: flowers, leaves, stalk, roots and pollen.

As a rule, plant dermatitis is classified in three groups: irritant dermatitis of plants; eczematous contact dermatitis; and phyto-photo dermatitis.

4.3.1. Irritant dermatitis of plants

This type of dermatitis is the consequence of contact with plants containing irritant substances, such as Achillea millefolium, Brassica nigra and Ranunculaceae. All persons in contact with these plants react, and the penetration of a substance into the skin depends on the thickness of the latter’s horny stratum. This dermatitis is particularly common among children.

It takes the form of small red pimples on the parts of the skin that have been in contact with the plant. In children especially, reactions in the form of blisters and pimples can be observed, even
on the lips (if a child has been chewing buttercup stalks). In adults there is erythema, with irritation and burning if contact is repeated, and eczematous dermatitis may result.

It has been suggested that the substance responsible for the irritant effect of the buttercup is produced in the tissues by the decomposition of a glucocide.

4.3.2. Eczematous contact dermatitis

This is an allergic disease. It appears after an earlier sensitisation to a particular species or a related species. Thus only persons who have previously been sensitised suffer from the disease. Usually the hands, neck and face are attacked, and gardeners and florists are usually the victims; children are rarely affected. With some species, sensitisation is achieved only after repeated contact.

(1) Primula obconica is, with a few very rare exceptions, the only one of the 600 species of the primula family that causes an eczematous contact dermatitis. The antigen is distributed all over the plant, but is concentrated more in the small glandular hairs of the leaves and stalks. The dermatitis affects the face, neck, arms and hands, and sometimes the eyelids. In the latter case, there is itching and a burning sensation; the eyelids are red and oedematous, and are often covered with blisters and linear pigmentations.

Sometimes diagnosis is difficult—as when there is only one transitory red patch with itching, or rapidly diminishing redness with inflammation of the eyelids. Lesions of the mucous membranes, which are uncommon, are accompanied by coryza, conjunctivitis, fever and diffuse red patches. In cases of repeated contact, the eruption may be very extensive and may be accompanied by fever and general symptoms.

(2) Toxicodendron radicans or Rhus toxicodendron (poison ivy) is the cause of the majority of cases of eczematous contact dermatitis. This dermatitis is characterised by typical blisters or vesicles on the surface that has been in contact with the plant. However, often only erythema or small pimples are evident, and the clin-
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A clinical picture will depend on the degree of sensitivity of the victim at the time of contact and on the quantity of allergen. The clinical manifestations appear from eight hours to ten days after contact; the longer the exposure to the allergen, the sooner the dermatitis will appear and the more severe it will be.

3. Lily rash consists of irritant pimply lesions. It mainly affects workers who cut, tie or pack wild or cultivated narcissi.

4. Bulb finger is an affection of the fingers of workers who are sensitive to the bulbs of tulips, hyacinths, garlic and onions. From 12 to 24 hours after contact, the symptoms will be pins and needles, heightened sensitivity and redness at the tips of the fingers. Repeated exposure will bring about a thickening and hardening of the superficial layer of the skin, together with cracks at the tips of the fingers.

5. Chrysanthemum dermatitis: the clinical signs appear progressively after repeated contacts. Once the subject has become sensitised, contact with the plant causes erythema and irritation. On subsequent contact the skin becomes infiltrated.

6. Pollen dermatitis is manifest in erythema and irritation due to an antigen that is found in certain plants of the ambrosia or related families whose pollen is airborne. Because the pollen can come into contact with any part of the bare skin and can even get under clothing, the location of the dermatitis makes diagnosis possible. This is because it can easily be distinguished from dermatitis due to photosensitivity or medicaments, which is bounded by the edges of clothing.

4.3.3. Phytophoto dermatitis (dermatitis bullosa striata pratensis)

Phytophoto dermatitis is caused by exposure to ultraviolet light of skin that has been in contact with certain plants. Anyone may contract it. The plants responsible are *Anthriscus sylvestris* and *Pastinaca sativa*, among others.

Clinically, this dermatitis takes the form of striped or irregular blisters appearing on exposure to sunlight, from 12 to 24 hours
after contact with the plant. After recovery, a pigmentation remains and persists for some months. The disease is commonest among children, agricultural workers and troops on manoeuvres.

4.3.4. Diagnosis

Questioning is essential for diagnostic purposes. The clinical signs may be helpful if characteristic linear lesions are found. However, tests are necessary in principle to confirm the diagnosis. The tests consist in applying to the skin of the back for 48 hours 1 cm² of a leaf of the plant suspected of being responsible for the clinical signs. The piece of leaf is covered with cellophane secured by sticking plaster. The place is examined after 48 and 72 hours and on the fifth and seventh days. The reaction is positive if erythema with oedema and small cracks are observed. Some reactions are less important, or are delayed, or both. It is essential to perform these tests, at least on normal persons, because it is always easy to distinguish the eczematous reaction in a positive test from a simple irritation. Tests performed over a period of months with dried petals or leaves produce weaker reactions. Sometimes when a test is performed, false positive reactions may occur. To avoid them, a sufficient interval must be left between the end of the clinical signs and the test.

If no particular species is suspected, tests must be performed with all the groups and varieties of plants with which the subject has been in contact.

When the plant responsible has been found, sensitivity to related species must be determined for the purpose of avoiding subsequent harmful contacts. Moreover, the patient should be advised to stop growing the plant in question.

4.3.5. Prognosis

If plant dermatitis has been correctly diagnosed and if the patient avoids all contact with the plant responsible, the disease is quickly cured and the prognosis is good. Desensitisation is difficult, and the only preventive measure consists in avoiding contact.
4.3.6. Prevention

Horticulturalists suffering from plant dermatitis must be advised to stop growing species that are harmful to them. In the case of Primula obconica, for example, subjects can be advised to grow Primula malacoides or Primula sinensis, which have no allergic properties. If the diagnosis has been accurate, hyposensitisation can be tried, since complete desensitisation is impracticable. However, if exposure continues after hyposensitisation, the subject may become resensitised.

4.3.7. Treatment

Local treatment consists in the application of saline compresses, 1:40 Burow’s solution (aluminium acetate solution) or hydrocortisone cream.

Antihistamines are often used.

If the symptoms are very pronounced or are accompanied by general disorders, rest in bed is advisable. Corticosteroids may be given, beginning with large doses which are gradually reduced. Antibiotics may be given as a precautionary treatment, but they are sometimes also necessary because of secondary infection of contact dermatitis.

4.4. Pathology due to snake bites and insect, spider and scorpion stings

4.4.1. Snake bites

Between 30,000 and 40,000 persons die from snake bites every year. The largest number of cases are recorded in south or southeast Asia; in the United States, between 2,000 and 3,000 persons are bitten annually.

Most snake bites occur in rural areas, and the frequency seems to depend to a large extent on occupational factors. It has been estimated that two-thirds of the victims are bitten at their work-
place in the forest or the field. The frequency is higher among men, also for occupational reasons, but a considerable frequency among children shows that they do not realise the dangers of snakes and play with them.

The frequency of bites increases in the rainy season when flood water drives the snakes out of their hiding-places, and also in the spring and summer when there are more people in the country, either working or spending their holidays. Bites are more serious after the winter or a period of fasting, because of the accumulation of venom in the snake’s glands.

Snakes are carnivorous reptiles. They eat irregularly and live in isolated places. They travel slowly (5 to 7 km/h) and cannot overtake a man who is running. The jaws can come out of joint and allow a large prey to be swallowed. Snakes are probably deaf because of the degeneration of their hearing apparatus. The tongue has tactile and olfactory receptors, and pits in the cheeks are organs for detecting the direction of heat, enabling warm-blooded animals in the vicinity to be located.

The venom is produced by special salivary glands comparable with the salivary glands of mammals, and two fangs in the upper jaw are equipped with a canal by which the venom can be injected into the victim’s body. The fangs make one or two round and deep wounds, the distance between which varies according to the size of the snake. Sometimes there are also small wounds made by the teeth in the lower jaw. Only one bite can be seen if the snake has attacked from the side.

Some physical features distinguish poisonous snakes from harmless ones. Poisonous snakes, except for the coral snake, have a triangular head, two pits between the eyes and the nostrils, elliptical pupils, two fangs and only one tail-plate.

Non-poisonous snakes have an ovoid head, no pits, round pupils and a double tail-plate. There are no fangs, but their absence is difficult to verify because they may be joined to the palate. The bite of a non-poisonous snake produces a series of superficial U-shaped marks. Coral snakes resemble non-poisonous snakes, but they have two fangs.
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Snake venom is composed of proteins, some of enzymatic origin. Its composition varies with the species and sometimes within one and the same species; it is also affected by the physical condition of the snake and its manner of life.

There are two main types of poison: haemotoxic (injuring the red cells of the blood), and neurotoxic (acting on the nervous system).

The viper family produces a venom that is essentially haemotoxic but possesses certain neurotoxic properties. On the other hand, the venom of the coral snake is essentially neurotoxic and only slightly haemotoxic.

The quantity of venom injected by a snake may range from a few drops to 2 ml. Usually less than a lethal dose is injected. The role of the venom is to paralyse the victim and to aid in digesting it.

The venom is rarely injected intravenously; more frequently it is injected into the subcutaneous tissues from which it spreads deeper into the body.

In man the venom may be neutralised by the antibodies of the organism and by antidotes used in therapy.

Snake bites do not always have harmful effects. The snake may be ill or may have used up its venom by biting and eating a prey a little earlier, or again the venom may have been absorbed by the victim’s clothes.

Local symptoms

The place bitten is the seat of an intense burning sensation with steadily increasing pain that spreads over the limb or body, sometimes following the neurovascular axis. The pain is very severe and persists until necrosis. Fever is exceptional; the temperature may rise to 37.7°C and more, and last for approximately two to six days.

At first, the wound has the appearance of a whitish ring around the bite; the ring then becomes reddish, bluish-green or purple as
the result of the invasion of red cells. Oedema spreads rapidly. The place is usually hard for the first 24 or 48 hours and then softens. The swollen part is coloured by the red cells and the plasma extravasated in the subcutaneous tissues. The oedema may spread for 24 to 72 hours, depending on the amount of venom injected, but after 12 hours it attains 75 per cent of its final volume. The extent of the oedema is a valuable indication of the site of the poisoning and the amount of venom received. If the final swelling is less than 3 cm, there will be no serious general phenomena; however, 30 per cent of the victims have a swelling of 4 to 8 cm and nearly all the victims with a swelling of more than 8 cm have been seriously poisoned.

If the upper limit does not reach the knee or the elbow, the poisoning may not be serious.

After one or two hours, pain is felt in the regional lymphatic ganglia, which become soft and swell slightly. The oedema sometimes spreads to the neck and the tongue; in such cases adequate treatment is needed, failing which there will be oedema of the larynx with asphyxiation.

Local destruction may be considerable, in the form of necrosis or gangrene. Necrosis occurs in 1 per cent of the cases; it is preceded by phlyctenas which surround the bite from the third or fourth day. It is usually superficial and rarely reaches the tendons, muscles or bones.

Necrosis is due to the high local concentration of venom. It is therefore more common with bites on the fingers or toes, or with bites from large snakes that inject a large quantity of venom, or again when the fangs have penetrated deeply into the muscles.

Necrosis is followed by bacterial infection that may be transmitted to the neighbouring joint. Infection also occurs after makeshift local surgery. In the majority of cases, the necrosis is black and dry. Extensive necroses occur chiefly in inhabitants of sub-desert zones far from any medical centre, who, after receiving some makeshift treatment, take several days to reach the centre.

The necrosis seems to be brought about by intravascular coagulation in the local blood vessels, leading to ischaemic necrosis.
(local anaemia due to diminution of the circulation of the blood) in the area that they irrigate. Dissection of the necrose area shows that a whole muscle may be affected while the adjacent tissues are normal, even though they may be close to the bite—a circumstance that suggests ischaemic gangrene due to vascular damage. However, the venom acts directly on the muscle by reason of its proteolytic properties (dissolution of albuminoid or proteic substances).

**Symptoms of general damage**

The first symptoms of general damage may appear after a few minutes or a few hours. Usually some hours elapse before the toxic picture is complete. Prostration or a syncopal condition may be the result of emotion. The victim’s general symptoms are headaches, nausea, vomiting, abdominal pains, watery diarrhoea and violent muscular spasms or convulsions.

Haematuria, melena, prolonged bleeding time of puncture wounds or the bite, haemoptysis, haematemesis, and haemorrhage of the vital organs are the consequences of failure of coagulation.

Failure of coagulation may occur only 20 minutes after the bite, or at the latest five-and-a-half hours afterwards. To detect it, the victim may be asked to cough hard and to spit into a receptacle, so that important symptoms will not be missed. The expectorated blood comes from the lungs and not from the gums, which rarely bleed and, if so, only much later.

The haemorrhage of the chief organs may be fatal. This is particularly true of cerebral haemorrhages, but these are rare because the respiratory system is affected before any other.

One can usually see a few discoid ecchymoses that are slightly raised and 3 to 15 mm in diameter (see figure 19). These are dis- colorations (sometimes black, sometimes brown or yellow) due to infiltration of variable quantities of blood into cellular tissues. They are rarely profuse and may be the consequence of an embolism in the arterioles of the connective circuit of the superficial subcutaneous vascular system.

Laboratory examination reveals blood that is non-coagulable or has an increased coagulation time; there is a reduction of plasmatic
Figure 19. Discoid ecchymosis, an effect of the coagulation defect. Profusion is found only in very severe systemic poisoning.

fibrinogen, the prothrombin time is longer and the sign of capillary fragility (Rumpel-Leede test) is generally positive.

The haemorrhagic syndrome persists for three or four days. In victims who have not been given any antidote, the hypocoagulability of the blood has been found to last for a week, and coagulation is defective for several days afterwards, although the clinical signs of the haemorrhagic syndrome have disappeared.

There have been reports of heart disorders, possibly due to dehydration and shock.

Snake venom is said to act on the plasma, producing substances that cause spasms in smooth muscle, vascular dilation with hypotension and lessening of the blood flow. These conditions may lead to thrombosis in an artery or coronary arteriole.

A syndrome of acute renal insufficiency has also been reported with haematuria and albuminuria.

The neurotoxic effects take the form of nausea, vomiting, salivation, difficulty in speaking, somnolence, unsteady gait and paralysis of the eye, face and spinal muscles. The tongue may be paralysed, and it may be impossible to swallow or to evacuate bronchial secretions because of paralysis of the intercostal muscles and the diaphragm. There is sometimes a feeling of heaviness or sleepiness accompanied by visual disorders, and sometimes also difficulty in opening the mouth. The paralyses caused by snake venom are reversible if proper treatment is given and they usually disappear in four or five days without any after-effects.

Generalised muscular pains and the presence of myoglobin in the urine are signs of poisoning by the bite of a sea snake (*Hydrophiidae species*).

Anaemia may follow loss of blood, either local or from the hollow organs. It may be aggravated by the existence of a concomitant disease conducive to anaemia. Massive haemorrhage may cause death, sometimes 12 days after being bitten.

In some parts of Africa, conjunctivitis due to snake venom is frequent. Snakes spit their venom, which they can project from four to six times over a distance of 4 m until they have exhausted the store accumulated in the poison glands. It is projected in the form
of a fine spray through the orifice in the fangs. African snakes can also inject the venom by biting.

Shock may occur after 24 hours. It becomes manifest as a profound apathy with loss of appetite, persistent thirst, weak and rapid pulse, pallor, abundant perspiration and loss of consciousness (see figure 20).

**Diagnosis**

Diagnosis is usually determined by the presence of wounds. If no wound is visible and the subject claims to have been bitten by a snake, diagnosis can be carried out after a period of observation.

The presence of wounds is not enough to justify a diagnosis of snake bite, especially if the snake has not been seen by the patient and if their appearance is not characteristic. The patient may have been bitten by another animal. With snake bites the pain steadily increases, unlike other bites with which the pain tends to decrease, with the exception of the bite of the black widow spider.

If the local phenomena are intense, the diagnosis will incline towards a bite by a viperine rather than a colubrine species (unless a tourniquet has been applied long enough). A precise diagnosis can be made only after the snake has been killed and examined. As a rule, the victim is not in a condition to give the details needed to identify the species in question.

**Prognosis**

According to statistics of 138 fatal cases, only 4 per cent of the deaths occurred within an hour of the bite, as against 17 per cent within 6 hours and 64 per cent between 6 and 48 hours. There is therefore time enough to give medical treatment.

The quickness of death depends on the position of the bite and the amount of venom injected. If the injection is massive, death may occur within half an hour, but this is very rarely the case.

The prognosis has much improved since the introduction of anti-venom serums, but these must be administered in good time.

The majority of deaths are due to lack of medical treatment.
Figure 20. Appearance of a patient bitten two days previously on the right hand. This illustrates most of the features of very severe systemic poisoning: the apathy of shock, limb swelling extending to the trunk, extensive blisters, and oozing from the bitten hand.

Source: ibid.

Prophylaxis

Prophylaxis consists essentially in wearing long trousers and top boots and in being particularly careful where one puts a hand or a foot in a region of dense vegetation that is reputed to be infested by snakes. It is also necessary to take special measures to destroy the snakes.
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Treatment

Everyone should know the species of snakes inhabiting the region in which he works, and also the local means of securing first-aid and medical treatment. Bites of harmless species should be treated, because the victim is hardly likely to be reassured by being told that the snake was harmless. However, the risk entailed by specific treatment is serious enough for it to be given only in the cases in which it is really indicated.

If the diagnosis is not certain, therapy will be confined to symptomatic treatment: treatment of the victim's anxiety and local treatment of the bite, while keeping the victim under observation for at least 24 hours and periodically testing the coagulation time in order to detect signs of poisoning and, if necessary, to begin treatment against it.

In India a large number of plants used to be considered effective in the treatment of snake bites and were used haphazardly. Systematic investigation of 322 of the 400 plants esteemed for their curative properties gave negative results.

The person bitten should be kept still to reduce the rate of propagation of the venom. All stimulants for the circulation should be forbidden, especially alcoholic drinks. The victim should be quickly conveyed to a hospital. A health education campaign should be undertaken to convince the public of the seriousness of snake bites and the need for prompt hospital treatment. On admission to hospital, victims should be given antidotes for tetanus and gas gangrene.

Broad-spectrum antibiotics should also be given in cases of necrosis, because the toxic action of the venom on the leucocytes and other phagocytes weakens the body's natural defences.

A mild sedative may be given to control anxiety. The pain must be treated, and sometimes it will be necessary to administer analgesics. The local application of heat or cold (ice-bag or ethyl chloride bomb) is contra-indicated.

Local treatment. The purpose of local physical treatment is to prevent the spread of venom. Physical methods have been used
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for centuries, but in recent years they have been resorted to only where specific treatment was unavailable—for instance, when the victim was a long way from a centre where he could be given antitoxic treatment, or when he had been bitten by a big snake.

A tourniquet is applied to the limb bitten, just above the bite, to impede the flow of lymph while allowing the venous circulation to continue. If the tourniquet is too tight, irreversible tissue lesions may result. Hence, it is necessary to loosen the tourniquet for 90 seconds every 15 minutes. A tourniquet that is correctly applied should be left in place as long as is necessary. It impedes the absorption of venom and allows some of it to be extracted by surgical methods. Moreover, venom that penetrates gradually into the circulation can be destroyed in the liver. However, the retention of the venom for a time by means of a tourniquet near the wound may conduce to local necrosis of tissue.

Puncturing the wound made by the fangs may allow the venom at the base of the bite to be removed, but it is liable to aggravate the lesions in tissue by bringing the venom into direct contact with it. During the past few years, this practice has been abandoned; it does not seem to have been effective and it creates risks of infection and necrosis of tissue.

A considerable amount of venom can be extracted by suction, which may be practised at 15-minute intervals for 8 to 12 hours. Suction may be applied by mouth or a pump, or by alternating positive and negative pressure with the help of a special appliance. Oral suction carries a risk of infection, but the venom does not constitute any risk for the operator unless he has lesions in the mouth.

The local application of permanganate of potash, gold chloride or carbolic soap has proved effective by reducing the toxicity of the venom, but cauterisation by strong acids or permanganate of potash must be avoided.

Necroses are very difficult to cicatrise, and the process is very slow. If necrosis is extensive, skin grafts or even amputation may be necessary.

For conjunctivitis, irrigation with sterile water is sufficient. Diluted antidote may be added to the water locally.
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*Diet.* Liquids are given for three or four days after the bite. In cases of weakness or giddiness, black coffee or strong tea can be given. If there are difficulties in swallowing, liquids can be administered intravenously for three to seven days.

*Treatment by antidote.* The high proportion of deaths from snake bite has been considerably reduced by the introduction of a specific polyvalent antidote.

Antidotes are prepared by hyperimmunisation of horses against one or more venomous snakes. The stability and efficacy of the various sera on the market differ widely.

The Wyeth antidote is a powder that must be dissolved before use. It keeps for five years. The Habu antidote is sold in ampoules containing 20 ml of the dry product, purified and frozen in a plastic box and kept dry by silica gel. It too must be dissolved in distilled water before it is used.

The purpose of treatment by antidote is to neutralise the venom that the organism has not been able to destroy, and also to deal with the general phenomena resulting from poisoning by snake venom.

Specific therapy can be employed only as curative and not as prophylactic treatment. The therapeutic effect on the general phenomena is spectacular, but local phenomena are unaffected. Consequently, it is necessary to identify the clinical signs of poisoning before administering antidotes.

Antidotes based on the venom of certain snakes may be effective against bites of snakes of the same species, and to a lesser extent against bites from different species: thus, the specific antidote for pit vipers is not effective against the coral snake but it may be of some use if no specific vaccine is available.

Antidotes based on horse serum may cause painful local reactions or allergic reactions in persons who have been sensitised or who are predisposed. Allergic reactions may be very violent and more serious even than the bite that is being treated. Consequently, measures must be taken to prevent and treat this possible complication. Close questioning will disclose the personal and collateral

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allergic antecedents of the victim as regards injections of heterologous serum. The sensitivity of the conjunctival sac or the skin can then be tested.

The reaction to the antidote may be anaphylactic, with serious hypotension leading to death, or it may be slight, with itching, coughing and fever.

The risk of these complications justifies medical treatment in a hospital, where all means are available for treating any shock and where the dangers of administering serum can be reduced to a minimum.

The dose of antidote should be sufficient for the quantity of venom assumed to have been injected. It will vary from one to five ampoules, depending on the size of the snake, on the interval between the bite and the start of treatment and on the clinical condition.

4.4.2. Insect stings

Insects sting more readily on hot and clear days when they are hampered in their search for nectar, and after heavy rain when they cannot find any nectar. They are attracted by bright colours and do not pay much attention to white clothes. They are also attracted by some scents and cosmetics.

At least 100,000 persons are stung every year by insects, but statistics of fatalities are very vague: some deaths due to insect stings are counted as sudden deaths from supposed heart diseases. It is usually impossible to identify the insect responsible for the sting, for it is seldom captured. However, the insect can be identified retrospectively by taking account of the time of the sting, the place (field, forest, etc.), the presence of nests of insects in the vicinity (underground, in trees, in bushes, in cellars) and the presence or absence of a sting.

In the bee, the sting is normally retracted in a compartment of the abdomen (the sting chamber). At the moment of stinging, the sting is protruded. Bees sting only once because they cannot recover their sting, which (together with the poison sac, intestines
and muscles) remains in the victim after the bee has flown away (see figure 21). The poison sac continues to empty itself because of the contraction of the muscles still attached to it. It takes three or four minutes to empty the sac, and it is therefore very important to take out the sting as soon as possible after the stinging.

Other insects save their sting; they can therefore sting several times by injecting a certain quantity of poison each time.

Symptoms

A clear distinction must be drawn between local irritative reaction and allergic reaction. A person who is stung has a certain reaction with the injected liquid, which is probably related to acetylcholine.

An allergic reaction occurs only in persons who have been sensitised; it is due to the proteic fractions of the poison that are also found in the bee’s body. Among the Hymenoptera (a large order of insects, including bees, wasps and ants—see figure 22), from four to six antigen proteic fractions have been found. Two of them are common to all the members of the order; the others are confined to a particular group.

Pollen has been found on the sting, and this might be held responsible for the allergic reaction. A first sting may sensitise a person for a later sting, which would initiate an allergic reaction; sometimes, however, reactions to subsequent stings are progressively weaker and a natural immunity develops.

There is a rapid succession of general symptoms: headaches, nausea, vomiting, dizziness, weakness, dyspnoea, collapse, unconsciousness, shock. The sooner the general symptoms appear, the more violent the reaction. If there is shock, it appears within five minutes.

Sometimes there will be small areas of bleeding under the skin, abdominal cramps, coughing and convulsions.

Pathological anatomy

Autopsies have disclosed congestion of internal organs, haemorrhages characterised by small patches under the skin and in
Figure 21. Stinging technique of honeybee. (Top) A female worker honey-bee in act of inserting the barbed sting into the skin. Once the sting is thrust into the skin, the honey-bee cannot withdraw it. (Middle) As the honey-bee pulls and tugs to free herself after inserting the sting, she is disembowelled and will fly away to die. The bowel, muscles and venom sac are left behind with the sting, which is embedded in the skin. (Bottom) Honey-bee sting with venom sac (x 4).

Figure 22. Serious and potentially fatal anaphylactic reactions may be caused by stings from arthropods of the Hymenoptera group of insects. Source: ibid.

the mucous membranes and the central nervous system, dilatation of the right heart with mycorditis, acute pulmonary oedema and gastro-enteritis.

In many cases, lesions of the central nervous system have been found with cerebral oedema, haemorrhagic meningitis, and so on.

**Diagnosis**

Often the victim pays no attention to an insect sting and does not report it to a doctor. A case is reported of a woman stung by an
insect who, within a few minutes, felt giddy, with faintness and difficulty in breathing. She called a doctor and was just able to write “stung” in the list of telephone numbers before losing consciousness. This one word enabled the doctor to give suitable treatment in good time, and the woman’s life was saved.

Persons who are as susceptible as this should carry on them a card stating that they are allergic to bee stings, and perhaps indicating the treatment to be given if they are found unconscious.

Sensitivity to insect poison can be diagnosed by means of separate extracts of some of the most dangerous kinds. The method is to start with very weak solutions and progress until the concentration that sets off the reaction is reached. This concentration multiplied by ten is taken as the starting-point of the hyposensitisation treatment.

In sensitisation tests, several injections can be given in one sitting, but one must beware of the cumulative reactions which are sometimes dangerous.

**Prophylaxis**

The rapidity with which the symptoms appear and the impossibility of giving the requisite treatment promptly create problems of prophylaxis.

Once the allergic nature of the symptoms has been recognised, prophylaxis will consist of the adequate hyposensitisation of persons who are hypersensitive to insect stings. Persons who have already suffered a general reaction after an insect sting should be given prophylactic treatment, because sensitivity may increase after each sting and the clinical picture may worsen considerably. The treatment should be given by a medical specialist.

During the treatment, patients are advised always to carry on them a first-aid kit with a tourniquet, pincers for removing the bee sting and the medicaments prescribed by the doctor. If this is done, patients will have the means required for essential first-aid treatment ready for use at the first alarm.

In order to achieve hyposensitisation, it is desirable to use a mixture of antigens affording protection against a number of in-
sects, because a patient may be made sensitive to different antigens and it is difficult to identify them in any given case. It is even very difficult sometimes to determine to which particular insects a person is sensitive. An extract of the total body of the insect will give a more effective antigen than the contents of the poison sac alone. The reason for this is that the poison sac contains less antigen than the whole body.

The antigen used in the majority of cases is an extract of a mixture of frozen live insects. Dead insects cannot be used because of post-mortem changes in their proteins. The extracts on the market are in the form of a powder from which a buffered glycerin solution is prepared for tests or treatment. It should be remembered that there is a refractory period of 10–15 days after an insect sting when the tests could give negative results. Accordingly, desensitisation should not be undertaken until a few weeks after the patient was stung.

Injections should be carefully controlled, because an overdose or an intravenous injection might set off a dangerous reaction.

Hypoallergenic patients who have been stung again have developed only local reactions. However, it is advisable that they should always carry their first-aid kit with them, so that they can treat themselves immediately if the general symptoms should appear after a sting.

*Treatment*

Treatment should be given immediately after a sting, for the time of survival is sometimes very short. First of all the sting should be extracted, care being taken not to squeeze the poison sac. The application of a tourniquet near the place stung will slow down the absorption of poison if a limb has been stung. The tourniquet should not be too tight (the pulse should be perceptible) and should be loosened frequently. The wound should be cleaned and fresh compresses applied to it. Cold will slow down the absorption of poison and reduce local pain. Local itching can be reduced by the application of antihistaminic creams. Medical treatment should be given and supervised by a specialist.
4.4.3. Spider bites

The average fatality rate for bites of spiders is 4 per cent, but is especially high among children. Death usually ensues from 12 to 24 hours after a bite, but it may occur after a few days or even a week.

It is the female spider that is responsible for most bites. They are inflicted from May to October, at times when young spiders are born.

*Lactrodectus mactans*, the black widow spider, is extremely dangerous. The female’s body is about 1 cm long with a red spot on the abdomen. *Loxoceles laeta* and *Loxoceles reclusus* have a poison with local narcotic and haemolytic properties in addition to the neurotoxic properties common to all spider poisons.

The poison of *Lactrodectus mactans* is 15 times more virulent than that of the rattlesnake. It is produced in glands in the cephalothorax. The distal segments of the first pair of chaliceræ have two curved processes in the form of fangs which inject the poison.

The poison has a necrotic and hypertensive action; it also acts on the cerebral cortex and the vasomotor centres of the dorso-lumbar marrow. It excites the central and peripheral nervous systems, causing muscular spasms.

**Symptoms**

The local symptoms of a spider bite appear after two hours and the clinical picture is complete in a few hours. An irritation appears at the site of the bite, with pain beginning after 15 to 60 minutes and spreading steadily over the limb affected and then to the trunk. The pain is almost unbearable and is accompanied by anxiety, painful cramps in the muscles of the trunk or of a limb, hypertension, nausea, vomiting, headache, sweating, salivation, contractions and shivering. Sometimes the symptoms persist for several days if no specific treatment is given.

After bites by *Loxoceles laeta* or *Loxoceles reclusus*, anaemia and haematuria may appear.
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Differential diagnosis

In a number of cases, differential diagnosis must be undertaken for perforated ulcer, pancreatitis, volvulus of the small intestine, nephrectic colic, coronaritis and similar diseases. In the case of spider bites, the abdomen does not exhibit signs of urgency.

Treatment

The patient is put to bed, especially if he suffers from high blood pressure. To reduce the pain and delay the absorption of poison, ice-bags are applied to the wound.

When the pain has been moderated by the treatment, a test for sensitivity to the antidote is made. The antidote is the serum of a horse hyperimmunised by the poison of *Lactrodectus mactans*.

4.4.4. Scorpion stings

Scorpions are arthropods. They range from 2 to 20 cm in length, and the glands that produce their poison are in the terminal segment. At the end of the tail is a sting by which the poison is injected into the victim. Scorpions do not attack human beings; stings are due to accidental contact.

The majority of scorpions make a non-lethal poison that gives rise only to a pronounced local reaction. In the poison of some species of North African scorpions, two proteins have been found (the scorpamines). Their action is purely neurotoxic and, all other things being equal, they are more toxic than snake venom; however, the quantity injected by the scorpion is much smaller.

The poison of the lethal species of scorpions has an elective action on the nervous system. The fatality rate due to scorpion stings is much higher among children and old people (among children under five years of age it amounts to between 1 and 2 per cent) than among adults.

Symptoms

Pain sets in at the wound and there is a burning sensation. If the sting comes from *Centruroides sculpturatus* or *Centruroides getschi*, there is also local hyperaesthesia.
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Stings from species with a lethal poison produce general symptoms as a result of their neurotoxic action; this makes itself felt quickly and lasts from 4 to 48 hours and sometimes longer. The victim is agitated, he weeps, his nose runs, he vomits and he suffers from epigastric pains, heightened abdominal sensitivity, fever, profuse sweating and shock.

The convulsions that occur are of the tonic type. They are accompanied by trismus and other muscular spasms. Generalised spasms of the flexor and the extensor muscles cause alternating postures. There are also difficulties in speaking, swallowing, breathing and making movements. In addition, the tongue feels thick and there are mydriasis, diplopia, temporary loss of sight and sphincteric incontinence.

The convulsions resemble those produced by strychnine poisoning, and they may be fatal if they are not treated in time.

Cardiovascular symptoms also occur, including increased heart rate, disturbances of rhythm and very weak pulse. The electrocardiogram shows evidence of myocarditis. Death may ensue from acute pulmonary oedema.

Treatment

Local treatment consists in applying a tourniquet in order to delay the absorption of poison. It should be loosened every five to ten minutes. An ice-bag can be applied to the wound for at least two hours (the ice must not be applied directly to the skin), again in order to delay absorption.

The pain can be reduced by a local injection of a solution of procaine and adrenaline. Morphine and substances related to it are contra-indicated, as it has been demonstrated that they have a synergistic effect on the activity of the poison and that this effect is proportional to the dose.

Breathing may be improved by oxygen therapy.

The most promising treatment, however, is to administer the immune serum. If no specific antidote is available, one for a related species can be used.
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If no immune serum at all is available recourse should be had to local and symptomatic treatment.

Prophylaxis

Prophylaxis consists essentially in educating the population to avoid infested areas, inspect bedding before retiring and shake clothes and footwear before putting them on. Prevention also consists in making the house vermin-proof, destroying weeds and removing stones and other objects round about the house. Insecticides such as kerosene and chlordane can be used to spray the surroundings of houses and workplaces.
5. Diseases particularly related to agricultural work: zoonoses, infectious and parasitic diseases

5.1. Introduction

Zoonoses are generally defined as diseases and infections transmitted directly between animals and man. In this comprehensive treatment of the problem, however, the term will be given a wider interpretation to include diseases common to animals and man. This definition includes the paprozoones, which are not transmissible directly between animals and man but have a common inanimate reservoir, such as soil.

The Joint ILO/WHO Committee on Occupational Health considered zoonotic diseases of occupational significance to agricultural workers in a report published in 1962. The classification of diseases proposed by the Committee is generally adopted in the following discussion. The space allotted here to each disease is proportionate to its estimated importance as an occupational disease of agricultural workers.

A comprehensive report on zoonotic diseases of occupational significance to agricultural workers in the United States has recently been completed. This report deals with many conceptual problems which are inherent in the subject, regardless of geographical location; moreover, many of the diseases considered therein are important in other parts of the world.

Certain problems that arise in any discussion of zoonoses should be kept in mind. First of all, there is a severe lack of specific data

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2 *State-of-the-art report on safety and health in agriculture*, comparative medicine addendum (Oakdale, University of Iowa Institute of Agricultural Medicine for the National Institute of Occupational Safety and Health, 1974).
5. Zoonoses, infectious and parasitic diseases

on the occurrence of zoonoses in the human population. In many cases, the agricultural workplace and the home environment are one and the same; thus, it is often very difficult to separate strictly occupational zoonotic diseases from general environmental zoonotic diseases. Second, there is the geographical element: for various reasons, certain zoonotic diseases are more prevalent in some areas than in others. Third, the existence of zoonotic diseases is dependent on many factors, such as the host, the agent, the environment, time and space, and will be influenced by anything influencing any one of these factors. Changes in agricultural technology and urbanisation are very important factors in this respect. Any examination of zoonoses must therefore take account of these limitations and problems; moreover, for want of adequate and reliable data, any decisions regarding the occupational nature of many of the diseases can only be subjective.

5.2. Viral and rickettsial diseases

5.2.1. Classification and geographical distribution of diseases caused by Russian tick-borne complex

<table>
<thead>
<tr>
<th>Area</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>British Isles</td>
<td>Louping ill of sheep.</td>
</tr>
<tr>
<td>USSR</td>
<td>Russian tick-borne or spring-summer encephalitis.</td>
</tr>
<tr>
<td></td>
<td>Bi-undulant meningo-encephalitis or diphasic milk fever.</td>
</tr>
<tr>
<td></td>
<td>Omsk haemorrhagic fever.</td>
</tr>
<tr>
<td>Central Europe</td>
<td>Tick-borne viral encephalitis.</td>
</tr>
<tr>
<td>India</td>
<td>Kyasanur forest disease.</td>
</tr>
</tbody>
</table>

There are several strains of these viruses. The differences between them seem to lie in the severity and degree of central nervous system involvement. The louping-ill strain shows fewer pathological effects in man than the other diseases mentioned. Russian tick-borne encephalitis shows relatively greater pathology.
Omsk haemorrhagic fever and Kyasanur forest disease show little central nervous system involvement. However, systematic and haemorrhagic manifestations are described.

Epidemiology and occupational characteristics

Russian spring-summer encephalitis is characterised by short seasonal outbreaks in the spring, summer and autumn. These depend on the activity of the vector. The infection is also believed to be transmitted by the ingestion of milk from infected goats, which may result in urban epidemics. The resulting infection has been called bi-undulant meningo-encephalitis or diphasic milk fever. The disease occurs in rural areas among agricultural workers.
and, particularly, in forested areas among woodcutters. Animal carriers of the viruses include sheep, goats, small mammals and birds (see figure 23). Hens' eggs may also contain the virus from infected birds. Transmission occurs through the bite of ticks when tick-infested animals are handled, through the crushing of ticks and through contact with rodents, wild birds and wild rats. The fact that the disease occurs mainly among young adults points to its occupational character. The tick carriers in the USSR are *Ixodes*
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*persulcatus* and *I. ricinus* (see figure 24). The dissemination of the disease is thought to result from the immigration of feral birds and rodents.

Diagnostic serology includes serum neutralisation and complement fixation. Detectable serum-neutralising antibody may persist for 20 years following infection with Russian strains. The complement-fixing antibody is of short-term duration.

**Clinical picture.** The diseases produce a variety of clinical manifestations. Infection may be inapparent or mild, and may escape diagnosis unless laboratory aids are used. The diseases may also be more severe, with possible permanent disability or death. The onset is sudden, with generalised systemic infection such as fever, severe headache, malaise, generalised pains and gastrointestinal manifestations.

A biphasic body temperature rise is usually (but not always) seen. The first phase lasts for 5 to 10 days and the second for 8 to 12 days. The second phase is accompanied by signs of central nervous system involvement. The peripheral blood shows a mild leucocytosis in infections involving the nervous system.

The central nervous system manifestations include spinal, bulbospinal and ascending types of paralysis and encephalitis. Paresis or paralysis, usually of the flaccid type, may be transitory or permanent. Sequelae may include chronic spasticity and epileptiform seizures. Other symptoms may include nystagmus, vertigo, and visual and mental disorders leading in some conditions to delirium and coma.

Vomiting, diarrhoea and albuminurea may be marked in Kyasanur forest disease, which is usually more serious and more frequently fatal than Omsk haemorrhagic fever.

**Diagnosis.** The diagnosis depends mainly on symptoms and history. The highest incidence of the diseases occurs in occupations in forest areas or where contact with ticks or infected animals is probable. Virus isolation from the blood is possible through chick embryo tissue culture or mouse inoculation. Serological diagnosis
5. Zoonoses, infectious and parasitic diseases

by complement-fixing tests or neutralisation tests gives reliable results.

_Treatment._ There is no specific treatment, but the use of convalescent human or hyperimmune horse serum or goat serum may be of value. This is given early in infections with the neurotropic strains. Hospitalisation is important, as fluid and electrolyte therapy are often essential. Permanent disability (such as partial paralysis) may be prevented by physiotherapy.

_Prevention._ Formalised mouse brain vaccine has been used in the USSR with good results. Vaccine prepared from sheep cord and brain has also been found successful. Such vaccine is given to potentially exposed workers in tick-infested areas. Serum prophylaxis may be helpful following tick bites in endemic areas and in accidental laboratory exposures. General protective measures, such as protective clothing, are also recommended for populations at risk. Rodent control and the use of insect repellants are valuable preventive measures. In order to exterminate ticks, aerosol pumps using acaricide have been used to good effect, and sheep dipping may also be of help in controlling them. Milk should be boiled or pasteurised whenever there is a possibility of contamination by the virus.

5.2.2. **Q fever**

This is a disease caused by a rickettsial organism, _Coxiella burnetii_. The primary cause of the disease is occupational exposure in agricultural work. Several epidemics of Q fever were reported in the armed forces during the Second World War and in some urban areas. These were found to originate in contacts with contaminated environments in the villages and in the ingestion of contaminated raw milk. The disease is recognised as an important public health problem in some parts of the world, and is a reportable disease in many of these areas. There have been no reports from northern European countries, including Scandinavia.
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Epidemiological and occupational aspects

Causative organism. *C. burneti* is an obligate intracellular parasite. However, it may survive for long periods in the environment, and is quite resistant to heat and desiccation. It can survive temperatures above 60°C in milk suspensions. In man, the major sources of infection are cattle, sheep and goats.

*C. burneti* exists in wildlife as well. Feral mammals and birds have found to be naturally infected. Ticks are believed to be an important vector in the wildlife form of Q fever. The epidemiological relationship of the domestic and feral forms of Q fever is not fully known.

Transmission to man. The inhalation of aerosolised organisms in dust is generally considered to be the most important route of transmission in man. In the USSR, transmission by ticks has been considered a major route of human infection. Milk from infected cows or goats is also an important potential source of infection. Aerosolised organisms are thought to be an important mechanism of transmission in domestic animals also.

The organism settles in the mammary glands and uterus of infected domestic animals. During pregnancy, the organism multiplies in the uterus, and on parturition it is present in high numbers in the placenta and uterine discharges. The livestock environment may become heavily contaminated at this time.

Occupational exposure. From the occupational point of view, most cases of Q fever occur in workers handling animals and animal products on the farm. The husbandry of milking herds and the handling of infected meat, hides and skins offer potential sources of infection. The use of livestock wastes for fertiliser or fuel are also possible sources of occupational or environmental exposures. Several outbreaks of the disease have been reported among abattoir workers. Cases of indirect contact have been reported in persons laundering shepherds’ clothing, persons passing through contaminated environments and persons transporting infected animals.
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Course and clinical features. There are no characteristic symptoms for Q fever. This makes diagnosis difficult. The symptoms and clinical course are quite variable, and may often be confused with influenza. Upper respiratory symptoms may occur, with coughing and signs of pneumonia. Gastro-intestinal disturbances may also be seen. The incubation period varies from 14 to 26 days, with an average of 20 days. The onset of the disease is usually abrupt, with fever, chills, malaise and myalgia. The disease generally runs a mild course in man, and recovery may be spontaneous.

Fatalities are rare and usually associated with complications such as progressive cardiovascular involvement. Treatment in acute stages is with broad spectrum antibodies.

Diagnosis. Laboratory assistance is usually required for diagnosis. Complement fixation or micro-agglutination are the standard techniques. Antibodies may reach a detectable level by the seventh day of the disease and an optimum level after 20 days. Isolation from the blood may be accomplished by inoculation of chick embryo yolk sacs, or of guinea pigs during the febrile stage of the disease.

Prevention

Little interest has so far been shown in the prevention of Q fever, probably because of its low economic significance as regards livestock and because of the usual mild nature of the disease in man. However, the potentially serious complications of this disease are not widely known, and public health education and control programmes to this end would be justified. There are also good grounds for making Q fever a reportable disease.

The control of Q fever depends largely on its prevention in domestic animals. Trial vaccinations of dairy cattle have been quite successful but have not been administered regularly. The serological screening of milking herds, with subsequent isolation of the infected animals, may be a step towards establishing “clean” herds.
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A vaccine for humans is available, but is not in general use at present. A drawback to its use is that it may cause allergic reactions in sensitive individuals. Intradermal tests are advisable before vaccination.

Environmental sanitation is essential if the disease is to be controlled. At parturition, animals should be kept under observation and the placentas should be immediately removed and disposed of in a sanitary manner.

Heat treatment of milk is essential. Pasteurisation at 61°C may not kill the organisms; a higher temperature for a longer period is required.

5.3. Bacterial diseases

5.3.1. Anthrax

As with most zoonotic diseases in man, anthrax throughout the world is severely under-reported. Approximately 9,000 cases are reported to the World Health Organization each year. Considering that many countries do not report the disease at all, or do so inadequately, its annual world-wide occurrence has been estimated at approximately 100,000 cases.

The majority of reported human cases are associated with agricultural work; the remainder are mainly industrial in origin. Most industrial cases are related to the skin and hide industry, especially where there are no quarantine regulations concerning imported hides and skins. Anthrax is endemic in countries in central Africa, Latin America, some parts of Asia and parts of North and South America.

Epidemiological and occupational aspects

Causative organism. Anthrax is caused by Bacillus anthracis, which is transmitted to man through direct or indirect contact with domestic animals or animal products. This organism, in its spore form, is very resistant to chemical and physical factors and
5. Zoonoses, infectious and parasitic diseases

can survive for years in soil, hides, skin and bones. Anthrax spores can transform to the vegetative form and multiply under appropriate environmental conditions, increasing the risk of animal and human exposure.

*Occurrence in animals.* Anthrax is mainly a disease of cattle, sheep and horses. The primary route of transmission in animals is by ingestion; rarely by insect bites. The illness is usually peracute in animals and leads to rapid death. The organism multiplies in the blood and may spread to most body tissues. It is excreted in the urine and faeces.

*Occupational exposure.* Anthrax is essentially an occupational disease of agriculture and industry. In agriculture, the handling of infected animals leads to human exposure. Farmers, herdsmen, shepherds, animal skinners and persons working with hides and skins are at risk if the disease exists in their livestock. In some cases, farmers may be exposed to anthrax from contaminated vegetables or from soil. Fertilisers made of animal bones may contain anthrax spores. Foodstuffs of animal origin and other agricultural products may also be contaminated.

The skin, hair, wool, meal, bones, horns and hooves of animals that have died of anthrax are likely to be contaminated with spores. Direct man-to-man transmission is not known.

In industry, anthrax has been a problem in countries where wool, skins and hides are used in manufacturing. In many countries, anthrax is a compensable occupational disease.

*Anthrax in man.* Anthrax occurs in three primary forms: cutaneous, respiratory and gastro-intestinal:

1. **Cutaneous anthrax:** the organisms usually enter through breaks in the skin, especially on areas exposed during work (hands, forearms, face, neck). The lesion begins as a small pruritic papule which becomes enlarged and more inflamed, with small vesicles. The central portion coalesces to form a blister which may rupture and discharge a clear serous fluid. The anthrax organism is usually found in this fluid. The lesion
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proceeds to local necrosis, and in severe cases extensive oedema is observed, with lymphangitis and swelling. There are mild systemic symptoms of fever and malaise. The regional lymph glands are tender and enlarged. If effective treatment is begun, the condition regresses; otherwise, in 5 to 10 per cent of untreated cases, the condition ends fatally, as a result of progressive cellulitis, sepsis and bacteraemia.

(2) Pulmonary anthrax is generally a much more serious disease than cutaneous anthrax. Septicaemia and respiratory embarrassment from massive oedema of mediastinal nodes result in a high mortality rate. The handling and sorting of infected wool commonly leads to respiratory infections.

(3) Gastro-intestinal anthrax is not often reported. It occurs among rural people in Asia and Africa where infected meat is eaten undercooked. The mortality rate from gastro-intestinal anthrax is lower than that from pulmonary anthrax, and the manifestations are less severe. Diarrhoea and other acute gastro-intestinal manifestations are the main symptoms.

Prevention

The control of anthrax depends on a series of preventive measures directed towards animal reservoirs. Early diagnosis of the disease in animals is essential to help to prevent dissemination within the herd. The health education of farmers is important. Laboratory assistance is also important in diagnosis. Sick animals should be condemned and the rest of the herd should be observed and vaccinated. Animals that have died of anthrax should be covered with lime and be buried on the spot in pits 2 m deep. Alternatively, the carcass may be incinerated.

Live animals may be effectively vaccinated against anthrax with spore vaccines. Vaccination must be carried out regularly (every six months to one year).

Contaminated soil, vegetables and implantations are difficult to detect, let alone control. Health education may help to teach workers to avoid areas that may possibly be contaminated.
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In many countries, imported hides, skins and wool from suspected areas are disinfected with steam, formaldehyde, mercury salts or formic acid. All imported animal products, such as bones and fertilisers, should also be disinfected by appropriate and effective methods.

Although cases reported following the ingestion of milk or meat are rare, in order to avoid contamination from infected animals sanitary methods should be used to handle milk products and meat, which should be pasteurised or thoroughly cooked. Meat from known infected animals should be condemned.

In several countries, the vaccination of humans has given promising results. In occupational groups where anthrax is a potential risk, workers were vaccinated using a cell-free vaccine. Three doses were given over a period of 18 months.

Human cases of anthrax, particularly cutaneous anthrax, are treated with penicillin or broad spectrum antibiotics or sulphonamides.

5.3.2. Brucellosis

Brucellosis is transmitted to man through contact with goats, cattle, cows and hogs, or contact with contaminated products of these animals. There are three species of Brucella: B. melitensis (associated with sheep and goats), B. suis (swine) and B. abortus (cattle). The disease is fairly cosmopolitan in distribution, and has been recognised as being of major importance in the Mediterranean area, the USSR, central Europe, North America and some parts of Asia. Synonyms for brucellosis in man are undulant fever, Malta fever and Mediterranean fever.

Epidemiology

Occurrence in animals. Infection in animals may persist for years or for a lifetime, and may spread continuously or in bouts to other animals or to man. In the Mediterranean area and in parts of Asia, infected sheep and goats are the primary reservoirs of brucellosis for man. In North America and central Europe, cattle
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used to be the primary source, but more recently *B. suis* has been responsible for most human cases in these areas, due to the relative decrease in the borne disease as a result of established control programmes. Direct and indirect contact of infected to non-infected animals is the principal means of inter-species transmission. The reproductive tract is a primary target organ for chronic infection with brucella. The primary clinical sign of most animal infections is abortion, and the organism may be shed in high numbers in the placenta and uterine fluids. The animal contaminates by this route.

*Transmission to man.* Brucellosis is mainly an occupational disease of farm workers. As with most zoonotic diseases, accurate rates are not available because this disease is universally under-reported. This may be due to lack of awareness, to insufficient medical services or to the difficulty of reaching a diagnosis using ordinary clinical methods. The available reports show that such agricultural occupational groups as shepherds, livestock breeders, milk handlers, dairy workers, meat packers, slaughterhouse employees and meat inspectors are especially at risk of contracting brucellosis.

In man, the *Brucella* organism enters the body through skin abrasions, the mucous membrane of the mouth or nose, the respiratory tract or the alimentary canal with ingested food (see figure 25).

The handling of infected animal tissues is an important source of infection, especially in abattoir workers. Veterinarians in particular may be infected during their attendance at the abortion or parturition of cows. The transmission of the disease from man to man is very rare. The ingestion of contaminated milk is the primary non-occupational source of infection for man.

*Clinical picture.* The course of the disease in man is quite variable—from mild and insidious to very acute and severe. The first type is known as the “intermittent” type, because of the repeated course it follows. The second is the malignant form of brucellosis. Generally, the symptoms are a feeling of weakness, sweating
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Figure 25. Pathogenesis of brucellosis

Seizures, chills, headache, joint pains, neuralgia and visual disturbances. The appearance of any one or more of these symptoms and their severity depend on the degree of virulence and the susceptibility of the individual. The clinical signs may include fever, hepatosplenomegaly, jaundice and lymphadenitis. Skin lesions have been described but occur infrequently. In some cases, coughing and even cardiac manifestations may be observed. The acute phase may lead to disability for a few weeks. Although the mortality rate is low, a chronic form of the disease may evolve if it is untreated or undiagnosed and inadequately handled. More severe and disabling forms may occur as a result of chronic conditions and complications. In one report, it was estimated that 10 per cent of the cases evolved to chronic brucellosis for such reasons. In chronic brucellosis, several complications due to chronic localisation of the organism are described. Chronic debilitating spondylitis and bone osteomyelitis with joint suppura-
tion may occur. The lumbosacral spine and pelvis are sites of predilection, often with permanent disability. Other complications (such as subacute bacterial endocarditis, hepatic necrosis, hepatitis and liver cirrhosis, genito-urinary chronic cystitis, pyelonephritis and meningeal involvement or encephalitis) have been described. Pneumonia due to *B. suis* and ocular derangement with optic atrophy and septic retinitis have also been reported. In some cases of chronic brucellosis, neuropsychiatric disorders are observed.

**Diagnosis.** Laboratory assistance is important in order to establish a diagnosis of brucellosis. Serology is the most common specific laboratory procedure used. Isolation attempts by blood culture and animal inoculation may be helpful. Skin tests have also been used, but chiefly as a screening technique. Clinically, brucellosis must be differentiated from other febrile diseases such as leptospirosis, typhoid fever, Q fever and a host of other bacterial, viral and rickettsial diseases.

**Treatment.** The treatment of brucellosis with antibiotics and antibacterials is difficult, owing to the intracellular location of the organism. Some success in shortening the course of the disease has been reported with tetracyclines, streptomycin and sulphanamides. However, the drugs have to be used for quite extended periods, in comparison with the length of time required to treat non-intracellular bacterial diseases.

**Prevention**

The prevention of brucellosis is of major importance, and will result in a subsequent decline of the disease in the human population. The following measures of prevention are suggested:

(1) Vaccination of herds reduces the occurrence of brucellosis. The vaccination of animals may be started early (for instance, at under four months in calves) to ensure longer protection, and to avoid confusing active infections with vaccination reactions.
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(2) Animals should be serologically tested for Brucella agglutinins and the reactors should be isolated and slaughtered. An approved serological procedure should be used.

(3) The vaccination of human high-risk exposure groups may be considered in some countries.

(4) The periodical serological testing of risk groups would provide data on surveillance which may be utilised with other epidemiological data to monitor and modify control programmes.

(5) The education of workers as regards personal hygiene is important in order to avoid contamination of clothes, hands, other parts of the body, superficial wounds and food. Measures should also be taken to ensure the proper use of protective rubber gloves and clothing by workers engaged in handling potentially infected animals and animal products. Environmental sanitation and the disposal of wastes from herds are other important measures. Milking should be carried out under strict sanitary supervision.

(7) The proper engineering procedures should be utilised to prevent exposure to airborne infection in abattoirs and other high-risk sites. Personal protection (face shields, respirators, and so on) should be considered for temporary usage in lieu of proper sanitary engineering measures.

(8) The pasteurisation of milk before drinking or manufacturing milk products is important.

5.3.3. Leptospirosis

In the year 1886 Weil described a few cases of infectious jaundice with fever, nervous symptoms, liver enlargement and renal involvement. The causative organism Leptospira icterohaemorrhagiae was identified in Japan in 1916. The disease leptospirosis was thereafter recognised in many countries and became known under several names: Weil's disease, leptospiral jaundice, spirochaetal jaundice, infective haemorrhagic fever and mud fever.
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Leptospirosis occurs in many animals and in man in all countries of the world. Endemic areas for leptospirosis include most of Europe, Asia (India, Indonesia, Japan), South America, North America, Australia and New Zealand. Reports from Africa are scarce: however, a few studies have demonstrated the presence of leptospirosis in rats in Egypt.

Epidemiology

Causative organism. Leptospirosis is caused by many different serotypes of L. interrogans. The different serotypes vary in pathogenicity, in the symptoms produced in man and in epidemiology. Leptospirae have limited resistance to environmental stresses, and are quite susceptible to an acid pH and other physical and chemical factors. They are killed by exposure to a temperature of 45°C for 30 minutes; at 70°C they are killed within 10 seconds. Salt water or direct sunlight kills the organism. Leptospirae can survive for long periods in moist soil, mud and fresh water, particularly if the pH is slightly alkaline.

Occurrence in animals. Particular serotypes are most commonly associated with a particular host or set of hosts. In some areas, cattle, swine, sheep, goats and horses become infected. In addition to domestic animals, a variety of wild animals are affected, such as foxes, raccoons, squirrels and other mammals. The host animals may, without obvious clinical signs, become chronic renal shedders of leptospirae, leading to the excretion of large quantities of organisms in the urine.

Rats (a very important reservoir) and dogs are usually infected by L. icterohaemorrhagiae and L. canicola respectively. Specific serotype host relationships vary with geographical location. Thus, for epidemiological reasons, it is important to know these relationships if the disease is to be controlled. Leptospirosis is transmitted through direct or indirect contact with urine or tissues from infected animals. Water is an extremely important medium for transmission between animals and from animals to man. Leptospirosis has very great economic significance in most livestock-
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raising areas, particularly as regards the breeding of cattle, sheep, and swine; abortion is the primary manifestation.

*Occupational exposure and transmission to man.* Reports of leptospirosis in different parts of the world show that the disease is most common among agricultural workers. Animal carriers such as rats excrete contaminated urine into suitable media (for example, water or mud) in fields; workers in rice and sugar-cane plantations are especially at risk, since they spend such a long time in contact with water and mud. The major route of infection is through abrasions, cuts and cracks in the skin. Bare-footed workers with cracks in the skin of their feet (resulting from long exposure to water or possibly from injuries) pick up leptospiroaee as they walk through contaminated mud or water (see figure 26). The leptospiroaee also enter the body through the mucous membranes of the mouth, nose, nasopharynx and conjunctiva. The ingestion of contaminated food or water is probably a minor source of infection in man. The transmission of leptospirosis from man to man is also of minor importance.

In agriculture, workers in irrigated rice-fields, canal workers, livestock producers, abattoir workers and veterinarians constitute the majority of high-risk groups. Occupational leptospirosis has also been reported in groups other than agricultural workers—for example, fish workers, coalminers, sewer workers, builders, soldiers and laboratory workers.

Non-occupational exposures may occur during bathing in polluted waters. A substantial proportion of cases of leptospirosis are reported as being due to swimming and other external uses of polluted waters, such as washing of clothes. There is a seasonal variation in the rate of leptospirosis in man. Moderate or warm climates and the warmer seasons of the year seem to be associated with a higher incidence of the disease. In very hot or very cold climates there are fewer cases. Rainfall and wet seasons are favourable to the spread of infection. Occupational activity may be one of the factors explaining such seasonal variations. Other factors could be related to the activity of carriers or to variations in
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Figure 26. Mode of transmission of leptospirosis to man

- Cattle
- Swine
- Dogs
- Rodents
- Contaminated water or mud
leptospiroae excretion in urine from season to season. In areas
where the disease is endemic, there are frequent outbreaks from
one year to another, depending on environmental and host factors.

Disease manifestations in man. The incubation period is 4 to
19 days, with an average of seven to nine days. The severity of the
disease in man may vary tremendously from minor involvement to
severe hepatorenal failure and death. Severe cases have been
associated primarily with *L. icterohaemorrhagiae*. Although the
virulence varies with different strains of each serotype, other
serotypes usually produce less severe symptoms in man.

In acute cases of leptospirosis, sudden fever is a characteristic
initial symptom. It is associated with headache, stiff neck, muscular
pains, ocular manifestations of redness and blurring of vision,
nausea and vomiting. The fever lasts for seven to nine days and is
usually biphasic, with icterus, prostration and meningeal irritation
appearing gradually. Purpuric haemorrhages into the skin occur
in many cases, indicating severe toxaemia. Epistaxis, haemoptysis
and haematemesis may occur.

Other clinical signs may be hepatosplenomegaly, oliguria,
albuminuria and the passage of renal casts. Blood urea is elevated.
The jaundice deepens as the disease progresses into the second
stage, when a drop in body temperature is usually seen. During
the convalescent or third stage, the signs of the disease abate
gradually, with improved renal function and diuresis, and the
jaundice begins to fade.

In some cases of leptospirosis, meningitis is the most prominent
feature. Pure meningeal forms occur mildly, and are rarely fatal.
Neurological involvement is indicated by severe headache, muscu-
lar weakness and changes in reflexes. The cell count of the
cerebrospinal fluid (CSF) increases, with polymorphonuclear cells
or lymphocytes predominating. The CSF protein level may also
increase.

The disease must be differentiated from other conditions causing
infectious hepatitis, meningitis or primary nephritis. The occupa-
tional history, as well as the circumstances of exposure (if they can

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be ascertained), may be of help in this respect. The clinical manifestations and history, together with laboratory assistance, will serve to identify the disease.

Temporary disability from leptospirosis usually lasts for two weeks to two months. In rare conditions, the time lost owing to disability from leptospirosis may amount to four or six months. No information is available on the extent of claims due to such disability among occupational groups in different countries. The disease usually ends with the recovery of liver and kidney function. In rare cases, permanent ocular disturbances may ensue. Some patients may suffer from headache in bouts of migraine after recovery.

Laboratory diagnosis. Attempts to isolate leptospires from the blood may be successful during the febrile stage of the disease, and isolation from the urine may be possible in later stages. Direct culture or animal inoculation may be used. However, it usually takes two to four weeks to determine whether cultures are positive. Serological procedures are most commonly used. Paired serum samples should be tested. At present the standard test is still considered to be the microscopic agglutination "lysis" test. However, the macroscopic plate agglutination test is also considered an effective screening test.

Prevention

In many countries, preventive measures for the control of leptospirosis are enforced by legislation. These measures are concerned with host carriers (such as rodents and other animals), the control of environmental contamination and prophylaxis in man.

Measures against host carriers. In areas where rats are important reservoirs of leptospires, systematic control measures against rats and mice should be undertaken. Trapping appears to be inadequate; poisoning may be effective if done properly. Warehouses for grain storage in villages could be fumigated. Such stores should be made rat-proof from the outset by covering all
holes and crevices and ensuring that ventilation grids, pipes and other inlets are suitably designed and externally sealed. Concrete or metal containers are preferable for grain storage in terms of rodent control.

The carrier state in cattle and other domestic animals may be controlled by serological testing and/or culture and by subsequent treatment with dihydrostreptomycin.

The vaccination of animals has been effective in preventing the clinical signs of leptospirosis, but does not necessarily prevent renal shedding of the organism. The vaccine should incorporate serotypes in the bacteria that are important in the specific region. At present, twice-yearly vaccination seems to be the most effective.

*Environmental measures.* The disinfection of large water surfaces, such as rice-fields, is impractical. In many areas, environmental measures to improve rodent control are the most effective. Potentially infected animals should be denied access to water intended for public use.

*Prophylaxis in man.* It is important that personal protective equipment, such as boots and gloves, be provided for men working in the fields. Personal hygiene by early identification and proper treatment of cuts and abrasions, as well as protection from contaminated water and soil, are other important prophylactic measures.

A safe and effective vaccine for the immunisation of human beings has been sought for a long time. The field trials of an experimental vaccine have been encouraging. However, at present no vaccine is in regular use for prophylaxis in man.

Early diagnosis and treatment are important if the severe consequences of the disease are to be avoided. When discovered in its early stages, leptospirosis can be treated with purified antiserum of high potency. The effectiveness of such therapy has been established. Other methods of treatment using antibiotics could also be tried. In addition to the treatment of renal failure by fluids and diet, electrolytic balances should be adjusted and symptomatic treatment should be given.
5.3.4. Tetanus

Tetanus is a serious disease of man that has been known since early recorded times. The causative organism, *Clostridium tetani*, is a bacillus that produces a soluble, lethal, neural toxin affecting the peripheral and central nervous systems. The disease occurs throughout the world, particularly in tropical and warm climates.

In developing countries, especially those with large agricultural communities, the onset of tetanus after injury represents an important public health problem. Approximately 1,000 persons suffering from tetanus are admitted to the hospitals of Sri Lanka each year. In some areas of India, health surveys in rural areas have shown that tetanus is one of the ten major causes of morbidity and mortality.

Tetanus is a compensable occupational disease among farm workers in many countries in Europe and South America and in the USSR.

**Epidemiology**

*Causative organism.* *Cl. tetani* is an anaerobic spore-forming bacterium. The vegetative form is quite sensitive to environmental stresses. However, tetanus spores are highly resistant. They can withstand boiling in water for one hour and resist the action of a 5 per cent solution of carbolic acid for five to ten hours.

The tetanus bacillus is a normal and harmless inhabitant of the intestinal tract of herbivorous animals. The excreta of horses and cows are the major source of soil contamination with *Cl. tetani*, particularly when wastes from these species are used as fertiliser. When excreted with faecal material, the organism does not multiply in the soil but forms spores and spreads to vegetables, street dust, clothes and land. Most soils, even in forests, have been found to contain tetanus spores. These survive for many years and may be carried to other areas by the wind. People continuously exposed to contaminated sources, such as manure, may become carriers of the organism and may therefore excrete tetanus bacilli in their stools. When ingested by animals or man with vegetables from the
fields, the spores are not affected by gastric secretions; favourable conditions exist in the intestinal tract for their growth and multiplication.

*Transmission to man.* The disease in man is caused by the contamination of a wound with soil containing tetanus spores. An anaerobic situation must be established for the organism to multiply and produce the toxin. Deep puncture wounds are most likely to establish anaerobiasis. However, the toxin can develop in most types of breaks in the epithelium when this is contaminated.

In the anaerobic condition of tissues, the organism grows and multiplies. It remains localised without spreading to other parts of the body. The toxins produced are disseminated from the site of infection to the target tissue by a mechanism that not is fully understood at present.

*Clinical features.* The symptoms appear in susceptible individuals after an incubation period of one to two weeks, depending on the nature and degree of contamination of the wound.

With a heavily contaminated wound with ideal growth conditions the incubation period is usually short. The disease is characterised by fever, sweating, insomnia and muscular spasms. At first tonic and clonic muscular contractions occur near the site of infection. The neck and jaw muscles are then involved, with painful acute spasms of prolonged duration. Other muscles may also become involved, and death may result from spasm of the glottis with syncope, cardiac paralysis and continuous diaphragmatic contraction. The disease may last from a few hours to some weeks, again depending on the nature of the wound and the degree of contamination.

*Epidemiology.* A fair number of cases of tetanus in mankind occur as a result of navel infection in new-born babies, the result of imperfect sanitation during delivery. There seems to be a higher prevalence of tetanus among the Negroid and Hindu races. As more cases occur in men than in women, as a seasonal variation exists in that most cases occur during the summer months and as
most cases occur in rural areas, the occupational nature of the disease is confirmed.

*Occupational aspects.* Workers engaged in agriculture run an increased risk of contracting tetanus: such workers are particularly susceptible to cuts and abrasions, and owing to the nature of the work environment it is likely that wounds will become contaminated with soil containing tetanus spores.

*Prevention*

Effective prevention of tetanus is achieved by active immunisation, using tetanus toxoid. The massive immunisation of occupational groups at risk is still necessary. In view of the substantial risk of contracting tetanus following an injury and in view of the fatal consequences of the disease, such mass action is fully justified. In many parts of the world, most children are given the tetanus toxoid after the third month of life, usually in combination with other antigens.

As regards occupational groups at risk, immunisation is usually carried out as part of the health programme for workers. In the event of injury, antitoxin may be used for prophylaxis if the immunisation history is not available. Active immunity is preferable to passive immunity, because there are fewer side reactions and because better protection is afforded.

*Handling injuries and treatment*

When an injury occurs during work in the fields or where contamination is possible, special care should be taken to cleanse the wound thoroughly and to remove all dirt. Punctures should be freely opened, necrotic tissues should be removed surgically and drainage should be established. Antiseptics (such as hydrogen peroxide) should be applied to the wound. Antitoxin or toxoid should be administered, according to the particular situation. The use of antibiotics may be considered in some cases.
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5.3.5. *Tuberculosis as an occupational health problem in agriculture*

Most cases of human tuberculosis caused by the bovine strain have been due to the ingestion of unpasteurised milk. However, a large number of cases have occurred as a result of occupational exposure.

In many countries, control programmes have greatly reduced tuberculosis in the bovine population. Australia, Canada, Denmark, Finland, the Netherlands, New Zealand, Norway, Sweden, Switzerland, the United Kingdom and the United States are among the countries that rarely report cases of tuberculosis of bovine origin. However, bovine tuberculosis is still a major public health and occupational problem in Africa, Asia and South America. *Mycobacterium bovis* accounts for 10 to 25 per cent of all human tuberculosis in these areas.

*Epidemiological and occupational aspects*

*Causative organism.* Mycobacteria have unusual resistance to antiseptics and chemotherapeutic agents. Tubercle bacilli usually survive exposure to acidic or alkaline media for prolonged periods. This characteristic helps in isolating the organism from pathological materials. In sputum or excreta, tubercle bacilli can survive for long periods in a dry state.

Four types of tubercle bacilli have been described: the bovine, human, avian and atypical varieties. The bovine and human types may affect a wide range of animals and man. Domestic animals such as cattle, swine, goats, horses and dogs are mostly affected by the bovine type but may also be affected by the human type. Avian tubercle bacilli mainly affect domestic fowl, parrots, swine, goats and rabbits; they affect cattle to a lesser extent.

The human and bovine types are the principal agents of tuberculosis in man. Bovine tubercle bacilli may cause lesions in man that are equally as serious as those due to the human type. Infection in man usually involves the intestines, cervical lymph nodes, bones and joints. Pulmonary tuberculosis of bovine origin also occurs; it is as serious as infections involving the human strain.

*Bovine tuberculosis.* In many countries, a high percentage of the
cattle is infected with the bovine type of tubercle bacilli. This infection mainly affects dairy cattle, causing considerable economic losses and danger to public health. The higher incidence of tuberculosis in dairy cattle may be accounted for by the particular demands placed on them by milk production, which lower their resistance, and by the fact that they usually live in much more crowded accommodation than beef cattle. The greater age to which dairy cows live may also be a contributing factor.

Most tuberculosis in cattle occurs through the inhalation of infested dusts or aerosols. Infection by ingestion is relatively uncommon; much larger doses are required to cause infection by that route than by inhalation. The primary lesion in cattle is usually an alveolar or bronchio-alveolar focus with a corresponding caseous lesion in the regional lymph nodes. Cattle may become infected with the human strain of *M. tuberculosis*, but usually this does not present as severe a problem as does the bovine strain, at least on a herd basis. Man may become infected with the human strain from cattle. The avian strain rarely occurs in cattle, but more frequently affects swine.

Bovine tuberculosis also occurs in many other species of animals—for example, dogs, cats, pigs and horses. Monkeys can contract the disease and may transmit it to cattle and to humans. Pigs and horses usually play a minor role in transmitting bovine tuberculosis to man.

Man may acquire bovine tuberculosis by drinking contaminated milk or eating improperly cooked contaminated meat, by inhaling contaminated air in farm buildings or by direct contact with infected animals or their tissues.

The manifestations of bovine tuberculosis in man are generally dependent on the mode of transmission of the disease. If the source is contaminated milk, they may be gastro-intestinal with mesenteric lymph gland involvement. Lung intake leads to typical pulmonary tuberculosis with considerable destruction of pulmonary tissue, caseation and cavity formation. Direct contact with infected animals or their tissues or their excreta will result in localised surface wounds on the hands or arms.
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It will be clear from this brief survey that farmers and cattle-breeders are in high-risk occupational groups for exposure to tuberculosis during work with infected cattle. Other workers, such as butchers, meat inspectors and veterinarians, as well as persons handling cultures of bovine tuberculosis in the laboratories, are also at risk.

*Avian tuberculosis.* Most species of domestic birds, as well as wild birds, are susceptible to avian tuberculosis. The disease is more common in poultry than in other birds and occurs to a greater or lesser extent in all parts of the world.

Lesions in birds are those associated with generalised involvement of the visceral organs, following ingestion: some may result in pulmonary or cutaneous involvement.

The transmission of avian tuberculosis to man is rare, but may result from continuous contact with infected poultry or birds. Infection may penetrate through scratches in the skin, usually from beaks or claws of birds; or through the alimentary tract, from eating eggs laid by infected hens. Pulmonary tuberculosis of avian origin in man is not reported.

Tubercular lesions in man resulting from contact with poultry may take the form of cutaneous ulcers or subcutaneous nodules. Poultry farmers and breeders are susceptible to such conditions.

*Diagnosis.* The diagnosis of tuberculosis in man depends on the detection of tubercle bacilli. Culture, acid-fast strain and animal inoculation may be used to identify the organism in sputum or infected tissue. Recourse may be had to tuberculin skin tests in many early and occult cases. Radiographs may be useful in many cases.

*Prevention*

*Control in animals.* The eradication of bovine tuberculosis in the cattle population is essential to the prevention of the disease in man. The detection of infected cattle by tuberculin testing and the subsequent removal of positive reactors has proved very effective in the eradication programmes of several countries. Physical exam-
infection of cattle, without the intradermal test, will reveal only a small percentage of infected animals.

The tuberculin reactors are removed by slaughtering and the infected parts are disposed of. The animals’ quarters are then disinfected and the herd is retested at a later date, until all reactors have been removed and the herd is free of infected animals. Different types of tuberculin are used in different countries. The purified protein derivative (PPD) is the preferred tuberculin because of its purity and ease of standardisation. In the production of tuberculin, strains of the human type of the tubercule bacillus are widely used. In some countries, the bovine type is preferred as being more specific. There is no conclusive evidence on the validity of this assumption.

*Milk and meat hygiene.* The proper pasteurisation of milk has eliminated the primary source of bovine tuberculosis in the human population in several countries. Although cases transmitted by the ingestion of contaminated milk are not occupationally related, pasteurisation can be very effective in controlling a significant part of the public health hazard of bovine tuberculosis, as can meat inspection programmes. In these programmes, infected cattle or tissues are condemned, and contaminated meat is thus prevented from reaching the public. The proper identification of carcasses at the abattoir allows infected animals to be traced back to their herds of origin. Test and slaughter methods performed on the herds of origin help to reduce the total amount of bovine tuberculos.

Vaccination with attenuated bovine strains, *Bacillus Calmette-Guérin* (BCG), confers some protection. There is strong evidence that its application in humans has reduced tuberculosis infection to a marked degree where the number of tuberculosis individuals is high.

The vaccination of cattle with BCG has proved to be of no practical value. It increases the sensitivity to tuberculin, and the intradermal test thus becomes worthless as a diagnostic aid.
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5.3.6. Tularaemia

This disease occurs in various wild mammals (primarily rodents and lagomorphs) in North America, Asia, Europe, Japan and the Scandinavian countries. It also affects domestic sheep. In man, the disease is transmitted by several routes of infection: handling of rabbit carcasses, direct contact with infected animals, tick bites, inhalation of infected dust, contamination of drinking water by diseased animals, and ingestion of improperly cooked meat of infected animals.

Epidemiology and occupational characteristics

Causative organism. The disease is caused by the bacterial agent Francisella tularensis. The organism has been isolated from a large number of animals, mainly wild rabbits, hares and their ticks, squirrels, rats, mice, sheep and wood ticks. The transmission of the disease among animals occurs through breaks in the skin, or through blood-sucking insects such as ticks, lice, flies and fleas. Domestic animals are more resistant than wild breeds. The highest susceptibility is noted in rabbits, guinea pigs, beavers and ferrets. Man is also highly susceptible.

Occurrence in man. Tularaemia is an occupational hazard for hunters, butchers, cooks, agricultural workers, lumbermen, forestry workers, sheep shearsers, fur farmers and laboratory personnel.

Wood ticks such as Dermacentor variabilis and D. andersoni harbour the organism in the coelomic tract and epithelial cells of the gut wall. The organism is excreted in the faeces. The horse-fly Chrysops discalis (see figure 27) also harbours the organism.

There are no differences in susceptibility in man owing to sex, age or race. The disease may occur in the spring and summer months or in winter.

Clinical picture in man. Four forms of the disease are recognised in man:
(1) The ulceroglandular type produces a skin papule leading to ulcers and enlarged regional lymph nodes. It may be associated with pulmonary and/or meningeal involvements.

(2) The oculoglandular type mainly affects the conjunctiva of the eye in association with enlarged regional lymph nodes.

(3) The glandular type is associated with enlarged nodes at the site of infection.

(4) The typhoidal type is manifested by a generalised febrile illness and pneumonia. This type is contracted by inhalation and has a high mortality rate.

The incubation period is one to ten days, with an average of three to four days. The onset of the disease is sudden, with headache, chills, body pains, vomiting and fever. The primary lesion,
as previously described, is usually in the skin or the eye, with enlarged lymph nodes. The associated fever lasts for two to three weeks, with periods of remission and recurrence as in septic fever. Mortality rates are usually fairly low, except for the typhoidal type.

*Diagnosis.* Diagnosis depends on the history and the clinical examination. Isolation of an organism may be attempted from nasal and bronchial secretions or from exudate of local wounds; specific blood antibodies may be detected in the second week of infection and remain present for several years. Skin tests may also be used as a diagnostic aid.

*Treatment.* The disease is amenable to treatment by sulphadiazine, tetracycline and chloramphenicol, streptomycin and the synthetic penicillins. The last two antibiotics seem to be the most effective. In many, the disease usually confers permanent immunity.

*Prevention*

Prevention must rely on wide-ranging sanitary measures, the use of personal protective equipment, immunisation, health education and rodent control.

Health education helps to develop alertness and awareness among the potentially exposed population. Protective gloves should be used in handling and cleaning wild rabbits. Protective masks may be used to prevent dust inhalation in animal houses. Water supplies which may be contaminated by wildlife should be sterilised before drinking. Cuts, insect bites and animal bites should be thoroughly cleaned and disinfected. Rabbit meat should be thoroughly cooked. An effective method of meat inspection is necessary. Rodent control and the building of rat-proof stores in rural areas are also helpful.

Bacterins confer minimal protection, but may modify the course of the disease in man. A living activated vaccine has been used among exposed populations in the USSR with success. In view of its encouraging results in man, this living vaccine strain may prove to confer protection.
5.3.7. Glanders

Glanders mainly affects horses, mules and donkeys, and is only occasionally transmitted to other animals and man. The disease is characterised by modular lesions in the internal organs, lungs, skin and the mucous membrane of the respiratory tract. In man, it is a serious disease and is very difficult to manage clinically. It has been suggested that man may have some natural resistance to glanders which accounts for the low human incidence.

Epidemiology

The disease is caused by the bacterial agent Malleomyces mallei. The organism is not resistant to heat or light outside the body, nor to germicidal action.

Incidental hosts, such as dogs, cats and camels, may rarely be involved. In animals, the disease is transmitted through direct contact with infected animals or through food, air or water contaminated by secretions and discharges from the lesions of infected animals. Skin abrasions and the mucous membranes also serve as portals of entry for the infection. In man, abrasions or wounds may become contaminated during the handling of infected animals. Transmission through the respiratory tract and mouth is less frequent. Man-to-man transmission occurs only very rarely. Mortality was substantial in the past, when the incidence of the disease was higher than at present.

Agricultural workers are most frequently affected by the disease. Farmers, stablemen, knackers, blacksmiths and veterinarians are potentially exposed.

Clinical picture. The disease may appear in one of three forms: acute, subacute and chronic. The lesions may be limited to the skin; this form of the disease is called farcy. It may be systemic with pneumonia.

Farcy is first characterised by redness of the skin, the swelling of regional lymph glands and a pustular eruption. The acute form is associated with fever, vomiting, diarrhoea, rapid pulse, dry
tongue, delirium and coma. Nodules appear along the lymphatic system, leading later to a pustular formation which ruptures and forms ulcers (farcy buds). The joints nearby are affected. Septicaemia, involvement of the lungs and orchitis may also develop.

The chronic form of the disease is less serious, but nevertheless extremely painful, with frequent exacerbations. The disease is associated with subcutaneous abscesses, ulcers and sinuses. Deep ulcers in the nose may also be observed.

*Diagnosis in man.* The diagnosis of the disease in man depends on the history of contact with horses or other equine species. The organism may be isolated and demonstrated from the lesions. Intraperitoneal injection in guinea pigs leads to inflammation of the testes and scrotal sac. This test, called the Strauss test, is characteristic for the disease. A complement fixation test should be performed and is helpful in diagnosis.

*Prevention*

Progress in controlling the disease in equines has led to its near disappearance in man; however, the disease is still endemic in certain areas of the world. Control in equines depends on the early detection of affected animals. This may be done by clinical examination, bacteriological studies, serological tests and mallein reaction. Animals with glanders must be slaughtered and their carcass disposed of. The infected premises and equipment must be thoroughly disinfected. New animals introduced into the group should at once be put under observation and tested by mallein; the test should be repeated annually. Common feeding and drinking troughs should be abolished. No vaccines for animals are available, and the treatment of cases is of doubtful value.

Great care should be taken in the laboratory when working with the organism. Human cases should be isolated, as the disease may be transmitted from one person to another.

*Treatment*

The treatment is mainly symptomatic: nursing, surgical management of abscesses and the application of chemotherapy and
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antibiotics. There is no specific antibiotic against the disease, but sensitivity tests may indicate which is the most effective. Sulphadiazine in large doses over long periods is helpful. Mallein endotoxin has been tried in some cases with promising results.

5.3.8. Melioidosis

In many respects, this disease is similar to glanders. It is primarily a disease of animals, both domestic and wild, and is seen occasionally in man. The disease is characterised by the formation of nodules in the spleen, lungs, liver and lymph nodes, which may suppurate. Lesions of the nasal mucosae or localised infections in the meninges, joints and bones are also described.

The disease was first recognised in Burma and Malaysia. It mainly occurs in south-east Asia (India, Indonesia, Democratic Kampuchea, Singapore, Sri Lanka, Thailand, Viet Nam) and in Australia, Chad and the Philippines. There are also reports from Canada, France, South Africa, the United Kingdom, the United States and Venezuela. Although the incidence is low, cases of melioidosis in man have been on the increase since the 1960s.

**Epidemiology**

*Causative organism*. The disease is caused by the organism *Bacillus pseudomallei* or *Malleomyces pseudomallei*. This bacterial agent has been isolated from soil, mud, rice-fields and stagnant ponds. It has relatively high resistance to environmental stresses, and remains viable for eight weeks in tap water and for up to 27 days in urine and faeces. The organism is readily killed by heat and common disinfectants other than phenol and lysol. It shows a certain resistance to drying.

The primary animal hosts seem to be sheep, goats and pigs. Cats, dogs, horses and cattle are more rarely affected. Rats are thought to be an important reservoir.

*Mode of transmission*. The mode of transmission from animals to man is not known with certainty. There is no evidence of a
primary animal reservoir of the disease for man. In tropical climates, the organism remains viable for several weeks in water contaminated by infected animals. Water may be a primary source of infection for man. Water used for irrigation could be responsible for the infection of agricultural workers through direct contact with the water or through ingestion. Lacerations or cuts could predispose to infection in contact with water.

The role played by insects or rodents in natural transmission is not clear. When experimentally infected, some insects, such as the red flea and the mosquito (Aëdes aegypti), can transmit the disease to laboratory animals. Man-to-man transmission is not reported, although the organism can be isolated from human excretions. The fact that most cases reported are in adult males suggests an occupational origin.

Clinical picture. The disease in its acute form may resemble severe generalised diseases such as cholera, typhoid and glanders. Mortality may be high in the acute form. It is generally a serious disease in man. Some cases, however, run a mild course, and may frequently occur even in subclinical form, which may pass undetected.

The septicaemic form is characterised by the occurrence of abscesses, pustules and sinuses. Consolidation and abscesses in the lungs, the liver, the spleen and sometimes the kidneys are an important feature. The disease lasts for three to four weeks, leading either to death or to convalescence. There is a chronic form of the disease which is characterised by general debility and the occurrence of multiple abscesses in internal organs, as well as pustules and vesicles on the skin that resemble variola.

Diagnosis. Diagnosis depends on the application of the Strauss test reaction using meliodin, which is similar to mallein. This is a heat-stable product and can be used in male guinea pigs to indicate infection.

The effective treatment of the disease depends on the use of sulphadiazine. Cauterisation is also applied to superficial lesions.
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Prevention

Prevention depends on general sanitation and on its detection in animals by serological tests using melioidin, followed by the elimination of positive reactors and disinfection.

Potentially exposed populations should avoid stagnant water and use disinfectants. No vaccine is available to protect man against the disease. Prophylaxis with sulphadiazine and antibiotics such as chloramphenicol can be tried.

5.3.9. Erysipeloid

Erysipeloid is principally an occupational disease in man caused by the organism Erysipelothrix rhusiopathiae. This organism causes economically significant disease in swine and turkeys. It is also associated with fish. Man may contract the infection during the handling of these species. The disease occurs throughout the world, and is a public health problem.

Epidemiology

Causative organism. E. rhusiopathiae is a highly resistant bacterial organism. It is resistant to relatively high concentrations of phenol, drying, pickling and salting, but it is susceptible to moist heat. The organism may survive in soil for long periods.

The disease in swine. The disease in swine is called swine erysipelas or swine fever. It is transmitted in pigs by the oral route. It occurs usually in one of three forms. The first of these is the acute form with a sudden onset, high temperature, rapid course and high fatality. There is a generalised septicaemia which affects all the organs of the body, with marked damage. The second form is the chronic disease, which is characterised by vegetative endocarditis of the heart valves with associated changes in other organs. The third form is the arthritic form in aged pigs; this affects most of the joints of the animal.

There is also an urticarial condition in swine erysipelas, which may occur either in association with the above internal conditions
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or independently. It involves skin damage and probably gangrenous sloughing.

*The disease in birds. E. rhusiopathiae* is pathogenic to turkeys, hens, geese, ducks and pigeons. Turkeys are the most common avian species affected. It gives rise to acute septicaemia affecting all organs.

*Transmission to man and clinical picture.* Apart from the active clinical disease, there is also a carrier state in pigs. Swine can harbour organisms in the alimentary tract and tonsils and contaminate the surroundings from oral and faecal emanations. The handling of meat from animals that have died from the septicaemic form of the disease may be a cause of infection in man.

Fish may also harbour pathogenic organisms in the slime coating on their skin, leading to the infection of scratches on the hands during handling of fish.

Erysipeloid in man is characterised by a painful and itchy swelling at the site of infection in the skin. The area is reddish-purple with associated necroses. The regional lymph nodes may become enlarged. Arthritis may develop in adjacent joints.

The disease in man very occasionally takes a generalised form with cutaneous involvement. Septicaemia is rare.

The mild, localised cutaneous form occurs mainly among workers handling swine, fish and poultry. It is sometimes known as fish-handlers' disease or pork finger, or by other common terms used in the meat- and fish-processing industry.

Workers on farms using the manure of infected animals, or peelers of root vegetables, are also susceptible. Workers in other occupations, such as veterinarians, meat handlers and bone-button workers, are similarly at risk.

*Prevention*

The prevention of the disease depends on its control in swine and other affected animals or fowl. The vaccination of swine, using living cultures and serum together, has been found to produce lasting and reliable immunity.
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Immune serum may be used in the treatment of cases in pigs and also in prophylaxis. Passive immunity resulting from serum applications lasts some weeks and may save potentially exposed animals. Prevention in man also depends on the methods used for handling meat, fish and infected animals or their excreta. Health education is important, as is personal protection through the wearing of boots or gloves. Care should be taken to prevent the contamination of breaks in the skin when fish and pork products are handled.

The treatment of the disease in man relies on the use of antibiotics. Penicillin and tetracyclin or chloramphenicol usually effect a cure.

5.4. Parasitic diseases

5.4.1. Ancylostomiasis

Ancylostomiasis, commonly called hookworm disease, is caused by a nematode worm which is cosmopolitan in distribution. The disease is endemic mainly in the warmer regions of the world. If untreated, it may lead to severe anaemia and debilitation. Agricultural workers and others living in rural areas are potentially exposed.

Hookworms are fairly host-specific, as are most intestinal parasites, in that each animal species has its own species of parasites. Ancylostoma duodenale (see figure 28) and Necator americanus are predominantly human parasites. However, the skin of man may also be infected with juvenile forms of hookworm of cats, dogs, cattle and sheep.

Epidemiology and occupational aspects

The parasite. The adult human parasites live in the duodenum of man, sucking blood from capillary vessels which are lacerated by the teeth or cutting plates (see figure 29). After copulation between males and females, eggs are produced in vast numbers and large quantities escape in human excreta. In the proper surround-
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Figure 28.
_Ancylostoma duodenale_.
Adult worms:
*left*, male; *right*, female.

ings (such as warm, moist and loosely packed soils, for instance on the sides of canals or in the shade of trees on plantations), the ovum (see figure 30) develops rapidly into an embryo and larva. Two days later, the larva assumes a filariform appearance. Infestation in man occurs following the invasion of the skin (see figure 31) by filariform juveniles, which are attracted by the heat of the body to the surface of the bare skin (usually the foot). From there, they
make their way into the bloodstream and lungs. They penetrate into the bronchi, where they are coughed up; they are then swallowed and parasitise the small intestine. The infection of man with the filariforms of animal species of hookworm occurs by direct skin contact or through contact with soil that has been contaminated with faeces from infected animals. However, the infection is limited to the skin, and adult forms never appear in the gut of man.

**Occupational aspects.** Ancylostomiasis is a rural disease, associated with agricultural work. Warm, moist soil, shade and high humidity are favourable to its propagation. Those mainly affected are warm field workers. In areas where the custom and practice allow direct contact of the skin with soils fertilised with human or animal excreta, persons are potentially exposed to the risk. Occupational exposure in such circumstances is not easy to control, for it depends on the habits and customs of the rural population and the feasibility of disposal of human excreta. The methods and tools
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Figure 30. *Ancylostoma duodenale*. Ovum.

Figure 31. Skin invasion by *Ancylostoma duodenale*.
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(whether manual or mechanical) used in cultivation, as well as the social and economic conditions of the farm workers, are other important factors.

Other non-occupational groups may also become infected through contaminated soil. Children in villages playing in the fields or farmlands and women helping their husbands or passing through infested areas may also contract the disease.

Other occupational groups, such as miners, may acquire the disease through working in locations with contaminated soils.

In some heavily infested areas, the disease may affect 90 per cent of the population. Other areas may have a much lower incidence. In the case of bare-footed workers, work with the legs and feet in mud leads to the transmission of infestation and the penetration of larvae through the skin.

Clinical picture. Ancylostomiasis is a chronic debilitating disease with a number of symptoms which vary according to the extent of infection, the nutritional status and the general health status of the infected individual.

When the individual is also suffering from malnutrition or another disease, the blood-sucking activity of the parasite leads to hypochromic microcytic anaemia, and this may decrease the capacity for work and cause disability.

The period of invasion, when the larvae are penetrating the skin, may be associated with dermatitis and vesicular eruptions at the site of invasion, such as the foot. In the chronic stage of the disease, with intestinal infestation, the symptoms may include abdominal discomfort and digestive disturbances in addition to pallor, palpitations and dyspnoea resulting from anaemia. Infection with filariforms of animal hookworms (cutaneous larval migrans) results in an intense inflammatory dermatitis with pruritis, and eruptions which correspond to the migrating larvae. Because man is an incompatible host for these parasites, they are unable to complete their life-cycle and the migrating larvae are usually walled off in the subcutis.

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Infection is confirmed by the discovery of hookworm eggs in the faeces. Species recognition is by microscopic examination of adult worms.

*Treatment.* Ideally, treatment should be with non-toxic drugs without side effects, bearing in mind the anaemic and otherwise constitutionally debilitated condition of the patients. Tetrachlor-ethylene and bephenium hydroxynaphthoate could be used. Bephenium is preferred with *A. duodenale.* Combined treatment using both drugs is sometimes administered.

The treatment consists in taking the drug on an empty stomach or after the use of purgatives, with subsequent purgation for the evacuation of the worms. The diet must be supplemented with protein and iron, particularly in advanced cases. Cutaneous larval migrans is treated by oral thiabendazole, or by stopping the cutaneous migration of parasites with trichlorethylene.

*Prevention.* The prevention of ancylostomiasis in areas where it is endemic is fraught with difficulty on account of the prevailing environmental, social and economic conditions. Basic requirements for control include the following action:

1. An epidemiological investigation should be made of the areas concerned, in order to define the degree of infestation of individuals and the prevalence of the disease in relation to occupational, social and age factors, environmental factors and available sanitary facilities. The presence in the population of other parasitic infestations, infectious diseases and malnutrition should also be explored.

2. A plan of disease control, suited to the circumstances obtaining and the facilities available, should be drawn up. The following measures are recommended:

   (a) environmental sanitation. The possibility of establishing sewage systems should be studied. In areas where this is not possible, alternative methods of providing suitable latrines in villages and farms should be tried;
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(b) improvement of housing and of roads (by asphalting or other means of paving). This measure will help to reduce the possibilities of transmission;

(c) mass treatment and individual protection. Action to detect cases in the population should be followed by the treatment of all affected persons, with re-examination to ensure its effectiveness. Treatment helps to interrupt the life-cycle of the parasite by the prevention of egg shedding. Individual protection should be provided by the supply of leather or rubber boots to agricultural workers. This is an effective protective measure, but unfortunately it is sometimes impracticable; and

(d) health education and community development. All measures taken to prevent parasitic diseases should be directly associated with health education activities at all levels. Children at school should be instructed in the subject, in the risks run and in the means of avoiding infection. Farm workers should be trained how to avoid contamination of the soil by the parasite.

5.4.2. Schistosomiasis

Schistosomiasis, also known as bilharziasis, is one of the major public and occupational health problems in many tropical and subtropical regions of the world. It is found particularly in rural areas, where it affects large sectors of the population. The number of infected individuals has been estimated at 200 million. The high incidence of the disease and the reduction in physical efficiency and earning capacity resulting from its complications are grounds for particular concern.

The introduction of new and extensive irrigation schemes helps to spread the infection to new areas and to increase its incidence. The migration of labour may also be a factor in spreading the infection. Overcrowding and urbanisation, when compounded with insanitary water supplies and inadequate sewage disposal, are other factors in the maintenance of the disease.
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Figure 32. Ova of *S. mansoni* (left) and *S. haematobium* (right)
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The disease in man is caused by three major species of schistosomes: *Schistosoma haematobium*, *S. mansoni* (see figure 32) and *S. japonicum*. *S. haematobium* occurs mainly in Africa and in south-west Asia. A limited focus is known in India, and a small European focus in southern Portugal now seems to have disappeared. *S. mansoni* may overlap *S. haematobium* infection in many places. It prevails mainly in Africa, south-west Asia, north and east South America and in some of the Caribbean islands. *S. japonicum* is less widespread, being found mainly in China, Japan and some parts of south-east Asia.

Animal-pathogenic schistosome species which may occasionally infect man include *S. bovis*, *S. mattheei*, *S. intercalatum*, *S. margrebowiei* and *S. rodhaini*.

Although it is difficult to estimate precisely the economic losses due to the disease in areas where it is endemic, some studies have shown that they can be substantial. They are due mainly to a loss in productivity of individuals and groups due to chronic conditions, the direct loss incurred by treatment of the disease, and the time lost through absenteeism.

Pathology and mode of infection

The following brief account of the life cycle of schistosomes will help in understanding the methods used to control them. The schistosoma worms are trematodes having an asexual life in mollusc snails (see figure 33) and a sexual phase in man or other mammals. The ova of the schistosomes are excreted with the urine or stools of affected persons. When they reach fresh water in streams, canals and ponds, the ova hatch and free-swimming larvae (miracidia) escape.

The miracidium (see figure 34) reaches the snail in the water, and in this host it becomes converted into primary and secondary sporocysts. The latter produce fork-tailed larvae called cercariae (see figure 35). Leaving the snail, the cercariae swim about until they reach their definitive host (man or some other mammal) and enter the skin. In the vertebrate host, they travel along the lymphatic
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Figure 33. The intermediate snail host of schistosomes

Figure 34. Miracidium
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Figure 35. Cercaria
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system or the bloodstream, reaching the right side of the heart to enter the pulmonary circulation and the lungs, and then migrate to branches of the hepatic portal veins. After the worms become adults (see figure 36), they pair and migrate to the mesenteric veins where egg-laying begins.

*S. mansoni* and *S. japonicum* deposit their ova mainly around the rectum and colon, whereas *S. haematobium* deposit theirs in the walls of the bladder. The ova of *S. mansoni* and *S. japonicum* are therefore found in the faeces and those of *S. haematobium* in the urine. If the ova find their way to a body of fresh water, the life cycle is repeated (see figure 37).

The common pathological complications of the disease include chronic urinary tract lesions (*S. haematobium*) and liver fibrosis (*S. mansoni* and *S. japonicum*).

Several studies have been made of the carcinogenic action of human schistosomiasis. Some authors have attributed an increase in bladder cancer to *S. haematobium*, while others report the occurrence of liver cancer. So far, no concrete evidence has been put forward to substantiate these claims.

Occupational exposure usually occurs through contact with infested water. The person's skin is penetrated by the cercariae whilst swimming, during irrigation, whilst working in rice paddies or whilst engaged in other agricultural activities involving contact with water. The disease is not necessarily occupational, as it may occur during non-occupational activities involving the use of water in infested areas. The disease is most prevalent among adults. However, its occurrence in the young and the aged, as well as in women, illustrates the importance of non-occupational exposures.

**Symptoms**

The first evidence of schistosome infection is very similar in all three species and results from the penetration of the skin by the cercariae. Some persons are particularly sensitive to cercariae and develop dermatitis or, in some cases, urticaria. These reactions disappear within a few days. The subsequent clinical picture, however, varies with the species:
(1) *S. haematobium*: the major sign is haematuria, which may be profuse or occult. There is localised bladder and urethral pain, cystitis (with pain in advanced conditions) and pyuria. Later in the course of the disease, oliguria and uraemia may occur. Generally the patient experiences lassitude, muscular pain, digestive upsets and pain in the back. However, a majority of adult males may not exhibit these general symptoms in areas where the disease is endemic.
(2) *S. mansoni*: these are mainly intestinal in origin. Bouts of diarrhoea with blood and mucus are observed. Between these episodes, vague discomfort may be observed. In advanced conditions, liver fibrosis with hepatosplenomegaly and ascites occur. Now that schistosomiasis is being treated in most countries where it is endemic, these signs are becoming less frequent.

(3) *S. japonicum*: as with *S. mansoni*, the symptoms are mainly intestinal. The infection may produce serious illness with marked damage to liver and spleen.

**Diagnosis**

The most practical method of diagnosis is the identification of schistosome ova in the stools or urine. With this method, how-
ever, the condition can be diagnosed only after egg production has started. If the eggs are evacuated frequently, no difficulty is encountered in their detection by microscopy. Concentration techniques may be required to identify ova. Certain quantitative techniques can be used to count schistosome ova in the urine or stools. The most commonly used techniques for internal schistosomiasis are the filtration staining method of Bell and the Kato thick-smear. In urine, the eggs can be detected by examining the deposit after centrifugation or sedimentation in a urinalysis glass.

Tests to indicate the percentage of viable eggs evacuated by schistosome-infected patients are useful in epidemiological surveys. When urine and stool samples are negative, a rectal biopsry may be necessary to detect the eggs. Intradermal skin tests and complement fixation are immunological tests that may aid in diagnosis in the absence of ova. These latter techniques are also good for epidemiologic surveys.

Treatment

Chemotherapy may be used to treat individual cases or populations that are at high risk. Antimony compounds are commonly used in the treatment of schistosomiasis. Sodium and potassium antimonyl tartrate are known to give excellent results when properly administered for a defined time. Other compounds such as potassium antimony dimercaptosuccinate (Astiban TWSb, Stibocaptate) have also been reported to give satisfactory results with all types of schistosomiasis. Intermuscular injections of stibophen (Fouadin) and antimony lithium thiomalate (Anthiomaline) may be given. The antimony drugs, although of proven efficiency for treatment, have certain toxic side-effects that should be borne in mind.

The toxicity of the schistosomal drugs has stimulated the development of other compounds which in time will probably replace the antimonials. A product for oral administration, lucanthone hydrochloride, has been utilised in several countries with good results for *S. haematobium*, but is of little use with *S. japonicum* infections. Another oral non-antimonial nitrothiazole derivative,
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Niridazole (Ambilhar), has proved to be effective against *S. haematobium* and *S. mansoni*. In a few cases, however, this drug produced severe side-effects. Other oral products are available, but all have certain undesirable side-effects.

An organic phosphorus insecticide (Dipterex) is under investigation for oral administration. It appears to be quite efficacious for the treatment of *S. haematobium*.

In general, it can be stated that, in early cases, the treatment of schistosomiasis is effective and the prognosis is good. In late complicated cases, with chronic urinary disease or liver fibrosis, the prognosis is not favourable.

**Control**

The interruption of the life cycle of the parasite is the immediate aim of a schistosomiasis control programme. The eradication of the disease must be viewed as a long-term objective.

The prevention of the spread of schistosomiasis to new areas is an important aspect of public health work in many developing countries. Migration, the concentration of inhabitants, the type of irrigation and the relative efficiency of sanitary systems in relation to urbanisation are all factors to be considered. In addition, the specific factors related to the ecology and the epidemiology of the disease, such as personal habits, intermediate hosts, availability of fresh water and types and degree of infection, should be thoroughly investigated before control is attempted.

So far, methods for the control of schistosomiasis have not been as successful as had been hoped. It is difficult to state which approach will be most effective in controlling the disease. Action in the field of environmental sanitation, health education, mass therapy of the human host and control of the snail intermediate host has proved to be more or less effective depending on local conditions. All these methods have their advantages and disadvantages. They can be used alone or in combination.

*Environmental sanitation*. Sanitation measures should be an integral part of a public health programme and should not be re-
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garded as specific anti-schistosomiasis measures. Such measures generally attempt to prevent the transmission of schistosomes to snails and from the snails to man. Therefore, all possible action should be taken to bring about the sanitary disposal of human excreta so as to prevent bodies of water from being contaminated. The provision and use of latrines are essential, but behaviour patterns take a long time to change in some parts of the world.

With regard to the use of water by man, there is no doubt that a supply of clear, uninfected or sterilised water for washing, bathing and household services will reduce the risk of exposure of infection.

*Health education.* The gradual over-all improvement in the social, economic and educational status of the population can be expected in time to lead to greater public awareness of the dangers of the disease and of the means of preventing it. Health education may be given in rural schools, factories and plantations and during public gatherings.

*Snail control.* Snail control is generally considered to be the most rapid and effective means of breaking the transmission chain in schistosomiasis. Several methods are used. Environmental control is intended to alter the ecology of the snail habitat to render it unfavourable to snails. This can be done by filling-in, drainage, increasing the velocity of water flow, straightening streams, removing vegetation, and so forth. The choice of measures will depend on local circumstances. Water management is particularly important in irrigation areas and where certain types of fish rearing and agriculture are practised.

Snails may also be controlled by biological means. Although there are several reports of the harmful effects on snails of predators, parasites and competitors, results in the field have, on the whole, not been encouraging.

The most effective way to control the snails is through the application of molluscsicides. Copper sulphate has been used for many years at a concentration of 40 ppm. The following compounds are at present being used in control operations:
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(1) Bayluscide (Niclosamide) is considered the molluscicide of choice. It has been used in the field at concentrations of 4–8 ppm, is highly toxic to all stages of snails and displays very low toxicity to mammals.

(2) Frescom (N. tritylmorpholine) is the most active molluscicide. It may be used in the field at a concentration of 4 ppm.

(3) Sodium pentachlorophenate (NaPCP) has been in use for some 15 years. It may be applied at a concentration of 10 ppm. A combination of this compound with copper sulphate has also been used.

Mass treatment. Treated cases must be re-examined to ensure that they are free of the organism. Prophylaxis may be maintained by keeping the patient on the antimony drug. If, as a result of mass treatment campaigns, all infected individuals could be stopped from excreting viable ova for a period long enough to permit the decontamination of water and to deprive snails of the miracidia, with reinfection being prevented at the same time by continued therapy, this would lead to a marked reduction in the incidence of schistosomiasis.

Prevention among occupational groups

Control programmes for factory and plantation workers are potentially very effective. Pre-employment medical examinations can be readily carried out. All infected individuals should be treated. Routine monitoring programmes should be established to ensure that no shedders exist. A proper sanitary water supply and effective means for the treatment of human waste should be provided for the workers, and educational programmes may be introduced for them. These measures, if carried out regularly, can be very effective in reducing infection within a specific workforce.

5.4.3. Leishmaniasis

There are three types of this disease: the mucocutaneous or American type (espundia, uts); the cutaneous type (oriental sore);
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and the visceral type (kala-azar, or black fever). The first type may be more frequently related to occupational exposure than the last two and is therefore considered here.

American leishmaniasis is a parasitic disease caused by infection with *Leishmania braziliensis*. It occurs among persons working in forests and on cacao and tea plantations in most countries of Central and South America, where the disease is endemic, as it is in Mexico. The parasites are transmitted to man by sandflies of the genus *Phlebotomus*. The fly carries the parasites from the infected lesions of man or from the animal hosts to the abraded skin of others.

The initial lesions occur mainly in the uncovered parts of the body, with ulceration and infection. Vesicles and pustules become confluent and closed, and are associated with lymphangitis and adenitis. Other forms of the disease may affect the nasal septum and the upper respiratory tract, including the pharynx, larynx and trachea. The palate displays proliferative forms of granulation which interfere with eating and respiration. Sometimes these forms are complicated by oedema of the glottis and respiratory tract.

Control of the disease relies on sanitation, the application of insecticides and the spraying of areas where flies may breed, such as rubbish heaps, damp areas and animal houses. Other means of insect control and repellants are desirable. Housing should be located away from infested areas.

5.4.4. *Contagious eczema* (orf)

This is a highly infectious viral disease of sheep and goats; man may contract it through infected animals. The causative agent resembles pox viruses in morphology and generally in the type of lesions produced. In sheep and goats, the disease affects the face, lips and buccal mucosa, as well as the teats and udders. It is first vesicular, then pustular and ulcerative with scab formation.

In man, the disease is transmitted by contact, particularly if there are abrasions or cuts in the hands. When sheep are handled, slight
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abrasions may occur from burrs present in the wool. Vesicles develop mainly on the hands, but other parts of the body which come into contact with infected animals may also be involved. Regional lymphadenopathy is often seen. The vesicular lesions are usually painless, and proceed to eschar formation when the vesicles rupture.

The disease is quite common in agricultural workers who work with sheep. Prevention is mainly through disinfection of abrasions and cuts (if present) after infected sheep have been handled, and through the use of protective gloves and clothing when infected animals have been handled or treated. Vaccine application is not appropriate as it produces an effect similar to that produced by the disease itself.

5.4.5. Milkers’ nodules

The disease is caused by the pseudocowpox, a virus similar to variola virus, and is limited to local infections of the udders and teats of dairy cows. It can be transmitted to man in the process of milking. The lesion takes the form of an erythematous ulceration and swelling around the site of infection. The disease may be epizootic within a dairy herd, and is transmitted between cows mainly by man through the process of milking.

The disease is transmitted to the cow milkers by direct contact with lesions on the teats and udders of infected cows. Lesions in man are thus almost always found in the hands and fingers; they are fairly typically vesicular, proceed to eschar formation and usually heal without a scar unless complicated by secondary bacterial infection. Regional lymphadenopathy may be seen. Generalised disease is rarely seen in man. A vaccine is available for the prevention of the disease in cattle. A high standard of sanitation and insect control will also help to prevent the disease in cattle. Isolating the infected cattle from the milking line, and milking them last of all, will certainly help to prevent the disease from spreading in the herd. The introduction of mechanical milking operations helps to prevent transmission of the disease to man. It is essential for a
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milk to wear gloves when treating or milking infected cattle, and for any worker coming into contact with infected animals to wash his hands afterwards. Prevention in man is brought about chiefly through education.

5.4.6. Psittacosis

This disease derived its name from its first recognised hosts, psittacine birds. Psittacosis has also been known as parrot fever, since many cases of the disease in man were contracted by owners of pet psittacine birds and by those who sold them in pet shops. More recently, the disease has been found in many other species of birds, such as poultry and pigeons. The disease is now also known as ornithosis, to reflect this broader host range.

The agricultural groups at risk for this disease are, first, workers in poultry-processing plants, and second, poultry breeders. Workers eviscerating the birds are at highest risk, and outbreaks in processing plants are not uncommon. Poultry breeders certainly are at risk when in contact with infected flocks. Clinically, the disease in man is characterised by fever with upper respiratory involvement. Although the disease may have a prolonged recovery time, most cases recover without complications, particularly since the advent of broad-spectrum antibiotics.

Control is by public education, by monitoring the sale of psittacine birds and by personal protection for poultry breeders and workers in poultry-processing plants. The reduction of the disease in poultry flocks will go a long way towards protecting agricultural workers from exposure. Flocks may be screened with a complement-fixation test to detect infected birds. The treatment of the flock with broad-spectrum antibiotics is quite effective.

5.5. Other infectious diseases affecting agricultural workers

By the very character of his work, the agricultural worker is often exposed to the infectious diseases that exist in nature. Many of these
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diseases may indeed be closely associated with the rural towns and villages in which he lives. Therefore, it is sometimes difficult to establish a direct occupational link for some of these diseases, since, although the agricultural worker is often susceptible to them, they may often be more directly related to the rural environment itself. Most of the diseases examined in this section fall into this category.

5.5.1. Rabies

This viral disease is transmitted to man mainly by the bite of a rabid animal, or occasionally through breaks in the skin contaminated by the saliva of infected animals. Rabies affects many feral and domestic species. It is found throughout the world, although the number of cases reported in man is not great. The disease leads to serious nervous system manifestations in man and usually ends fatally.

Agricultural workers are probably more exposed than others, since they have more opportunity for contact with affected animals. Prevention of the disease depends primarily on the vaccination of domestic dogs, public health education and pre-exposure immunisation for high-risk exposure groups. Post-exposure treatment includes immediate and thorough cleansing of the wound, adequate laboratory support for diagnosing rabies in the animal responsible, and proper post-exposure immunoprophylaxis. The control of stray dogs and cats, the identification of feral reservoirs and the control of the latter in certain situations is important in preventing human exposure.

5.5.2. Viral encephalomyelitis

This is a group of diseases caused by viruses which have the common characteristic of being mosquito-borne. The disease in man occurs during the summer months when mosquitoes are active. The diseases are endemic in many areas of the world. Since transmission to man occurs from the bite of mosquitoes, and since
feral birds are the primary reservoirs of the virus, people living in rural areas are more likely to be exposed to the disease.

*Eastern equine encephalitis (EEE).* This disease has a rather limited incidence and occurs mainly in the eastern and mid-central regions of the United States. It is caused by a relatively virulent virus which has many hosts. Wild avian species, such as pheasants, are the primary reservoirs. The main mosquito vector in the transmission of the disease seems to be *Culiseta melanura.* There is a cycle of transmission between wild birds and mosquitoes, by which horses and men become infected.

*Western equine encephalitis (WEE).* The virus produces infection, which is usually subclinical, in many domestic and feral animal species. The disease occurs in horses and mammals and in domestic fowl, wild birds and sparrows. Horses are usually clinically affected, exhibiting disturbances of the central nervous system. Transmission to man is by the mosquito *Culex tarsalis:* through its cycle and transmission in many hosts, man becomes accidentally infected.

*St. Louis encephalitis (Type C encephalitis).* This disease occurs mainly in Panama, Trinidad and the United States. It is clinically indistinguishable from WEE. The virus has been isolated from mosquitoes and wild birds. There are indications of a mosquito/wild bird cycle, with occasional human infection in men working in forests.

*Japanese encephalitis (Type B encephalitis).* This occurs mainly in Japan, the eastern shores of China and the eastern USSR. Many outbreaks have been reported. Transmission is mainly by *Culex* mosquitoes. The principal reservoirs of infection are wild birds and pigs. Cattle, horses and other domestic animals may get the disease. Its occurrence in man is accidental and mainly affects workers in paddy fields. More than 40 million vaccinations have been performed in Japan.

*Murray Valley encephalitis.* Many cases of encephalitis reported in Australia are caused by virus infection due to mosquito trans-
mission. Some *Culex* and *Aedes* strains were found experimentally to be capable of transmitting the disease. Serologically, the virus is closely related to the Japanese (B) and St. Louis (C) viruses.

The encephalitis group (WEE and EEE) produces central nervous system damage, leading to severe headache, muscular pain, lethargy, amnesia, ataxia, tremors and convulsions. Recovery may follow the acute stage in ten days. Permanent paralysis is uncommon. The diagnosis of this entire group of diseases can be aided or established by complement-fixation tests.

Vaccination for EEE and WEE is regularly practised for equine species. Vaccination of man is of limited usage, but may be considered for exceptional circumstances where exposure is heavy. Mosquito control is the most logical and effective prophylactic measure.

No vaccine exists for prophylaxis against St. Louis (C) encephalitis. For Japanese (B) encephalitis, a vaccine consisting of an 18 per cent suspension of infected mouse brain in normal saline solution inactivated by formalin has been used by the armed forces in affected areas. The introduction of measures for mosquito control reduces the possibility of the transmission of the disease to man. Mosquito control depends on the elimination of breeding places or the use of chemicals to kill the larvae of mosquitoes, the use of mosquito repellants, the spraying of vector insecticides, the use of nets and screens in the houses of infested areas and health education.

### 5.5.3. Yellow fever

This is a viral disease of common occurrence which is also transmitted by mosquitoes. Severe epidemics have been reported in Africa and South America. The mosquito *Aedes aegypti* (see figure 38) is responsible for the spread of urban yellow fever to man from man or from infected animals. In areas where these mosquitoes have been eradicated, the disease has become less prevalent or has virtually disappeared. However, the disease still persists
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Figure 38. Mosquito *Aëdes aegypti*
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in various areas between mammals and mosquitoes. Jungle yellow fever is transmitted to man from animals by other mosquito species.

Clinically, the disease is characterised by acute fever, with chills, headache, backache, nausea and vomiting. There is a tendency for haemorrhage. Albuminuria is present, especially when the fever is high.

Diagnosis depends on the clinical picture, complement-fixation and serum neutralisation tests. The identification of viruses from the blood is possible with intracerebral inoculation of mice.

The control of the disease depends on mosquito control. Vaccination against yellow fever is also possible, with an immunity of six years or more following the injection. Care should be taken to avoid reactions from egg protein vaccine in allergic individuals; in such instances, vaccination must be done using a minimum dose.

5.5.4. Dengue

Dengue is a widespread viral disease transmitted by the mosquito *Aedes aegypti*. It usually takes a benign course, with systemic infections that may be accompanied by an acute onset of fever and severe headache. Pain in muscles and joints as well as a maculopapular rash are early symptoms. A petechial rash occurs at the termination of the fever.

Laboratory diagnosis depends on serological tests using acute and convalescent sera.

In cold climates, the virus is unlikely to multiply and the level of transmission by mosquitoes is lower.

The control of the disease depends on the suppression of mosquitoes by the usual methods.

5.5.5. Rocky Mountain spotted fever

This is a moderately severe fever, caused by *Rickettsia* organisms. It is typhus-like, with a profuse macular rash, and is transmitted by
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bites of infected ticks. In the western United States, *Dermacentor andersoni* is the primary vector. The habitat of this tick is mainly wooded areas, and thus outdoor occupational groups are exposed. In the eastern United States, the main vector is *D. viriabilis*, a tick which is found in more domestic surroundings, resulting in exposure to the general population. The disease in man occurs in spring and early summer, and is chiefly associated with the tick season.

The rash mainly affects the trunk and limbs. At first it is macular, later becoming petechial. There may also be involvement of the vascular system.

Diagnosis depends on the detection of antibodies by serological tests. Complement-fixation and immunofluorescence tests are the most frequently used.

Tetracycline and chloramphenicol are effective antibiotics for the treatment of the disease in man. They should be given early in its course.

The control of Rocky Mountain spotted fever is by prevention of tick infestations. It is most important that protective clothing be worn during work in infested areas, and the examination of the body in order to detect and remove ticks upon returning from work is essential. Tick repellants are also used. Immunisation appears to be effective but is limited to persons at high risk.

5.5.6. *Scrub typhus (tsutsugamushi fever)*

This disease has been known in Japan since 1878 and was originally known as Japanese flood fever. It occurs among persons whose work or environment is associated with water. People who work in forests are also at risk. The disease is found in the Far East, Australia, India, Papua New Guinea and Sri Lanka. The reservoir and cycle of infection appear to be from the mite *Trombicula akamushi* to rats and mice and back to mites. Men are accidentally infected while working in, for instance, partly cultivated areas near the jungle, which are infested by rats. In man, scrub typhus may occur either in sporadic or in endemic forms.
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The disease is caused by *Rickettsia tsutsugamushi*, which can infect experimental animals, monkeys and chick embryos.

Clinically, the disease evidences a sudden onset with fever, rigors and headache. The fever is remittent in most cases. A macular rash develops on the sides of the chest and abdomen. A characteristic feature is the occurrence of a punched-out ulcer, covered with a blackened scab, which marks the site of the mite bite. Lymph glands in the area of the eschar are sometimes enlarged. Other generalised manifestations, such as delirium, twitchings, tachycardia, low blood pressure and pulmonary symptoms, are also observed.

Laboratory diagnosis may be performed by complement-fixation tests and also possibly by Felix-Weil tests.

The control of the disease depends on mite and rat control. Workers’ camps should be located away from infested areas. Antimite chemicals may be used either for killing or for repelling. Dimethyl and dibutyl phthalates and benzyl benzoates can be used to kill the mites. The addition of these substances to laundered clothes keeps them mite-free.

The immunisation of potentially exposed workers has been attempted. Prophylactic treatment with antibiotics has been adopted with success, especially in combination with vaccination.

5.5.7. Plague

Plague is a serious disease which caused great pandemics in the past. The disease occurs sporadically today, and there are still endemic areas in parts of Africa, south-east Asia and South America. The disease is spread to man by the bites of fleas which carry the organisms from infected rodents. The primary reservoirs are feral rodents; thus people living or working in rural areas are potentially more frequently exposed. When a plague epidemic occurs, there is no occupational differentiation and any person may be exposed and contract the disease. However, herders, farmers and persons in similar occupations may be at relatively greater risk of contracting plague when it occurs sporadically in endemic areas.
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Plague is caused by the aerobic bacillus, *Yersinia pestis*. The disease occurs in three forms: bubonic, septicaemic and pneumonic. Bubonic plague is characterised by enlarged lymph glands, bacterиемia and a vesicular or carbuncular lesion at the site of the flea bite. This form is usually not too severe unless left untreated. The septicaemic form may develop as a secondary phase of the bubonic form. The pneumonic form is the most severe, and may result as a secondary phase of the septicaemic form, or by primary inhalation of infectious droplets from infected patients. Severe general manifestations are seen in the latter, with rapid deterioration of the patient.

The treatment of plague depends on the use of streptomycin or chloramphenicol, in combination with sulphonamides. Plague control is founded on anti-rat measures and the vector control of fleas. The immunisation of man against plague is achieved by anti-plague vaccines which are prepared from encapsulated organisms (*Y. pestis*). The disease is now generally on the decline in most countries.

5.5.8. Dermatoses

The skin of agricultural workers is particularly susceptible to infections caused by excessive sweating, extremes of temperature, contact with the soil, insect bites, poor or inadequate clothing and lack of bathing facilities. Sweating alters the pH of the epidermis, turning it into a more favourable medium for growing bacteria and fungi and for harbouring insects. Sunburn may increase the possibility of infection and inflammation. Diabetes, buccal sepsis, superficial and intestinal parasites and minor trauma and abrasions on hands and feet are contributory causes to the development of complications, such as lymphangitis, erysipelas, abscesses or gangrene. Elephantiasis may result from repeated attacks of lymphangitis due to parasitism (for example, filariasis).

Some of the dermatoses that occur most commonly in agricultural workers are examined below. Certain of these are specifically occupational, such as rice workers' dermatosis and milkers'
nODULES. Others may occur as endemic diseases affecting the whole population of villages.

*Rice workers' dermatosis.* This disease is caused by cercariae in stagnant pools and flooded fields. Lymphatic infiltration of the papillary layer of the skin and around the blood vessels is observed. Erythematosis and papular eruptions may be observed. Conditions of dermatophytosis are also described.

*Cutaneous mycosis (ringworm).* Animals constitute an important reservoir of fungal infection in humans. In animals there is a high prevalence of ringworm, which is transmitted to man through direct contact, or contact with contaminated fomites. Ringworm infection of cats and dogs by *Microsporum canis* is common. The affected cat may appear quite normal or may show small scattered areas of hair loss. The fungus infection occurs by direct transmission to exposed areas, particularly wet and hairy parts.

In man, another condition of suppurative ringworm may occur among rural workers as a result of contact with ringworm-infected cattle. The causative organism is *Trichophyton verrucosum*, which affects cattle during the winter months when they are kept in barns. Indirect transmission may occur from the infected hairs of animals.

Another possible source of infection is *T. mentagrophytes*, which occurs in rodents and can therefore cause a substantial incidence of ringworm infection in rural areas. The groups affected by these diseases are farmers, dog breeders and persons handling animal carcasses and hides. Children in rural areas may develop ringworm of the scalp from contact with cats and dogs.

Ringworm is a resistant skin infection. Prevention is through control of the infection in animals. A high degree of general environmental sanitation and good animal management practices will help to prevent the disease in animals. Treatment with griseofulvin, which is an oral antibiotic, together with the clipping of hair and the local use of fungicides, helps to control the disease. Public education will help to prevent the disease in man, for example by teaching how to identify and avoid the infected sources. The early detection and treatment of cases in man is important.
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5.5.9. Actinomycosis

This infectious disease is caused by various micro-aerophilic fungi which produce nodular, infiltrated lesions with central necrosis and chronic subcutaneous abscesses and sinus tracts. The lesions are chronic. Infection through the teeth, gums or tonsils results in infiltration of the jaw and neck. This condition is known as “lumpy jaw” in cattle. Usually there are yellowish-grey granular masses of fungi, which are described as “sulphur granules” and help in the identification of the organism in tissue. Other organs, such as the lungs, may be involved.

The disease is not highly transmissible from one infected animal to another, and the organism is usually introduced through injury. Although most cases in man are probably contracted directly from the soil, man may contract the disease from infected cattle, swine or horses. The disease occurs sporadically in all parts of the world.

Treatment is usually undertaken with iodides, sulphonamides and penicillin. Surgical intervention may be employed to remove sinus tracts. X-ray therapy is also used.

5.5.10. Echinococcosis (hydatidosis)

Hydatid disease results from the infection of man with larval stages of the tapeworms Echinococcus granulosus and E. multilocularis. Hydatid disease has a wide distribution, especially in sheep- and goat-raising areas of the world. The adult tapeworm lives in the small intestine of carnivorous animals, principally canine species. Man is an accidental intermediate host for the larval hydatid stage of either species of the worm. Ingestion of the infectious agent is the mode of transmission. In agriculture, plants may be contaminated by the excreta of infected animals; contact with such infestation through the handling of food and water containing infected matter leads to transmission to man.

In man, the disease is characterised by cyst formation, mostly in the liver. The cysts are filled with fluid that contains the developing parasite. There is an inflammatory and fibrous tissue reaction around the cysts.
5. Zoonoses, infectious and parasitic diseases

5.5.11. *Other diseases of potential occupational significance to agricultural workers*

<table>
<thead>
<tr>
<th>Disease</th>
<th>Occupations exposed</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Diseases caused by viruses and rickettsia</em></td>
<td></td>
</tr>
<tr>
<td>Catscratch disease</td>
<td>Cat handlers, dog handlers, veterinarians; the aetiology is thought to be a virus, but is not documented at present.</td>
</tr>
<tr>
<td>Newcastle disease</td>
<td>Chicken handlers, poultry houseworkers, turkey handlers.</td>
</tr>
<tr>
<td><em>Diseases caused by bacteria</em></td>
<td></td>
</tr>
<tr>
<td>Listeriasis</td>
<td>Animal handlers, cattle breeders.</td>
</tr>
<tr>
<td><em>Other fungus diseases</em></td>
<td></td>
</tr>
<tr>
<td>Aspergillosis</td>
<td>Bird handlers, grain mill workers.</td>
</tr>
<tr>
<td>Coccidioidomycosis</td>
<td>Farmers, fruit pickers, shepherds.</td>
</tr>
<tr>
<td>Histoplasmosis</td>
<td>Farmers, poultrymen.</td>
</tr>
<tr>
<td>Sporotrichosis</td>
<td>Berry pickers, farmers, florists, foresters, gardeners.</td>
</tr>
<tr>
<td><em>Other diseases caused by parasites from mites</em></td>
<td></td>
</tr>
<tr>
<td>Grain itch</td>
<td>Barley and malt-house workers, cotton-seed handlers, farmers, grain elevator workers, straw-board makers.</td>
</tr>
<tr>
<td>Creeping eruption</td>
<td>Cat and dog handlers, veterinarians, and so on (from infected larvae from dog and cat hook-worm eggs).</td>
</tr>
</tbody>
</table>
6. Organisation of occupational health services and medical inspection of labour in agriculture

6.1. Introduction

The Joint ILO/WHO Committee on Occupational Health has given the following broad definition of occupational health:

Occupational health should aim at: the promotion and maintenance of the highest degree of physical, mental and social well-being of workers in all occupations; the prevention among workers of departures from health caused by their working conditions; the protection of workers in their employment from risks resulting from factors adverse to health; the placing and maintenance of the worker in an occupational environment adapted to his physiological and psychological equipment and, to summarise: the adaptation of work to man and of each man to his job.¹

There are two main reasons why it is essential to organise occupational health services in agriculture: first, a large proportion (and, in many countries, the majority) of the working population consists of agricultural workers; and second, technical progress in agriculture throughout the world (development of mechanisation, increased use of chemicals, concentration of livestock) has considerably increased occupational risks.

However, the organisation of occupational health services in agriculture is influenced by a number of factors peculiar to the rural environment and to the particular nature of agricultural work:

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(1) Unlike industrial workers, agricultural workers are usually dispersed in remote rural areas where public services generally may be insufficient. However, although this dispersal is characteristic of agriculture, its importance should not be exaggerated. Concentrations do exist in agriculture, the most striking examples being the large plantations of cotton, tea, fruit trees, and so on. Moreover, although the dispersal of workers makes it difficult to organise occupational health services, it is not an insurmountable obstacle, as the satisfactory situation in the building industry shows.

(2) A wide variety of jobs are performed by the agricultural worker, especially in small undertakings. Nevertheless, there is a similar variety in other industries and this has not hampered the organisation of occupational health services.

(3) For the most part, the work is done in the open air and consequently the worker is exposed to all weathers.

(4) In all countries, the agricultural environment suffers from a certain technical backwardness as compared with the industrial environment. Tradition in agriculture often hampers the application of modern techniques, of which occupational health services are only one example.

(5) Although new forms of work organisation have made considerable progress in agriculture, the fact that the performance of agricultural work is so dependent on weather conditions is a considerable obstacle to more efficient operation. Thus, while the speed of a production line in industry can be accurately planned, a sudden change in the weather—a rainstorm, for example—will compel the farmer either to work faster or to stop working altogether. Moreover, these changes in the weather can sometimes completely alter working conditions: for instance, plans for the application of pesticides in favourable conditions will be upset if a sudden wind springs up from the wrong quarter—the favourable conditions become both difficult and dangerous.
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(6) The agricultural worker’s private life and his working life are often interwoven. In certain kinds of undertaking it may be possible to separate the two; however, in most cases the existing situation seems likely to continue for many years to come. Furthermore, as agricultural work is carried on in the countryside, it is subject to the risks inherent in a rural environment, with the workers being dependent on the general standard of public health in such matters as the provision of an adequate water supply and protection against vermin and insects. These factors have a considerable bearing on the health problems of a particular area.

(7) Agricultural work is very often a family affair, and sometimes all the worker’s family—children, women, old people—share in it to a greater or lesser extent. The absolute necessity to care for these people modifies the traditional form of occupational health services, which are generally intended for the workers alone.

These and other factors peculiar to agricultural work fully justify the organisation of agricultural health services. Their nature will, however, vary according to the country, the district, the local crops and the method of growing a particular crop. It is impossible to deal here with the needs of each special case, from the large undertaking to the small family farm, but an attempt will be made to establish the general principles which should be observed in order to improve the working and living conditions of the agricultural worker.

6.2. Practical organisation of agricultural health services

Agricultural health services are governed by the general principles for occupational health services drawn up by the Joint ILO/WHO Committee on Occupational Health, with due allowance being made for the special nature of agricultural work.
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6.2.1. Preliminary investigation

As mentioned above, agricultural health services should be organised according to local conditions and needs. The type of service provided will therefore be based on as exact a knowledge as possible of these conditions and needs and of likely future developments.

In the first place, it is essential to ascertain as precisely as possible the number of full- and part-time workers, both permanent and temporary. This figure will include women and children, as well as those doing industrial work followed or preceded on the same day or during the same week by agricultural work. Inevitably, it will be virtually impossible to arrive at an exact figure, but information that is even approximately correct will be useful. It will also be necessary to know the number of persons needing medical supervision, and where they are to be found.

In the second place, the hazards to which workers in a particular area are exposed must also be ascertained. This is a very difficult task. However, fairly reliable results may be obtained with the co-operation of the various organisations which are in a position to know these risks—for instance, the service organisations (fire, civil protection, health and first-aid) and other bodies such as insurance institutions, if any.

While serious accidents, acute poisoning or the effects of particularly adverse working conditions are likely to leave clearly visible effects from which the degree of risk can be assessed, other hazards are difficult to evaluate (chronic conditions due to noise, vibration, cumulative poisoning, and so on).

Some form of administrative organisation for the reporting of risks is indispensable. This should cover all the aspects involved and be equipped to undertake statistical analysis. However, such organisations are often lacking in one respect or another, where they exist at all.

The attention of the medical profession should be drawn to the possible existence of chronic risks, since these often escape notice and, when discovered, are not reported to a competent authority qualified to assess them.
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The same is true of epidemiological information, of which there is a world-wide lack. Admittedly, some of the chief epidemic or endemic diseases have been studied; but in all countries, even those with high sanitary standards, epidemiological research has been generally neglected. Investigations should be conducted at the local, regional, national and even international levels. As well as studying pathology in man and beast, the investigators may expect to discover reservoirs of infection in man, in domestic and wild animals and in the ecological environment, together with the vectors capable of transmitting infection. These specialised investigations could either be confined to one disease and cover a fairly large area or be confined to a small area and extend to all possible diseases. Their organisation calls for considerable resources and the co-operation of all the specialists taking part, and their preparation must be psychological, administrative and technical.

The purpose of psychological preparation is to draw the attention of the public authorities and the population at large to the importance of the investigation and the need for it, to persuade the authorities to give the investigators all the help they require, and to persuade the public to submit willingly to all the questionnaires and samplings to which they will be subjected. The population should be prepared by a campaign mounted by the health services (administrators, doctors, veterinary surgeons, social assistants), by propaganda in meetings and in villages, by the regional press and radio, and by various audio-visual demonstrations presented by educational or occupational bodies.

The administrative preparation involves approaches to ministries and subordinate organisations, to prominent persons or representatives of the people, and to educational, trade union and religious authorities.

The technical preparation comprises the mobilisation of the necessary staff and equipment. In addition to a permanent secretariat, the staff will consist of permanent technical personnel (bacteriologists, virologists, parasitologists, doctors or veterinary surgeons, ecologists (mammalogists, entomologists)) and tem-
6. Occupational health services and medical inspection

porary technical personnel (botanists, pedologists, climatologists, psychosociologists, economists, accident prevention specialists, industrial hygienists).

Financial resources should be made available for the necessary research equipment (sampling apparatus, marking apparatus, travelling laboratory) and the laboratories which are to examine the samples taken. These should be chosen for their competence and geographical location.

The investigation will include a general survey of the region, classifying its geographical, geological, cadastral, demographic, economic, social and medical features. Research may be planned and the human and animal diseases of the region explored on the basis of the findings of the survey. The actual study will consist of clinical examinations of people and animals, paraclinical investigations (allergies, radiological examinations, and so on) and sample-taking from people and domestic and wild animals (a carefully completed record card should accompany each sample).

The appropriate season should be chosen for each investigation according to its nature, and provision should be made for repeating the examination if necessary.

This epidemiological investigation can serve as a model for general health, traumatic and toxicological investigations.

Of course, such investigations call for sizeable resources, and not all of them can be undertaken; but they may become possible in the future, little by little, depending on the resources available in the countries concerned. Even investigations of limited scope will furnish a wealth of information.

As part of the preliminary inquiries to be made before agricultural health services are established, it will be necessary to study the labour legislation in force, as regards the possibility of extending it effectively to agricultural workers, and to provide for the amendments and additions needed for the administration of agricultural health services. It will also be necessary to draw up an inventory of all the existing medical, veterinary and health services and their activities, as well as a census of other related professional and auxiliary personnel available to deal with the
problems of agricultural medicine. Naturally, none of these activities can be undertaken unless adequate financial resources are available. The scale of the activities will clearly have to be adapted to the funds at the investigators’ disposal.

6.2.2. Implementation

Role of occupational health services

The role of occupational health services is considered in the ILO’s Occupational Health Services Recommendation, 1959. The following paragraphs take account of the particular features of agricultural work in this connection.

*Pre-employment, periodic and special medical examinations (biological, radiological, etc.).* Workers should be medically examined on engagement and at yearly intervals thereafter, except for certain classes of workers, such as pregnant women, young persons, workers exposed to special risks and others in special circumstances, for whom the doctor will decide the appropriate periodicity. Examinations should also be made on the resumption of work after an absence due to illness or accident. In this way, the doctor will be able to gain an idea of a person’s state of health and, being aware of his working conditions, to advise him as to the kind of work best suited to him. The examinations should make it possible to diagnose troubles in their initial stages, to see whether satisfactory preventive measures have been taken, and if necessary to assign the worker to another job.

However, changes of job, although easy to arrange in large enterprises, are much more difficult in small family undertakings. In such cases, it is advisable to consider carefully the possibility of transferring the worker to another occupation, with due regard being had to his physical and mental aptitudes and the state of the labour market.

*Surveillance of the working environment.* It is obvious that a doctor who has to judge the aptitude of persons for certain jobs,
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and to supervise them medically in the light of the risks to which they are exposed, must be familiar with their working conditions.

Job analysis is an essential part of the doctor's work, enabling him to advise the technicians on the methods to be employed to avoid risks (handling of poisons, contagion risks, and so on). He should also check that the personal protective measures recommended have been applied (clothing, respirators, and so on).

**Prevention of accidents.** In agriculture, the proportion of occupational accidents attributable to the human factor is particularly high. It is essential that the agricultural health service should help actively to instil the safety spirit in the workers by seeing that personal protective equipment is properly used and maintained and by encouraging the adoption of appropriate safety measures.

**First aid.** In view of the difficulties of establishing effective first-aid services in a rural environment, the role of the agricultural health service should include initial and regular subsequent training of first-aid personnel.

**Health protection measures.** The closer the agricultural health service is to the workers, the easier it will be for it to advise on health measures, whether these measures are concerned with investigations or with the organisation of prevention against scourges such as tuberculosis or malaria. The rural doctor can protect people against certain risks by active immunisation (for example, vaccination against tetanus) or by chemotherapy (for example, distribution of quinine derivatives in malarial zones.)

**Surveillance of the hygiene of sanitary installations.** The doctor must be an adviser in matters of hygiene and health. He should inspect the workers' housing and the various sanitary installations (water supply, shower baths, drains, latrines, and so on). The inspections will also provide opportunities for educating workers in health and hygiene.
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Consultations on health problems. In the event of occupational or other disorders, agricultural workers should be encouraged to go to the agricultural health service for information on any questions of health or hygiene. Such personal contacts will increase when the doctor has been able to gain the confidence of those in his care, and they will be extremely useful for the prevention of risks and for the promotion of health education, especially as regards nutrition.

Medical treatment. The rural doctor might participate in the treatment of mild conditions. However, if medical aid is difficult to obtain, the doctor may be allowed to give any necessary treatment to agricultural workers and their families.

Health education. The doctor has a major role to play in educational health programmes, as regards both general health problems (general hygiene, nutrition, and so on) and problems relating to occupational hazards and the protection of workers. He should maintain close contact with all the different bodies concerned with questions of the farmers' health, safety and welfare in rural areas, as well as provide direct advice on occupational health in agriculture.

Co-operation with other medical services. The doctor must not work in isolation from other medical services. He must rather establish contact with other kinds of preventive medical services (health agencies, school and sports medical services, and so on). He must meet other practising doctors and visit hospitals and supervisory medical services (participation in inquiries into occupational accidents and diseases, furnishing opinions on resumption of work jointly with medical inspectors of insurance institutions).

Agricultural health services must be operated in close co-operation with the local veterinary services, practitioners and/or administrators in order to deal with zoonoses, and with agricultural technicians and labour inspectors.

Records. It is essential that records be kept up to date if the agricultural health service is to function efficiently. This is all the
more important if the doctor is to monitor a worker's state of health and to follow its evolution, particularly as regards chronic affections and cumulative poisons.

The statistical processing of records by a central organisation would make it possible to undertake comparative studies at the regional, national and international levels. This is particularly so if the health services are to take part in research (inquiries, specialised examinations) and in education.

Operation

The number of qualified medical personnel available determines the way in which agricultural health services may be operated:

(1) In countries where there are enough doctors (say, one doctor for every 800 inhabitants, with the term "doctor" covering both general and specialist practitioners and supervisory and preventive personnel), provision may be made for occupational health doctors specialising exclusively in agricultural medicine.

(2) In countries where there are not enough doctors but where there is no serious shortage, an agricultural health service may be incorporated in a system of preventive medicine serving the population among which the doctor concerned works (e.g. public health service, industrial medical service).

(3) In countries where there is a chronic shortage of doctors, which unfortunately is the commonest case (it is estimated that about two-thirds of the world’s population have no means of obtaining adequate medical treatment), the agricultural doctor may consider participating in the treatment of workers and their families.

Operational conditions

Fixed or itinerant medical installations may be provided, according to the density and the distribution of the population of agricultural workers. Fixed installations are to be preferred, because better health services can be provided than with itinerant in-
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installations, with, at the same time, minimal restrictions on the occupational health doctor.

*Characteristics of a health service*

Two basic conditions are essential for the organisation of a successful health service. First, the doctor must be competent. His competence in handling the problems of occupational health in agriculture can be enhanced through specialised educational programmes that take account of the continuing evolution of agricultural technology. Second, the doctor must have sufficient time in which to practise agricultural medicine. Ideally, the maximum workload for a full-time doctor would be 3,000 workers.

*Resources*

The doctor must, of course, be provided with the installations and equipment needed for his work, as well as the necessary nursing staff, secretarial staff and possibly social staff in support.

*Type of organisation*

Agricultural health services can be organised either on a geographical or on an occupational basis. Obviously, the greater the degree of specialisation, the greater the efficacy of the service, since the doctor will not have to be a specialist in so many areas.

However, apart from a few exceptions (large undertakings, single-crop areas), it is practically impossible for an occupational health doctor to specialise in agricultural medicine. Sometimes, even in rural areas, artisans and tradesmen for whom there is no suitable occupational health service available can be cared for by the agricultural health service. Nevertheless, when feasible, specialisation in agricultural medicine is to be preferred.

*Finance*

In many countries, agricultural workers constitute only a small proportion of the wage-earning population. Farmers may reasonably be asked to help to finance agricultural health services from
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which they benefit. It is, however, unlikely that agricultural health services would be set up by private initiative.

6.3. Medical inspection of agricultural work

Experience has shown that, even in countries where legislation on occupational health exists, it is not always applied according to its expressed aims. Sometimes employers and workers, either because of the pressure of production or because of their failure to understand the importance of occupational health—the significance of which has not always been fully appreciated, perhaps because of its relatively recent creation—have not been too keen on submitting to medical examination, or have made the doctor’s task more difficult.

Since occupational health in agriculture is a comparatively new discipline, many subjects are obviously still very imperfectly understood. The co-ordination of investigations into a particular morbid condition or the working conditions of a particular agricultural occupation can be extremely useful in filling some of the gaps in knowledge. There is no doubt that a medical inspectorate should work in close collaboration with the universities to this end. Moreover, a university is particularly well equipped to undertake certain investigations. Its relationship with the inspectorate will enable it to direct its research work towards the most current and urgent problems.

We may consider an inspectorate as being responsible for a certain geographical region, so that it gains an idea of the different problems of that region. However, since inspectors should have the same qualifications as practising doctors, it would be extremely difficult for them to be specialists in all areas of medical knowledge, if only because of the range and complexity of medical science. Hence, the regional inspectorate should be backed up by a gen-

1 See also ILO: *The role of medical inspection of labour* (Geneva, 1968)
eral inspectorate which would not only co-ordinate all the regional inspectorates but also maintain a staff of specialists for matters such as accidents, poisonings and zoonoses. The general inspectorate would thus be able to advise practising doctors or to undertake specialised investigations with a good chance of success.

Under which administrative authority should a medical inspectorate of agricultural labour be placed? Possible authorities are the ministry of agriculture, the ministry of labour or the ministry of health. The answer to the question depends largely on the national administrative policy of the country concerned. What is essential is that a competent medical inspectorate should exist.

6.4. Problems of education and training in occupational health and hygiene in agriculture

One of the distinguishing features of agriculture is that it is carried on in an essentially rural environment where working and living conditions are often closely interwoven. It is therefore necessary to provide the agricultural population with suitable education in hygiene. Hence, agricultural technicians and health officials should have received some training in occupational health and hygiene in agriculture.

6.4.1. Education

As has been seen, in most cases the whole family takes part in agricultural work in varying degrees, or at least lives in the working environment. It is therefore necessary to educate both children and adults, including elderly persons whose useful experience has sometimes been nullified by technical advances in agriculture (by the introduction of new poisons, for example) or by developments in sanitation. To this end, all practicable methods should be employed. Publications may include posters, pamphlets, leaflets and press articles. Since many persons will be illiterate, the spoken word must not be neglected: lectures and broadcasts will be useful.
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Furthermore, good use may be made of audio-visual means such as lantern slides, films, television (when it exists) and visits to model villages and model farms.

As regards the question of where health education should be given, the school clearly seems to be the best place for reaching most of the children. However, since education in a rural environment is hampered by a lack of schools, transport difficulties, bad weather, the daily round of work on the farm, and the seasonal employment of children in the fields, the school is not enough and children should also take advantage of courses for older people.

For adults, including the elderly, meetings could be organised in the village, either in administrative or cultural centres or in the premises of agricultural associations. If as much advantage as possible is taken of the various means available, the lessons in health and hygiene will be all the more effective.

The teachers of health education will, in the first place, be health officials, nurses, scientists and engineers, social assistants, pharmaceutical chemists, veterinary surgeons, doctors and perhaps health administrators; but those who have some influence by reason of their function should not be overlooked—members of the teaching profession, whether general or agricultural, technicians, agricultural consultants or administrators and, sometimes perhaps, persons in holy orders.

Specialised services such as medical services, agricultural health services and agricultural medical institutes may be expected to organise health education campaigns. Notwithstanding these campaigns, the day-to-day work of the different authorities will be the most important.

Health education programmes must be both specific and comprehensive. First, they must be specific enough to provide adequate background knowledge for specific remedial solutions to be recommended, and yet general enough so that existing hazards and risks will be recognised and appropriate solutions sought. Topics such as housing, water, waste disposal, nutrition, epidemiological risks, poisoning and accidents should be included.
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In conclusion it must be stressed that the multiplicity of problems facing the agricultural sector should be no reason for neglecting education in health and hygiene. It is quite certain that some rural housing problems, for example, will not be solved without adequate economic resources. However, in many cases, simple and inexpensive methods will be sufficient to bring about considerable improvements in both living and working conditions.

6.4.2. Training

Trainees can be divided into two groups: health officials and agricultural officials.

Health officials

Nursing, social and health personnel. Both in countries where there are enough medical personnel and, with more reason, in those where there are not (and where, accordingly, responsibilities are greater, training briefer and general education poorer), these officials should be aware of the new techniques in agriculture and of their dangers.

Pharmaceutical chemists. In some countries, pharmaceutical chemists can play an important role in health education, prevention and first aid. While some subjects that are useful in country districts, such as cryptogamy and toxicology, are normally taught, it would be desirable to give better instruction on occupational health in agriculture, especially to newcomers to agricultural life.

Veterinary surgeons. The veterinary surgeon has, by his training, a good knowledge of the diseases affecting animals, especially infectious and parasitic diseases. He should in addition be well grounded in agricultural medicine because of his contacts with farm workers and his important functions in the field of epidemiology (since prevention should be for both man and animal) and toxicology (where human and animal problems are the same). Thus he is well qualified to take part in preventive and educational activities.

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*Doctors*. Throughout the world, the problems of agricultural medicine seem to be treated no more than superficially in ordinary medical courses. However, specialised courses should be provided both for those who are actually practising general or specialised medicine in rural areas and for those who are preparing to practise agricultural medicine.

*Health administrators*. In some countries, it is becoming increasingly common for health administration to be entrusted to non-medical personnel. The lack of general medical qualifications of these people is an added reason why they should be trained in agricultural medicine, especially when they are working in a rural area. Such training facilitates their understanding of the problems they are likely to encounter.

*Agricultural officials*

*Heads of undertakings*. Both on small family farms and in large specialised undertakings, those responsible for management should spend some time learning about agricultural medicine in the course of their training.

In both elementary agricultural schools and highly specialised institutes, courses in agricultural medicine that will be relevant to the educational level of the students must be organised. During his training, the agricultural student acquires the habit of keeping in touch with doctors, so that the medical problems that he will subsequently meet with in practice may be solved more easily.

*Agricultural administrators*. Agricultural technicians, engineers working on the development of rural zones or on agricultural buildings and agricultural labour inspectors should all, within the ambit of their responsibilities, be familiar with the problems of occupational health in agriculture. Because of the decisions they take and the advice they give, they have an extremely important part to play in education and prevention.
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6.4.3. How to train

As well as the courses in theory that may be given to health and agricultural officials, practical courses are highly desirable. It would be particularly useful for health officials to take courses in agriculture so as to improve their knowledge and understanding of agricultural technology.

For agricultural officials, it would be useful if they spent some time at medical institutions or services so as to gain a better insight into the problems of rural doctors.

Model farms for the various branches of agriculture could provide courses for health and agricultural officials where, inter alia, they could exchange views on common problems.

The instruction would not always take the same form. Depending on the future career of the students, courses could be organised by agricultural technicians and/or the staff of agricultural health services, agricultural medical inspectorates and agricultural labour inspectorates.

6.4.4. Where to train

Courses in agricultural medicine, both for health officials and for agricultural officials, should feature in the curricula of educational establishments. However, because of the almost complete lack of such courses in these establishments at present, some post-university or recurrent education or training should be provided.

Education is apt to become sterile if it is not activated with the catalyst of practical experience. Congresses are a valuable means of educational regeneration. They should be organised at the regional, national and international levels. They may be confined to a single subject or cover a number of subjects. Everyone who might be interested (and not merely doctors) should participate. The expression of opposing points of view often leads to fruitful exchanges of information and the clarification of obscure ideas, or to the initiation of research and investigation into hitherto unexplored areas. Comparisons between regional and national experience are equally beneficial.
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6.4.5. Provision of training

The university

The teaching staffs of universities are particularly well qualified to conduct training courses. Their experience in research and teaching enables them to master the subjects, express them with the maximum of clarity, keep abreast of changes and (provided that they have the necessary resources at their disposal) undertake research into practical needs.

The medical inspectorate

The medical inspectorate plays an important role in providing agricultural health education in undertakings. It should therefore also co-operate with the universities in the provision of training in occupational health and hygiene. On the practical side, the inspectorate can arrange visits and participate actively in investigations and research projects.

Practitioners

Rural practitioners' experience of specific everyday problems enables them to make a positive contribution to training. They can also participate in education and research through the work of their learned societies and in collaboration with the inspectorate and the universities.

Institutes

Effective collaboration between the various persons and establishments concerned with training in occupational health and hygiene can most often best be achieved within the framework of institutes. Depending on needs and resources, these institutes may be national or regional in scope.

The institutes discussed here may specialise in agricultural medicine or form part of a health institute or an occupational medical institute.

Their personnel should include not only teachers, medical inspectors and practitioners but also all who can contribute to the
declared educational and research goals. Some of the staff would be directly concerned with agricultural medicine (veterinary surgeons, toxicologists, bacteriologists and virologists, physiologists, agricultural engineers), while others, indirectly concerned, would make useful contributions of a statistical, psychological or sociological character.

The function of these institutes would be to harmonise education and research. Their three fields of action would be education, research and evaluation.

The importance of evaluation cannot be over-emphasised. It is poor practice to undertake research without knowing whether earlier work has been undertaken on the same subject and, if so, what its results were.

Such an institute, serving simultaneously as a centre of agricultural medicine, a documentation centre and a statistical office for the rational processing of information, should be supplemented by a centre for conditions of employment, which would be concerned with job analysis, rationalisation, ergonomics, and so forth. There should also be a centre for occupational pathology, which would study toxicology, epidemiology and various advanced medical and technical subjects as the need arose, and a centre for psychosociology.

The need for collaboration between the different disciplines must be especially stressed.

6.5. Organisation of first aid

The organisation of first aid in agriculture is particularly difficult—first, because the accident generally occurs at a place far removed from a treatment centre; and second, because of the inadequate means of communication and transport. It is therefore advisable to make advance provision for the administration of first aid, in order that disastrous mistakes, or indeed a total inability to save the life of a person in danger, may be avoided. The plans should provide for medical care as well as for first aid.
6. Occupational health services and medical inspection

6.5.1. First-aid training

The agricultural doctor should do his best to encourage people to become proficient in first aid. First-aid attendants should be chosen from among both men and women, and even from among young people of either sex, who often take a great interest in first aid.

First-aid training may be organised at the agricultural doctor’s medical centre. According to the trainees’ opportunities for attendance, it could be either spread over several weeks or concentrated in a short period. However, in countries where training is provided by public or private organisations, the doctor should get in touch with these and preferably co-operate with them.

The training course should include the study of different accidents and the methods of administering first aid to the victims, with particular emphasis being laid on the mistakes to be avoided. However, the specific content should follow approved standards of first-aid practice; special attention should be given to problems which are particularly significant in the country or agricultural area concerned.

Practical exercises showing whether the lessons have been well learned are indispensable, and the award of a certificate after success in an examination is often an encouragement to the trainee.

First-aid personnel should also be provided either with pamphlets or with multigraphed instructions with which they can subsequently refresh their memories. These instructions should be simple and clear.

The training of first-aid personnel should not lead to the neglect of arrangements for medical care, and in no case should the administration of first-aid treatment cause delay in summoning a doctor. The purpose of first aid is to do what is necessary pending the arrival of a doctor. It must not be looked upon as makeshift treatment which, if unsuccessful, can be remedied by the doctor. The administration of first-aid treatment and the summoning of the doctor should be, and in most cases can be, simultaneous.
Health and hygiene in agriculture

When there are good roads, transport by ambulance is practicable; however, for various reasons (bad roads, no roads, impassable roads) provision must sometimes be made for transport by helicopter. Every sizeable treatment centre should have a landing place for helicopters.

The ambulance or helicopter should be so equipped that treatment can be begun during the journey—for instance, with resuscitation apparatus, perfusion equipment, oxygen and first-aid equipment.

6.5.2. The treatment centre

Some treatment centres specialise in certain injuries (such as burns, poisoning or snake bites). If rapid transport is available, the victim can be taken straight there. If this is not possible, he must first be treated at a primary care centre staffed by competent doctors.

6.5.3. Poisoning prevention centres

The frequency of poisoning, not only of agricultural but also of industrial, domestic and medicinal origin, is enough to justify the creation of poisoning prevention centres. The diversity of agricultural chemicals is too great for anyone but a specialist to diagnose and treat cases of poisoning correctly. Modern methods of resuscitation require specialist personnel. It is therefore desirable to provide for the establishment of such centres, especially in rural areas.

These regional poisoning prevention centres could act as both treatment centres and information centres. If treatment cannot be given, information becomes necessary.

To keep the information provided by the regional centres up to date, it would seem desirable to set up national centres that would centralise and disseminate new knowledge.
Guide to further reading

Publications of the International Labour Office


Publications of the World Health Organization

Some other ILO publications on occupational safety and health

Encyclopaedia of occupational health and safety
Contains about 900 alphabetical entries of an essentially practical character relating to various trades, occupations, processes, machines, substances, affections and so forth. The emphasis throughout is on the hazards for the workers and the safety and health measures to be taken. Numerous photographs, drawings and charts. Analytical index. In two volumes.
ISBN 92-2-101000-7  Price: 180 Swiss francs. Also available in French

Ergonomics in industry, agriculture and forestry. Occupational safety and health series, No. 35
ISBN 92-2-001656-7  Contributions in English, French, German and Russian

Man at work. Studies on the application of physiology to working conditions in a sub-tropical country. Occupational safety and health series, No. 4
ISBN 92-2-100910-6

Safe use of pesticides. Occupational safety and health series, No. 38
ISBN 92-2-101826-1

Forestry. CIS bibliographies, No. 12. Also available in French

ILO codes of practice—
Agricultural work
ISBN 92-2-100194-6

Forestry work
ISBN 92-2-100017-6

Protection of workers against noise and vibration in the working environment
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ISBN 92-2-100975-0

These ILO codes of practice and guides to safety and health are also available in French and Spanish editions (except*)
GUIDE
TO HEALTH AND HYGIENE
IN AGRICULTURAL WORK

Work on the land is man's oldest occupation. For thousands of years he has tilled the soil and bred livestock to feed and clothe himself. Agriculture remains of great economic importance today. With the application of modern technology to crop production and stock rearing, however, the many health problems which are inseparable from work in the fields and on the farm generally have become more numerous and, often, more serious. Consequently, the risks to which the agricultural worker is nowadays exposed are many and varied.

This guide completes the triad of ILO publications intended for the protection of the agricultural worker from occupational accidents and diseases. With its predecessors (the ILO code of practice entitled Safety and health in agricultural work and the Guide to safety in agriculture), it provides a wide spectrum of information on agricultural health hazards and their prevention which should be useful to developing and developed countries alike.

Price: 27.50 Swiss francs
ISBN 92-2-101974-8