THE MISCELLANY

OF

THE TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION

(REGISTERED)

Editor: DR. M. C. CONDILLAC, M.M., M.R.C.S.

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CONTENTS

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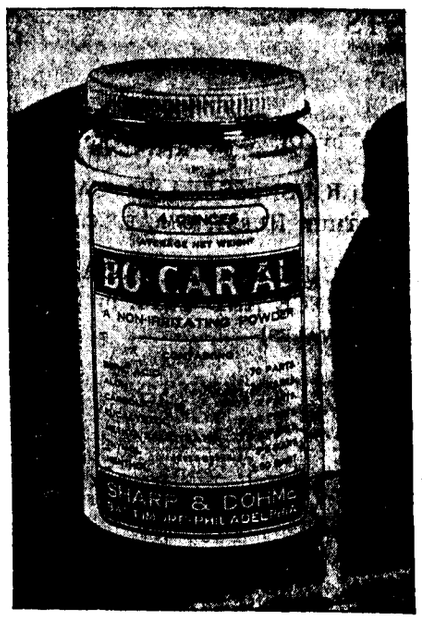

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Tests for Renal Efficiency

BY

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HAVING made a complete clinical examination of the patient, and having found protein in the urine together with an abnormal deposit, the medical practitioner may desire to extend his examination by applying tests of renal efficiency. Viewed in this light, i.e., as an extension of the clinical examination, renal efficiency tests are often of great value. Naturally in the majority of cases the chemical tests simply confirm the opinion already drawn from the clinical examination, but in so doing they give additional confidence. Occasionally the chemical tests are indispensable, or necessitate a revision of the conclusions drawn from the clinical examination.

Objects of Kidney Function Tests

(1) Differential diagnosis from non-renal diseases.

(2) In unilateral disease, diagnosis of the side affected.

(3) Estimate of degree of damage to each kidney separately, or estimate of damage to one kidney and "proof" that the other is functionally efficient.

(4) Estimate of total damage: estimate of risk of uraemia.

(5) Repeated estimations of kidney function (unilateral or combined) at intervals as an aid to prognosis; or,

(6) As an aid to treatment.

Conditions of Testing

Tests may involve the examination of the "combined urine" from the bladder, with or without catheterisation; or of the segregated urines obtained by ureteric catheterisation, or of the blood. Lastly examinations of blood and urine may be combined in order to ascertain the state of affairs on both sides of the kidneys.

The substance estimated may be an ordinary constituent of the blood and of the urine; or it may be an artificial product administered by mouth or parenterally for the purpose of the test, e.g. dye tests. In practice estimations on the urine have very largely been given up, owing to the difficulty of obtaining a complete collection of the twenty-four hours' urine. Finally, provocative tests may be employed. In such tests, relatively large doses of substances are administered in order to force the kidney to work hard. In this way some measure of the reserve power of the renal tissue is obtained; the kidney is working at a disadvantage, and defects in function may be rendered obvious which would escape notice using other tests. An example of a provocative method is the urea concentration test.

The Functions of the Kidneys

The main functions of the kidneys are:—

(1) EXCRETION of
   (*a*) Waste products of metabolism.
   (*b*) Poisons, chemical and bacterial (these may or may not have been rendered innocuous by the liver before reaching the kidneys).
   (*c*) Other foreign substances.

(2) Regulation of the OSMOTIC PRESSURE of the blood chiefly by control of salts excreted, especially chlorides.

(3) Regulation of the HYDROGEN-ION CONCENTRATION of the blood.

(4) CONSERVATION OF THE BASES—particularly sodium of the blood and body-fluids. The chief mechanism is the conversion of urea into ammonium carbonate in the kidneys; a double exchange then takes place, the ammonium salt is excreted in the urine and the sodium is conserved as bicarbonate in the blood.

(5) KIDNEY SYNTHESIS, for example, the conjugation of benzoic acid with glycine to yield hippuric acid (benzoyl glycine).

Tests Employed

(1) The urea concentration test (Maclean and De Wesselow).
(2) Blood Urea.
(3) The Blood Urea clearance test. (Van Slyke.)
(4) Dye tests.
   (*a*) Indigo-carmine.
   (*b*) Phenol Red or Phenolsulphonephathalein.
(5) Chlorine content of Blood and Urine.
(6) CO_2 combining power of Plasma. (Alkali reserve plasma bicarbonate)
(7) Blood cholesterol.
(8) Indicanaemia.
   (*a*) Diazo Test,
   (*b*) Oxidation to Indigo Blue.
(9) Blood Non-protein Nitrogen.
(10) Blood Creatinine.
(11) Urinary Creatinine.
(12) Blood Uric Acid.
(13) Diastase Tests.
(14) Sugar content of Blood and Urine.

The Urea Concentration Tests

Principle—The object of this test is to force the kidneys to CONCENTRATE urea by flooding the blood with urea, and by withholding fluids for eight to twelve hours beforehand.

Technique—(*a*) Performance of Test in Ward, etc.

Nothing to drink overnight, e.g. nothing after 10 P.M. Next morning at:—

5-58 a.m. Empty bladder completely.
Label specimen "O".

6 a.m. Drink the dose of urea.

7 a.m. Empty the bladder again.
Label specimen "1"

8 a.m. Pass urine again.
Specimen "2."

9 a.m. Pass urine once more.
Specimen "3."

(*b*) Laboratory Determinations.—Measure the volume of urine in cubic centimetres and estimate the percentage of urea in each of specimens 1, 2, and 3. The volumes are measured in order to detect excessive diuresis. The hypobromite method for the estimation of urea is sufficiently accurate for clinical purposes, in that only relatively gross variations in urea concentration have any significance.

Urea prescriptions for an adult:—

R	Urea	15 gm.
	Tincture of orange	1 C.C.
	Water.....to	100 C.C.

Interpretation of Results

(*a*) Concentration of urea. The information required is the MAXIMUM CONCENTRATION of urea. It does not matter whether this occurs in the first, second or third hour. Usually the maximum is found in the second or the third hour but occasionally in the first hour. It is, therefore, advisable to determine the percentage of urea in each of the three hours, but if time is pressing the second or third hour sample may be analysed first, and if satisfactory further analysis may be omitted.

Normally the maximum concentration exceeds 2·5 per cent frequently being greater than 3%. Figures between 2·5 and 2 per cent may be regarded as within the boundary zone. Their significance should be carefully assessed in the light of

all the evidence available, and if any doubt remains as to the efficiency of the kidneys, further tests should be performed, e.g. an estimation of the blood urea. Figures below 2 per cent generally indicate renal inadequacy always assuming that the test has been properly executed.

Value of the Test

On account of its simplicity the test has been used extensively in all branches of urinary disease. It is very useful in clinical work, and undoubtedly will reveal the presence of a serious degree of renal inefficiency in the vast majority of cases.

The urea concentration test is a provocative test. That is to say, by flooding the blood with urea an attempt is made to provoke the kidneys to excrete urea at higher concentrations than on an ordinary diet. It is possible that in the case of a damaged kidney such provocation makes a call upon some of its "reserve." Possibly for this reason the urea concentration test is more sensitive than many other tests, such as the blood urea test.

(To be continued)

The Physical Basis of "Biliousness" and "Wind Round the Heart"

BY

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[*From the British Medical Journal*]

"BILIOUSNESS" OR A "TOUCH OF LIVER"

AMONG the commonest conditions of which patients complain is a "touch of liver" or a bilious attack. As neither is mentioned in most text-books of medicine, it is natural that a recently qualified doctor should conclude that there is no such thing and should be driven to explain to the sufferer that what he puts down to his liver is really a disorder of his stomach, his bowels, or his nerves. But the patient remains unconvinced, and when next he wakes up with a dull headache, a dirty tongue, no appetite for breakfast, and the conviction that life is not worth living, he will tell his wife that his bad temper is due to his liver, and the doctor be damned. It is a curious fact that there is no equivalent in French or German for the word "liverish" or a "touch of liver," though a bad-tempered Frenchman is said to be "unbilieux." There is also no record of the word "liverish" being used in England before the first quarter of the nineteenth century, and I am inclined to think that it was introduced by soldiers and civilians who came home to nurse their damaged livers in Cheltenham and Bath, as it was in India that the frequency of hepatic abscess first led Army surgeons to recognize the premonitory symptoms of a disordered liver.

I believe that the patient who says he is liverish is often correct, and that functional disorders of the liver are actually far more common than organic diseases. The liver has more ways in which it can be attacked than any other organ. Poisons absorbed from the stomach, intestines, gall-bladder, and spleen reach it in the blood of the portal vein; poisons

and organisms in the systemic circulation are conveyed to it by the hepatic artery; infections may ascend the bile ducts from the duodenum, especially in the presence of achlorhydria, and infection may be carried by lymphatics from the wall of the gall-bladder.

Alcohol is the commonest cause of liverishness, but there are many others. The intestinal toxaemia which results from the habitual abuse of purgatives, though never from untreated constipation, and all forms of food poisoning and intestinal infections, such as those with typhoid and various parenteric bacilli and with *Amoeba histolytica*, as well as such general infections as septicaemia and malaria, may injure the liver. With the exception of alcohol and amoebiasis they rarely produce enough damage to give rise to symptoms unless two or more are associated together. But an acute "liver attack" may occur in all these conditions as a result of indulgence in an amount of alcohol which formerly would have had no ill effect, and indiscretions in diet, fatigue, and excitement are often contributory causes. A patient of mine, a man of 58, found it difficult to decide whether a bad liver attack was too high a price to pay for his sole pleasures in life, betting at race meetings and gambling at Monte Carlo, both of which invariably brought one on even if he drank no more alcohol than usual.

The liver is well provided with means for dealing with toxins and bacteria, but it is not always capable of destroying them when they arrive in excess. The hepatic cells are then damaged and their functional capacity is impaired. The damage in the early stages is not irreparable, and although microscopical examination would doubtless reveal the presence of structural changes, they are at first of a type from which complete recovery can take place. The ordinary clinical methods of physical examination do not give any safe guide to the diagnosis of these early stages of hepatic disorder, but fortunately in the laevulose test we have a means of recognizing them. Our experience of over a thousand cases at New Lodge Clinic in which the possibility of hepatic insufficiency required consideration has led us to regard it as a very reliable test of liver function. We now consider a combined one-hour and two-hour

rise in the blood sugar of more than 25 mg. per 100 c.cm. after taking 50 grammes of laevulose as suspicious, and of more than 30 mg. as certain evidence of hepatic insufficiency.

Alcoholic Hepatosis

It is obvious that cirrhosis of the liver is only the last phase of a very long-standing disease, the early stages of which have passed unrecognized. As the condition is one of toxic degeneration rather than inflammation it is more suitably described as hepatosis than hepatitis. Whereas the death rate per million living from cirrhosis of the liver has fallen from 248 in 1902 and 185 in 1912 to 81 in 1922 and 50 in 1932, alcoholic hepatosis is quite common, especially among the well-to-do. The familiar symptoms of "the morning after the night before" are the symptoms of acute alcoholic hepatosis, and the laevulose test shows that definite hepatic insufficiency is present and disappears completely within a few days. Table I shows the results of the laevulose test in five healthy young men on the morning before a "night out" compared with the morning after. In each case there was a greater rise in blood sugar with the second test than with the first, which showed an actual fall in four instances. The rise was greatest in A and B, the only ones who felt any the worse for the experiment.

TABLE I

Blood Sugar in mg. per 100 c.cm. Fasting and One and Two Hours after taking 50 grammes of Laevulose in Five Healthy Young Men, (1) the Morning Before, and (2) the Morning After a "Night Out"

	A	B	C	D	E	Total
(1) Fasting ...	86	80	100	100	94	
1 hour ...	88	71	98	111	111	
2 hours ...	74	64	95	87	105	
Total rise ...	—10	—25	—7	—2	28	—16
(2) Fasting ...	133	82	69	126	114	
1 hour ...	143	87	63	145	139	
2 hours ...	143	87	71	125	121	
Total rise ...	20	10	—4	18	32	+76

Habitual over-indulgence leads to a chronic alcoholic hepatosis, which is still capable of complete resolution even if it has been present for many months. It is only when it has continued without intermission for years that irreparable damage is done, but the nodular hyperplasia caused by new-formed liver tissue may even then compensate for this more or less completely, although areas of complete necrosis are gradually replaced by fibrous tissue, the first stage of cirrhosis having at last developed.

Over-indulgence in alcohol is a very elastic term, what is excess for any given individual depending upon the vulnerability of his liver cells, which varies greatly in different people. It has nothing to do with his general reaction to the effects of alcohol, as alcoholic hepatosis is not often associated with alcoholic psychoses or peripheral neuritis. The premonitory symptoms of cirrhosis are mainly hepatic in origin, through chronic oesophagitis and, less frequently, chronic gastritis may contribute to them; but the latter is not necessarily present, a normal curve of acidity without any excess of mucus being often obtained with a fractional test meal.

Young men and girls who indulge in cocktails three or four times a week may develop alcoholic hepatosis. Their anxious parents are apt to ascribe their sallow complexions, loss of appetite, mental torpor, and irritability to incipient consumption or other serious disease, but the well-marked hepatic insufficiency disclosed by the laevulose test and the tender and often slightly enlarged liver reveal the true nature of their malady. Complete recovery with disappearance of liver tenderness and return to a normal response to the laevulose test quickly follows a period of rest with a light diet and abstention from alcohol.

Table II gives some examples of the results of the laevulose test in alcoholic hepatosis before and after treatment. Patient No. 6 had drunk a bottle of champagne a day for twenty years, and was so ill that he was thought to be dying from some obscure abdominal complaint. His liver was large, hard, and very tender. After three months on a strict diet his liver was no longer palpable, and he returned home feeling perfectly fit

and able to play tennis and golf for the first time for five years. No. 7 had been drinking from one to three bottles of whisky a day for over a year, and also recovered completely.

The tender liver often aches, and a man "with a liver" has generally a fairly accurate knowledge of its surface anatomy. He is never jaundiced, and latent jaundice, as shown by a positive indirect Van den Bergh reaction is uncommon; when present it disappears as rapidly as the abnormal response to the laevulose test.

In early cases, after a short period of strict treatment, it is only necessary to forbid alcohol in any form between meals,

TABLE II

Lævulose Tolerance Test in Alcoholic Hepatosis Before and After Treatment

Case	Sex and Age	Interval between Observations	Rise of Blood Sugar above Fasting Level in mg. per 100 c. cm. after taking 50 grammes Lævulose		
			1 hr. after	2 hrs. after	Combined 1 and 2 hrs.
1	F, 22	22 days	51	15	66
			23	10	33
2	M, 32	32 „	45	89	134
			6	30	36
3	F, 56	9 „	44	49	93
			16	18	34
4	M, 49	9 „	44	7	51
			15	-15	0
5	M, 56	20 „	49	0	49
			13	-11	2
6	M, 52	3 months	73	71	144
			0	-13	-13
7	M, 45	6 weeks	58	31	89
			10	-6	4
8	M, 40	24 days	51	15	66
			12	-22	-10

when it is rapidly absorbed and reaches the liver in a comparatively concentrated form. But in more advanced and chronic cases it is useless to advise anything short of complete and permanent abstinence. This advice is easier to follow than might be expected, as the patients are never dipsomaniacs and have no uncontrollable urge to drink. They drink for conviviality and because they like it or find it difficult to complete a business deal without celebrating it with a drink. A badly damaged liver is abnormally vulnerable to such toxins as alcohol, and even a small dose is poisonous. But more important is the psychological fact that with his first drink a man's judgment is impaired, and though he may have intended to take no more he will have a second and a third, and so on, *ad infinitum*. He is only safe when teetotal.

Amoebic Hepatitis

In 1923 Lieutenant-Colonel Gordon Covell published in the *Guy's Hospital Reports* a paper in which he showed that amœbic dysentery was always accompanied by hepatic insufficiency as measured by the lævulose test, even in the complete absence of symptoms pointing to disorder of the liver. Since that time we have always performed lævulose test in the many patients who, on returning home after long residence in the East, complain of general unfitness in the absence of any obvious organic disease. They look pale and sallow, though they are neither anæmic nor jaundiced and Van den Bergh's reaction is generally negative. They have frequent liver attacks—headache with a dirty tongue, anorexia, discomfort in the region of the liver, and constipation or alternating constipation and diarrhœa. The liver is often slightly enlarged and tender, and the lævulose test shows that hepatic insufficiency is present. Though alcohol and various infections may have contributed to this result the condition is mainly due to a mild chronic form of amœbic hepatitis. The patient may have had definite attacks of dysentery, but more frequently he has had nothing else than an occasional attack of diarrhœa, and sometimes not even that, yet the cæcum and ascending colon are often tender, and the appendix has generally been removed for "chronic appendicitis" without benefit. It is remarkable

that this condition, which never leads to abscess formation, can persist for several years after returning to England and yet be amenable to treatment. It often remains unrecognized owing to too much reliance being placed on the presence of cysts of *Entamœba histolytica* in the stools as essential evidence of the existence of amœbiasis.

Complete recovery follows a dozen injections of 1 grain of emetine. The patient not only loses his symptoms but the liver ceases to be palpable and the lævulose test shows that the hepatic insufficiency has disappeared (Table III). To avoid the risk of recurrence a second series of six injections of emetine should be given at the end of three months and then every six months for a couple of years.

TABLE III

Lævulose Tolerance Test in Amœbic Hepatitis Before and After Treatment with Emetine

Case	Sex and Age		Rise of Blood Sugar above Fasting Level in mg. per 100 c.cm.		
			1 hr. after	2 hrs. after	Combined 1 and 2 hrs.
1	F, 53	Before treatment...	71	31	102
		After treatment ...	16	18	34
2	F, 40	Before treatment...	55	30	85
		After treatment ...	6	0	6
3	F, 33	Before treatment...	34	14	48
		After treatment ...	18	-2	16

Bilious Attacks and Migraine

What is called a bilious attack in a child is in most cases the equivalent of migraine in an adult. One or other parent is often a sufferer from migraine, and the child is likely to develop it when he grows up. The term "bilious attack" is apparently derived from a misinterpretation of the fact that it often terminates with bilious vomiting. There does, however, appear to be some obscure connexion between the liver and migraine in a proportion of cases in both children and adults (Hunt, 1933). We have never found any sign of hepatic insufficiency as shown by the lævulose test, and Van den Bergh's test is negative both during and between the attacks. But when in the course of our investigations we aspirated a specimen of bile through Einhorn's tube after making the gall-bladder and bile ducts evacuate their contents by injecting magnesium sulphate into the duodenum, a number of adult patients asked to have the process repeated, as they felt clearer in their heads and altogether less "bilious" than they had done for months. In these cases the gall-bladder was often found to be tender, especially shortly before and during an attack. It is apparently the mechanical effect of unloading the liver of accumulated bile which has this unexpected result, as in every case the bile was quite normal and there was no evidence of co-existing cholecystitis or gall-stones. Such patients derive benefit from stimulation of biliary drainage by means of Epsom salts and olive oil. The former should be given in concentrated solution fasting every morning, the dose being the largest which can be taken without causing looseness, and a tablespoonful of the latter should be taken three times a day half an hour before meals.

A large proportion of sufferers from migraine know from experience that fatty food induces an attack, and among those who have not discovered this for themselves it is common to find that the exclusion of eggs, cream, fat meat, fried food, and chocolates helps to prevent migraine. It is interesting to observe that these are just the cholesterol-rich foods which frequently upset patients with diseases of the gall-bladder, and although there does not appear to be any special liability to cholelithiasis among the migrainous, it seems not improbable that a disordered liver may be one factor in the complicated pathogenesis of migraine.

"WIND ROUND THE HEART"

Aerophagy

There is no physical basis for the wind which troubles most people who complain about it; it is on their brains; and neither in their stomachs nor round their hearts. Nearly thirty years ago I described experiments which showed that the only adequate stimulus to the sensory nerves of the alimentary tract

is tension on the fibres of its muscular coat (Hurst, 1911). A slight increase gives rise to the sensation of fullness and a greater rise to pain. The tension depends upon the postural tone of the muscle fibres and the volume of the contents of the segment concerned. A sensation of fullness and pain may result from increased tone of the muscles with no change in the contents, or upon increase in the volume of the contents with constant tone. The sensation of fullness or pain can be relieved when some or all of the contents consist of gas, which can be evacuated by belching from the stomach or as flatus from the colon. Everybody has had experience of gaining relief in these ways, and consequently the average person is inclined to ascribe a sensation of fullness in the stomach or colon to wind, although in most cases it is due to other causes. Success confirms his suspicion, but instead of accounting failure to a wrong diagnosis he assumes that wind is present but cannot be expelled. In his futile attempts to eructate he swallows air until enough is present in his stomach to be easily and loudly evacuated. The relief which follows is partly psychological and partly due to the fall in intragastric tension. As the discomfort which initiated the aerophagy is often due to achalasia or spasm of the pyloric sphincter, the primary discomfort may be overcome, as the further rise in intragastric pressure resulting from the aerophagy may be sufficient to force the pylorus as well as the cardia. Sodium bicarbonate gives relief in precisely the same way as aerophagy: carbon dioxide is evolved by the action of the acid gastric juice, and the increased tension in the stomach forces gas through the cardia and fluid through the pylorus. In both cases the patient brings up gas, and it is difficult to convince him that this is not the product of fermentation in his stomach. But owing to the inhibiting action of free acid on bacterial activity and the continuous passage of food into the duodenum fermentation cannot occur; we have found that even in complete achlorhydria very little gas is produced by fermentation unless pyloric obstruction is also present.

Neurotic patients may get so much satisfaction from belching wind with a resounding noise that they continue to swallow air, and the whole process is repeated again and again. Eventually attacks of aerophagy occur without any preceding epigastric discomfort, the condition then having all the characteristics of a tic. The action, though originally serving a useful purpose, no longer does so; its repetition depends upon the subconscious gratification which it gives to the patient, who is able to restrain it by an effort of will, but after a short time gives way to an overwhelming impulse to continue, though often ashamed of his weakness.

Aerophagy can be cured by explaining the true cause of the "flatulence" in language suited to the patient's intelligence. He is instructed to make no voluntary effort to eructate, however much he may desire to do so. At the same time an attempt must be made to discover and relieve the primary cause of the discomfort which led to the aerophagy, whether this is some form of functional dyspepsia or of organic disease, such as gastritis, ulcer, or cholecystitis.

Intestinal Carbohydrate Dyspepsia

The most common cause of intestinal flatulence is carbohydrate dyspepsia (Hurst and Knott, 1931). The envelopes of root vegetables are softened but unbroken by cooking. Under normal conditions amylopsin of the pancreatic juice penetrates the envelope in the small intestine and converts the starch into soluble sugars, which diffuse into the surrounding media and are absorbed. When owing to abnormally rapid passage through the small intestine some of the starch reaches the caecum the same thing happens there. But whereas in the relatively sterile small intestine very little undergoes fermentation, in the caecum and ascending colon bacteria are so active that fermentation occurs before the sugar has time to be absorbed. Gases and organic acids are produced, the former causing colonic distension and discomfort, and the latter, if in great excess, irritating the mucous membrane sufficiently to cause diarrhoea. The stools are acid, contain many starch granules, and produce much gas on incubation. During the day the gas collects in the splenic flexure and causes discomfort, which is often mistaken by the patient for gastric flatulence and so gives rise to aerophagy. During the night, when the splenic flexure is no higher than the pelvic colon and rectum, the gas moves onwards, and the discomfort in the lower abdomen is often enough to interfere

with sleep until relief is obtained by the passage of odourless flatus. Intestinal carbohydrate dyspepsia can be controlled by giving a diet containing no root vegetables or rice; in severe cases the use of other starchy foods must also be limited. As there is no deficiency of amylopsin the administration of vegetable diastase is useless. The number of enterococci in the stools is increased owing to the excess of carbohydrate in the colon acting as an excellent culture medium; the intestinal flora return to normal when the condition is relieved by dieting. It is obvious that nothing could be more futile than treating cases of this kind by vaccination with the patient's enterococci or by intestinal lavage, though many patients receive such treatment owing to failure to look for starch in the stools and to see whether fermentation occurs on incubation.

Eventration of the Diaphragm

In certain conditions gas accumulates in the stomach or the colon immediately beneath the diaphragm under excessive tension, with the result that the left dome is displaced upwards into the thorax, so that it is higher than the right dome and may even give rise to dextrocardia. Eventration of the diaphragm produced in this way is very much commoner than that resulting from congenital atrophy of its musculature, for which the name has generally been reserved.

(*a*) AÉROGASTRIE BLOQUÉE

The gas bubble seen with the *x*-rays under the diaphragm is derived from air swallowed with each mouthful of food and drink. It is permanently absent only in achalasia of the cardia, in which the contents of the dilated oesophagus act as a lock, preventing the passage of swallowed air. Excess is normally removed by unconscious silent eructation in the day and through the pylorus at night, when the horizontal posture makes this possible. In rare cases eructation is prevented by spasm of the lower end of the oesophagus just above the sphincter owing to the presence of a simple œsophageal ulcer (Hurst, 1934a). Enormous quantities of gas collect in the stomach and may cause intense pain, increased each time any food or saliva is swallowed owing to its admixture with air. Failure to recognize

the true nature of the condition may have a deplorable effect upon the patient's nervous system. This was the case in a man of 76, who had suffered from "gas pains" since 1926. In 1929 and again in December, 1931, he had a small hæmotemesis; on both occasions he was *x*-rayed, but nothing beyond excess of gas in his stomach was discovered. Early in 1932 he noticed that when he swallowed there appeared to be an obstruction at the entrance into his stomach, as the passage seemed to "shut up" in a painful spasm whenever food reached it, this being followed by regurgitation of the food with excess of mucus. An *x*-ray examination again showed no abnormality, except an enormous gas bubble in the stomach, which caused the left half of the diaphragm to be elevated above the right. He was regarded as hysterical and was treated by various forms of psychotherapy on the Continent, in America, and in England, but without success. His condition became progressively worse, and when I first saw him in January, 1932, he was intensely depressed and was terrified at the mere idea of eating. When a meal was brought into the room his face became anxious and his hands shook. Several minutes elapsed before he could persuade himself to lift the fork in his trembling hands to his lips, and he masticated much more than was necessary in order to put off the moment of swallowing as long as possible. He had lost much weight and strength. As it seemed impossible that such severe dysphagia could be purely nervous in origin, especially in view of the history of hæmatemesis, a further radiological examination was made, and Dr. P. J. Briggs succeeded in demonstrating the presence of a deep chronic ulcer just above the cardiac sphincter. The enormous gas bubble in the stomach was clearly a result of the œsophageal spasm caused by the ulcer interfering with the escape of swallowed air upwards, just as it interfered with the passage of food downwards. He was treated with a diet of citrated milk and with atropine, and rapidly lost this pain and dysphagia; he became comparatively happy, but the physical signs of fear which occurred when food was brought into his room persisted in a lesser degree after the disappearance of the associated emotion, presumably as a result of a conditioned reflex. The ulcer became much smaller, but after many weeks it had not healed owing

to the development of cicatricial narrowing, and eventually a gastrostomy was performed. This resulted in complete healing with disappearance of the occult blood from the stools. The cicatrization of the ulcer led to almost complete obstruction; this would probably have been overcome by gradual dilatation had he not developed a rapidly fatal attack of acute mania.

A similar condition of aérogastrie bloquée may result from dislocation of the cardia, which is generally caused by excess of gas in the neighbouring splenic flexure. The excess of gas escapes from the stomach when it can reach the pylorus on assuming the horizontal position. In the morning no excess is present, but with each meal the quantity increases until the afternoon, when the accumulation produces a rise of pressure sufficient to cause pain, the diaphragm at the same time being displaced into the thorax. Immediate relief follows the passage of a stomach tube. One patient with this condition had learnt to lie down at 3 p.m. every day when the pain was at its height. In half an hour he was comfortable, as the gas had passed into his small intestine, and after another hour he evacuated some of it per anum, but enough remained in his splenic flexure to maintain the dislocation of his cardia. Permanent relief occurred only after he had evacuated the gas from his stomach by a tube at 3 p.m. every day for a week, so that no excess passed through his pylorus to collect in the colon.

(*b*) MEGACOLON AND AÉROCOLIE BLOQUÉE

Megacolon is the result of interference with the normal neuromuscular mechanism controlling the last stage of the act of defaecation, in which the passage of faeces from the pelvic colon into the rectum causes the latter to contract and the sphincter ani to relax. When owing to degeneration of Auerbach's plexus or other causes the sphincter does not relax (achalasia), faeces and gas are retained, and the rectum and especially the more thin-walled pelvic colon become progressively dilated (Hurst, 1934b). The pelvic colon finally forms a huge loop, which reaches the left and occasionally the right dome of the diaphragm. Megacolon is the commonest cause of eventration of the diaphragm. I have seen thirty-five cases in private

practice—twenty-four in males and eleven in females, including four boys and one girl (so-called Hirschsprung's disease)—compared with two cases caused by congenital atrophy of the diaphragm and four cases of aérogastrie bloquée. It is remarkable how trivial the symptoms of megacolon may be, the condition often being recognized by the discovery of an enormous gas-containing loop of pelvic colon under the diaphragm in the course of a radiological examination of a patient suffering from nothing more serious than chronic constipation. In a minority of cases recurrent attacks of severe pain from partial volvulus of the dilated pelvic colon occur. Gradual dilatation of the anal sphincter and complete evacuation of the colon generally result in complete and permanent relief, although the pelvic colon remains abnormally distensible. In exceptional cases a partial twist of the uppermost part of the pelvic colon prevents the escape of the accumulated gas and painful attacks of partial volvulus may continue. It is sometimes possible to evacuate the air-lock by means of a tube. In other cases it may be necessary to excise the volvulus during a quiescent period. I have never found it necessary to advise sympathectomy.

The Seriousness of Tuberculosis in India as Shown by a Study of Incidence and Type

BY

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[*From the Indian Medical Gazette*]

OUR knowledge at present as regards the incidence of tuberculosis in India is very meagre, as there have been few scientific investigations and these very limited in extent. Apart from these few investigations, our main knowledge rests on general observations deduced from dispensary and hospital records and mortality statistics. As regards health in the large cities, because of the better facilities for collecting statistics there, it is known for certain that tuberculosis is exceedingly prevalent, but what the conditions are in the villages is largely a matter of conjecture. Many detailed surveys will have to be done over large and varied areas before we can get any real idea as to what is the actual state of affairs.

Tuberculin surveys

One of the ways of getting knowledge as to the extent and distribution of tuberculous infection is by tuberculin surveys of many thousands of people in different parts of India and in different communities, under differing conditions, urban and rural.

Ukil (1930) gives figures for a tuberculin survey of 6,500 individuals very predominantly urban, and a jail population, and a positive reaction was found varying from 11 per cent in those of five years and under to 75 per cent in those of 60 years and over. He also shows that the figures for rural areas are much less than for urban and that the positives increase as the conditions approach the urban.

Tuberculin survey of 6,665 individuals in South India

A tuberculin survey was carried out by the U. M. T. Sanatorium in parts of Chittoor District and its environs, 3,015 in 1937 (Benjamin, 1937) and 3,650 in the present year. In these two surveys were included three *taluk* headquarters towns with a population varying from 4,000 to 10,000, two large villages with a population each about 1,000, and a number of small villages with populations varying from 50 to 300. In some of the villages, the whole population was tested. In addition, the survey included four schools in Madanapalle, one in Vayalpad, two in Chittoor, two in Kolar Town in the Mysore State, and an industrial school in Katpadi in North Arcot District.

The test was done without any selection in the towns and villages, for all ages and both sexes. All communities, Hindus, Christians and Muslims, were included. The majority tested were of the poor and middle classes, as these formed the bulk of the population of the areas tested. But a number of well-to-do families was also tested.

In this tuberculin survey, the total number tested was 6,665, composed of Hindus 4,201, Indian Christians 1,668 and Muslims 796.

From these, 163 persons are omitted—Hindus 43, Christians 101 and Muslims 19—as they were coming into personal contact with tuberculous patients in the wards of the sanatorium during their daily work and, as such, were specially exposed to infection and could not be considered as a sample of the normal population.

There is no question of tuberculization of the villages proceeding from the sanatorium, as most of the villages and all the small towns tested were miles away from the sanatorium. It is interesting in this connection to note that 227 persons living in the sanatorium compound, but not attending on patients, showed a percentage of tuberculin positives similar to that seen in the village group.

Technique

The method used for the tuberculin test was that of von Pirquet, according to the modern technique, with a scarification of the skin about half an inch long through tuberculin already applied to the skin, using, of course, the usual aseptic precautions. Readings were generally taken after 48 hours, a few only being after 72 hours, and the intensity of the reaction was noted under different heads. A positive was recorded when there was palpable œdema and redness of a width of more than 2 mm. When the width was from 3-5 mm., it was noted as +; from 6-10 mm. as + +; over 10 mm. as + + +. The tuberculin employed was Burroughs, Wellcome & Co., Old Tuberculin, Human (T).

We realize that the Mantoux intra-dermal test is the more sensitive, and has the advantage over the von Priquet test, that in a particular individual successive tests can be done with graded doses and the results compared. This should be the test used when the group tested is under control, and it is amenable to such a test.

In the communities such as those with which we had to deal, it was impossible to use the Mantoux test involving an injection, except in a few educated individuals.

In this connection it is interesting to note that such an eminent tuberculosis worker as Bardswell (1938), in a recent tuberculosis survey in the island of Cyprus, had to abandon even the von Pirquet test, and do Morro's inunction test because of the difficulty of getting the co-operation of the population. Aeimbeck (1938) is of the opinion that the accuracy of the von Pirquet reaction, according to modern technique, is very good, and that the margin of error, when compared to the Mantoux test, is only 1·8 per cent in persons below the age of 15, and only 3 per cent between the ages of 16 and 30. The von Pirquet test has been variously estimated to be equivalent to 1 in 10,000 dilution used for Mantoux (Dow and Lloyd, 1931), and 1 in 2,000 (Matthews, 1931), and in the African survey reviewed by Cummins (1935) as roughly comparable to the intradermal test with 1 in 5,000 dilution of tuberculin.

TABLE I

Results of a tuberculin survey

Community		TOWNS				VILLAGES				TOTAL			
		Adults		Children		Adults		Children		Adults		Children	
		Number	Positives	Number	Positives	Number	Positives	Number	Positives	Number	Positives	Number	Positives
Hindus ...		1,401	562	1,664	183	594	168	499	13	1,995	730	2,163	214
	Per cent	40·1	40·1	10·9	11·6	28·3	28·3	6·2	6·2	36·6	37·8	9·9	9·9
Indian Christians...		574	192	647	72	127	35	219	20	701	227	866	92
	Per cent	36·9	36·9	11·1	11·1	27·6	27·6	9·1	9·1	32·4	32·4	10·6	10·6
Muslims ...		332	172	317	51	61	38	67	13	393	210	384	64
	Per cent	51·8	51·8	16·1	16·1	62·3	62·3	19·4	19·4	53·4	53·4	16·7	16·7
TOTAL ...		2,307	926	2,628	306	782	241	785	64	3,089	1,167	3,413	370
	Per cent	40·1	40·1	11·6	11·6	30·8	30·8	8·2	8·2	37·8	37·8	10·8	10·8

Analysis of findings

As a result of this tuberculin survey, we find that the average percentage of positives for the whole group is 37·8 for adults and 10·8 for children under 15 years of age. In small towns, children under 15 years of age showed 11·6 per cent positive and in the villages 8·2 per cent. Adults showed 40·1 per cent in the towns and 30·8 per cent in the villages. This is shown in Table I.

When we analyse the total number investigated according to different communities, we find that there is very little difference between the Hindu and Indian Christian groups, but a marked difference is found in the Muslim group, the percentage of positives being much higher in this group than in the two other groups, both in towns and villages, and also among adults and children. Compared with 36·6 per cent and 32·4 per cent positives for Hindus and Christians respectively, Muslims show 53·4 per cent positives for adults, and 16·7 per cent for children, as compared with 9·9 and 10·6 per cent for Hindu and Christian children. While among Hindus and Christians the percentage of tuberculin positives is higher in the towns than in villages, among the Muslims the opposite is found. The *purdah* system and special social habits among the Muslims may increase the chances for tuberculous infection and consequent higher percentage of positives in this community, but why the villages show a higher percentage than towns cannot be explained. As this group comprises only 777 individuals, it is too small a number on which to form any opinion; but it would be interesting to see in further investigations whether this higher percentage in the villages persists, and, if so, what are the special factors contributing to this.

It is of some importance to note the intensity of reaction. In Europe the intensity of reaction is not of such great importance among adults, but among small children an intense reaction is usually associated with active disease. Among Africans, the significance of a tuberculin reaction has been found to be similar to that among children in the European races. Hypersensitiveness to tuberculin is considered a serious matter in Africans and other primitive races, as this hypersensitiveness is not associated

with an immunity, but just the opposite, and, therefore, is a danger signal. What the significance of sensitivity to tuberculin means in India, we do not as yet know.

In Table II are given the reactions found in the investigation.

It will be seen from this table that 56·4 per cent of the positives were reactors with a reaction between 3 and 5 mm. wide; 31·5 per cent with a reaction of 6 and 10 mm. wide; and 12·1 per cent with a reaction of above 10 mm. wide. If we group the two stronger reactions together, we find that as regards community, Muslims have as many as 61·3 per cent of the strong reactions, whereas Hindus have 40·5 per cent, and Christians 37·6 per cent.

The higher percentage of strongly positive reactions among the Muslims coincides with a higher percentage of infection rate among the same community as shown by tuberculin tests.

It was possible to persuade 69 of those showing strong reactions, and a few with suspicious symptoms, to come to the sanatorium for *x*-ray and other examinations. Of these, seven were found to be suffering from active tuberculosis, five of them with cavities and positive sputum. Six of these seven were engaged in their ordinary work.

TABLE II

Strength of von Pirquet positive reaction

Community	Number	+	Per cent	+ +	Per cent	+ + +	Per cent	+ + and + + +	Per cent + + and + + +
Hindus ...	944	562	59·5	280	29·7	102	10·8	382	40·5
Christians ...	319	199	62·4	81	25·4	39	12·2	120	37·6
Muslims ...	274	106	38·7	123	44·9	45	16·4	168	61·3
TOTAL ..	1,537	867	56·4	484	31·5	186	12·1	670	43·6

When we analyse the figures with regard to occupation (Table III), we find the highest percentage naturally among

those who attend on patients then come the police, merchants and clerks, and those with indoor occupations. The group with the lowest percentage among adults is those engaged in agriculture. Those whose occupation could not be definitely ascertained and also those where the numbers were less than ten in any single group are all grouped together in the miscellaneous group.

It is evident from the figures given by the above survey that the villages in the area surveyed are no longer free from infection, and are not 'virgin soil' in the formerly accepted sense of the word, and yet this area is comparatively isolated with some of the villages about 25 miles away from rail and main roads, is sparely populated, and has one of the best climates in South India.

TABLE III

Occupations and tuberculin positives

Occupation	Number	Positives	Per cent
Children under five years ...	465	17	3·6
School children	2,769	409	14·8
Ward boys	29	25	86·2
Ward ayahs	11	9	81·8
Police (constables and inspectors) ...	41	26	63·4
Clerks	52	31	59·6
Doctors (medical students) ...	23	13	56·5
Merchants	243	136	55·9
Cooks	53	25	47·2
Sweepers	30	14	46·7
House servants	85	39	45·9
Carpenters, artisans and goldsmiths ...	141	64	45·4
Tailors	108	49	45·4
Nurses and compounders ...	61	28	42·4
Hotel-keepers	15	6	40·0
Teachers	151	60	39·7
Peons	53	20	37·7
Housewives	542	184	33·9
Dhobies	44	13	29·6
Coolies	450	130	28·9
Agriculturists	441	116	26·3
Miscellaneous	853	246	28·8
TOTAL ...	6,665	1,660	

THE TYPE OF PULMONARY TUBERCULOSIS

Variation in type in different races

General observations of tuberculosis among European races and a study of their *x*-ray films, and post-mortem examinations, reveal that a comparatively large proportion of these show a tendency to chronic disease with localization, and marked fibrosis round tuberculous foci and cavities, but this state of affairs does not hold good for other races. Investigations done in America among the negroes and in Africa among the native population show an entirely different type of disease. A recent investigation done in Tanganyika (Wilcocks, 1938) shows a preponderance of cases of acute 'galloping consumption' and bilateral disease. Fibrosis is not common, and, when it occurs, does so chiefly in middle-aged people. The lung bases are frequently attacked. Cavitation is frequently seen, but it is rare to see a zone of fibrous tissue round these cavities. Massive consolidation is occasionally found, and miliary spread often occurs.

Type of disease found in patients in India

A detailed study has been made of the type of tuberculosis found in 2,158 Indian, Anglo-Indian and European patients admitted to this sanatorium since 1930, all of whom were diagnosed as suffering from pulmonary tuberculosis. With a view to the determination of type, the following points were studied: the amount of fibrosis, evidence of previously healed lesions, tendency to dissemination, the question whether one or both lungs were involved, the amount of cavitation, all as shown in *x*-ray films; the evidence of resistance to the disease as shown by the length of time between onset of symptoms and admission to the sanatorium, the activity of the disease as shown by blood examinations (Schilling count and sedimentation test).

The results are shown in table IV.

TABLE IV

Analysis in percentage of findings in 2,158 Indian, Anglo-Indian and European patients

	INDIANS		ANGLO-INDIANS		EUROPEANS	
	Number 2,021	Percentage	Number 94	Percentage	Number 43	Percentage
Fibrotic	108	5·3	12	12·7	17	39·5
Exudative	1,879	93·0	81	86·2	26	60·5
Pleurisy	34	1·7	1	1·1	...	...
Previously healed lesions ...	321	15·9	30	31·9	19	44·2
Acute-initial, 1 to 24 months	1,560	77·2	67	71·3	27	62·8
Chronic-recrudescent ...	461	22·8	27	28·7	16	37·2
Localized	397	19·6	21	22·3	24	55·8
Moderately disseminated ...	783	38·7	45	47·9	16	37·2
Markedly disseminated ...	841	41·6	28	29·8	3	7·0
Unilateral	581	28·7	20	21·3	18	41·9
Bilateral	1,440	71·3	74	78·7	25	58·1
With cavities	1,482	73·3	61	64·9	21	48·8
Cavities with soft walls ...	1,395	94·1	50	82·0	13	61·9
Cavities with moderately hard walls	87	5·9	11	18·0	8	38·1
With single cavity ...	704	47·5	32	52·5	14	66·7
With two or three cavities ...	385	26·0	14	23·0	4	19·0
With multiple cavities ...	393	26·5	15	24·5	3	14·3
Activity of disease shown by Schilling count and sedimentation rate—						
Low	157	9·3	13	16·9	5	17·9
Moderately high ...	315	18·7	13	16·9	12	42·9
High	1,215	72·0	51	66·2	11	39·2

From this Table it will be seen that among the 2,021 Indian patients, only 5·3 per cent showed any evidence of marked fibrosis, while 93 per cent showed a predominantly exudative type of disease. Previously healed lesions, as denoted by calcified spots either in the lung or hilus, were seen in only

15·9 per cent. Only 19·6 per cent showed a tendency to localization, while as many as 41·6 per cent showed marked dissemination. In 71·3 per cent of the patients, both lungs were involved. As regards cavities, 73·3 per cent of the patients showed cavitation, 52·5 per cent of these showing more than one cavity; the walls of the cavities in 94·1 per cent appeared soft, and only 5·9 per cent showed evidence of even moderately hard fibrous walls. As regards resistance to the disease, judged by the duration of symptoms before the patients came to the sanatorium, 77·2 per cent of the patients belonged to the 'acute-initial' group, according to Cummins' classification, that is, with continuous symptoms of less than two years' duration, and only 22·8 per cent to the 'chronic-recrudescent' group, that is, with symptoms lasting more than two years, or with symptoms followed by intervals of good health. As many as 72·0 per cent showed high activity of disease, as judged by the blood.

The Europeans are very few in number, being only 43, but among them the type of disease approximates to what is found among Europeans in the west. In the group 39·5 per cent showed evidence of considerable fibrosis and 44·2 per cent of previously healed lesions; 55·8 per cent showed a tendency to localization of the disease.

The figures for the 94 Anglo-Indian patients indicate a type of tuberculous disease in the lung between that of the Indians and Europeans.

Another investigation into the distribution of lesions in the lung (Table V) shows that as high as between 71·2 and 73·1 per cent of the patients have lesions in the middle zone of one or both lungs, and 32·6 to 33·6 per cent have lesions in one or both bases. It is recognized that the more chronic type of the disease is found in the apical and sub-apical zones of the lungs, and that lesions in the middle and basal zones are usually of the more acute type.

The results of these investigations show that for Indian patients the disease is of a very serious type; it is acute, rapidly developing with little tendency to show a natural

resistance and healing. The allergic condition is such that there is a severe exudative reaction followed by a rapid breaking down of tuberculous tissue with a resultant cavity formation.

TABLE V

Distribution of Lesions in the lungs

	RIGHT LUNG LESIONS				LEFT LUNG LESIONS			
	Apic	Sub-apic	Mid-zone	Base	Apic	Sub-apic	Mid-zone	Base
Number	1,141	1,368	1,438	658	973	1,220	1,478	678
Percentage	56·5	67·7	71·2	32·6	48·1	60·3	73·1	33·6

Conclusions

In the earlier part of this paper a review has been made of a tuberculosis survey in a limited area and of a study of the type of disease found in Indian patients. The survey, of course, is too small and limited to be able to form from it any general conclusions for the whole of India or even for districts or areas outside that surveyed. It does, however, support the general impression that tuberculous infection is widespread even in the villages.

The study of the type of the disease found in Indian patients can perhaps be used for a general conclusion, as the patients included in the study have come from all parts of India, and little difference has been noticed in the type of the disease in patients coming from different parts of the country. It may be concluded that for Indians as a whole tuberculosis is a very serious disease, because of its tendency to show itself in an acute, rapidly developing form. This reveals a general lack of immunity among Indians as a race.

The African investigations, already referred to, show that, as long as the Africans remain undisturbed in their own natural surroundings, tuberculosis does not appear as an epidemic disease, but it spreads comparatively slowly, and there are a large number of relatively mild and chronic forms of the disease with some severe cases. But when once the conditions of life

are changed, by, for instance, migration to the mines, the situation becomes quite different, and a great many succumb to a very acute type of the disease.

In India we seem to have a similar situation. There is a widespread incidence of tuberculous infection, as far as can be judged; at the same time the immunity response is very low. Conditions of life in India are rapidly changing. The villages are being rapidly brought into contact with towns and industrial centres through improved means of communication. A liking for the amenities of civilization attracts people from the villages. There are more opportunities for infection in the increased concentration of people for education as well as for industry. The poverty of the villager makes him easily lured to the town in the hope of quickly getting rich there. Life in the city is usually associated with much increased strain both mental and physical. Not only is this true in the city, but it is equally true wherever civilization is making headway, even in the villages.

The changing conditions of life in India to-day, taken in conjunction with wide-spread infection and a low immunity, make tuberculosis an increasingly grave problem.

The Trichinopoly District Medical Association

Minutes of the General Body Meeting held at the Government Head Quarters Hospital, Trichy, at 5 p. m. on Saturday the 17th September 1938.

THE General Body Meeting of the above Association was held at the Government Head Quarters Hospital, Trichy, when 26 members were present.

Dr. T. N. Subramanian Iyer presided and Dr. Balammal, M.B., B.S., was At Home to the members.

After refreshments the Secretary read the minutes of the previous meeting and was adopted.

The following cases of Clinical Interest were demonstrated:

1. Fusiform Tumour. (Lympho Sarcoma) left side of the neck for differential diagnosis by Dr. Raman Menon, F.R.C.S., Trichy.
2. Pernicious Anaemia (Cured by blood transfusion) By Dr. T. V. Srinivasan, M.B., B.S.
3. Melanotic Carcinoma leg (Amputation).
4. Recurrent polypus nose.

   (*b*) Removal of foreign body under Broncoscope.
5. Rickety Liver for differential diagnosis.

This was followed by an interesting Film Demonstration by Bayer on blood.

After the President's concluding remarks the meeting terminated with a vote of thanks to the Chair and to the District Medical Officer, Trichy.

Minutes of the General Body Meeting held at E. R. High School, Trichy at 5 p. m. on Saturday the 20th August.

A General Body Meeting of the above Association was held at E. R. High School, Trichy, when 26 members were present.

Dr. C. E. R. Norman, the President of the Association, presided and Dr. T. S. Balasubramanian was At Home to the members.

The Secretary read the minutes of the previous meeting. The amended resolution submitted by the Sub-Committee regarding the Honorary System was passed.

There was Clinical Demonstration on Tropical Ulcer of the foot from the Government Hospital, Trichy, by Dr. T. V. Srinivasan.

After the President's concluding remarks the meeting terminated with a vote of thanks to the Chair.

O. R. BALU,
Hony. Secretary.

The Trichinopoly District Medical Association
TRICHINOPOLY

Statement of Account up to the end of September 1938.

RECEIPTS	Rs.	A.	P.	PAYMENTS	Rs.	A.	P.
Cash Balance ...	64	1	3	Clerk's pay up to Sept. from 15th Aug. '38 ...	9	0	0
Bank Balance	210	4	2	Hire of Office for Aug. '38	5	0	0
Subscription from Members	55	0	0	Additional Clerk in the absence of old Clerk.	5	0	0
Subscription from Advertisements	42	13	0	Stationery ...	2	0	0
				Printing ...	16	0	0
				Postage	12	12	0
				Baksheesh to peon for G. B. Meeting ...	1	0	0
				Sweeping Charges ...	0	4	0
				Cycle repairs ...	3	4	0
				Bank balance at the end of the month (including the deposit of the Clerk) ...	268	5	2
				Cash balance ...	49	9	3
TOTAL ...	372	2	5		372	2	5

1st October 1938.

M. C. CONDILLAC,
Hon. Treasurer.

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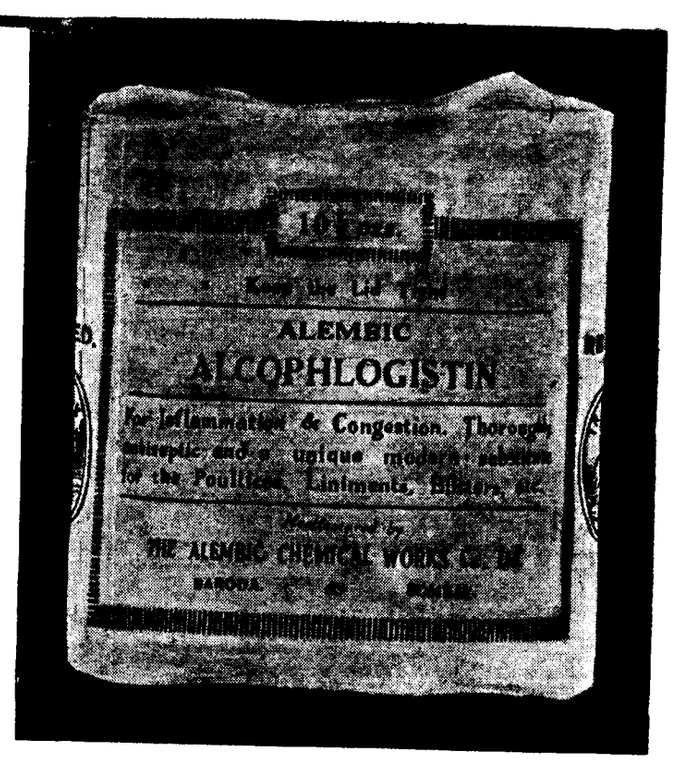
(REGISTERED)

The Editor requests contribution on Medical, Topical and News Subjects from Members of the Association and other medical men. Contributions should be sent typed in duplicate and signed by the contributor. In case where it is desired to write under a nom-de-plume the signature must be included for the information of the Editor as a token of good faith.

October, 1938.



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CONTENTS

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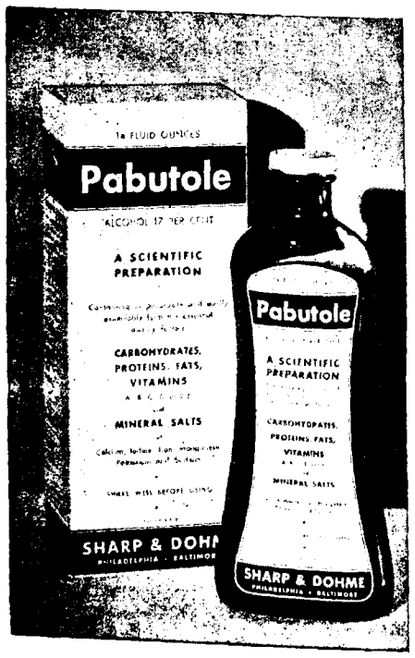

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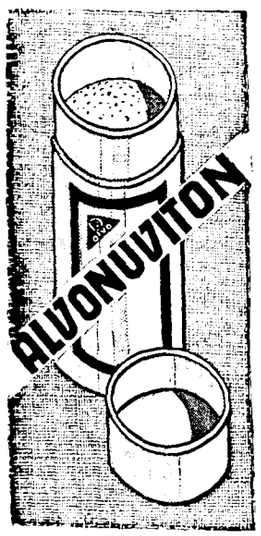

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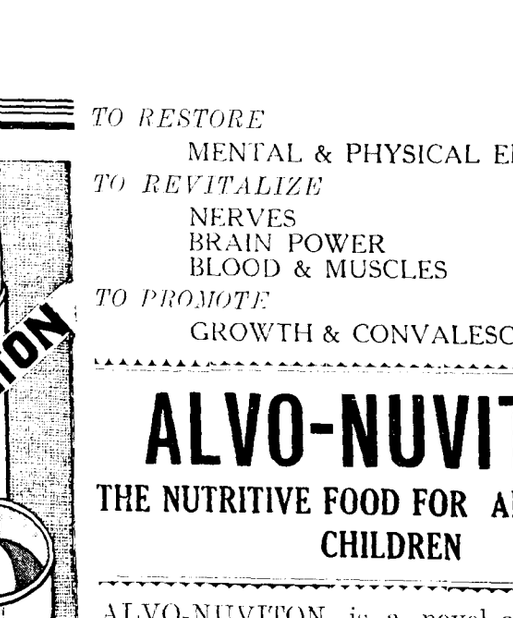

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The Treatment of Intestinal Amoebiasis

BY

P. D. BHAVE, M. B. B. S., D. T. M.

AMOEBIC dysentery is one of the very common diseases of mankind and it is believed that as much as 10 to 15 per cent of the world population are sufferers from this malady. It is particularly prevalent in the tropics and in India, it is a great source of sickness and invaliding, particularly among the working class such as mill-hands, dock-labourers and other menials. The disease is rarely seen in its acute form, the common form being the chronic variety and this is the reason why good many cases do not come to the notice of the medical practitioners.

The cystic stage is the resistant as well as the infective stage of Entamoeba Histolytica. The cysts can live outside the body for some days and cause new infection when they are swallowed with water or food. Cysts do not develop in the small intestines but do so in the upper part of the large intestines where plenty of fluid is present. Excystition results in the escape of a single quadrinucleate amoeba through a minute perforation in the cyst wall. These amoebulae then enter the crypts and produce the typical lesions. They spread along the submucosa which is the chief seat of action of the amoebae. Amoebae act by means of their proteolytic and tissuelytic substances which they produce. Some of these find their way to the portal radicles and are carried to the liver

and other organs where they produce the typical non-inflammatory necrotic amoebic abscess. Entamoeba histolytica does not live on the intestinal contents as other amoebae do but it lives on redigested tissues and red blood corpuscles and this fact clearly distinguishes it from other amoebae. The chief habitat of E. histolytica is not therefore the intestinal cavity but the intestinal submucosa wherein it produces its typical tissue-lytic action. This is in short the pathology of amoebiasis which is necessary, to understand the treatment properly. The following is a synopsis of the numerous treatments advocated by various eminent workers in the field.

The remedies used against the amoebic dysentery may conveniently be classified under two main headings A. Curative and B. Palliative.

A. Under the curative remedies, the following are included.

I. The vegetative alkaloids of ipecacuanha which pre-eminently include emetine and its preparations.

II. Vegetative alkaloids of Holarrhena Antidysenterica which include the Kurchi products.

III. The synthetic drugs amongst which the following require special mention: Carbarsone, Yatren, Stovarsol.

B. Under the palliative remedies we have the following two important 'ones: i. Plantago Ovata or Isabgul, ii. Aegle Marmelos or the Bael fruit.

It is very essential to give a note of warning regarding the drugs used in the curative treatment of intestinal amoebiasis. All these are as potent in action as they are toxic. It is therefore essential that the dosage should be within the margin of safety for each particular drug; yet, short courses and smaller doses will not eradicate the disease, but will only ameliorate the acute symptoms. The treatment to be efficacious, should be continuous, prolonged over a sufficient period and the doses must be adequate. Complete eradication of a chronic infection is a pretty difficult task because in a good many of these supposedly cured cases, the infection persists at a sub-clinical level as in chronic malaria and the patient's own natural powers of resistance is able to keep the parasites under check.

The Criteria of cure: In amoebiasis, temporary or clinical cure and permanent freedom from infection most often go as synonyms. Some years back authorities took cure to mean freedom from all symptoms and the absence of amoeba (both vegetative and cystic forms) in the stools for a period of at least six months after the cessation of the symptoms. This criterion was of little practical use because once the symptoms have ceased it is rare for a patient to come back to the hospital off and on for six months just to know if the eradication of the infection is complete or not. Recently the authorities at the Carmichael Hospital for Tropical Diseases, Calcutta, have formulated a practicable criterion of cure. A case is considered as cured for all practical purposes when six stool examinations done on six different days within a fortnight after the cessation of the treatment are negative as regards the amoebae (both vegetative or cystic forms) and Charcot Leyden crystals.

The Treatment of Acute Amoebic Dysentery: A case of intestinal amoebiasis may start as an acute case or more often as a chronic one but with occasional acute exacerbations. The treatment falls under the following important heads: (*a*) Rest, (*b*) Diet, (*c*) Medicinal.

It is absolutely necessary that a patient with acute symptoms must be put to bed. Bed-pan should be used to prevent exhaustion from too frequent and copious purging. Rest is essential both to the body as well as to the intestines. Complaints of tenderness of calf muscles will be relieved by massaging and hot water bottles. Chills, exposure to cold or fatigue both during the treatment and for some months afterwards should be carefully avoided.

Diet in Amoebiasis: The main lesions of amoebiasis are in the intestines and it is therefore essential that this organ gets functional rest so that it would better resist the amoebic invasions and the recovery be more rapid. Diet is therefore of great importance in the early stages of the disease. For the first few days fluids and easily assimilable foods such as chicken broth, rice water, rice-kanjee, egg albumin, barley water, etc., may be given. The diet should be simple nutritious

yet one yielding low residue. It should not irritate the bowels. A high vitaminous diet is beneficial in aiding rapid healing of amoebic ulcers. In later stages diluted or citrated milk, butter-milk, Benger's food, Horlick's milk, etc., may be allowed.

Such strict dieting is not necessary in the subacute or chronic cases but all the same spices, chillies, excess of tea, coffee, alcohol, and smoking, raw meat, etc. should be avoided or at least restricted. Milk, buttermilk, sago-pudding, arrowroot, soft boiled rice, etc., and vitamins in some form or other should be allowed liberally. Patients supposed to be cured may at times suffer from looseness of bowels, abdominal discomfort, etc., because the gastrointestinal tract in some cases remains very susceptible for a long time even to slight irritations. Exposure to chill, excessive fatigue or indiscretion in diet may precipitate an attack of looseness of bowels.

Medicinal Treatment of Acute cases: The following are the two drugs of choice: (1) Emetine, (2) Carbarsone.

Ipecacuanha was used for the treatment of amoebic dysentery in old days as far back as 1829. The root was used both in the form of powder and decoction. Ipecacuanha soon fell out of use because of the difficulty of maintaining adequate doses. The treatment in the old days had to be a very prolonged one and the doses were too large. On both these accounts, patients were very reluctant to undergo treatment with ipecacuanha. Moreover it also caused undesirable symptoms such as vomiting, headache, cardiac depression, etc. Rogers was the first to start emetine injection for the treatment of amoebic dysentery.

The latest and a very useful treatment is the one advocated by Brevet Col. Chopra (Director, School of Tropical Medicine, Calcutta) This method is in practice in the Carmichael Hospital for Tropical diseases, Calcutta and in many other hospitals.

Brev. Col. Chopra's treatment: Patient is resting in bed with flannel abdominal binder on. He is kept warm by blankets and hot water bottles. Diet is restricted to fluids given at frequent intervals in small feeds, mainly barley water, rice-kanjee, chicken broth, egg albumin, etc. Bismuth carbonate in two drachm doses is given three times a day throughout the period of treatment. Emetine hydrochloride one grain

daily is given for six consecutive days intramuscularly half an hour after the first dose of bismuth. Bismuth carbonate is supposed to act as an alkaliser of the intestinal contents and thus making the intestinal contents unfavourable for the amœbæ. It is given in a tumblerful of water. Three days rest is given and again a course of three more injections of emetine is given. Emetine being toxic and cumulative drug the bowels should be kept moving regularly by giving saline aperients if necessary. In cases with acute abdominal pain and tenesmus, one may be required to give morphine 1/6 grain. Usually starch-opium enema (tincture opii min. 40 & mucilage of starch 1 ounce) suffices to relieve the tenesmus. Turpentine stupes and poultices to the abdomen are also beneficial.

Pharmacological action of Emetine: It is essential to know the pharmacological action of emetine so that one could easily avoid toxic symptoms. Emetine is one of the three alkaloids of ipecacuanha. It is a white amorphous powder turning yellow on exposure to air. Emetine hydrochloride is a crystalline white powder and is the one used for injections. It is easily soluble in water and alcohol. Emetine is a protoplasmic poison and a local irritant. Emetine though effective, cannot be given by mouth, because of its irritant action on stomach giving rise to nausea and vomiting. Emetine increases the tone, circulation and movements of gastrointestinal musculature. It depresses the excitability and conductivity of the heart muscle. It is rapidly absorbed from mucous membranes or from the muscles but is slowly excreted; it is therefore a cumulative drug. About 1/6 of the drug is excreted by kidneys while the rest 5/6 is excreted from the intestines; it thus reaches the intestines in a good amount. Elimination continues for five to nine weeks; it is therefore important not to repeat emetine injection course for some three months after the first course.

Toxic Symptoms of Emetine: Alimentary: Nausea, vomiting, griping, looseness of bowels; other signs of toxicity are low blood pressure, muscular weakness, cramps, feeling of faintness, cardiac arrhythmia and œdema both cerebral and pulmonary may occur.

Cardio-Vascular System: The harmful action of emetine is most marked on the myocardium. Emetine depresses the excitability and conductivity of the myocardium thus producing slowing and dilatation of the heart. In toxic cases heart gets irregular, auriculo-ventricular dissociation occurs and death supervenes from ventricular fibrillation.

Therapeutic dose of Emetine: Total dose of emetine hydrochloride should not exceed 9 to 12 grains. Daily dose for adults is 1 grain intramuscularly. The dosage has to be reduced in case of debilitated persons and in persons with low bodyweight. In case of women the dose should be a little less. For children under three years, ¼ grain from 3 to 6 years, ⅓ grain per day may be given for 9 doses. Deutsch (1925) recommends the following doses for children.

0·01 to 0·02 grm. for a child of 6 months.

0·03 to 0·05 grm. for a child of 1 year and a total quantity upto 0·12 grm. For older infants a total quantity upto 0·15 grm. can be given.

Emetine can be given by mouth in keratin coated pills but it produces nausea and vomiting.

Colonic irrigation with emetine (1·0 gram of emetine in 1000 c.c. water or saline) has given good results at the hands of some workers; but it is also irritating. Emetine hydrochloride should preferably be given intramuscularly because there are less sensory nerve-endings there and therefore less pain. If given subcutaneously it is very painful and may produce inflammation or even necrosis. The usual sites of injection are the buttock and the shoulder. When emetine is being given, it is absolutely necessary for the patient to be in bed and the doctor must daily examine the pulse and blood-pressure. No sooner the characteristic emetine pulse (weak, rapid and compressible) shows itself, emetine should be stopped as further administration will bring about ventricular fibrillation and death. A few patients show idiosyncrasy to emetine and in them even a single injection may produce stiffness and pain in the muscles lasting for several days. The pain caused by emetine can be considerably lessened by dissolving it in 1% solution of carbolic acid. Emetine has been given intravenously but it does not give any special

advantage. On the other hand it often produces nausea, vomiting and even cardiac depression. The intravenous dose should not exceed ¼ to 1 grain and should be dissolved in at least 15-20 cc. of sterile saline.

After emetine treatment the patient should not over-exert; no sooner the pulse goes up, the patient should be advised to go back to bed so as to avoid heart complications. Emetine can safely be given to pregnant women. After therapeutic doses of emetine, the concentration of the drug in circulation is never more than 1 in 150,000 and such weak dilutions can never precipitate abortion. In menstruating women, the treatment is best started after the period is over, but in urgent cases requiring immediate treatment, no serious harm will be done by starting the medication at once.

In acute cases, emetine acts like a charm. This is because the bowel is in a hyperaemic condition and therefore emetine reacts on the invading amoebae in the crypts and the submucosa and the site of ulcers. Whenever there are chances of a hepatitis as shown by tenderness in the liver region emetine should be used in preference to carbarsone. In amoebic hepatitis emetine again acts like a magic. In such cases 2 to 4 injections of emetine followed by carbarsone, has been found to be effective. The other combinations of emetine are generally used in chronic cases.

Carbarsone: It is a very potent but a toxic compound of arsenic. It is a white crystalline powder with no taste or odour. It is available in capsules of 0·25 gram each. It is less toxic than stovarsol and at the same time a more efficient amoebicide. It is given by mouth in hard gelatin capsules. The dose is 0·25 gram twice a day for 8 to 10 days, with a cup of tea. The average total dose is 75 mgm. per kilo and given in 10 days. In therapeutic doses it has no ill effects on the liver. In cases of hepatitis emetine to start with and then carbarsone works well. The drug, beside having action on other intestinal amoebae has the advantage of being easy to take. It is also cheap. Carbarsone is very effective both in chronic as well as acute cases. During carbarsone treatment it is necessary to keep the bowels open with saline aperients. No alkalies should be given when carbarsone is being given.

The other known remedies for acute intestinal amoebiasis are those advocated by Acton and Knowles, Deck and Rogers.

Acton and Knowles: The patient is put to rest in bed; saline purge or castor oil ½ ounce with tinct opii 15 minims and routine fluid diet is given. Bismuth carbonate in 1 to 2 drachm doses is given every four hours to decrease the acidity of the colon contents. Exactly 2½ hours after the first dose of bismuth, emetine is given intramuscularly, one grain daily for eight days. Then rest is given for three days and again a course of emetine 1 grain daily for 3 or 4 days.

Deck's treatment consists of a similar course of emetine injections but he advocates larger doses of bismuth.

Roger's treatment (1929): Rest in bed; fluid diet and 1 grain emetine injected daily for 8 days followed by the following mixture at bed-time for one week:

Pulv. Ipec gr. xx to xxx		Tartaric acid gr. x	
Mucilage	dr. i	aqua	oz. i

The treatment of subacute and chronic cases:—This is really of prime importance because these are the cases which cause so much economic loss to the country on account of sickness and invalidity. These are also the cases that neglect treatment because the symptoms are not of acute type. The chronicity may follow an acute attack or it may start as such from the very beginning. In the latter type balance is established between the rate of invasion of the amœbæ and the reparative process in the bowels. The following are the various symptoms and signs met with in the chronic cases: looseness of bowels, alternating often with constipation, there is thickening and tenderness of the cæcum and of ascending colon; loss of weight, mental irritability, etc. Some of the secondary conditions which are the outcome of chronicity are worth-while mentioning here. These may be grouped under two heads:

I Direct due to septic absorption from the ulcers;

II Indirect.

Direct secondary conditions: I. Pyelitis. 2. Dysenteric arthritis. Complications 1. Hepatitis. 2. Amœbic abscess of liver and its sequelæ such as perforation into the lung or

abdomen producing septic peritonitis, pseudo-appendicitis, pelvic abscess, etc. 3. Amœbic abscess in other parts of the body such as in brain, spleen, etc.

Indirect conditions: Patient may come to the doctor for the treatment of one of the following conditions and not for amœbic dysentery, which is really the chief cause of the complaint. These conditions are allergic in nature and are supposed to be due to the liberation into the blood stream of poisonous pressor bases from the amœbic ulcers:

1. Asthma. 2. Allergic skin eruptions. 3. Neurasthenia. 4. Leucoderma. 5. Migraine, etc. In some of the cases of leucoderma, cysts of E. histolytica have been found and such cases have improved with emetine treatment.

The treatment of chronic cases falls under the following heads:—

1. drugs, 2. diet, 3. palliative remedies. The treatment of chronic cases presents considerable difficulty. In these there is a considerable thickening and fibrosis of the intestinal wall, fibrosis being particularly marked round about the base of the ulcers. The amœbae though less in number are all walled off by the fibrous tissue and the amœbicidal drugs and emetine cannot find easy access there. This is one of the reasons why treatment of chronic cases is rather patience-trying and unsatisfactory. The other causes of failure of treatment are as follows:

There is always existing secondary infection of the gastro-intestinal tract with B Coli, Flexner's group of dysentery organisms, Bact. pseudo carolinus, Bact. mucosus and streptococci etc. Since all these increase the acidity of the intestinal contents, emetine is rendered partially inert.

Diet: This has been fully dealt with in connection with the treatment of acute amœbic dysentery; chillies, spicy food, alcohol, and raw meat should be avoided. No article of food which would leave a large residue should be taken.

Drugs: for the treatment of chronic cases a large variety of drugs are used, the main ones being 1. Emetine Bismuth iodide, 2. Emetine periodide, 3. Vegetable alkaloids of

Holarrhena Antidysenterica (in English it is known as Bitter Oleander); it includes extract Kurchi liquid and kurchibismuth Iodide, 4. Organic arsenicals including carbarsone, yatren, and stovarsol. 5. Kaolin, osmokaolin, kaylene, silal, rivanol, dihydranol, arsemetine, gavano, ravitt paste, auremetine, emetol, vivoform, anayodin, etc.

Carbarsone is of special value in chronic cases. The doses and other things have been already dealt with.

Holarrhena Antidysenterica: Kurchi alkaloids are as effective as emetine in the treatment of intestinal amœbiasis. Injections are very painful and cannot be given. Fortunately the total alkaloids of kurchi can be given in large doses without any untoward effects. Acton and Chopra of the School of Tropical Medicine were successful in preparing an efficient compound of total alkaloids of Kurchi with Bismuth iodide. They named it as Kurchi Bismuth Iodide (K. B. I.). Acton & Chopra (1933) advocated the following treatment in chronic cases:—

Soda bicarb gr. 60.
Soda citras gr. 40.

To be given half an hour before the dose of Kurchi Bismuth iodide. The dosage of K. B. I. is 10 grains (0·6 gram) twice a day for 10 to 20 days. In obstinate cases they advocate extract kurchi liquid with or without isabul for long periods extending over three to four months. The dose used is 1 to 2 drams of Extract Kurchi Liquid daily.

Emetine-Bismuth-Iodide: 2 grains daily at bed time for ten nights. It is given in a dram or two of liquid paraffin. It causes gastric discomfort, nausea and vomiting; therefore, tinct. opii minim 15 or Chlorodyne or omnopon 1/6 gr. may be given half an hour before each dose. During treatment the patient should be kept in bed and given light diet. Treatment should be stopped if severe prostration or cardiac depression develops. The drug should be given as a loose powder in a hard gelatin capsule or in a paper cachet. It decomposes in the alkaline content of the duodenum, into emetine and iodine. It is found that one may have to resort to 2-3 courses to get complete cure. In no case should the drug be tried for more than 3 courses. It is found that the dosage and the duration of treatment in the succeeding courses requires to be increased. Usually the drug produces diarrhœa about the middle of the treatment but this is often beneficial because in addition to its specific amœbicidal action, it keeps the bowels open. Some authors advocate a combination of Emetine 1 grain intramuscularly with E. B. I. 2 grains daily for 6 to 9 days.

Auto-Vaccines

Chronicity is very often due to presence of secondary infection with B. Coli, B. Mucosus, B. Pseudacarolinus, organism of Flexner group; etc. It has been found that if a course of auto-vaccine made from these intestinal invaders is given prior to drug treatment the results are more satisfactory and the cure is rapid. Auto-vaccine treatment is therefore strongly advocated in chronic and relapsing case.

Emetine Periodide. (E. P. I.) Dose is 2 grains (0·13 gram) three times a day for 15 days. The drug is combined with 5 grains of exiccated ox-bile which is said to enhance the action E. P. I. It is the least toxic of all the emetine compounds and yet as effective as E. B. I. If necessary the course can be repeated after 10-15 days. It does not produce nausea, vomiting, or any depression of the heart. In no case more than three courses are to be given. The drug can be given in combination with emetine and stovarsol.

Stovarsol:—The dose is 0·26 gram in pill form twice a day for 10 days. To avoid toxic symptoms (optic neuritis) more than 8 grains per day should not be continued for more than 8 days. The drug is easy to administer and is well tolerated by patients. It is therefore of special value in practice. It has also hæmatinic properties. For obstinate cases the drug is best combined with emetine injection or with emetine bismuth iodide. The following doses are advocated for children 0·05 to 0·08 gram twice a day for infants under 1 year, 0·08 to 0·10 gram twice a day for infants 1 to 2 years. The drug sometimes produces measlelike rash and dermatitis.

Yatren (Synonyms: Loretin, Quinoxyl, etc.) It is given as a pill or as a cachet (8 grains in each): the total daily dose being 15 to 22 grains. The course extends for one week, and then

for three days a week for the following two weeks. It is also given as a retention enema consisting of 200 c.c. of 9/ solution. The enema should be retained for as long as possible. Before giving the enema, the bowels should be washed clear of all mucus by means of 2% soda bicarbonate solution. According to Akashi (1926) Yatren kills amoebae not only from the intestinal wall but also from the cavity of the intestine.

Kaolin; Osmokaolin; Kaylene; and Rivanol have been tried by some workers with success; but these have not been universally favoured. The drug treatment should always be controlled with repeated stool examination and if possible with a sigmoidoscopic examination. Recently it has been found that by bringing about a change of the intestinal flora, cure is more readily obtained in obstinate and relapsing cases. This is brought about by giving the patient curds, whey, lactose and lacto-bacillus tablets. All these help the growth of the lactic acid organisms which soon overgrow the dysentery organisms and finally completely replace them.

Palliative Remedies in Amoebic Dysentery: These are really of some importance because they help the healing of the ulcers. Two of these require special mention viz., Isabgul and Bael Fruit. Other Palliative remedies include Acocua clumus, Castela Nicholsons, Solaleva Bark, etc. Plantago Ovata (Isabgul): In India, it is grown in Sind, Punjab, and the U. P. When the seeds are steeped in water, a gummy mucilaginous substance is formed. It is odourless and tasteless. It is the pericarp of the seeds which swells up into this gummy substance. In India even laymen know how very useful Isabgul is in chronic dysentery. The mucilaginous substance coats the surface of ulcers and protects it. It also absorbs intestinal toxins. It is thus both an emolient and demulcent. It may be given alone or with Ext. Kurchi. Two to four tablespoonfuls of the seeds are shaken up in a glass of water and the whole thing is drunk. A little sugar may be added for taste. It is given twice a day. Roasted seeds are said to have better astringent action. Some people prefer to use just the pericarp of the seeds and this is stirred in water and taken. **Aegle Marmalos** (Bael fruit). It acts by its astringent effect. Raw and fresh fruit is preferable to dry or ripe fruit. Main action is due to its

demulcent action and partly to the small amount of tannin present in the fruit. It may be used in the form of sharbat or drink, marmalade or jelly or as liquid extract in 1/2 to 1 dram dose thrice daily.

Prophylactic treatment. In connection with this it will be worth-while mentioning a few words here with respect to the carriers who are mainly responsible for keeping up the infection as well as its spread. Carriers may be (*a*) healthy, (*b*) convalescents, or (*c*) contacts. In a healthy carrier there is a balanced mutual toleration between the host and the amoeba. A healthy carrier does not show any symptoms of infection. Yet he may any time go down with an acute attack of dysentery no sooner there occurs a lowering of the colonic mucosa and this is brought about by invasion of the heamolytic streptoccus into the amoebic ulcers. This is helped by indiscretion in diet, exposure to cold or by excessive fatigue. Carriers may not show any symptoms yet they will be passing infective cysts in their stools. They as a rule do show some vague symptoms and are very often bowel conscious. A carrier may show one or more of the following symptoms. 1. Irregularity of bowel action, looseness of bowels and constipation alternating; 2. Vague abdominal discomfort after food; 3. low grade erratic type of fever; 4. loss of appetite; 5. secondary anaemia, etc. As the carriers are the main source of infection, we should take care to see that our servants and cooks, who have to handle food and water, are not carriers and in cases of doubt a microscopic examination of the stools for infective cysts should be made. This precaution is particularly of importance in respect of servers and cooks engaged in public eating houses, college messes, etc., and proper drug treatment should be given to the carriers. Food should be protected from flies and care should be taken to prevent contamination of water and uncooked vegetables. As the infection is very prevalent in the poor classes, improvement of general hygienic and sanitary conditions is of primary importance. People should be educated in personal hygiene and cleanliness. In doubtful cases stool examination should be made and proper treatment given.

The Causes of Hæmorrhage from the Rectum

BY

J. P. LOCKHART-MUMMERY, F.R.C.S.

Emeritus Surgeon to St. Mark's Hospital for Diseases of the bowel

[*From The British Medical Journal.*]

VISIBLE blood may be passed per anum under a variety of circumstances and from many different causes. Such an occurrence is always alarming to the person concerned, and the medical attendant will at once be called upon to give a reason for it and reassure the patient as to its meaning. It will be necessary to account for the bleeding at once, or at least to arrange for some examination to be made which will reveal the cause. While it is advisable to reassure the patient, it is never wise to tell him or her that a little bleeding is of no serious consequence and to treat it by ordering a few suppositories. Such guesswork methods are dangerous both to the patient's well-being and to the doctor's reputation. It is therefore of some importance to consider what the cause of the hæmorrhage may be and how the source and reason for the bleeding may best be ascertained.

Naturally a large hæmorrhage will give rise to much more alarm than a small one, but it does not follow that the former condition is the more serious or has the gravest significance. Any bleeding, whether small in quantity or not, calls for immediate attention, and should never be neglected, as not infrequently very slight intermittent bleeding is the first sign of a malignant growth somewhere in the intestinal tract; nor is it safe to assume that because the patient is quite a young person malignant disease can be ruled out. I have seen a child of 8 years with carcinoma of the rectum, and although the condition is rare under the age of 40 there are quite enough cases to make us very cautious.

It is a curious and not unimportant fact that patients who have suffered for some months from what they describe as

'slight bleeding" at stool often exhibit a much more serious degree of anæmia than those who have had one large hæmorrhage. The probable reason for this is that patients are prone to attach much less importance to small often-repeated bleedings than to a single severe one.

Internal Piles

The commonest cause of hæmorrhage from the rectum is unquestionably internal piles, and this probably accounts for quite two-thirds of all the cases of bleeding from the rectum. I have seen patients who, as the result of frequent hæmorrhages due to piles, have been bled white and whose blood count had been reduced to under 3,000,000 red cells per c.mm. Not long ago I had in this hospital a patient with internal piles who was a professional pugilist. He had been bleeding at stool for over a year, and he was so reduced by it that he could hardly walk across a room without feeling faint. His skin was a greenish white and his red cell count was under 3,000,000 per c.mm. Before operating for the piles it was necessary to transfuse him with human blood. But such severe anæmia is unusual as the result of piles.

Bleeding due to piles can generally be recognized, because a simple examination of the rectum with a speculum will reveal the presence of easily prolapsible internal piles, and also because the characteristic form of bleeding is for the blood to drip or squirt after the bowel has been relieved. It is well to remember that the presence of internal piles does not exclude the possibility of a carcinoma higher up the bowel.

Carcinoma

The next commonest cause of bleeding from the rectum is carcinoma. When the blood is coming from a growth in the rectum or colon it is generally mixed up with the stool instead of being passed after it, as is usual with piles, so that the evacuations are red or brown from the presence of blood. The amount of blood, as a rule, is not considerable, and often it is present only intermittently. It occasionally happens that a sudden and very severe rectal hæmorrhage is the first symptom of a growth in the rectum or colon. When this occurs it

is due to the growth having ulcerated into a small artery. I have known several such cases, but they form only a very slight proportion of the total cases of carcinoma of the large bowel.

The diagnosis is as a rule readily established if a finger is passed well up into the rectum. Quite 80 per cent. of rectal growths occur within reach of the finger, especially if the patient is examined bimanually. If nothing is felt, an early examination with the sigmoidoscope should be made. It is hardly necessary to stress the paramount importance of diagnosing a growth in the bowel at the earliest possible moment, when alone a satisfactory chance of curing the patient by operation exists—a chance which will be seriously jeopardized by a delay of some weeks or months. In diagnosing the cause of the bleeding, therefore, the first requisite is to eliminate the possibility of a growth in the rectum or sigmoid, and the only way to do this is by making a careful digital and sigmoidoscopic examination of the rectum. I often find that patients have been examined by means of a barium enema and radiographs when a simple digital investigation of the rectum would have revealed the lesion. An x-ray examination should only be ordered after a digital and sigmoidoscopic examination has given negative results.

Apart from carcinoma of the rectum, an innocent adenoma will cause hæmorrhage. The amount and frequency of the bleeding may be quite considerable if the adenoma is a large one. These adenomata can usually be felt by the examining finger, but as they often are soft and velvety they may be missed by those who are not accustomed to examining the rectum. They can, however, be very easily seen if a speculum is passed. They should always be freely removed, as there is grave risk of their becoming malignant. They should be looked upon as a precancerous condition.

Peptic Ulcer

Hæmorrhage due to a bleeding duodenal ulcer, as is well known, generally results in tarry stools or melaena, owing to partial digestion of the blood on its way down the alimentary tract. When, however, the bleeding is very copious, or occurs

rapidly, and the bowel is not loaded, the blood may be seen in the stool as bright fresh blood similar to that which is present when the source of bleeding is in the rectum or lower colon. This is rather confusing, as in the absence of very definite symptoms of duodenal ulcer the source of the blood may be unsuspected. I have known this happen on several occasions, and in one case it was some time before it was realized that the patient was bleeding from a duodenal ulcer and not from his large bowel.

A very rare and obscure cause of rectal hæmorrhage is a peptic ulcer in a Meckel's diverticulum.

A few years ago a girl aged 15 was sent to me from South Africa for treatment. The history was that since the age of six she had been liable to sudden and very severe hæmorrhages from the rectum. In three of the attacks she had nearly died, and on five occasions it had been necessary to give transfusions. Beyond the bleeding there were no symptoms at all. Most careful examination and x-ray investigation had entirely failed to reveal any cause for the condition, and except for a secondary anæmia her blood picture was normal. I was quite unable to make a diagnosis, and finally decided to explore her abdomen. I then discovered a Meckel's diverticulum five inches long, with a peptic ulcer at the end of it. I removed this, and since then she has had no further bleeding.

Cobb (1936) collected 100 cases of peptic ulcer in a Meckel's diverticulum. Of these, seventy-one had hæmorrhage, and in nearly half the cases there was no other symptom. In cases where there are repeated attacks of profuse rectal hæmorrhage, especially in young persons, the presence of a peptic ulcer in a Meckel's diverticulum should be suspected. It is usually impossible to demonstrate the diverticulum by radiographs, but this was achieved in one of the cases described by Cobb.

Angioma of the Rectum or Sigmoid

The curious condition is congenital and resembles a cirsoid aneurysm of the skin or a cavernous naevus. The wall of the rectum and colon is occupied by a mass of huge blood vessels just beneath the mucous membrane. These enlarged vessels may be arteries or veins, or a combination of both.

In a case which I have under my care at the present time some of the vessels when looked at through the sigmoidoscope are as large as the aorta, and can be seen pulsating. The angioma in this patient starts about four and a half inches up the rectum and extends for fifteen inches almost to the descending colon. This patient had to have an abdominal section, and I thus had the opportunity of examining the bowel from the abdominal aspect. The engorged blood vessels in the affected portion of the colon made the tissue quite firm and stiff, like erectile tissue, and this applied also to the mesentery, which consisted of masses of enormous blood vessels.

This patient is now over 60 years of age, but all his life he has suffered from occasional severe haemorrhages.

There are some twelve of such cases on record, and most of them resulted in death from bleeding. One patient, whom I attended for some years, eventually died in Paris from a sudden severe hæmorrhage. The condition is not one which lends itself to surgical treatment. The great size and number of the feeding vessels preclude the possibility of excising the affected portion of bowel.

Local Injury

A few years ago I was consulted by a lady, 60 years of age, who had a sudden rectal hæmorrhage while in apparently good health. During the following twelve hours the bleeding was continuous and she lost over two pints of blood. On examination nothing could be found, except that the lower bowel was full of blood-clots and fresh blood was still coming away. The patient was put to bed and treated with cold rectal douches containing hazeline and the administration of morphine. This stopped the bleeding, or at any rate considerably diminished it, and next day after washing out the bowel I was able to make a thorough examination with a sigmoidoscope. I then saw that there was a small cut about a quarter of an inch long in the wall some two and a half inches up the rectum. This cut had partly divided a small artery, which could be seen pumping when the clot was removed. The bleeding was stopped by cauterizing the bleeding spot and there was no further trouble.

It subsequently transpired that this lady had swallowed some broken pieces of china a fortnight previously, and I have no doubt that a fine chip had cut the mucous membrane.

I have also seen bleeding from the rectum due to injury resulting from a man breaking a shooting-stick while out shooting pleasants and impaling himself on the lower broken end. The hæmorrhage in this case was much less important than the question of damage to the bladder and peritoneum. Although the broken shooting-stick had gone into the rectum for six inches or so the peritoneum was not injured, but the back of the bladder was torn. The patient was fortunate enough to survive this injury.

Hæmorrhage from the rectum may, of course, complicate operations upon the rectum, and this I shall refer to again later.

Ulcerative and Haemorrhagic Colitis

Ulcerative colitis or proctitis may cause serious bleeding, but as a rule the septicæmic condition quite overshadows the bleeding. A blood count should always be made from time to time, as any considerable degree of anæmia is certain to prejudice the patient's chances of recovery, and should be corrected by transfusion.

Hæmorrhagic colitis causes very serious bleeding, and in this condition the bleeding is the chief symptom. The ætiology of this variety of colitis is still uncertain: many people believe it to be a form of ulcerative colitis; but in typical cases there is no ulceration, and my own opinion is that it resembles pellagra of the skin, without its characteristic blood picture. The great majority of the sufferers are young adolescent women, in whom the condition appears intermittently, often over many years.

One patient of mine, who was married and had one child, lived in a garden suburb on the outskirts of London. One evening her husband returned home to find a large pool of blood just outside the front door and a trail of blood all along the passage inside the house. He not unnaturally thought that his wife had been seriously injured or even murdered, but on reaching the kitchen he found her cooking the dinner. She was standing in a pool of blood, but was unaware of the fact.

Although the bleeding in these cases may be very considerable it is seldom really serious, and I have never heard of a fatal case. Rest in bed and hypodermic injections of thrombokinase, in the shape of haemoplastin or one of the other similar preparations, usually soon control the bleeding. Recurrences are common, however, and until we know more about the ætiology of the condition we can do little to prevent them. It is usual to administer calcium in these cases, but there is some doubt about the efficacy of this treatment.

High Blood Pressure

Bleeding from the rectum may occur in persons with very high blood pressure, and may be profuse. The bleeding almost certainly comes from a vein in the rectum or colon that has ruptured. I have seen several such cases where the patient had had a really severe hæmorrhage, but when subsequently examined with the sigmoidoscope the source of the bleeding could not be determined accurately. In one patient, an elderly lady, the blood pressure previous to the haemorrhage was as high as 260 mm. Hg. and there were signs of hardened arteries.

The hæmorrhage in such cases is beneficial rather than otherwise, and if the cause is known the patient should be treated by occasional venesection when the pressure gets high, instead of being allowed to bleed vicariously from the bowel. When a sudden and severe hæmorrhage has occurred in an elderly subject this cause should be considered and the blood pressure taken. Naturally, after the hæmorrhage the blood pressure will have been considerably reduced, so that it is important to ascertain, if possible, what it was before the hæmorrhage.

A case is reported by Elgood (1930) of a woman who had profuse hæmorrhage from the rectum, resulting from an extra-uterine pregnancy having ruptured into the lower end of the colon.

A cause of rectal bleeding which is very uncommon in this country, but common enough in Egypt, is infection with the parasite *Bilharzia haematobia*. There are two varieties of this parasite; in one case the ova perforate into the bladder

and cause hæmaturia, but in the other they perforate the rectal mucosa and cause rectal hæmorrhage. I have seen two such cases in England. An examination with the sigmoidoscope reveals numerous minute hæmorrhagic ulcers in the mucous membrane, and if one is fortunate the characteristic parasites can be recovered from scrapings from these ulcers. The only treatment is by means of tartar emetic injections. In Egypt such cases are very common, and in time the rectum becomes a mass of granulomatous tissue.

Methods of Controlling Rectal Haemorrhage

Hæmorrhage from the rectum is a complication which may follow any operation upon the rectum, and it happens in about 1 per cent. of cases after all such operations. The most common time for a hæmorrhage is about the eighth day after operation. It is essential that anyone who is going to look after rectal operation cases should know how to control the bleedings, as it always occurs unexpectedly and the necessity for immediate attention will be urgent.

The first thing to do when bleeding of any consequence is noticed is to pass a short piece of rubber tubing, about three-eighths of an inch in diameter and three or four inches long, through the anus. The tube should have a safety-pin or a piece of thread through the outer end to prevent it slipping entirely into the rectum. If the bleeding is coming from some small vessel this simple procedure will generally stop it, and if the bleeding is more serious the tube will allow the blood to escape and not accumulate in the rectum: in other words, the tube will prevent a concealed hæmorrhage. If the bleeding restarts after a few minutes it is clear that it is not under proper control, and the surgeon should proceed to pack the lower end of the rectum. As a rule this can quite well be without an anæsthetic if done by the following method. A piece of rubber tube about six inches long and the three-eighths of an inch in diameter is taken and a thin strip of dry gauze about an inch wide is wrapped tightly round one end in a figure of 8, so as to form a bulge; this should be just small enough to be pushed through a tubular speculum. The speculum is well greased and is passed into the rectum; the tube

with the bulge at one end is then pushed through the speculum until the bulge is above the speculum and the latter can be removed. The bulge of gauze on the tube will now be in the rectum and the end of the tube protruding from the anus. When the tube has been drawn down as far as it will come a large safety-pin is put through it about one inch from the anal opening. A long strip of gauze is then wrapped round the tube, outside the anus but inside the safety-pin. As this wrapping increases it will tend to pull the gauze bulge on the other end of the tube hard down against the bleeding point, and if properly done this will at once stop the bleeding. The plug should be left in place for forty-eight hours, the patient being given morphine if he feels much discomfort. Should the bleeding restart it will at once be evident, as fresh blood will come through the tube.

Another way to stop bleeding is this: Cut off the finger of a rubber glove and, after passing the end into the rectum, plug gauze into the open end so as to well distend the closed end inside the rectum. To prevent the plug slipping in, a safety-pin and a roll of gauze are used outside the anus. The tube method is the more satisfactory as the glove finger is apt to tear. If a plug such as this fails to control the bleeding the patient should be given an anaesthetic, and when all the blood has been douched away the bleeding-point can be looked for with a speculum. It is generally very difficult, or impossible, to pick up the vessel satisfactorily, and it is better to plug the rectum properly with a gauze plug and tube of suitable size.

Indigestion

BY

DR. D. SRINIVASAN

Sthenic Dyspepsia

WOMEN are prone to get indigestion more often than men. Genarally they are elderly maiden ladies and widows who are temperamentally neurotic and often consult the doctor for indigestion. They give a history of "Sinking in the Epigastrium" 2 or 3 hours after meals and they feel better immediately. They take some food. They are awakened in the early hours of the morning with heart burn, pyrosis, sneezing, hiccoughing and 'asthmatic' attacks which, however, rapidly subside as soon as they are able to disperse the wind of which the stomach appears to be full. Among those who suffer from the sthenic variety of dyspepsia, there are a very large number of patients who are pursuaded by their own reasoning that they want "Supporting", that their condition is due to general debility and nervous weakness; and that large and frequent meals are absolutely necessary to maintain their body fit. They become gluttonous and idle. Even if they are properly explained the real state of their condition they form an opinion that the doctors are quite unsympathetic towards their suffering and lose their faith in doctors and their advice.

An attempt should always be made to guide them properly regarding their diet and the necessity of good exercise in the open air. Even if we spend some hours in explaining the pathological condition it is not a waste as it would prevent a human soul from becoming a nuisance to their relatives and to the society in general. In case the doctor is not able to imbue confidence it is better to lose a patient than to share in the responsibility for the arterio-sclerosis and other serious conditions which sooner or later are bound to follow. Patients who pity themselves dwell on their weakness in heart-rending descriptions to pursuade the doctor to their view. So it is highly necessary that we should see the pitfalls and get accustomed to discredit

some of their woeful tales without letting them know that we do not believe the statements made by them. Our motto should be to guide them gently but firmly.

Treatment. The very first thing to be done is to clean the primaviæ. A dose of Calomel say 3 grs. at bed time followed by a saline purge in the morning will do good in clearing the toxic intestinal contents. Calomel acts not only as an indirect chologogue purgative but also as a powerful intestinal antiseptic. A gay and dissipated life should be forbidden. So should alcohol and tobacco. Warm baths may be ordered but here in India since everybody bathes at least once a day it need not be stressed. He should be instructed to masticate his food well which should be light but nourishing.

Regarding drugs we should prescribe those which have antacid properties. Patients should be asked to take the prescribed antacid immediately on the commencement of the symptoms or before the expected onset. Among the antacid I think the time-old soda Bicarb. fits in nicely. It is a good antacid pure and simple, very cheap and available anywhere and at any time. There is a disadvantage in using it in cases of flatulent and initiant dyspepsia for it has no sedative action and produces more gas. Here Bismuth comes to our aid. It is not only an antacid but also a good sedative. Bicarb. is the favourite of many reputed physicians but some prefer Bismuth Subnitrate. I do not like subnitras since it tends to constipate. If however one wishes to use it, one point should be borne in mind not to add Soda bicarb. to it as a lot of CO_2 is generated and this will aggravate the symptoms. Liq. Bismuth Ammoncitras also helps very much when given as mixture. In these drugs we have to prescribe more than the pharmaceutical dose as it is very insufficient.

mist Carminativa 4
soda Bicarb. not gr. 10 but gr. 20 to
Liq. Bismuth amm. citras 4
¼ part four times a day after food.

If the patient has not improved after giving alkalies and sedatives one should seriously revise his diagnosis as Colonic trouble sometimes stimulates gastric trouble and in that case

Calomel and high Colonic lavage will relieve the patient. In sthenic dyspepsia common salt should be omitted or consumption should be limited very much as Munatic acid in the gastric juice is believed to be common salt.

Asthenic Dyspepsia

As the term goes it is a common complaint of asthenic individuals. Over-worked and under-nourished persons, convalescents and nulliparous women of middle age in poverty-stricken conditions are the usual victims. In Trichinopoly many are from among the poor clerical staff and also in boarding houses, orphanages and convents for whom I was the medical attendant for a few months. They complain of dull aching pain in the chest which passes through the back—The pain is not relieved by food but gets more profound. They suffer from flatulence and palpitation and are generally constipated with stomach tibes and cracked lips. In these cases the Purgatives though they move the bowels have no action on the gas. On examination of the abdomen we find a very thin and badly developed abdominal wall. They take no interest in life and they are averse to any exercises as they imagine themselves too weak to do them.

Treatment. Coffee and tobacco should be restricted. Bowels should be carefully cleaned. Mineral waters like vichy water do good. The presence in them of common salt which we have seen is contra-indication in sthenic dyspepsia is here an advantage. Most of these waters are best taken in the morning and their effect is enhanced by the association with each dose of a tumbler of hot water.

Having regard to the deficiency of HCl. our object should be to supplement it. The dose should be enough to meet the requirement. The minimum dose of 15 minims combined with Pepsin and Strychnine may be given after food. The usual prescription is as follows.

acid hydro-chlor. dil. iss
liq. Strychnine m. 15
Pepsin Powder gr. 60
Aqua
1/3 part i. o. after food.

Along with this we can add quinine bihydrochlor. Liq. morphine and Liq. arsenicalis, which are quite compatible not only with each other but also with the other ingredients of the mixture. When the condition is of long duration it is better to add m. 10 of morphine hydrochlor. At the same time general tonics and injections of campolan and vitamin B. I think a patient without any other complication should do well with this treatment unless there are other complications which should be suitably dealt with.

The Trichinopoly District Medical Association

The Minutes of the General Body Meeting held at the Government Head Quarters Hospital, Trichy, at 5 p. m. on Thursday the 20th October '38.

THE General Body Meeting of the Trichinopoly District Medical Association was held at the Government Head Quarters Hospital, Trichy, on 20th October '38 when 26 members were present.

Dr. C. E. R. Norman presided. There was a lecture about 'Menengitis' by Dr. C. E. R. Norman and it was supported by Dr. Rama Menon.

There was an 'At Home' to the members on the occasion.

After refreshments the Secretary read the minutes of the previous meeting and was adopted.

The following cases of Clinical Interest were demonstrated by Dr. Rama Menon, F.R.C.S., Trichy.

1. Fibro Adenoma of the Shoulder Joint.
2. Rhumatoid Arthritis.
3. Compound Fracture of Tibia.
4. Liver abscess.

After the President's concluding remarks the meeting terminated with a vote of thanks to the chair.

O. R. BALU,
Secretary.

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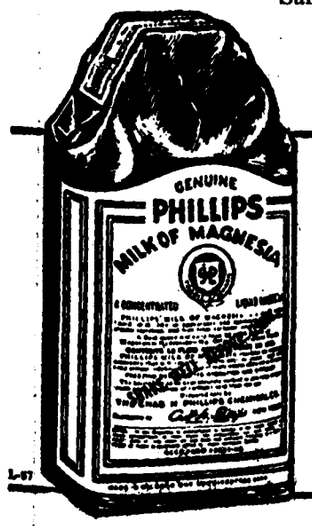

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(REGISTERED)

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November, 1938.

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OF

THE TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION
(REGISTERED)

Editor: M. C. CONDILLAC, M.M., M.R.C.S.

DECEMBER 1938

No. 12

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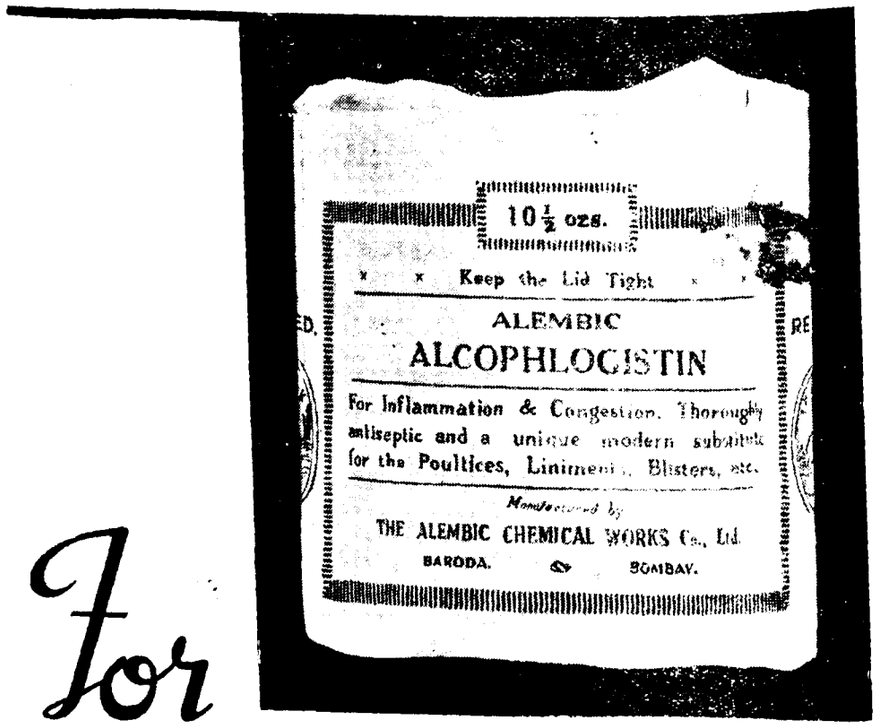

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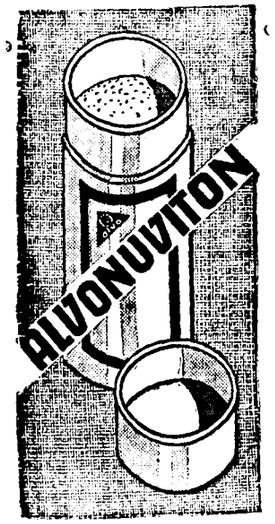

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Vol. V] DECEMBER 1938 [No. 12

Future of Medicine in India

BY

CAPTAIN E. S. GOPALAN, M.B., M.S., A.I.R.O., COIMBATORE.

(*From the Journal of South Indian Medicine*)

AMIDST the innumerable problems, political, social, economic and religio-political *alias* communal, which India has to solve and is attempting to solve, there is one problem, the problem of medicine in future India, which must be handled with tact, courage and considerable foresight. A great responsibility rests on the present generation of medical men to evolve a plan suitable to the peculiar conditions in which India stands, as regards the so-called different systems of medicine prevailing here. India is peculiar in that it has a rich heritage of its own, of a glorious past, in every sphere of human activity. It is not like the case of Africa, America or Australia, where the uncivilized and uncultured aborigines, met with practical annihilation on coming into contact with civilized races. India has changed, is changing and is going to change, but any amount of impact with other civilizations, can never shake it out of its deep foundations of a transcendental past. Of course, the recent centuries, including some centuries of the pre-Mussalman period, were comparatively a period of decline in medicine. But compared to the state of medical knowledge obtaining in contemporary European states, India was far ahead, even in its period of decay. No useful purpose will be served in trying to

find out the causes of decay, but suffice it to say that the one important cause was the unquestioning faith in all the proven and unproven medical dogmas of the past and the undue glorification of everything that was past without regard to one's own observations and experiences. Even in Europe this unquestioning faith in Hippocrates and Galen, giants though they were, was responsible for so much delay in the discovery of the circulation of blood by Harvey, an event of outstanding importance, in that it is the veritable foundation of the modern scientific medicine.

Historical Background

An unbiassed historian of the modern medical system, I refuse to call it allopathy or western system, must have to trace its origin in the ancient civilizations of the world, India, Egypt and China. Of these Indian civilization is the oldest. Indian medicine did go out of India. The Mahomedans imbibed it, who in turn gave it to Europe. In Sir Walter Scott's "Talisman", it is mentioned that during the Crusades, King Richard I, the lion-hearted, was treated for fever by a Mahomedan adversary by an effervescing draught. During the recent centuries this system took a scientific turn, and has come back to India with the advent of European civilization in India. India has been sleeping all these centuries, and when it woke up, like the proverbial Rip Van Winckle, it sees a new kind of medical system foisted on it as the official system. It looks upon it as an adversary, trying to kill it outright, little knowing, that the modern system is a great great grand-child of its own, who has returned to India, to greet the grand-parent after tremendous experience in its sojourn in various continents. The present problem is how to reconcile the grand-parent represented by the ancient Indian system and the world-experienced rationalistic and inquisitive grand-child. The grand-child must know, that the venerable grand-parent has still some treasures kept safely and secretly which will be bequeathed to the grand-child, on proving its bonafides that it will utilise the treasures properly and if necessary polish it out of its rust. The grand-parent also must know, that the child is born of his own flesh and blood, that he has an open mind, and will not hesitate to improve the treasure, after studying it, classifying it and incorporating it into its own bank account. The grand-child is bound to be immensely richer, if it cares for the hidden treasures of the grand-parent.

It was a great pity, that "Ayurveda", the science of life, did not, in the recent centuries, proceed towards its realization of its logical goal. Otherwise, it would not have been left to Europe to put fresh scientific foundations so lately as the 19th century. India should have done it at least a thousand years ago and India and the world would have been the gainer. But it was not to be. Now that the modern system, though developed recently, has shown remarkable progress, based as it is on vital ancillary sciences, it behoves us all to enrich it in every possible way. It shall put this matter in another way. Supposing India had not given any quarter to any European power and had continued to the present day ruled by the successors of Aurangazib or Shivaji, can any of us say that the modern scientific system might not have come to India at all? Japan was never conquered by any European power but still it has thoroughly imbibed the modern scientific system and is paying the European countries in its own coin. Possibly without the European advent, India might have taken to the modern system a little later, but not never. As in the case of railways, telegraphs, automobiles, aeroplanes, wireless, etc., India is bound to be modern with or without its European connexion two centuries ago. So, to accuse the modern system as a system foisted on India by a foreign nation down the throats of protesting Indians, is a ridiculous interpretation of an inevitability under modern conditions of international exchange of thoughts, ideas, culture and trade. If I am to judge the events of the future world, not the immediate future with its warring camps each small group calling itself a nation out to civilise the rest with its pet slogans and dogmas, but a future war-weary world, I should conceive it bound by common ideals, standards, culture and a greater civilization shorn of its war-provoking rakshasas or demons. In that greater civilized world, I want India should play its part nobly and well.

Ancient Indian Systems suited to Indian Constitution?

There is a fallacious popular saying that the Indian ancient systems alone are suitable to Indian constitutions under all

circumstances. Some of our political leaders also are victims of this fallacy and since our future destiny lies in the hands of our politicians, we must do propaganda to negative this fallacy. The human anatomy is the same everywhere and the human machine acts and reacts alike to external stimuli in identical manner. Drugs do not discriminate between black man, white man or yellow man. All drugs act equally and to the extent of their specificity on all constitutions. The organic materia medica has its replica in the leaves, roots, barks, and seeds of Ayurveda. The inorganic materia medica has its replica in the baspams and metallic preparations such as those of mercury, arsenic, copper etc., of Ayurveda and Siddha. The malted preparations have its counterpart in the lehyams and gulgandhs. The medicinal springs and baths of Western Europe have its equals in the hot springs in the neighbourhood of Babrinath and elsewhere. The system of massage has its well-studied rival in the famous Malabar systems of Navarakizhi and Dhara. Fundamentally the systems are the same. The drugs are the same or very similar. The drugs of the modern pharmacopœa are not grown in England or France exclusively. All the drugs of the modern pharmacopœa can be grown in India, and when that happens, you cannot say they are Western drugs. They are as much Indian as any other Ayurvedic drug. Even powerful drugs like Digitalis, Belladona, Nux Vomica, etc., are grown in India and exported to the European continent. So there cannot be any quarrel as to the geographical habitat of the drugs. As a matter of fact India, though poor as a country, is rich in its drugs of any variety and there is great future for Indian drugs of every denomination in the future world market. So even if India were to practise cent per cent modern system, it need not and should not depend for its drugs on any foreign country. Throughout the history of medicine, in all countries, empiricism did play the dominant part. Even to-day, in the modern system, empiricism has its own place and we cannot do away with it altogether. Even mistakes become discoveries if we are able to judge the mistakes properly. The mighty experiences of our forefathers, in ancient India, fell into unworthy hands, who did not have the capacity to enrich it but who claimed finality for all the truths, half-truths and even mistakes

of their forefathers. Hence our stagnation in the logical progress of the evolution of Ayurveda. At least we shall learn a valuable lesson by the ignorance and foolhardiness of our more immediate forefathers.

The Genesis of the Modern System

Not long ago, medicine practised in the European countries was, as Ayurveda has been in India these centuries, as much empirical, dogma-ridden, and authoritarian. Even at the time when the East India Company set its foot on the Indian soil, the English system was, considering the present advances, very primitive and did not possess any outstanding merit over our Indian system. A text-book of medicine, published one hundred years ago, in England, will make even a third year student, in a Medical College, look with contempt at the achievements and curious ideas of diseases which medical men entertained at that time. Most of the tremendous progress has been recorded within the last three-quarters of the century, and to this progress every nation, including India has contributed. So the modern system of medicine is strictly speaking international and no nation has the exclusive right or the monopoly of its discoveries. The system did not originate in England and come to India along with the merchandise.

To the foundations and the superstructure of this international system practically every country in Europe, America, India, China, Japan, Australia and New Zealand etc., has contributed, even as India has contributed to the international knowledge in Physics, Chemistry, Botany and Mathematics. The full life cycle of the malarial parasite was worked out in India. Our knowledge of plague and its prevention was worked out in Bombay. The dreadful Kala-Azar has lost its dread, thanks to the gift of a distinguished Calcutta physician to the suffering humanity. The names of Noguchi, that martyr to yellow fever, Kitasato, Shiga, etc. are not European but Asiatic. In Europe, not only England, but France, Italy, Germany, Russia, Czecho-Slovakia etc., have all contributed to the system. Keeping these facts plainly before us, to call the system as Western, allopathic or foreign, is a gross misrepresentation of a really international system.

The merit of the Modern System

The fundamental merit of the Modern System is that it has fully utilized the vast treasures of the physical and biological sciences in return for what medical men themselves formerly contributed to them. The physical and biological sciences have revolutionized the world in the 19th and 20th centuries, in all fields of human activity. We all deplore and condemn the prostitution of science for the creation of engines of human destruction, but all the same, we cannot but see, that in every sphere of our activity, the results of these discoveries have pervaded very minutely. Whether some of us want these or not, whether all these developments are eventually for our good or not we have to admit, that the present international life is dominated by it. Even the present Madras Provincial Government, having immense faith in the spinning wheel, does not hesitate to spend money for hydro-electric projects and radio-receivers for villages. It shows that the Government, in spite of its concern for the unemployed humanity, have to admit the usefulness of such of these latest innovations. It is but right they do it. In a similar way, the modern system of medicine, is entrenched, in secure scientific foundations by the advancement of the physical and biological sciences. The sciences of chemistry, physics, biology, anatomy, physiology, bio-chemistry, pharmocology, morbid anatomy and pathology, bacteriology, psychology etc., have been utilized to the full in illuminating the origin, progress, effects, diagnosis, prevention and treatment of diseases. The pure clinician might deplore so much intrusion of the ancillary sciences into his domain, but he has no right to restrict the uses of these sciences. In the region of traumatic surgery of bones, no doctor will be safe medico-legally, in a future enlightened India, if he does not demand a skiagram of the injured part. The so-called sprains of the wrist and ankle, of the olden days have been, in some cases, proved to be minute fractures of the navicular or the internal malleolus respectively. Without skiagrams, the fractures will be missed and the lesions thought of lightly. To give another example, cystoscopy and pyelography, utilizing the Edison's vacuum lamp, has placed urology on a firm foundation as an exact science. These are only two random

examples of the saying "Knowledge is Power." To ignore these triumphs of modern medicine, is to deny truth and what all it stands for. Charaka and Susruta, if they were to relive today, will gladly incorporate these modern procedures into their system, with an advantage to the science. Why not we, descendants of these illustrious men, catch a little of their spirit, at least?

Theories in Ayurveda

It must be admitted that man, in spite of all the epoch-making discoveries, is still imperfect. There is no limit to the knowledge which a man can have. Doubts there were, there are going to be. Perfection in all knowledge is impossible. Even if I am given two thousand years of life, and I go on discovering and discovering, at the end of my life, the sum-total of my knowledge will be very negligible compared to the things I will be ignorant of. Even in the modern system, some of our pet and so-called established theories, may be disproved by our descendants. Bacteriology was priding itself that it had settled the causation of some microbic diseases, but, De' Herelle's bacteriophage, as ultramicroscopic things, inimical to the bacteria themselves, have to be explained convincingly. We do not yet know the causation of cancer, though the theories of cancer, from Virchow's cell inclusions, to Lockhart-Mummery's theory of Gene-Mutation, are a bewildering and imposing study. So all the theories of the Modern system, as well as the Ayurvedic system, must be judged in the historical perspective. A correct theory or hypothesis might establish a science even as the Atomic theory established chemistry. But when another theory, like the election theory, explain the phenomena better, the original theory must be given the go-by even as the old Phlogiston theory is mentioned now-a-days to ridicule it. Some of the theories have given great impetus to the state of sciences then existing and should be given a proper niche in the history of that science. But to hold on to it in spite of the odds of proofs against it, is to deny truth. The tri-dosha theory is considered to be the basis of Ayurveda. It may explain many disease phenomena and their therapeutics. But to hold fast to it and to it alone, to the exclusion of all the recent developments, and to bend it and twist it to explain

all and sundry diseases including traumatic manifestations, endocrinology, etc., is the limit. If today you discover adrenalin and acetylcholine as the sympathetic and para sympathetic effects respectively, the tri-dosha theory comes in to give an ingenious explanation. If to-morrow you discover prolan A and prolan B and correlate its effects on the ovary and corpus luteum, the tri-dosha theory comes in anyhow. It may be right but you have to prove it on your own platform. At least you must make an attempt to do it. If you seriously attempt it, you cannot but utilize the resources of modern science. You cannot live on the patriotism of your leaders alone for all time to come, if you ignore facts and truths. Any nation, which disregards truth, must come to grief eventually. If you cannot explain a thing or if I cannot explain a thing, it would be manly for both of us to say we do not know.

Greatness of Ayurveda

But having said all these against the unscientific attitude of the present-day Ayurvedic practitioners, I cannot but feel a thrill, when we recognise how great Ayurveda was, judged according to contemporary conditions, and in the historical perspective. I, perhaps, an unworthy descendant of the great medical men of yore, who were scientific enough, according to the conditions then prevailing, cannot but feel a genuine pride, when I read that even the science of Surgery, leaving alone medicine, was very advanced, and operations such as lithotomy, lithotrity, intracranial explorations, delivery of the child by forceps etc., were in vogue. These could not have been achieved by minds pinned to dogma and tradition. There must have been original minds, thinking minds and minds free from bigotry and authoritativeness. Toleration has been the watchword of all our great men of the past in every field of activity. The ancient Indian mind was always open for receiving truths from any source and that instinct is not dead even now. India has never been inactive in its researches on the properties of matter, mind and soul. The mightiest thinkers did differ from each other even fundamentally even as modern scientists and philosophers. If there had been implicit faith in authority handed to them by their forefathers, how are we to explain the three systems of Advaita, Visishtadvaita and Dwaita based

on the Gita and Upanisheds. Probably future scientists may agree to Sankara's conception of the identity of matter and energy even as the alpha and beta rays in radium emanation represent both matter and energy in contradistinction to the gamma rays which represent only energy. We must also take into consideration the fact that the Indian races have managed to live on till the twentieth century under the Indian systems of medicine, old as they are and imperfect as they are, judging from the present standards. We did not die as a race because of the want of the scientific system. Even serious illnesses and chronic illnesses have been cured under the Indian systems. Leaving apart the question, whether medicines alone cure diseases, the fact must be admitted that these cures are attributed to the Indian systems. Any amount of our pride on our scientific system, must occasionally meet with humiliation on a case getting ultimately cured by a so-called "quack". These everyday failures attributed to the scientific system, is not the fault of the system as such, but the narrowness and bigotry of the proud allopathic practitioner. So though the scientific system is broad-based, rationalistic, convincing and achieved notable triumphs, yet its imperfection must be acknowledged, if we have any regard for truth, and must learn to profit by its failures, and be ready to incorporate remedies and systems of treatment, from any source and I should say, even from an ignorant unlettered quack knowing only Kattumathirai (கட்டு மாத்திரை) and Vayu ennai (வாயு எண்ணெய்). When Jenner can take a hint from a milk-maid who asserted she could never get small-pox and utilized it to protect humanity against the scourge, when Owen Thomas and Sir Robert Jones could learn and utilize the tricks learnt from bone-setters and osteopaths, in the practice of orthopaedics, we must have no hesitation to learn anything from any source. Even the toad may have "a precious jewel in its head." After all Ayurveda and other systems of Indian medicine are as old as the Indian civilization, and we have to credit it with a vast amount of clinical experience, empirical use of drugs and a vast pharmacopœia specially the organic pharmacopœa. Even without a proper diagnosis in the modern sense, without morbid anatomy and histology without any of our ancilliary sciences to help us in even our trivials, that the ancient practitioner could achieve remarkable

systems and incorporate the fruits in the scientific system gradually and continuously. If we make a start now and work vigorously we can expect to assimilate everything of Ayurveda and the other systems in the course of 25 years with five five-year plans. After 25 years we can see the rejuvenated Ayurveda, the mighty Indian prodigy, of my dream. The thing must be done and finished before the existing practitioners of Indian medicine die out.

Who should do Research?

To do intensive work for these twenty-five or more years, we require specially qualified men who after the M.B.B.S. degree should devote 3 years of study in the Indian systems and must continuously practise in Hospitals where the Ayurvedic system only is in vogue. The present L.I.M's. are not sufficient. If the L.I.M. standard is raised to my expectations and the L.I.M. students, wherever they are trained, are made to appear for M.B.B.S. degree of the University, then they will do admirably. But money can be conserved and duplication prevented by abolishing the present L.I.M. course and enticing graduates to study Indian systems for three years with a view to do clinical research in hospitals dealing in Indian medicine only. Such hospitals need not concern themselves with surgery, midwifery, ophthalmology etc. Any cases requiring surgical aid must immediately be transferred to suitable other hospitals. So also other hospitals must send suitable cases to the Ayurvedic hospital.

Along with clinical research, in the same institution, there must be pharmacological research by whole-time workers. These workers must be highly trained and must be numerous to do intensive and extensive research. I should insist on the following qualifications for a pharmacological research worker in Ayurveda (1) B.Sc. or B.Sc. honours with chemistry and botany as indispensable subjects, (2) M.B.B.S. degree, (3) Two Years' Clinical study in an Ayurvedic hospital, (4) Three Years' training in a pharmacological research laboratory preferably in an institute where research in Ayurvedic drugs are going on. With such qualifications, and with a good number, with these qualifications, working in close collaboration with the clinical side of the Ayurvedic hospitals, surprising results can be achieved.

Indian Pharmacopoeia

Side by side an Indian Pharmacopoeia must be built. Every five years the pharmacopoeia must be revised and the results of the researches must be incorporated If within 25 or 50 years the Indian systems are exhausted, the Ayurvedic hospital will become automatically a modern hospital and the research section can go on doing research in Chinese medicine etc. So there will be only one system, an Indian system and those system wallahs ever emphasising on the differences, will be silenced for fever.

M.D. in Ayurveda

For those men who may work in the Ayurvedic hospital after the M.B.B.S. course, the University can introduce an M.D. in Ayurveda along with the other branches in medicine.

Conclusion

The plan suggested by me, or on some such lines, is the only solution for this pressing question. The present L.I.M. can never solve the problem. With the abolition of the L.M.P., at any rate in Madras, the L.I.M. will naturally become the outcaste, in the medical profession in the future. India, with its teeming millions, can afford to produce any number of highly trained medical men and women. Half standards and lowering of standard will be ruinous in the end. The authorities, who control our destinies, must look far ahead and must visualize the future possibilities of a step in the right direction.

Treatment of High Blood Pressure

BY

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BEFORE the subject of actual treatment is taken up a few general remarks may not be out of place. Causes of raised blood pressure are many and the treatment of every case cannot be exactly the same though the general principles would apply well to all of them. It has to be remembered that in a good number of cases the raised blood pressure is not secondary to diseases of the kidney and belongs to the so-called primary group. This group has been variously named by different authors; thus the terms hyperpiesia and essential hypertension refer to this group. The real underlying cause of most of the cases of this group is not well understood at present, though it is now believed that the cause is probably to be found in a pressure substance which may be infective, metabolic or endocrinic in origin. The cause being not known there can be nothing of the nature of a specific or radical treatment and the treatment of necessity has to be directed towards general lines. An important fact to be remembered is that whatever may be the cause of raised blood pressure it has a profound influence on the working of the different organs of the body. Thus the kidney is accustomed to working at a high pressure gradient. So it has been said that "patients with hyperpiesia of some standing present an optimum blood pressure at which the organs as a whole work most satisfactorily and any attempt to lower this drastically is a real disservice to the patient" (John Hay, 1936). Besides, there is no doubt that in many cases the raised blood pressure is a manifestation of nature's defensive reaction against some known or it may be as yet unknown cause. This conception is not a new one and we find in the writings of Oliver the statement, 'high arterial pressure is sometimes a necessary evil' (Halls Dally, 1934). So in a subject suffering from high blood pressure, if the pressure

is brought down suddenly without due regard for the functioning of the important organs of the body the result is a most unhappy one.

Another important fact that one should remember in this connection is that every extra pound of flesh beyond the optimum weight for a given hyperpietic means added load to the already embarrassed heart. Obesity which is only too often encountered in subjects of high blood pressure should therefore receive as much attention of the physician as any other recognisable underlying causative factor.

In a case of hypertension in the early stages the blood pressure is often liable and more readily amenable to treatment and so in this condition as in many other diseases an early diagnosis is of paramount importance for the successful treatment.

General Management

(*a*) *Desirability of disclosing the sphygmomanometric readings to the patient.*—When raised blood pressure is first detected, it is disputed whether the patient should be told about it. I think it all depends upon the temperament of the patient. If the patient is highly nervous it is not desirable to disclose to the patient at once that he has been suffering from high blood pressure but the physician has his responsibilities with regard to his patient; so in such cases the diagnosis should be disclosed to some responsible relative of the patient who may inform the patient about the high blood pressure gradually and cautiously.

In other cases the patient himself may be informed about the existence of high blood pressure and at the same time he should be assured that this should not frighten him. It should also be explained to him that his co-operation in the treatment with regard to following out details as much as practicable is of vital importance.

In this connection I may cite instances of 2 of my recent cases. The first, a demonstrator of the Presidency College consulted me complaining of various nervous and digestive disorders. When I brought the sphygmomanometer for taking his blood pressure the patient suddenly jumped out of the examination

table being extremely frightened and said out, "No, doctor, not that one. If you would now tell me that I am suffering from blood pressure I would simply drop down dead here." I assured him then with all the arguments at my command and told him that for all probability he was not suffering from high blood pressure and that only to complete the bed-side examination I wanted to take his blood pressure. Fortunately he was not suffering from high blood pressure but in case such a person is found to have raised blood pressure it would not certainly be advisable to tell him that then and there. The other, a lawyer of Calcutta consulted me for his high blood pressure informing me that he was compelled to change his physician twice in 3 months. He had to change his first physician when he discovered that he was injecting distilled water. This doctor had given out to the patient that he was suffering from some functional heart disease and that the injections would do him good but the name of the injection was kept a secret to the patient. The other physician had told him the correct diagnosis but the very day the patient discovered that the physician was not telling him the correct manometric reading he left him. So the patient requested me to give out to him the exact readings always. He has so far consulted me five times and I have always given him the correct readings. The patient has always been keen about the readings and has always kept a record of these. These two cases illustrate how the temperaments of patients differ.

(*b*) *Regulation of mode of living with regard to rest and exercise.* Rest ordinarily means physical rest. Rest is of considerable importance in the treatment of high blood pressure and the nature and amount of rest necessary for a particular case has to be decided on the merits of the case. Relative rest is a necessity for practically every case but if the pressure is very high and produces symptoms and if there is reason to believe that high pressure has lasted for a pretty long time without any real treatment and if there are evidences of myocardial weakness a preliminary period of rest in bed is desirable. If, however, rest in bed is recommended indiscriminately for every case, one would recognise too soon that many subjects who could have led a useful and relatively active life

for at least some more time is converted to neurotic invalidism. What is necessary for many a case is that he should enjoin greater rest in the intervals of his activities.

Mental rest, that is freedom from worries and anxieties is even more important than physical rest. To achieve mental rest detailed directions should be given taking into consideration the nature of work and the financial condition of the patient, so that he might adjust his activities and minimise the mental strain.

Some amount of exercise is helpful for many cases. The beneficial influence of moderate exercise in some patients is illustrated by a case reported by Gager (1930). A woman physician (aged 50) at the age of 46 had 150 mm. of Hg. systolic pressure, at 48 she had 210/110 and the pressure had since reached 230/130. 'Rest in bed had only a transient effect on the pressure and was decidedly boring to this lady of active habits. Deciding to give up bed and resume social activities she discovered that after relaxation of her dancing, her pressure fell to 150, lower than during the period of complete physical inactivity.'

(*c*) *Diet*.—So far as diet is concerned excess of meat and fish is to be avoided. The meals should be small and dry. There is no necessity of undue restriction of fluid; restriction of fluid, in fact, may be harmful in some cases. Whenever possible fluids should be taken in between and not with meals. Salt need not be unduly restricted for most cases of essential hypertension.

(*d*) *Removal of septic foci*.—Septic foci when discovered should be removed as these might have a possible bearing on the ætiology of the raised blood pressure in certain cases.

(*e*) *Cure of bowels*.—Constipation influences the blood pressure of hypertensive subjects possibly in 2 ways: firstly by a possible toxic absorption and secondly by acting on the nervous system reflexly. Bowels should therefore be kept regularly moving. Even if the patients think that the bowels are acting satisfactorily an opening dose of saline purgative once a week or oftener has a decided beneficial action on the blood pressure.

In some cases periodic enemata and colonic wash-out are desirable. Those who are habitually constipated should take some laxative, preferably a lubricant at bed time daily and a hydragogue purgative once or twice a week.

Drug Therapy

(*a*) *Iodine and iodides* Iodides are being used for high blood pressure for a long time indeed and have been used possibly much more commonly than any other drug. In the syphilitic group it is easy to understand its rationality but in the nonsyphilitic it is difficult to realise how it acts and, in fact, whether it has got any action at all or not on the blood pressure. Until recently potassium iodide was very commonly used. At present, however, there are many preparations of iodine in the market such as iobramag, iodocitrane, colloiode drops and the like which are reputed not to produce iodism and which are often being used in the place of potassium iodide.

(*b*) *Sedatives*—Sedative drugs lower the receptivity of the nervous system and are quite helpful in this disease as they allay anxiety and relieve many of the nervous symptoms such as insomnia, headache, pain in the nape of the neck etc. Even when the blood pressure is not lowered by sedatives, the symptoms are relieved; sodium bromide, ammonium bromide or Peacock's bromide may be given. Barbiturates in some form may also be conveniently used. I have obtained satisfactory results with Theominal (which contains theobromine and luminal) in many of my patients.

(*c*) *Vasodilators*—The nitrite group: All the members of this group have the property of dilating blood vessels and are potent agents for lowering blood pressure but their action is temporary. For reasons already stated indiscriminate use of nitrites is to be strongly deprecated. As a rule, in mild and early cases these drugs should not be used and they should be restricted for cases of some standing in which the blood pressure is very high. Their greatest usefulness lies in averting attacks when one is threatening due to sudden rise of pressure from any cause above the already existing levels. Amyl nitrite

(m iii) and glyceryl trinitrate (gr. 1 100) which cause rapid but short lasting lowering of pressure may be used for such cases. Sodium nitrate (gr. ½), Erythrol nitrate (gr. ¼) or mannitol nitrate (gr. ¼) may be used for other cases where more lasting action is desired. The action of mannitol nitrate lasts longer than most of the other members of the group. Other drugs: Veratrone may be used to reduce the blood pressure in eclampsia. The action of mistletoe is similar to that of nitrites. Preparations are now available in which various vasodilators or vasoregulators have been combined with sedatives. Jocapral (which contains promina), theobromine and iodocalcium triethanolamine) and Agatheosan (which contains phenylethyl barbituric acid, theobromine and nitroglycerine) belong to this group and may be tried in cases of very high blood pressure.

Organotherapy

Various organic extracts are reputed to possess general or selective vasodilator action. They have been frequently used in the treatment of high blood pressure sometimes with benefit. Padutin is a pancreatic extract of complicated structure and may be given to cases of high blood pressure showing evidences of angiospasm.

Lacarnol is another organic tissue extract and its indications are somewhat similar to that of padutin. Thyroid extract may be used with advantage in obese hypertensive subjects. It must not, however, be used for cases in which there may be the slightest suspicion of there being a thyrotoxic element in the causation of the raised blood pressure. Insulin, liver extract, testicular and ovarian extracts belong to this group and have been recommended by various workers but I have no personal experience with these drugs.

Preparations like hypotensyl (which contains mistletoe, liver and pancreas extracts), detensyl (which contains mistletoe, extracts of liver, pancreas and lungs) belong to this group.

Treatment of Symptoms and Complications

For the ordinary symptoms of the disease what has already been described would be sufficient. In some cases, however, some of the symptoms such as headache, precordial pain or

discomfort seem to be refractory to ordinary treatment and in such cases these symptoms should receive special attention. Intractable headache is often relieved by application of leeches to the temple and similarly precordial pain by application of leeches to the precordium. Lacarnol and euphyllin are often successful in relieving intractable precordial pain.

Amongst coaplications, important are heart failure, cerebral vascular accidents and renal failure often described as failure of the pump, failure of the tubing and failure of the filter respectively. Heart failure and cerebral hæmorrhage are much more commonly encountered than uraemia in cases of essential hypertension. Other important complications are acute œdema of the lungs and hypertensive encephalopathy. For hypertensive attacks, measures for lowering the blood pressure should be adapted and drugs which have the reputation of relieving angiospasm may be tried.

If these fail to relieve the symptoms, lumber puncture should be tried. For heart failure, acute œdema of lungs, apoplexy and uraemia, their appropriate treatment should be given. It may be mentioned in passing that in many cases of cardiac asthma occurring as a complication of hypertensive heart disease, morphine and its derivatives often act as a charm.

Miscellaneous Therapeutic Measures

Application of leeches has already been referred to. Sometimes venesection may prove to be of real value for relieving the symptoms of refractory cases for it gives direct relief to an embarrassed circulation by withdrawal of fluid: massage, diathermy ultraviolet rays, X'rays and various operative procedures are being tried in recent years for essential hypertension (Halls Dally, 1934; Fishberg, 1930). These have more academic than practical utility and as I have no personal experience with these measures I would not like to discuss their value. It may, however, be pointed out that the multiplicity of therapeutic measures that have been suggested and are being tried in recent years clearly indicate the unsatisfactory state of affairs still existing regarding the treatment of high blood pressure.

Analysis of Cases and Case Notes

In the course of last one year (20-5-37 to 20-5-38) amongst my private patients there were 23 cases of high blood pressure. The age of the patients varied from 21 to 65.

Only 2 were females and the rest were males. In 9 cases the cause of raised blood pressure was chronic nephritis and the remaining cases belonged to the non-nephritic group. In addition to giving general directions with regard to physical and mental rest, diet and care of the bowels, as a rule, I tried first iodides, bromides and sedatives in some form or other. If these failed to relieve the symptoms I tried vasodilators and other measures. I have tried to follow up all the 23 cases referred to above but I have at present no knowledge of 7 cases due to lack of co-operation on the part of the patients. To my knowledge only one case has died in the course of the year. This was a case of a young man of 21 suffering from nephritic hypertension. At the time of observation his blood pressure was 230 150. He preferred hospital treatment and died of heart failure at the Carmichael Medical College Hospitals. I have got up-to-date informations regarding the remaining 15 cases. In 2 of these the result of treatment has not been at all satisfactory. Both of these cases have got nephritic hypertension. In 5 cases though the blood pressure readings have not been much influenced, the symptoms have been relieved to some extent. In the remaining 8 cases the blood pressure readings and symptoms have both shown definite improvement. To speak in general terms, the result of treatment has not been at all satisfactory in the nephritic group and the best results have been obtained with purgatives and sedatives and not with vasodilators. In the above analysis of cases I have not included the hospital cases observed by me during the period as I could not follow them up over any length of time. Brief notes of only 3 of the other cases are given below:

Case No. 1.—Mr. K. K. S., Hindu male of 25, a sixth year medical student, consulted me on 30th August, 1937 complaining of thickening of arteries and visible pulsation of the brachials, the radials and other superficial arteries. He had no palpitation,

dyspnœa, precordial pain, headache or any other symptom. Had he not been a medical student, as there were no subjective symptoms, he would not have sought medical advice. Family history of high blood pressure or arteriosclerosis- negative. No past history of nephritis. On examination all the superficial arteries were found to be grossly thickened but not particularly tortuous and these were pulsating visibly. Aortic second sound slightly accentuated. Blood pressure 155/105. Fundus oculi – normal. Urine—Sp. gr. 1015. No abnormality detected. Teeth and tonsils healthy. By way of treatment he was given directions regarding his diet and he was advised saline purgative once a week. Theominal one tablet twice daily and iobramag one tablet three times a day. He carried on with this treatment for 4 months stopping theominal and iobramag from time to time. At the end of this period his blood pressure became 145/95. There being no symptoms he stopped all medicines at this stage. His present blood pressure is 150/95 without any symptoms as before and the arterial condition remaining same. The interest of this case lies in arteriosclerosis and high blood pressure occurring at an early age without any apparent family history and in treatment having but little effect on the blood pressure reading.

Case 2. Mr. H. M. M., Hindu male of 62, came under my treatment on 15th January, 1938, complaining of a bad type of headache, palpitation, intolerance for loud noises, constipation, flatulence and a sense of throbbing of the whole body. He was afraid of even getting out of his bed. That he had raised blood pressure was first detected 6 months previous to this. On examination – an obese old man with thickened arteries. Blood pressure 210/120. Heart enlarged (apex-beat in the sixth left intercostal space in the anterior axillary line). Fundus oculi—sclerosis of retinal arteries but no exudate or haemorrhage. Urine—Sp. gr. 1012, trace of albumen but no casts. Biochemical examination of blood not done. He was advised saline purgative 2 or 3 times a week. In addition he had a carminative mixture, theominal one tablet twice daily and Peacock's bromide half a drachm three times a day. He had rapid relief from all his symptoms and the blood pressure came down to 190/110 in about a fortnight's time. The effect was not however lasting

and in a month's time his blood pressure went up to the original level with reappearance of headache and palpitation. At this stage some modification of the treatment was made and Padutin (10 minims three times a day) was added. This however had no effect. Then Agatheosan was prescribed one tablet three times a day. In two weeks' time the blood pressure came down to 170/105 but he had a new complaint in the form of extreme weakness. As it was thought that the rapid and marked lowering of the blood pressure might be responsible for the feeling of weakness Agatheosan was stopped for a week. The blood pressure rose to 195/115 and the weakness lessened. Thereafter Agatheosan was again ordered one tablet on alternate days. The blood pressure then became 180/110. The patient was however still getting palpitation. 4 leeches were then applied over the precordium after which the palpitation disappeared. The blood pressure of the patient is now maintaining at 180-185/110. He now gets occasional headache and has no other complaints. He is walking about short distances but is not fit for any active work yet.

Case No. 3.—Mr. S. C. G., Hindu male, aged 32, consulted me on 28th May, 1937 complaining of insomnia, headache, occasional throbbing sensation within the head and peculiar attacks of one year's duration. The attacks occurred at varying intervals and usually followed physical or mental strain. The attacks, as described by the patient, were characterised by flashes of light before the eyes, giddiness and severe headache soon to be followed by a state of confusion compelling the patient to take to bed. Only on one occasion the patient lost consciousness. The duration of the attacks varied from a few minutes to several hours. He never had any aura. Vomiting, convulsion or tongue biting never occurred. He sought medical advice at the time of the very first attack and he was told by his medical attendant that he was suffering from high blood pressure and that the pressure at that time was 175/115. Since that time he had taken various medicines including nitrites but without any appreciable benefit. On examination—he was an obese young man of short stature. Height—5 ft. 2 inches; weight—11 stone 2 pounds (normal for the height—8 st. 7 lbs.). Blood pressure 160/110. No significant abnormality in other

systems. There was no history of past nephritis. Urine—Sp. gr. 1018. No albumen or casts. I thought that his attacks were due to temporary rises in blood pressure above the already existing high levels and were of the nature of hypertensive attacks. I prescribed for him a low caloric diet, a day of fast once in the week, walking a distance of 2 miles every morning, Agarol every night and a saline purgative once a week. In addition he had Padutin, Theominal and potassium iodide. There was distinct improvement in his symptoms in about a fortnight's time. He carried on with this treatment for 4 months stopping Theominal from time to time and using Peacock's bromide instead at those times. At the end of this period his weight came down to 10 st. 3 lbs. and his blood pressure was 135/95, he had no symptoms and there had been no attacks.

At this stage all the medicines were stopped one after another and he was asked to carry on with the general directions with regard to diet and care of bowels and was asked to report from time to time. Now full one year has passed, his blood pressure is maintaining at 135/95 and he has been keeping free from symptoms having had no attacks during this time.

In conclusion, I would like to emphasise that while treating a case of high blood pressure, the aim of the physician should be to treat the patient as a whole and not his blood pressure alone.

(Read at a clinical meeting of the Calcutta Branch of the Indian Medical Association on 16th June, 1938.)

Editorial

Castes in Medicine

THANKS to the efforts and vision of Sir Frank Connor, I.M.S. and our Health Minister a caste will soon have disappeared from the ranks of Medical Men in this Presidency. That the measure is a revolution and will bring no little self respect and satisfaction, there is no doubt.

We have however growing up among us another big division that need scarce exist; and which, in years, is going to give us a deal of trouble.

Whatever branch of Allopathic or Indigenous system of medicine one practises, we practise the art of healing, and anything that helps to improve this art should be included and grouped under one head and from one comprehensive study.

The East gave the West its civilization. It was an evolutionary necessity that the earlier civilization had to give of its best surviving influences.

The art of healing was one of these. The Indian system of medicine was one of our earliest systems. It was the genesis of the Allopathic or Western system. It crept into the West through early travellers and explorers.

With the decline of Eastern progress, came the decline in the progress of the art of healing; while the legacy it gave to the West continued to grow and be nurtured. Its study based on experience, scientific observation and research has to-day given us what one should call scientific medicine, for this form of medicine has progressed with the rest of the progress around us. It has survived the test of time; it has been adopted by three quarters of the world's population and by 99% of its intelligentsia.

On the other hand, what of Eastern forms of medicine? They have remained at a standstill. To-day they are nothing but a list of secret remedies handed down from father to son.

The principles enunciated hundreds of years ago and based on the reasoning and research of what in the scale of progress was a comparatively dark period, cannot survive the scientific reasonings of to-day. It stands apart, a necessary evil in our slowly progressing country; for it is the answer to the ignorant and lowly paid peasant and labourer; and bread to the quack and pseudo-scientific healer.

To-day sentiment, nationalism, antipathy to things 'foreign' are going to perpetuate a system that is but a parody on the scientific system that was born of, and developed from this very system. In the course of development, the scientific system has cast aside what could not be reconciled with reason and research.

To foster the study of Indian Medicine as such, is to foster this parody. It is the setting up of a parallel system.

It was the lack of anatomical and physiological study as we know it to-day and which we accept as the basis of correct medicine and surgery, that was responsible for the casting aside of earlier tenets of the art of healing and its evolution. There seem no grounds to justify this going back; not even the cry of the rural areas; not even economy. Is it correct one may ask to give the poorer and those who need it most this second-rate help; and reserve the better aid for those less in need of it?

How can one reconcile this vestige of 'dark' days with our present-day advance in politics, government and industry?

Early Medicine or Eastern Medicine gave birth to an improved and developed Western Medicine. In giving birth it died; for it lacked the sustaining support of anatomical and physiological truths; it was the child of a naturally limited reasoning and limited observation.

Are we to-day going to perpetuate this effete system by trying to give it back these truths it lacked, at the same time maintaining principles that are contrary to them?

What is the teaching of Indian Medicine to-day? A questionable standard of general education with an indifferent

and elementary knowledge of anatomy and physiology, is grafted to hospital practice which is a return to the ancient rule of thumb therapeutics; and a contradiction of the earlier training.

If there was any good in 'Indian Medicine' it has been absorbed in progressive medicine of the West or rather the modern medicine of 80 of the earth. What may be conceded is that a few remedies remain to be tried out and proved. This certainly does not call for setting up of a parallel system which has only sentiment and antipathy to things foreign to support it.

Rural needs, I mention apart. To-day the rural medical problem is not the outcome of unemployment; but the misemployment of doctors and lack of modern medical aids in the villages. Indian systems of medicine continue to flourish here only because the poor and the superstitious abound in these areas; and it is an easy field for the quack.

If there is good in Indian Medicine let it be worked out and proved through investigation in scientific laboratories and in the wards of modern hospitals. Let those who hold these hoary secrets but have neither the training nor the opportunity to prove their value, work with the more advanced scientific medical men; and contribute jointly to advance in healing.

The bastard of bias and sentiment can never know truth and progress.

Only scientific truths can advance the progress of the correct art of healing.

The Trichinopoly District Medical Association
TRICHINOPOLY

Statement of account for the month of October 1938

RECEIPTS				PAYMENTS			
	Rs.	A.	P.		Rs.	A.	P.
Cash Balance ...	49	9	3	Clerk's salary ...	18	0	0
Bank Balance ...	268	5	2	Rent ...	10	0	0
Advt. Charges ..	48	3	0	Postage ...	9	11	0
Members Subscription.	56	0	0	Sweeping Charges ...	0	4	0
				Stationery ...	2	6	0
				Cycle repairs ...	1	0	0
				Baksheesh to Peon ...	1	0	0
				Advt. Charges ...	4	6	0
				Printing ...	1	4	0
				St. Joseph's Industrial School Press for printing of Miscellany ...	100	0	0
				Bank Balance at the end of the month ...	132	13	2
				Cash Balance ...	141	5	3
TOTAL ...	422	1	5		422	1	5

1st October 1938.

M. C. CONDILLAC,
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