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NEPHRITIS AND NEPHROSIS*

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NEPHRITIS (BRIGHTS DISEASE): In 1836 Richard Bright gave a graphic description of the disease which has not yet been surpassed. Yet more than 100 years after him, we are still to confess that by no means have we a cure for this condition. We are still in the dark regarding the mechanism of its progression and at times the exact diagnosis also is difficult. This unsatisfactory state of affairs is continued in the aetiology and classification of this disease too. The pathology, however, has been clarified of late.

The classification is still vague and, as Addis says, each student of nephritis constructs his own. Volhard and Fahr classified this disease as acute, sub-acute and chronic with glomerulo-nephritis as one disease in its various stages, and with a different entity called nephrotic syndrome which simulated the sub-acute form. Ellis, on the other hand, classified this differently which, though appealing, the rationale, is still not yet satisfactory.

VOLHARD AND FAHR:

(1) Acute glomerulo-nephritis
(2) Subacute
(3) Chronic glomerulo-nephritis
(4) Nephrotic syndrome—A separate entity

ELLIS:

(1) Acute glomerulo - nephritis Type I.
(2) Chronic Nephritis.
(3) Type II Nephritis (Nephrosis)

From the aetiological and symptomatological points, Ellis seems to be correct; but in the physio-pathological view he falls short and Volhard and Fahr seem to be more rational.

GLOMERULO - NEPHRITIS—TYPE I OF ELLIS:

In 80%, it is preceded by an upper respiratory infection due to haemolytic steptococci (Group A of Lancefield).

*Reprinted from the Nilgiris District Medical Association Souvenir—1960.

It is encountered mostly in children and adolescents. It starts abruptly characterised by proteinuria, haematuria, hypertension and oedema along with general symptoms like malaise, vomiting, headache and slight pyrexia. About 80% recover completely. A small percentage, less than 5, die during the attack by acute hypertensive encephalopathy, cardiac failure and uraemia. In another 3%, the oedema and hypertension persist and progress slowly with loss of renal function and death occurs from uraemia. The remaining cases improve except for protien, few R. B. C. and casts in the urine. Such persistence after six months is called latent nephritis. But slowly and steadily the renal function is affected, blood pressure rises and death occurs from azotaemia and uraemia. This is called Chronic Nephritis of Ellis. This may take five to ten years. The term latent is used, as recovery is still possible in that state.

PHYSIO - CHEMISTRY :

The kidneys have a vast reserve power. Major portion of the nephrons take rest and work in shifts. It will be appreciated that the nephrons filter 170 litres of the waste products in 24 hours and reabsorbs 168·5 litres and the necessary constituents back in the tubules, leaving only about 1·5 litres of the urine containing the waste products as solutes. Thus even if a large portion of the kidney is damaged or removed, the mechanism can go on normally but only with less of reserve power.

Before proceeding further, since one wishes to assess the damage to the kidney easily and correctly, certain tests are considered important. *The water concentration and dilution test* gives the most accurate information of all the renal function tests. This test is simple. The patient has breakfast as usual but has no more to drink till next morning. The only urine collected is the one that he passes on getting up from sleep the next morning (i. e. about 22 hours after the commencement of the test). Another specimen is taken one hour later. He is allowed to walk and a third specimen is taken after another hour. The specific gravity of the urine should be not less than 1·022 in one of the specimens atleast. In severe renal impairment, the specific gravity does not rise over 1·010. The range 1010 to 1021 represents lesser degree of renal damage. *In oedematus subjects the test is of no value.* If a man's BUN is over 100, the test should not be done. This test is of better value than urea clearance test.

Blood chemistry is of prime value as far as BUN and plasma albumin is concerned. The carbon-di-oxide combining power is necessary in azotaemic conditions.

PHYSIO - PATHOLOGY OF NEPHRITIS :

Acute nephritis is mainly a disease of children and adolescents. Apart from the necessary infection of upper respiratory tract, it is thought that there must be an allergic diathesis inherent in the child. There is a definite allergic form of reaction from the bacterial toxins. The result of this action is to produce generalised capillitis. The severity of the nephritis that follows has no relation to the severity of the original infection. There is a defenite lag of period from infection of tonsils, teeth or skin to the onset of nephritis. In a few individuals, there may occur during infection haematuria (benign haemorrhagic nephritis); but the urine contains no casts or albumin, nor are there any other symptoms.

The generalised capillitis leads to increased permeability and ischaemia of the organs. Ischaemia of the kidney is followed by a glomerulo-tubular damage and when the ischaemic phase is over, leakge occurs in the area.

The same process acting on the subcutaneous tissues results in oedema. In the heart, it produces acute heart failure and pericardial effusions. The lungs show it off as pulmonary oedema and the brain as hypertensive encephalopathy.

The accute stage is followed by recovery by epithelialisation. 80% of the children recover and about 10% pass on to the sub-acute stage of Volhard and Fahr. The albumin escapes as long as there is damaged nephrons and till they are fibrosed and go out of function. This continuous loss of proteins alters the osmotic pressure and oedema continues.

In the general order of procedure, this hydraemic stage gets less and less acute and passes on to the chronic phase. The damaged nephrons having become hyalinised and fibrosed go out of function and albuminuria stops. The kidney is able to function normally even with reduced nephrons but under certain handicaps. This phase is known as a state of *compensated renal failure.*

To carry out the normal amount of filtration with reduced nephrons, the filtration pressure has to be raised and consequently the general blood pressure gets raised. This leads to *systolic hypertension* at first and later diastolic hypertension with its attendant changes in the fundus and secondary changes back into the kidney. The heart hypertrophies to cope up with the extra work. *This systolic hypertension is compensatory and has to be maintained.*

The solutes that are passed have to be sent out at a certain concentration only. Further, the reabsorbing power of the kidney is also impaired. Hence there is polyuria with difficulty of concentrating the urine and slowly and steadily the urine gets fixed at a specific gravity of 1010. Along with this dilute urine, sodium gets naturally excreted more than normal due to the excessive volume of fluid lost. Thus there is excessive water and sodium loss from the body and in this phase there should be no restriction of water or salt intake. If they are restricted, the early onset of decompensation sets in and lack of sodium may result in prerenal uraemia.

Slowly and steadily the phase leads on to the last stage of *decompensated renal failure.* The kidney is unable to throw out the waste products and there is nitrogenous retention. The acid base balance is upset and acidosis sets in. The azotaemic and acidotic phase ends in uraemia and death.

CLINICAL SYMPTOMS :

The onset of acute nephritis is abrupt with gastro-intestinal symptoms, slight pyrexia, oedema, oliguria and dysuria. The oedema is due to increased capillary permeability, a small degree of cardiac failure, oliguria and sluggish liver which fails to neutralise the A. D. H. hormones.

Headache is common, but amaurosis is the early sign of the onset of hypertensive encephalopathy with its attendant convulsions and coma. Acute cardiac failure with dyspnoea is not common. Effusions into the various serous cavities may occur and

lungs may show pulmonary oedema. Increase in the blood pressure in the uncomplicated case is not much. This may be due to vasospasm and renal ischaemia. If oliguria is prolonged, hypertension also may be prolonged. The duration normally of acute nephritis is about four days and restoration to normal occurs. The warning of encephalopathy is headache, a sharp rise in blood pressure and fundal change from retinitis to papilloedema.

Urine: There is a generalised shut down of nephrons with oliguria. Anuria is rare. In the tubes the highly acid media precipitates the albumin into casts and the extruded R. B. Cs. and W. B. Cs. adhere to it forming blood granular casts. There is massive proteinuria. *By the fourth day* mild polyuria occurs, oedema disappears and repair takes place. The last to disappear is albumin in the urine.

Blood Chemistry: There is a slight rise in blood urea; but blood protein is normal. There is a slight acidosis and blood cholesterol in normal. Here we have to differentiate the nephritis from focal nephritis (at the height of infection), embolic nephritis (as in sub-acute bacterial endocarditis) and collagen diseases.

SECOND PHASE OF NEPHRITIS:

Instead of complete recovery, albuminuria continues till the damaged nephrons go out of function. Cylinduria is rare. There is also slight generalised oedema. This may go on to the latent phase which still can produce complete recovery in two years.

The oedema may be intermittent or steady. The cardio-vascular system is normal. The fundi are normal. The renal function tests and blood pressure are normal. This has to be differentiated also from true lipoid nephrosis and amyloidosis.

Prognosis: The disease goes on to the third stage in two to ten years. Death may occur occasionally from hydraemia or mainly from secondary infection. Death is not due to renal failure or hypertension.

THIRD AND FOURTH PHASE:

The first is *compensated renal failure.* There is compensatory hypertension and polyuria. Tests of renal function will show slow renal failure but not marked. There may be no retention of nitrogenous products. But there is a persistent loss of electrolytes which should be replaced along with water loss.

Decompensation: The slow loss of electrolytes becomes too much and waste products are retained and mount up in the blood. These result in acidaemia and proceeds to uremia. Polyuria → Loss of water → Loss of chloride → Hypochloraemia → Pre-Renal Azotaemia → Uremia.

(Salt and water to be replaced)

Loss of calcium → Demineralisation of bones → Tetany and Rickets.

(This is aided by acidaemia and calcium has to be replaced) Early in this stage the B. U. N. (Blood Urea Nitrogen) is normal. The cardio-vascular system shows gross changes. There is hypertension a necessary one and hence the heart hypertrophies greatly. The eye grounds show albuminuric retinitis. Anaemia supervenes from bone marrow depression.

Acidosis → Fall in renal function →
↓ Changes in fundus BUN rises.

Fall in CO 2 combining power.
Fall in calcium.
Fall in chloride.

Prognosis — Slow but eventual uremia and death.

This has to be *differentiated from essential hypertension,* where death is from cardiovascular accidents or coronary thrombosis and uraemia is rare and *Acute nephro-sclerosis* of essential hypertension where renal failure is late. Further, the above causes are *rare in children.*

Uraemia: Two types are met with here — the prerenal and renal with the same result. There is retention of nitrogenous products of metabolism. The first to go up is urea. As urea mounts the chain of reactions follow. Urea increases the permeability of the capillaries and diaphoresis takes place. This allows phenols and magnesium two cerebral depressants into the tissues and guanidine — a cerebral irritant. Thus coma or fits supervene. Further the excessive urea is deposited in various tissues of the body, the serous cavities and mucous membranes where it acts as an irritant. Deposits of urea in the mouth, oesophagus and stomach gives rise to bad taste, retching and vomiting. The same in the intestines causes diarrhoea—symptoms commonly seen in uraemia.

Deposits of urea in the pericardium results in uraemic pericarditis and in the lungs - pulmonary oedema and in the brain - cerebral oedema and fits.

Steadily as the uraemic condition progresses, the CO_2 combining power falls, oliguria sets in and death intervenes.

TRUE NEPHROSIS: (Ellis Type II)

Some light has been thrown on this condition by renal biopsies and electron microscopic examination of the basal membranes and the tubular basis has been brought out and yet it is not clear. We also know that there is no known method of prevention of this disease. This also occurs mostly in children.

The onset is insidious with progressive oedema. The renal function is normal and there is no hypertension. There is massive proteinuria. The generalised oedema may be alarming. Death occurs mostly by intercurrent infections. This is a progressive disease and the rate of complete cure is only above 5–15%; and more cures are seen in younger children (about 20% — Lipman).

The causes of this condition is unknown. It is said that there is an increase in aldosterone production. There is a certain interference in protein synthesis. There is a hypercholestremia (over 400 to 800 mgs%). The plasma albumin falls and is always less than 3 G%. The total protein also shows a drop. The basal metabolic rate is low. (This may be confused with myxoedema where there is no albuminuria). The great drop in the blood gamma globulin level induces infection which is the major cause of death here. Occasionally diuresis occurs spontaneously and complete recovery ensues.

Management: The treatment of these conditions is still empirical and symptomatic. The daily urinary output should be recorded along with the blood pressure and weight of the patient. The blood urea should also be estimated.

IN THE ACUTE PHASE:

1. The child should be completely at *bed rest* for 4 weeks. Proteinuria should be the guide for its duration.

2. **Diet:** Restricted proteins as well as salt and fluids. In children with fever the fluid is not to be restricted strictly.

Fruit juices are best avoided for the first 48 hours and in severe cases with fever and vomiting, as it has a high content of potassium. It may be added once diuresis sets in. Starvation is good for the first 48 hours with sips of glucose water. Calorific value should be basal. If the case is not acute, solid food could be given.

A low protein diet is as follows:

Milk 7 oz.; Bread 3 oz. (or one large Iddly) Rice and vegetables.

Later proteins 20 grammes, dry type of food with low sodium chloride and water 1000 ccs. with calorific value of 1200 is to be given. When oedema subsides, a generous diet with more proteins could be given but still keeping down salt and fluids.

Normal diet is resumed ten days after guiac lest is negative (i.e. no haematuria microspically).

3. Antibiotics are to be given in the febrile cases the best being Penicillin in low doses, as the urinary excretion is poor.

4. Vitamin E by parenteral route shortens the duration of the illness and also prevents complications to some extent.

5. Antihistamines — Value is doubtful.

6. Tonsillectomy if of no use; but later may prevent recurrence.

7. Diaphoresis, purgatives, diuretics are strictly contraindicated.

COMPLICATIONS:

Hypertensive encephalopathy — occurs in less than 5% of the cases and death occurs from heart failure or fits. This is an emergency and requires rapid and adequate treatment and full recovery is possible in most cases.

(a) Magsulph is useful in children orally in doses of 1 to 2 drachms. It is given not for purgation or dehydration but for sedation. This could abort an attack.

(b) Paraldehyde 2-4 c. c. Intramuscular or more as necessary to control fits. Phenobarbitone is to be avoided.

(c) Blood urea being low, *ganglion blocking agents are indicated* in low dosage as there is poor urinary clearance.

Ansolyen 1/2 mg. or one mg. I. M is useful and may be repeated but keep the child propped up in bed.

(d) Venesection may be outmoded but occasionally is a life saving measure and about 200 to 250 c. c. could be removed safely in a child.

Anuria—is rare, and Bull's regime or its modification is to be resorted to. Surgical measures are of no value.

Acute cardiac failure—is most uncommon and mostly is fatal as the normal cardiac stimulants and glucosides are of little value nor any diuretics could be used in this condition obviously. Here again venepuncture is of most value.

AFTER-CARE OF NEPHRITIC CHILDREN:

The children have to be watched for one year. They must avoid exposure, wet clothes, overactivity and should have a periodical checkup of urine, blood, blood pressure and fundus examinations.

In the sub-acute and latent phase only diet is important and in the strictly latent phase, there need be no restriction of diet. High proteins should be given in the sub-acute stage. But all infections should be treated carefully and a periodical check up of blood pressure, blood and urine should be conducted every three months for two years.

CHRONIC PHASE:

This phase comprises the compensated and the decompensated phases. The writing on the wall is there and one has to keep it as far away as possible. The natural growth of children is restricted and their activities are curtailed. The urine concentration test is near normal to start with but goes down rapidly. These patients should be up and about as far as possible at their normal avocations.

In the *compensated phase*, the diet should be ordinary one. A slight restriction of protein is indicated but there should be *NO restriction in salt and water.* The hypertension is compensatory and should be maintained unless it is alarming. Blood urea, blood sodium, blood calcium and urine examination should be done frequently along with examination of fundus and taking of blood pressure. In a few cases salt may have to be augumented if there is hyponatraemia or hypochloraemia which itself may produce uraemia or even convulsions.

In children normal schooling is advocated.

There is a concomitant anaemia. The haemoglobin falls with the rise of blood urea nitrogen (BUN) and small transfusions are of great help. Convulsions may occur from hyponatraemia or hypertensive encephalopthy, the former being remedied by salt intake and the later treated as enlisted before. But blood letting is contraindicated here, as these patients are already anaemic.

Tetany may occur from loss of calcium and it has to be replaced. Acute infections, if supervening, have to be treated meticulously, as they may produce hypopotassaemia with its attendant dangers. Penicillin is the best drug to be used here. Uraemia is terminal and has to be treated as anuria.

TYPE II NEPHROSIS OF ELLIS OR NEPHROTIC SYNDROME:

In this stage of hydraemia, the main point is prevention of water retention. The fluid intake is to be restricted drastically as also salt. We have to raise the *Colloidosmotic pressure.* A high protein diet is to beg iven. Plasma infusions are of doubtful value. Plasma expanders like polyvinyl pyrrolidine (Dextraven) are of some use. For *removal of fluid* mercurials are the best. The chlorthiazides are also of value. The idea should be to remove fluids as long as the body reacts to these drugs. If oliguria is extreme and there is no response to these drugs, their use should be restricted.

A. C. T. H. AND CORTISONE:

Under the view that nephrotic syndrome is allied to the collagen diseases A. C. T. H. was tried. It was found that the œdema increased alarmingly; but when the drug was stopped, there was a dramatic diuretic response lasting many days. ACTH has been replaced mostly by Cortisone and its later derivaties like Prednisolone and Dexamethasones. Cortisone or its other products to the equivalent of 200 mg. of Cortisone is given daily for 4 to 5 days. The œdema increases,

the face becomes moon-shaped. The drug is gradually reduced and diuresis takes place copiously. A plain antibiotic like Penicillin may be used concurrently to guard against any intercurrent infection. One has also safely used the milder diuretics at the end of the course to augument the action of steroids. This may completely bring normalcy in the child but a small maintenance does of cortisone may have to be given for a few weeks.

REFRACTORY CASES :

Southey's tubes and acupuncture of the œdematus tissues have been tried in a few cases. Intercurrent infection like *measles* has produced remarkable remissions but its imitation as a cure is rather dangerous.

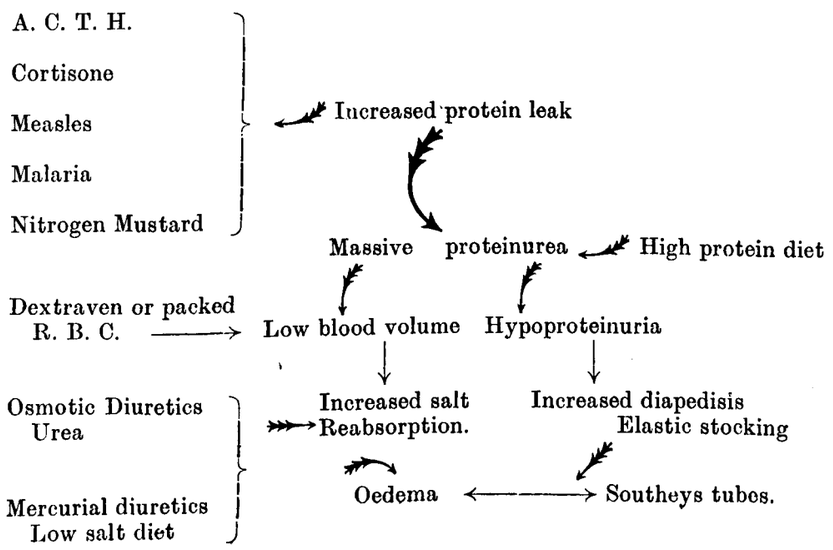
NITROGEN MUSTARD :

In four of our refractory cases which were not amenable to the above regimen and who were hopelessly ill, mustine was given in four biweekly doses. The response in three cases was good out of whom two had a year's remission on follow up. This method is dangerous and has no true scientific basis and is not to be attempted generally.

In nephrotic patients, routine plasma, albumin, and urine estimations are to be done along with urea clearance test. Routine weight and examination of urine is to be done. When remissions occur from the hydraemic state, the children should be sent back to school. Great care should be taken to avoid intercurrent infections.

After many relapses during the course of many years, a small proportion recover completely, but others may go on to chronic nephritis described before. It is rather a happy note that nephrosis in children have more than 20% recovery rate.

The treatment of nephrotic syndrome could thus be summarised as :



SUMMARY AND CONCLUSION :

The genesis, progress, course and management of nephritic and nephrotic diseases is discussed briefly here. The polyglot attack in the treatment clearly brings out the ignorance that exists about these diseases even in the present century of advancement and one has to end with a note on the unsatisfactory state that still exists. It is hoped that more light would soon be thrown on the kidney diseases by modern studies on kidney function with Radio-active Isotopes, when it is hoped treatment may get more rationalised.

VERY EFFECTIVE AUTO-SUGGESTIVE LONGEVITY PRAYER

Every day, in every way,
By the help of loving God,
I'm getting better & better,
Also getting healthier & happier,
For, all my organs & glands normal,
My Heart & Soul are great Powers,
Amazingly triumphant every hour,
I'm the master of my Fate,
I'm the victor of my Will,
I always abide by Nature's laws,
I'm free from drink & smoke,
I'm free from anger & worry,
Hence I'll surely live a century,
All because my sub-conscious mind,
Readily yields to my conscious mind,
I repeat this prayer just before & after sleep,
The best propitious time for it to act and keep.

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THE SUCCESSFUL GENERAL PRACTITIONER

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The role of a general practitioner has been undergoing constant change for the past fifty years. Originally nearly all doctors were general practitioners, and there were many eminent men among them. With the progress of medical science - a progress that has been both wide and deep and has opened up new and unexplored vistas - it has become impossible for one to be conversant with the entire field of medical science. Thus the era of specialisation dawned. An increasing number of doctors took to specialisation, and a steadily decreasing number became general practitioners, with the result that it was feared that there may not be any more general practitioners in the country. But wisdom prevailed and now there is a welcome trend towards a hormonious balance between the specialists and the general practitioners.

The general practitioner, to-day, has a definite role to perform and an important function to discharge. It is estimated that he can care for 85% of all illnesses, either alone or in consultation with a specialist. The general practitioners are spread all over the country, while the specialists mainly concentrate in the cities and big towns. It is on the general practitioner that the sick people depend upon for guidance and it is he who attends to all the various kinds of maladies from the simple whitlow or abcess, to acute and serious conditions such as acute abdomen. Further, the general practitioner makes treatment available at or nearer homes, in town or out of the way places, day or night as circumstances demand, unlike the specialist who can and will render service only in his clinic. Hence, the general practitioner shoulders the ultimate responsibility for the care of the sick and thus the health of the nation. Hence, it is appropriate and opportune to consider what constitutes a successful general practitioner.

Let us ponder over what contributes success to a general practitioner. Is it the large number of patients that turn to him or the amount of money that he can earn from his practice or is it his ability and skill to attend to various kinds of illnesses or is it his pleasing personality or the esteem with which he is held by his patients? It can only be said that it is a combination of all these virtues in some proportion or other. We will consider in detail the essentials of a successful general practitioner.

The general practitioner should possess the personality which wins the confidence of the patient. He maintains intimate contact with his patient and his family and maintains the important doctor-patient relationship. For this he should have good bedside manners which essentially consist of personal interest, kindness, sympathy, friendliness, understanding, cheerfulness and humour. He should be resourceful and tactful to understand and study the needs of his patient. The patient is studied as a

whole, as a human being with a mind and not merely his affected part or organ. A pleasing personality does not mean to please every one, but it needs firmness, if need be, as in fussy patients. Another virtue for a good doctor is a good memory for names. Patients admire and like the doctor who remembers them and are kind and sympathetic and who evince interest in their welfare and they choose him as their family doctor. The patient should feel and look upon his doctor not only for the relief of his physical suffering but also as a guide, adviser and a companion in his mental affiiction and worries.

Next to his personal qualities, come his professional ability and skill. His professional knowledge should be on sound basis and he endeavours by his constant desire and efforts to keep abreast with latest advances in all the different fields, of medical science. He keeps up the standard of his professional knowledge and skill by visiting hospitals, clinics, seminars, etc., and by reading carefully selected journals. If the family doctor is to contribute his proper share to the well being of his patients, he must know the recent developments as they occur not only in diagnostic and therapeutic technics but also in social madicine, industrial medicine and so on. Apart from keeping in touch with recent advances, he needs also much revision. This he does by reading journals, the only method available in rural areas or by attending to one hour of prearranged lectures or study every week or by attending clinical meetings, or by discussion with his colleagues and specialists. Like his professional knowledge and skill, which should be adequate to do real service to the sick, his surgery should be well equipped with necessary appliances, instruments, and personnel, so that at no time he should feel helpless especially in emergency cases.

The general practitioner is learned in humanities too, to enable him to study the patient's environments, way of life, and also the factors that influence his mind and body and the needs of his patients. His association with the patients is real and intimate, and he is able to find out the contributing factors to his illness.

The general practitioner should be available for visits day and night, in town or out of the way places and on holidays too, if patient's condition requires. He gives his best thought and attention to his patients. He treats them during their illness and is responsible for continuing care of his patient. He advises his patients and their families on their health matters and plays a vital role in social and preventive medicine. Aware of the conditions under which the patient lives and works and at the same time being ready and willing to help in all their difficulties and to share their joys, he is not merely a skilled professional, but an expert guide and a beloved companion.

His prime duty is to assess the clinical signs and symptoms by his clinical acumen and to arrive at a diagnosis. He should be well versed with the simple clinical laboratory investigations, which he can provide in his own surgery. The clinical assessment for diagnosis is highly imperative especially where facilities for laboratory and other aids for diagnosis do not exist and consultation with a colleague or a specialist is not possible as in rural areas or in out of the way towns or the patient cannot afford them. When he is

afronted with a case in similar umstances, he arrives at a possible nosis and exercises his knowledge skill to do his best for the ery of his patient. Where facilities exist, he calls in a specialist for consultation in the best interest of the patient. False prestige or other considerations should not prevent him from doing so. He helps the specialist with his knowledge about the patient, his antecedents and the present history and course of illness, etc. He follows the case until the recovery of the patient. By meeting the specialist and discussing with him, he can improve his knowledge in that particular field. It is worth mentioning that he forms a nexus between the patient and the specialist. Sherer compares him to a central executive such as a company chairman or a cabinet minister with a number of specialised advisers whom he consults on specialised problems.

The general practitioner resists temptations for politics and fends off fascination for a prospective fortune from industry or other avocations. Fear that his own profession may not provide enough for him and his family and for his old age may distract him to more enterprising avocations. Distracted into fields other than his own, he cannot be a true general practitioner, a person of dedication to his fellow human beings and for relief of human suffering.

He upholds his prestige and that of his colleagues. Professional jealousy or personal pride should not allow him to talk lightly of his colleagues. He scrupulously avoids criticisms about his professional brethern, especially with his patients, lest it affects the esteem, affection and the confidence the patients have in him. He is ready to realise his errors and helps others when they need them.

There is one thing mentioned against the general practitioner that he is not able to keep abreast with the latest advances in the field of medical science and his basic knowledge also deteriorates for lack of revision. It is a pity that he is relegated to the background with the growth of specialisation. But the real need for a family doctor, who can render assistance at times of need, in or near the patient's home, has given fresh impetus to the general practitioners, who also now assert their rightful place in the maintenance and preservation of the health of the citizens. The vast medical science with its progress so deep and wide makes it impossible for a general practitioner to master it. Hence, he can form a group practice, in which a few general practitioners can join together and each can improve his knowledge and skill in a particular branch of medical science of his own choice and help one another. But the general practitioner should guard against assuming the role of the specialist and confine himself to the role of a general practitioner pertaining to that field.

To sum up, the general practitioner is defined as a doctor who is in direct touch with his patients, who accepts continuing responsibility for providing or arranging them general medical care, which includes the prevention and treatment of any illness or injury affecting the mind or any part of the body. He is the real family doctor — the Friend, Adviser, Host and Guest of his patients. Through the long years of his life and even during his old age, he continues to dedicate himself to the sacred principles of his chosen profession. With all this, the general practice remains the most exciting and satisfing life any man can ask for.

NUTRITION AND HEART DISEASE*

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I feel that I should preface these remarks with a note of apology: I have not worked, I am not working now, and I do not contemplate working in the near future on heart disease. My interests are first as a potential sufferer. According to the statistics of the World Health Organization, my moving from Paris to Boston and becoming an American has increased my chances of dying of heart disease by a factor of almost seven times. I am also interested as a nutritionist, because there is increasing evidence that nutritional factors are of importance in heart disease. Finally, I am interested as a research worker; while I have not worked on heart disease as such, I have worked on some of the regulations of metabolism, particularly on regulation of food intake and regulation of lipogenesis, and I believe that these subjects are not only factually relevant to the problem of heart disease but may furnish a guide as to possible methods of attack on some of the more vexing aspects, particularly cholesterol metabolism.

Let us start by examining the possible relationship of obesity to heart disease and review the facts; my little pause between "the" and "facts" is meant to convey that facts not only are few in that field, but also are not necessarily in agreement with widespread opinions.

According to some recent speakers and to many of the recent press releases, the association between obesity and coronary heart disease is so striking that it has always been observed by good physicians. It has been said that (making allowance for the fact that coronary heart disease as such is of recent diagnosis) Hippocrates himself had recognized the relationship between something like coronary disease and obesity.

When you look into this, as Keys has done, you find extremely little on the subject of obesity in Hippocrates. Actually, there are only three remarks on this subject in those of his treatises which have survived: one on the infertility of fat women among the Scythians, and two sentences about obesity in "Aphorisms." Aphorism number 35 says: "In all maladies, those who are fat about the belly do best. It is bad to be very thin and wasted there".

This remark is never quoted in lectures on obesity and instead there is very frequent quotation of aphorism number 44 which says: "Sudden death is more common in those who are naturally fat than in those who are lean". So much for the ancient writers.

The evidence of a strong correlation between obesity—or, rather overweight—and heart disease, first noticed by Rogers in 1901, and so well presented by Dublin, Marks, and their collaborators,[1] does not need to be recalled here. One may recall that mortality from all causes in overweight men, is 150 per cent of the normal. In overweight women it is 147 per cent, and the difference

* By Kind Courtesy of American Journal of Public Health — Volume 50, Number 3.

is particularly striking for heart disease—142 per cent for men, 175 per cent for women.

The implication usally drawn from these data is that obesity causes heart disease. This is a type of reasoning which is dangerous: if one looks at the same table, one finds that the mortality from tuberculosis among overweight insured men and women is very low—21 and 35 per cent, respectively, and that suicide is also very significantly lowered—70 per cent, as compared to the normal mortality.

Yet nobody has suggested that overweight as such protected against, say, the infection by tubercle bacillus: It has been shown by a careful English long-term study that the association between overweight and decreased mortality from tuberculosis is not an artifact due to the effect of a wasting disease, but the result of a decreased tuberculosis morbidity among obese individuals.[2]

Similarly, the decreased incidence of suicide among the overweight has never been ascribed seriously to obesity as a causal factor: inability to drag yourself to the window or get through it, or impossibility of getting strong enough rope to hang yourself if you are overweight.

It is obviously more reasonable to assume that there is an association there, that some of the factors that make for obesity also make for decreased tendency to suicide. So I think we should be wary of interpreting too literally such correlation through causation, even though the fact is uncontrovertible that the association is striking in any statistics which involve a large enough number of cases.

Now by contrast, when one looks at reports of smaller and supposedly more careful studies, one finds almost every opinion as regards the possible association of obesity, overweight, and heart disease. For example, in a study of five and ten-year survival rates at the Mayo clinic conducted on about 6,000 patients, it was found that by and large, the obese patients did better than the normal—70 versus 59 per cent surviving after five years, 44 versus 35 per cent after ten years.[3]

Again this does not necessarily mean that obesity protects against the consequence of angina pectoris; in fact, it could suggest almost the reverse; that obese individuals may be more likely than nonobese subjects to have mild cases (from which they then survive). Again considerable difficulty is encountered in interpreting results.

Results of necropsies are by no means clear either. To give some examples, a study by Rosenthal showed no correlation between atherosclerosis and obesity.[4] A study by Wilens showed a strong correlation between obesity and overweight; and furthermore included the interesting suggestion that a wasting disease or inanition actually decreased the degree of atherosclerosis even when this was already established.[5]

Some of the studies done during the war, particularly in Finland and in the Netherlands, showed a correlation between decreased caloric intake and decreased atherosclerosis.[6,7] Actually, there were many variables other than caloric intake even among nutritional factors (qualitative as well as quantitative changes took place as a result of dietary restriction).

In a study which Wright* conducted in about a thousand patients, there was, if I am not mistaken, very little correlation between weight and myocardial infarction.[8] The study which Keys conducted in his laboratory on a group of about a hundred coronary patients showed no difference in weight distribution from that observed in a hundred similar nondiseased patients.[9]

It has been suggested recently that the correlation between fatness and heart disease is better than that between weight and heart disease. Recent findings on small groups of patients have indeed tended to show that even though there was no difference between the weight of coronary patients and those of normal individuals of the same age, the coronary patients were fatter as detected by calipers. Such studies are, however, difficult to interpret because one of the effects of coronary disease is probably decreased physical activity and this could account for increased shift from muscle to fat. Considerations of body composition may be also involved in the findings of Breslow on the relatively superior health record of overweight longshoremen.[10]

In a study reported by Wilkins, Roberts, and their collaborators,[11] in the October, 1959, issue of Circulation, there was a strong correlation between weight and atherosclerosis as seen at autopsies. The study done in Framingham by Dawber, Moore, and Mann[12] suggests a definite degree of correlation between overweight and heart disease when the population itself is used to establish its own standards. Initial results indicate that very overweight individuals are more prone to have heart disease than very underweight individuals, with a wide zone in between where it is difficult to observe a correlation. It also appears that the correlation may be more directly one between overweight and hypertension with the effect on heart disease secondary to the occurrence of hypertension.

Generally speaking, I think that it is fair to conclude from these and from many other studies that there is a correlation between overweight and heart disease, but that there are a number of qualifications which must be kept in mind. First of all, overweight appears to be only one of the many contributory factors involved. If the sample is too small or the selection of the experimental group is slanted one way or the other, the correlation may disappear. The correlation is good when one looks at population groups made up largely of very thin peole, such as the Bantus or the population of southern Italy—and particularly in populations which have, like these two, very low serum cholesterol.

The correlation is also good if one compares extremely obese with normal individuals. However, between these extremes in weight, unless there is a very large group, such as those described by the insurance companies, or that studied in the United States Army which was made up of 22,000 officers,[13] the effect of obesity is not that definite and may be lost. Therefore, I believe it is a fair conclusion that in very large groups obesity is a factor in heart disease, which justifies weight reduction as a preventive measure, at least for most individuals.

Now if we try to go a little deeper into the problem, and think of

* Irving S. Wright, M. D., Cornell University Medical College, New York, N. Y.

possible mechanisms, we encounter the possibility that obesity has a differential effect on atherogenesis and on coronary disaster. For that matter there appears to be a definite difference between the effect of obesity on the coronary artery and the effect of obesity on the aorta. The correlation between obesity and atherosclerosis is much stronger for the coronary artery than it is for the aorta.[11]

We also have the possibility that both obesity and heart disease are linked by association rather than by causation. One possible link is the role of exercise and physical activity. We have shown first in experimental animals and later in man [14-16] (and this has been confirmed by a number of laboratories in other parts of the world) that contrary to general thought (e.g.,[17,18])—while the increase in physical activities for an already active individual is accompanied by an increase in food intake, a decrease in physical activity below a certain point is not accompanied by a decrease in food intake, and the individual becomes fat instead.

Now, inasmuch as Morris[19,20] in particular, has shown that there may be a strong correlation between lack of physical activity and incidence of heart disease, and there is a strong correlation between lack of physical activity and overweight, we have to acknowledge a possibility that both overweight and heart disease may be in part due to the same factors, rather than one "causing" the other.

Another possibility is the following: We know about eight or ten different forms of obesity that are present in exprerimental animals. These have been divided into two classes.[21,22] One we have called regulatory obesities, which are essentially due to a dysfunction in the regulating mechanism—for instance, hypothalamic obesity, or the obesity one can produce by punishing an animal unless it overeats. Such forms of obesity in experimental animals are not accompanied by hypercholesteremia. By contrast, the metabolic obesities occur where a primary metabolic lesion causes the hyperphagia. Examples are the obese-hyperglycemic syndrome, the so-called yellow obesity, and the obesity due to ACTH secreting pituitary tumors. Such obesities show hypercholesterolemia or disorders of cholesterol metabolism as part of the syndrome. Thus it is quite possible that in speaking of "obesity" and "heart disease" and implying that there is one obesity and one heart disease we are using a primitive type of thinking. There are a number of forms of obesity which have various relationships to the many aspects of the disease process.

To give but one last example: in hypothalamic obesity, although there is no effect on cholesterol, prolonged hyperphagia affects the kipneys[23] and a long term hypertension without any concomitant effect on cholesterol metabolism may be the result of hyperphagia.

Finally, there is the possible interrelationship between genetics and the effect of fat diets in animals. Different types or strains of animals react quite differently to a high-fat diet.[24,25]

The relationship of atherosclerosis and heart disease to nutritional factors other than calories has been discussed in at least three different ways: (1) The possible direct linking of coronary disease to nutrition and particularly to fat in-take. (2) The

possible linking of nutrition to cholesterol metabolism, leading to a possible relation of cholesterol levels to coronary disease. As Berry has commented about this hypothesis: The first half of the evidence (that leading from nutrition to cholesterol, the "halfway house") is trodden broad and fat, but the second half, the road from cholesterol to heart disease, is as yet poorly blazed. (3) The possibility of a relationship of fat intake either to atheroma or thrombosis as components of coronary disease with emphasis on clotting time; this is supported by claims of increased and more prolonged rise in blood lipoids following ingestion of fat in coronary patients.

The first hypothesis is largely based on surveys. These have been widely discussed, so that we shall not spend much time on them except to recall that Keys found[26] (or thought he had found) a relationship between heart disease and total fat intake. Yudkin[27] found no correlation, working on the same data. Yerushalmy and Hilleboe[28] found that the apparent association decreased if additional countries were considered. Bronte-Stewart found a correlation with the ratio of solid fats to oils, but left out a number of countries.[29] Hollingsworth could find no correlation between the increase in incidence of heart disease in Britain between the pre- and postwar period and changes in fat consumption.[30] Among others, one of the most recent comparisons — that of Jolliffe and Archer[31] is particularly interesting, in that it is more sophisticated and tries to relate death from heart disease not to one component, but to the interaction of various components.

Even if we limit ourselves to experimental studies on human subjects (as distinct from epidemiological studies– or experimental studies in animals), we find ourselves faced with the following collection of conclusions:

Keys suggests that the cholesterol-lowering ability of certain fats is related to the amount and ratio of the saturated and polyunsaturated fatty acids they contain. The saturated acids raise the serum cholesterol level and are twice as active per gram in raising it as the polyunsaturated or unsaturated acids (which have the opposite effect) are in lowering it. Monounsaturated acids have very little effect.[32]

Ahrens appears to believe that the lowering of serum level is related to the total degree of unsaturation and to the iodine number, presumably with the mono and polyunsaturated acids all being effective in proportion to their degree of unsaturation.[33]

Kinsell concludes that the polyunsaturated acid (linoleic) is the primary factor.[34]

Bronte-Stewart states that the addition of a saturated oil to a highly unsaturated oil does not necessarily elevate the serum cholesterol level produced by the unsaturated oil.[35]

Beveridge claims that hydrogenation of corn oil, which decreases the unsaturation of this fat, has very little influence on the ability of corn oil to lower the serum cholesterol.[36]

Others have found that by contrast the effect of tung oil in increasing the serum cholesterol level cannot be accounted for by its fatty acid content.

Hegsted thinks he can demonstrate that the product obtained by multiplying the concentration of saturated acids by that of linoleic acid has a high negative correlation with the serum cholesterol levels.[37]

In other words, there is a wide diversity of views, but an almost general acceptance of the idea that by and large saturated acids tend to elevate the level of cholesterol.

There are many other factors which we do not have time to discuss. They have been reviewed recently by Portman.[38] Among others, these are dietary cholesterol, a question which has just been revived by Beveridge[39]; the level and kind of protein in the diet; the very interesting suggestion that purines and pyrimidines may be important in determining serum levels of cholesterol; the posible influence of various carbohydrates: sucrose, lactose, and starch seem to have very different effects on cholesterol levels at least in experimental animals; fiber may be a factor. And then a number of minerals have been involved–vanadium, cobalt, magnesium, calcium, and iron. Vitamins, nicotinic acid, pyridoxine, pantothenic acid, choline, B-21, inositol A, C, and their possible mutual interactions have also been mentioned.

Well, what can we conclude from all this? The thing which strikes me as a physiologist interested in regulations in general is how little has been done on the problem of regulation of blood cholesterol. First of all, is this level regulated by a mechanism insuring its near constancy and reacting to decrease and increase, or is this level just a resultant of various influences, some increasing it, some decreasing it, but none of them subjected to a feedback control? There are very few studies pertaining to that problem. One which deserves to be cited is the interesting work of Siperstein and his colleagues showing how cholesterol inhibits the synthesis of new cholesterol, probably at the mevalonic acid stage.[40] But even that is only a small part of the feedback; and the first step should be to get some idea of the general way in which the regulation works even without knowing the details of the mechanism. Is the regulation of blood cholesterol precise? How long does it take to get back to a given level? How is it effected by general physiological phenomena? And so on. This is the kind of study which has been well done for glucose and which we have tried to do for food intake. It has also been done quite well for blood pH, and for water intake. As far as I know at present nothing quite like this is being done on blood cholesterol.

It seems to me that animal studies should concentrate on this question because that is what laboratory animals can best be used for — to find fundamental mechanisms. By contrast, laboratory animals are very poor instruments in general to predict small or long-term effects as they may apply to man. This is where human studies are essential and where it would be particularly interesting (even though I realize the difficulties are formidable), to see what the very long-term effects of, say, feeding the various unsaturated fatty acids would be.

From the point of view of treatment, we are, I believe at the point at which diabetes was before the discovery of insulin; and perhaps the only way open to us at present is to recommend a return to the simplest virtues: abstemiousness in diet (low calories, not too much saturated fat, not too much fat for that matter); enough physical activity; if possible, avoidance of unnecessary psychological stress; and I presume that Dr. Hammon will add–less smoking.

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ELECTRICAL PHYSICIAN*

DR. G. NOVINSKY, Physician; V. BYKOV, Phisicist and A. GRUNDMAN, Desighner.

The patient suffered for a long time from headaches and dizziness. After examining him in a special neurological hospital, the doctor surmised that the patient's complaints were caused by neurasthenia. However, soon he arrived at a new diagnosis — migraine and subsequently arteriosclerosis. But none of these diagnosis was correct. The true cause of headaches was discovered by a simple logical apparatus designed by the authors of this article. When the data about the patient was inserted in the apparatus, it gave a different diagnosis — swelling of an auricular nerve in the brain. The operation confirmed the diagnosis.

Why the diagnostic apparatuses are necessary? Frequently physicians meet with considerable difficulties in establishing correct diagnosis of illnesses and the analysis of all symptoms, which at times form the most intricate combinations. Having examined the patient and collated results of various analyses, X-ray readings and other data relating to the patient, the doctor finally makes a diagnosis based on his logical conclusion. In this diagnosis he must take into account all data received, draw on his experience in the past, recollect what he has learned from various sources on certain diseases and of similar cases dealt with formerly.

If, for instance, the doctor has a case when the symptoms of the illness are expressed in fits of headaches, dizziness, nausea or vomiting, but there is no pain in the eyes, then he diagnoses migraine.

As times the solution of logical tasks in diagnosing illnesses is very difficult. The doctor's all-embracing logic, just as the logic of any man, lacks sufficient quickness, accuracy and automatism. Trusso, the famous French clinicist of the nineteenth century, was absolutely right when he declared that most of the diagnostic errors made by the physicians are due not to lack of their knowledge, but to the fact that some of them are unable to recollect at the right time certain substantial symptoms of illness and to mobilize in time all their knowledge and experience to make correct diagnosis.

It is at this juncture that cybernetic mechanisms must come to the doctor's aid — logical machines for discerning separate groups of illnesses. While not replacing the doctor in his thinking, they will help him in checking the correctness of their diagnostic deductions, compel him to think deeper and not to feel satisfied with what has been accomplished.

Recently we proposed a new diagnostic device for defining illnesses accompanied by headaches. This device took into account 5,000 of such cases. During the statistical treatment of the data, we sorted essential symptoms from accidental, and among the latter important from the unimportant.

* By Kind Courtesy of the U. S. S. R. Embassy in India.

Essential symptoms are those without which the given illness does not occur. Thus in the case of migraine, it is fits of headaches. Accidental symptoms are those which may or may not be. Thus, for instance, migraine may or may not be accompanied with dizziness, nausea and vomiting. But of these symptoms, nausea and vomiting are important, and dizziness is of secondary importance. Results of the study of several thousand cases of illnesses are quite sufficient for making a correct diagnosis.

The whole device consists of a small box, on one side of which 29 - one contact on-switches (to conform with the possible number of symptoms) are attached and one 23 - contact change - over switch (to conform with the number of possible illnesses). Within the box 23 panels are set in, where each symptom is represented in the form of an electrical resistor. By rotating the change - over switch, any of the 23 panels may be switched into an electric circuit of a battery and galvanometer. By the turn of one - contact switches on the panel, resistors are joined to the circuit.

The resistors representing the essential symptoms are switched into series consistently, while the accidental ones having the same value in parallel, so that the hand of the galvanometer deflected in proportion to the number of accidental symptoms.

Proceeding to diagnosis with our instrument, the physician turns several one contact on - switches, thus introducing into the device the symptoms of the illness. Then he begins to revolve the 23 - contact switch, passing all its 23 positions and watching until the deflection of the galvanometer hand is the greatest. The position of the switch which caused it determines the name of the illness.

The absence of an essential symptom will cause no deflection of the hand. If the largest deflection of the hand is the same at several positions of the switch, this means that the final diagnosis cannot be made and additional examination of the patient is needed. The physician can see, however, within what range the diagnosis can vary. In theses cases the instrument helps to determine what additional examination is needed for the patient.

In determining a limited range of diseases such as nervous, ear, infantile and other diseases rather simple logical apparatus can be quite useful. A preliminary test of the apparatus described was successful and showed that application of such instruments is sure to help the physician in such complex and subtle art as diagnosis is.

ABSTRACTS AND EXCERPTS

FAMILY STUDIES BY STUDENTS: In 1958 and 1959, the fourth-year medical students at Lucknow, who number about 160 per year, were each alloted a family having a registered case of pulmonary tuberculosis and were asked to write a medical and social history of the case. Before they did this, they were given a schedule of study showing what information they must seek, and they attended a demonstration at which a case was presented in accordance with this schedule. Health visitors from the tuberculosis department introduced the students to the families, and the students consulted the departmental records of the cases. Each case-history was evaluated by me and the student was called to clarify points in the history and discuss the study. Occassionally, he was asked to revisit the family or consult the records again.

The students on the whole did an excellent job, and some made instructive sketches to show the environment in which the families were living. Their interest was not confined to writing the medicosocial case-histories; where necessary they examined the sputum of patients and contacts, and took the contacts to the tuberculosis clinic for screening, Mantoux testing, and B. C. G. vaccination. A few patients who had defaulted from the clinic were persuaded by the students to revisit it for the usual check-up. Some of the students, going beyond their task, visited the neighbouring families to find out whether there were other cases of tuberculosis and what was being done for them.

(Dr. B. G. Prasad - Lancet - October, 1960 - p. 757)

"PROGRESSIVE PATIENT CARE": Dr. J. D. Spillane, M. D., F. R. C. P., Consultant, Cardiff Royal Infirmary, writing on NEW AMERICAN MEDICAL SCHOOLS (B. M. J. September 10, 1960), says "You cannot travel far in the U. S. hospital world nowadays before hearing the phrase "progressive patient care" (P. P. C.). It is a concept which has aroused considerable interest and is in operation at Stanford and Gainesville. The U. S. Public Health Service has published in 1959 a booklet of 80 pages entitled "*Elements of Progressive Patient Care*" which is based on studies made by a team which was appointed in 1956. P. P. C. is defined "as the organisation of facilities, services and staff around the medical and nursing needs of the patient". Patients are grouped according to their degree of illness and need for care. The general hospital of the future is envisaged as a community health centre concerned with the care of long-term as well as short-term patients. There are five elements in the present concept of P. P. C.; four are contained within the general hospital: (1) *the intensive care unit*, where critically ill patients are concentrated regardless of diagnosis; (2) *the intermediate care unit*, where the average hospital patient is to be found; (3) *the self care unit*, for ambulant patients who are physically self-sufficient; and (4) *the long-term care unit*, where skilled medical and nursing care play an important part.

It has long been obvious that not all patients entering a general hospital require the full resources of a traditional ward. The indignity of being routinely put to bed has been surprisingly tolerated by the public. The patient who is admitted for diagnostic investigations or for treatment such as physical therapy or radiation, the diabetic in for regulation and advice, the post-coronary patient who requires observation of his tolerance of activity, the patient with mild emotional disorder - are examples of hospital patients who need only simple hotel-room type of accommodation. It should be possible to provide for them in a more homelike atmosphere with only the necessary supervision and guidance. The patient's schedule can be outlined for him on admission, he takes his meals in a cafeteria or restaurant, and restrictions are minimal. A good case can be made out for the " ambulant wing ".

The argument for the intensive-care unit is less convincing, but it may have a part to play in the smaller hospitals of the future. At this end of the scale we have the desperately ill patient whose life is going to be saved only if constant observation by a highly trained staff skilled in the handling of modern apparatus and in emergency treatment, can be provided. It cannot be said that this level of care can always be guaranteed in the general wards of smaller hospitals. In large hospitals with special departments the need for such a unit should not arise. The types of patient who might be nursed in such a unit are: major surgical cases released from the recovery room, multiple injuries or burns, major haemorrhage, and myocardial infarction. The team should be skilled in the observation of vital signs, the measurement and recording of fluid intake and output, the detection of complications, the administration of i. v. fluids and oxygen, the techniques of suction, catheterisation and bladder irrigation, wound dressing, anticoagulant therapy and the management of the various forms of respiratory paralysis. Some observers suggest that 60% need the normal intermediate care, 20% intensive care, and 20% self care.

(B. M. J. September, 10, 1960)

MODERN TREATMENT OF LEPROSY: During the last 20 years, leprosy has changed from being a hopeless horror to being a relationship between host and invading pathogen that can be successfully challenged, and there is the cheerful conviction that most patients with leprosy can eventually be cured, though it may still take a long time. The fundamental advance during this period has been the introduction of dapsone (diamino-diphenyl sulphone) by J. Lowe and others. This has now become the standard treatment, given in doses approximating to 300 mg. twice weekly. It has the advantages that it cures most patients in the long run, it never produces resistant bacilli, it seldom produces dangerous complications if given with reasonable care, and it is cheap. All the same, dapsone is not the perfect drug for leprosy, and there have been many attempts to produce drugs which act more rapidly or which are less apt to produce untoward effects or lepra reactions.

In the first place, there have been attempts to modify or mask the sulphone molecule by substituting one or both of the terminal amino groups, or by using diaminodiphenyl sulphoxide (which has one less oxygen atom than dapsone). Some of these compounds may have slight advantages over dapsone, but they are not so great as to supplant the parent compound. A more hopeful compound is Ciba 1906 (D.P.T., 4-butoxy-4-dimethyl-amino-diphenol-thiourea). This was first tried by T. F. Davey and G. Currie five years ago and considerable experience has now been gained with it. Briefly, it is given orally as 2 g. daily for an adult. It is slightly more active than dapsone, as judged by the disappearance of bacilli in lepromatous cases. In two to three years signs of drug resistance may appear, so it should not be continued after that period. Its chief advantage is that it is less toxic than dapsone; in particular, it is less harmful to the liver and it has less tendency to provoke lepra reactions or erythema nodosum. Accordingly it is the drug of choice for debilitated patients or for those on dapsone who have complications such as psychosis, neuritis, or the reactions already mentioned. It is rather expensive, especially when the much bigger dosage is taken into account; on the other hand, if it allows treatment as on out-patient instead of as an in-patient, it may be an economy in the long run. There is some evidence that its therapeutic effect depends on its conversion to a more active metabolite.

The next drug was "etisul" (diethyldithiol isophthalate), which is somewhat unconventional. Compounds of this type (ethylmercaptans) had long been known to be active against acid-fast bacilli, but their clinical application was hindered by their stench. However, acceptable preparations have been provided by the addition of suitable perfumes, and etisul can now be given twice weekly by inunction over a large area of the body. When given to lepromatous patients in this way, Davey found that there was often a remarkable reduction in the number of bacilli in biopsy specimens of skin, a change in their morphology, and a distinct clinical improvement in the patient. These effects are greater than can be produced by other drugs and they have been confirmed elsewhere. After three to six months, signs of drug resistance may appear, and the patients tend to become weary of the treatment. Accordingly this compound is best given in conjunction with dapsone or Ciba 1906, and after three months the etisul is discontinued.

Many other drugs have been tried, but none have been outstandingly successful. Cortisone and related steroids have been found valuable in controlling lepra reactions provoked by dapsone; and unless their administration is unduly prolonged, they do not seem to do harm weakening the body's defences against this infection.

Among other aspects of leprosy on which research is proceeding, the nature of the lepromin reaction has been critically reconsidered. It had always been assumed that a positive lepromin reaction was analogous to a positive tuberculin reaction and that it indicated hypersensitivity of the body induced by the infection specifically to certain chemical fractions of the leprosy bacillus. But now evidence is accumulating that the lepromin reaction may depend only on raised sensitivity to almost

any foreign substance introduced into the skin. Secondly, great efforts are being made to induce leprosy bacilli to multiply either in laboratory animals or in tissue cultures. Limited multiplication — namely, two to four generations — of the rat leprosy bacillus has been obtained in tissue cultures by J. H. Wallace and colleagues and R. J. W. Rees and P. C. Wong. Material from lepromatous patients has also been inoculated into animals and growth of acid-fast bacilli has been obtained in black mice, in hamsters, and in chimpanzees. The past history of leprosy is strewn with so many premature claims that all reports must be regarded with scepticism until it is proved beyond all doubt whether the bacillus which has been isolated is really Hansen's bacillus or only some previously unrecognized mycobacterium, of which unfortunately there are many. Nevertheless, leprosy has now changed from a subject of despair to one of active, and optimistic research.

(B. M. J. August 27, 1960)

METRIC SYSTEM IN BRITISH MEDICINE: When the next edition of the *British Pharmacopoeia* — an official medical guide to drugs, formulas, and doses — is published in 1963, the metric system will be used for the first time.

To ease the problem of doctors in practice who are in the apothecaries' system, it is proposed to introduce the metric system in stages. In the first instance it will be used for bottles of medicine.

It is impossible to make the change without Parliament's approval, and the General Medical Council of Britain is proposing to have the system adopted by a private Bill. However, Lord Cohen of Birkenhead, a member of the Council, has undertaken to see if an amendment can be introduced to the Weights and Measures Bill at present before Parliament.

(British Information Services)

ERRATA

1. Vol. XXVII No. 5 November 1960 "**Some Aspects of Respiratory Disease in Practice.**" — On Page 83 Line '5' in the first column instead of "and in the belief that psychological effects are dubious" read as "and in the belief that the psychological benefits of the bottle of medicine are undoubted even if the pharmacological effects are dubious."

2. In the X-Ray plates of the above article, the pretreatment picture of the case in fig. 10 was omitted in printing and it showed "opacities in the right mid and upper zones resembling P. T. in a medical student after an attack of respiratory catarrh."

3. Vol. XXVII No. 6 December, 1960 "Insulin Therapy in Diabetes Mellitus" — Page 111. The full designation of the author is given below: Dr. R. S. Rajagopalan, M. D., D. T. M., Medical Registrar, Stanley Hospital, Madras.

ASSOCIATION NOTES

BRANCH NOTES

Anamallai Branch:

1. A clinical meeting was held on the 20th August, 1960, at the residence of Dr. N. S. Menon, the President. Dr. E. Harris gave a talk on "Common Surgical Conditions of the Rectum and Anus".

2. A clinical meeting was held on 17th September, 1960, when Dr. T. V. Sivanandam, President of the State Branch, addressed the members on "Pitfalls in Medical Practice".

3. A clinical meeting was held on 22nd October, 1960, when Dr. Padmajan Pillai gave a talk on "Nephritis".

4. A Clinical meeting was held an 19th November, 1960, when Dr. R. Perumal gave a talk on "Some Conditions Causing Pain in the Throat".

Coimbatore Branch:

A combined meeting of the Coimbatore District Medical Association and the Clinical Association of the Government Head-Quarters Hospital, Coimbatore, was held on Saturday, the 17th December 1960, at 5-30 p. m. in the Head-Quarters Hospital, Coimbatore.

Dr. G. T. Gopalakrishna Naidu, Vice-President of the Association presided over the general session and Dr. V. Selvaraj, District Medical Officer, Coimbatore, presided over the clinical session.

A resolution expressing condolence on the death of Dr. K. Sriramamurthy of Udumalpet, a member of our Association, was passed — all the members observing silence for two minutes.

Several interesting cases were demonstrated by the members of the various departments of the Head-Quarters Hospital.

This was followed by a film show on "Piles and The Early Detection of Rectal Cancer" arranged by Messrs. William R. Warner & Co. Bombay.

Madras City Branch:

A clinical meeting was held at the Madras Medical College at 6-30 p. m. on 5th November, 1960, with Dr. Sriramalu, the president in the chair when Dr. E. Dunlop, Hunterian Professor of the Royal College of Surgeons of England and Cecil Joll Medallist of the Royal College delivered an interesting lecture on "The Problems of the Mouth and Throat Cancer". Being an eminent Cancer Surgeon in a leading Cancer

Hospital in Australia, he gave an authoritative exposition of cancer surgery in mouth and throat with projection of very interesting lantern slides showing his operation technique. He advocated radiotherapy only in instances when the cancer surgeons failed to give satisfactory results.

Madura Branch :

The monthly meeting was held on 17th December, 1960, under the Presidentship of Dr. E. S. Chellappa, F. R. C. S., Madurai. Dr. M. D. Ananthachari, M. D. formerly Principal, Madurai Medical College, Madurai, gave an interesting lecture on "The Problem of Amoebiasis".

Trichy Branch :

A monthly meeting was held on Saturday the 17th December, 1960, at the Medical Association premises under the Chairmanship of Dr. P. V. Sundaram. A condolence resolution on the demise of Dr. D'Costa, an oldest lady doctor of Trichy was passed all members standing in silence for 2 minutes.

Dr. M. V. Bhat, M. S., Professor of Operative Surgery, Madurai Medical College and Surgeon, Erskine Hospital, Madurai gave a talk on "Hamaturia — Its causes and treatment".

LETTERS TO THE EDITOR :

"EYE RELIEF CAMP"

I understand that there will be an eye relief camp shortly at *Tuticorin*, to be conducted by one Dr. Sharma of Bihar. Several camps have been conducted by the above doctor in various parts of the Madras State, and from all these camps lot of people who have lost their eyes completely as a result of his operations are being treated in the various local eye institutions. At the recent Madras State Ophthalmic Conference held at Vellore last month, a resolution was passed condemning such work.

I request the Government and the responsible citizens to take immediate steps to see that such havoc is not produced by these camps.

I hope that this letter will find an immediate place in your esteemed Journal.

Madurai,
9—1—1961.

(Sd.) DR. A. SATHAR, L. M. P., L. O.