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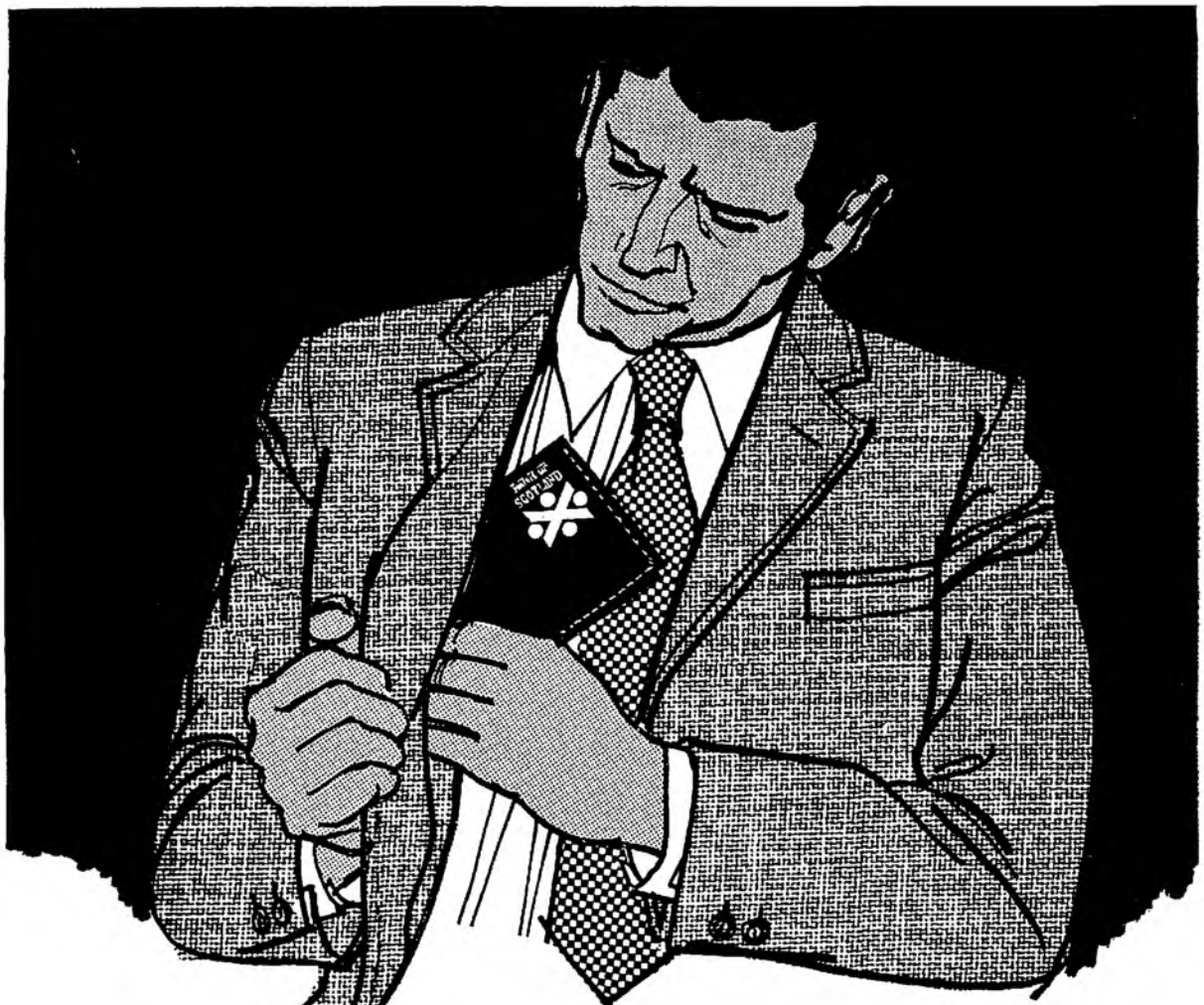

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EDITORIAL

"My Chinese uncle, gouty, deaf, half-blinded,
And more than a little absent-minded,
Astonished all St. James's Square one day
By giving long and unexceptionally exact directions
To a little coolie girl, who'd lost her way".

—Robert Graves.

But what became of him later? If he had lived in Edinburgh his future would have been disturbingly bleak. At present the South-East Region has the least number of long-stay beds in Scotland. Care for the elderly has become one of the darkest bêtes-noires of the N.H.S., and, as far as expenditure goes, one with potentially the most voracious appetite.

It is customary that certain platitudes about lack of finance be now meted out. This is readily understood, and yet, is the present situation economically logical? Owing to lack of more suitable facilities, many old people are now quietly slipped into acute wards in general hospitals although their diseases are more social than medical. At a rough estimation it costs £3,500 to maintain an old lady for one year in a teaching hospital — the house officers' despairing comparison with a year at the North British Hotel is perhaps unfair but nonetheless revealing.

In the wards the presence of long-stay patients in relatively large numbers poses certain very tangible problems. It is, apparently, a policy of the Royal Infirmary of Edinburgh that 20% of beds in every medical ward should be occupied by geriatrics, but in some wards this figure is greatly exceeded. At the time of writing, one female — officially on acute medical — ward in the R.I.E. contains 15 long-stay beds. It is obvious that with such a complement the space for truly medical cases is greatly reduced — thus the weary assembly of beds down the centre of the ward is started. These long peninsulas of beds make the patients feel insecure, make nursing extremely difficult and make the teaching of students well-nigh impossible.

Solutions to the problem have been assiduously discussed and sighed over. It seems that merely to build carefully-designed hospitals for long-stay geriatric patients would do little to alleviate the situation: the staffing problem would still remain.

Certain incentives for geriatric nursing do already exist. For instance, there is a special allowance of about £200 per year to State Registered Nurses who look after geriatric patients (for some incomprehensible reason not payable to nurses in the R.I.E.), but other inducements are obviously required. At present the only way of overcoming the chronic shortage of nurses is to indulge more freely in the use of the married (part-time) nurses who wish to return to work after starting their families. Obviously if they are to be enabled to do this crèches must be made available for their children. Financial gains must be increased — the problem cannot be resolved under the present tax scheme; an income tax incentive scheme is desperately required to tempt these nurses back to work; at present it is just not financially worth-while.

People generally look for romance before they leap on to a band-wagon. Care of the elderly offers little of this. It is, however, one of the few subjects that can do little but gain from repeated publicity. The maturity of a society is said to be reflected in the manner in which it cares for its most vulnerable members — for this reason, and for more selfish anxieties about our own future — we should take care to look after them well.

THE BIOCHEMISTRY OF SCHIZOPHRENIA

J. R. Smythies, M.D.

For many years there have been two schools of thought concerning the aetiology of Schizophrenia. Some psychiatrists have been impressed by the disturbed family relationships and early upbringing that is commonly seen in cases of schizophrenia and have felt that the condition is largely psychogenic: that is that anyone subjected to these malign influences would develop the disease. Other psychiatrists have felt that schizophrenia results from a genetically determined metabolic disorder and that the disturbed behaviour results from a brain with specific faults in its biochemical mechanism.

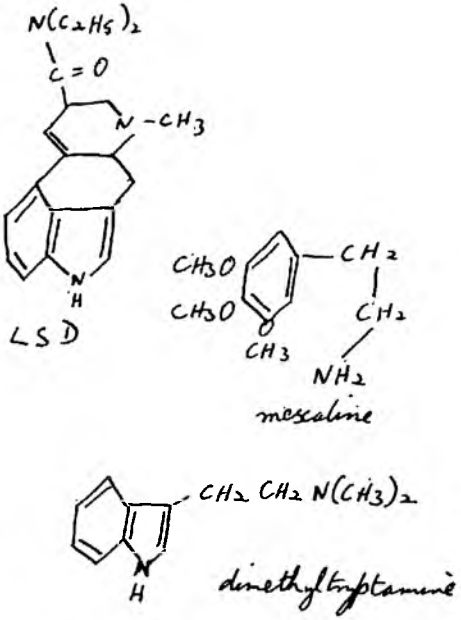
Two recent studies of what happens to the children of schizophrenic mothers who have been removed from their mothers shortly after birth and reared in foster homes have provided powerful, and I believe conclusive, evidence in favour of the latter view. This work was carried out by Heston in Oregon and by Rosenthal and Kety in Denmark. The results showed that these children in foster families nevertheless developed schizophrenia at the same rate (about 12% — as compared with the normal expectancy of 0.8%) as do the children of one schizophrenic parent reared by their biological mother. A control group of adopted children of normal mothers reared in similar foster homes showed no increased incidence. Then the foster families in which these children actually developed schizophrenia were compared with those in which the children remained normal, and no difference could be detected between them. Both lots appeared to be ordinary families. Thus what counts for the development of schizophrenia appears to be the genes and not the early family environment. Moreover many of the children of a schizophrenic mother, who did not develop actual schizophrenia, were abnormal having various severe forms of psychopathic personality. This would therefore account for the fact that many of the relatives of schizophrenics have abnormal personalities and for the fact that many schizophrenics come from peculiar families. However environmental influence also play a role in the genetic expression, which is of course true for any gentically determined condition, such as diabetes, and even for infections, such as tuberculosis. In identical twins, if one twin has schizophrenia, the other is affected in only about 40% of cases. But the affected twin is almost always the smaller and weaker with signs of relatively defective intrauterine nourishment.

The prognosis of the illness also depends, of course, on environmental and psychogenic factors. But the present evidence is that most schizophrenics are fated from birth to develop the condition.

If then schizophrenia is genetically determined, presumably the faulty gene or genes are expressed by some faulty enzyme(s) whose

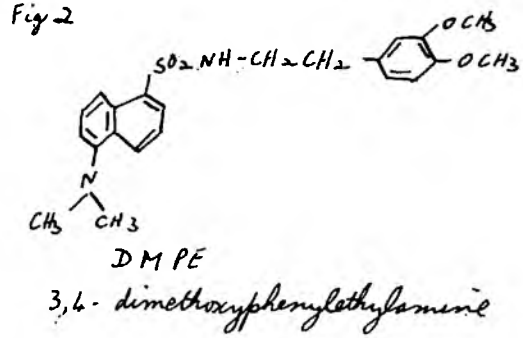
malfunction leads to disorders in the brain mechanisms underlying thinking, emotional responses and the generation of belief and behaviour. In search for clues as to what the system concerned might be people have studied the mode of action of drugs that can induce a schizophrenia-like reaction (i.e. hallucinogens such as LSD) and those that are of therapeutic value in the illness (i.e. the phenothiazines and butyrophenones). To take these in turn. The hallucinogens produce many different effects in different people, but sometimes they include a 'bad trip' which is an acute psychosis with many points of similarity with an acute schizophrenic breakdown. The formulae for some well-known hallucinogens is shown in figure 1. It will be noted that many are close chemical relations to the neurotransmitters serotonin and dopamine, the simplest relation being O-methylation, N-methylation or both.

Fig. I.



This relation ship led Osmond, Harley-Mason and Smythies to suggest in 1952 that schizophrenia might be associated with some disorder of catecholamine metabolism so that mescaline-like compounds were produced in the brain such as DMPE (fig. 2). Although this compound has been shown to be a normal constituent of urine (possibly of dietary origin) there is evidence that its levels are not raised in schizophrenics. However it is possible that schizophrenics may be abnormally sensitive to it, particularly since it has been reported that schizophrenics are abnormally sensitive to the psychotoxic properties of its close chemical relative 2,3,4-trimethoxy phenylethylamine (fig. 3). This possibility can easily be tested by experiment.

Fig 2



Another approach to this problem derives from the discovery made by Pollin et al in 1961 that some chronic schizophrenics react to l-methionin with an acute psychosis. This has now been confirmed by four groups of workers. In some cases the psychosis shows features of a toxic psychosis and in others of an acute schizophreniform psychosis. About 50% of schizophrenics react and the rest show no reaction at all. It is not possible to predict on clinical grounds which patients will react and which will not. The dose of l-methionine required is quite small (10-20 C/day) and this dose produces no reaction in normal people. Recently an unconfirmed report from Poland claims that methionine increases the excretion of dimethyltryptamine in schizophrenics but only in those that react to methionine. Thus the basis of the methionine effect may be to increase transmethylation processes in the brain since methionine is the origin of the methyl groups in all transmethylation reaction in the body. Other unconfirmed reports in this field are that schizophrenics do not react with a toxic psychosis as normal people do to the methionine antimetabolite MSO (methionine sulphoxamine) at a dosage of 300 mg/day. Also that schizophrenics methionine metabolism is different from normals. Methionine with a C_{14} label on the labile methyl group was given to schizophrenics and normal controls and the rate of excretion of $C_{14}O_2$ was measured.

This was much slower in the schizophrenics indicating an overactive transmethylating pool.

However we cannot assume that methionine induces its effects by some action on the transmethylation system, for it has other potent biological actions. For example 20 G l-methionine a day will cause an upset in the uptake of all the other amino acids and it will also affect secondarily tryptophan metabolism. The problems of how it does produce its effects in schizophrenics can only be settled by further experiment.

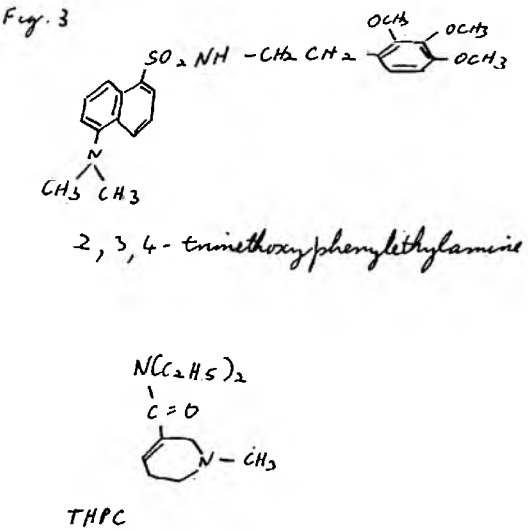
We can now ask how do hallucinogens produce their effects on brain function for that may give us a clue as to the site of the disorder in schizophrenia. Their most marked property is to block central serotonin mechanisms.

This has been determined both by microinjection techniques onto the surface of individual neurones and by the following technique. Most of the serotonin containing neurones in the brain locate their cell bodies in the raphe nuclei of the brain stem. From here the axons are distributed all over the rest of the brain. This system seems to control slow-wave sleep amongst other things. If recordings are made from this nucleus and d-LSD injected intravenously an abrupt cessation of firing results.

In addition to these drugs that mimic schizophrenia, we now know of several classes that alleviate it and are used in its clinical treatment. This includes the phenothiazines (such as chlorpromazine), the butyrophenones (such as haloperidol) and certain diphenylbutylpiperazines (such as pimozide). These have a very wide range of biological effects but prominent amongst them is the inhibition of catecholamines. Pimozide, in particular, has potent anti-dopamine actions. This suggests that schizophrenia may be associated with excess activity in the adrenergic and particularly the dopamine system, or it may not be the absolute level of activity in these systems that may be as important as their relative level or balance. If the serotonin system is blocked there may be a relating imbalance of the adrenergic or dopamine systems. The therapeutic action of the anti-psychotic drugs may thus depend on their ability to reduce this (relative) excess activity in the adrenergic system so that it comes back into balance with the serotonin system again.

Another way to treat schizophrenia would be to develop drugs that would so act on the serotonin receptor in brain that the binding of hallucinogens (such as dimethyltryptamine) would be blocked but serotonin itself could still bind. We are currently working on developing such a drug based on the 'top half' of LSD. Figure 3 shows a compound we have called THPC (N-methyl-1,2,4,6 tetrahydropyridine carboxamide). This is a close approximation to the top half of d-LSD. If it were to bind in the same locus as where d-LSD itself acts, clearly d-LSD itself could no longer bind, but mescaline itself, which approximates in shapes to the A + B rings of d-LSD, could. Therefore one would predict that THPC would block the effects of d-LSD but potentiate the effects of mescaline. This prediction was tested using a conditioned avoidance test in rats that enables us to measure the 'psychotomimetic' activity of a drug and was confirmed. THPC was also shown to block the effects of dimethyltryptamine. Clearly then THPC may be useful in the treatment of an LSD psychosis and if schizophrenia is caused by a compound like dimethyltryptamine, THPC might prove

Fig. 3



of therapeutic benefit, whereas if the compound responsible was more like mescaline, THPC should exacerbate the illness. Thus THPC can be used as a molecular probe to try and determine the nature of the psychotoxin of schizophrenia. Even if it had no effect, this would provide useful evidence — i.e. that no compounds similar to dimethyltryptamine or to mescaline were involved.

Now although the bulk of recent research

in schizophrenia has been based on the transmethylation hypothesis, other lines of study have produced some promising results. It has been known for years that chronic schizophrenics have a peculiar odour. Recently a claim has been made that the substance responsible has been identified (fig. 4). This was found in the sweat of 10/10 schizophrenics and in no normal controls. It did not appear to be the product of some bacterial flora peculiar to chronic hospital wards. The compound certainly has a most characteristic and penetrating odour. However it belongs to a chemical family — a branched chain unsaturated fatty acid — never before connected with brain function. In fact the only compounds I know of related to it that have a known biological activity are the bee ovarian hormones, Queen Substance and Royal Jelly, but this may be no more than a coincidence. At any event if this report is confirmed, vigorous efforts should be made to trace its metabolic origin.

A very large number of other investigations have been carried out in schizophrenics but none has achieved any very notable results. Sometimes positive results have been claimed but subsequent work has shown these to be artefactual or due to malnutrition, non- specific stress and other similar factors. This very lack of success has had negative results. The answer to the problem of schizophrenia can only come from an intensive research effort directed towards discovering its biochemical basis. Unfortunately the skilled biochemists and other biological scientists needed to work on such a programme are not attracted to what has proven over the last 50 years to be so unrewarding a field. The bulk of biochemical research in psychiatry is currently focussed on the biochemical basis of depression, in the form of very extensive work on the biochemistry of cerebral amines — their metabolism, uptake, storage and release mechanisms — which has already paid handsome dividends including Nobel Prizes to two of the main workers in this field. In contrast the field of schizophrenia research is almost totally neglected both in Europe and North America which is strange when one considers the enormous economic cost to the community and the cost in human suffering and blighted lives that schizophrenia brings about. It is not commonly realized that about one in 120 people will develop the illness at some point during their lives and that slightly less than one-quarter of the hospital beds in the country are occupied by schizophrenics.

However, now that we possess at last one valid fact about the physiology of schizophrenia — the methionine effect — the immediate way ahead is clear. The biochemical basis of the effect must be determined. Is it due to altered transmethylation reactions, to changed patterns of aminoacid uptake, to disturbances in tryptophan metabolism or to some other factor? The answer to this question may well lead to other research programmes that in turn may further our understanding of the biochemical basis of one of the still outstanding problems of medicine.

THE NATUROPATHIC APPROACH

C. Leslie Thomson, B.SC.

That word 'approach' reflects an essential feature of Naturopathy — its philosophical attitude to the problems of life. It is no mere collection of empirical techniques, although like any practical system it had its beginnings in many fields of observation and in trial and error. Its theories are co-ordinated with facts in a consistent fashion which justifies its being called a method .

What this implies may be clarified by considering a person who has headache, and who consults a variety of individuals about his problem. The first might prescribe an analgesic, perhaps combined with an anti-depressant. Another could offer to sell him extracts from vegetable tissues, but essentially intended to have the same effects as the ethical prescription. Still other advisers would propose to make the sufferer unaware of his distress, by some form of mental exercise, suggestion or counter-irritant.

On principle, the Naturopath rejects all of these. All may work, but not by rectifying the causes. He has more affinity with the manipulator who, finding undue tensions or misalignments in the neck, by suitable adjustments diminishes strain and so gives relief. But Natuopathic philosophy demands that before applying even so apparently obvious a remedy one must ask the question 'why?'. Headache and neck tension may be due to various primary causes, more often a combination of several. Without seeking to discover at least something about these, one cannot give advice or treatment likely to be of more than transient benefit.

It is more tragic than comic that many people believe that 'a disease is the sum of its symptoms'. This fallacy is profitably exploited in the sale of every kind of nostrum and in encouraging many forms of symptom treatment. The Naturopath believes that any diseased organism is disordered *before* the appearance of signs or feelings of 'dis-ease'. Logically, true corrective treatment must be related to conditions which preceded the symptom. Even if these conditions no longer exist, they can at least be recognised, and reasoning about the patient's problem becomes possible.

In everyday terms, most illness is the result of the way most people live. A person who is properly nourished, well exercised, rewardingly employed, happy in his relationships and in an unpolluted environment has an excellent chance of good health. These factors alone cannot guarantee freedom from illness, because others may undermine his constitution. However, one can say with assurance that to the extent that the individual's existence is deficient in one or more of the items on that list, his chance of good health is diminished. Accordingly, we look to all these aspects and more.

Perhaps more important than any is an understanding of disease processes. We believe that disease is no accident; it always has rational causes, although one may have to seek far in time and space to find them. (By this we mean neither astrological mysticism nor the vilification of creatures and substances of microscopic or molecular smallness. Bacteria and viruses are interesting and complicating factors in many conditions, but are themselves influenced by pre-existing disorders.)

The Naturopath's immediate aim is to discover what guidance his patient requires in such routine matters as diet, exercise, occupational effects, emotional adjustment and the avoidance —as far as possible — of environ-

mental perils. Today, none of that sounds controversial, although it aroused bitter resentments little more than a generation ago.

However, orthodoxy still rejects our attitude to acute illnesses. These we regard as having not only logical causes but constructive purposes. That is, simple colds, fevers, rashes, sicknesses or diarrhoeas have eliminative or stress reducing significance. We see them as analogous with spring-cleanings in a household — a time of disturbed routine, of discomfort and commotion, yet with a wholesome purpose. A system which has its occasional spring-cleanings — as necessary to deal with accumulations of waste and to effect general readjustment — remains in good working order throughout a long and useful lifetime. But if these unpleasant yet useful processes are promptly arrested by symptomatic treatment, the system declines into a state of obstruction and impaired function. This is the situation in which chronic disease is most likely to develop.

We prefer to give our help to those still in good health, so that they may maintain it and realise their potentials. We have much to offer to young people and athletes; the beneficial effects of proper dietetics upon dentition, general development and muscular endurance are everyday instances. For people of any age, we, have a useful range of emergency aids from reduction of pelvic stress (often miscalled 'slipped disc') to treatment of injuries, wounds and burns. (For over 150 years, we and our predecessors have, with outstanding success, treated burns with cold water — a method only now beginning to be partially adopted in orthodox circles.)

And here it may be well to record that we place high valuation on surgery in more serious accident cases or in vital obstruction. Convalescence can be markedly assisted by Naturopathic routines, and in less-urgent circumstances we have often co-operated most successfully with surgeons, by promoting an improvement in the patient's tissues before operation, so that healing is rapid, neat and strong.

Those who have long been chronically ill, and who have suffered extensive degenerative changes, are likely to turn to Naturopathy as a last resort. Obliquely flattering though this may be, true cure in such cases is impossible — because some member, tissue or a part of a vital organ has already been destroyed. Nevertheless, that good, workable recoveries can sometimes be effected is an impressive testimony to the tremendous self-healing powers of the human system — when unopposed and understood. These cases are also evidence of intelligence and determination on the part of the patient. And there should be no dubiety about this; Naturopathy is demanding on the patient. It calls not only for the effort of initiating routines of treatment (which gradually merge into daily habits) but also for the burden of individual responsbility.

We do not offer to 'cure' our patients. Our work is essentially educational, with the aim of enabling the patient to realise his own self-reparative and vital capacities. In the early stages we may have to assist by relieving physical stresses, to give guidance amounting at times to bullying or to ask the patient to do certain things on trust. But until we have explained reasons, and until the patient gains confidence in his own judgment, our work is incomplete.

We point to the cumulative menace of widespread and seemingly innocent customs — such as taking quantities of tea, coffee or soft drinks; all forms of smoking; the consumption of denatured foodstuffs; the use of 'simple remedies' for bowel sluggishness, stomach-ache or insomia — and to the many other devitalising effects of civilised existence.

On the negative side, we must explain that our philosophy has no real place for such amusements as intestinal irrigation, expensive thermal baths, elixirs, vitamin concentrates and a whole catalogue of so-called 'health foods' (which often prove to be merely costly packages of wastes from food factories and pharmaceutical laboratories). Our methods do not involve the patient in the purchase of such services or merchandise. Our incomes do not come from profit or commission on sales of remedies, 'health foods' or gadgetry.

More positively, we can practise our skills in manipulative therapy, all the way from simple massage for relaxation to really strong corrections. Even here, we do not work mechanically, since each case must be individually assessed; dealing with the causes of a disorder is obviously more vital than giving immediate relief. In dietetics, we have practical information accumulated over three generations of systematic observation, and going far beyond the elementary matters of calories, amino-acids, vitamins and minerals. Through two centuries, experience of hydrotherapy supports our use of simple and effective water applications in particulars circumstances.

We are familiar with the psychological

significance of certain symptoms and stresses. We are alert to the ways in which emotional strains may be intensified or diminished by altered physiological states, and how the reverse sequence can occur. Closely linked with these are problems of faulty posture— a broad field in which the patient's emotional background is as significant as his occupation.

Naturopathy is not, and makes no pretence to be, a substitute for medication. It cannot be produced in doses for specific ailments, and it is not primarily a resort for people when they are ill. It is a way of looking at life, and applying reasoned interpretation to what is observed, so that those who accept and practise its philosophy can give their systems a better chance of maintaining normal function, or of regaining it. As with any worthwhile way of life, it is not merely for the Sabbath but for every day.

Our first, fairly lengthy, interview with a patient may be mainly devoted to examination of physical indications of disorder, but often it is obvious that psychological strains are of greater account. Sometimes, we believe we identify the major factors promptly; in cases it takes time to establish communication — perhaps after a considerable interval for contemplation. What we suggest as remedial depends more upon what we recognise as basic causes than upon specific symptoms. Further, we cannot truly apply our methods to a reluctant patient; there must be willingness to understand and to make a personal effort. The patient does not 'submit to a cure'; he effects it himself, with our guidance.

To make this easier, we have residences where such material considerations as diet are provided for, and where it is convenient to give instruction and physical treatment. But this is only a temporary phase; an introduction to and preparation for the life-long acceptance of Naturopathic philosophy and practice. Although a patients' education there may be intensive, only a small part of his recovery usually takes place in such establishments; his real cure is a continuing process, conducted and carried out by his own physical, ethical and mental resources.

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PULMONARY INSUFFICIENCY IN THE CRITICALLY ILL

Malcolm F. MacNicol

Surgical Research Fellow Harvard University

In recent years clinicians concerned with intensive care have become increasingly aware that pulmonary insufficiency may develop shortly after the apparently successful immediate resuscitation of the critically ill. The incidence of this complication is high, a figure between 30% and 50% being generally accepted[1]. In its initial stages respiratory tract infection does not appear to play a part though bronchopneumonia may later supervene.

The impact of this adult respiratory distress syndrome (ARDS) on surgeons dealing with extensive trauma, sepsis, burns and heamorrhagic shock has been dramatic and since 1968, when the first conference devoted to the pulmonary effects of nonthoracic trauma was held [2], investigation into the problem has been energetic. Essentially two opposing viewpoints have evolved to account for the occurrence of ARDS. That they are interrelated should become clear in the following discussion.

AN IATROGENIC CAUSE?

Serious trauma, sepsis and burns share at least one thing in common with hemorrhagic shock: they all lead to a decrease in the circulating blood volume with reduction of tissue perfusion. Effective means of fluid replacement are now universally available and proper concern is given to replenishing losses of the formed elements of blood, plasma and electrolytes in the proportions required by the individual case. However the practice of fluid replacement to sustain an adequate circulation is not without its dangers. The exuberant use of crystalloid solutions, while preserving renal function, may so load the pulmonary circuit that there ensues an interstitial edema consequent on a lowered intravascular osmotic pressure. Therefore the clinician must be aware of the delicate balance that exists between a renal demand for adequate perfusion and a pulmonary sensitivity to fluid overload.

The pulmonary microcirculation may also suffer from infusions of blood banked too long or inadequately matched. In this context it is often not fully realised that the lung is the first vascular bed to meet and react with materials delivered by vein. Thus the sludge from old blood, or the potency of pharmacological agents, exert their maximum effect on the pulmonary vasculature. While the lung appears to perform remarkable feats of detoxification and inactivation this function can be outmatched, especially in the sick patient, and thereafter endothelial and alveolar cell damage will occur. High pressures of oxygen administration will also seriously impair pulmonary function but awareness of this danger has led to a cessation of such therapy.

Although ARDS cannot simply be explained by errors in clinical management, especially since it frequently develops prior to hospital treatment[3], it is salutary to remember that the clinician can worsen the respiratory function of a patient. Moreover any gross mishandling of drugs or fluids will have far more serious consequences in the critically ill patient than in his normal counterpart.

ENDOGENOUS FACTORS?

The other explanation of ARDS complicating nonthoracic injury stresses that anoxic, injured and inflamed tissue releases a variety of deleterious agents into the circulation. These products of proteolysis or membrane dissolution may be directly injurious to the lung or may trigger the release of other pathological factors. Systemically, activation of Hageman Factor will result in kinin formation and intravascular clotting. Locally, histamine and serotonin may be released in the lung. Normally the spread of these agents is contained by natural inhibitors. But in prolonged sepsis and severe trauma, especially allied to inadequate nutrition and hypoxia, the protective mechanisms of the body are frequently overwhelmed.

There is ample experimental data to suggest that endogenous agents, probably circulating in

pathological amounts, can seriously alter pulmonary morphology and function[4]. A number of factors have been suggested, with various degrees of clinical correlation, and include endotoxin[5], fibrinopeptides[6], microemboli[7], bradykinin[8] and fat embolism[9]. In any individual case of ARDS a number of these agents may be implicated, working directly or indirectly, and perhaps in a characteristic sequence yet to be defined.

CHARACTERISTICS OF THE LESION

Clinically the earliest manifestation of ARDS is physiological shunting in the lung. The results from both a ventilation — perfusion imbalance and a change in ventilatory pattern in which hyperventilation and a reduction of tidal volume are apparent. A shunt of over 20% is one of the most sensitive indicators of impending respiratory failure and few survive shunts of 60%[3]. Evidence of significant shunting on admission to hospital is associated with an extremely high mortality, therapeutic reversal of the damage incurred by the lungs proving well nigh impossible with currently available techniques.

The patient responds to the shunt with hyperventilation but cannot compensate for its effect so that the pO_2 falls progressively, reaching values below 60 mm of mercury. While a respiratory alkalosis is usually evident initially, being superimposed on the attendant metabolic lactacidemia of the severely ill patient, as ventilatory failure and increasing shunt develop the pCO_2 begins to rise. In most instances this is a terminal feature. Radiologically the chest is often normal at the outset, in spite of a developing hypoxemia. Within one or two days, however, a diffue mottling in both lung fields appears, to be followed by the X-ray features of consolidation and atelectasis. These changes occur either with or without the clinical signs and symptoms of the pneumonic process. Lung compliance also decreases and adds greatly to the work of ventilation. This is dramatically demonstrated by the increased pressures required to ventilate the lungs of the patient artificially. Occasionally pressures in excess of 20 cm of water are necessary, often making pressure cycled respirators ineffective.

Studies with radioactive indicators[10] have shown that the pulmonary capillaries are relatively impermeable to the sodium ion on its first passage through the normal lung. This characteristic is lost in patients with pulmonary insufficiency following major operation or injury. Pathological specimens of the pneumonitic lung clearly demonstrate the result of this deranged function and the following features are regularly seen with mocroscopy, although in varying degrees.

1. an oedematous thickening of alveolar walls.
2. focal atelectasis and in severe cases the formation of hyaline membranes.
3. erythrocyte and platelet intravascular engorgement, diapedesis and intraalveolar aggregation.
4. leucocyte proliferation, margination and adherencse to the pulmonary capillary endothelium.

Compared to normal leucocytes these cells contain irregular electronlucent areas and appear to have undergone partial degranulation[11]. As observed in the shock state, the emigration of these leucocytes is decreased. Therefore the membranes and cellular elements of the lungs present many of the features of the classical inflammatory response though certain differences are evident.

Accepting that the etiology of ARDS remains controversial and is almost certainly multifactorial, its effective treatment rests solidly on the accurate and early interpretation of clinical signs. A careful assessment must perforce include a watchfulness for tachypnea, frequent chest physical examination with accompanying X-rays, blood gases and electrocardiograms where indicated. Obviously care of the primary lesion is of paramount importance. In sepsis the eradication of any septic focus, whether by drainage, resection of nonviable bowel or amputation, is essential. Gram negative sepsis is so common that it is well to remember the high resistance of these organisms to the penicillins. Systemic kanamycin, gentamycin and polymyxin B are antibiotics of proven efficacy against most gram negative bacteria. In cases of fulminating peritonitis lavage of the peritoneal cavity with kanamycin dissolved in normal saline is also meeting with some success the critical need of the patient to counter bacterial and endotoxin invasion from the gut reservoir.

Careful assessment of the loss of vital fluids and electrolytes allows a reasonably accurate titration to be carried out in terms of acid-base balance, hydration and haemoglobin concentration, which should be maintained above 12.5 grams/100 mls if possible. The central venous pressure should not be allowed to rise precipitously, and while absolute values can be misleading, rapid fluctuations must be carefully monitored. In cases of a high central

may be of benefit and diuretic therapy may be necessary; however the increased pressure may simply reflect the rise in pulmonary artery venous pressure elevation of the head and chest pressure that accompanies pulmonary damage. Respirator support is essential in those patients showing a pO2 much below 60 mm of mercury. The decrease in lung compliance in ARDS has been attributed to a loss of pulmonary surfactant. Saline lung washes from patients with post traumatic pulmonary insufficiency reveal a decreased concentration of surfactant[1]. The question is whether this decrease reflects diminished production of surfactant as a primary problem of ARDS or whether it results from poorer extraction of the surface active agent from lungs made atelectatic by another mechanism. Whatever the cause of the diminished lung compliance may be, the maintenance of continuous positive pressure throughout the ventilatory cycle by a respirator is of great benefit and ensures that the occurrence of further atelectasis is minimised. With proper use of respirators and fastidious tracheal toilet patients with severe pulmonary insufficiency can be ventilated for several days.

A variety of drugs have been used in the treatment of ARDS. As already mentioned, antibiotics and diuretics are of proven efficacy provided that their use is appropriate to the situation. Steriods, perhaps on account of their membrane stabilising property, have been shown, at least experimentally[12], to be of some benefit, and in an attempt to reduce intravascular aggregates in the pulmonary microcirculation both heparin and low molecular weight dextran have been advocated. If vasoactive peptides are definitely shown to be circulating then the use of antoproteolytic agents to block their formation at source holds out much hope, as may the use of carboxypeptidases which destroy the kinins.

As a final concept it should be remembered that the lung is an actively metabolising organ and not merely a passive membrane exchanger of gases. Normally it deals efficiently with infective or toxic agents reaching it from the air or blood. However this capacity may be overcome under conditions of hypotension, severe injury and sepsis with resultant inflammatory changes and progressive impairment of pulmonary function. If ARDS is not recognised early a relentless fall in blood oxygen tension will seriously impair the recovery processes of the critically ill patient and at autopsy will be found a heavy, sodden and hemorrhagic pair of lungs with numerous areas of atelectasis and consolidation.

SUMMARY

A progressive and pernicious pulmonary complication that often attends nonthoracic trauma, sepsis, burns and circulatory disturbances is discussed. The reason for the rise in the incidence of this adult respiratory distress syndrome (ARDS) is complex but reflects paradoxically the apparent success in the immediate resucitation of the critically ill. Although the etiology of ARDS is probably multifactorial certain lines of treatment suggest themselves and should be vigorously pursued. Above all, early recognition of the syndrome may prevent the remorseless deterioration of the lung that so frequently accounts for the death of the patient.

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NEUROSURGERY - CONTEMPLATION OF THE FUTURE

J. Lawrence Pool, M.D.

When I was a medical student 42 years ago my neurological textbooks were replete with diagrams of the motor, sensory, visual, and speech areas of the cerebral cortex. It occurred to me then that perhaps the viscera might also have cortical representation and therefore why not try to find out if this were so? Sad to say I was dissuaded from investigative attempts on the grounds that time in medical school should be spent only on learning. (In this respect, I would urge that any of you with promising investigative ideas should put them to the test and not be persuaded to forget them. For sad experience has shown that if you don't do the job, someone else will!) Since my early days, much has been learned about the visceral representation in the cortex of the brain, to part of which I eventually contributed. It is now known, for example, that the vagus nerve nucleus has a representation in the subfrontal cortex, bladder and bowels in the juxtamotor cortex, cardiac and respiratory control in the cingulate and medial temporal cortex, and sex organs also in the latter. Pupillary changes may be elicited by stimulating the anterior cingulate gyri, intussception by stimulating posterior cingulate gyri, while blood pressure alterations may be induced on stimulation of numerous parts of the cerebral cortex. All this adds up to the now well-known fact that in addition to a motor and sensory cortex, there is also what might be termed a "visceral brain" in man and beast[9].

More recent and on-going studies in our laboratories[3] suggest there may also be what might be called a "metabolic brain". For example, stimulation of certain discrete areas within the hypothalamus and more remote but related parts of the brain by means of microelectrodes, in cats and other animals, induces an almost immediate and massive mobilization of fat, after a meal, into the circulating blood, and at the same time causes the blood to clot within a matter of seconds instead of several minutes. Here then is a possible mechanism by which coronary or cerebral vascular accidents may be produced. A stressful situation following a heavy meal may, via hypothalamic mediation, cause a dramatic increase in fat in the circulation. This mobilization of fats increases blood viscosity and the blood tends to clot rapidly. The combination of these two factors within a narrowed sclerotic coronary or cerebral artery could obviously lead to its occlusion. This preliminary work, although not yet definitive, suggests how the brain may play a role in causing a serious systemic disease, such as a stroke or coronary attack.

Other work in our medical center has indicated the presence of a new hormone-like protein, derived from the hypothalamo- pituitary system, which appears essential for synchronous peristalsis of the gut. Preliminary studies suggest that this hormone is deficient in persons afflicted with gastro-intestinal disorders such as ulcerative colitis, and that the latter can be relieved in some cases by administration of this hormone[6]. This perhaps is another example of metabolic control of body organs by the brain.

More familiar examples are fluid, electrolyte and sugar regulation by hypothalamic and brain stem nuclei, and endocrine control by the hypothalamic-medial temporal-pituitary axis. Recent work[12] indicates that even the pulmonary circulation may be drastically altered by the autonomic nervous system activity.

What has all this to do with the future? Increasing evidence points to the influence of the brain in regulating systemic activity. I would suggest that perhaps *all* body functions, metabolic, hematologic, endocrine, visceral, autonomic and even pulmonary circulation[12] may be controlled or regulated by the brain.

Further efforts should be made, I feel, to understand how the brain and its chemistry affects the various moods and emotions whose aberrations can lead to mental illness, including schizophrenia[10]. A good deal, of course, is known about the anatomy of these circuits such as that of the limbic system and its specific thalamic and hypothalamic links. The future, I would suggest, offers the possibility of a brand new and far more promising kind of brain-mapping, based on the well established fact that various cell types of the central nervous system, be they neurons or glial cells, have individual biochemical characteristics with respect to their nucleic acids, organelles, enzyme systems and synaptic transmitters. A dramatic example of this possibility is afforded by the fluorescent histochemical technique developed by Falck and Owman for detecting monoamine-containing nerve terminals[4]. This tool has already afforded us new insights into the function of the nigral-straital system that was impossible by conventional histological methods. The excitement generated by each new discovery in the metabolic pathways of L-DOPA, and the therapeutic success of this amine, may well be duplicated when more is understood about the function of other amines, particularly norepinephrine.

Sophisticated application of radioactive tagging techniques offer a new method of biochemical "brain-mapping". In this manner we can detect, for instance, transneuronal transfer of possible *trophic* substances in the central nervous system[5] and the differential up-take of steroids by various portions of the brain. This work has underlined the importance of temporal lobe structures in regulation of both cortical and estrogen function. These techniques may also further our knowledge of the metabolism of metal in the brain. Copper, lead, tin, mercury, manganese and lithium all have been linked to certain changes in brain and mental function. The recent work showing amelioration or control of manic-depressive states by administration of lithium carbonate indicates that investigation along these lines should also be rewarding.

Understanding of these biochemical-neural circuits could be important not only for a better understanding and therefore therapy of the usual run of mental and emotional illnesses, but also for those unfortunates plagued by life-long psychosomatic symptoms. The latter too often have been tossed off by our psychiatric brethren as being purely psychogenic in origin. Perhaps so, but it does seem that such symptoms may be truly organic in the sense that they become embedded in brain circuits as part of a self-inflicted conditioned reflex. (And at this point I must confess parenthetically that there is a very thin line between what some would call a psychogenic and others an organic pattern.) The main point to be made, I suggest, is that most psychosomatic complaints relate to the autonomic nervous system: stomach aches, anorexia, palpitations, sweating, a dry mouth, fatigue, constipation and perhaps sundry aches and pains including headache. Too little attention has been paid to a possible organic aetiology of such symptoms. Recent studies have shown that rats have so slowed their heart that they actually died of cardiac arrest. These and other experiments[3], indicating that cerebral control and conditioning is possible for *autonomic* nervous functions, suggest that psychosomatic complaints may likewise have become ingrained, conditioned reflexes. Experimental mapping of the involved circuits could, in the future, perhaps lead to specific stereotaxic method of relieving severe psychosomatic symptoms.

And finally, with reference to what Bell (1816) called hypochondriacs, which I suspect were often what we would call "psychosomatic" patients, he wrote as follows:[1]

> "In the Hypochondriac's feelings all is not imagination. Pains and odd sensations, attributable to external and remote parts, do actually proceed from the disturbance of internal nerves."

I would heartily second these prophetic remarks and suggest pursuit of brain mapping with this in mind. For it is clear we do not begin to know all we should about the nervous system. My orthopedic colleague, Professor Andrew Bassett, and others[2] have, for example, discovered that the internal as well as external parts of all bones of the body, including every Haversian canal, have a nerve supply. Where do these nerves go and what is their purpose? Is their role sensory, metabolic or perhaps trophic? Do they, like other nerves, enjoy cortical representation? Could it be, as your Dr. William Cullen (1710-1790) taught in 1760, that the normal state of the body is determined by "nervous energy" from the nervous system and affected by external stimuli[11]? These are additional questions for the future.

There are many other topics one could cite that need attention in the future. Suffice it

to say in closing that some of the more important include microsurgery to improve the circulation of the diseased brain; a better understanding and hence better treatment of some of the still mysterious forms of *hydrocephalus*; and perhaps the development of special techniques[7,8] or even implanted micro-electrical circuits fitted with miniature computers, to enable paraplegics to walk, and, as some are already attempting, to enable the blind to see.

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SKI-ING INJURIES

G. P. Mitchell, F.R.C.S.

Injuries sustained while ski-ing do not differ significantly from injuries due to other sports. Tears of the medial meniscus however are rare in ski injuries compared with football. The increasing frequency of the unstable boot top fracture due to the modern high rigid boot resembles the type of fracture sustained in a sliding tackle on the football field.

Discussion on ski injuries therefore should emphasise prevention rather than treatment. Statistics suggest certain common factors in the clinical history of ski injuries which indicates the need for a safety code which might reduce the incidence of injury and thereby increase the pleasure derived from this sport.

In this article common ski injuries will be mentioned, prevention will be discussed and a safety code for skiers will be suggested.

I. COMMON SKI-ING INJURIES

Ski injuries can be divided into (A) soft tissue injuries and (B) fractures.

(A) *SOFT TISSUE INJURIES*

1. *The knee joint.*

The most common soft tissue injury is the abduction rotation strain to the knee which causes a partial or complete rupture of the medial collateral ligament of the knee with associated capsular tear in the severe cases. The injury is usually a partial rupture of the medial collateral ligament and the medial meniscus is seldom involved. Waldie (1969) reported 125 cases of knee injury in which there were 102 cases of injury to the medial collateral ligament. There were 11 cases diagnosed as medial meniscus injuries in which 6 cases healed without operation and probably were not true meniscus injuries — there was only 1 bucket handle tear proved at operation. The relative infrequency of medial meniscus injuries from ski-ing as opposed to football is probably due to the fact that in the ski injury the unweighted tibia is abducted and rotated on the femur while in football the femur is rotated and abducted on the weighted tibia.

Statistically the infrequency of injury to the medial meniscus is of special interest to the orthopaedic surgeon. The problem is complicated by the skier who returns from a continental holiday with the history of an abduction rotation knee injury, sustained perhaps a week ago, which has been treated by a compression bandage to allow the patient to continue with the social if not the sporting activities of the holiday.

Clinical examination reveals a flexion deformity of the knee suspiciously like the "locked knee' of a meniscus injury. There is tenderness over the femoral origin of the medial collateral ligament but there is also tenderness over the medial meniscus. A definite effusion might suggest a meniscus lesion but many cases have only a slight or no effusion. This knee should not be subjected to immediate operation. The knee should be examined and fully extended under general anaesthesia and should then be immobilised in a plaster cylinder with full weight bearing for approximately 3-4 weeks. At the end of this period the incomplete tear of the medial collateral ligament will have healed sufficiently to allow normal rehabilitation. If the meniscus injury is present then the knee will again produce symptoms on mobilisation and arthrotomy will be indicated. Immobilisation in a plaster cylinder is initially the best treatment for a partial rupture of the medial collateral ligament which might otherwise give rise to symptoms for a considerable period.

Complete tears of the medial collateral ligament should always be treated by surgical repair because this allows accurate reposition of the torn ligament, repair of the associated capsular tear and accurate inspection of the joint to ascertain any injury to the medial meniscus.

2. *Injury to the Gastrocnemius and Tendon Achilles.*

Partial rupture of the Gastrocnemius muscle or complete rupture of the tendon achilles is

a relatively frequent ski-ing injury. The modern high rigid boot will reduce such injuries at the expense of "boot top" fractures of the tibia.

Complete rupture of the tendon achilles can be successfully treated by immobilisation of the ankle joint in full equinus but the majority of ski-ing surgeons would prefer a formal surgical repair.

Rupture at the Gastrocnemius tendon junction can be a very painful injury and in major tears the ankle joint should be immobilised in plaster. A below knee plaster does not abolish all symptoms but the patient is more mobile and rehabilitation is more rapid. Immobilisation in an above knee plaster abolishes all pain but greatly restricts mobility.

3. *Dislocation of the Shoulder.*

Dislocation not associated with other injury is usually found only in the shoulder in the skier and is a relatively common injury. The importance of this injury is that shoulder dislocation can be reduced by first aid measures either on the ski slope or in the local hotel thus avoiding the need of transportation of the patient to hospital and anaesthetic facilities.

The technique of reduction of shoulder dislocation without anaesthesia was described by McNair (1957) and the author has found the method to be successful in all cases treated either on the ski slopes or in the nearest habitation. The principle implies the positioning of the patient in a comfortable prone position on a table with the injured arm hanging free over the edge of the table. The pain is usually immediately relieved and muscle spasm can be further reduced by simple analgesics such as whisky or brandy provided the patient is treated in his home base. Pain disappears the moment the arm is dependent. The doctor then sits on the floor and applies gentle longitudinal traction to the arm, at the same time reassuring the patient, to allow full relaxation of the muscles. Usually at a very early stage of traction the shoulder reduces because the muscles have relaxed. Traction should be prolonged and gentle in apprehensive patients. In the rare event of reduction not being achieved by gentle traction, one hand is gently placed in the axilla while the other hand maintains traction. The humerus can then be displaced laterally into the joint from the subglenoid position achieved by traction.

The author has reduced shoulder dislocations on the ski slope by the same technique with the patient lying prone on the back of a man kneeling in the snow. Ski clothing does not have to be removed. The only difficulty is that the patients elbow must be flexed to the right angle in order to apply adequate traction without the patient's hand touching the snow. Such immediate reduction allows the skier to return to his base without assistance. This method of reduction is safe because the hand in the axilla can easily detect the crepitus of an associated fracture.

4. *Abduction injuries of the thumb.*

Abduction strains and complete rupture of thumb ligaments can occur from the thumb being caught in the strap of the ski stick or from impingement of the thumb in the mesh of an artificial nylon ski slope. Sprains of the medial ligament respond to plaster immobilisation but complete tear should be treated by surgical repair of the medial ligament which is frequently entrapped in the joint.

5. *Ligamentous injuries of the ankle joint.*

These used to be common injuries with the soft boot. Recently with the rigid high boot these injuries have decreased while tibial fractures occur more frequently.

The most common injury is a tear of the anterior talo-fibular ligament. Lateral rotation of the ankle may cause a tear of the inferior tibio-fibular ligament resulting in instability but this injury is often accompanied by a fracture of the fibula.

Rupture of the lateral ligaments should be treated by immobilisation in plaster.

Spademan (1968) reported that in lower limb injuries 15% occurred in the lateral ligament of the ankle joint and 12% occurred in the deltoid ligament.

(B) FRACTURES

Fractures occur most commonly in the tibia or in the ankle joint where they may be associated with rupture of the ankle ligaments. In the past ankle injuries were more common than fractures of the tibia. Recently however the number of fractures of the tibia has increased while ankle fractures have reduced. This is considered due to the modern high rigid boot which protects the ankle at the expense of the tibia. The fracture of the tibia in the lower third of the leg known as the "boot top fracture" is now a relatively common injury in the modern ski slopes.

The principle of treatment of fracture of the tibia and fracture dislocation of the ankle joints does not differ from standard practice of similar injuries from other causes and so will not be discussed.

II. PREVENTION OF SKI-ING INJURIES

Ski-ing injuries could be reduced by attention to (A) equipment (B) Adherence to the skiers safety code.

(A) Equipment.

In recent years there has been an increase in the types and complexity of so called "safety binding". There is really no such thing as a "safety binding" — there are only release bindings. The implication is that a release binding is only a safety binding when the release mechanism is adjusted to the technique, experienece and strength of the skier.

Reliable adjustment of a release binding can only be made by the skier himself provided he fully understands the mechanism of his own binding. The adjustment applied by ski hire shops is unreliable and even modern ski binding testing mechanism can only give an approximate assessment. The accurate assessment depends much more on the technical ability of the skier than on the weight and height of the skier. The skier who can adjust his own bindings has a greatly reduced chance of injury compared with the skier who hires skis and relies on the ski shop for the adjustment. Commercially it is not possible for the ski hire shop to spend the time necessary for the accurate adjustment of bindings to the strength, ability and boots of the customer.

Release bindings are only safe when the skier fully understands their release mechanism and is prepared to spend time testing and adjusting these bindings at slow speed and varying snow conditions. A binding set to safety for the moderate skier would release prematurely during a turn at speed by a highly competent skier of the same body weight. Ski hire is therefore less safe than one's own skis, which have been set from experience to one's own ability.

Space does not permit detailed discussion on the merits of relative bindings but some common errors can be mentioned. The curved sole of the old boot which does not have the rigidity of the modern plastic sole, the matching of different types of forward and heel release, the worn or kinked cable release and the bent cable clamp or inaccurately sited posterior heel cable release, the lack of antifreeze lubricant in certain bindings at high altitudes, &c. The conclusion is that there is no such thing as a safety binding and bindings can only be safe when personally adjusted to release before injury threshold.

The modern high rigid boot is responsible for an increase in "boot top fractures" of the tibia which did not occur with the old lacing boot. A strong, fit and experienced skier will benefit from the more rigid support which is not necessary or advisable for the beginner or moderate skier who is not fit. The danger of the modern high rigid boot can be avoided by adjustment of the boot to grip the heel but to allow some forward flexion of the ankle. In the conventional boot with 5 clips this will mean firm adjustment of the second top clip with a less firm adjustment of the upper or ankle clip.

(B) Skier's safety code.

Surgeons dealing with ski injuries find a constantly recurring theme in the history. This suggests that efforts to make the skier aware of the danger might reduce accidents. It is well known that the skier is most prone to injury in the first year, which in this country probably means the first two weeks of ski-ing. The accident rate graph gradually drops until after 8 years ski-ing there is only a small chance of injury. Waldie (1968) reported interesting statistics from the Scottish ski slopes which are approximately similar to reports from other countries. These statistics commend study:

58% of injuries occurred in beginners
36% in competent skiers
6% in expert skiers

54% of injuries occurred in the first year of ski-ing
14% of which occurred on the first day
33% of which occurred in the first week.

The influence of fatigue was noted by the fact that the majority of ski injuries occur at 12 mid-day and between 3 and 4 p.m., i.e. after approximately 2 hours ski-ing. McIntyre (1963) also found that most accidents occur at noon or late in the afternoon. Factors causing accidents are fatigue, inexperience, poor technique, speed and poor snow and over confidence in release bindings.

Relation of injuries to snow conditions in Scotland showed 45% in soft snow, 24% on piste and 27% on ice — other 4%.

Effect of release bindings: 80% of the injured wore apparently adequate release bindings which failed to release in 74% and opened in 26% (causing mainly upper limb injuries), 13% wore fixed bindings.

Spademan (1968) reported that safety bindings functioned in 60% of injured males but in only 27% of females.

The American ski patrol report of 1960 showed that 6 out of 10 injured skiers wore release bindings which did not release.

(1) You are particularly vulnerable to injury during your first year of ski-ing and particularly so during the first week. Injuries in Ski Schools are rare — you should ski only in Ski School during your first ski-ing holiday. The expense of Ski School is less than injury and you will reach proficiency earlier. In early years at least part of every holiday should be spent in Ski School.

(2) Study the side slip, which is the most important manoeuvre to negotiate awkward inclines and is the basis for the more advanced ski-ing techniques. The snow plough is only reliable at low speeds and is a dangerous technique to adopt at increasing speeds.

(3) Learn to fall correctly and do not hesitate to fall if in trouble. Fall whenever you feel out of control. Fall into the hill curled up like a ball with your skis together and elevated off the snow.

(4) Do not ski unless you are fit. Ideally you should attend dry ski school classes before your first holiday. Keep fit by jog trot runs. The one best ski exercise is to sit up against a wall with knees bent to a right angle until you can last 2 minutes in this posture. This will strengthen the quadriceps muscle and give confidence.

(5) Never have one last run of the day after you have left the ski school however well you think you have been ski-ing.

(6) Beware of the last run of the day when the sun sets in the spring and the piste becomes icy in a matter of minutes.

(7) Beware of the last run of the holiday which may be more memorable than you had intended.

(8) Do not run into deep snow if you are in trouble, it may turn out to be heavy or crusted and may well fracture your tibia.

(9) Beware of drift snow which can be very heavy and difficult.

(10) Beware of ruts in spring snow in the late afternoon. They will probably be frozen and dangerous.

(11) You are very vulnerable when fatigued. Always stop ski-ing when you feel you would like one more run. The majority of accidents occur after 2 hours ski-ing at mid-day or in the late afternoon.

(12) Do not ski by yourself, always have company.

(13) When you hit ice — if you cannot hold the edges then slide and keep the skis together, relax and let yourself go. Look out for patch of snow and be balanced so that you can stop or turn when you reach the snow.

(14) Release bindings — the only reliable release bindings are your own which you have learned to adjust. If you hire skis, try to understand the adjustment mechanism and do not hesitate to return to the ski shop at a quiet time of the day when the fitter will be free to answer your questions and carry out any further adjustments to suit your particular ski-ing ability.

(15) If you purchase a modern rigid boot adjust the clips to provide firm support over the heel but leave some freedom for forward movement at the ankle in the event of a fall. In the five clip boot this will mean firm support from the second top clip but more freedom from the upper or ankle support.

These rules are suggested not to curtail your enjoyment but to prevent injury and prolong your holiday. The more experienced you become and the more proficient your technique the less will be the chance of injury. Most honest experienced skiers will admit that most potentially dangerous falls could have been avoided with some forethought.

Ski-ing will always be dangerous to some extent and that perhaps may be one of the many attractions. Attention to the safety code, equipment and ski-ing technique will greatly reduce the possibilities of a serious injury and add to your enjoyment.

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A COMPARISON OF THE SCOTLAND YARD TEST AMIDOPRINE/H2O2 AND "OKOKIT"** IN THE ROUTINE DETECTION OF FAECAL OCCULT BLOOD

Concern about the possible carcinogenic properties of o-tolidine and benzidine has led to a search for less harmful reagents for use in the detection of occult blood in faeces. The new preparations — Amidopyrine and Okokit — were compared with the standard method of the Scotland Yard Test. Amidopyrine and Okokit are believed to be non-carcinogenic reagents. The active ingredient of the Ham's reagent used in the Scotland Yard Test is aminophthalhydroxide and is likewise not implicated in carciogenesis.

The chief aim of this study was purely practical in elucidating, for purposes of side-room testing, the efficacy of the three tests.

In the Scotland Yard Test, Ham's reagent (5 drops) is added to each labelled carton containing a faecal sample to be tested. This is followed by the addition of 5 drops of H_2O_2. The fluorescent blue / purple colour, indicating a positive result, appears immediately, and the shadow cast by the container wall is sufficient for the colour to be seen readily in daylight. No sample preparation is required and sequential testing of many specimens is possible using this technique.

For the test using Amidopyrine as the reagent a spatula is used to smear a small amount of faeces onto a square of filter paper. Amidopyrine (4 drops) is dropped onto the paper followed by 2 drops of H_2O_2. A purple colour develops within 1 minute if the test is positive. This colour may be partially obscured by orange staining of the filter paper by faeces and Amidopyhine when the test is only weakly positive.

"Okokit" requires the same initial step of smearing filter paper with faeces. An "Okokit" tablet is placed in the centre of the smear and three drops of "Okokit" reagent dropped onto it. A blue staining of the filter paper occurs with positive specimens. The colour lasts for a few minutes, but in weakly positive specimens may be hidden by the tablet.

Fifty samples of faeces were tested by these three methods. Eighteen were positive both with the Scotland Yard Test and with "Okokit", although of these specimens two were very weakly positive. Only sixteen positives were obtained using Amidopyrine, and the two negatives were not the same two with which "Okokit" gave a very faint positive.

** "Okokit" — supplied by Hughes and Hughes Ltd., Brentwood, Essex.

Results—

	Scotland Yard Test	"Okokit"	Amido-pyrine
Positives	18.	18.	16.
Negatives	32.	32.	34
Sensitivity	1 : 64,000	1 : 8,000	1 : 8,000
Speed of reaction	Immediate	1 - 5 mins.	Less than 1 min.

The Scotland Yard Test, which was the cheapest of the three tests, requires the least sample preparation, shows the most immediate colour change, and is the most hygienic method in that there is no need to handle the specimen. Practically it is the simplest of the three tests to perform and positive results are easily detectable with no delay. A further advantage of the Scotland Yard Test is the ease with which it may be used on the inverted finger of a glove used for rectal examination.

Of the other two tests, that using "Okokit" was considered to be the better. One reagent only was used and provided that the optimal amount of faeces is used, the test is totally reproducible.

Amidopyrine is the most complex reagent to use and may produce false negatives as staining of the paper may obscure a faintly positive result. The reagent also deteriorates rapidly.

All three tests were examined for their sensitivity using serial dilutions of erythocytes suspended in 0.45% (w/v) saline. The Scotland Yard Test was by far the most sensitive and may be considered by some to be too sensitive. However, in repeated estimations of the amount of occult blood in faeces, such a faint reaction as is given by trace amount of haemoglobin is not significant.

Thus, when the methods are compared on the bases of speed, simplicity, sensitivity and ease of interpretation — the most important factors in the side-room — the Scotland Yard Test is preferred on all counts. Next in order of preference comes "Okokit" because of its lack of ambiguity and relative ease of method. Amidopyrine is the lest preferred of the three tests.

V. E. M. Flynn, D. P. Kirk

SOCIETY NEWS

The two most significant recent events in the life of the Society were undoubtedly the Symposium on "Immunological Aspects of Cancer" held in the Autumn of 1970 and the visit by Dr. Issels from the Ringberg Klinik in the early Spring of 1971.

The Symposium, which was conceived, nurtured and presented by Ian Smith, then Senior President, attracted a great deal of attention in the Medical Press — especially in the British Medical Journal which covered the event in two consecutive issues of the magazine.

The Symposium was held in the George Square Lecture Theatre and the main participants were, "in order of appearance", Professor Klein, Dr. Allison, Professor Doll, Dr. Kinlen, Dr. Stuart, Dr. Burkitt, Dr. Bagshawe, Dr. Hamilton Fairley, Professor Alexander, Professor Sir Michael Woodruff, Professor Mathé, Professor Crile and, of course, as general co-ordinator, Ian Smith.

The list is impressive and yet equally distinguished assemblies occur frequently in the many "Congress Spots" of the world. Why did the R.M.S. Symposium have such a special appeal?

This can no doubt be accredited to all the willing and enthusiastic volunteers from the general body of the Society who cheerfully acted as chauffeurs, guides and ushers at all stages of the proceedings. As the contributors kept remarking, there was a tremendous atmosphere of interest and, although one obviously hesitates to become too effusive, goodwill throughout the whole event.

Dr. Issels' visit last February was of rather a different genre. He was invited to come to Edinburgh and present his views on the treatment of cancer to the Society. News of his rather controversial beliefs had filtered, mainly through the popular press and television, as far north as Edinburgh, but Dr. Issels appeared to have had few opportunities to present his theories to a scientifically — rather than emotionally — orientated audience.

His visit aroused a fair amount of excitement among the general press but this was played at as low a key as possible in order to avoid all the disasters of sensationalism. Nevertheless, the response from the medical public in Edinburgh (and beyond) was extraordinary. Dr. Issels gave his Address in the George Square Lecture Theatre with closed-circuit television link-ups to two lecture theatres in the Appleton Tower. A total of at least 1,100 people came to hear him.

In his talk Dr. Issels, together with Dr. Ganss, his Senior Registrar, outlined the rationale behind the techniques used in his treatment, with many slides and case histories as illustrations. Nowadays any apologist for a particular type of cancer therapy expects to meet with opposition and argument. This Dr. Issels did indeed encounter, but nonetheless, both he and the Society felt that the original intention of providing a forum for discussion had been accomplished. The Society has now warm memories of Dr. Issels, his wife and his son, and were delighted to make him a member of the Society at the dinner held in his honour after his speech.

On a less exotic, and less costly, plane were the traditional weekly meetings of the Society. Speakers at the Public Business talks and addresses included Professor Hinton, Mr. W. M. McQuillan, Dr. J. F. Munro and Dr. C. Mawdsley, with topics ranging from "Dying" and "Obesity" to "Peripheral Nerve Injuries" and "Parkinsonism". In the summer term came an address to the Society by Professor J. Laurence Pool from Columbia University, New York, the address being reproduced in part in this magazine.

At the Annual General Meeting of the Society there was some criticism of the dearth of Dissertations by members. This was due in part to pressure of time from the more spectacular events and we are glad to note that the 25th Session is providing its proper quota of Dissertations.

BOOKS

Urology and Renal Medicine. £1.25. Livingstone. J. E. Newsam and J. J. B. Petrie.

This unpretentious little book provides a concise, lucid resume of the medical and surgical aspects of urology. It allows no sharp demarcation between the two aspects of the subject — instead it presents integrated accounts of different topics, with renal physiology well-blended in and devoid of its usual mystifying aura of High Science.

It is the ideal book for the student in Final Phase who wishes to clarify and add to his existing knowledge, but would undoubtedly be useful both before and after this period of tranquillity.

Altogether it is to be highly recommended to all those interested in urology and renal medicine. S.M.R.

Medical and Veterinary Protozoology — An Ilustrated Guide. £6. Churchill Livingstone. K. M. G. Adam, T. Paul, V. Zaman.

This book was apparently designed to be used in conjunction with one of the various protozoological textbooks. With this in mind comments can only be favourable. It has a pleasing format, good colour reproduction and, of course, lots of pretty pictures.

It is however, difficult to imagine that it will appeal sufficiently to many people to entice them to purchase a copy as it obviously has a very limited appeal. A medical student at Edinburgh could well find it worthy of reference in his "Tropical Disease Week" when its many illustrations of plasmodia may well help to impinge the life-cycle of the malaria parasites on to his memory.

Apart from this, I can see very little opportunity for its use— perhaps it would be better appreciated by our veterinary colleagues.

C.L.

The Principles and Practice of Medicine. 10th Edition. £3. Livingstone. Davidson and Macleod.

If an Edinburgh medical student were cast away of a desert island, "Davidson" would most likely be the book he'd take with him — to keep in touch and to remind him of home. There can be few clinical students who do not possess a copy — it is the booksellers' hardiest perennial.

In this new edition the mixture is very much as before, but the pages are larger, the print clearer and (alas!) the paper more flimsy. There are two very welcome new chapters at the beginning of the book, one on Genetics, the other on Immunology; both of these are excellent and concise outlines of present thinking, with the usual useful lists of references at the ends.

The section on psychological medicine has no doubt failed to expand sufficiently to satisfy those with strong leanings in that direction, and this I think is all that can reasonably be expected from a conveniently-sized general medical textbook.

Sir Stanley Davidson declares in his editorial to this edition that owing to failing sight this will be the last for which he will be responsible — it is a pity he could not now persuade someone to do the service to surgical teaching that he did to medical teaching through his "Principles and Practice". A.N.F.

Hormone Assays and their Clinical Application. 3rd Edition. £5. Livingstone. John A. Loraine and E. Trevor Bell.

The Clinical Endocrinology Research Unit in Edinburgh may to some of the uninitiated be merely the place to which specimens are sent when some rather abstruse hormone level requires measurement. However, by endocrinologists all over the world it is highly regarded as one of the most important centres for research on hormone assays.

This book gives a resume of the work that has been done there in recent years. Although it obviously is an account of a specialised branch of medicine and, as such, is not of universal appeal, it nevertheless is a welcome reference volume for anyone interested in the subject as it is both clear in layout and fascinating in content. For the dedicated endocrinologist it is indispensable!

N. de P.

BOOKS

Cardiology. £1.75. Concise Medical Textbooks: Balliere. D. G. Julian.

This extremely useful book gives as comprehesive an account of all the cardiac topics as any student (and most doctors) is likely to need to know. Of especial use to the undergraduate fighting his way through a sea of gimmick-polluted "scheduled learning" and "aids to memory" cards from drug companies about E.C.G.'s is the chapter on the electrical activity of the heart which provides the reasons for the shape of a normal E.C.G. and the reasons for pathological aberrations — incidentally it also clearly explains about axis-devation, which was my own particular neurosis-producer.

There is also an interesting summary of radiology and the heart with silhouettes of radiological appearances in the presence of different disease, plus notes on cardiac catheterisation and its risks. All the standard subjects, such as coronary artery disease and rheumatic heart disease, are present, only in this book, unlike in some of the larger and more verbose texts on these subjects, they are clearly explained with the pages attractively laid out.

Towards the end of the book fly some of the canaries — the heart in glycogen-storage disease and pseudoxanthoma elasticum — but these are kept severely in control and are obviously designed to satisfy the majority rather than the avid fancier.

This is, indeed, a well-planned, attractive book which will undoubtedly be read by many.

P.A.T.

Gynaecology. £5.75. Churchill Livingstone. Novak, Jones and Jones.

There is usually something rather intimidating about American textbooks. They tend to be large, hopelessly over-detailed and, one feels, ostentatiously bursting with references.

This book is different. It has been written especially for the undergraduate, being an adapted and invigorated synopsis of Novak's "Textbook of Gynaecology". Nonetheless, it still retains a rather daunting amount of material for the completely uninitiated: there are, for instance, about 2½ pages on the Stein Leventhal syndrome alone.

This book has, however, been carefully and clearly laid out with numerous photographs (not all as enticing as that on the cover unfortunately) both in black and white and in colour, with a fairly comprehensive index. Altogether, it promises to be a useful book for many medical students — although perhaps more suited to the staid souls of Final Phase than to those taking their first tremulous steps in the subject in Fifth Year.

A.R.M.

The Voice and Voice Therapy. £4. Prentice Hall International. Daniel Boone.

The exasperated parent who is unable to tolerate his offspring's screaming will find in this book a good rationalisation for his attempts to stem the noise. Apparently children who shout too much are liable to develop "vocal nodules" leading his hoarseness; hoarseness, moreover, which increases as the nodules enlarge.

This, and other fascinating information — such as the cause of "falsetto males" — can be found in this book by Daniel Boone. It was intended for use by speech therapists and other such specialised workers rather than medical students, but it is well worth browsing through, though perhaps not purchasing a copy.

It consists mainly of quick explanations of different speech disorders, then concentrates on detailed accounts of methods of overcoming them. The chapter at the end of the laryngectomy patient provides the uninitiated who are anxious to learn more with a glimpse into the lives both of laryngectomees and of speech therapists.

Alice said that good books ought to have pictures and conversations: this book has a few, though perhaps not enough, of the medical textbooks' equivalents — diagrams and case-histories.

K.F.

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