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The Antiseptic

A Monthly Medical Journal.

Dr. U. Rama Rau, M.L.C. & U. Krishna Rau, M.B., B.S.

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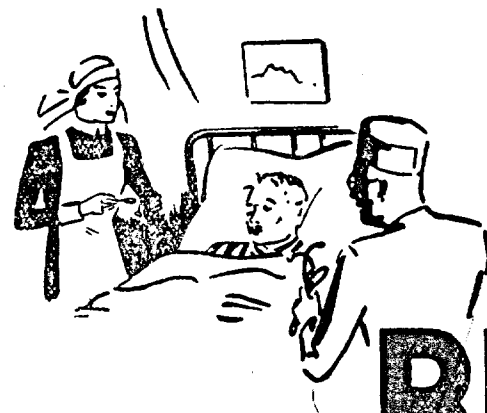
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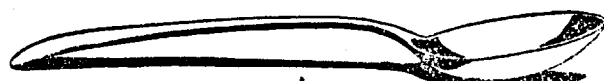


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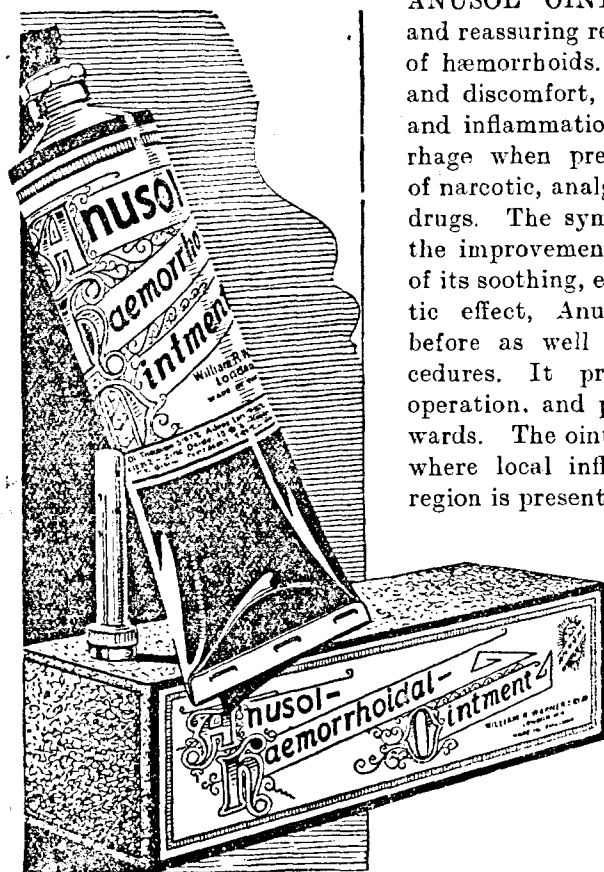
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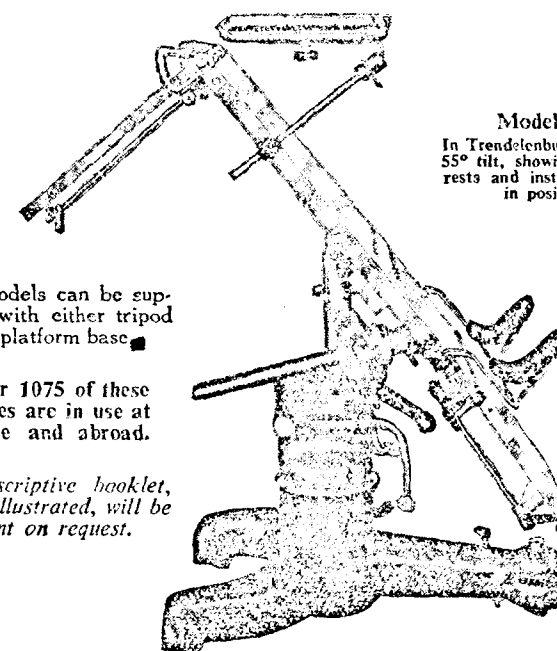
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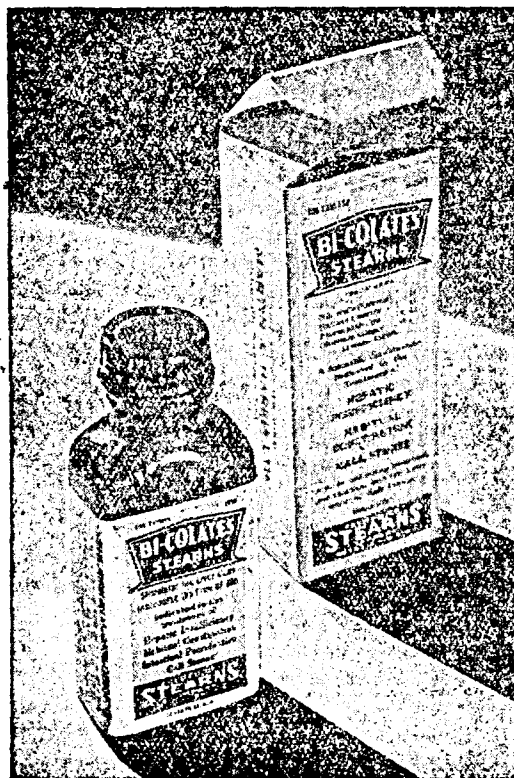
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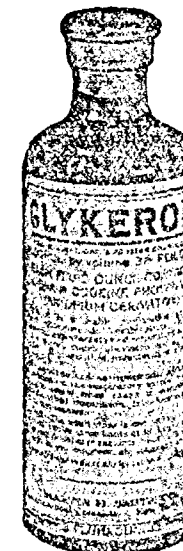
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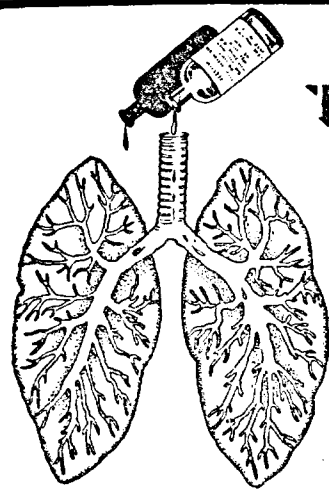
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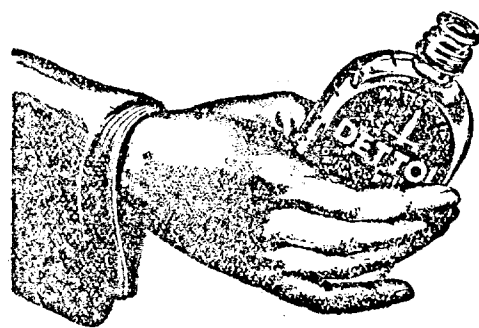


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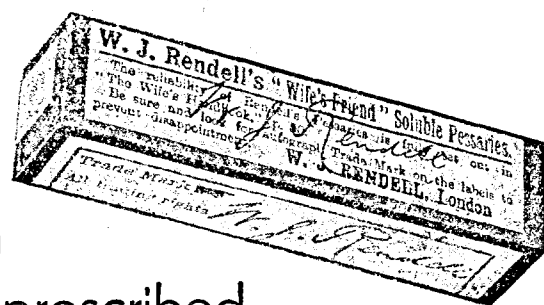
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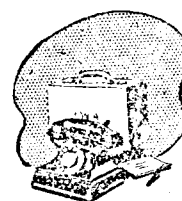
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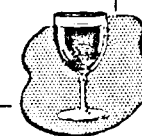
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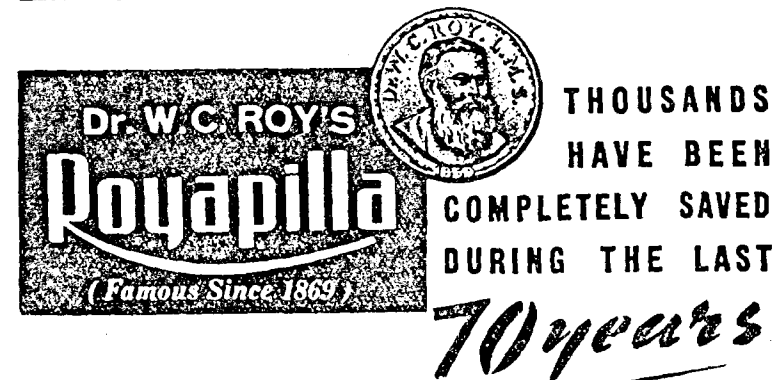
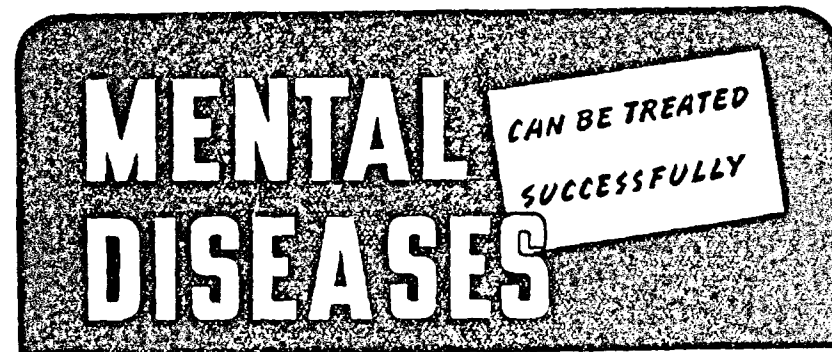
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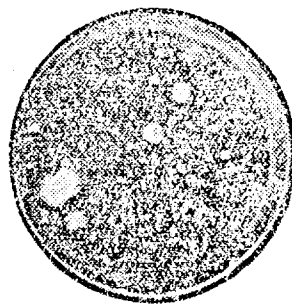
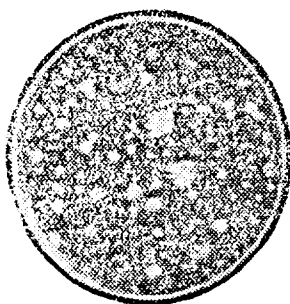
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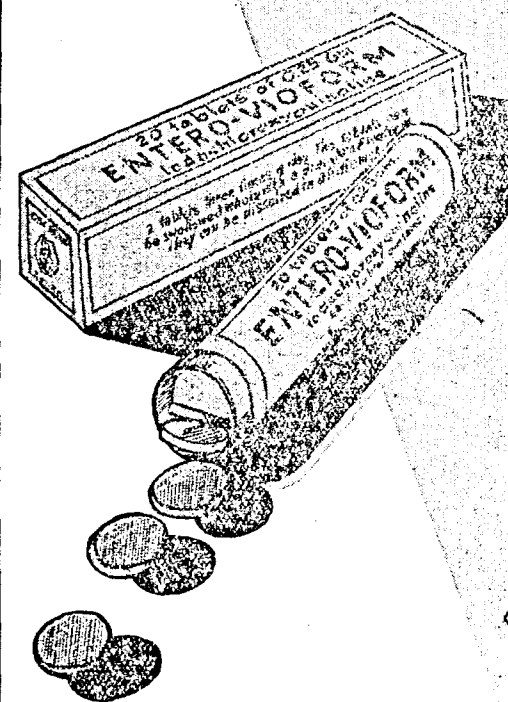
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Original Articles

Special Features of Diabetes in India*

BY

RAI BAHADUR DR. SHIAM MANOHAR LAL,

Lucknow.

IN a vast country such as India, peopled by races widely differing from one another in respect of diet, customs and climate, it is natural to expect that a metabolic disease like diabetes would present great variations. Bengal, where there is abundant rice and where Nature, without much effort, produces rich harvests, where people are more intellectual and less hard-working, is heavily afflicted with diabetes. In the Punjab, where they must work hard to produce wheat, where martial races abound, and where people are more industrious and hard-working, diabetes is comparatively rare. The Gurkha, though eating more rice than the people of the plains, does not suffer so much as the Bengali, being more hard-working. Mehommedans, who mostly subsist on a mixed diet, are not so frequently affected as the vegetarian Hindus, particularly the *Banias*, who are as a rule better off, accustomed to eating rich food and whose occupation of trade and commerce does not involve much physical exercise, though the business frequently gives them worry—a patent cause of chronic sympathetic irritation, leading to hyper-adrenalinæmia. The would-be diabetic is frequently a man of adrenal preponderance, which marks him off as a man of outstanding ability, be he a businessman, a lawyer, a judge or a banker. Favoured by luck and egged on by ambition, he does not allow his sympathetic and vegetative nervous system enough of rest. Perhaps, at the age of thirty, he has got all that others achieve at forty. By dint of sheer hard work—involving not much physical exertion—he has assured his future. As his business or profession flourishes, he has less time for regular exercise and amusements. Even walk is curtailed and an electric lift and motor cars save him even the usual exertion. Yet, he continues to eat, perhaps more than before, aided with the culinary art. In many cases, this excess of innate exuberance of energy finds an outlet in the worship at the altar of Venus. Excessive indulgence to calm and quell the *carnic concupiscence*,

* Specially contributed to 'The Antiseptic'

taxes the testicular secretion and inhibits the combustion of fats. Should this votary of Vulcan by day and Venus at night, become a worshipper of Bacchus too, the candle of life begins to burn fast at both ends. Excess of sugar in blood is deposited for sometime as fat under the influence of the pituitary. The fatty heart and muscles become less contractile, exertion produces breathlessness and early exhaustion. Lack of exercise causes muscles to become flabby. Now it may be, he falls ill with some minor malady, from which he takes a long time to recover, or he gets "boils" or even a carbuncle. It is usually at this stage that the Indian is diagnosed to be suffering from diabetes, for the first time.

It was for this reason that in India, in ancient times, every rich man was enjoined to be charitable. There is nothing like charity, good will and sympathy towards the unfortunate fellow-human beings, which soothes the nerves. Worship of God, meditation, entertaining of noble ideas, study of religious scriptures, pilgrimage, fasting and other austerities, serve to remove worry and anxiety and do away with jealousy, evil designs and base motives. The soothing of sympathetic ganglia, meagre food and seclusion from the scene of "worldly objects", restore the nervous equilibrium. Blood pressure, which invariably rises in such cases, falls; and the heart which has been quickened finds relief. Fussiness and hurry disappear. Sensual indulgence is diminished. Control of diet—an integral part of Self-Control—is the most important measure.

Here again, medical men sometimes make mistakes. It has to be remembered that in diabetes the inability to glycogenize sugar is not the only, nor, for the matter of that, the chief, defect. The most dangerous defect is the defective fat-metabolism. Diacetic acid, Acetone etc., are formed out of fats and the usual advice to cut down carbohydrates and supply the necessary calories by fats is pernicious.

In India, we seldom meet with true or malignant pancreatic diabetes in which the pancreas is organically defective. In most cases, it has been over-worked or the liver has become unduly loaded with fats, leaving little space for the glycogen to be stored. The pituitary factor is by far the most dominating factor. In most cases, the hyperactive pituitary and adrenals depress the pancreatic function. Hurried food does not give sufficient time for entero-kinase to reach the pancreas. In the majority of these cases, therefore, Insulin has not much effect as the glucose equivalent of Insulin-units is low. (On the contrary, a small dose of Insulin would markedly influence the blood and urine-sugar in a true diabetic). In these cases, it is the fat which is the harmful article of diet. Eating of much fats produces a craving for sugars and sweets. Cutting down of fats in diet produces a contrary effect. In planning the dietetic treatment, it is the fats—and not starches and sugars—which have to be greatly eliminated or entirely cut-down. It will be seen—surprising as it may appear—that in many cases the toleration for carbohydrates would greatly increase. Of course, this does not mean that the carbohydrates have not to be restricted. In most cases, they should not exceed 200 grammes in 24 hours, divided equally into 4 meals. Food should be rich in vitamins, particularly in vitamin B, which has an indirect influence on sugar metabolism. The carbohydrates should not be in a concentrated form. Too much of concentrated food is not capable of being properly disposed of. The pancreas cannot secrete sufficient Insulin *rapidly* enough, but, if given adequate time, it will meet the necessary requirement provided the advent

of glucose in blood is not too rapid. Foods containing 5% carbohydrates can be eaten with impunity; and even those containing upto 10% can be freely permitted. In a diluted form, mixed with much cellulose, which hinders rapid absorption, carbohydrates can be given in larger quantities.

Proteids should not exceed $\frac{1}{1000}$ of body weight per diem; if given more they may be not properly combusted and give rise to gout. If used as a source of energy both proteids and fats are converted partly into glucose; and during their inefficient combustion, ketonic bodies are frequently formed.

The use of Insulin is liable to much abuse. Its indications are definite. It is a powerful drug and must be used with caution and care. The indiscriminate use in all cases, in which sugar is detected in urine, is to be highly condemned. Blood sugar curve should be determined in all cases invariably before the use of Insulin is started. Urine should be daily examined for control. There are four definite ways in which Insulin may be used in different cases.

1. In all those cases in whom sufficient caloric value of food cannot be metabolised, Insulin has to be used, perhaps for a long time and in a few cases for the rest of life. Without Insulin such patients cannot live; they will continue to waste till they die from sheer exhaustion.

2. As a cure, it is used in severe cases. First of all, the basic metabolism is determined and the amount of different food stuffs found out which can be given without producing glycosuria. Then it is found out how much Insulin would metabolize a given quantity of excess of sugar in blood. Then the dose of Insulin is determined, which will cause sugar just to appear in the urine after 2-hours of meal. The quantity of carbohydrates should be given is just sufficient to make the total caloric value equal to 1800. Insulin is given in two divided doses. After a week or so, one day Insulin is stopped and the total quantity of sugar excreted in the urine found out. This quantity is divided by the units of Insulin given to render the patient's urine sugar-free. This ratio is called the glucose equivalent. Then Insulin is continued for some days more and very gradually diminished. If sugar re-appears in the urine, the dose of Insulin may be again increased. After a few trials, it will be found that less Insulin is necessary. The tolerance for Insulin diminishes after prolonged use and smaller doses of Insulin metabolise larger quantities of sugar. This line of treatment requires frequent blood tests and continuous urine tests. Patience is usually rewarded with success. Ultimately, after a few months, Insulin is by stages completely withdrawn. The patient's weight must not be allowed to increase. If in excess, it should be brought down to the normal by gradual starvation and not by continuous fasting in which there is a distant danger of producing ketosis.

Of course, a weekly fast does not do much harm provided the urine does not show traces of ketones and acidosis. Otherwise, instead of a complete fast, a diet consisting of fats—eggs and green vegetables—may be substituted. On those days, the patient should not take any Insulin.

3. In cases of emergency e.g. coma, cellulitis, carbuncle, gangrene, optic neuritis etc., Insulin has to be used plentifully combined with plenty of carbohydrates. In severe cases, it has to be injected intravenously with glucose. It is not realised as a rule that given intravenously

or rectally, glucose is not excreted in the urine, because it does not pass through the liver. The explanation is complicated and would occupy much space. In all diabetics with the advent of sepsis, glucose equivalent rises. Even ordinary diabetics in whom a moderate amount of Insulin was enough, would become Insulin-resistant and the quantity of Insulin will have to be increased. In all cases of diabetes suffering from acute illness, where food is restricted, and where there is, in consequence, a great danger of coma, Insulin and glucose must be administered even though the diabetic had not needed any before the onset of the acute illness.

4. *As an anabolic agent*:—In all cases of wasting, particularly in old people and in diabetic tuberculosis, in heart disease, in intrinsic hypertension of pulse due to sympathetic continuous irritation etc.

Insulin has its limitations as it is not the only internal secretion of the pancreas. It is believed that the interstitial tissues of the pancreas have another secretion (just as in the case of the testicle), which is necessary to maintain life. In depancreatized dogs, life cannot be maintained with Insulin; but if a portion of the pancreas is left, life can be prolonged with Insulin. This proves the existence of another pancreatic internal secretion besides Insulin.

There are many complicated experiments which throw a good deal of light on the sugar economy of the diabetic. If a healthy person is injected with Insulin and his blood transfused (with necessary precautions) into a diabetic, the result would be different in Insulin-sensitive and Insulin-resistant cases. In the former, the blood sugar would fall; in the latter, the blood sugar would be unaffected. In these, there is obviously an antibody (hitherto unknown) present. The presence of this antibody may be demonstrated in suitable cases by dropping Adrenalin into one of the patient's eyes; in the healthy, there is no effect, in the diabetic, madarosis is produced.

Further, connecting the adrenal vein of one dog with the jugular vein of another dog, and then injecting Insulin in the first dog, would produce hyperglycemia in the second. If the pancreatoco-duodenal vein of one of them is connected with the jugular vein of the other, the injection of Adrenalin in the first dog, would lead to hyperglycemia in the second. (FALTA). The increase in blood sugar excites normally the parasympathetic centres, leading to hyperinsulinization; diminution in blood sugar stimulates the sympathetic leading to adrenalization of blood. In this way, the two opposing forces keep the blood sugar in a normal condition of equilibrium.

In the diabetic, this balance is disturbed. Lecithin of brain too has also a glycolytic effect on the hepatic cells.

Diabetes, therefore, is not a disease of mere deficient oxidation of blood-sugar; there is an erratic stimulation of sympathetic ganglia and unportuous glycolysis in the hepatic cells. On this consideration is founded the treatment of diabetes by the injection of graduated doses of a suitable albumin.

In India, true pancreatic diabetes is rare and juvenile diabetes almost unknown.

* Manohar Nivas',
Aishbagh, Lucknow.

Diagnosis and Treatment of Amœbiasis*

BY

U. P. GANGULY, M.B. (CAL.)

Senior House Physician, Medical College Hospitals, Calcutta.

THE history of the development of the treatment of amœbiasis is an interesting study. Ever since the discovery of *Entamoeba histolytica* by LOSCH¹ various workers like COUNCILMAN and LAFLEUR² SCHAUDINN³ KOVACS⁴, and others, continued to work on the subject and differentiated various amoebae infesting intestinal canals of human beings into parasitic and coprozoic ones, and the pathogenicity of *Ent. histolytica* was duly recognised. KARTULIS⁵ (1887) found amoeba in the pus of liver abscess, confirming KOCH's⁶ observation in post mortem examinations of five cases of dysentery, of which two were complicated with liver abscess. OSLER⁷ also confirmed this in 1890. The discovery of the cysts of *Ent. histolytica* was made by QUINCKE and ROOS⁸ and rediscovered by Huber, who filled up the gap of our knowledge about it. WALKER⁹ gave the finishing touch to our knowledge about the pathogenicity of the *Ent. histolytica* and the cysts, although the details of infection—life history, staining reactions, culture and such others—were worked out by subsequent workers. ACTON¹⁰ laid emphasis on the finding of Charcot Leyden crystals in the faeces as diagnostic of the infection.

Regarding treatment, the value of Ipecacuanha was known in Brazil since mediæval ages. In 1864, PISO¹¹ introduced it in Europe as a secret remedy for dysentery. In 1817, PELLETIER¹² isolated Emetine which was first used in dysentery in 1829. In 1894, PAUL and COWNLEY isolated two alkaloids, Emetine and Cephaline. After *Ent. histolytica* was discovered as being one of the causes of tropical dysentery, VEDDAR¹³ found that Emetine kills *Ent. histolytica* in dilution of 1 in 100,000. CHOPRA¹⁴ found that in alkali medium, Emetine kills it in 1 in 200,000 dilution. The remarkable action of Emetine in relieving the symptoms of dysentery and presupplicative hepatitis led to its misuse. Toxic actions of Emetine, especially on the heart, began to be reported from all over the world and CHOPRA¹⁴ found that it produces a permanent damage to the heart even in therapeutic doses. CAPT. GANGULI¹⁵ issued a note of warning not only regarding its toxicity, but also its failure to cure nearly 80% of cases, unless the ordinary therapeutic doses *viz.*, six to eight injections of one grain of Emetine are followed by three grains of Bismuth Emetine Iodide for fifteen days. Bismuth Emetine Iodide fell into disuse on account of its extremely nauseating properties and on account of its toxicity. Besides, even if the patients are able to retain the drug, which few are able to do, at least twenty per cent of such cases get relapse even after several courses. Hence the necessity for a less toxic and more efficient drug was keenly felt. MANSON-BAHR and SAYER¹⁶ were of the same opinion. Various less irritating preparations were used, such as, Emetine periodide (E. P.I.), Aur-Emetine, Emetol, Paremitol, Gavano and such others were tried, but none of them was satisfactory.

In India, Kurchi (*holarrhena antidysenterica*) was used as treatment of dysentery by Indian medical practitioners, from pre-historic times. HAINES isolated an alkaloid from it, which he named *Conessine*. It was found that injection of this alkaloid in the same doses (*viz.*, one grain for

* Specially contributed to 'The Antiseptic'.

six to eight days) was as much efficacious as Emetine, without manifestations of any toxic effect noted in the latter. This drug has fallen into disuse on account of its extremely irritating action after injection. Similarly, Kurchi Bismuth Iodide fell into disuse on account of want of uniformity of extraction of the alkaloid from different barks. In any case, it had none of the disadvantages of Emetine Bismuth Iodide, but a more efficacious drug was called for.

A revolution in the treatment of amœbic dysentery was ushered in with the advent of organic arsenical preparations. In 1909, EHRLICH first prepared *Carbarsone*, which was used extensively in both acute and chronic forms of amœbic dysentery. Even experimentally infected kitten, which invariably died in spite of exhibition of Emetine, were saved by *Carbarsone*. It is however contra-indicated, when kidney and liver diseases are present in an amœbic case. All arsenical preparations should be used with extreme caution regarding the optic nerve. Besides, it has been found that the course has to be repeated in several cases, and yet nearly 25% showed relapse.

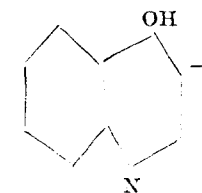
It must be understood, that it is impossible to kill all amœbæ in the system by any chemotherapeutic drug. It is proved that the ultimate destruction of all protozoal infection must be done by development of tissue resistance and antibodies in the system. Hence, a long course of tonic treatment is necessary in amoebiasis, malaria and other protozoal infections. In extensive intestinal ulcers the vitality of the body is lowered especially through the action of toxins of secondary infection of *B. coli*, Strepto- and Staphylo-cocci and such others. Long course of medicines is necessary to heal up the ulcers and to cure the original and secondary infections.

It has been suggested by WESTPHAL,¹⁷ that normally *Ent. histolytica* exists as a harmless amoeba in the lumen of the gut (*minute form*) and the extent to which it may become pathogenic, depends on the tissue resistance of the host, which may be broken down by bacteria. In the tropics, conditions favour the reproduction of *Ent. histolytica* and virulent stains thereafter issue. SWARTZWELDER¹⁸ showed that simple enteritis, by a strain of *B. coli* or some other organisms may be a predisposing factor causing increase in the virulence of *Ent. histolytica*. One has also to consider that mixed infection of both bacillary and amoebic and other protozoal organisms are very common in the tropics, and one infection helps to keep up the other. Similarly, DESCHIENS and DECOURT¹⁹ found that toxic enteritis caused by Croton oil may flare up amoebic dysentery in some carriers. It has been our experience to find frequent flaring up of dormant amoebæ even after a dose of castor oil. It is therefore necessary to be careful about these secondary infections and to keep the intestines free from pathogenic organisms by prolonged exhibition of such a drug which has destructive action on amoebæ as well as on secondary organisms. This drug should not have any deleterious action on heart, kidneys, liver, optic nerve and such others, even on prolonged use.

Fortunately, a preparation containing Iodine and Quinoline was first introduced by Behring and Co., which they named *Yatren* 105. GORDON²⁰, MANSON-BAHR²¹, YØRKE and ADAMS²², TURNER and JONES²³ all spoke highly of this drug in the treatment of amœbic dysentery. Various other workers, including MEGAW, ACTON, KNOWLES and CHOPRA of Calcutta

School of Tropical Medicine, gave trial of this drug and they found that this drug was very efficacious, but doses above 15 grains were liable to produce severe diarrhœa. Although KNOWLES pointed out that diarrhœa was essential for success in the treatment, the author has personal experience of several patients who were so frightened by the onset of diarrhœa, that it was impossible to induce them to continue the treatment. Besides, diarrhœa-producing drugs are unsuited for ambulatory treatment, and working patients do not like to be confined to bed during treatment. However, it has been found out that it has got an antiseptic action regarding secondary organisms infecting the ulcerated mucous membrane of the intestine and also a deleterious action against amœba. Had it not been for its disadvantage mentioned above, it would have been an ideal drug for after treatment of amœbic dysentery and for treatment of chronic cases and carriers, although it has been mentioned by CHOPRA¹⁴ that "it may not reach all the lesions of the bowels in sufficient concentrations".

Various other preparations of Iodine and Quinolic acid have been put into the market, of which two at least deserve special mention. One is *Enterovioform* and the other is *Enterozol*. The former is an Iodine derivative of Quinoline in which the chlorine group replaces the sulphonic group of Yatren, which causes diarrhœa. Enterozol is a simpler preparation of 4-Oxy-3 Iodo-Quinoline and the structural formula is as follows:—



The author has used both these preparations and obtained equally good results and without prejudice to either, he prefers Enterozol as cheaper of the two, although a few cases had idiosyncratic iodism after its use. A few case notes have been mentioned below, which uniformly proved the efficacy of the drug.

Method of investigation.—In the School of Tropical Medicine, Calcutta, no case has been declared cured unless six consecutive examinations of schools gave negative results. The author had to be content with three or four examinations as the patients refused to have more examinations done. Besides, extreme care was taken for diagnosis and differential diagnosis, so that no case of *Ent. coli* was misdiagnosed as *Ent. histolytica*. The diagnosis is not so easy as is commonly supposed. The slides were prepared, one containing emulsion of mucus and stool with normal saline, and the other with Lugol's solution. The former is intended for diagnosing vegetative entamœbæ and chromatoid bodies in cysts, and the latter is more suited for diagnosing cysts and number of nuclei. With normal saline, the chief characteristics of vegetative *Ent. histolytica* are: (1) clear hyaline pseudopodia thrown out during movement, and (2) ingested red blood cells. *Ent. histolytica* never ingests bacteria yeasts, fungi or similar food particles, like *Ent. coli*, unless it is dying when they are parasitised by bacteria, fungi and yeasts. It must be noted, however, that these are not the sole criteria of differential diagnosis of *Ent. histolytica* from *Ent. coli*.

PAVLOVA²⁴ found that locomotion, and formation of ectoplasmic pseudopodia could not be regarded as diagnostic. In cases of both the amœbæ, greatest activity was manifested at pH 6.5, the rate of locomotion being higher in body temperature, than in room temperature. In a fresh condition, neither of them does show any sharp differentiation between ectoplasm and endoplasm, and locomotion in both takes place *without formation of hyaline pseudopodia*, as is said to be the characteristic of *Ent. histolytica*. It is only when the *Ent. histolytica* becomes devitalised that it begins to throw out clear hyaline bigger pseudopodia. Whenever living amœba gets out of the gut wall, it begins to die. A dying amœba becomes sluggish in its motility and throwing out of pseudopodia becomes less frequent and the pseudopodia of both now becomes big and massive but only contains hyaline ectoplasm. The next differential point is the ingestion of red blood cells, and according to KNOWLES²⁵ "any entamœba containing included red blood cells is *ipso facto* *E. histolytica*". But even ingestion of blood cannot be sole characteristic of *E. histolytica*, as TYZZER and GEIMAN²⁶ found that in a case of intestinal obstruction, *E. coli* was actively ingesting red blood cells in the intestine.

In the precystic stage, when all red-blood cells are excluded and the amœba ceases to throw out pseudopodia, it is impossible to differentiate two kinds of amœba, unless staining methods are applied, when they are differentiated by characteristic position of their nucleoli.

In the cystic stage, also, the early formed cysts contain two nuclei, like those of *E. coli*, in which the characteristic glycogen vacuole is not always seen even in the latter. The presence of chromatoid bodies characteristic of *E. histolytica*, cannot be always relied, as a few *coli* cysts also show them, but not in the form of bars. The number of nuclei only can be relied on, as 53% of cysts of *E. histolytica* show fully developed 4-nuclear spherical bodies of small size, compared with large 8-nuclear adult cysts of *E. coli*.

The presence of Charcot Leyden crystals was always regarded as suspicious, for chemically they are the same as ethylimine, which are products of tissue digestion as happens in the asthma sputum through autolysis. But we have found rhomboidal crystals to be the characteristic of chronic amœbiasis, and not the long acinary types of crystals, found in stools. A finding of lozenge-shaped Charcot Leyden crystals is quite enough to indicate treatment with Enterozol.

Regarding other methods of diagnosis, in suspected cases of amœbiasis, especially chronic ones, sigmoidoscopy is not of much help in large proportion of cases, as the amœbic ulcers are more commonly found in ascending and transverse colon, unlike those of bacillary dysentery. But where ulcers are found in descending colon or sigmoid flexure, a swab taken from ulcerated part will usually show actively moving amœbæ in amœbic cases. Complement fixation test has been recommended, but PAULSON and ANDREWS²⁷ found that it is unreliable. BONSIGNORE²⁸ recommends "Histidine cutaneous test" in chronic cases, in which 1 c.c. of a 10% solution of Histidine is injected into the cutis. The immediate reaction is a local pallor of half an inch diameter, followed in one or two minutes by hyperæmia over the area lasting for 40 to 45 minutes. Failure of development of hyperæmia within 5 minutes indicates absence of amœbic infection.

The author has given trial to ten chronic cases in which *E. histolytica*

cysts were demonstrated, the reaction was positive in all. In ten cases of suspected amœbiasis, whose stools repeatedly gave negative results, Histidine reaction gave positive results in three. All these were treated with Enterozol and every one of them responded to this treatment. Only one was refractory after two courses of treatment, but with an exhibition of ultraviolet rays over the abdomen, the symptoms disappeared after a third course of Enterozol.

In conclusion, although we insisted on getting fresh stools from patients, it was not always possible to get them. It is not possible to diagnose dead amœba with certainty even in acute cases. In chronic cases, repeated examinations of stools were sometimes necessary, as in spite of repeated negative findings, a number of cysts are showered perhaps in the next stool, where even three or four cysts could be discovered in each microscopic field. The Histidine reaction gave us a very valuable clue to diagnosis, as we were almost certain to get it after persevering efforts.

Experiences with Enterozol.—Enterozol has been prepared in the laboratory of Union Drug Co. Ltd., and as has already been noted, is an Iodoquinoline preparation with 48% Iodine content. It is an amorphous powder, odourless and yellowish grey in colour. It is insoluble in water and ether and sparingly soluble in Acetic acid and moderately soluble in absolute Alcohol.

This preparation has been given trial in both acute and chronic cases of amœbic dysentery, and a very large number of other bowel troubles including colitis and chronic bacillary dysentery. It has been found to be generally efficacious in all varieties of such diseases, but in this article, we are chiefly concerned with amœbiasis.

Case notes.—Enterozol was tried in three acute cases of amœbic dysentery, twentyone chronic cases and three latent cases, in which Histidine test was positive, but no cysts or Charcot Leyden crystals were detected in the stools in spite of repeated examinations.

Acute amœbic dysentery.—*Case No. 1* :—S.K.S., aged 20, gave history of passing blood and mucus in his stools for the past three days, with gripping pain in the abdomen, which was constantly present day and night, but which became considerably increased before and during defæcation. He had a temperature of 99.6° F. on the previous day. On examination, his stools showed not only vegetative *Entamoeba histolytica*, but also Charcot Leyden crystals. According to KNOWLES²⁵, the presence of these crystals indicates that he was a carrier. Any how, this man admitted a previous attack of dysentery nearly two years back and since then, his digestion was bad. He was advised a liquid diet and one tablet of Enterozol thrice daily after meals. In five days, blood and mucus disappeared from his stools but his gripping pain persisted especially before going to stools which were still five or six in number in 24 hours. Enterozol was then given twice daily for 10 days. All the symptoms totally disappeared. His stools were examined three times, but no *Entamoebæ* or cysts were found.

Case No. 2 :—N. B., aged 35, gave history of an acute attack of dysentery, two and half years back, which was relieved after 12 injections of Emetine hydrochlor. (He could not mention the dose of the Emetine). Since that time, he has been suffering from indigestion, flatulence, and occasionally had unusual swellings of the abdomen. He got an acute

attack of dysentery since the previous two days. Stool contained both vegetative and cystic forms of *E. histolytica*; *E. coli*, and Charcot Leyden crystals were also present. The patient was anaemic, neurotic and extremely emaciated. His ascending colon was thickened. He had two courses of Enterozol, each course consisting of three tablets daily for ten days, after which an interval of two or three days was given. His symptoms were found abated to a large extent. A further examination of his stools revealed *Ankylostoma duodenalis*, which was eradicated by exhibition of Carbon tetrachloride. No entamoeba of any form was seen in spite of four examinations of stools. The patient was then given an iron tonic, after which, he was completely cured of all his symptoms including anaemia.

Case No. 3:—G. R., aged 19 years, was suffering from indigestion for the previous six months with occasional diarrhoea alternating with constipation. Two days back, he was attacked with a severe type of dysentery; his motions consisted of almost pure blood. He had intense griping pain and had no wink of sleep for the previous two days. Examination of stools showed both *E. histolytica* and *E. coli*. The left lobe of his liver was very tender. He was given Enterozol thrice daily for ten days. His symptoms were relieved 5%. After an interval of two days' rest, he had two further courses of Enterozol. His stools were examined four times on alternate days, but no entamoeba was found.

It will be seen from the above that although no dramatic result followed the treatment with Enterozol in acute cases, like that got by exhibition of Emetine, yet the ultimate results were far more satisfactory.

Chronic amœbiasis.—This series includes 21 cases, with various durations and various manifestations of symptoms of the diseases. Almost all of them complained that they occasionally passed mucus with solid stools and uneasy sensation during defæcation. Majority complained of indigestion and chronic constipation. Seven patients suffered from constipation alternating with diarrhoea. Sixteen patients complained of uneasy abdominal sensations after meals ranging from heaviness to actual pain. Five patients complained of peculiar ball-shaped sensation of wind under the hepatic region. Almost all of them elicited tenderness on palpation over the right iliac region. One patient complained of pain all over the body. Another had pain and tenderness of all the big joints of the body. Four patients were complicated with malaria, and four patients showed ankylostoma in their stools, in addition to amoebic cysts. These latter patients were invariably anaemic and much debilitated. Other helminthic infections were found in eight patients, of which two showed ova of *Ascaris lumbricoides*, three showed *Oxyuris vermicularis*, and two showed ova of both round and thread worms. Whipworms were present in three in combination with either round or thread worms. One showed larva of *Strongyloides stercoralis*.

Two patients showed *Giardia intestinalis*, who had to be treated with Atebrin four tablets for three days in addition to Enterozol before they were eradicated.

It so happened that some cases were treated symptomatically with Enterozol even when the first or second examination of stools did not reveal any *E. histolytica*. It was when the symptomatic treatment for dyspepsia or other complaints failed to relieve them, that further examination of the stools showed the cysts, especially after opening medicines.

Treatment of chronic amœbiasis.—Most of the patients, especially those complaining of constipation had preliminary treatment with Mist. Olei Ricini (B.P.) one ounce thrice daily, till bowels were satisfactorily evacuated. They were then put on a course of Enterozol, which consisted of one tablet thrice daily after meals for ten days. This course was repeated after an interval of a week. The patients were put on a light diet, consisting of rice, potatoes, green bananas, and white fish. Oranges and pomegranates were also allowed, if the patients liked. These two courses had been sufficient to eliminate symptoms in fifteen out of twenty one cases. The rest were more intractable, but four of them managed to get free from infection after three or four courses, but in one case, *E. histolytica* persisted even after four courses, although his symptoms were absolutely cured.

Results of treatment.—All the cases were relieved of 50% of their symptoms with one course of treatment. Seventeen patients (about 80%) were totally relieved of their symptoms after two courses, but the remaining four patients (20%) continued complaining of their vague symptoms, even after the eradication of the amœbæ, as shown by repeated examination of stools. Two of these patients became subsequently free from symptoms after the course of Enterozol was repeated several times. The remaining two became neurotic and nothing could relieve them.

It may not be out of place to remark that in spite of the disappearance of *Ent. histolytica* from stools, they may lurk in the tissues in latent forms for a long time. The ultimate destruction of all protozoal infection depends on the development of tissue resistance and antibodies. A long course of tonic treatment is necessary to cure malarial infection. So also, even after apparent disappearance of cysts from the stools, a long course of Enterozol and other tonics for improving digestion and such others should be undertaken. This tonic course of Enterozol may not be more than one tablet once daily for 10 days followed by another course a week later, along with digestives as already mentioned. This after-treatment should not be given up till the patient feels quite O.K.

Latent amœbiasis in which no E. histolytica was found in the stools, but in which Histidine test was positive.

Out of several cases, which complained of typical symptoms of chronic amœbiasis, such as chronic constipation alternating with either loose, undigested or even diarrhoeic stools, occasional appearance of mucus in stools, reappearance of griping pains for some days, followed by periods of quiescence, dyspepsia, wind in the abdomen, especially "ball" in the hepatic region, and such others, only three responded to Histidine test. These were all relieved by two courses of Enterozol. Formerly, they tried all sorts of medications, such as hepatic stimulants, digestive tablets and elixirs, antacids, carminatives, vitamin B complex and such others, with temporary or no benefit.

It may be mentioned that all these three patients had acute dysentery in the past, and all of them had Emetine injections after which they thought that they were cured.

I like to conclude this article with a note of warning against the use of Emetine in amœbiasis. It is a dangerous drug and has been found to cause general depression and muscular weakness and to cause permanent damage

to the heart even in therapeutic doses, as found out by CHOPRA¹⁴ and Ganguly¹⁵. With the advent of the new arsenical preparations like *Carbarsone*, Bayer's preparations 9945, and *Iodo-oxyquinoline* preparations like *Enterovioform* and *Enterozol*, the use of Emetine should be altogether discarded in amœbiasis, except in complications like hepatitis and the like. Their action may not be so magical like Emetine, but the ultimate curative action is far greater than that of the latter. The special advantage of *Iodo-oxyquinoline* preparations is that they can be used for long time till the infection is eradicated altogether.

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External Otitis*

BY

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EXTERNAL otitis is only a local manifestation of general furunculosis. It is frequent in summer months. Apart from mangoes or perhaps indulgence in sweets, the irritation of hair follicles by the summer heat and the excessive sweating in coastal towns and humid plains, do count as predisposing factors. This is proved by the fact that individuals suffering from external otitis are remarkably benefited by a change of climate to cool hills.

External otitis is comparatively rare in children as an independent disease unlike in adults. In the former, it usually appears as a part of various septic conditions of the scalp, so common in children brought up under unhygienic circumstances and in ill-ventilated rooms. It differs from the typical types, more frequently seen in adult life, in that in children the lymph glands around the meatus are more commonly inflamed with swelling and tenderness, or an abscess of considerable size manifests itself. Adults, and especially males, seem to be more often affected than females, probably due to greater chance of exposure to dust and irritation. Here, one should never forget to examine the urine for sugar, and very often this may be the first occasion when the patient discovers it.

In addition to the classical predisposing causes for furunculosis in general, such as chronic constipation, albuminuria or other debilitating conditions, the local causes are, irritation due to accumulated and impacted wax, and especially when it begins to develop into the condition of keratitis obturans, or the aspergillus infections so commonly met with under moist conditions. These affections, though not directly responsible, are patent causes of irritation or an itching sensation in the ear, necessitating the use of infected nail or an ear pick for relief.

Pathology.—The causal organisms are usually Streptococci, especially in adults, diabetics and the debilitated, though, in children, it may be Staphylococci. The staphylococcal type starts as a diffuse cellulitis in the walls of the external meatus, with slight swelling, redness and a severe pain. The swelling quickly develops, and in severe cases presents an erysipelas-like redness, and a clear defined progressing margin. Another variety, however, usually a mixed infection, limits itself into a blind, localised furuncle which later may point out into a small abscess. These furuncles may be single or multiple, and have a tendency to recur. In both cases, the infection starts in the hair follicles, and hence, the condition is found in that part of the external canal where there are hair follicles.

Signs and symptoms.—Pain is the outstanding feature. In some cases, especially in the so-called moist variety with a slight discharge, the pain may be so severe as to rob the patient of his night sleep, and incapacitate him from his daily work. The pain may spread up the side of the head and down to the neck. The swelling round the ear corresponds to the original seat of infection. The tissue in front of the tragus is swollen when the anterior wall is affected, or behind the ear over the mastoid and upwards towards the temporal region, when the infection starts in

* Specially contributed to 'The Antiseptic'.

the posterior wall of the meatus, which however, is not so frequent as the former. Any slight movement of the pinna gives rise to a severe pain, due to the tension put on the tissues lining the ear. Tenderness on pressure is marked, and found especially in front of the ear. Next in frequency comes the tenderness below the ear, particularly when the glands are involved, and rarely behind the ear. Tenderness over the tragus settles the diagnosis usually. A small degree of deafness may be met with, proportional to the degree of occlusion of the meatus, but is a very rare feature. Examination of the canal or the tympanic membrane with a speculum is a difficult affair, on account of the extreme tenderness, and sometimes on account of the occlusion produced by the swelling itself or the accumulated epithelial scales and debris in later cases. But in the early types or the deep-seated varieties, it may be possible to introduce the speculum, and furuncle definitely located by the naked eye or at least with the aid of a probe.

Diagnosis.—The pain in front of the ear has to be diagnosed from the referred pains from a carious tooth, diseased tonsils or affections of tongue. For pains behind the ear, one has to remember mastoid neuralgia and especially mastoiditis. The latter is very important in view of serious consequences if the diagnosis is mistaken. We cannot rely on a history of otorrhœa, nor the constitutional disturbances which usually accompany mastoiditis. Even a good view of the condition of the tympanic membrane may not be available. Before rushing to radiological examination, a simple tap at the tip of the mastoid, the posterior border or over the suprameatal triangle to elicit the typical mastoid tenderness differentiates this condition from the characteristic tenderness over the tragus in case of external otitis. Further, in mastoiditis we always find a peculiar sagging down of the posterior superior part of the meatus and a marked displacement of the pinna of the ear,—both of which are absent in external otitis.

Prognosis and treatment.—When the diagnosis of external otitis is established, one should refrain from the usual practice of cleaning the meatus with drops of Hydrogen Peroxide or the syringe. Moisture prolongs the condition indefinitely and chances of recurrence are greater. Further, syringing is extremely painful. If syringing is found essential in cases of profuse discharge, or to remove wax and debris, the canal should be thoroughly dried by means of ether or preferably the household hair drying hot-air electric douche. This gives temporary relief from the pain also.

In early stages and also for the initial inflammation to subside, soothing lotions, such as an 8% Aluminium acetate solution or a 10% Glycerine Ichthyol solution, are useful. These can be used for packing the canal and also as an external application. Under this treatment, the œdema disappears and the patient gets relief within 3 to 4 days, when a speculum could be introduced and a better view of the canal and the membrane obtained to detect the presence of any other concomitant disease. Further, Aluminium acetate has the additional advantage of drying up weeping eczematous conditions so common in the moist types of external otitis. When the canal is sufficiently open and the discharge has dried up, a simple application of Gentian Violet solution clears up the remnants of the disease.

One important point to be remembered in this connection is the immense relief the patient gets by the application of heat. It can be used

as the simplest bran-fomentation, or sand or preferably common salt tied in a piece of cloth and warmed sufficiently. The latter has the advantage of retaining the heat for a longer time. For the more fashionable, the same dry heat can be applied by means of a carbon lamp or the hot air douche mentioned above. But wet fomentations or poultices are not recommended as they have a tendency to give rise to an eczematous condition. Even the Glycerine Ichthyol packings can be used as often as is desired, preferably slightly warmed previously; but Glycerine carbolic is condemned as it does not prevent recurrence due to its tendency to attract moisture. Instead of applying the usual cotton-wool plug at the meatal opening, thermogenous wool retains the warmth inside to a greater extent.

A word of warning to those who like to have a spectacular effect of surgical treatment. Most cases are easily relieved within 4 to 5 days under the above treatment, and especially those who are seen sufficiently early, and those that do not have any other constitutional disorders. An early incision, instead of giving any relief, simply aggravates the pain, letting out a little dark blood, without any pus in it. Further, there is a great chance of the infection spreading to other hair follicles—thus prolonging the agony of the patient. Invariably, incisions are not called for, in the streptococcal cellulitic form, but may be necessary only in the later stages of a single large pointing out furuncle, when a small quantity of thick creamy pus can be demonstrated in adults. After ascertaining the maximum point of tenderness by means of a blunt probe, the meatus is disinfected and anaesthetised, preferably by a gauze packing soaked in 10% solution of Phenol with Cocaine. Subsequent dressing may be either a packing with the simple 10% Glycerine Ichthyol which has the advantage of also preventing further infection or with the following solution preferably warmed:—

R	Anaesthesia	...	gr. xv
	Absolute Alcohol	...	3 iiss
	Liqr. Aluminium acetatis	...	m xxx
	Glycerine	...	3 i

If the incision is well advised, the patient should get relief within 2 to 5 hours. In spite of the anaesthesia, incisions are so painful for the patient that every otologist would recommend the good old simple remedies for furunculosis in general, such as a few injections of staphylococcal vaccine or 2 to 3 injections of Collosol Manganese which give relief even for the streptococcal diffuse form. Sulphanilamide preparations, especially M & B 125 could be tried, as an alternative.

Lastly, a word about prophylaxis. Local causes of irritation giving rise to a scratching or picking the ear, are to be avoided. Immediate application of Aluminium acetate or Gentian Violet is a good preventive. Further, attention should be diverted to general conditions giving rise to dryness of skin or the tendency towards epidermal manifestations of allergic diathesis.

Tuberculosis of Lungs and the Ways to Wage War Against*

BY

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It is an agreed point that tuberculosis of lungs is causing the greatest havoc on the face of the earth, devitalizing the mankind. Mortality is increasing, not only as a result of tuberculosis, but also by other inter-current diseases owing to devitalization. The extent to which tuberculosis requires consideration from the preventive point of view, is not sufficiently represented by its accredited death-rates. Verily, the actual mortality may be no less than about 20 times the recorded mortality.

Anti-tuberculosis crusade is a world movement of first importance. Is extermination of tuberculosis of lungs from the world possible at all? It should be quite possible when once the causative factor is definitely made out and agreed to and when that cause is possible to be removed. The causative factor is now definitely known to be of bacterial origin by the prolonged researches of VILLEMEN (1865) and finally by the demonstration of KOCH (1882). This fact remains up-to-date undisputed by later scientific research workers. The disease having been proved infectious and communicable, it is clear that the infective process must be conceived and dealt with on the broad lines which had been applied effectively to other infectious diseases. The earliest trace of infection should be sought for and dealt with.

The following *ammunitions* are required to wage war against the dire enemy 'Tuberculosis of Lungs' to a successful termination and to eradicate the same from the world.

- I. Notification of the disease.
- II. Tuberculosis dispensary clinic.
- III. Establishment of sanatoria and supervised home treatment.
- IV. Establishment of tuberculosis colonies.
- V. Establishment of tuberculosis hospitals for incurable cases.

I. **Notification of the disease.**—It is a great necessity with the consequent advantages from the adoption of compulsory notification as any other infectious disease is. By notification of the disease, it does not mean that every case should be immediately transferred to a tuberculosis hospital for segregation as in other infectious diseases. This is a practical impossibility. In some instances, the removal of the patient to a suitable hospital will be sufficient. In many others, notification may discover a whole nest of the disease. Several cases of consumption may be found in one and the same household and the whole house may probably be infected.

But this will always prove a weakness of any scheme in which notification stands alone i.e., without an organized system for the treatment and relief of tubercular patients at their own houses or in one or other type of hospitals. It is at this point that the significance of the dispensary becomes apparent. The general practitioners and family physicians can best serve the campaign by notifying to the authorities concerned

*Specially contributed to 'The Antiseptic'.

immediately when they come across a case of consumption, for necessary investigation with regard to preventive measures.

II. **Tuberculosis dispensary clinic.**—This will play the most important part in eradicating the disease in many aspects. This clinic should receive and examine all cases, (1) keeping a record of every case with an account of the patient's illness, history, surroundings and present condition; (2) should make bacteriological examination of sputum and other discharges; (3) should give instructions to patients regarding how to treat themselves and how to prevent or minimize the risk of infection to others; (4) should dispense necessary medicines and sputum bottles-disinfectants and, where the patient's condition seems to warrant it, foodstuffs and the like; (5) should arrange for visitation of patients at their own homes by a qualified medical man and a specially trained health visitor for the double purpose of treatment and investigation into the state of the dwelling and general conditions of life, and the risk of infection to others; (6) the selection of patients for hospital treatment—of early cases for sanatoria and of late cases for incurable homes, and the supervision of patients after discharge from hospital. A dispensary such as this is a much more elaborate institution than ordinary dispensaries attending to out-patients. The latter is, however, primarily directed towards the treatment of the individual patient and little attention is extended to the wider aspect of the problem. It goes without saying that the clinic need be sufficiently and efficiently equipped to meet the various aspects and scope of the dispensary. The dispensary should also play an important part in the relief of the pecuniary distress which the disease so often entails. The families and dependents of affected persons must not only be safeguarded in respect of their health and thus kept fit for work, but where necessary, as for instance, when the chief wage-earning member is affected, the dispensary should take cognizance of the household means and, if desirable, put the household in touch with one or two public or charitable institutions. Suitable lines of employment of a light and open air kind for consumptives not requiring hospital treatment should be determined.

Towards the clinic, inquiries from all sides regarding tuberculosis should be directed. Patients in all stages of tuberculosis should be invited and, likewise, persons who may be anxious to know if they are tubercular. This great collecting centre constitutes the seat of diagnosis and classification of the varying types of the disease. This comes in turn to be a clearing house. Each case will receive appropriate guidance and direction whether at the centre itself or at one of several residential institutions with which it should stand in organic relationship.

III. **Sanatoria.**—There appears to exist a popular notion at the present time that the solution of tuberculosis problem has been found in the establishment of sanatoria for consumptives in different parts of the country. The establishment of sanatoria and hospital meet only one aspect of the question. Pulmonary tuberculosis is a disease of extremely varying complexion. Only a certain number of cases and early cases are suitable for sanatorium treatment. In respect of such patients existing, sanatoria can undertake the care of a small number only. Sanatoria are intended for selected patients with a view to cure. It is concerned with the effective arrest of the disease and the educations of the public as to how to treat and prevent it. The two conditions under which the Tubercle bacilli

cannot withstand are fresh air and sunlight. If the patients will only be out of doors—lying, sitting, walking or working according to the nature of the case—a large part of each day, and when indoors will allow access of fresh air day and night, satisfactory results may be obtained anywhere. It is erroneous to believe that a cure can only be effected and health maintained under conditions which their ordinary residence and station in life will not permit them to enjoy.

Until there is a sufficient number of sanatoria, supervised home-relief must play a chief part. In this, the problem is how best we are to afford a patient relief from suffering and distress, how best to minimize the risk of his becoming a source of danger to other persons, and how to limit more effectively the communication of the disease from the already affected person to other members of his house-hold, to the dwelling itself and also to persons brought into contact with the affected person at work or elsewhere. The chief source of infection is the expectoration of the consumptive. The great danger lies in the drying of the expectoration and the blowing about of the dried infectious material. The spread of consumption can be largely prevented if the following directions be obeyed. There need be no serious dread in ordinary intercourse with patients. The breath of the consumptive is not directly contagious. The patient should expectorate into a jar or a cup containing a disinfectant. The spittoon should be changed once in 12 hours at least. It should be cleaned with boiling water after being filled with the same. When the patient is out of doors, he should carry a pocket spitting flask. The patients should never spit on the street and should not use handkerchiefs for expectoration. If this has to be done, it may be burnt after use. Bits of rag or paper which may be used for convenience should be similarly treated. The expectoration should on no account be swallowed, for the disease may pass on to other organs. Consumptives should avoid kissing. Consumptive mothers should not suckle. If expectoration has been accidentally deposited on the floor or other object, it should be wiped and burnt and the surface of the object cleaned with a strong antiseptic. Rooms which have been long occupied by a consumptive should, before occupation by some one else, be carefully disinfected. Fresh air is the food of the lungs. The patient should occupy as airy a room as possible. It must be scrupulously dry. The windows should be freely open. When able, the patient should be out of doors once or several times during the day. He must avoid over-exertion, damp or chill, which would counteract the benefit.

IV. Tuberculosis colonies.—These should be established near sanatoria, where poor patients might be enabled for long periods under hygienic conditions to maintain themselves honourably by contributing to the common good of the residents. The workers should remain at the colony under medical surveillance. The character and amount of their work would be suitably gauged and directed. The produce of the colony resulting from the patients' labours would form an important supply for the sanatoria. The residents in the colony should be recruited by selection from patients whose disease had been successfully treated at the sanatoria, explaining to them its advantages in the shape of both physical and pecuniary benefit.

V. Hospital for advanced or dying patients.—This will play the most important part of the campaign against tuberculosis. For, it is these patients that throw about the Tubercle bacilli indiscriminately and thus

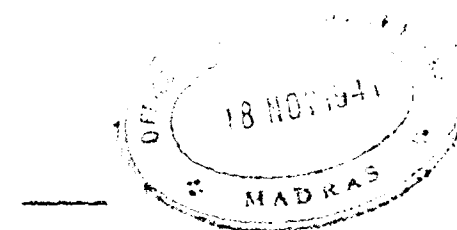
they are the greatest sources of infection endangering others. So, hospitals for these cases are even more urgent than the establishment of sanatoria, in the campaign against tuberculosis. It should be strictly enforced that these patients must, in the ordinary course, continue in the hospital until the end. If the hospital is to fulfil its mission, patients once admitted must not be discharged save for quite exceptional reasons. To make the patients a little better and then discharge them from further treatment is almost completely to negative the purpose for which the hospital was intended. By doing so, the patients feel satisfied and think that they are looked after—though we are quite helpless in the treatment of these cases. The sputum and other discharges should be properly disinfected and destroyed.

These hospitals will also play an important part from the research point of view. Most of these patients admitted in these hospitals will succumb. Post mortem examinations may be undertaken invariably on these cases and scientific notes of every case may be recorded on the pathological conditions of the disease which may be compared to the clinical findings of the respective cases. This will certainly lead to expansion of our knowledge on the subject, as to how the disease has progressed in every case, how the treatment done has been a failure and how best the treatment of such cases could be done.

Housing question relating to dwellings, lodging houses, schools, workshops, factories and offices must occupy a chief place in the preventive programmes against tuberculosis.

In the end, I do not think that the measures required for extermination of the disease are a practical impossibility. Where there is a will, there is a way. Success is certain, though not immediately, at least in the near future. The measures have only to be co-ordinated by all concerned such as the State, Local bodies, philanthropic and charitable institutions, community at large, the actual patients and finally the general practitioners as well as the medical officers-in-charge of the campaign. An impetus and a start have already been made by no less a personage than Lady Linlithgow and also by The Tuberculosis Association of India, and now it is upto others to cooperate and do their best towards the extermination of this dire disease.

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Caries of the Teeth*

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Definition.—Caries of the teeth is a process by means of which molecular death of a part of a tooth gradually takes place, due to the action, on the dental tissues, of various germs.

It is not a specific disease due to one special kind of germ, but a rotting away of the tooth from the action of one or more kinds of germs which have the power either of producing acid or liquefying collagen, or of doing both.

History.—Caries or decay of the tooth is more or less a disease of civilisation. Though it existed among the ancients—for, there are traces of this disease found in ancient skulls, and mention has also been made in ancient Hindu Medicine, of fillings with gold and silver, presumably for the decay of teeth—yet, its prevalence at that distant age was nothing compared to that at the present day. That it is a disease of civilisation is proved by the fact that even at the present-day primitive races are free from it, compared to the modern civilised races. It is further shown by the alarming extent of the prevalence of this disease among the American and European school children, 75—95 percent of whom are found to be suffering from caries.

Introduction—The mouth cavity even in the healthiest condition is a good incubator for all sorts of germs. In it are found all the conditions viz. the temperature, moisture, medium etc. that are needed by the germs for their development. Naturally, all sorts of bacteria are greater in number and more virulent in action in unclean mouths than in clean ones.

Prevalence of caries.—To what extent dental caries is prevalent in India could not be ascertained; for, there are no records available owing to the backward state of dentistry here. From what little information I have collected from my personal dental practice and from the experience of other colleagues, I put down the extent of this disease among Indians to between 30-55 percent.

1. *Ancient races*:—Statistics show that races like Romans and Egyptians which had reached a high degree of civilisation were more susceptible to dental caries than the races that lived under more primitive conditions.

2. *Existing primitive races*:—Skulls of practically all the races over the world have been examined and they show clearly that races whose principal diet is flesh are more free from caries than those existing only or wholly on vegetable diet.

3. *Modern civilised races*:—Dental caries has increased considerably not only in the human beings but also in the domestic animals.

Frequency and distribution.—The first permanent molar (Mandibular more than Maxillary) is most prone to caries due to un-hygienic condition of the mouth. The caries commences on the occluding surface. The second permanent molar comes the next—the lower more than the upper; then

the lower pre-molars, second more than the first; the upper pre-molars, upper incisors and, last of all, lower incisors which are practically immune to caries because of the cleaning action of the tongue and lips. Caries is more common in females than in males and more active during the period of growth.

Morbid anatomy and pathology.—1. **MACROSCOPIC APPEARANCE** (Naked eye appearances):—(a) *Enamel*:—The first sign is that, it loses its normal polish, and next a whitish spot appears and gradually becomes darker. When the process is slow, discoloration is more marked than when it is rapid. Later on, the part disintegrates and a cavity is left. The destruction advances until the dentine is exposed. When caries begins on the approximal aspect of teeth, the actual point of contact is not always decalcified, but around it is a zone showing decalcification. When decay of teeth has started on the approximal surface and reaches the under surface of the enamel of the crown e.g. on the molars and pre-molars, the enamel on that part appears bluish-white due to decalcified tissue which has not been removed by saliva. As the caries reaches the surface, enamel grows whiter and eventually becomes so thin that mastication fractures it and the under surface is found to be soft and cheesy. This is known as *Secondary Dental Decay*. In children, in whom rickets develops after the first year i.e. after the eruption of incisors, these teeth decalcify, become brown and leathery. In these children, the saliva is acidic.

(b) *Dentine*:—It becomes tough, hard and not soft and cheesy as with the case of enamel. After softening, it undergoes disintegration and acidity is formed and pigmentation develops. The process may spread in any direction with varying rapidity. In a tooth of ordinary structure, the cavity is cone-shaped. The apex of the cavity is directed to the pulp known as *penetrating caries*. In badly developed teeth, the decay extends laterally and undermines the enamel. In hypoplastic teeth, caries spread rapidly near the junction of the enamel with the dentine and causes the enamel to break away. In *rapid caries*, the dentine may come off in big masses. This condition is also known as *caries humida*. In this case, the action of the acid is more than that of the germs. When the process is slow, the action of the germs keeps pace with the action of the acid.

(c) *Cementum*:—It is less commonly affected than the enamel or dentine. Generally starting at the neck of the tooth, the cemental substances become soft and disintegrate leading to the formation of shallow cavities. The cavity becomes wide but seldom deep. An exception occurs at the junction of two roots of a molar tooth. The cavity here is often deep.

(d) *Nasmyths' membrane*:—For sometime, the crown of the tooth is covered with a thin layer of epithelium known as Nasmyths' membrane. It is also destroyed in caries but there is not much change in colour.

The pathological anatomy of caries consists of the destruction of Nasmyths' membrane, if present, by infiltration with germs to such an extent as to obscure all structure. A tough layer of soft tissue composed mostly of germs is often found adhering to the surface of enamel in the early stages of caries, and it is hard to say if this is altered Nasmyths' membrane or a product of germ activity. Its importance lies in the fact that it is very difficult to remove by the ordinary methods of cleansing teeth, and if it harbours acid-forming germs, by holding such acids in close contact with the teeth, it may greatly aid the process. On the other hand, it seems that at times such films may be protective.

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(e) *Phenomenon accompanying caries*:—(i) *Translucency*:—The part situated between the pulp and the carious process appears translucent. The affected area is cone-shaped, with its apex towards the pulp. There are many theories about the cause of translucency, but the most commonly accepted theory is the following:—The opacity of the normal dentine is due to the fact that the matrix and contents of the tubes have different refractive indices. If these indices approximate, the dentine becomes translucent, and we know that there is some change in the contents of dentine tubes which affects its refractive index.

(ii) *Pigmentation*:—The colour may vary from pale yellow to dark black. The more acute (rapid) is the process, the higher is the colour. It is produced probably by the action of 'chromogenic bacteria'.

2. *CHEMICAL CHANGES*:—There is a decrease in the amount of lime salts (1/13 of the original) with the loss of organic tissue.

3. *MICROSCOPIC APPEARANCES*:—(a) *Enamel*:—The surface is covered with a mass of micro-organisms. The enamel is decalcified between the rods and the interstitial cement substance. The enamel globules being set free are dissolved and washed away. The enamel may be penetrated to a considerable extent before the breaking down of the tissue occurs.

(b) *Dentine*:—From within towards:—(i) An unstained part in decalcified condition. (ii) The streaks (lines) of stain which are germs occupying the dentinal tubes. (iii) The globular masses which are also germs occupying the matrices and tubes also scattered irregularly. These have been termed as '*Liquefaction focii*'. (iv) Irregular masses occasionally form by the fusion of liquefaction focii.

(c) *Cement*:—It resembles caries of the dentine.

(d) *Pulp*:—It is inflamed causing a shooting pain.

Bacteriology.—A number of micro-organisms are associated with the production of dental caries but the proofs are not definite. The pathogenic organisms found in the mouth are:—(1) Staphylococi. (2) Pneumococci. (3) Klebs-Löffler or Diphtheria bacilli. (4) Tubercle bacilli (T.B.). (5) Odium or Saccharomyces albicans or Thrush fungus.

The non-pathogenic organisms found in the mouth are:—(1) Streptococcus brevis. (2) Micrococcus tetragenus. (3) Leptothrix buccalis. (4) Spirochaete dentium etc.

Of these, the Streptococcus brevis is the most constant, and is found in healthy as well as unhealthy mouths. What function these non-pathogenic organisms perform, is not definitely known.

It is interesting to note the comparative immunity of the system, against the attack of the pathogenic organisms that are mostly found in healthy or unhealthy mouths. The two reasons for it are:—(1) The bactericidal action of the saliva. (2) The resisting power of the body due to antitoxic property of the blood.

The organisms concerned with the decay of teeth are of two kinds.

1. *Acid-producers*:—They are anaerobic (capable of thriving without oxygen) e.g. Streptococcus brevis, Bacillus necrodentalis, Staphylococcus albus and aureus, and Sarcina albus.

2. *Liquefiers*:—(of dentine): They are aerobic (requiring oxygen for their growth) e.g. Bacillus mesentericus ruber, Bacillus gingivae pyogenes etc.

Process of caries and sources of the acid.—Caries is a chemico-parasitical process consisting of two parts: 1. Acid formation and softening of the tooth substances by means of these acids. 2. Liquefaction and digestion of the softened tooth-tissues.

For the working of this process, two things are absolutely necessary, viz., the bacteria, and such suitable food-stuffs (carbohydrates) which can be decomposed into acids by the action of these bacteria.

Lactic acid is the principal form of the acid which is formed from the carbohydrates which lodge on the teeth and undergo fermentation. In the case of *sugars* of the grape sugar group, galactose is directly fermentable. The cane-sugar and milk sugar are only fermentable by taking water into their molecules. The dextrose and levulose are acted upon by lactic acid ferment:

Cane-sugar + H₂O ← Dextrose + Levulose ← Lactic Acid ferment and gases.

In the case of starch, the Ptyalin (ferment containing in the saliva) converts it into grape sugar which is directly fermentable. Experiments have proved that when fermentable albuminous materials are combined with saliva, very little acid is formed. But, when such substances as meat undergo putrefaction, acids are formed, saliva neutralises these acids and the action of the saliva remains alkaline. Vegetable food is less likely to ferment when raw than when it is cooked.

When once the decalcification has taken place by means of acids, the remaining major portion of the tooth substance is dissolved by the action of bacteria which act upon the dentine in the same way as the pepsin of the gastric juice acts upon the albuminous materials. It has been shown that the primary decalcification of the enamel often starts around and not at the point of contact. This fact seems to indicate that the food in the fluid condition is maintained around the point of contact by capillary attraction. Again, in mouths which are kept quite free from the solid food, caries is often found in pits and fissures, etc., which clearly indicates that the disease must arise from the action of fluids and not solids. Solid food cannot ferment so rapidly as the liquid food, because, the particles cannot move about so freely in the solid substances as they can in fluids. With the solid substances, the acid formed by the germs remains just around them and cannot disperse; and when the acid is raised to a certain percentage, it does not allow the germs to grow. Some observers do not agree with this view of mine. The mucus secreted by the glands of the mucous membrane often has an acid reaction due to the presence of acid phosphates, lime, and soda; and it is a fact that this acid reaction is in some way related to the rapid caries in cases of mouth-breathers.

Spontaneous arrest of caries.—It may occur in milk or permanent teeth but only if they have living pulp. Among the permanent teeth, it is mostly met with in under-developed 1st permanent molars. The teeth show extensive loss of tissue, and the enamel of the masticating surface and of the sides disappears. The exposed dentine is dark and the probe will show that it is harder than normal. Microscopically, the dentine shows the

colour of caries which is more dark. Surface is irregular and the dentinal tubes end eruptively on the surface.

It is not clear how the arrest of the process is brought about. Probably, it is due to vital activity of the tooth itself, because the process is not arrested in pulpless teeth. Arrest of caries is met with in pulpless teeth as well, but the death of the pulp always occurs after the arrest of caries.

Experimental production of caries.—Perfectly sound teeth cut in pieces were placed in a mixture of saliva, meat and bread. Mixture was changed frequently for 3 months and kept at a temperature of 98.4°F., at the end the caries of the teeth was found but there was no translucency and pigmentation. If these teeth are exposed to coffee, tea, tobacco, fruits etc., shades of different colours develop.

Sound teeth, if placed in 2% glucose broth in test-tubes and *Streptococcus salivarius* added later on, showed penetration of dentine by germs from the pulp side after 14 weeks.

Teeth were kept in cow's milk (50 to 100 c.c.), the milk curdled and was acid in 2 or 3 hours; remained so for 2 to 3 days and then changed to alkaline with bad smell; dentine became soft with white opaque patches on the enamel and the dentine tubes showed germs in them.

Susceptibility and immunity to dental caries.—For this purpose, teeth are clinically divided into three groups:—

- (1) *Native*:—The teeth of primitive races.
- (2) *Sclerotic*:—(Hard) Hard teeth immune to caries.
- (3) *Malacotic*:—Soft teeth susceptible to caries.

For examination of certain markings on the enamel surface, the following method may be done:

The tooth may be washed with water, dried, surface cleaned with Zylol or Ether and Alcohol; Graphite may then be applied by cotton and the excess may be removed. It marks the pits and pitches and in this way the surface of the enamel is seen traversed by a series of parallel grooves and ridges to which the name '*Imbrication lines*' is given. These lines are mostly marked at a point $\frac{2}{3}$ of distance between the neck and the crown and they are more marked in the third type than in the second; and more in the second than in the first. These lines are intimately related to the striae in the enamel and they are the result of intermittent and interrupted calcium deposit. Hence imbrication lines can be regarded as an index of the degree of calcification of the enamel. Organic contents of the enamel is 1 to 2% (the rest is calcium); and increase in the amount of organic matter lessens the resistance of the enamel and helps caries. The caries follows the line of organic matter. The difference in the hardness of the enamel is not due to quantity but to the quality of the inorganic salts or calcium salts. Pits and depressions, cracks and fissures are pre-disposed to caries.

Influence of lime salts in the food.—In districts where the soil and drinking water are rich in lime, the teeth are sounder than in the places where lime is deficient. If the teeth are regular and in good position, the caries is less. Some mouths are self-cleansing and others are not. In the former, the caries is less than in the latter.

Saliva:—A strong flow of saliva flushes the mouth and removes the

particles of food from the teeth. If it becomes less and thick, caries increases. Highly alkaline saliva is the best means to check the progress of caries. Diet, rich in calcium, increases the alkalinity and the quantity of saliva and favours good quality of teeth. Potassium (part of the saliva) Sulphocyanide forms .1% of the total solids in the saliva. It has got no effect in any way on caries.

Pregnancy increases susceptibility to caries because calcium is required for the formation of bones of foetus; but there is no proof of it. Feeding in infancy influences the disease. Breast-fed children have the best teeth. Starchy foods given along with the milk to the babies is harmful.

Adenoids:—Because of mouth-breathing, it causes the food to adhere to the teeth, increases the number of germs and helps the caries.

Poverty:—Poorest children have the best teeth because they take most simple food and cannot afford starchy substances such as cakes and sweets.

Ætiology of dental caries.—There are several theories propounded by different investigators. They are: (1) chemico-parasitic, (2) inflammatory, (3) electrical, and (4) chemical.

Of these, the first namely, that "Caries is a chemico-parasitic process" as already described above, is the best and most accepted by the dental profession.

The form of relative positions of the human teeth favours the lodgement of food-stuffs in solid form or in solution upon or between the teeth. Of the three groups of food-stuffs—protein, carbohydrates and fats—the carbohydrate group is specially concerned in bringing about caries. Of the three groups of carbohydrates—cellulose, starch and sugar—cellulose undergoes no change in the mouth. Starch is first converted into dextrose and, by microbic ferments, into lactic and other organic acids in minute quantities. Sugars are directly fermentable and are converted into acids whereas cane-sugar takes up water and breaks up by ferment *Invertine* produced by bacteria into dextrose and levulose.

Cane-sugar + Water (*Invertine*)—Dextrose + Levulose directly ferment and produce *lactic acid* by the action of germs. These acids attack the teeth where they are produced i.e. wherever the food is solid or fluid and has lodged on or in between the teeth and they lead to decalcification, first, of the enamel or cementum and later on, of the dentine. The decalcification of the enamel results in its complete destruction. Because of the decalcification, the organic matter does not hold together and as it is of nitrogenous nature, it undergoes decomposition by the action of bacteria in the presence of moisture and suitable temperature. This decomposition is brought about by a ferment produced by the bacteria in the mouth. The prevalence of caries in modern races is due to the soft character of food and increased consumption of carbohydrates.

Predisposing causes of caries.—The causes of dental caries may be divided into two main parts:

1. *Local*:—(1) Connected with the mouth. (2) External.
2. *General*:—(1) Heredity. (2) Pregnancy. (3) General environment.

LOCAL CAUSES:—(1) *Connected with the mouth*:—The formation of the teeth has much to do with the occurrence of caries. *Teeth with weak structure are more prone to caries than well-formed teeth.* A normal arrangement of the teeth and the gums in Nature is a great safeguard against the caries. This arrangement is that "all the teeth come in contact with

one another by one single point forming the V-shaped spaces in between them, which are filled up by the gum tissue." This arrangement will enable Nature's cleansers (the tongue and lips) and the artificial means such as brush and tooth-pick to keep the teeth clean. But any condition (*e. g.*, abnormally placed teeth or loose and receded gums) which alters this natural arrangement, will tend to promote the development of dental caries by creating spaces facilitating lodgement of food-stuffs and micro-organisms.

(2) *External causes*:—The acid and mucoid condition of the saliva due to dyspepsia, febrile conditions and pregnancy, is a potent cause of caries. The acid condition works as a direct cause, while the other works indirectly by sticking on to the teeth and so helping the food-particles to stick on them. Such food as has the tendency to lodge on teeth-surfaces and to undergo fermentation is the kind that best promotes the inception of caries. *Hence, the carbohydrate food is more likely to cause caries than the proteid or meat diet. It is said that meat-eating individuals are more free from caries than those whose staple food is carbohydrates.* But the experience of medical practitioners and dental surgeons in India is otherwise. It is a common experience that Indians who practically live on carbohydrates are infinitely more free from dental caries than any meat-eating European nation.

GENERAL CAUSES:—(1) *Heredity*:—Heredity does play some part in the causation of dental caries. Its action can be considerably neutralised if proper attention is paid to the food and the habit of cleansing of the mouth of the individual.

(2) *Pregnancy*:—Some women show marked tendency to caries of the teeth during this period. *This may be due to the acid condition of the saliva or to the vomiting attacks during this period.* This pre-disposition during this period is not constant, for there are instances of mothers of many children possessing good teeth and showing no such pre-disposition at all. It is believed that this pre-disposition to caries has its origin in the beliefs amongst mothers that: (a) it is natural for a mother to suffer dental troubles during this period, and (b) that she must let her teeth and mouth severely alone during this period, lest any effort at cleansing on her part would hurt or disfigure the foetus. If only mothers could be made to keep their mouths clean during this period, this factor would cease to be a cause of dental caries and it would be in the interests of both the mother and the baby, that pregnancy should cease to be a predisposing cause of caries.

(3) *General environment*:—It is a fact that not only the human teeth have deteriorated and are still deteriorating in the present generation, but that the whole constitution is undergoing a similar change; and that the resisting power of the present generation is much lower than that of our ancestors. Under these circumstances, the satisfactory cause of this must be sought for in the general environments of the present generation than in any particular local condition. *So, it is believed that the present-day cooking and the mad rush of modern civilisation are responsible for the great prevalence of this disease.* The present-day culinary art needs the food-stuff to be free from fibrous parts, and so prepares it that the cooked food is liable to stick on to and in between the teeth, and undergo acid fermentation in the mouth, thus starting the process of dental caries. The second factor works by lowering the vitality of the teeth and by leaving little time for the individual to carefully attend to the hygiene of his mouth and teeth.

Detection of caries.—The detection of caries in an early stage is often difficult. *Inspection* may detect a white spot on the enamel, a small hole, an altered translucency of a part of the tooth, a darkening of an area, or the lodgement of food, and it is necessary to inspect every part of each tooth that is accessible. Then a *fine probe* may detect a widening of fissures and deepening of natural pits and holes between the teeth or at the margin of fillings, or may pierce friable enamel or softening dentine, or cause pain when passing over an eroding surface. The passing of silk or strands of wool between teeth will detect roughness.

Signs and symptoms of caries.—(1) Lodgement of food stuff. (2) White spot in the place of decay. (3) White spot turning black with the advancement of decay. (4) Cavity formation. (5) Beginning of pain, though very slight. (6) Pain becomes severe on the cavity extending to the pulp.

As described above, the pain comes on at a later stage in the process of decay. It is due to irritation of the fine ends of the nerve in dentinal tubules and will vary with the situation and extent of the cavity. If it is on the occluding surface, change in the temperature or introduction of an irritant will produce pain which increases as the cavity deepens. In cavities under the gums or on the approximal surfaces, the pain is often not felt until the pulp is involved. The pain varies also with the temperament of the individual. Some patients do not complain of any pain even if the disease is advanced. One may be better advised not to wait for the treatment till this symptom appears, for then the treatment becomes more painful and more troublesome.

Treatment of dental caries.—(1) Preventive or prophylactic. (2) Remedial.

PROPHYLACTIC TREATMENT:—1. *Mechanical*:—The mechanical process is brought into operation when the hard fibrous portion of the food that is usually consumed by these uncivilised people, thoroughly scrubs the teeth clean during the action of mastication. The teeth are also scrubbed clean by the strong movement of the muscles of the tongue and lips, and the coarse fibrous matter contained in the food-stuffs.

2. *Chemico-physiological*:—This process is brought about by the antiseptic property of the saliva, and its amylolytic action on certain parts of the food-stuffs.

3. *Hydro-dynamical*:—This process operates by the parts of the mouth continually washed by the constant flow of saliva, and by its being forcibly pumped through the crevices between the teeth, and by the muscles of the tongue and lips.

4. *Bacterial*:—This process operates through certain bacteria, whose presence is beneficial to the health of the mouth cavity. So, taking Nature as our guide, our preventive treatment must consist of: (1) the adoption of firm, fibrous food stuffs that would scurb the teeth clean and call forth the play of the muscles of mastication, and (2) encouraging the flow of the saliva, in order to wash clean the interspaces between the teeth, and to exercise its antiseptic action. This can be best done by introducing in to our meals natural organic acids such as are found in fruits, especially as a final course. This line of preventive treatment, if scrupulously followed, would suffice; but very often, it is found that some artificial means at prevention may be needed in addition to these.

5. *General instruction*:—The prevention of caries is the ideal of dentistry. Insist upon breast-feeding, if practicable. A diet in early

years which requires efficient mastication should be given. Avoid pulpy foods. Give crust or toast instead of bread soaked in milk. Better give a piece of bone instead of minced meat. Avoid the use of carbohydrates which are easily fermentable. Sugars are most harmful and so the sweets should be forbidden. The last food-stuff given in a meal should not be of sugar or starch. The meal should finish with a fruit or cheese in the case of children. Fruits are specially useful as they are slightly acidic and stimulate the flow of saliva. Give meals at regular hours to children and encourage mouth breathing. Avoid tender teeth because they hinder mastication and because the natural action of mastication is interfered with. Treat them by filling or extraction. The proper cleansing of teeth after the meals is most important. It should be done after every meal. Nothing should be eaten between the meals. The cleansing of teeth is especially needful after the last meal of the day. Brushing the teeth early in the morning only is useless. The longest period between the meals is the night during which teeth are left uncleaned, and just after cleaning in the morning, they are given a fresh lot of fermentable material by eating a break-fast. The kind of tooth-brush is not so important as the manner of using it. Whatever it may be, it must be kept properly cleaned. The best way is to dip it in a strong solution of Sodium chloride after use, and leave it to dry in direct sun-light. Another method equally useful is to keep the brush permanently in a glass of 3% Lysol. It will be softer than it would, if it were kept dry but it will not rot as it does if left out in the air in an ordinary way. There is no need to wash the Lysol out of it before use, because it is a good tooth-wash, alone or with a paste or powder. A dentifrice may be prescribed in the form of liquid, paste or powder. For emergency mouth-wash, one of the following may be selected:—

Chloramine	... 100 parts per 10,000	Thymol	... 100 parts per 10,000
	(Nearly 1%)		(nearly 1%)
Chinosol	... 80 parts per 10,000	Hydrogen Peroxide	... 100 " " "
		(H ₂ O ₂)	
Lysol	... 50 " " "	Boric Acid	... 300 " " "
Mercury Chloride	... 2 " " "	Potassium Permanga-	
Formaline	... 5 " " "	nate	... 50 " " "
Phenol (Acid Carbolie)	100 " " "	Potassium Chlorate	... 200 " " "

The order is of efficiency.

Prevention of caries by artificial means:—As dental caries is a chemico-parasitical process, naturally something counteracting the chemical and parasitic processes, must form the keynote of the preventive treatment. So, the treatment must consist of antiseptics to counteract the effects of parasites, and antacids to counteract the effects of acids that result from the chemical action.

Antacid mouth-wash:—

℞ Magnesium Carbonate ... 3 iv.
Aqua Rosæ ... 3 iv.
Prepare mixture.

Sig: One teaspoonful to be kept and sluiced about in the mouth, morning and night. Shake before using.

Acid mouth-wash:—

℞ Acid Boric ... gr. xv.
Eucalyptol ... ℥ ii.
Tin. Card. Co. ... ℥ x.
Aqua Rosæ ... ad. 3 i.
Mft. Lotio.

Sig: To be used as mouth-wash in $\frac{1}{2}$ a cupful of warm water, 3 or 4 times a day.

Tooth-powder:—

℞ Crete Præcip ... 3 i.
Pulvis. Iridis ... gr. xxx.
Oil of Cloves ... ℥ xx.
Otto Rosæ ... ℥ iii.

Prepare powder.

Sig: To be used with tooth brush, in the morning and at bed time.

Brushing of teeth.—Too vigorous brushing should be avoided as it produces grooves on the teeth especially when it is done on the same direction. Brush up and down and inside the teeth especially the lower ones.

If gingivitis, tell the patient to massage the gums towards the teeth with finger and thumb or with soft tooth-brush. Scaling and burring of teeth should be done, if necessary.

Remedial treatment of dental caries.—It is a purely dental treatment, and consists of the removal of the decayed portion, the sterilisation of the adjacent healthy tooth tissue, and the filling up of the cavity with suitable filling materials. There are many materials used for the filling of carious teeth. Each has got its advantages and disadvantages. The following are the substances commonly used. (1) Gold. (2) Amalgams. (3) Osteoplastics. (4) Synthetic cements. (5) Gutta Percha. (6) Porcelain. (7) Solutions of gum resins with cotton wool as temporary filling.

Ideal filling:—It must be hard, capable of standing any chemical action of the mouth. It should be non-irritant. It should be non-conductor of heat and electricity and it should be easy of adaptation. It should retain its shape and its colour after insertion. The colour should resemble the natural tooth. At present, no material fulfils all these conditions.

When dental caries is extensive and the crown of the tooth is destroyed, the tooth can be contoured up by means of an artificial crown. In cases of slight caries in adults or the extensive superficial caries in children, the remedial treatment within the reach of the medical practitioners is the use of drugs such as Silver Nitrate and Absolute Alcohol. *This treatment must consist of the application of the saturated solution of pure sticks of Silver Nitrate twice or thrice at intervals of a week, and the application of Absolute Alcohol once a day during the intervals.* During the application of these drugs, saliva must be kept away from the parts treated. These drugs act by precipitating the protoplasm in the dental tubes. Beyond this treatment, a medical man may do the radical treatment *viz.* the extraction of the tooth.

Conclusions.—The medical practitioner would be better advised to stop short at palliative treatment, and leave the legitimate dental treatment to a properly qualified dental surgeon.

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Cases and Comments

Non-Specific Acute Intestinal Toxæmia

BY

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ONE reads about and sees cases of chronic intestinal toxæmia having its origin in the abnormal chemical changes in the intestines. Here, the chemistry of the intestines is at fault and some toxin or toxins of an indefinite nature are produced and after absorption cause various and varying symptoms. The symptoms thus produced are numerous and are both mental and physical and, even if not serious, they certainly are responsible for a good deal of suffering and misery. Every practitioner of medicine is familiar with the cases of the so-called "dyspepsia with the accompanying neurasthenia." All these cases are of a chronic type and the toxæmia of a non-specific nature. The acute intestinal toxæmia of specific origin is also recognised. *e.g.* cholera, ptomaine poisoning, dysentery; typhoid etc. But no mention is made of acute toxæmia of a non-specific nature. This obscure but serious type of toxæmia, producing serious and distressing symptoms which can possibly become fatal, has not received the attention it deserves.

I had the opportunity of watching the results of the treatment given by Hakims, which invariably consists of the elimination of the effete matter produced by the putrefaction or preponderance of one of the four humours. Thus, the administration of purgatives constitutes the sheet anchor of the Unani treatment. In many a case where doctors were afraid of giving any opening drug except the mildest laxative, lubricant or enemata on account of the collapsed condition of the patient, the Hakims had the courage guided by the principle of universal purgation to administer a fairly strong purgative attended in some cases with surprising and at the same time gratifying results. Out of many such cases which came under my observation, I mention one case.

I. Cases of collapse due to acute intestinal toxæmia.—An old gentleman, a near relation of a doctor-friend of mine, was suddenly taken ill and this doctor was in constant attendance. Being a friend of the family, I used to visit him daily although he was under the treatment of other senior doctors and had the opportunity of studying this case. Before he fell ill, his general health was fairly good for his age, which was above 70 years, and he used to lead an active life in spite of his advanced age. The only flaw in his health was chronic constipation and dyspepsia. One day, he was too weak to leave his bed, being in a state of collapse, and subsequently for about a week, he remained very much in the same condition in spite of the best treatment. During this period, his blood pressure used to range between 70 to 80 (systolic) and 40 to 50 (diastolic) and he was sometimes pulseless with cold clammy sweats. Injections of Adrenalin, Atropine, Strychnine, Cardiazol, Camphor-in-oil, Ether etc. had to be given frequently, while Brandy and other stimulants were administered by mouth. His bowels were moved by gentle laxatives or enemata. His condition was causing grave anxiety and he was almost given up. As I had seen several cases of collapse caused by intense toxæmia from the intestines, I suggested to my friend that salines might be given a trial, but he and

other doctors regarded it as dangerous in view of the serious condition of the patient.

After about a fortnight, the patient changed the treatment as the scientific system had failed to give him any relief and as he had greater faith in the Unani system. As is usual with Hakims, who treat most cases by beginning with purgatives, the Hakim who was now called in also began his treatment with a strong purgative, and contrary to the expectation of others, the patient's condition became rapidly better and, by the evening of the same day, he felt stronger. On the day following the purgative, I went to enquire after his health and found him cheerful, cursing the doctors. His pulse was regular, stronger, with better tension and volume, and its rate was 80 per minute. He regarded the sphygmomanometer as a humbug, and it required some persuasion before he agreed to allow me to take his blood pressure which was 122 systolic and 80 diastolic. The Hakim kept his bowels open giving him at the same time some digestives and carminatives. The improvement in his condition was uninterrupted, and in about a week's time, he had resumed his usual life.

To the same category belong the following two cases which I select at random, having seen over a dozen cases of this type. In every case, every possible care was taken to exclude heart disease and some cases have been followed up for some years.

A young man of about 25, of good physique and previously quite healthy, had during the winter of 1938, a few motions due perhaps to some unusual article of diet or due to some indiscretion. He was in a collapsed condition with weak and scarcely perceptible pulse, with cold extremities and cold sweats. A dose of Seidlitz powder and a carminative mixture containing Spt. Ammon. Aromat. and some intestinal antiseptics were prescribed. After some 6 hours, he was reported to have had a few more motions and was much better and stronger. His bowels were kept open with one or two doses of Mist. Alba, and he was allowed gradually to return to his normal diet. After another two days, no further treatment was thought necessary.

I have come across similar cases and when treated with stimulants only they drag on for a long time, till Nature asserts itself, changes the chemistry of the bowels and eliminates or neutralises the poison.

A lady of about 30 years of age had been suffering for a week from an acute exacerbation of a chronic *B. coli* infection of the urinary tract. The most serious aspect of her illness was her general condition which was causing anxiety to her medical attendants. She had become very weak and her pulse rate used to range between 125 and 145 and the blood pressure between 80 and 100, and Cardiazol, Coramin, Veritol and other stimulants had to be administered whenever her pulse became feeble or perceptible, or when she had an attack of collapse with scarcely perceptible pulse, cold sweats and cold extremities, and this happened several times in 24 hours. Her bowels were kept open by lubricants only. Her general condition to my mind could not be explained only by the acute phase of a chronic infection and some other cause, besides the *B. coli* infection, was also at work. I attributed her extreme weakness to the abnormal condition of her colon and was prepared to give salines, but the other doctors who were also attending on her did not see eye to eye with me on this point. The case dragged on till, on the 8th day of her illness, Nature

came to our rescue. Unexpectedly and without any aid, she had 14 motions within 6 or 7 hours, and a good quantity of the faecal matter was eliminated. These motions changed the whole picture. The pulse rate fell to 110 to 116 and her blood pressure rose to 110 (systolic). The restlessness had passed off and so had the attacks of collapse, never to recur subsequently in the course of that illness. The change was dramatic and left the medical attendants to deal with an uncomplicated case of *B. coli* infection without any further need of rushing with stimulants to fight out attacks of collapse. Her fever and urinary troubles were relieved under proper treatment in due course of time, and she made an uninterrupted and uneventful recovery.

I have purposely avoided taking notes of cases occurring during the cholera season, as the collapse could be attributed to specific toxæmia.

II. Cases of hypotonia due to intestinal toxæmia.—Another common manifestation of acute toxæmia, originating in the bowels, is giddiness. Slight movement of the head causes giddiness which may be so severe as to cause fainting. Patients attempting to move about often fall and injure themselves. The trouble starts almost suddenly, and gradually becomes worse unless treated properly. There may be some headache and blurred or coloured vision. These cases go on for days and sometimes for weeks till their bodies rectify the defect. If suitable treatment is given, they recover quickly. The cause of giddiness, the most prominent symptom, is the fall in the blood pressure combined with perhaps the action of the toxins, whatever they may be, on the nervous system.

Examination in these cases reveals a weak, slow pulse, low blood pressure which is invariable, and general depression. There is no abdominal discomfort and generally no constipation to direct one's attention to the real cause. A lady of about 40 years of age, one morning, felt giddy and soon was confined to bed. The giddiness became worse, making movement difficult and dangerous. On attempting to move about, she fell down several times and injured herself. On examination, her pulse was slow (64 per minute) weak and low-tensioned, and her blood pressure was 96 and 40 (her usual pressure in health used to be 125 and 80). She was given two Stearn Bicolates at night and a double dose of Seidlitz powder in the morning with a carminative mixture every 4 hours. Her diet was restricted to liquids and she was encouraged to drink plenty of plain and aerated water. With each motion she felt better and after 8 motions, she felt completely relieved. Her pulse was stronger and blood pressure 120/76 in spite of the action of the bowels and the semi-starvation.

This case has been cited as an example. Such cases are common, especially during the hot and rainy season. There is no complication and they all end in recovery.

III. Summer toxæmia.—Another type of patients is to be seen during the summer and rainy seasons. These patients feel lethargic, listless and irritable and complain of lack of concentration and of general weakness. The appetite is impaired, they eat less and drink more water than usual. Generally, there is some constipation. The pulse is slow, weak and of a low tension. Naturally, the patients themselves and their doctors attribute all this to heat. They are both right and wrong. They are right in so far as the heat is the real cause and they are wrong since these symptoms are mainly due to the abnormal chemical changes occurring in the intestines

probably under the influence of the heat, and humidity. If the bowels are opened and kept open, it is astonishing how these very patients again become active, shake off their lethargy and irritability, can attend to their work better, have better appetite and do not feel as thirsty as they did before.

For the sake of convenience, I have classified these cases into three categories: (1) Cases of collapse. (2) Cases of hypotonia. (3) Cases of summer toxæmia.

The first two are certainly acute cases differing only in severity, and of these, only the first group is serious. In all cases of collapse, the possibility of previous heart disease was carefully excluded. The third group can be regarded as sub-acute. In all these cases, there is generally very little to direct the attention at once to the abdomen which can thus be overlooked. There may be some looseness of the bowels or mild constipation, but as a rule, both are so insignificant that even the patients fail to mention them. The prominent symptoms which attract the attention of both the physician and the patients are: the collapse, the giddiness, and the general lethargy in the three groups respectively. A quick-acting but mild purge, specially of the saline group with some intestinal antiseptic, brings about rapid and surprising cure within a short time. In the cases of collapse at the outset, stimulants have got to be given in order to support the strength of the patient till the toxins have been eliminated. But stimulants alone do not cure these cases. Symptomatic treatment, on the other hand, unnecessarily prolongs the trouble till Nature comes to the rescue or the accidental administration of any opening drug cuts short the disease. How and why some strong poison or poisons are formed and only in a limited few, while others escape this catastrophe, and what the nature of these poisons is, are questions which are impossible to answer at present. Is it not possible that the substances resulting from the defective chemical changes in the intestines are more universal and only a few are susceptible to it as happens in allergic conditions? Whether it is due to the mischief of a particular micro-organism suddenly becoming active or whether it is only due to the chemical changes proceeding on abnormal lines, I do not know.

In passing, it is interesting to note that, during the course of various diseases which we are called upon to treat, constipation affects their course and severity adversely and its relief alleviates the discomforts of the patients and reduces the severity. Here, naturally, we think that it is the elimination of the toxins peculiar to the disease which produces the salutary effect. But is it not possible that the chemistry of the bowels too is so modified by the disease as to result in the formation of some non-specific toxin as well? In some cases *e.g.* malaria, lung affections, many ordinary *E. N. T.* troubles and many surgical cases etc., the relief after bowel action is more than can be accounted for by the mere elimination of the specific toxins alone.

Role of Liver in The Treatment of Blackwater Fever

BY

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A CASE of Blackwater fever came under my observation and treatment a few days back. I adopted the routine treatment but supplemented it with Campolon injections, 2 c.c. every day for 4 days, during the course of the disease. As the first injection was followed by marvellous results in the general condition of the patient, and stopped his uncontrollable vomiting, I became very much encouraged and continued the injections.

History.—A patient named R., aged 16 years, male, Xian, belonging to Coovercolly Estate, had a sudden attack of chill and fever on the 10th night. He was seen in the morning by about 9 a.m. on 11-7-'41, when the temperature was 106°, and pulse rate 140. The patient was complaining of severe headache and backache especially in the loins. He told me that there was a slight difference in the colour of his urine. Next sample of urine was collected and examined. It was portwine in colour, and on further examination, revealed the presence of haemoglobin. There was incessant retching and vomiting. The colour of the vomited material was bright yellow and suggested that it was nothing but bile. The motion was semi-solid and coloured pale yellow. On account of the severe vomiting, the patient was unable to retain even sips of glucose water, soda water, and alkaline mixtures. Usual remedies for vomiting like Rectified Iodine and alkalies were of no use. The patient became very weak, emaciated and jaundiced. The conjunctivae, skin, and mucous membranes were yellow. Spleen was found enlarged about 4 inches below the costal margin and liver about 1 inch. Tenderness was elicited over the liver and splenic regions on gentle palpation.

Previous history.—The patient had suffered from an attack of small-pox in his childhood. He had suffered from a previous attack of blackwater fever and had then been treated successfully here alone. Last time, the patient had the disease by the end of June, 1940, and this time, in the early part of July, 1941. So, there seems to be some definite relationship between the season and the disease.

Treatment adopted.—Patient was shifted to the hospital and kept warm. Warm soapwater enema was given to empty the lower bowels. One pint of warm rectal saline with Sodibicarb and glucose was given every 4 hours. Sips of glucose water with Sodibicarb and sodawater were given. Sips of barley water also were given, but all fluids taken by mouth were brought out.

Medicines:—Plasmoquin Co. containing Plasmoquin 1/6 gr. and Quinine 2 grs. tablets were given—2 tablets three a day for the first 3 days and 2 tablets twice a day for 2 more days. The temperature was gradually coming down by about half a degree every day. On the 2nd day, the patient was very bad, the heart showing signs of failure. The urine passed was very small in quantity and the vomiting very severe. Injections of Strychnine Digitalin were given every day and stimulant mixture every sixth hour. I gave one Campolon injection 2 c.c. at about 4 p.m. on the 2nd day. By about 10 p.m., the vomiting completely stopped all on a

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ANTERIOR CRURAL NEURITIS—K. B. SARMA

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sudden and the patient was able to retain medicines and fluids administered orally. His general condition also showed very much improvement. The patient spent a better night after the injection. Next day morning, the temperature came to 102° but the colour and quantity of urine passed remained the same. I continued the Campolon injections for 3 more days until the temperature touched normal. Blands pills (5 grs.), 2 pills twice a day, were given from the second day and even now continued.

The urine cleared up completely on the 5th day and the patient was passing larger quantities.

Salient features of the case.—(1) Relapse. (2) Beneficial effect of liver in controlling the severe vomiting and improving the general condition of the patient.

I request my learned friends to try Campolon or any other liver therapy during the acute stage in cases of blackwater fever and observe the results.

QUESTION:—Whether the beneficial results I observed after the Campolon injection were actually due to the liver or due to the natural waning of the disease process?

Anterior Crural Neuritis

BY

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ACUTE anterior crural neuritis may be the result of septic absorption, chill, gout, or it may be part of the picture of a multiple neuritis, as in diabetes or alcoholism.

This is comparatively rare but is usually mistaken for sciatica. The distribution of the pain is more over the front of the thigh to the knee, though it resembles sciatica in usually commencing around the back of the hip. The pain is apt to be paroxysmal and may be intensely severe probably the more so than is usual in sciatica. The knee-jerk may be diminished or lost, just as in a proportion of cases of sciatica, the ankle-jerk is lost. Wasting of the quadriceps is met with only in chronic cases, as in diabetic neuritis and in gross lesions of the nerve as when damaged by pelvic growths.

The treatment of this form of neuritis will be on general lines by Anodyne liniments, fomentations, or Antiphlogistine packs, rest in bed and internally Salicylates or Aspirin, with combinations of Pyramidon or Phenazone with Codein or Heroin and Medinal. Cachets of Pyramidon, Heroin and Medinal may be used to relieve the pain at night and to induce sleep, and in the most severe cases, injections of Morphia or Heroin may be necessary.

An Interesting Case of Menorrhagia

BY

DR. (MISS) JIWANLATA

Resident Superintendent, Mrs. Girdhar Lal Maternity Hospital, New Delhi.

MRS. K., aged 33 years, Hindu, was admitted for severe bleeding P. V. for about four weeks.

History of present illness.—The patient got her period normally but it did not stop and bleeding became very profuse, she passing a number of clots at times. She also had pain and attacks of faintness off and on. Periods were regular otherwise, but she had two such attacks, one about seven years back and the other three years back, but those two attacks lasted only for a few days and were easily controlled by treatment, especially injection of Ergotinine citrate.

Children—Nil; **Abortion**—one, 14 years ago; **Premature**—one about 11 years ago; **Not pregnant** since then.

General condition.—Slightly anæmic. Very nervous and depressed: otherwise nothing noteworthy. P. V.—Uterus retroverted. Slightly movable, otherwise n. a. d.

Treatment.—The patient was given a mixture containing Calcium chloride, Tr. Hydrastis and Ergot. She was also given Cal. Lactate gr. xxx every day. In addition, she was given injection of Ergotinine Cit. gr. $\frac{1}{100}$ and Inj. Calcium Gluconate 10 c.c. intravenously. A number of injections were given. Locally, she was given hot douches between 100° to 110° temperature 3 to 4 times a day, but with no improvement. I then did dilatation and curetting. There was some improvement as the bleeding became much less and for a day it stopped, but recurred again. The above treatment was repeated but no appreciable improvement was observed. She was given additional treatment of Sistomensin tablets, Aletris Elexis (Alembic) Uteron (B.C.P.W.) one injection of Stypticin, Inj. Ergometrin Tartarate, Inj. Pituitrin. Inj. Hæmoplastic serum, Erbolin capsules, and so on, but the condition persisted. Of course, she was given large doses of Iron (Ferri et Ammon. Cit. in mixture) in addition.

At last, I thought of trying iced douche. It was given twice daily, and for the first 12 hours, she was asked to use ice bag for the lower abdomen. The next day, there was a remarkable change, the bleeding becoming less. Second day, it stopped for 12 hours and third day it stopped completely. Iced douches were continued for three days more and now the patient is completely free from the trouble after suffering for two months.

The case is of interest as I have not come across iced douches being given for any trouble. In this case, I feel sure that if it was used earlier, it would have saved me and the patient so much of anxiety, but as I had not heard of this form of treatment, I took long to try it. I had even suggested hysterectomy. I do not know why it should not be used oftener. I can only think of two drawbacks, one perhaps fear of introducing sepsis and the other producing shock. To avoid the former, one can use boiled water brought to zero temperature in a refrigerator and to avoid the second trouble, one can use it cautiously and little first, and if there is no shock, about two pints of it can be easily given.

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Editorial

Recruitment to the I. M. D.

IF the conditions and terms of recruitment of the medical graduates to the Emergency I. M. S., on which we made our observations in the last issue of the Antiseptic, are unsatisfactory and require suitable modifications to make them acceptable to the profession, much more so are they in the case of recruitment of licentiates to the Emergency I. M. D. The Working Committee of the Indian Medical Association, which met at Calcutta on 12th July 1941, is therefore correct in stating that "a mere appeal to the patriotic sentiments of medical men to accept either an Emergency Commission in the I. M. S. or an emergency appointment in the I. M. D. would be ineffective unless fundamental changes are made and the terms and conditions of emergency services now offered are materially altered".

What the Government offer to the licentiate joining the Emergency I. M. D. is very inadequate and unsatisfactory. Starting as a Jamedar on Rs. 75 with a Civil allowance of Rs. 50 per month, he may, if the emergency lasts for 25 years, ultimately become a Subedar on Rs. 160! It does not require much effort to see that his allotted status and position in the Army does not befit his education as a medical man, nor is his remuneration such as would maintain his family at home in reasonable comfort and security.

Even at the very commencement of the present war, the All India Medical Licentiates' Association took up this matter seriously and the results of its two years of efforts are the granting of Civil allowance of Rs. 50 and the change of designation to Assistant Surgeon. But the Association's main demands remain yet to be fulfilled, and they are, in our view, neither excessive nor exaggerated. They are stated very clearly in its Standing Committee's Resolution dated 15th July 1940:—

- (1) "That the status and emoluments offered to the medical licentiates have been reported upon by the regional Branches of the Association as utterly unattractive and unacceptable and even fall far short of the terms offered during the last war of 1914-18"
- (2) "That in view of such an opinion from the Branches, this Committee considers that the present terms of recruitment are so

meagre and unsatisfactory as would not evoke any response from the medical licentiates in as much as their present educational standards are more or less equivalent to the medical graduates who are offered the King's Commission in the I. M. S., with fair emoluments, and as their practice will be left unsecured and unprotected during their absence on military duty".

(3) "This Committee therefore urges upon the Government the necessity of instituting Military Assistant Surgeon Branch of the Indian Section of the Indian Army, for the purpose of allotting to medical licentiate recruits under 10 years of service or practice on a graded scale of pay of Rs. 250-10-450, leading to the conferment of King's Commission."

(4) "In the case of licentiates of the Standing of 10 years and over in the medical register, in service or in private practice, who may be called for military duty, they may be directly recruited to the temporary I.M.S. cadre (Emergency Branch) with King's Commission in consideration of the experience they have already acquired."

The resolution of the Central Council of the Indian Medical Association also likewise demanded that the licentiates in the I.M.D. should start with a rank and seniority in consonance with their experience and standing in the profession and that 10% of them shall be granted the honorary King's Commission.

The Government would be well advised not to flout this unanimous opinion of the organised profession, particularly in view of the fact that its demands, as we have said earlier, are neither excessive nor exaggerated. What the profession earnestly seeks is that the licentiates should be put on par with Military Assistant Surgeons with regard to their status, emoluments and prospects in the Indian Army. As the Government themselves observe: "The present-day Sub-Assistant surgeon is a well-educated and qualified man and there is no reason to retain the title of *Sub-Assistant Surgeon* which might suggest that he holds the same subordinate status as his original predecessor". But a mere change of designation without corresponding changes in terms and conditions of service is monstrously futile, and unless the Government see the wisdom of avoiding such proposals of varying degrees of barrenness, and of implementing in their stead, the minimum demands of the licentiates, their response to appeals to volunteer for military service would indeed be very poor, as we understand it is so already.

Of course, the Government may be within their rights if they tap the strength of the subordinate medical service which is relatively greater than the Provincial Medical Service, the ratio being 6:1, but that does not take away the need for initiating fair and equitable terms, conditions and status, appropriate to the present standing of the licentiates in the body politic of the profession. The Government must realise that the medical licentiates in private practice hold a great stake in the profession and they are not likely to barter it for a position which neither adds to their dignity nor enhances their future prospects.

Gleanings From Medical Press

Medicine and Therapeutics

Anaemias and Their Treatment.—By J. R. Davidson, M.D., Neyyoor. It has been allotted to me only to introduce this subject of 'Anaemias and their treatment', and I hope that all have come prepared to add to the discussion.

The subject of physiology is one that I feel sure ought to take more of our attention than perhaps it does. So often we find ourselves dealing with slight errors and anomalies of physiology rather than pathological conditions and many of the anaemias fall into this type of disease. I would therefore like to call your attention to a modification of a diagram that appeared in the *American Handbook of Medicine, 1938*, showing the normal physiology of blood production and destruction.

We will see illustrated the three main factors in the production of blood, and deficiency of any of these will give rise to anaemia. Nutritional deficiency due to a deficiency of any one of the food factors, iron

B. Antipernicious anaemic factor. 1. Addisonian. Pernicious anaemia. 2. P.A. of pregnancy. 3. Macrocytic anaemia of sprue, idiopathic steatorrhoea, dithioriocephalus infection, etc. 4. Tropical macrocytic anaemia. 5. Macrocytic anaemia of liver disease.

C. Vitamin C.

D. Thyroxin. Myxoedema. 2. Due to excessive blood destruction.

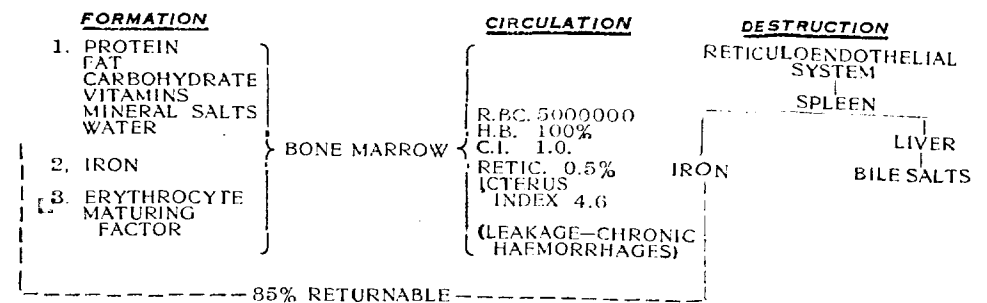
A. Primary haemolytic anaemia. Familial and acquired acholuric jaundice.

B. Secondary haemolytic anaemia. Paroxysmal haemoglobinuria. Acute haemolytic anaemia of Lederer. Sick-cell anaemia. Haemolytic anaemia of infancy. 3. Aplasia of the bone marrow.

A. Idiopathic.

B. Secondary causes; heavy metals, Benzol group, poisonous gases, radio-active materials, (anaemia of blood donors).

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deficiency or the specific type due to a deficiency or absence of the anti-anaemic or erythrocyte maturing factor. It may be due to an insufficient supply or to inefficient absorption.

In the circulation we must not forget the possibility of leakages causing anaemia such as bleeding haemorrhoids, and excessive menstrual loss. If also the normal destruction is wrong then again we will see different types of anaemia resulting. The advantage of keeping such a diagram uppermost in one's mind is that we are not then so likely to place all our cases of anaemia into one category and not think of the underlying cause. The following full classification is according to the deficiency that is the cause of the anaemia:

Classification of Anaemias:—1. Anaemias due to deficiency of factors essential to normal blood formation.

A. Iron. 1. Chronic nutritional hypochromic anaemia. Chlorosis, and hypochromic anaemia of pregnancy. 2. Nutritional hypochromic anaemia of infancy 3. Post haemorrhagic anaemia.

Miscellaneous.—1. Splenic anaemia (Banti's disease).

2. Polycythaemia. (a) Polycythaemia vera. (b) Enterogenous cyanosis.

3. Haemorrhagic diseases. (a) Purpuras. (i) Primary; (ii) Secondary. (b) Haemophilia.

4. Leukaemias.

5. Lymphatic diseases—Hodgkin's disease Lymphosarcoma.

6. Agranulocytic angina.

7. Infectious mononucleosis.

8. Reticulo-endotheliosis Gaucher's disease. Niemann Pick's disease. Hand-Schuller-Christian disease.

The diagnosis of the different types of anaemia is usually fairly easy and the important guides are the history and the laboratory findings, a complete blood examination and a urine and stool examination.

There are two blood examinations that are of special aid in the diagnosis.

First the size of the erythrocytes, remembering that the normal size is between

5.5 μ and 8.0 μ with an average at about 7.7 μ . The average size of the red blood cells in the bone marrow is about 7 μ —10 μ and even up to 15 μ .

The second which is much more reliable and easy to perform is the reticulocyte count.

TREATMENT.—As regards treatment I intend to deal only with iron deficiency anaemia which is found to some degree in most cases.

The normal iron need of an individual is approximately ten m.g.m. daily, but the need varies according to the metabolic rate and in women according to the menstrual loss and with pregnancy. An adequate supply of iron is normally found in an adequate diet, which as already shown, is an essential in the proper formation of blood. Ten m.g.m. of iron would normally be found contained in a diet with a quantity of green vegetables. Under normal conditions this is enough for the need of the individual but may become inadequate if there is excessive menstrual loss or during the added strain of pregnancy the supply may not be adequate to the requirements.

The first step therefore in the treatment of anaemia in women (and it is in women that one sees the majority of the chronic deficiency anaemias) is to attempt prophylaxis by seeing that the patient has an adequate diet and correcting any gynaecological faults either organic or endocrine in origin.

During pregnancy an attempt may be made to increase the amount of the iron-containing foods and of the general nutrition of the diet.

For the general cases education of the public to an appreciation of a more balanced diet is most important.

CURATIVE MEASURES.—*Treat the cause first.*

GENERAL MEASURES.—1. *Rest.* During the period of treatment for anaemia the patient must rest in bed. He may be allowed up to attend to the calls of nature but otherwise he should be strictly in bed.

2. The often forgotten *symptomatic treatment* that makes the patient feel better during the time that the blood regeneration is taking place and makes him feel that something more is being done for his condition.

(a) **GASTRO-INTESTINAL SYMPTOMS;** the main being ANOREXIA. Most of the patients have an achlorhydria and therefore are given hydrochloric acid in some form

Acid Hydrochlor dil. oz. 1.
Glycerin Pepsin B.P.C. oz. 2.
dr. 1 during meals.

Many have an excessive mucous secretion in the stomach which may give rise to the achlorhydria, and an alkaline powder given after meals may prove beneficial:

Sod. Bic.
Bis. Carb.
Mag. Carb. a.a. oz. 2.
Sig. dr. 1. t.i.d. p.c. ex. aq.

Constipation may be troublesome and the best treatment is Mineral oil with Cascara, Agarol, Petrolagar.

Vitamin B given as Bemax or Marmite as a medicine.

Two or three Yeast tablets daily.

Note that in general experience the modern massive dosage of iron gives rise to diarrhoea rather than to constipation.

Glossitis sometimes needs treatment as a symptom but in the ordinary anaemias there is seldom need to give any special treatment.

Two and a half per cent chromic acid in water (10 gr. to the oz.) is a good local application.

Dysphagia needs no definite treatment. Assurance that it will go as the anaemia gets better.

(b) **NERVOUS SYMPTOMS.** For the various neuritic symptoms give vitamin B, in form of Marmite or Bemax or Yeast. In the very severe cases injection of vitamin B daily. For the anxiety state that so often is present due to family worries: Chloral and Bromide aa gr. 15. m. and n. or Phenobarbitone gr. $\frac{1}{2}$ l. m. and n.

SPECIFIC IRON THERAPY.—Iron salts are used in many tonics but the amount of iron in such is usually of a negligible quantity.

There are five main compounds of iron used: (1) Metallic iron. (2) Ferric salts. (3) Ferrous salts. (4) Inorganic salts with complexions. (5) Organic iron combinations.

Soluble iron salts give rise to certain difficulties as unpleasant taste, stain the teeth, irritate the stomach and act as astringents causing constipation and colic.

The ferric salts are most likely to cause these undesirable actions and on the whole are ineffective in the treatment of anaemia. The last group of organic combinations are very pleasant to take but are useless in the amount of iron they supply and are also not effective in the treatment of severe anaemia.

Reduced iron is effective but must be given in large doses.

The soluble ferrous salts are the most effective but have the big disadvantage of being very rapidly oxidized to the ferric state. Therefore the insoluble ferrous salts are of more use and the most popular of them is Blaud's pill or the iron carbonate and also the saccharated iron carbonate. The disadvantage of these preparations is again the tendency for them to oxidize and become so hard that they pass through the alimentary canal unchanged.

The scale preparations are therefore under all circumstances the most reliable.

Parenterally iron preparations are not of much value as it is difficult to give large doses without giving rise to toxic symptoms.

Main success of treatment is to give any suitable preparation in sufficiently large dose.

60—90 gr. of Ferri et Ammon Cit. daily is as good as any.

The standard of cure should be to aim at getting a rise of at least 1% of haemoglobin for each day of treatment.

Perhaps a more recent line is the addition of some copper and manganese to the iron preparation as catalysts and one does get effective results from the compound pills of Ferrous Sulphate as made by Glaxo.

Other preparations are:

Ferri et Ammon Cit. gr. 30 t.i.d.
Ferrous Sulphate Co., tab. 1 t.i.d.
Ferrous Carbonate (bipalatinoids).
Ferrous Sulphate with Yeast.
Tab Ferrous Chloride.

Colliron.—*Journal of Christian Medical Association.*

The Pain of Peptic Ulcer.—By C. ALLAN BIRCH, M.D., M.R.C.P., D.C.H., D.P.H.. The fact that pain is a common and usual symptom of gastric and duodenal ulceration is taken for granted. But when we inquire into its causes we find that even to-day they are improperly understood. A brief consideration of the observations on which the theories of pain causation are based may help us to obtain greater insight into the mechanism of ulcer pain.

The two most popular theories are that pain is caused by hydrochloric acid or that excessive contractions of the stomach cause it. There is evidence for both these views and while it is possible that both play a part it must be remembered that other factors help to determine whether any given patient feels pain or not. The stomachs and nervous systems of individuals vary in sensitiveness and this may vary in the same patient from time to time.

Ulcer symptoms come and go even when the ulcer is still demonstrable and there is no close relation between a recurrence of pain and increase of acidity of the stomach. Ulcers of the stomach have been shown to be at times insensitive to acid introduced by a stomach tube. Hence the patient or his ulcer must at times become insensitive. It is known that a patient is more sensitive to pain when his ulcer is troubling him.

Ulcer pain often ceases when a holiday begins or when a hæmatemesis puts the patient into hospital. A sudden fall in gastric acidity is not the reason for this. A sudden removal from a worrying environment may be the explanation. Some patients, however, worry more and feel their troubles more when, for example, their illness by removing them to hospital removes their livelihood.

Libman classified patients into hyper- and hyposensitives according to their response to pressure on the styloid process. A hyper-sensitive patient with an ulcer and taking a light diet may have more pain than a hyposensitive patient with an apparently identical ulcer taking a heavy diet. This

difference tends to be accentuated if a hyper-sensitive lives alone and is given to gloomy pondering. These generalisations must be borne in mind in the consideration of any given case.

Let us now discuss the acid and spasm theories. Because alkalies will rapidly relieve ulcer pain, it is often argued that acid must be the cause of the pain. Patients with achlorhydria, however, can develop ulcer with typical pain. These ulcers with achlorhydria are acute and superficial but the pain they produce and also that of syphilitic and some carcinomatous ulcers associated with achlorhydria can be relieved by alkalies.

But an amount of alkali insufficient to neutralise the acidity of the gastric contents will often relieve the pain and a small amount of food can act in a similar way. Sippy said: "The pain of ulcer may be entirely absent when the aspirated stomach contents show even high grades of free hydrochloric acid."

Powerful cramp-like spasms of contractions of the stomach are thought to cause pain. Hunger pains are thought to be due to such contractions and these can certainly occur in patients with achlorhydria and no ulcer. But these contraction waves are rhythmic and ulcer pain is steady. Also the powerful contraction waves occurring during digestion are not painful. Recording balloons in the stomachs of ulcer patients sometimes show great activity without pain and also pain occurs without much activity. The degree of muscular activity of the stomach has something to do with pain. The hunger type of ulcer pain tends to come on at 11 A.M. and 4 P.M., which are the peak periods of increased irritability of the whole gastro-intestinal tract. Ulcer pain coming on later in the day may be accentuated by the fact that the patient is tired.

In some patients with typical ulcer symptoms no lesion of the mucous membrane can be shown by any method. It is thought that in these "pseudo-ulcer" cases acid irritates the mucosa until it becomes red and sore, though not ulcerated. It has been shown that dilute hydrochloric acid trickling over the gastric and duodenal mucosa of dogs will gradually exhaust its defensive power, so that redness and ulceration finally result. This explains why if food is taken soon after pain starts it is more likely to relieve it than if it is taken after an interval.

Peptic ulcer may cause a dull aching pain between the scapulae. This does not necessarily mean that the ulcer is adherent to the pancreas or other structures. It is probably a referred pain.

When all the evidence is summed up it seems that in most cases pain, particularly of the steady recurring type, is due to irritation by hydrochloric acid. Intermittent and rhythmic pain may be due to contraction waves pulling on the base of the ulcer. Waves of contraction and pyloric spasm may

also cause pain by interfering with emptying and thereby increasing the acidity of the gastric contents.

The fact that many measures which relieve ulcer pain all diminish acidity points to acid being the cause of the pain. Thus emptying the stomach relieves pain and if gastric contents are neutralised and reintroduced pain does not occur. Histamine by increasing the secretion of gastric juice may cause pain. Another point in favour of the acid theory is the fact that pulling on the base of a duodenal ulcer at operation under local anaesthesia will cause pain which can be relieved by the injection of alkali into the duodenum.

The stopping of ulcer pain should not be an end in itself since the absence of pain does not mean that the ulcer is healed. Nevertheless when pain is severe, measures to counteract it, although part of the general treatment of ulcer may have to be accentuated.

These are:—(1) *Alkalies*.—The soluble ones, e.g., sodium bicarbonate, give the most prompt relief, but have the disadvantage of causing a secondary rise of acid secretion. Prolonged dosage may produce alkalosis. Magnesium trisilicate, which has a prolonged physical rather than a chemical action, is preferable for general use. It does not cause alkalosis. A good plan is to use it at night and to combine it with soluble alkalies for use during the day.

(2) *Milk*.—This neutralises its own volume of decinormal hydrochloric acid. In a patient with a rapidly emptying stomach its effect may soon be lost and it is wise to combine it with biscuits or take it as a thickened drink.

(3) *Atropine* in doses of gr. 1/100 is helpful where spasm is the cause of pain.

(4) *Phenobarbitone*, gr. ½ in the morning and gr. 1 at night, helps to cut down the sensitivity of the patient to his pain.—(*Medical World*).

Fever therapy in diseases of the nervous system, Samuel H. Epstein and Harry C. Solomon. M. Clin. North America, Philadelphia, 24: 1555-1572, Sept. 1940.

In neurosyphilis the three outstanding factors relating to the mechanism of artificial fever therapy are: (1) The destruction of invading spirochetes by elevation of body temperatures; (2) the biologic response to nonspecific foreign protein shock; and (3) the alteration produced in the brain parenchyma by the malarial organism.

Malaria is well established throughout the world as standard treatment for parietic neurosyphilis. Since 1931 electropyraxia has been used. Comparative evaluation of the two methods is difficult because there are hardly two groups of cases in literature that are comparable with each other. Electropyraxia is indicated especially in the Negro race, many of whom are immune to malaria.

Cardiac and renal diseases are contraindications to the use of malaria. The authors stress the value of the combination of tryparsamide with fever therapy. They say that tryparsamide is capable of producing beneficial results similar to those produced by malarial therapy and that a considerable number of preliminary tryparsamide injections greatly enhances the possibility of therapeutic success in the treatment of parietic neurosyphilis.

The therapeutic effect of fever upon the course of tabes dorsalis is not so striking. Serologic reversal is frequently accomplished but clinical cures are not readily forthcoming. In the authors' experience tabetics with cord bladders are prone to develop serious complications due to infection. Some relief in patients with gastric crises has been obtained. In optic atrophy malaria is of distinct value in arresting the progress of visual loss.

In early meningeal syphilis, neither tryparsamide nor fever therapy is advisable at the start of treatment, but may be used after 2 or 3 years of treatment in patients with resistant spinal fluid serology. Only about a third of the patients with vascular neurosyphilis show good results. Malaria is contraindicated on account of the deleterious effects upon the circulatory system.

Accumulated clinical evidence indicates that fever therapy adequately prevents clinical neurosyphilis. Patients who receive adequate chemotherapy and later malaria treatment showed no clinical progression. Therefore in recent years, mechanical hyperpyrexia has been employed in asymptomatic neurosyphilis. The authors recommend, as the best method of treatment of resistant neurosyphilis, combined artificial fever therapy and chemotherapy.

The results of fever therapy in cases of juvenile paresis are not so favourable as in the acquired disease. The authors' experience with malaria in this disease has yielded unsatisfactory results, and this was true with electropyraxia also. In a series of 30 cases of juvenile paresis, 3 patients developed interstitial keratitis subsequent to fever treatment and while receiving arsenical treatment, and 4 patients of the series of 4 cases of nonparetic congenital neurosyphilis who were not given fever therapy, likewise developed interstitial keratitis. This would appear to be more than a coincidence. The hypothesis is so advanced that the febrile condition enhances the possibility of the appearance of an interstitial keratitis (a gummatous lesion) in a patient with an essentially parenchymatous disease.—V.D.I.

Treatment of Trichomoniasis in Males

—Although *Trichomonas vaginalis* infestation in males is thought to be comparatively rare and is certainly so when compared with the incidence of the condition in females, there is evidence to show that it is commoner than generally supposed. In many cases, no doubt the presence of the organism causes

symptoms, but routine examination of cases of chronic gleet would probably reveal trichomonads in a fair proportion. The protozoan, should it succeed in penetrating between the epithelial cells lining the urethra, may produce stricture and infection of the prostate and bladder; and kidneys may give rise to serious symptoms. Accurate diagnosis and a knowledge of the conditions which favour the growth of this protozoan are essential for successful treatment. Excellent work on this has been done by Liston, who found that the organism thrives best at a pH of about 5.5, but cannot long survive at a pH of 4—that of the normal vagina—or at one of 7 to 7.5. Early treatment should therefore be the aim, and an attempt should be made to render the urine neutral or slightly alkaline as quickly as possible. For this purpose the bicarbonates and citrates of potassium or sodium are most useful, and they should be persevered with because of the tendency to relapse if treatment is stopped too soon. On the other hand, it would appear, in theory at any rate, that mandelic acid therapy in the form of calcium mandelate might be successful, but it is doubtful whether it would be possible to keep the pH at a low enough level for a sufficient length of time to destroy all the organisms. Acriflavine, given orally in half-grain "enteric-coated" tablets, and sulphani-lamide have been tried with doubtful success. Various local remedies, including devegan or stovarsol, silver picrate, and negatol, have been used successfully in cases of vaginitis. Negatol may be employed in the male as a urethral irrigation or instillation in a strength of 1% to 2%. Experience has shown that routine irrigation with the usual antiseptics, such as potassium permanganate, is not only useless but may be positively harmful, though some workers employ mercury oxy-cyanide. Where the infestation is only superficial, maintenance of a urine pH of 7 to 7.5 will probably be successful, but when the submucous tissues are involved, local treatment is indicated. In all cases, failing to clear up with alkalis an endeavour should be made to locate lesions in the urethral canal. Infected follicles and submucous infiltrates, which if left untreated, might lead to stricture formation, should be dealt with by either massage over a straight sound or the use of Kollmann's dilator. In some instances it may be necessary to employ the cautery, which should always be followed by dilatation, as strictures are more easily prevented than cured. If symptoms of prostatitis occur, appropriate treatment consists in regular massage and diathermy. Cystitis has been treated successfully by daily irrigation with 1 in 1,000 nitrate of silver, and pyelitis by lavage of the pelvis of the kidneys with 2% mercurochrome; in both these conditions it is worth while keeping the urine alkaline. It cannot be too strongly stressed that success in treatment depends on starting it before the organisms have got out of reach beneath the mucous membrane, and on maintaining it for some time after all symptoms have subsided. Nor should it be forgotten that in the case of a married man the wife is almost

certainly infected also, and it is useless to treat the one without treating the other.—(B. M. J.)

Prolonged Jugular Compression; A Diagnostic Test of Neurologic Value.—R. B. AIRD (*Archives of Neurology and Psychiatry* 45:633, April 1941) reports the use of prolonged jugular compression as an aid to the diagnosis of neurologic lesions; such compression causes an exaggeration of the signs and symptoms of tumors and other space-consuming lesions of the brain and spinal cord but not in other types of lesion. The jugular compression is exerted by an ordinary sphygmomanometer cuff, folded lengthwise and wrapped about the patient's neck; a pressure of 40 mm. mercury, maintained for ten minutes, has been found to give the most satisfactory results; and is usually well tolerated by the patient. The usual neurologic examination should be made before the jugular compression is applied, and repeated during compression with special attention to the tests most likely to show changes. In 100 cases in which this test was done, positive results were obtained in a high percentage of cases of tumour of the brain and spinal cord and in herniation of the intervertebral disk; that is, in these cases, the jugular compression caused an exaggeration of some sign or symptom already present, or developed some new sign or symptom. In cases of degenerative diseases of the central nervous system, convulsive states of unknown origin and post traumatic syndrome, the test was negative. In many cases this test confirms the diagnosis made by other methods, but in certain cases in which the diagnosis was obscure, it "contributed valuable evidence" toward making the correct diagnosis or the localization of a tumour. In cases of brain tumour, the signs, most frequently exaggerated or newly elicited by jugular compression, were facial paralysis (of central origin), asymmetry of deep reflexes and visual field defects; in spinal cord tumor, sensory and reflex changes were chiefly affected by the jugular compression test; and in herniation of the intervertebral disk, pain and sensory changes (reflex changes less frequently).—(*Medical Times*).

Vitamins in Peptic Ulcer.—H. Field Jr., W. D. Robinson and D. Melnick (*Annals of Internal Medicine*, 14: 588, October 1940) note that evidence of vitamin C deficiency is frequent among peptic ulcer patients, such patients are apt to avoid the use of fruits and vegetables voluntarily, and the usual ulcer diet is definitely deficient in this vitamin. On the other hand, the characteristic pathology of vitamin C deficiency indicates that this deficiency might have "a deleterious effect" on the healing of peptic ulcer, and might be a definite factor in bleeding. Of 58 patients with peptic ulcer, in whom the plasma ascorbic acid values were determined, 39 showed values below 0.5 mg. per cent ("the beginning scurvy level" of Farmer and Abt). There were 8 patients with values above 0.7 mg

per cent; of these, 2 had been given supplements of synthetic ascorbic acid. Bleeding from the ulcer occurred in 12 of the 58 patients, either at the time of admission or while under treatment at the hospital; in 10 of these 12 cases the ascorbic acid values were below 0.40 mg. per cent; in 2 cases the bleeding occurred after the patients had been in the hospital on an ulcer regimen for eighteen and twenty-six days respectively; the ascorbic acid values in these cases were 0.30 and 0.25 mg. per cent. These findings indicate that in the treatment of peptic ulcer, the vitamin C deficiency should be corrected promptly; this is best done at the beginning of ulcer treatment by the injection of ascorbic acid in moderately large doses, "perhaps 200 mg. four times a day," until the plasma ascorbic acid is 0.9 mg. per cent or higher, or until 2 gm. of ascorbic acid has been given (if plasma values cannot be determined). Thereafter, 100 c.c. of orange juice or 50 mg. of ascorbic acid should be given daily by mouth. Other vitamins may also be deficient in the ulcer diet, but the vitamin C deficiency is evidently of the greatest significance.—*Medical Times*.

The Curative Treatment of Diphtheria.

—By G. E. DONOVAN, M.Sc., M.B., D.P.H., Medical Officer of Health for the Llŵchwr Urban and Gower Rural Districts.

The three essentials in the treatment of a case of diphtheria are: early adequate serum administration, rest in bed and skilled nursing.

SERUM:—The patient suspected to be suffering from diphtheria should be injected immediately with antitoxin. This should be done even before sending off the swab for bacteriological confirmation. When the clinical picture is typical, negative bacteriological findings should be ignored. The case mortality of the disease is progressively doubled for every 24 hours delay in instituting serum treatment. The total dose of antitoxin considered necessary for the case should be given at once, not as a series of relatively small doses. In severe cases half the serum should be given intravenously and the other half intramuscularly. If the initial dose has proved inadequate as shown by the spread of membrane, a second injection should be given. The best site for intravenous injection in an adult is into one of the veins of the antecubital fossa, or in the case of a child, into the internal saphenous or internal jugular vein. The most convenient site for intramuscular injection is the middle third of the outer side of the thigh. Subcutaneous administration should never be employed. The smallest therapeutic dose of antitoxin is 4,000 units; this may be adequate for a mild case with small scattered patches confined to the tonsils of not more than 48 hours duration. If both tonsils are completely covered with a patch of membrane, 8,000 units would be considered appropriate. If the faucial pillars are also involved, 10,000 units would be the minimum, but if the membrane has spread

on to the soft palate, not less than 20,000 units should be given. If the membrane spreads to the base of the uvula, 30,000 units is the minimum, and if the uvula is also involved, 40,000 units. Should the membrane have extended to the nasopharynx and a nasal discharge be present, 60,000 units should be given. If the disease be of longer than 48 hours duration, when first seen, these doses should be substantially increased. Cases of gravis diphtheria and so-called "bull neck" should be given a minimum total dosage of 120,000 units. In such very severe cases, the dosages could be as high as 220,000 units, half of which is given intravenously and half intramuscularly. Children require a dose almost similar to adults; under two years, the dosage could be two-thirds of the adult dose. A practitioner should rather over estimate the severity in giving antitoxin. A dose of 10,000 units is sufficient for anterior nasal diphtheria and when the larynx alone is involved, a dose of 20,000 units is adequate. Those with nasal or laryngeal diphtheria combined with faucial lesions require larger doses. Diphtheria confined to the conjunctiva, genitals or wounds, is relatively non-toxic, and a dose of 8,000 units is generally sufficient. The desired result after the administration of antitoxin is general improvement and shrivelling of the membrane, commencing from 12 to 24 hours.

Concentrated anti-toxin prepared by precipitation methods contains about 2,500 units per c.c. Serum prepared by enzyme disaggregation, followed by differential heating results in a smaller antitoxin globulin molecule with increase in concentration, say 6,000 units per c.c. so that the volume and protein content of the therapeutic dose is much reduced. O'Meara of Dublin (*The Lancet*, Feb. 15, 1941, p. 205) believes that commercial serums lose some of their potency for the hypertoxic type of diphtheria, which is generally due to the gravis strain, by their method of preparation. He claims special properties for a serum which was clinically tried at Cork Street Hospital by McSweeney (*The Lancet*, Feb. 15, 1941, p. 208) on hypertoxic cases, and their published results seem to be very good. Their serum is not commercially available and cannot be purchased by the medical man who wishes to try it.

COMBINATION OF BLOOD TRANSFUSION WITH COMMERCIAL ANTITOXIN:—This treatment was first suggested by me in *The Lancet* (March 1, 1941, p. 293). It is based on the theory that the average adult is immune to the type of diphtheria prevalent in his district. His blood contains specific antibodies for the disease and not for just one factor of it. A transfusion from such an adult would also benefit the patient by introducing healthy blood to his own, and diluting his circulating toxins. Hypertoxic cases are given 60,000 units of commercial serum intramuscularly and 60,000 to 100,000 units intravenously. The blood is given by the drip method and need not be heated. The average quantity injected is one pint, but this

depends on many factors such as age, etc. The results are eminently satisfactory and improvement is noted very quickly in the case.

UNDESIRABLE AFTER-EFFECTS OF SERUM:—These are mainly anaphylaxis and serum rash or serum disease.

Preventive Treatment. The practitioner should in the routine case put the following questions to the patient before giving serum.

1. Do you sneeze, cough or develop asthmatic symptoms whilst in the neighbourhood of horses?
2. Do you suffer from asthma, hayfever, or attacks of nettle rash?
3. Have you received previous injections of serum?
4. Did you suffer from serum sickness?

If the answer to these questions is in the negative, antitoxin could be given by any route. Serum sickness may follow the injection but it will be without danger to life. If the patient answers in the affirmative to question No. 1, he should preferably be immunised with an immune ox serum. If the history is in any way suggestive, a skin test may be applied. If the answer to either of the questions 2 and 3 is in the affirmative, antitoxin could be administered by the intramuscular route with comparative safety following desensitisation, but care must be exercised when it is given intravenously.

Skin Test. Individuals liable to reactions can be detected by this test. If 0.1 c.c. of a 1/50 dilution of sterile horse serum in sterile normal saline is injected intradermally, an urticarial weal will appear in a person sensitive to horse serum. The test is negative if no weal appears within an hour of the injection. As a control, an equal volume of sterile normal saline should be injected intradermally in the opposite arm.

Desensitisation. Inject subcutaneously at intervals of five minutes successively, 0.1 c.c., 0.25 c.c., 1 c.c., 2 c.c., 5 c.c. of serum. If no symptoms occur, continue with complete dose; if symptoms occur, wait for half an hour before the next injection. The first few c.c.'s of the balance of the serum should be injected, when doing intravenous injection, very slowly into the blood stream and the patient carefully watched for the development of untoward symptoms.

Treatment of immediate reactions and after-effects. If anaphylactic symptoms appear, the administration of serum must be immediately stopped and 10 to 15 minims of adrenalin injected intramuscularly. Should the reaction be severe, adrenalin should be supplemented by the subcutaneous injection of atropine sulphate grain 1/100. The general treatment for shock should be applied. Serum rash may occur seven to fourteen days after injection. The patient usually suffers great discomfort from pruritis due to the rash. Adrenalin, minims 5, subcutaneously affords relief. Some practitioners prefer to give ephedrin by mouth in doses of grain 1/6 to 1/2 every six hours. The value

of the customary administration of the calcium compounds is debatable. A lotion made up of calamine lotion with 1 per cent phenol added, relieves itching. Aspirin, grains 10, helps in relieving the headache and pains in the muscles and joints.

MANAGEMENT.—The patient is best treated in hospital. Isolation may be maintained at home, but it is difficult to obtain satisfactory management unless the expense of night and day nurses can be incurred. If the patient is isolated at home, he should be nursed in a separate well-ventilated warm room, preferably at the top of the house. A sheet constantly moistened with a disinfectant should be hung outside the bedroom door to break aerial connections between the room and the rest of the house. Probably the real virtue of this sheet is that it is a constant reminder to the rest of the household of the presence of infection and the necessity for care. Separate utensils should be utilised and these should be disinfected or boiled after use. Visiting should be prohibited or at least actively discouraged. Unless facilities are available for these things, it is best to send the patient to an isolation hospital.

Rest:—This must be absolute. The patient must lie flat in bed. He must be kept in a recumbent position for the whole period during which cardiovascular complications may rise. This period varies from not less than three weeks in the mild cases to as long as ten weeks in severe cases. In the earlier stages, the patient lies flat on his back without pillows, but this may be modified slightly if discomfort results, as this produces restlessness and sleeplessness, as both are inimical to absolute rest. The maximum number of pillows allowed during this period is one. Owing to the risk of cardiovascular complications, any attempt to sit up, reach over to a chair, etc., must be prohibited. The patient should not even feed himself according to some authorities. He can be taught to feed himself from a plate on his pillow without raising his head. Sitting up in bed is dangerous and fatal syncope may occur. The patient should be washed and fed. A simple enema should be given on alternate days to avoid the risk of straining at stool. The patient should be lifted on to the bed pan. Movements such as changing the patient's position in bed should be carried out by nurses.

When convalescence starts, he may be allowed increasing liberty but the transition to greater activity should be gradual. At first, pillows should be given; then he should be allowed to sit up in bed; then wash and feed himself, later he should be allowed to get out of bed, to lie on a couch at first, to sit on a chair and, lastly, to walk about. The rate of progress will vary according to the severity of the attack and the response of the cardiovascular system to increased exertion.

Diet:—This is a controversial subject at the present time. There is a great difference between a desirable diet and what one can actually procure in war time. The latter will vary from time to time. Since I do not

purport to be a prophet, I have thought it best to give notes on what would constitute a desirable diet in this disease.

Onset Diet. Milk, with glucose at each meal, water and occasional orange juice between.

Continuation Diet. Milk with weak tea, jelly, rice pudding made with milk, crustless bread and butter, small quantity of fresh fruit.

Recovery Diet. Breakfast: Egg sandwiches, tea-Lunch: Cocoa made with milk, plain biscuits. Dinner: Potatoes, gravy, mince meat, sometimes a second vegetable, fresh fruit.

MEDICATION.—Glucose is of great value in combating toxæmia in severe cases. It can be given as a solution of 20 grammes of anhydrous glucose dissolved in 40 c.cm. of sterile saline intravenously. Glucose lemonade normally should be freely administered to every diphtheritic patient. Dextrose therapy provides a good means of combating cardiovascular failure. Some authorities recommend small doses of strychnine by the mouth; personally, I do not believe in this, as it is of doubtful value to give strychnine to a patient who needs absolute rest. Atropine, coramine and adrenalin are used but there is no unanimity that they are any good. Digitalis is contra-indicated as these patients are liable to varying degrees of heart block. Morphine in repeated small doses, according to some, may tide a patient over a dangerous period of restlessness. Others believe that it is positively dangerous in this condition. Ammonium bromide, grains 5, repeated in 6 or 8 hours if necessary, is a much safer remedy. The routine administration of alcohol has nothing to commend it.

SYMPTOMATIC TREATMENT.—*Cardiovascular Failure.*—Circulatory failure remains one of the terrors of diphtheria. The fatal cases can be divided into two classes, which are probably quite unrelated. In the first, in which the onset is early, the patients usually die within the first seven days, while in the second they do not go downhill until the second week and do not die until the 18th day or even as late as the fourth week. The early form of circulatory failure is probably due to cardiac failure and the later type bears close resemblance to Addison's disease. The patient should be kept without pillows and the foot of the bed slightly raised. Vomiting should be prevented by feeling the patient rectally or giving glucose intravenously. Radiant heat from a cradle may prevent coldness of the extremities. Drugs are of doubtful value. Cortical hormone is of value in relieving the condition, which resembles that of Addison's disease.

Paralysis.—Adequate early serum treatment is the best prophylactic against the nervous complications of diphtheria, also, as stated earlier, a blood transfusion helps. Vitamin B₁ has neither a prophylactic nor a therapeutic effect on the paralytic symptoms of this disease. This was shown by

Bannister and myself in a recent article on the Value of Vitamin B₁ in Diphtheria (*B.M.J.*, March 8, 1941, vol. 1, p. 359). Thickening of the fluids prevents regurgitation in paralysis of the palate. In pharyngeal paralysis, the foot of the bed should be raised to promote drainage of mucus through the nose and prevent broncho-pneumonia. The pharynx can be kept clear of residual mucus by digital swabbing or electrical suction. Atropine 1/200 grain b.i.d. may be of some help in the treatment of these cases. Nasal or rectal feeding is desirable and when the paralysis is fully established, essential.

Respiratory paralysis, invariably accompanied by pharyngeal paralysis, is effectively treated by mechanical respirators of the Drinker type or a Bragg-Paul pulsator. In rare cases, paralysis of the extremities may sometimes be severe enough to call for massage and electrical treatment. All varieties of diphtheritic paralyses generally clear up completely in time.

LARYNGEAL DIPHTHERIA.—In addition to antitoxin, the treatment of this form of diphtheria is either expectant or operative. The former suffices in mild cases and some moderately severe cases. Operative measures are indicated where the seriousness of the attack, the progression of the dyspnoea, the increasing restlessness or the severity of paroxysms indicate that relief is not being provided. If delayed until the patient is cyanotic, the prognosis is much worse. The treatment can be summarised under four headings:—

- (1) Serum.
- (2) Direct laryngoscopy.
- (3) Expectant measures.
- (4) Operative measures.

Serum.—The amount required depends on whether or not there are associated faucial or nasal lesions.

Direct Laryngoscopy.—This is desirable if possible:—

- (a) To see if membrane is present in the larynx.
- (b) To take a direct swab.
- (c) To aspirate mucus and membrane; it is sometimes possible to relieve dyspnoea and avoid operative measures by removing quantities of membrane.
- (d) To perform direct intubation if required. Tracheotomy instruments must be at hand in case aggravation of the obstruction demands immediate relief.

Expectant Measures.—(a) Antispasmodics: The patient is put into a steam tent. An easy arrangement is to put two draught screens around the head of the cot or bed and to fix a sheet so as to form a roof about a foot above the head of the patient—steam being introduced from a bronchitis kettle on either side. Alternatively a crude form of bronchitis kettle could be used and no attempt made to make a steam tent. The patient should be kept flat; light fomentation to the neck is of value in loosening the membrane in the larynx. Atropine or belladonna is occasionally helpful.

(b) Sedatives, for instance, chloral and bromides, help to quiet the patient.

Operative Measures. (a) Aspiration has been mentioned under the heading of Direct Laryngoscopy. If it gives relief, it may be repeated two or three times a day until dyspnoea subsides.

(b) Intubation may be performed by sight through a direct laryngoscope or by the old method of touch. It is not a method for the general practitioner.

(c) Tracheotomy. This is the usual operative method and in times like the present, it is desirable that every medical man should be able to do it. It is preferable not to use a general anaesthetic; local anaesthesia may be employed if time allows. As speed is often essential the operation often has to be carried out without any anaesthesia. The operation must not be deferred until the patient is cyanosed. Everything and everybody must be ready before the operation. The patient should be prepared in bed and is immobilised by wrapping a blanket around his body enclosing the arms. He is held firmly on the table without obstructing the respiratory movements. The head is held rigidly in the midline by an assistant. A pillow under the shoulders brings the trachea nearer the surface. The practitioner stands on the right of the patient and having sterilised the skin makes quite sure of his landmarks before any incision is made. The thumb and the middle finger of the left hand grip the thyroid cartilage on either side and the index finger is placed lightly on the lower border of the cricoid cartilage. The first incision cuts the skin and the second goes through the trachea between the cricoid cartilage and the isthmus of the thyroid gland; or one "stab" incision may go straight through. The usual length of incision is one inch. It is absolutely essential that the incision be made exactly in the mid-line or the trachea may be missed. This is due to the fact that the calibre of the trachea in children under five years is that of a cigarette. One should be absolutely sure that the trachea has been opened before proceeding to the next stage; this is readily known as air rushes in and out and the patients sometimes cough. The tube should not be hurriedly put into the trachea. Dilators are inserted into the tracheal wound and the patient is allowed to cough out any membrane through the tracheal opening. This is generally accompanied by a marked improvement in colour. Fragments of membrane may be removed by the membrane forceps. The violent coughs made, following the opening of the trachea, usually clear the upper respiratory passage and then the tube

can be put in. Bleeding readily stops when the tube is in position. When the operator is certain that the tube is in the trachea (by feeling the air enter and leave) and that bleeding has ceased, he should tie the tapes of the tube around the neck and finally insert the inner tube. The operation should be performed very quickly, that is, from a few seconds to a few minutes. Oxygen should be available and pituitrin 0.5 c.c. injected before incision helps to diminish shock. If there is reflex stoppage of breathing, artificial respiration should be done. McSweeney (*The Practitioner*, June, 1929, p. 701) recommends that the belt of a Bragg-Paul pulsator, which has been put on loosely before the operation, should be buckled and the apparatus switched on by an assistant the moment respiration becomes shallow. Electrical suction through the tracheal wound often remove an obstructing piece of membrane further down the trachea.

The after-treatment of tracheotomy is important. The patient is put back to a warm bed and preferably kept not too flat. A little steam helps for the first few hours after the operation but should then be discontinued. The inner tube should be removed for cleaning every few hours. The outer tube should not be taken out unless obstruction occurs after the operation, when it can be removed, dilators at once inserted, and the obstructing piece of membrane removed. On the third day the orifice may be lightly plugged with sterile cotton wool and the effect on the patient noted. If restlessness recurs the plug may be removed. It is generally possible to do without the tube on the fourth day. Broncho-pneumonia, surgical emphysema and "retained tube" are occasional complications. Broncho-pneumonia should be treated with oxygen, M. & B. 693, etc. Surgical emphysema quickly clears up. As regards retained tube, if the patient requires a tube longer than four days, it is desirable to put in a fresh tube for the one inserted in the operation. Daily efforts must be continued to educate the patient to breathe *per vias naturales*.

TREATMENT OF THE CARRIER STATE.—Those carrying virulent bacilli generally have some pathological condition in the throat, nose or ear, which favours propagation of the organism. The rational treatment of carriers is the removal of the underlying abnormality.

A diphtheritic case should not be discharged from hospital or released from isolation until three consecutive swabs from the throat and nose have been reported negative for *C. diphtheria*.—(*Medical World*).

Surgery

Surgical treatment of Angina pectoris.

—The attainment of precision in our conceptions of the nature of a disease rarely fails to stimulate therapeutics. The accumulation of evidence for the theories that the pain of angina pectoris is due to the

accumulation of pain-producing substances released by an imperfectly oxygenated myocardium, and that in angina of effort the essential anatomical lesion is narrowing of the coronary artery, has led to several attempts to improve the condition by

surgery. Attempts to remedy the fundamental disturbance in the arterial supply to the heart muscle have been made by Beck and O'Shaughnessy, the former grafting pectoral muscle and the latter omentum to the heart. Both operations appear to provide new collateral vessels to the myocardium, and published case reports indicate some improvement in the clinical condition of the patients; but it is not yet known to what extent these operations lengthen life. Mansell reported in 1940 that seven of O'Shaughnessy's original fifteen patients were dead. Another operation which has not yet passed the experimental stage is proposed by Fauteux of Montreal, who has found in dogs that the high mortality from resection of the ramus descendens of the left coronary artery is reduced by previous ligation of the vena magna cordis; he proposes venous ligation to prevent subsequent attacks of coronary infarction and to relieve angina of effort in man. This operation is an example of a practice in surgery based on a principle which is not recognized by orthodox physiology; for while every physiologist would agree that venous ligation increases venous and capillary pressure, it is difficult to see how it accelerates blood flow, which is one of the effects claimed. A rather different approach to the relief of angina is to interrupt the sensory nerves carrying the pain impulses from the heart by removing the upper thoracic sympathetic ganglia or by injecting them with alcohol. Successful operations yield results that appear to be eminently satisfactory in relieving anginal pain, and there is no evidence that the patient who is deprived of the warning to limit his activity lives a shorter time thereby. But there are few cases of angina in which the pain is unilateral and in which interference with the nerves on one side suffices to abolish the pain. The operation is an extensive one, and the alternative alcohol injection, unless performed most skilfully, is apt to be followed by intractable intercostal neuralgia. Finally, we come to an operation the basic principle of which remains comparatively obscure—namely, total thyroidectomy. This was introduced by Levine and Blumgart to relieve congestive failure, and was based on the well-known benefits conferred on the heart in Graves's disease by subtotal thyroidectomy. It aims at reducing the cardiac work by diminishing the basal metabolic rate, which after operation can be maintained at the desired level by administration of thyroid. Although it has now largely been abandoned as a therapeutic measure for congestive failure in the absence of hyperthyroidism, it survives in the treatment of angina of effort, and published series of cases suggest that it has been of real benefit in many. Thyroidectomy was thought at one time to relieve angina by intercepting the sensory paths, but it seems more probable that the results are directly or indirectly due to removal of thyroid secretion, as in many patients relieved by operation pain recurs if too much thyroid is given.

The reader of most of the original papers

dealing with cardiac surgery is apt to emerge with a confused idea of what these operations do and of when they should be employed. That there is doubt as to the effects of these operations is regrettable. Every therapeutic measure constitutes an experiment. Results can in many instances be assessed with considerable accuracy, and in a few more accurately than in angina of effort, for in this disease Wayne and his colleagues showed that the amount of exercise, performed under standard conditions, which is necessary to produce pain is remarkably constant, it is therefore a simple matter to demonstrate what measures increase and what diminish exercise tolerance. Repeated exercise-tolerance tests under standard conditions could scarcely fail to throw new light on the precise effects of these various operations. As to their place in the treatment of angina, most would agree with Sir Maurice Cassidy in his remarks concluding a discussion on the subject at the Royal Society of Medicine; "As I tell all my anginal patients, this is a much-maligned disease. We are impressed by those unfortunates who succumb to or are crippled by coronary infarction; we forget those very numerous patients who live to a ripe old age in spite of their angina, or die from some other disease. Some of them in fact cease to have angina; others learn so to regulate their lives as not to be worried by anginal pain. Nevertheless, I agree that there is a limited scope for surgery in the treatment of this disease." In our present state of knowledge the most suitable patients are those whose lives are rendered excessively difficult by restricted exercise tolerance or unbearable by repeated attacks of severe pain at rest. Choice of operation will be determined to some extent by the nature of the surgical skill available, but should become a simple matter as experience accumulates.—(B. M. J.)

Painless Appendectomy.—RALPH L. GORRELL, M.D., Clarion.

Pain following an abdominal operation may be greatly decreased by the injection of a local anesthetic solution into the region of the incision or lateral to it. It may also be lessened by the use of a transverse incision and by the avoidance of any procedure during the operation, which would cause pain or discomfort to the unanesthetized patient, such as picking up the skin with towel clips, clamping rough towels on the edge of the incision, blunt and rough dissection, spreading of tissues, and incising sensitive structures like the skin, fascia and peritoneum without first locally anesthetizing them. Crile's work on anoci-association was fundamentally much more important than is realized at the present time. Leriche has shown that pain is in itself a disease.

Technic: If the operation is carried out under spinal or nerve block, anesthesia (which prevents painful impulses from reaching the central nervous system, and in itself is a valuable means of preventing

postoperative pain), the incision is made transversely at the level of the anterior superior spine, or lower, if the point of maximum tenderness is inferior to that point. This incision may be four inches in length and yet the patient will have little pain if care is taken to cut cleanly, and blunt freeing of the rectus fascia is not done.

The line of incision is infiltrated with procaine solution or eucupin-procaine solution, one-half of one per cent, if the operation is performed under general anesthesia. The rectus fascia and peritoneum are also infiltrated, so that no sensitive tissue is incised until it is anesthetized. A further refinement is to infiltrate the mesoappendix with the solution before clamping or ligating the appendiceal artery. The time for the operation is reduced if adrenalin is added to the procaine solution, since oozing is obviated and with it the clamping of many tiny veins.

The edges of the incision are protected with soft latex rubber sheeting or dam and the bowel is packed back with the same material, so that gauze packs never touch the delicate serous covering of the intestine. Preferably, the cecum is isolated by tilting the patient to the left so that the small bowel falls away from the ascending colon and the cecum is seen without the necessity of pressing on the intestine or inserting packs of any type.

No drains are ever used unless the peritoneal cavity is full of free pus, although after the removal of any acutely infected appendix a strip of rubber or cigarette drain should be left down to the peritoneum, to prevent the formation of a wound abscess. A catheter (French size 18), with several additional holes cut out, may be slipped through the opening left in the cecum by the removal of the appendix and secured by two purse string sutures, if the appendix is severely inflamed or if peritonitis is present. Its use often permits the patient to pass gas, thus relieving or preventing "gas pains" and may prevent the later use of ileostomy or duodenal tube for relief of distention.

Post-operative Care: One-sixth grain of morphine is used each night for two nights. Mild discomfort is relieved by nembutal ($\frac{1}{2}$ grain), acetylsalicylic acid (5 grains) or Dover's compound-aspirin tablet every four or five hours. The adhesive tape is cut along the gauze at the end of twenty-four or thirty-six hours, to permit the slight distention that follows any abdominal operation. Overlooking of this minor point causes the patient to suffer unnecessary discomfort.

Immediate postoperative feeding: It is a strange commentary on the logical processes of surgeons' minds that they ordered no food to be given to patients either before or after operations, for many years, and at the same time noted that those patients, subjected to emergency operations, often had less discomfort. We treat the patient as a normal individual both before and after operation. He is encouraged to eat a full meal the evening before operation and is given sufficient sedation that digestion is not hampered by

apprehension. If the operation is performed under local anesthesia, food is taken at noon; if under general anesthesia, a few bites of solid food are taken on the evening of the operative day. The patients are required to take a little solid food at each meal time thereafter, despite anorexia, nausea or slight distention. Liquids are given in very small amounts during the first forty-eight hours, but foods with high water content (ripe banana and carefully peeled apple) are given every two hours to prevent thirst. Saline solution or five per cent dextrose solution is given by hypodermoclysis if nausea persists or if vomiting appears (usually in one case out of five). If definite distention appears, a duodenal tube is inserted but this is necessary usually only if the appendix is ruptured, and the insertion of a cecostomy tube at the time of operation often relieves gas.

Results: These patients usually have little pain in the incision; they move about in bed freely and notice few or no gas pains late in the second or early on the third day. The few which may appear are readily relieved by one tap water enema on the morning of the third day. They are a joy to the nurses because they sleep all night, do not need to be catheterized, probably because of lessened pain in the incision, and do not make demands on their time. The average patient with acute, non-ruptured appendicitis, is up on the edge of the bed on the third day and in a chair on the fourth day. Even the most complaining patient is ready to go home on the seventh day. Early walking seems to help cut-down pain in the incision.

Eucupin: Eucupin is a long-acting local anesthetic which is bactericidal. Its chemical name is isoamylhydrocupreine, indicating that it is a synthetic alkaloid, closely related to quinine. The manufacturers have kindly supplied us with quantities sufficient to carry out clinical research. Because of a slight burning sensation on injection, it is usually made up with one per cent procaine solution. It is also furnished in oil solution for prolonged anesthesia after rectal operations, and in iodized oil for the injection treatment of low back pain.

After completion of the appendectomy and suture of the peritoneum, from 30 to 60 cubic centimeters of eucupin solution with procaine one per cent are injected into the peritoneum, rectus muscle and skin. The addition of four drops of epinephrine prolongs the period of anesthetization. Occasionally, I have used eucupin in oil for a more prolonged effect but have not had the courage to inject amounts large enough to cause analgesia for several days, inasmuch as these are all private patients. It would be very valuable if a charity surgical service could inject oil soluble in eucupin in a large series of cases, to observe the possible incidence of sloughs and delayed healing, as well as the lessened period of hospitalization.

de Takats employed eucupin and a combination of one of the more rapidly acting anesthetics and epinephrine in 100 operations, which included herniotomy, thyroidectomy and minor surgical procedures

about the head and neck, and reported a postoperative analgesia lasting about twenty-four hours. The solution did not cause tissue reaction nor any disturbance in wound healing (microscopic section and clinical observation). He feels that the powerful antiseptic property of eucupin is an advantage in ensuring sterility of the injected fluid as well as that of the operative field. This article was found on exploring the literature. Our incisions have healed normally. Davis technic of performing a transverse incision was used. Except for the first case noted below, no patient had fever after twenty-four to forty-eight hours but for the usual fever of a mild nature, 99 to 99.8 degrees afterwards.

SUMMARY:—Postoperative pain may be largely prevented if a few simple steps are taken.

1. Injection of eucupin-procaine solution into the lateral edge of the incision when suturing is done.
2. Infiltration of skin, rectus muscle and fascia and peritoneum with procaine solution before incising them.
3. No procedure is carried out which would be painful to the unanesthetized patient; clamping skin with towel clips, use of rough towels on edge of incision, rough or blunt dissection, or forcible separation of muscles in line of incision, letting the patient's back remain unsupported by a small pillow.

4. Encouraging immediate postoperative feeding.—*Journal of Iowa State Medical Society.*

The Etiologic Role of a Long Omentum and its Proper Consideration in Attempting to Effect a Cure of Inguinal and Femoral Herniae by Operation.—By Ottokar Tenoppy, M.D., F.R.C.S. and F. M. Al Akl, M.D. Brooklyn, N. Y.

In considering the operative treatment of hernia, whether in text books or in current magazine articles, attention seems to centre upon the structure of that region of the cavity wall through which the hernial mass protrudes, while hardly any mention is made of the contents of the cavity and the role which they play in the causation of hernia, primary or recurrent. The prevailing impression seems to be that hernia is due to one or a combination of the following factors:

1. Weakness of the structures of the cavity wall.
2. Abnormally large fascial apertures.
3. Defects in the wall due to—(a) improper fetal development (patent processus vaginalis, openings in the diaphragm, etc.), (b) surgical incisions, and (c) accidental wounds or infections with sloughing of tissue.
4. Increased intracavitary pressure.

Yet, supposing we had a patient whose abdominal wall was weak, abdominal rings

wide—a patent processus vaginalis—and tense abdominal muscles, but had no organs inside his abdomen, could such a patient ever have a hernia? The answer would naturally be "No". Now, instead of supposing that the patient has no abdominal organs at all, let us say that the patient has all his abdominal organs, and add that all these various organs are attached to the abdominal parietes through short pedicles and mesenteries, and that the patient has a short omentum; and ask if such a patient could have an inguinal or femoral herniation of any organ totally enveloped by peritoneum? The answer would again be: No.

From the illustrations one can readily see the role which an unduly long omentum would play in the causation of hernia; yet, foregone as this conclusion might seem, its implication is not easily understandable. To prove this point further, the following simple experiment was performed at the morgue. A fresh cadaver with a reducible, complete inguinal hernia was chosen for this experiment. With the hernia completely reduced, the abdomen of the cadaver was compressed manually with the result that the hernial mass reappeared in the groin and descended into the scrotum. The hernia was reduced and the procedure was repeated several times, the hernia appearing each time upon pressure. The abdomen was then opened through a lower midline incision, the part of the omentum beyond the pelvic brim was trimmed off, the mesentery of the lowest loops of bowel plicated, and the abdominal incision closed. Subsequent compression of the abdomen resulted in no hernial protrusion. The result of this experiment seemed to prove conclusively an issue that had long been controversial in our conferences.

It seemed clear that while the incision of the hernial sac and the reconstruction of the abdominal wall was well in order for the radical cure of hernia, the removal of redundant omentum was an important additional step in cases where the omentum was long enough to be accessible through the neck of the sac.

Anatomists describe the omentum as quite short at birth, scarcely extending down to the umbilicus, but extending down to the summit of the bladder in the adult. It hypertrophies and becomes elongated with years and is often encountered as a tremendous mass in the scrota of aged patients with complete inguinal hernia. Watson in his chapter on the etiology of inguinal hernia seems, like most other surgeons, greatly impressed by the sacular theory of the causation of hernia, and does not mention the part an unusually long omentum would play in the formation of a hernia. This omission becomes particularly striking when he quotes Raw and Murray, who have found 68 peritoneal sacs (52 femoral, 13 inguinal and 3 umbilical) in bodies of 200 people who never had any history or external evidence of hernia. The fact that Nobbe found that 35% of males live and die with a patent processus vaginalis, while the incidence

dence of inguinal hernia according to Berger is no more than 8 per cent in males, amplifies greatly the etiologic role of intra-abdominal organs.

While one cannot excise all that one finds in the hernial sac, or perhaps plicate with safety the mesentery of protruding loops of bowel, the excision of the omentum when indicated by excessive size has been harmless in our hands. A search of the literature revealed no ill effects reported. We therefore deliver the omentum through the neck of the sac and apply traction to its free border by means of clamps. One or more hemostats are then passed through avascular areas in the omentum at its exit through the neck of the sac. A long continuous ligature is then grasped between the jaws of the hemostats and pulled through the omental apron. The ligature is cut at the sites where the clamps had been applied, and the omentum is tied off in small segments by means of the resulting smaller strands of cat-gut. The rest of the omentum is clamped beyond the ligatures to prevent return flow and the omentum is severed between the ligatures and the clamp. Once the severed stump is inspected and no bleeding seen, the ligatures are cut short and the stump is allowed to retract into the abdominal cavity. This done, the neck of the sac is transfixed and the wall is reconstructed as required.

While this procedure might seem objectionable to those who are particularly conscious of the uses of "the policeman" of the enough of the omentum is usually left for it to attack offenders of the peritoneum when disposed to do so.

Because of the frequency of recurrence in those past middle age, excision of the omentum becomes particularly indicated in that age group whose herniae are, among other causes, due to omental hypertrophy, and in whom wound healing is not so rapid and union not so strong as in younger individuals.

It is true that the omentum increases in length by age, therefore its shortening at the time of operation would at least relieve the freshly repaired walls of immediate pressure, whether due to abdominal distention, vomiting or coughing during the critical period of healing, until enough union has taken place for the reconstructed wall to withstand the pressure of the lengthened omentum.—*Medical Times.*

Interpretation of the Visualized Gall-Bladder.—George Turner, M.D. El Paso, Texas. Before the advent of opaque dye in 1924, perfected by Graham, *et al.*, for rendering the gallbladder visible, information concerning the condition of the gallbladder had to be gained from whether or not it contained opaque stones, possessed a wall of sufficient density to cast a contrast shadow, and from deformities of the duodenum caused by adhesions from the diseased viscous to adjacent structures. These are yet cardinal points in the diagnosis of gallbladder disease and none the less important since the advent of the dye. A flat film of the gall bladder area before the dye is given and a careful

barium study of the stomach and duodenum after the dye films are completed make an important part of the examination.

Experience has taught that the wall of the normal gallbladder is so thin and deeply buried that it will not produce a contrast shadow on a flat film. In case the wall has become sufficiently dense through fibrous change and consequent thickening to produce a shadow in contrast with the surrounding tissues, a diagnosis of chronic cholecystitis can safely be made. This information is again important in determining the emptying time or degree of filling of the gallbladder with the dye. A light shadow of the same density as that obtained before administration of the dye means no filling or complete emptying if the first films made after taking dye showed it filled.

Calcium density stones are readily demonstrated and are not often confused with any other condition. The characteristic laminations, translucent shadows encircled by a rim of calcium, small, multiple and shot-like shadows, or the typical faceted arrangement serve to differentiate gallstones from a stone in the right kidney pelvis, gland calcification or irregularly calcified rib cartilage. Dye administration is important after stones have been demonstrated to determine if there be function of the gallbladder, its size and contour, and to further identify the stone shadows within the gallbladder. It is necessary occasionally to use the dye and also to make pyelograms of the right kidney where there is a single gallstone and also a right kidney stone. A lateral view is helpful in making this differentiation.

A barium study of the stomach and duodenum will give information concerning the condition of the gallbladder in most cases of gallbladder disease. In acute and subacute cholecystitis, motility of the stomach and duodenum is increased with no point of irritation of either of these structures. Approximately 50 p. c. of the cases of chronic cholecystitis will show deformity of the cap or second section of the duodenal loop from adhesions. These adhesions are characterized by fixation and deformity of the loop without ulcer defect or irritability. Occasionally adhesions will be sufficiently dense and contracted to produce obstructive narrowing, in which case hypergastric motility, irritability and spasm of the duodenum, together with clinical symptoms of pyloric obstruction, will be found.

After more than fifteen years experience with opaque gallbladder dye it has been shown that oral administration is the best method of giving it. Reactions following oral administration are less frequent and less severe than when given intravenously and there are not so many contraindications. Most radiologists have discontinued the use of the dye intravenously. Jaundice, acute cholecystitis with or without stones, and advanced cirrhosis of the liver are contraindications for its use. Interpretation of the results from oral administration are coming to be well understood. Granting that the proper dose of dye has been given and that the films are made properly, failure of the

gallbladder to fill is a definite criterion of a pathologic condition. To be quite sure that instructions for taking the dye have been properly followed it is best to repeat the dye before reaching a final conclusion of a blocked duct in non-filling instances.

There is no place in radiology where details of technic are more important than in the study of the gallbladder. Any break in the chain of details necessary to produce films to bring out properly contrasted soft tissue shadows in the abdomen are likely to lead to an erroneous conclusion. The usual routine films consist in a flat view of the gallbladder area before giving the dye. The dye is given orally following a fat-free or nearly fat-free evening meal. The first film is made the following morning approximately fourteen hours after taking the dye and immediately after a cleansing enema. It is best to give the enema immediately before making the film to avoid refilling of the transverse colon with gas or unabsorbed dye sufficiently to obscure the gallbladder shadow. Again an enema shortly before making the film will produce enough stimulation to the gallbladder to make it fill, whereas no filling would be found on a film made before the enema; or it would be found empty again if too much time elapsed between the enema and the film. One or more films are made at this time and other six hours following the stimulation of a fatty meal. If sufficient dye has been given and absorbed the gallbladder will be found to show some degree of filling on the morning films unless it is blocked by cystic, hepatic, or common duct obstruction.

In cases of marked ptosis or enlarged liver the gallbladder is likely to be directly in front of the spine, or more rarely below the pelvic brim, and its outline obscured on a film made according to the usual technic. In these cases it is necessary to elevate the right hip and thus rotate the patient to the left to throw the shadow from in front of bone. A lateral view is occasionally necessary and useful.

The afternoon films usually show the normal gallbladder empty of dye. The length of time required for complete dye elimination is in proportion to the amount of dye taken. The normal gallbladder is more likely to be empty six hours after fat stimulation following one dose of dye than it is after two or more fractional doses. Elimination is slower following a large than a

small dose. The fact that it is not completely empty six hours after fat stimulation does not mean that it is diseased. A reduced size with a regular outline only means that the process of emptying is not complete. If it remains of approximately the same size and configuration as before the fat stimulation, evidence of abnormal retention is indicated.

Opaque stones may be obscured by the dye, though their outline can usually be followed by a contrast greater than that of the dye. The outline of the negative stones is contrasted by a density of the dye greater than that of the stones. Sand is difficult to demonstrate though it will often appear as a small shell-like margin taking the shape of the fundus. Small, foamy, negative stones are best seen on films made in the standing position. They form a zone of negative shadows at the middle third of the gallbladder.

Evidence of cholecystitis is shown by variations in shape, size, and function. Generalized contraction with poor function is one of the most common findings. Irregular contraction with resulting deformity is common. The middle third and tip of the fundus are the most usual locations for fibroid contraction. Deformities produced are curving, flattening of the medial edge, cuff-like constriction with bulb formation at the fundus, and folding. Pointed or "tented" defects indicate adhesions and may be either to the duodenum or, more rarely, to the colon.

Anomalies of the gallbladder are very rarely seen. Duplication is the most common. Diverticula are also rare. Localized contraction and erosion about a stone sometimes form an auxiliary pouch.

Adhesions about the gallbladder are not always caused by inflammation arising within it. It may occasionally find itself "in bad company" and does not stand alone as a trouble maker with adjacent structures. Adhesions following acute duodenitis or a penetrating duodenal ulcer are found with no other involvement of the gallbladder.

Tumors arising from the mucosa show characteristic constant defects. Papilloma is the most frequent. These defects appear at either margin and remain constant through any degree of filling or phase of emptying. They are usually small.—*Texas State Medical Journal*.

Dermatology

Plantar Warts—By AUSTIN FURNISS, L.R.C.P., L.R.C.S., L.D.S., D.P.H.

Plantar warts are true warts occurring on the soles of the feet, where they appear as rough, hard, yellowish masses. They are commonly met with in children, and are due to a filter-passing virus. The external appearance of a plantar wart is no true indication of its size, since, owing to its situation, it is subject to more or less continual pressure and grows upwards into the skin of the

sole, forming a small cavity for itself. Although they may occur on any part of the sole, they principally affect pressure parts such as the balls of the toes, the metatarsal pads, and the heels. Often several are present and they are round or oval in outline, and are either level with or slightly raised above the under surface of the foot. They are usually painful on pressure, the pain sometimes being so severe as to cause a limping gait, which, if at all prolonged, may

cause definite deformity. It has always been thought that warts in other situations are infective; and in the case of the foot it has been shown that they are infective in origin, being transmitted from infected bath mats, towels, and the like. They do occasionally form after prolonged irritation, such as that caused by an embedded splinter, or by a nail sticking through the sole of the shoe. On examination with the microscope of a section of a wart there is seen an overgrowth of the prickle cell layer, prolongation of the interpapillary processes, and pronounced hyperkeratosis. The corium beneath is stretched and flattened, and often there is a small artery running into the base of the wart. These warts are always sharply outlined and rough on the surface, which is sometimes streaked with black due to dirt ingrained between the hyperkeratotic processes.

Girls are much more frequently affected than boys, the proportion being almost two to one. Numbers of cases are seen at school clinics, and it would appear that the condition occurs in epidemics, sometimes on quite a large scale. To give an idea of its frequency, figures from the *Bulletin of Hygiene* (vol. ii, 1936) may be given. The well-known skin specialist, Dr. Lomholt, of the skin department of the Finsen Institute, Copenhagen, issued a summary of a census of 1400 unselected school children. He found that 6 per cent. of the girls and 3.8 per cent. of the boys were infected.

Methods of Spread.—Evidence points to the bath, including swimming and shower baths, as a fairly common method of infection. Bath mats used in common appear to be a means by which the virus is conveyed from one person to another. Home infection can also take place, as some cases have been found in which a brother or a sister has been found with the disease at home.

Symptoms.—Children come to the clinic because they complain of pain on walking or running. On examination the wart or warts are readily seen and may be of quite a large size. They are tender to the touch—sometimes very much so. They are almost invariably found on parts of pressure—the heel or ball of the foot, a fact which makes their treatment all the more difficult. Sometimes the condition is not detected until the child is seen to be walking awkwardly or painfully. This induces a bad habit of walking, which, if neglected, may lead to actual deformity of the foot.

Methods of Prevention.—These will be considered in detail, as it is obviously better to prevent this painful and troublesome condition than treat it. More can be done in the case of children at boarding-schools than in other schools. A regular foot inspection should be carried out, and infected children, if they are boarders, should (a) be segregated in separate dormitories; (b) have their own bath and changing rooms; (c) not be allowed to use the school swimming-bath; (d) have separate shoe and boot lockers, and their shoes and boots should be cleaned after those of the unaffected children.

Infected children should wear white cotton

socks, which should be boiled every night. In the presence of an epidemic, boarding-school children should have their feet sponged with methylated spirit after a bath, and their shoes should be occasionally swabbed out with a 1:50 solution of lysol or similar preparation. The bath should also be swilled out with a similar or weaker solution after use. A disinfectant should also be applied to the sidewalks, foot-board, and diving-boards of the swimming-baths. As bath mats used in common appear to be a means by which the virus is conveyed from one child to another, each infected child should have his or her own bath mat, or, better still, two towels—one for standing on and one for use in drying.

Treatment.—The best treatment consists in their removal with a small sharp spoon, under a general anaesthetic if many are present; under a local anaesthetic if there are only two or three. The best local anaesthetic to use is a 2 per cent. solution of novocain, 1 c.c. of which is injected into the base of each wart. This injection may, however, be very painful. It is quite easy as a rule to shell out the little glistening warty mass, but it is absolutely essential to see that every trace of the warty substance is removed, otherwise there will be recurrence. It has been mentioned that there is often a small artery running into the base of the wart; sometimes there is profuse bleeding from this artery, but it may be stopped by applying firm pressure with a pad and bandage for about a quarter of an hour. The electro-cautery is a valuable instrument to have in these cases. If available, the base of the cavity may be touched with the cautery heated to a dull red; this not only stops the hemorrhage, but it also removes any remaining traces of warty material. The cavity left after the operation can be got to heal in about a fortnight by the daily application of pulv. bism. subgall. If the skin of the sole is soft and thin, most plantar warts respond to salicylic acid and collodion paint, or they may be destroyed with nitric acid. In other cases, however, where the skin is thick and leathery, erosion, CO₂ snow or X-rays are usually required. When freezing with CO₂ snow is carried out, the application should be of four to five minutes. Heat cauterization and diathermy are not very satisfactory. X-rays is usually very effective, and it has the advantage of rapidly relieving pain. Whatever method is adopted, it is a good plan to remove the horny lid over the wart with 20 per cent. salicylic acid plaster, applied for a week or two before treatment. X-ray is probably the best form of treatment, a pastille dose and a half being given, care being taken to completely screen the surrounding skin. If this application fails, a second dose should be given after a month's interval. This method of treatment offers the best prospect of cure in intractable cases.

Most clinic doctors use remedies which are used for the treatment of warts in general, such as strong acids, nitric and trichloroacetic and alkalis, the surrounding skin being

protected by vaseline, salicylic collodion, (1:7), or this preparation with zinc chloride. A useful paint can be made by adding 5 or 8 gr. of cannabis indica to salicylic collodion, the parent being instructed to apply the preparation by means of a glass rod or a camel-hair brush. Salicylic collodion used by itself does not achieve much, but combined with zinc sulphate or cannabis indica often causes the warts to disappear. Strong nitric acid carefully applied with a glass rod—employing the same precaution for protecting the skin—

is sometimes efficacious. Bleeding sometimes occurs as a result of this treatment, owing to the corrosive action of the acid, so that, if possible, the blood supply to the wart should be cut off by ligature of the base. After all traces of the wart have been removed it is advisable that the child should be kept under medical observation, and any tendency to foot deformity—which is occasionally met with—as previously stated, should be corrected by suitable footwear and remedial exercises.—*Prescriber, Jan. '41.*

Book Reviews

Materia Medica of Pharmaceutical Combinations and Specialities—By Dr. U. B. Narayana Rao, Managing Editor, "Medical Digest" and "Indian Journal of Venereal Diseases, Bombay, Second Edition, Published by "Medical Digest", Girgaum, Bombay. Price: Rs. 7-12-0.

Since the publication of the First Edition, there has been an enormous increase in the number of medicinal specialities that have been marketed in this country and Dr. U. B. Narayana Rao, for the benefit of the Medical Profession, has compiled them into one book. It gives valuable information with regard to the various pharmaceutical combinations and specialities. This second edition includes new products which, together with those in the previous publication,

amount to over 4000. The sections on Sex-Hormone Therapy and on Vitamins, printed at the end of the book, are useful. The Editor has taken every pain to make this edition useful to the practitioner, Chemist and the Druggist. The book is handy, neatly printed and well got-up.

May & Baker's Specialities—Notes on.—We have received a copy of the above, which contains information about their new products with revised notes. The Therapeutic Tables have been completely revised and rewritten in the light of the most recent knowledge and should prove a useful means of reference to the application of their products in the treatment of a wide variety of conditions.

Forthcoming Publication

Mr. Dorothy A. Bennett, Sales and Promotion Manager, University of Minnesota, announces that a book on **The History of Drs. Mayo, Their Clinic, and the Mayo Foundation** by H. B. Clapesattle, the trained historian and chief editor of the University of Minnesota Press, will be published under the auspices of the above Press, on 17-11-'41.

There is hardly any medical man in the world that has not heard of the famous Drs. Mayo and their institution in Rochester, in New-York State, U.S.A. The phenomena of achievement of the Drs. Mayo, the small town clinic that grew into international fame was little understood. The modesty of the men and their conformity to the medical ethics, discouraged any publicity during

the life-time of the Drs. Mayo. In the end, however, in order to divest themselves of any connection with it, they authorised the University of Minnesota to publish, through the University of Minnesota Press, a book on the history of the Drs. Mayo, the Mayo Clinic, and the Mayo Foundation.

The book now to be published is an independent study, written by H. B. Clapesattle. It is a story of how the Old Doctor Mayo, himself a social and medical pioneer, passed on to his sons a passion to learn every one and everything and how they passed it on to their colleagues till it became the living spirit of a great institution. The book will bring all these elements into focus in an important human drama of unequalled interest. The book will surely be quite welcome to every medical man.

Announcement

The next issue of *The Antiseptic* will be a Special Number on **Diseases of Old Age—Their Prevention and Treatment.**

As usual, it will be an enlarged edition, containing several contributions and will be out on the 15th October, 1941.

Institutions and Associations

The Mysore State Medical Department—Annual Report for 1939.—A total of 330 Medical Institutions were functioning in the State at the end of the year 1939 as compared with 311 at the close of the previous year. The number of beds available for in-patients was 3,210 as against 3,056 in the previous year. The total number of in-patients treated in the various hospitals was 65,285—an increase over the previous year. The number of out-patients treated was 61,40,706, as against 56,12,993 in the previous year. The total number of Parturition cases was 33,093, of which 13,808 were done inside the institutions.

The expenditure on the Medical Department during 1939 was Rs. 19,82,217. The Scheme for Subsidised Medical Practitioners in rural areas is progressing and 9 more appointments were made during the year. A grant of one lakh of rupees was made during the year for providing an up-to-date surgical and other equipments to the major hospitals in Mysore and Bangalore. The State interested itself in Anti-Tuberculosis work. From the report it is seen that Malaria is the most prevailing disease in the State and that diseases of the Respiratory System and of the Digestive System also claimed a large number of victims. From the report, we see that a large number of patients attended the Ayurvedic and Unani Dispensaries in the State in spite of the fact that treatment is not free. The report gives a number of other facts and figures and valuable statistics which speak volumes of the ability with which the Mysore Medical Service under the able guidance of Rajasevasakta Dr. B. K. Narayana Rao is working.

The Government Ophthalmic Hospital, Madras—Triennial Report for the Years 1938-40.—From the above Report, we note that more than 35,000 out-patients and 5,000 in-patients were treated in 1940, and that 2216 cases of Kochweeks conjunctivitis, 1708 of trachoma, 2323 of chronic conjunctivitis, 3105 cases of cataract and 6469 cases of anomaly of refraction and disorders of the mechanism of accommodation were treated among a great number of cases of other diseases. We find that the Hospital trained in 1940, 166 M.B., B.S. students, besides many other classes of students. It may also be noted that many new amenities have now been provided for the patients such as a Dining Hall, a Radio Set etc., from donations from philanthropic gentlemen as well as from the Hospital funds. This Hospital is doing very good work.

The Union Mission Tuberculosis Sanatorium, Arogyavaram—Annual Report for 1939-40.—This Sanatorium was opened in 1915 and ever since, has been working satisfactorily. The number of patients admitted during the year was 385 and 378 were discharged. The total number of patients treated during the year was 631. The daily average number of patients treated has

been 255 for the whole year, as against 238, for the previous year. The number of beds available during the year was about 244, but extra beds were fitted temporarily as there was a need. Of the 382 patients discharged, 305 were found to be suffering from pulmonary tuberculosis. Of these, 264 are included in detailed statistics and of these 210, or (79.6%) were advanced cases when admitted. Of these advanced cases, about 98 or 46.7% were discharged as much improved and 19 or 9% as improved.

These figures are mentioned only to emphasize the very great importance of sending the patients for treatment as early as possible. During the 25 years of the existence of the Sanatorium, 5,789 patients have been discharged. Out of this number, 4,050 or 70% have obtained positive results. Out of the 264 patients discharged, artificial pneumothorax treatment was done to 199. Sanocrysin treatment was given to 132. During the year, 1,601 X-ray photographs were taken. Surgical procedures such as thoracoplasty and phrenic nerve operations were done on a number of cases. The Sanatorium is also equipped with first class laboratory where during the course of the year, 52,000 examinations of various kinds have been done.

On 19th July 1940, the Sanatorium celebrated its Silver Jubilee. This year was marked by the beginning of the T. D. D. course of training for doctors started by the Government of Madras—the first of its kind in India. The practical training for this course is being given in Madanapalle. During the course of the year, a number of extensions have been built and the Sanatorium is much improved.

All these facts go to prove the popularity of the Sanatorium and the very excellent way in which it has been managed, first by Dr. C. Frimodt-Moller, and later by Dr. P. V. Benjamin and his very energetic and enthusiastic staff of assistants. We congratulate the sanatorium on its splendid work.

The Coimbatore District Medical Association Annual, 1940.—The Annual presents us with 19 original articles on various topics of interest, among which may be mentioned 'Ectopic Gestation' by Dr. Ida S. Scudder, and a Symposium of Pulmonary Tuberculosis by various writers.

The Annual Report for 1940 at the end of the Annual shows that the Association is doing good work and that it is one of the best organised medical Associations of South India. We are glad to find it reported that the membership of the Association has increased from 57 to 107, and that it is hoped to increase it to the total of 200. Besides one annual meeting in January 1940, five general meetings and nine ordinary meetings, mention may be made of two joint meetings held by the Association—one with the Trichinopoly District Medical Association in July 40, at Perundurai, and

the other with the Malabar Medical Association in September 1940 at Palghat. The Association maintains a Reading Room with nine different journals placed on the table. We find that the Association is in want of accommodation, and a library, which, we wish, it will have in the near future. We also hope it will be able to form itself into a branch of the Indian Medical Association.

The Kistna Dist. Medical Association, Masulipatam.—(The Kistna Dist. Branch of the Indian Medical Association.)—At a monthly meeting held on the 23rd August, 1941, under the presidentship of Dr. N. N. Sujee, D. M. & S., Dt. Medical Officer, the above Association nominated the following President and three Vice-Presidents of the Andhra Provincial Medical Association for the ensuing year.

Dr. B. S. Ramachandra Rao, President.
(1) Dr. P. Veeriah Chaudary, Guntur, (2) Dr. B. Tirumala Rao, Vizag, (3) P. Gurumurthy, Rajahmundry, Vice-Presidents.

Then the letter from the Honorary General Secretary of I. M. A., Calcutta, dated 4-8-41 was read, which contained the resolution passed by the Working Committee regarding the recruitment to the Emergency Commissions in I.M.S. and I.M.D.

During the ensuing Clinical Meeting:—

Dr. J. Venkatarama Sarma, M.B.B.S., & L.O., Masulipatam, demonstrated a case of some cerebral complaint and explained the pathology of the same.

Dr. C. B. Ethirajulu, M.B.B.S., Assistant District Medical Officer, Kistna, demonstrated a case of Dermoid Cyst which started at the Frontonasal Centre and discussed the differential diagnosis.

Dr. Miss R. Lazarus W.A., Masulipatam, demonstrated two interesting cases of Abdominal Tumours, and the differential diagnosis was discussed. And also demonstrated a rare case of Gumma Breast in a woman aged about 35 years.

The President, in his concluding remarks, discussed the differential diagnosis of the above cases and the meeting dispersed at about 7 p.m. with a vote of thanks.

The Indian Chemical Manufacturers' Association, Calcutta.—Members of the above Association met Lieutenant General

Sir Gordon Jolly, Director-General, Indian Medical Service, at the premises of the Association at tea on 21-8-41, and discussed with him the following subjects relating to the Chemical and Pharmaceutical Industry in this country. Mr. J. N. Lahiri was in the chair.

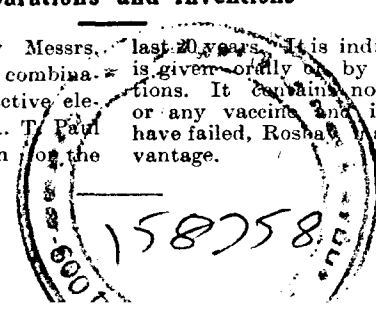
- (1) Purchase of medical supplies from England over a long forward period.
- (2) Manufacturing and importing activities of the Government Medical Stores Depots.
- (3) Purchase of vaccines and sera by the Government from Indian manufacturers.
- (4) Difficulties experienced in exports of drugs and medicines out of India, and
- (5) Necessity of giving permission to private manufacturers who undertake manufacture of opium alkaloids.

Lt. General Sir Gordon Jolly gave an assurance that the policy with him was to get all available medical supplies from this country and import only those items which were not being manufactured already or which could not be manufactured in the near future. As regards permission to manufacture opium alkaloids, he stated that the matter rested with the Central Board of Revenue. He, however, promised to lend his support to the request of the manufacturers in this connection. Among those who attended the meeting were: Bt. Col. Sir R. N. Chopra, Dr. J. N. Ray, General Taylor, Major General S. P. Bhatia, Col. W. C. Paton, Dr. H. Ghosh, Mr. B. Maitra, Mr. Mohamed Hanif, Sir Hari Shankar Paul, Dr. B. N. Ghosh, Mr. Madanlal H. Vakil, Dr. A. K. Sen, Dr. M. A. Rahman, Mr. H. D. Nag, Mr. B. Das, and Mr. J. Chkravarty.

All-India Ophthalmological Society.—The Committee of the All-India Ophthalmological Society announces that the subject for the next award of the ADENWALLA MEDAL AND PRIZE IN OPHTHALMOLOGY will be 'Changes in the Cornea due to Malnutrition'. The prize will be awarded to the best work on the above subject and is open to all ophthalmic specialists resident in India. The essays must be submitted to the Secretary, All India Ophthalmological Society, Flitcham, Marshall's Road, Egmore, Madras, before the 15th November, 1942.

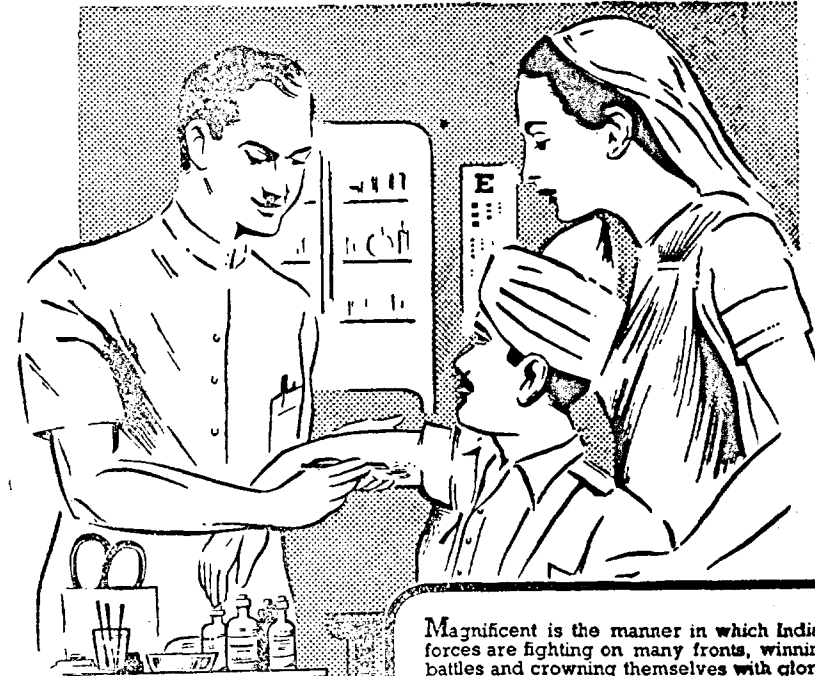
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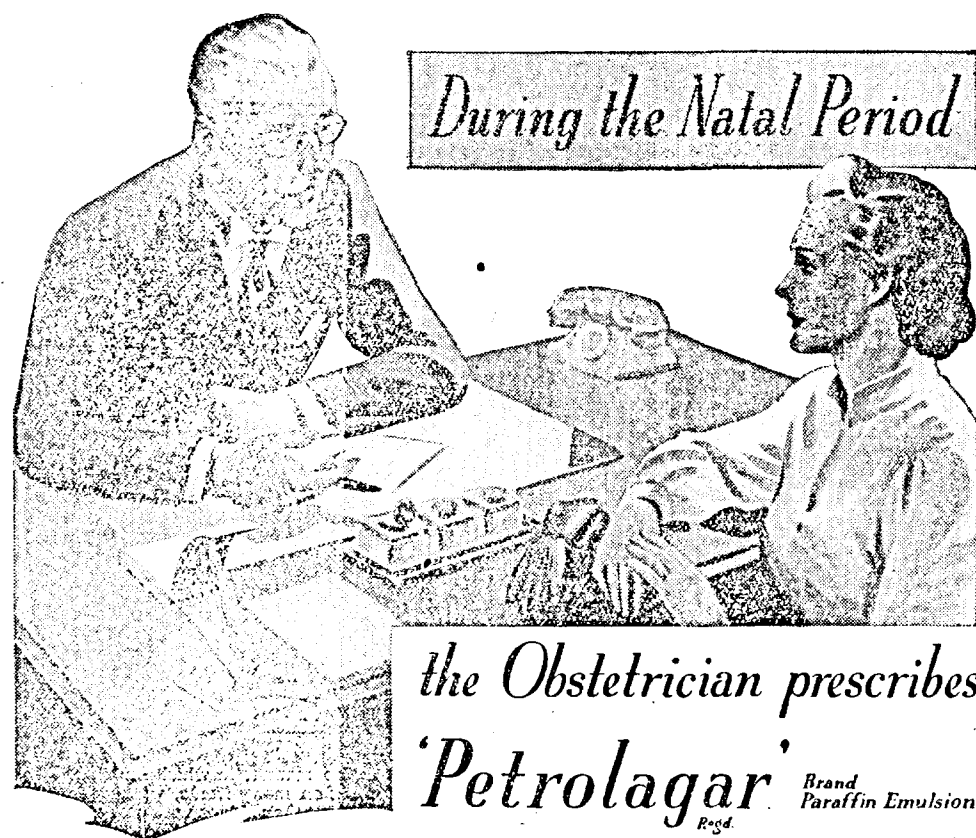
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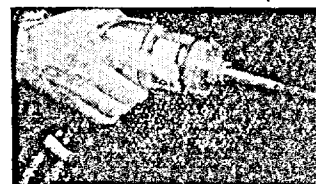


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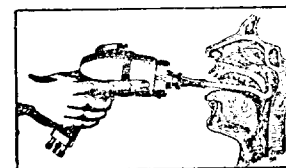
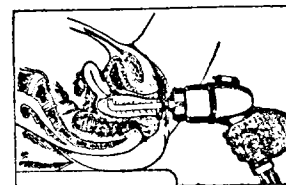
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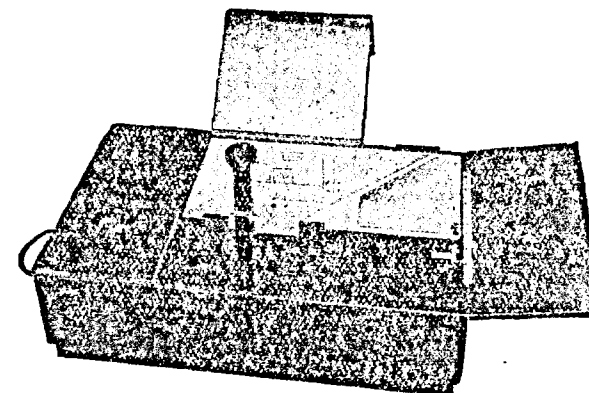
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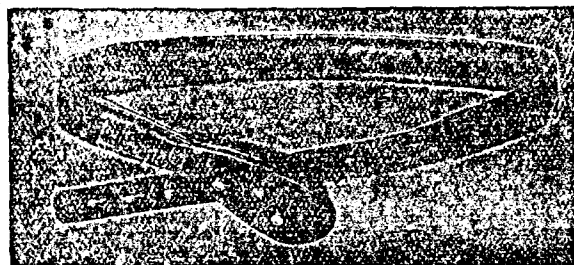
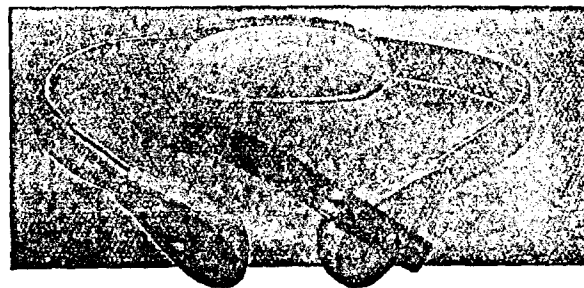
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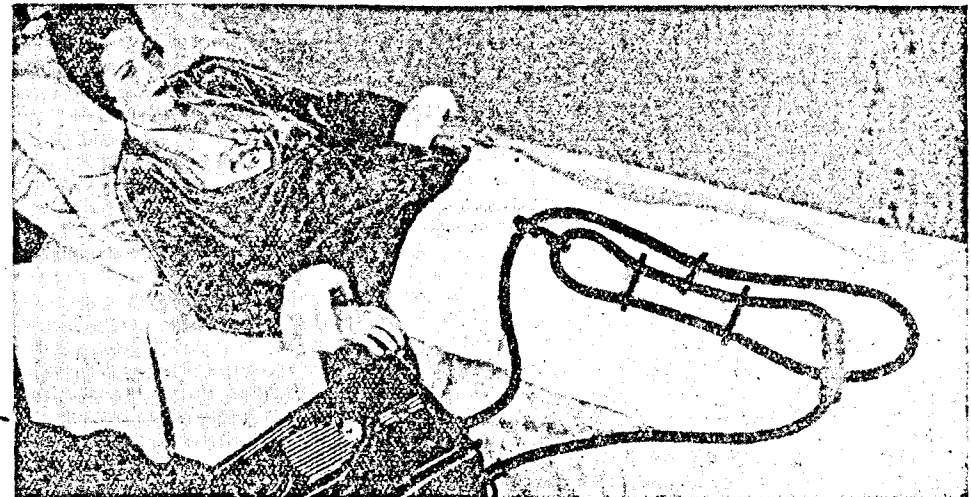
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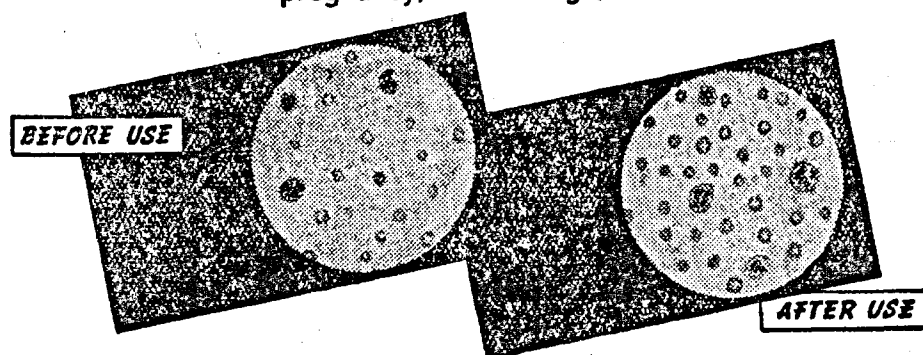
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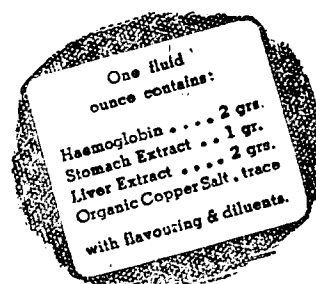
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CALCUTTA :: :: BOMBAY.

Madras Agents:—N. DASAI GOWNDER & CO., 41, Bunder Street.



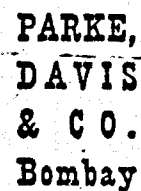
■ Liquid Citralka decreases curd tension when added to whole milk formulae.

Liquid Citralka is indicated for prevention and treatment of acidosis and for alkalizing urine. Common clinical conditions in which **Liquid Citralka** is of value as supplementary treatment include:

Diabetes ————— ectomy and tonsillectomy
in children)

Liquid Citralka is not indicated for gastric hyperacidity.

■ Liquid Citralka is supplied in 8-oz. bottles.

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