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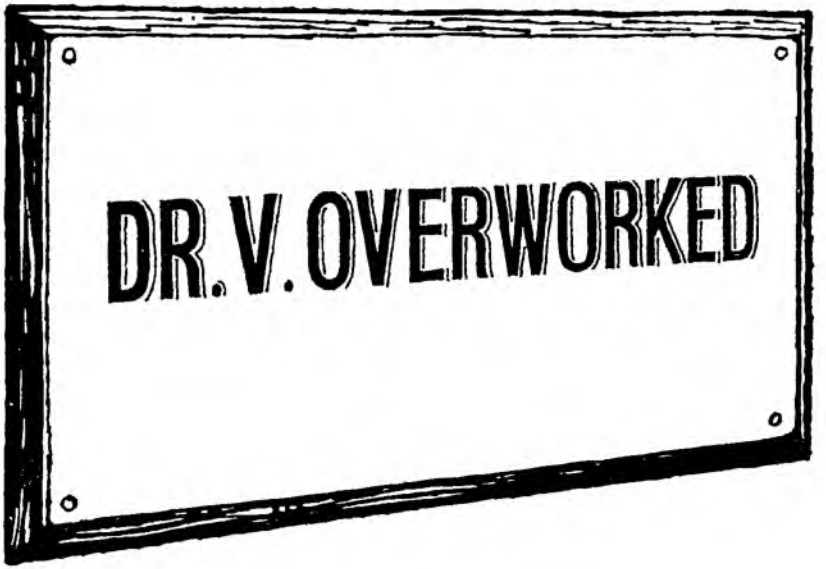

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THE DESIGN OF THE ORGANISM

R. B. FISHER, Department of Biochemistry, University of Edinburgh

—being part of an address given to the Society on 15th November, 1963

Medical students and clinicians prefer — for different but equally good and acceptable reasons — to be presented with simple and easily comprehensible explanations of physiological and biochemical phenomena. But, as I have been saying to my students for years, the body was not designed for the convenience of the medical student or the clinician, if, indeed, it can be regarded as having been designed at all.

The popular notion of what it was designed for is that every discernible aspect of its functioning is there for the benefit of the organism, and it is a popular minor intellectual sport to 'explain' a physiological observation in terms of what you would have put it there to do. The exponents of this sport rarely, if ever, pause to think what happens when man designs a complex piece of machinery. When a team of able experts designs an aeroplane they can be moderately certain of a few things: it will fly: its speed will be in the expected region: its range will be much as hoped. But it is so complex that the inter-actions between many of its parts and functions are unpredictable. So the machine that is built to the design is put into the hands of a large team of other experts who spend years measuring hundreds of its properties in flight, and it will undergo successive small modifications intended to nullify the undesirable features that are disclosed by this study. When all this has been done with enormous care, industry and intelligence, it is still possible to leave the machine in possession of some such disastrous property as the unsuspected susceptibility to fatigue of the airframe of the Comet.

Personally, I am firmly opposed to viewing the body as 'designed'. Quite apart from the dangers inherent in the easy superficial explanations of functions to which such a view inevitably leads, it leaves out two matters of importance.

In the first place, by satisfying one that everything is there, in the most subtle and intricate collaboration, to maintain the status quo, it makes it very difficult to understand the occurrence of any but the most gross of pathological processes. In the second place, it involves ignoring the conclusions of basic biology.

Biologists in general accept the notion of evolution by natural selection. By the nature of things, they cannot prove how evolution has occured. But they have been able to observe it in action in some instances, and the biological phenomena which could form its basis are beyond dispute.

For instance, it is beyond dispute that errors occur in the duplication of chromosomes and that chromosomes can segregate during cell division in irregular fashions, so that parts of two chromosomes can interchange, or fragments can break off and be lost, or more than the normal complement of chromosomes can appear in a germ cell. Chromosome abnormalities in man in association with structural and functional abnormalities are now frequently being reported as the result of recent advances in the technique of study of chromosomes.

We can lump together all the random abnormalities of chromosome reproduction as mutations and we can call the bearer of such an abnormality a mutant. The theory of natural selection suggests that evolution is due to the survival of a mutant form because it possesses greater ability to adapt to its environment than does the original form, the 'wild' type.

Before we can understand the application of this notion, two points have to be taken into account. First, in a sexually reproducing organism, it does not seem likely that a single error, resulting in a unique mutant, could have much success in originating a new species. But it is known that particular mutations occur naturally with quite a high frequency, that is,

a particular kind of cell is prone to particular kinds of reproductive inexactitude. For instance, before modern haematological advances made life supportable for the haemophiliac, that is before he had any significant chance of surviving to the reproductive age, new cases turned up with regularity, and it was clear that the mutation regularly occurred spontaneously. So, with a mutant form that does not confer a reproductive disadvantage, it is to be expected that it will accumulate in a population much more rapidly than if it had to rely for its spread on the progeny of a single mutant.

The second point is a more important one. Modern evolutionary studies have indicated that successful new forms are most likely to arise on the fringe of the habitat of the wild type. The fringe of the habitat is the region which is disadvantageous to the wild type, but just not sufficiently so to exclude it. The new form, then, does not have to be better adapted to the conditions of the major part of the habitat, but does have to be better adapted to the special features of the fringe area.

The consequences of this can be seen if some special instances are considered. An anaerobic bacterium, incapable of tolerating more than a trace of oxygen, can live in the deep layers of the soil. A mutant form capable of tolerating higher oxygen concentrations or of living fully aerobically could arise at the upper fringe of this habitat and could invade the upper layers of the soil. There is no reasons why a second mutant could not arise from this one capable of tolerating lower oxygen concentrations, so that it could re-invade the original habitat. If it were incidentally more capable of tolerating the particular conditions of acidity of the lower soil than the original form it could compete with it successfully.

Two important considerations arise from this example: first, that successive mutations do not necessarily mean successive refinements of adaptations to one environment and, second, that since it is more likely than not that successive mutations will be at different sites, the double mutation will retain the properties conferred by the first mutation, even though they have no specific value in the new environment. Remember, in this context, that evolutionary success does not mean that the successful form is perfectly adapted to its environment. It merely means that it is better adapted than is any contemporary competitor for that biological niche.

This implies that complex organisms must be expected to possess numerous features that were introduced a long time ago in evolution which have never disappeared because the pressure of selection against them has either never occurred or has never been strong enough. Recapitulation in ontogeny is sufficient evidence that this sort of thing occurs, but we rather tend to suppose that this is merely an anatomical matter, and we take comfort from such facts as that the pronephros and the mesonephros disappear during development, and that we are left with a respectable modern metanephros to look after our excretory needs.

But I think that we take comfort too easily, and that we are not really knowledgeable enough to be able to say how far cellular architecture conforms to an efficient pattern and how far it is cluttered with lumber from the evolutionary past. It is certainly true that there are anaerobic micro-organisms which contain cytochrome oxidase for which they cannot be seen to have a use and that there are marine worms containing a kind of haemoglobin which holds oxygen with such tenacity that the tension at which it will give it up is too low to support the life of the organism. Plants manufacture alkaloids for which no biological function can be found, and the form of the Peppered Moth which is now commonest all over England is so conspicuous in its black livery against the bark of clean trees, on which it rests with open wings, that it is an easy prey to birds. This last instance is particularly striking, because 60 years ago the common form was a pale speckled creature which is quite inconspicuous in this situation.

I believe that these considerations imply that the functioning of living organisms is based on crude basic mechanisms which have been overlaid from time to time by accretions, many of which are no longer particularly useful, which have tended to moderate the imperfections of the basic mechanisms in particular ways. Such arrangements might be expected to breakdown when stressed quite trivially in particular ways, and I believe that we could be better occupied in endeavouring to study the properties of these conglomerations than in searching for broad principles of design.

Have you ever thought of skeletal muscle as a kind of Heath Robinson contraption? Muscle cells at rest have rather a low oxygen consumption. As soon as the cells are stimulated to contractile activity the oxygen requirements rises enormously, and the cells are so constituted that oxygen lack or lack of a substitute

source of energy puts them rapidly into a state of contracture comparable with rigor mortis.

Yet, as soon as a group of muscle cells contracts, it exerts on the blood vessels running within it a pressure sufficient to occlude the blood supply: a process equivalent to putting a plastic bag over your head before you run for the bus.

The first stage adjunct to the muscular apparatus which offsets this major defect in design is the provision of a glycolytic mechanism. This enables the muscle to obtain some energy from the breakdown of carbohydrate to lactic acid without need for oxygen. But since the blood that brings oxygen also brings glucose, this mechanism is of little use without another, the mechanism of glycogen storage and breakdown.

The combination of these two adjuncts is of limited use: muscle cannot store much glycogen, and the glycolytic breakdown of carbohydrate provides only a fifth or a sixth of the energy that can be obtained by oxidation, so that the store lasts only a short time. In addition, the lactic acid formed in glycolysis makes the muscle more acid than usual and militates against function. So, even with these aids, the inherently bad design of muscle makes it difficult to maintain tonic activity or long-terms phasic activity, as in walking or flight, for any length of time. With these mechanisms, we still do not have an effective basis for rapid flight from an enemy, on the ground or in the air.

However, a third supplementary mechanism exists. This is the provision of myoglobin. Myoglobin is closely related to haemoglobin in function, but it occurs in the muscle cells themselves. Whereas haemoglobin consists of four peptide chains, each of which is combined with a haem molecule which is responsible for the capacity of the haemoglobin to bind a molecule of oxygen, myoglobin consists of one such peptide chain combined with one molecule of haem. There seems to be no clear relation between the sequence of amino-acids in the haemoglobin peptides and that in myoglobin. The two haem-proteins appear to have arisen separately in evolution.

Myoglobin will still retain its complement of oxygen when the oxygen tension is much below that in arterial blood, but it gives up the oxygen at a tension which is still high enough to be of use intracellularly. Thus it holds on to oxygen inside the cell until the supply from outside has run out and then prolongs the period during which the muscle cell can function aerobically. It does for the muscle cell what the Chancellor of the Exchequer wishes that he could do for the economy: it irons out the swings in the relation between supply and demand.

You will note that this arrangement is not aiding the glycolytic mechanism. It is as though someone had thought that though glycolysis was quite a good notion for a beginner, it wasn't really good enough, and it would be better to try to solve the problem another way. The interesting thing is that the myoglobin mechanism is not only of a different kind from the glycolysis mechanism but it has quite a different incidence.

Whereas all skeletal muscle appears to possess the capacity for glycolysis and for the storage of glycogen, only certain muscles in certain species possess myoglobin. Think of the white meat and the dark meat: the pectoral muscles of the almost earth-bound domestic fowl have little or no myoglobin: those of the game birds, pheasant, grouse and partridge, for instance, are richly endowed with it.

Thus we can anatomize the apparatus for the provision of energy in muscle into elements which are independent, elements which are not inevitably brought together in every case, elements which fall into classes of basically functional and of superadded elements which serve to moderate the imperfections of the basic apparatus in fashions which are sometimes co-operative but sometimes independent.

Note that if cellular organisation is of this kind, then there is a strong probability that the superadded elements will introduce properties that are disadvantageous in some conditions, and keep in mind at all times that unless you are prepared to take the position that evolution is over and that neither man nor any animal whose physiology has been thoroughly studied is susceptible to modification capable of adapting the organism more fully to its present environment or to any environment at the fringe of its habitat, then you must conclude that in some context or other the organism has imperfections.

The simple explanations that we all need to enable us to focus on physiology are only a beginning of our study. If we accept these neat notions as true physiology then we are accepting a picture of man as man would have designed him had he had the forethought, and, at that, this is a picture of a design that has not been test-flown.

National Health Service—Some Achievements

By HUGH A. RAEBURN, S.A.M.O., South Eastern Regional Hospital Board

The National Health Service has now been in existence for over fifteen years, and is firmly established as one of the features of Twentieth Century Britain. There are many criticisms of it and many defects and deficiencies; one has only to read the newspapers—particularly the correspondence columns — to have them pointed out. However, it is good to look at the service's successes and achievements, not in any sense of complacency, but rather in the spirit of Sir Winston Churchill's famous minute, written in 1942, on the possibility of establishing artificial harbours on the Invasion Beaches of Europe:—

> "Let me have the best solution worked out. Don't argue the matter; the difficulties will argue for themselves."

The National Health Service consists of its three branches — the Hospital Service, the Environmental Services for which the City, Burgh and County Councils are responsible, and the General Practitioner service. If it is the achievements of the Hospital Service which are discussed, it is because the writer's experience has been in this field. All three branches have their own and equally important contribution to make.

To understand the achievements and difficulties of the Hospital Service, the background against which it was initiated and developed must be appreciated. The present century is a time of change, two features of which are of particular relevance:—

(a) The development of a social conscience, with the acceptance by all that the State has a responsibility for providing help for its less fortunate citizens, culminating in the concept of the Welfare State with its health service, education, unemployment benefit, insurance benefit and the like. As far as the sick person is concerned, the concept of Welfare State means that everyone should have the treatment necessitated by their condition, irrespective of means, and that illness should not impose an undue financial burden on any family.

(b) Since Lister first introduced antiseptic methods into surgery a hundred years ago, the advance in the scope of medical science has been both dramatic and revolutionary, and advances and discoveries have been most striking in the last twenty-five years. Diseases are now becoming amenable to investigation and treatment where, not so long ago, they were closed books. Medicine has made great use of the advances in the basic sciences and technology. The various methods now in use call for different skills, but the common factor of all is the technical complexity of modern investigation and treatment, calling inter alia for highly trained and experienced staff, and highly specialised and expensive equipment.

The increasing complexity of medical treatment has inevitably resulted in its becoming very costly. The Annual Hospital Service Budget for the United Kingdom is now of the order of £648 millions (for Scotland alone £74 millions). No body but the State could face such expenditure.

It is perhaps not always realised what the cost to patients in hospital would be if they had to meet it themselves—e.g., in a general hospital the average cost per patient per week is £29; in a teaching hospital this rises as high as £33. The cost of treating a patient in highly specialised units is even more.

There may be nostalgia for the days of the voluntary hospital and the voluntary system in general, but there would seem to be no real alternative to the provision of the Hospital Service as a State responsibility. Acceptance by the State of the responsibility for services started by voluntary effort is not, of course, a new thing, and has happened before; even the Army and Navy were at one time provided by private enterprise! In view of the scale of this "take-over" operation, one achievement is that the National Health Service has worked.

The transfer of hospitals to the ownership in England of the Minister of Health and in Scotland of the Secretary of State was a revolutionary change, in some ways comparable in magnitude to the changes following the Dissolution of the Monasteries in the time of Henry VIII. One achievement is that this revolutionary change has been of evolution rather than revolution. The Hospital Service demonstrates the British genius for compromise. Many of the features of the old system are retained. In particular, although the hospitals now form a part of the National Health Service, the concept of voluntary service has not been lost. Voluntary hospitals were traditionally administered by Boards of public-spirited citizens serving without payment, as also did members of Local Authorities. It is perhaps not sufficiently realised that members of Regional Boards and Boards of Management serve without payment, and in an article such as this on the achievement of the Hospital Service, mention must be made of those men and women, all of them busy, who continue to contribute so much of their time and varied experience to this humanitarian work.

Another striking feature of the Hospital Service is that the continuance of the interest taken by people in different areas in their hospital has been maintained, and this is demonstrated by legacies being left, and donations made, to the hospital. It should be noted that the State, when taking over the hospitals with all their liabilities, did not take over the sometimes very large sums of money held as Endowment Funds. In Scotland a special Commission reviewed and redistributed these funds. Part was allocated to the different hospital groups, with re-allocation between those groups to enable all to benefit. This provides an annual income which can be expended as thought fit in the general interest of the patients, thus enabling amenities and comforts to be provided—the "extras" which contribute so much to the welfare of patients. The other part of these funds is administered by a body called The Scottish Hospital Endowments Research Trust and provides for the encouragement of research. In this way the intentions of the donors are honoured.

An achievement of the Hospital Service therefore, is that it has demonstrated that there is nothing incompatable between a State service and the continuation of the concept of voluntary service and effort.

Our hospitals have a varied origin. There were the great voluntary hospitals, some of which were founded centuries ago, and general municipal hospitals, usually of Poor Law origin, but transferred to local authorities under the Local Government Act of 1929. There were also hospitals established to carry out the public health functions of local authorities— e.g., infectious diseases and tuberculosis hospitals. Then again there were the mental hospitals, usually under the aegis of Local Authorities, but in some cases under private foundations. To say there was rivalry between these various types of hospital perhaps does not give a true picture. Rivalry in itself is not unhealthy, but the attitude, with of course, many notable exceptions, is better described as a "sheep and goats" attitude. One type of hospital tended rather to ignore the existence of the other, resulting in the duplication of facilities and much waste of effort. It can be claimed as an achievement of the Health Service that hospitals now regard themselves as part of one integrated service with, no doubt, a certain amount of healthy rivalry. It is thus possible to plan the most effective use of facilities, particularly important in view of the high cost of their provision.

The integration of the hospitals into one service has, it can be claimed, developed a much greater feeling of mutual respect between

medical staff in one field of medicine and their colleagues in other fields. In the past, the staff of voluntary hospitals in particular perhaps tended to look on the staff of infectious diseases and tuberculosis hospitals, and of mental hospitals, as different from themselves. No doubt due partly to the wise provision that conditions of service should be the same for staff in all branches of medicine, this attitude is now a thing of the past.

Reference must be made to one possible cause of difficulty which has been avoided. In spite of the fact that the State is responsible for the Hospital Service, including financial responsibility, clinical freedom has been maintained. In certain legal cases, it was decided that a hospital authority is vicariously responsible for the professional acts of medical staff. This means in effect that a hospital authority may be responsible for damages resulting from professional acts of those staff. It is to the credit of the Hospital administration that, although the hospital authorities may have to pay the piper in the way of damages, they have not attempted to call the tune in the way of laying down the treatment which doctors should provide. Of the many criticisms of the Hospital Service, so far as is known, interference with doctors' clinical judgment has never been one of them—in itself quite an achievement.

In 1948, when the National Health Service started, the Hospital Service took over a very mixed bag of hospitals. Some were reasonably modern and provided moderately good accommodation. Others can only be described as Dickensian. Two examples must suffice. In one hospital the nursing staff were accommodated in cubicles at the end of greatly overcrowded and poorly decorated wards; in another large hospital, dealing with acute patients, there was no X-ray plant, patients having to be sent to an X-ray department a number of miles away.

During the early years of the service, most of the money available for building had to be used on a "make do and mend" basis to enable the existing hospitals to continue in use at reasonable standards. The situation has now changed, and hospital authorities are now devoting most of their funds to major schemes involving the provision of entirely new hospitals. The task is a complex one, and those concerned have had to gain their own experience. There is now a very much better appreciation of such factors as the demands on hospitals and the matching of facilities to be provided.

One success of the Health Service is the quite considerable improvement in the standards of hospital accommodation and equipment—already achieved. And the stage has been set for quite outstanding developments in the design of hospitals—some buildings completed and others being planned.

A hospital is a most complex organisation, and quite apart from the care of patients, has to deal with many of the traditions of a big business organisation. An achievement of the Health Service is the development of team work by the members of many different professions and trades. In the care of patients, the part doctors and nurses play in hospitals is well known. The important role of administrators, accountants, finance officers, engineers, physicists, biochemists, physiotherapists and occupational therapists, electricians and many others is not always appreciated. For example, in one complex operation, the skills of persons with twelve different professional backgrounds were required.

It can be claimed that the National Health Service has some considerable achievements to its credit. Some may say that such achievements are mainly in the abstract realm of "attitudes" and hypothetical "might have beens". Be this as it may, attitudes and similar abstractions have a profound influence on the working of any service. They are very long-lived and in many ways are more difficult to alter or correct than material deficiencies.

In conclusion, reference must be made to the problems which the Health Service has still to tackle. These are (a) shortage of, and the need for improvement in, accommodation and equipment; (b) organisational difficulties to ensure the best use of facilities available. It is obviously wrong for a patient only requiring simple treatment to be treated in a hospital with many and extensive ancillary departments available.

There is one problem which will be always with the Health Service—as it is with any other service—conflicting demands. It never happens that the availability of staffing, equipment, finance, etc., is sufficient to meet the demands on them; the assessment of priorities will remain one of the most difficult problems of the Hospital Service.

SURVIVAL IN COLD WATER

From a dissertation to the Society on 18th October, 1963

By J. R. ROBERTSON, Esq., B.Sc.

There is a note in a paper written in 1943 by Kritchley which mentions that after a ship had been torpedoed in an area where the water was 29°F, only one man out of a crew of ten survived longer than half an hour in the water.

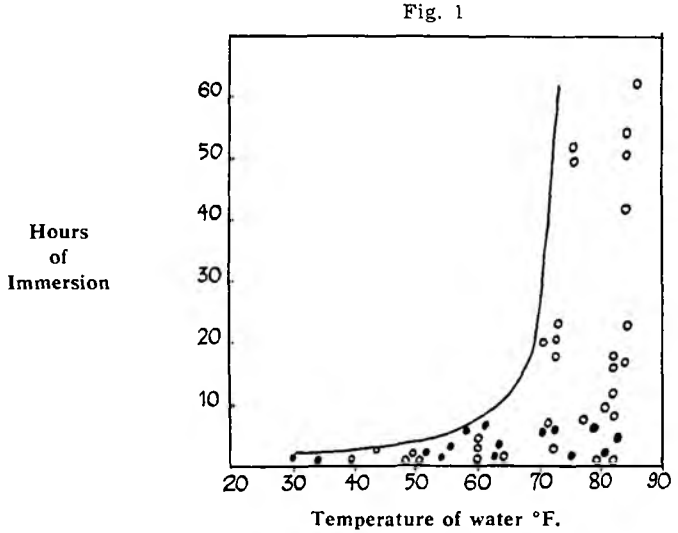
Losses during the war resulting from cold immersion were catastrophic, accounting for perhaps two-thirds of the people who lost their lives at sea.

In 1945 this question was reviewed by Molnar. He collected a number of records of incidents involving immersion in cold water from the files of the Bureau of Medicine and Surgery U.S. Navy, Washington. He chose those records which included the temperature of the water, or an accounted note of the date and locality so that the temperature could be read off from maps, and some indication of the duration of immersion.

Each of these records is represented by a point on Fig. 1. The continuous line is drawn above all of the points, and it indicates very roughly the point of demarkation between survival on one side and death on the other. Each point below the line represents an individual who has survived. This measure, of course, cannot be expected to apply in all cases. It is only a rough guide. Many other factors may contribute to shortened survival such as drowning in heavy seas, fainting, exhaustion and injuries. Molnar points out that the line represents a hyperbola. This indicates perhaps that the product of the deficiency in water temperature and the survival time equals a constant. That is to say death occurs once a *critical body temperature* has been reached.

Alexander reports on cold immersion trials carried out in Dachau concentration camp.

Fig. 1



These trials are of limited value because the height and weight is given in only five of the subjects used. The subjects in question were clothed in aviators garb; that includes headgear and a rubber or kapok life-jacket. Fig. 2 shows the results of thirty-six experiments. The white dots represent subjects who survived the immersion, whereas the black dots represent twelve subjects who perished. Individual variation is marked. The chart includes the results of another experiment carried out by Spealman. The subjects were immersed in water as low as 50°F, the lowest temperature that he considered safe. From the results he calculated a "safety limit" which is represented by the lightly dotted line. The heavily dotted line is a rough average of the Dachau results. The continuous line is taken from the previous chart, and evidently represents the outside limit of tolerance. Few will survive longer than it indicates. One hour in water at 50°F can be expected to kill 50 per cent. of people. This is a generalisation, but provides a figure that is easily remembered. Conversely it could be said that few more survive a drop in rectal temperature below 78.5°F.

Fig. 3 is based on the fall in rectal temperature over the first hour of immersion at various water temperatures. It is based upon Spealman's figures, with the Dachau figure marked in the circle at the top of the graph. In water at 75°F the fall in rectal temperature is 2°F per hour, at 55°F it is 5°F per hour, and at 45°F the fall is about 12°F per hour. For succeeding hours of immersion this cooling rate can be produced in a linear fashion until the lethal temperature is reached. There seems to be a number of people who survive rather longer than these measurements suggest. In such cases the cooling, subsequent to the first hour, probably occurs exponentially, and not in a linear fashion. Perhaps the same subjects can withstand a greater fall in rectal temperature than is considered usual. This, however, is only surmise.

If, on the other hand, the temperature of the water is reasonably warm, that is to say above 60-68°F, the rectal temperature falls to a new level within the first hour, achieving a new equilibrium. Heat production is stepped up by three to six times the usual rate in response to the cold, and soon keeps pace with the heat-loss, albeit at a lower internal body temperature. This explains why there is a such a very sharp increase in the number of people that survive long hours of immersion once the temperature gets above 60-68°F (Fig. 1). In colder water no equilibrium is reached.

In water the body cools at just twice the speed that it would in air at the same temperature, although water conducts heat twenty-five times faster than air. The limiting factor is the conductivity of the body, which cannot be reduced below a certain level once vaso-constriction is complete, then the internal gradient of temperature will depend upon such factors as body-build and the clothing worn. The greater heat-loss in water is also contributed to by the following factors. First of all there is a greater effective surface area presented by the body to the water; this is because one leg cannot exchange heat with the inside of the other, nor the arm with the lateral chest-wall. Secondly the layer of water immediately surrounding the body has a greater specific heat than a similar layer of air. And thirdly the water-layer is more readily disrupted. These factors cause the external surface temperature of the body to be lower than it would have been in air at the same temperature. The internal heat gradient is therefore steeper, and heat-loss will therefore be quicker. In water at 60°F and below, the internal body temperature continues to fall. When it is below 90°F you reach the paradoxical situation of heat production being impaired for want of heat energy. At 80 to 88°F it is barely at the B.M.R.; cardiac and respiratory irregularities occur, which if allowed to continue, lead to death.

There are certain factors which influence the maintenance of body temperature in the water, some of which were investigated by Keatinge in 1959. The question of work is quite important, because up to then it was not known whether the survivor should be encouraged to swim around vigorously in the water or not. The results produced were quite interesting. They found that in water at 5°C and 15°C, work tended to quicken the fall in rectal temperature. This was so whether the men were clothed or not, whether they worked as hard as possible or only a little, and whether the water was vigorously stirred or not. In water that was above 25°C the opposite was found. Work reduced the fall in body temperature and was presumably beneficial. But since water at this temperature is far less dangerous anyway it does not matter so much. The musculature was in better condition after work, and

Fig. 2

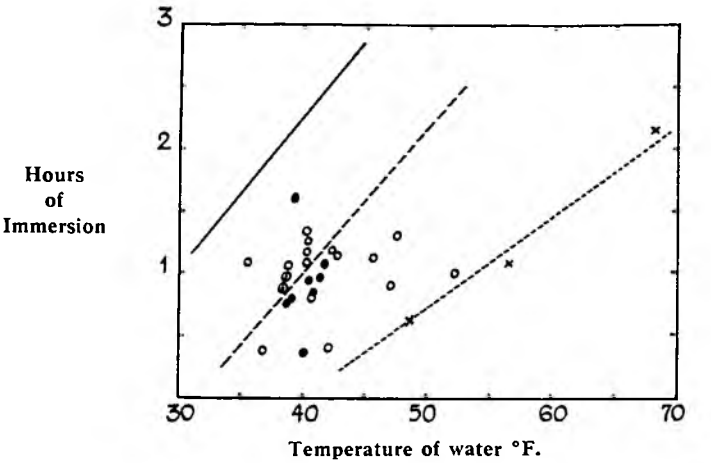
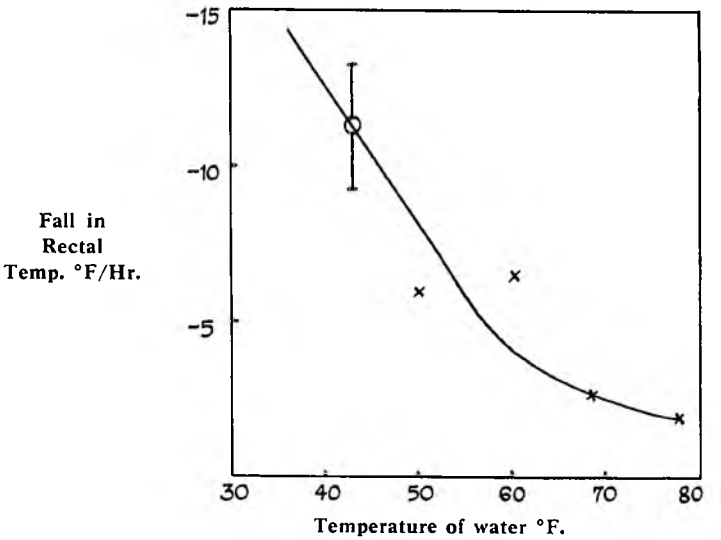


Fig. 3



this meant that they could climb on to a raft more easily.

As regards the effect of clothing, the deep body temperature did not fall as rapidly in the clothed subject as in the unclothed, especially when they did not move. This was also true of the superficial temperature. The standard clothing was not waterproof.

Three other items were investigated; meals, previous exposure and the drug hyoscine. If a heavy meal had been taken three-quarters of an hour beforehand, then a greater risk of developing cardiac irregularities occurred, especially in very cold water. Previous exposure made little difference. It appeared that whether a man was warm or cool, had taken exercise or remained still before being immersed, the effect upon the falling rectal temperature was only very slight. Those who were hot when entering the water cooled more rapidly, but remained slightly warmer than the others throughout the experiment. Hyoscine produced quite definite hyperventilation in some cases, explaining perhaps the muscle-cramps that were experienced by one subject. The same man did not have cramp at any other time. Since hyoscine is the standard remedy for sea-sickness, this may present a hazard, but not a major one.

The question of hyperventilation brings us on to another problem. That is the changes in alveolar p.CO_2 which are produced in a subject immersed in cold water. Suggestions have been made that the alveolar p.CO_2 level became dangerously high in a man who worked hard in cold water.

This is quite obviously untrue. On the first occasion when men sat unclothed in the water at 15°C their end-tidal p.CO_2 decreased by an average of 1.8 mm.Hg. in the first four minutes. At 5°C the decrease was 4.5 mm.Hg. These are end-tidal measurements, and represent an even larger decrease in alveolar p.CO_2, perhaps to the extent of endangering the normal working of the ventricles. However, this situation had approximately righted itself by the end of a twenty-minute period. Extra systoles were recorded on a number of occasions. In one case they were multiple. This happened in water at 15° and below. It is tentatively suggested that ventricular fibrillation is one cause of sudden death on immersion into cold water.

By far the most spectacular finding was the cooling-rate of the body in relation to the thickness of the subject's sub-cutaneous fat. Fig. 4 shows the fall in rectal temperature in degrees Centigrade during a thirty-minute immersion in water at 15°C. The abscissa is the reciprocal of the thickness of a fold of skin. A number of measurements, using calipers, are made from specified sites on the body, and the mean of these is the value used. On the one hand a man whose skin-fold thickness was 10 mm. only loses 0.5°C in half an hour, whilst on the other a man whose skin-fold thickness is only 5 mm. loses four times as much—that is 2°C in half an hour.

The problem of immersion in cold water is part of the larger problem of survival. Having realised the full extent of the threat, it has been investigated and certain facts are emerging. For example, the rapid loss of heat from the body is made worse by movement. The importance of clothing and fat in protection against the cold, has led the Navy to experiment with special rubber "Survival Suits", which can be put on in a matter of 20-30 seconds before jumping into the water. They are worn over the clothing, thus making sure of a thick layer of insulation. The design has not been perfected yet, but tests carried out with a number of these suits have given very encouraging results. Meanwhile the danger remains very real. It is rather horrifying to think what would have happened in the recent sea-disaster, had the sea temperature been the same as that surrounding the iceberg which sank the "Titanic".

Medical Research Council: Reports prepared for the Royal Naval Personnel Research Committee—Survival at Sea Sub-committee.

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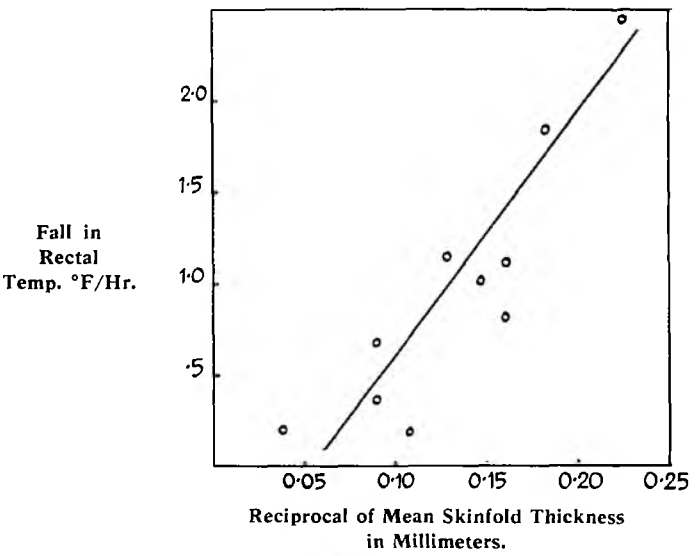
Fig. 4



RES MEDICA

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On Wednesday, February 19th, the triennial meeting for the revision of laws was held and as usual the question of admitting ladies was brought up. But this time, something unusual happened. By an overwhelming majority it was decided that ladies be admitted to the public business of the society. So it was that ladies were present at Professor Sir Dugald Baird's address on the following Friday. It must be noted that on two or three previous occasions ladies have in fact been present at meetings of the society, so that precedent had been set for this decision.

The society must now decide on the admission of ladies to full membership, and a motion proposing this will be brought before the society at the start of the next session. If ladies are to be admitted to full membership, this motion will have to be passed by a two-thirds majority at two consecutive business meetings. The widest publicity will be given for these meetings, and the first will take place at private business on the night of the inaugural address of the 228th session, in October of this year. It is hoped that a really good attendance will be registered at these important meetings.

We note that the Royal Charter is worded so that 'any person' may become a member of the society, and that the exclusion of ladies from membership has never been an actual law of the society—the society seems merely to have neglected the existance of the opposite sex. Our own view is that ladies should have been admitted long ago, and we hope that the society has the courage to drop its inhibitions and learn to come to terms with the facts of life. We acknowledge that some people view the admission of ladies as a disaster, and we respect this view when reasonably expressed. Our editorial opinion is not necessarily that of the society at large, though we do try to express the opinion of the majority of active members.

The society has enjoyed a most interesting session, with the new regulations allowing much more time for the discussion of medicine by the streamlining of the business meetings, and the reduction in their number. Six evenings per term have been devoted entirely to medical topics, and the pre-clinical first private meetings have proved quite an attraction to both junior and senior students.

An excellent visit to Peel Hospital was made on February 5th, the outing being very well attended. The annual film show was held the same evening, and this was also a great success.

It seems that the society will have to leave Melbourne Place at the end of the next session. The new hall will probably not be built for some years, and so we will be in temporary premises for possibly ten years. In the meantime, the Royal College of Surgeons has most generously offered us the use of premises at 3, Hill Square; these rooms will provide storage and office space. We are extremely grateful to the Royal College, and look forward to making good use of their hospitality.

The Appeal Committee also deserve our thanks, wider publicity is deserved for the immense amount of work they have done for the society. Under the dynamic leadership of Sir Derrick Dunlop, this committee is ensuring that the future of the society will be as great as its past.

MENTAL HOSPITAL NEEDS AND BEDS

On April 1st, 1961, in the Lancet, the trends in mental hospital populations were subjected to statistical analysis,* and the conclusion was drawn that the number of mental hospital beds in England and Wales could be reduced to nearly half, by 1975. The authors of the analysis were a medical officer of the Ministry of Health and a Government statistician, and the present intention of the Ministry of Health is to act on their recommendation.

The authors had attempted to estimate the numbers of mental hospital beds which would be required by short-, medium- and long-stay patients by 1975. The estimate for long-stay patients was necessarily a statistical projection, involving the run-down of present residents and the build-up of a new, chronic population. In the cases of the rundown of present long-term residents, they estimated that all would be dead or discharged by 1975. This is certainly at odds with the expectations of clinicians. A letter to the Lancet soon after pointed out that while the figures used to support this statistical projection were interpreted as for a straightline graph, in fact the figures formed a curve, with all the patients dead or discharged some time long after 1975.

The analysis can be criticised on other grounds. The figures for admissions and discharges were taken from the late 1950's, which years were atypical in that an upsurge of interest in the rehabilitation of chronic patients was taking place, and most patients capable of discharge were discharged at that time; again, "tranquillisers" were at that time coming into vogue and allowed a temporarily high discharge rate. More topically, the proposed reduction takes little count of the rapidly rising readmission rate.

Anyway, on the basis of this analysis, the Hospital Plan for England and Wales (1962) envisages a reduction in mental hospital beds of from 3.3 per 1,000 of the population in 1961, to 1.8 per 1,000 in 1975. By contrast, the Hospital Plan for Scotland (1962) describes a recent run-down in mental hospital bed requirements of about 1% per year, but declines to quote a figure for future requirements.

The patients to be discharged will be largely chronic schizophrenics. They might be discharged to their families, to lodgings, or to a hostel run by the local authority. Experience has shown it most unlikely that a significant number will be accepted by their families. In Edinburgh, an attempt to form a list of lodgings willing to accept mental patients failed entirely. So the answer can lie only in providing hostels.

In this respect the local authorities are in no way ready to cope with a greatly increased number of mental patients living in the community. In 1962 there were in England and Wales 18 hostels for the mentally ill with a total of about 350 places. Scotland has only one hostel, in Aberdeen. Another will soon be opened in Edinburgh. The provision of suitable buildings, suitably staffed, is painfully difficult, and expensive.

Estimates as to how many such hostels will be required vary widely. If patients are to be transferred from hospital to hostel on the scale made necessary by the Hospital Plan, then up to 150 mental patients per 100,000 of the community will have to be reaccommodated.

In short, the plans of the Ministry of Health are ambitious, but may well be based on misleading data. Unfortunately, a prophecy such as this one can be self-fulfilling—bed requirements will certainly seem to fall when physicians can read that their hospitals are scheduled for closure. It may be that the result of governmental over-optimism is already becoming obvious; in some areas patients over-promptly discharged are returning to hospital via the police courts.

* Tooth, G. C. & Brooke, E. M. 1961, Lancet *1*, 710. "Trends in Mental Hospital Population and their effect on Future Planning."

The Status of Antibiotics

By J. A. RAEBURN

From a dissertation read before the Royal Medical Society on Friday, 1st November, 1963

History of anti-microbial therapy [1]

Many attribute the earliest recognition of an antibiotic effect to Sir Alexander Fleming. However, in 1877, 50 years before Fleming's discovery, Pasteur and Joubert described the phenomenon of bacterial antagonism; the process whereby the growth of certain species is inhibited in the presence of others. In the particular case of the anthrax bacillus they found that growth was inhibited in cultures contaminated with 'common bacteria' (those types now known as the Enterobacteriaciae). Shortly afterwards the term "antibiosis" was introduced for such antagonism.

The problem that faced these early workers was to discover substances with selective toxicity—substances which destroyed bacteria in concentrations having no effect on the cells of the body. Without this selective action, an anti-microbial substance is no more than an antiseptic. In medicine today it is important to remember this distinction. It is difficult to justify the use of antibiotic sprays to disinfect surgical wards, a use in which it cannot be said that selective toxicity is required.

In 1928, Sir Alexander Fleming recognised the effect on the growth of staphylococci of Penicillium notatum, a fungal contaminant. The mould had caused the lysis of the surrounding staphylococcal colonies. Ten years later Florey and his co-workers published the first paper on the clinical use of penicillin.

Since 1939, increased research into the development of antibiotics has resulted in over a dozen being available for clinical use. We must remember, however, that for each antibiotic that has found a place in therapeutics today, there are many hundreds that were isolated but which were subsequently found to be too toxic for clinical use.

Present clinical problems

When a clinician decides to treat an infection with antibiotics two problems face him; firstly drug resistance and secondly drug toxicity.

A. Drug Resistance

It is convenient to consider bacterial resistance to antibiotic action as being either congenital or acquired. We could regard those bacterial species outwith the spectra of individual antibiotics as being congenitally resistant to such drugs. Treatment of infections whose causative organisms show such resistance to the antibiotics in use, is doomed to fail.

More pressing at the present time is the acquired resistance of bacteria which were originally susceptible to given antibiotics. What is the nature of such resistance? What changes occur in bacterial structure to cause it? A rational approach to this problem would be to determine the precise modes of action of all antibiotics and to investigate the changes occurring as resistance develops. The table below summarises the likely modes of action of some antibiotics in common use.

ANTIBIOTIC	*MODE OF ACTION*	*TYPE OF ACTION*
PENICILLIN BACITRACIN	Inhibits cell wall synthesis	BACTERICIDAL
STREPTOMYCIN	Interferes with carbohydrate metabolism	BACTERICIDAL
CHLORAMPHENICOL	Interferes with protein synthesis	BACTERIOSTATIC
TETRACYCLINES NOVOBIOCIN ETC.	????	BACTERIOSTATIC

Since precise knowledge of the mechanism of antibiotic action is lacking, still less is known of the nature of the changes occurring to drug resistant types. Two opposing theories [2, 3] have been suggested. These have implications that are fundamental to the rational treatment of infection.

I. The Genetic Theory

This theory states that in bacterial populations, mutants which are less susceptible to the drug arise spontaneously, and that the production of these mutants is independent of exposure to the drug. Subsequently they thrive at the expense of the more sensitive strains if their environment contains quantities of the drug.

II. The Adaptive Theory

In this theory it is postulated that the drug is a direct stimulus to the development of resistance.

If we are thinking, as we should be, of the status of antibiotics in future years, it is the second theory that gives more cause for optimism. For if the use of antibiotics were restricted this would lessen the stimulus to the development of resistance. Conversely, if resistant strains arise despite restricted use of antibiotics, there is cause for concern.

The evidence for each theory cannot be included here, but most workers agree that the genetic hypothesisis is more able to explain certain accepted facts. In accepting this expert view, we should realise that the same mechanism need not operate in all cases. To support such a compromise, we need only think of the different patterns of developing resistance. For example with the tetracyclines, the first stage in the development of resistance forms strains that are resistant to only slightly increased concentrations of the drug. Subsequently resistance develops to higher and yet higher concentrations. By contrast, the resistance which develops to streptomycin may '*ab initio*' be of uniformly high level.[4] The pattern of developing resistance differs; it is likely that so too will the mechanisms of developing resistance.

Taking account of both theories, we can construct sensible rules for the antibiotic treatment of the individual patient. Moreover rational therapy benefits not only the individual but also the hospital community, for fewer drug resistant strains arise.

Guiding rules for antibiotic therapy

I.

To be most effective treatment must be started early in the course of the infection before many organisms and hence many resistant mutants have developed (genetic theory). Here a balance must be struck, for treatment must often be delayed until the sensitivity of the caustative organism is known. The following table shows a number of diseases caused by micro-organisms that have a consistent susceptibility to antibiotics. In such cases early treatment can be instituted on the strength of a clinical diagnosis (see table ii).

A corollary to this first clinical rule would be that in chronic infections of the lungs, the urinary tract, etc., little will be gained by hasty 'blunderbus' treatment. In such situations, two or three series of careful bacteriological investigations may be required before rational antibiotic therapy can begin.

II.

When the chosen treatment is started, there must be no delay in providing effective tissue levels of antibiotic. (Both theories.)

III.

Levels of antibiotic above the minimum inhibitory concentration (MIC.) of the infecting bacteria must be maintained long enough for all the causative organisms to be eradicated (adaptive theory).

If the drug concentration at any stage in treatment is below the MIC of the bacteria, not only is growth enabled to continue, but further, bacteriological evidence indicates that some stimulation of growth may occur. (See plate.)

IV.

Antibiotic must be eliminated from the body as rapidly as possible after successful treatment:

a) In order that the normal bacteriological environment is quickly restored.

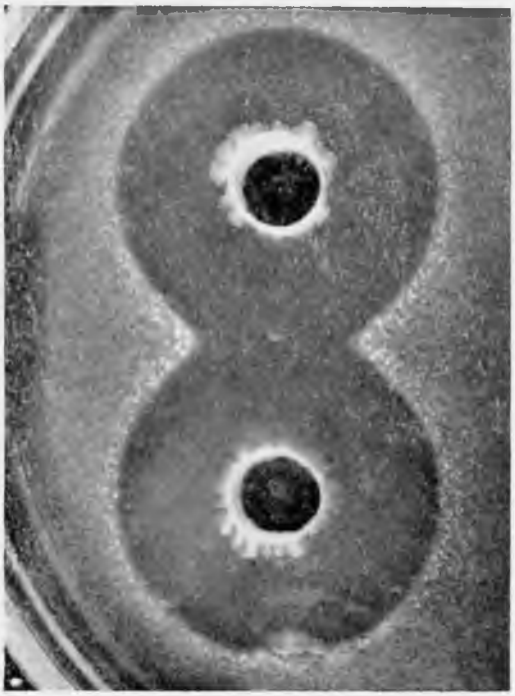
b) Lest the results of clearance tests are falsified.

e.g. After the oral treatment of dysentery, antibiotic levels that are significant may persist in the faeces for some time. In such cases, certain patients are declared free from infection although they still harbour the causative organism, temporarily masked by high residual levels of antibiotic.

CLINICAL DIAGNOSIS	*USUAL CAUSATIVE ORGANISM*	*APPROPRIATE ANTIBIOTIC*	*DURATION OF TREATMENT*
Lobar pneumonia Erysipelas Scarlet fever	Pneumococcus Haemolytic Streptococci	Benzyl penicillin 100,000 units/6 hours	5 days
Enteric fever	Salmonella (typhi and paratpyhi)	Chloramphenicol 250mg./6 hours or Ampicillin[6] 1 gm./6 hours	14 days 14-28 days
Typhus fever	Rickettsiae	Tetracyclines 250mg./6 hours	7 days
Brucellosis	Brucella	[illegible]	14 days
Meningitis	a) *Haemophilus influenzae	Chlorampyenicol 250mg./ 6 hours	5 days
	b) *Pneumococcus	Benzyl penicillin 20,000 units intrathecally +250,000 units intramusc	7 days
	c) *Meningococcus (dose for child of 1½ yrs.)	Sulphadiazine 500mg./6 hours Benzyl penicillin 250,000 units/6 hours	5-7 days

* Distinguished on the gram-stained film of the CSF.

Table ii



This photograph shows part of an agar plate on which staphylococci have been evenly distributed. Centrally two 8mm. cups have been punched out and in each a known amount of streptomycin has been placed. Diffusion of the drug through the medium has caused inhibition of the growth of the staphylococci inside large rings surrounding the cups. The rings of inhibition are sharply demarcated by zones of increased growth. At this point, the antibiotic concentration is just below that which causes inhibition of growth. If such sub-inhibitory concentrations of drug exist in the body, then similar increased growth may occur. The maintenance of adequate levels at the site of infection is of especial importance when using bacteriostatic antibiotics.

Having discussed the phenomenon of drug resistance and how it affects the treatment of individual cases, let us examine the 'natural history' of resistance in the community. The following table shows how the percentage of resistant strains of Shigella sonnei has changed in the last seven years. The figures quoted are the approximate values for the Edinburgh area.[9]

Therapeutic agent	Percentage resistant	
	1955	1962
Sulphonamides	97%	98%
Streptomycin	2%	27%
Tetracyclines	0%	3%
Chloramphenicol	0%	0%

It is readily seen that in the case of the first three substances there has been an increase of resistance. There has been no increase in resistance to chloramphenicol, for in this area the physicians, well aware of this drug's tragic side effects, do not use it in the treatment of dysentery. In the Glasgow area, where more chloramphenicol is used, the percentage of resistant strains is now about 2%. It is difficult to deny the obvious conclusion that with increasing use of antibiotics in a community, the proportion of resistant strains increases.

B. Antibiotic Toxicity

The vital property of anti-microbial agents for parenteral use is selective toxicity. No matter how great this is, it is certain that all antibiotics at present known will in some situations have toxic effects, the degree of severity of these being dependent on the level in the body. Although the many possible therapeutic disasters that could be caused by antibiotics cannot be elaborated here it must be emphasised that no antibiotic can be administered without the danger of ill effects. The deduction follows that a simple rule must be applied before ordering a course of antibiotic therapy:—

Antibiotics should only be used in severe or potentially severe diseases.

The use of highly toxic antibiotics can only be justified if such use is deemed to be life-saving, and if adequate facilities exist for the estimation of blood levels of the drug. It is depressing to think of the fatal marrow aplasias that have followed the empirical treatment of minor catarrhal conditions with chloramphenicol.

It has been suggested that the severe effects of the common cold can be forestalled by the prophylactic use of antibiotics, and that since this illness affects millions of people the result of such prophylaxis would be of great economic significance. However, as a result of the widespread use of antibiotics for this purpose, the secondarily invading bacteria would soon become resistant to the antibiotics employed. The economic benefit would be short-lived. Serious thought must be given to the question of treating such minor complaints.

SUMMARY

In this brief review the attempt has been made to pose certain questions regarding the policy for the use of antibiotics today and in future years. There was no space to consider still more controversial subjects such as the prophylactic use of antibiotics[5,7] or the value of antibiotic combinations.[8] The rational use of these drugs is not simple, and if further indiscriminate usage continues, the problems of the future will become still more alarming. Until now, the development of new antibiotics has kept pace with the steady increase in resistance to those used currently. It is foolish to suppose that the development of new drugs will continue. Our legacy, from the previous generation has been one of powerful drugs which can cure the most severe infections. If we misuse this, the legacy of future generations will be one of multiple drug resistance in infections more terrible than any we at present know.

ACKNOWLEDGMENT

In the preparation of this article I have had much assistance from the staff of the Infectious Diseases Unit, the City Hospital. In particular I must thank Dr. J. McC. Murdoch who has given me invaluable support and encouragement.

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PHYSICAL SIGNS IN THE CHEST

Part II

By PROFESSOR JOHN CROFTON

THE SIGNIFICANCE OF PHYSICAL SIGNS IN THE CHEST

In this section some of the causes of the common physical signs are listed.

Mediastinal shift.

Mediastinal shift to one or other side is detected by *deviation* of the *trachea* or of the *heart*. Naturally the trachea is more often deviated by abnormalities in the upper part of the chest, or occupying the whole of one side of the chest, and the heart by abnormalities in the lower part of the chest. If the heart is deviated to the left the apex beat will be deviated to that side. If there is no obvious cause for enlargement of the heart one can provisionally assume, depending on the later detection of consistent physical signs, that the deviation is due to mediastinal shift. It is less easy to be sure of deviation to the right, especially if the abnormal physical signs are present in the right lower zone and dullness at the right base makes it impossible to detect the right border of the heart. Inward deviation of the apex beat, unless it is gross, might be due to the patient having a relatively small heart. In this case initial impressions may have to be confirmed by x-ray.

The mediastinum may be *pushed* or *pulled* to one side.

Pulling of mediastinum to one side: It will be concluded that the mediastinum is pulled to one side if the abnormal physical signs in the chest are found on the same side. The common causes, in order of frequency, are:

(1) *Collapse of the lung*
(2) *Fibrosis of the lung*
(3) *Old pleurisy or empyema*: This requires a little elaboration, as this cause is relatively unfamiliar. If a patient has a large effusion or empyema, sometimes gross thickening of the visceral pleura develops over the lung collapsed down by the fluid; this exudate may later organise, so that the lung is covered by a cuirass of thickened pleura. This cuirass prevents the lung re-expanding when the fluid is absorbed, so that the thoracic space can only be occupied by deviation of the mediastinum and pulling in of the chest wall. In these cases deviation of the trachea is accompanied not only by deviation of the mediastinum to the appropriate side but also by considerable flattening and immobility of the whole chest wall, the so-called "frozen chest".

Pushing of the mediastinum to one side: In this case the abnormal physical signs will be found on the side opposite to that to which the mediastinum is deviated. In order of frequency, the causes are:

(1) *Pleural effusion*
(2) *Pneumothorax*
(3) A very large mass: This is relatively rare but the trachea is sometimes deviated by a tumour, cyst or aneurysm in the upper mediastinum or, very occasionally, in the upper lung.

Dullness to percussion.

Common causes are:

(1) *Consolidation of the lung*
(2) *Pleural effusion*
(3) *Collapse* of the lung
(4) *Fibrosis* of the lung

Less common causes are:

(5) *Thickened pleura*

(6) *Raised diaphragm*: This may be due to an abscess or tumour in the liver or to subphrenic abscess. The liver dullness is often raised in obese people, but then both diaphragms are raised and there is relatively symmetrical dullness at both bases.

(7) *Tumour*: Tumours or masses in the lung are seldom large enough to give rise to dullness, except by blocking a bronchus and producing collapse, or by giving rise to pleural effusion or pleural invasion. Nevertheless, occasionally a large tumour may give rise to dullness. The relatively rare primary tumours of the pleura may also result in dullness to percussion.

Bronchial breathing.

Common causes are:

(1) *Consolidation* of the lung: Classically this occurs in lobar pneumonia, although signs of patchy consolidation may be found in bronchopneumonia. Fibrosis may sometimes be dense enough to cause bronchial breathing and actual consolidation may of course be due to a pulmonary infarct. In the latter it is commoner for there to be diminished breath sounds but classical signs of consolidation are sometimes found.

(2) *A thin layer of pleural fluid*, sufficient to cause collapse of the superficial alveoli but not of the bronchi so that the bronchial sounds are conducted to the surface. An early pleural effusion may simulate the classical physical signs of consolidation, with dullness and bronchial breathing at the base. At this stage the fluid is not sufficient to cause deviation of the mediastinum. Within 48 hours the fluid has usually increased sufficiently for there to be diminished breath sounds and diminished vocal fremitus at the base, the bronchial breathing may then be lost or be heard only over the upper and thinner part of the effusion in the midzone of the chest. The mediastinum may also have become deviated to the opposite side.

Less common causes are:

(3) *Collapse* of the lung, when this is not due to blocking of a main bronchus but of the smaller peripheral bronchi. The bronchial sounds may then be carried through the collapsed lung to the surface.

(4) *Pneumothorax*: It is much more common in pneumothorax for the breath sounds to be merely diminished but occasionally, as mentioned above, breathing of the cavernous or amphoric type may be heard.

(5) A large *cavity* in the lung: As already mentioned, it is rare for a cavity to give rise of itself to abnormal physical signs, but if it is very large and very near the surface of the lung it may do so. If the cavity is exceptionally large the bronchial breathing may be relatively low-pitched and "cavernous"; if it is rather less large the breathing may be a little higher-pitched and of the amphoric type.

Rhonchi.

As already mentioned, the bronchi are narrower on expiration and therefore expiratory rhonchi are commoner than inspiratory, but rhonchi may be heard in both phases of respiration.

Generalised rhonchi are due to (1) *Bronchitis*
(2) *Asthma*

Localised rhonchi are due to

(1) *Mucus in a bronchus*. It may be possible to displace the mucus by coughing, thus altering or abolishing the rhonchus.

(2) A *tumour* or other cause of localised bronchial stenosis.

Crepitations.

Fine crepitations may occur in

(1) *Pneumonia*: They are usual in bronchopneumonia and occur in the later stages of lobar pneumonia.

(2) *Tuberculosis*, either localised as in pulmonary tuberculosis, or generalised, as in the very late stages of miliary tuberculosis.

(3) *Collapse*, although in collapse there is often merely diminution in breath sounds, with no added sounds.

(4) *Bronchitis*: Commonly the crepitations are medium rather than fine. Fine crepitations suggest bronchiolitis or small areas of bronchopneumonia.

(5) *Pulmonary congestion* due to left-sided *heart failure* or to *mitral stenosis*. Crepitations are virtually always present in left-sided heart failure and common in mitral stenosis.

Medium crepitations occur in

(1) *Bronchitis*, particularly at the bases.

(2) *Fibrosis*, either localised fibrosis or generalised fibrosis. The crepitations often seem to have a "sticky" quality on auscultation. Sometimes the medium crepitations are an indication of accompanying bronchiectasis.

(3) Sometimes in *bronchiectasis* although in this condition the crepitations are usually coarse.

Coarse crepitations.

These are almost always due to *bronchi-*

ectasis, although they may sometimes occur in bronchitis.

THE PHYSICAL SIGNS OF COMMON CHEST CONDITIONS

In this section will be listed not all the conceivable physical signs, as stated in text-books, but the physical signs which are most frequently present and which are most helpful in making the diagnosis.

Consolidation.

(1) *Diminished movement* of the affected side.

(2) Marked *dullness* to percussion, although this will only be present in lobar consolidation and absent with patchy consolidation.

(3) Increased vocal fremitus, sometimes useful in distinguishing from a thin layer of fluid.

(4) *Bronchial breathing.*

(5) *Aegophony* and *whispering pectoriloquy.*

(6) In the early stages of consolidation there are usually no crepitations but *fine crepitations may be present* later.

(7) A *pleural rub* is often present or the catching of the breath on inspiration may indicate the presence of dry pleurisy.

Collapse.

(1) *Diminished movement.*

(2) *Deviation* of the *mediastinum* to the *same* side.

(3) *Dullness* to percussion.

(4) Commonly *diminished breath sounds,* but sometimes, if the collapse is due to obstruction of the smaller bronchi and the larger bronchi remain open, *bronchial breathing.*

(5) *Crepitations* may or may not be present.

(6) *Aegophony* and *whispering pectoriloquy if* there is *bronchial breathing.*

Pleural effusion.

Pleural effusions, of course, may be of all sizes and may be difficult to detect if the effusion is very small.

(1) *Diminished movement.*

(2) *Deviation* of the *mediastinum* to the *opposite* side. This is only detectable when the effusion is relatively large.

(3) *Stony dullness* to percussion.

(4) *Vocal fremitus absent* or *decreased.*

(5) *Breath sounds diminished* or, if the layer of fluid is a thin one, and quite commonly over the upper part of the effusion, *bronchial breathing.*

(6) If *bronchial breathing* is present, *aegophony* and *whispering pectoriloquy.*

On the whole the *shape* of the upper level of *dullness* in pleural effusion is not very helpful. Classically this rises into the axilla and is lower anteriorly but in practice this is not very helpful. *Stony dullness, absence of vocal fremitus* and *diminished* or *absent breath sounds* are the main diagnostic physical signs.

Pneumothorax.

(1) *Diminished movement.*

(2) *Deviation* of the *mediastinum* to the *opposite* side, only if the pneumothorax is large.

(3) *Percussion note more* resonant on that side than on the opposite side.

(4) *Diminution* in *breath sounds,* which is much the most common physical sign although, as mentioned above, *cavernous* or *amphoric breathing* may sometimes occur.

(5) *Pleural clicks:* These clicks are occasionally heard in a left-sided pneumothorax in the cardiac area and in time with the beating of the heart. They are usually due to the approximation and separation of the two layers of pleura lying over the heart when these are separated only by a very thin layer of air during systole and the two layers come together during diastole. As the heart contracts the two sticky layers of pleura separate with a click.

Diminution in *breath sounds* is the only *consistent sign* of pneumothorax. A spontaneous pneumothorax is usually suspected on the strength of the history. If diminution of breath sounds is found on one side, with a normal or hyper-resonant percussion note, pneumothorax should be suspected. Deviation of the mediastinum will only be found if the pneumothorax is large. Rather quaint physical signs, such as "the coin sound", have been described in pneumothorax but are archaic relics of the pre-x-ray period.

Hydropneumothorax.

In hydropneumothorax fluid is present as well as air. The classical signs of pneumothorax may be found in the upper part of the chest and those of effusion in the lower part of the chest, usually with a well-defined upper border of dullness for the effusion; the position of this

may vary with the patient's position. The classical physical sign of this condition is "Hippocratic succussion", or splash sound. The patient is asked to sit up and his whole chest is violently shaken. The splash may be then heard either with the ear against the chest wall or with the stethoscope. If the patient is at all ill, it is less traumatic to take an x-ray!

Fibrosis.

The physical signs of fibrosis depend very much on the extent of the condition. Often fibrosis may be obvious enough on an x-ray, though no relevant physical signs have been elicited. Fine fibrosis may, of course, not even be detectable on the x-ray. Gross fibrosis may be localised, for instance after old pulmonary tuberculosis, or generalised, as in such conditions as sarcoidosis or diffuse interstitial fibrosis.

Localised fibrosis may cause

(1) *Diminished movement.*

(2) *Deviation* of the mediastinum to the same side.

(3) *Bronchial breathing* or *diminished breath sounds.*

(4) *Medium crepitations* which are often "sticky".

(5) *If* there is *bronchial breathing* there will be *whispering pectoriloquy* and *aegophony.*

Generalised fibrosis does not usually give rise to diminished movement, dullness or deviation of the mediastinum. It usually has to be diagnosed by x-ray, but there may be generalised medium crepitations.

Bronchitis.

(1) The chest may be *barrel-shaped* or sometimes *long* and *thin.* In a barrel-shaped chest the *subcostal angle* is *widened.*

(2) The *accessory muscles* of respiration may be used.

(3) Owing to the inflation of the lung, or the presence of emphysema, the *cardiac* and *liver dullnesses* may be *diminished.* The liver dullness may lie as low as the eighth rib or below. The liver may be several finger breadths palpable in the abdomen owing to the lowering of the diaphragm.

(4) *Breath* sounds may be *diminished,* particularly at the bases.

(5) There may be a *wheeze* audible with "the naked ear".

(6) *Generalised rhonchi,* most often expiratory, but frequently also inspiratory. *Crepitations* may be heard, particularly at the bases, and are usually medium in type.

Asthma.

(1) There may be signs of *inflation* of the *chest* of the same type as in chronic bronchitis.

(2) There may be *audible wheeze.*

(3) Classically there are *expiratory rhonchi,* although these may be *inspiratory* also. Sometimes, in severe asthma, there may be no rhonchi and the chest may be nearly "silent" with great diminution of breath sounds. There are usually no crepitations. However, in *cardiac asthma crepitations* are often present.

Bronchiectasis.

There may be various physical signs of accompanying underlying conditions, such as fibrosis or collapse. The classical physical sign of bronchiectasis is the presence of —

Coarse crepitations persistently present on different occasions in the same areas of the chest.

CONCLUSION.

It should be emphasised again that physical signs are only to be taken in conjunction with all the other evidence and, in any serious condition and indeed in almost any chest condition, to be supplemented by radiological investigation. Other investigations, of course, are often necessary. Nevertheless, physical signs are often a very important clue as to what is wrong with the patient. In general practice, and in hospital after hours, an x-ray may often not be available or may properly be deferred until the morning. Rhonchi and crepitations cannot be detected by x-rays and are important evidence about the condition of the underlying lung. Therefore the student is well advised to become practised in physical signs, maintaining at the same time a proper cynicism about his capacity. If one is doubtful whether something is present, it is usually wiser to assume that it is not! Do not be like one famous self-confident Chief who, as a house physician, used to infuriate his consultants by marking in ink on the x-ray the cavities which *he* was able to detect by physical signs but which the radiologist was unable to elicit by what the house physician considered an inferior technique!

Book Reviews

THE GROWTH AND DEVELOPMENT OF THE PREMATURELY BORN INFANT. C. M. Drillien. Pp. 376. Price 50/-. E. & S. Livingstone, Edinburgh, 1963.

This book describes the layout and planning of a survey to determine the effect of various factors on the baby of low birth weight. It goes on to set out the differences in the effects of many of these on the premature and normal control groups studied.

There is a multitude of data on the problems of prematurity, but for the student not adept at "table scanning" it would be helpful to have a comprehensive summary of the main findings.

In all, this is a book for the specialist, but the introductory chapter concerning definition and aims of the study, and also the difficulties and planning of such a survey make useful reading for the relatively uninitiated.

R.J.H.McL.

A PORTFOLIO OF CHEST RADIOGRAPHS. B. T. Le Roux and T. C. Dodds. E. & S. Livingstone Ltd. Edinburgh and London 1964. Pp. 366. Price 60/-.

The purpose of this book is to acquaint the student with the common radiographic abnormalities in the chest. Over 750 X-rays taken from the collection of the Regional Thoracic Unit in Edinburgh are used to illustrate common features. The emphasis is on seeing the abnormalities, not once but many times, so that the range of the normal and the abnormal can be appreciated.

Each plate is described in a brief legend which gives clinical details and also a succinct account of the main radiographic features—the type of description required of the candidate in oral examinations! The short text gives the radiological approach to each group of conditions with some mention of the type of treatment that would be necessary and the further assessment of progress. Thus the student can study radiographic abnormalities within the context of treatment and prognosis.

The quality of the plates is first-class throughout with a minimum of loss of contrast, a common fault in the reproduction of X-rays. The criticism anticipated by the authors, that the book has a surgical bias, can be offset by the fact that throughout an approach to chest X-rays is taught that is applicable to films of any condition, medical or surgical.

The price may seem excessive, but it is realistic considering the many illustrations. Expense may debar all but a few students from purchasing this book, but it shall certainly be read by many.

J.A.R.

TEXTBOOK OF MEDICAL TREATMENT. Sir Derek Dunlop, Sir Stanley Davidson, Stanley Alstead. Ninth Edition. E. & S. Livingstone Ltd., 1964. Pp. 979. Price £3 5/-.

The authors of any textbook of medical treatment have a number of difficult tasks. They must attempt to blend valuable, established methods with recent developments, and to give a balanced discussion where views differ. Full consideration must be given to important and common conditions, but the rare illnesses, so dear to the heart of the more academic physician, must not be neglected. The authors should lead the reader to a full understanding of the management of patients, yet the book should not be unwieldy, and must be up to date.

The high reputation of this text is based essentially on its success in carrying out just these tasks. The new edition is in the tradition of its predecessors. All chapters have had some alterations, and six have been completely rewritten by new authors. It is remarkable that at a time in which new editions are usually synonomous with new additions the editors have managed to shorten it by ten pages. A small change, perhaps, but one in the right direction. Undoubtedly the 'Textbook of Medical Treatment' shall continue to be a valuable source of knowledge and advice for both student and practitioner alike.

D.L.W.D.

THE EYE IN GENERAL PRACTICE. C. R. S. Jackson. Third Edition. E. & S. Livingstone Ltd., 1964. Pp. 164. Price 25/-.

This excellent little book, already well established as the most popular text amongst students in the ophthalmology class, has been further improved by the addition of a number of simple diagrams which simplify the understanding of clinical findings. It is pricipally this modification which has caused the slight increase in size. There is no doubt that these diagrams are worth many words of explanation.

A brief glossary has also been inserted, and this will be found very useful to the beginner. The text remains very much the same as in the second edition, but some revision has been made, and to keep the book down in size, some of the less important material deleted.

The illustration of branch vein thrombosis has been rotated and now shows the lesion at its commonest site, the upper temporal vein as described in the text.

Though the price has increased by four shillings since the previous edition, there is no doubt that this book will remain both a favourite with students and a very convenient guide to the general practitioner.

R.R.S.H.

THE EVOLUTION OF PSYCHIATRY IN SCOTLAND. Sir David Kennedy Henderson. E. & S. Livingstone Ltd., 1964. Pp. 300. Price 32/6.

This book contains many personal reminiscences on the development of psychiatry from 1907 up to the present day, of one who has lived through a phase of revolutionary growth. As such it forms an interesting record and valuable source of reference of those who were prime movers in Scottish psychiatry.

The earlier chapters are concerned with the story of the French influence on the Edinburgh School, telling how the first series of systematic and clinical lectures on mental diseases was instituted by Alexander Morison in 1823. Morison had studied in Paris under Pinel and Esquirol in 1818, and subsequently became a firm disciple of Esquirol. In 1823, Morison drew up a plan to establish a Professorship of Mental Diseases in Edinburgh but the University turned this down, it being considered too specialised a topic! It was not till nearly one hundred years later, 1919, that the chair of psychiatry was endowed.

It is unlikely that many students would wish to buy this sort of book, especially as much of the more important events are mentioned in Sir David's well known textbook, however, for those interested in the details of Scottish psychiatry, or those interested in the reminiscences of a very remarkable man, this book will provide interesting reading.

R.S.

NOTES ON CLINICAL SIDEROOM METHODS. Board of Medical Studies, University of Edinburgh. E. & S. Livingstone Ltd., Edinburgh, 1963. Illustrated, 7s. 6d.

In 79 small pages, this booklet describes the basic principles and the techniques of all those tests which can be carried out in the sideroom of the normally equipped ward. Obviously a book of this size cannot go into details of the interpretation of such tests and so it in no way competes with the larger texts in this respect.

However it does present in a most useful manner the sort of information which one actually requires when faced with the practical problem of how to do a particular test. Every student at this University is faced with just such a problem in part of the final examination, and surely every student in the country will meet such problems in the natural course of his career. A thorough acquaintance with this book will prove invaluable to all clinical students and all housemen.

When called to perform some emergency test in the middle of the night, even the most bleary-eyed will find the instructions simple to follow, and there is no doubt that the value of any test depends first upon its correct execution!

Prepared by clinicians, it is completely down-to-earth, and does not include anything that is not of genuine importance or any technique which is not in current use.

The minimum of essential illustrations increase the value of the book as does the convenient index. Without doubt, this will become a minor classic.

R.R.S.H.

A SYNOPSIS OF GASTROENTEROLOGY. By G. N. Chandler. 200 pp. 12 illus. Price 27/6. John Wright & Sons Ltd., Bristol 1963.

I often wonder what group of people uses a book of this kind; that is a synopsis of one of the major medico-surgical specialties. For those whose main interest is gastroenterlogy, it has little or no value on account of its brevity and for those whose interest lies outside this field, the material contained in this book is largely included in any comprehensive general text-book of medicine. However, such criticism can be levelled at all books of this type and within these limitations this text is in general a good one. One advantage of this kind of book I suppose is that it can be kept up to date more easily than general text-books. In the main, this is so with this book, but why does the author devote half a page to a completely obsolete investigation like the fractional test meal and only the same space to the much more important maximal histamine test. In this latter section, he makes no mention of the interpretation of the results of this test and in what ways the information obtained from it may be useful to the surgeon in his choice of the best type of operation in any given case.

Apart from this, the section on peptic ulcer is a good and comprehensive one as are the sections on small and large bowel disease and in particular on steatorrohoea and ulcerative colitis.

One major omission I feel, is adequate subsections on the psychiatric aspects of certain gastrointestinal diseases such as peptic ulcer, ulcerative colitis and Crohn's disease.

In conclusion this book is a good example of its kind but I feel with rather a limited appeal.

I.D.S.G.

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