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1-55

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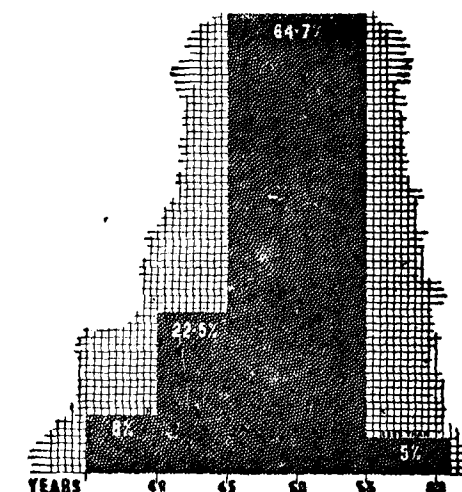
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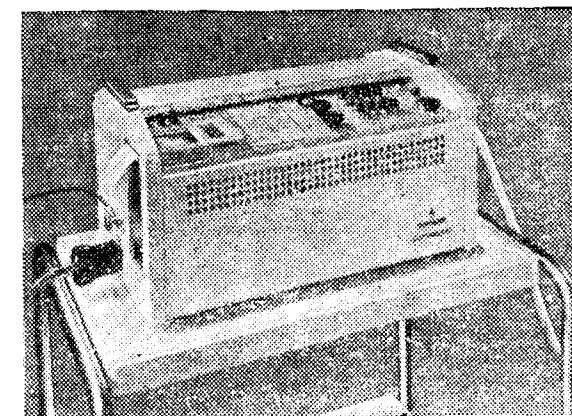
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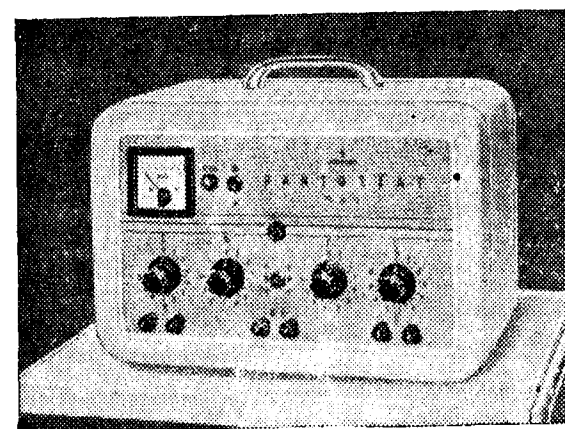


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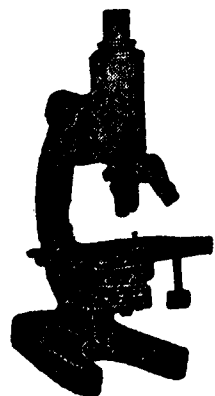
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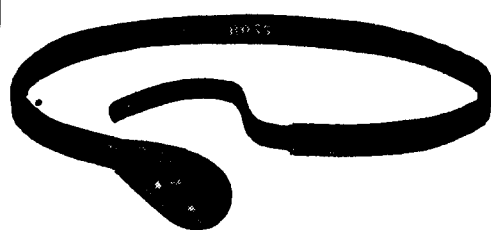
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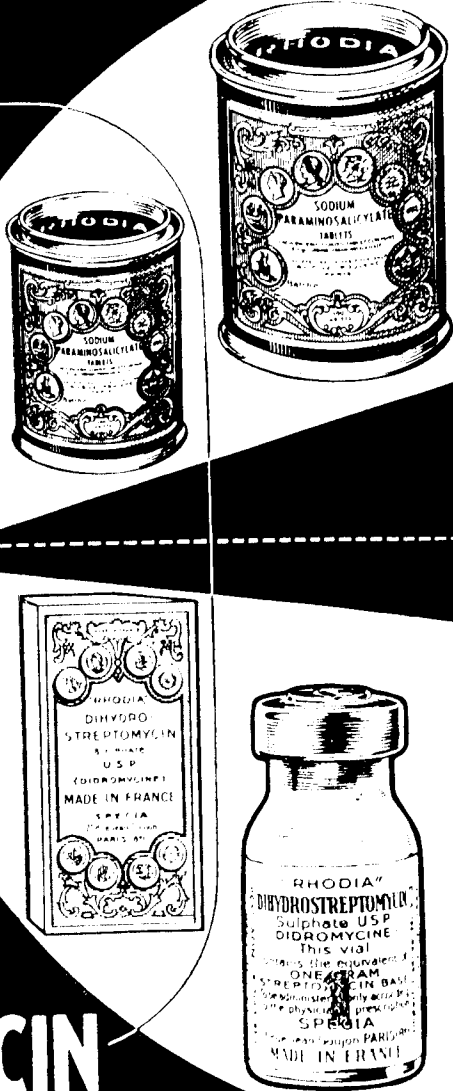
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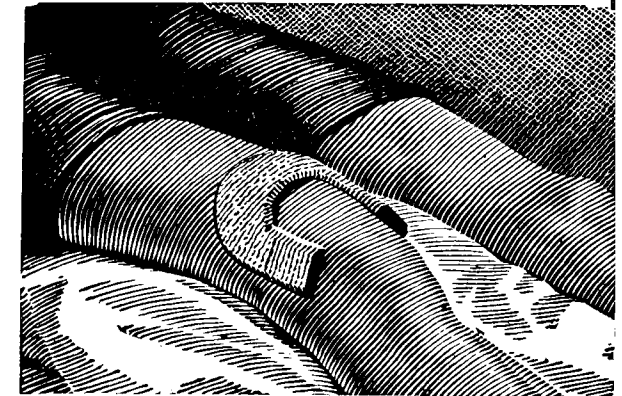
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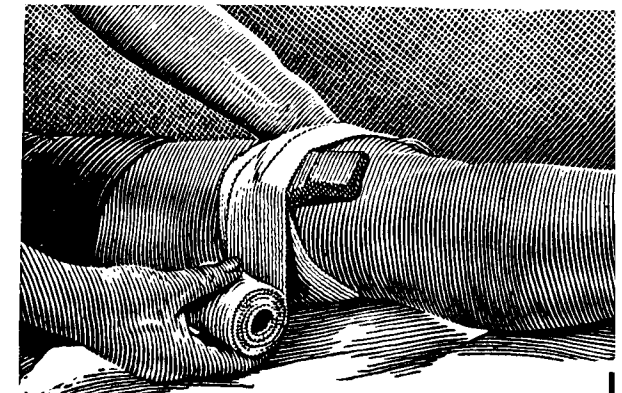
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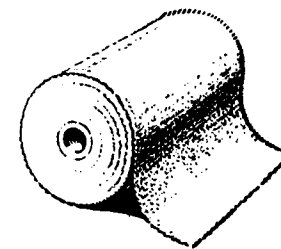
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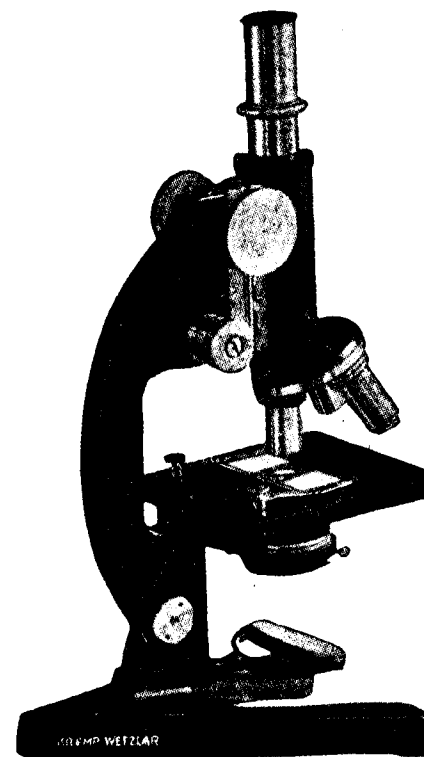
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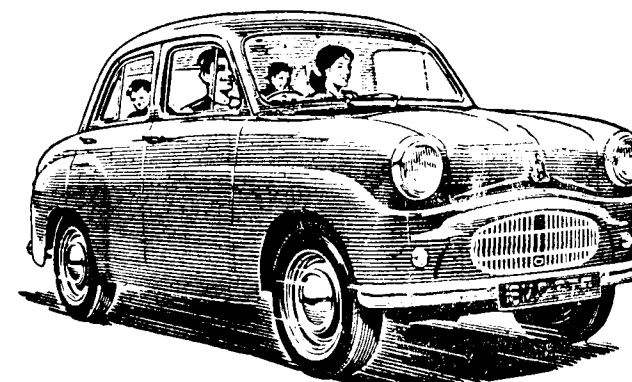
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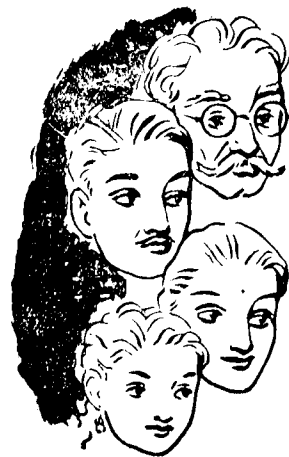
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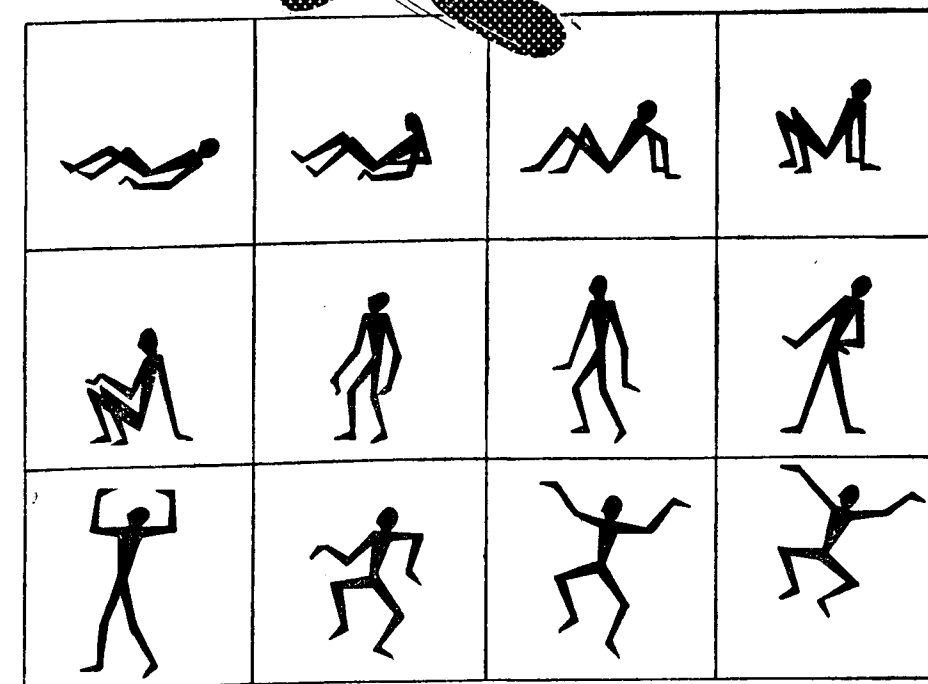
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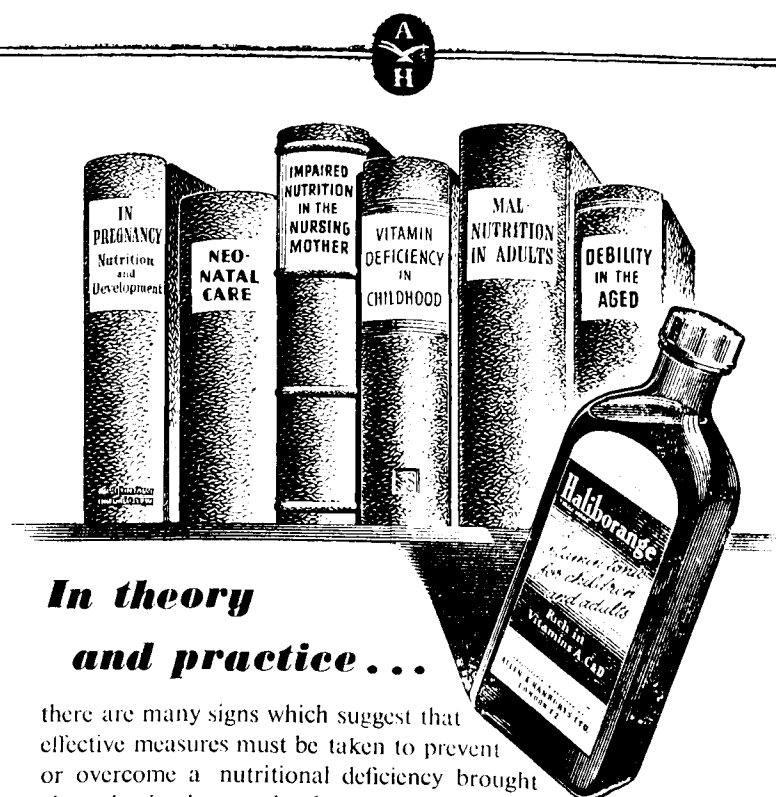
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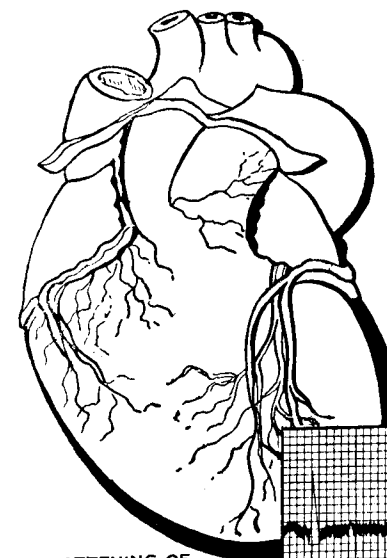
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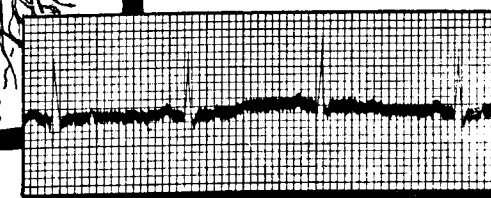
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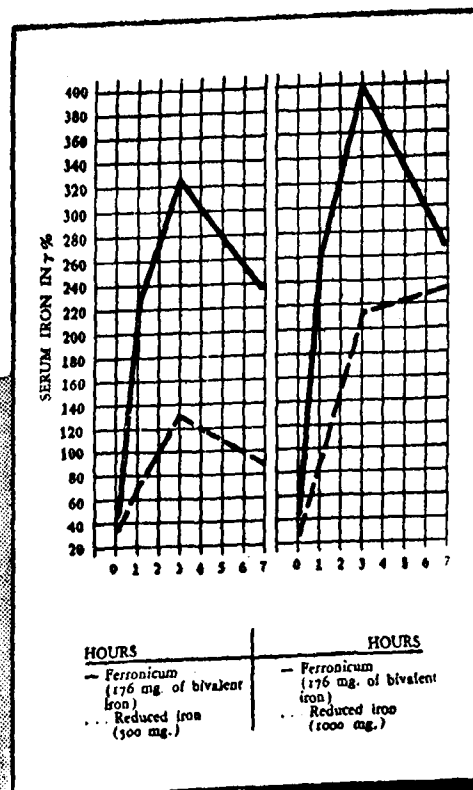
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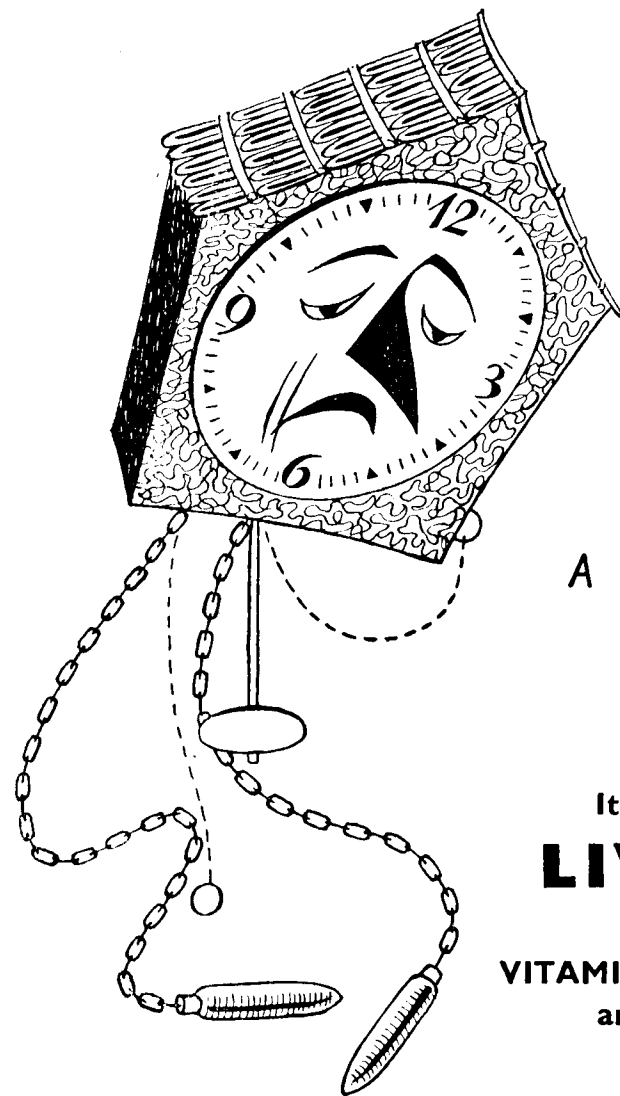
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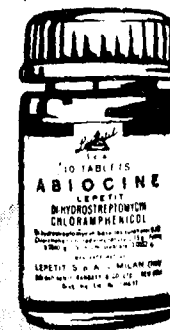
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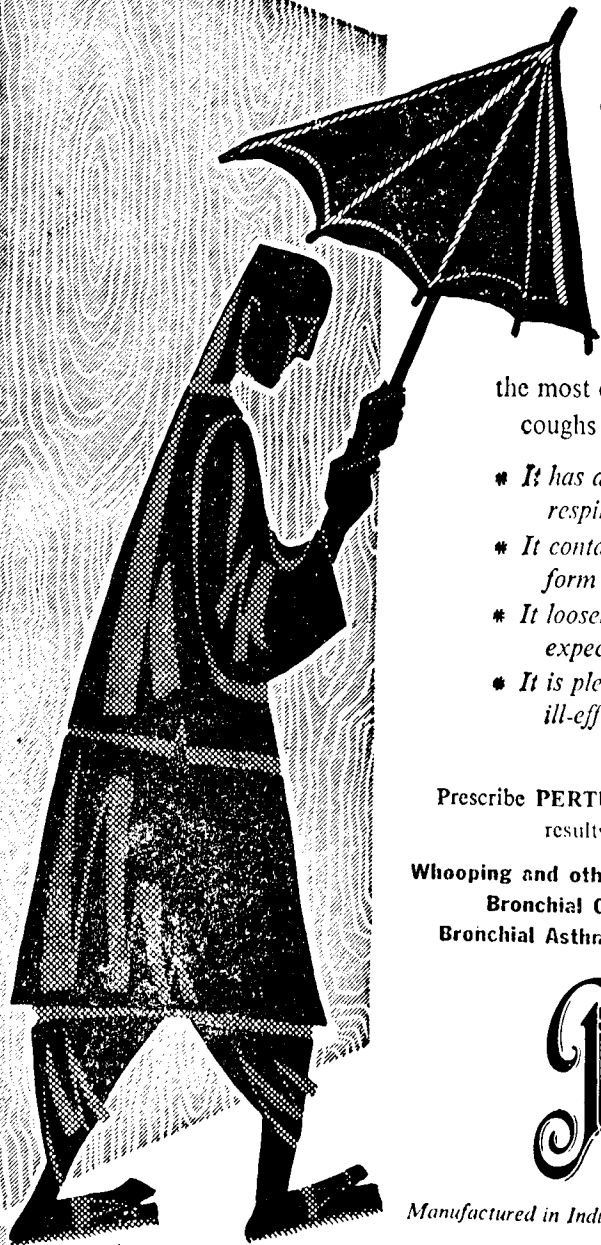
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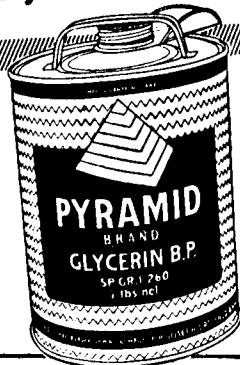
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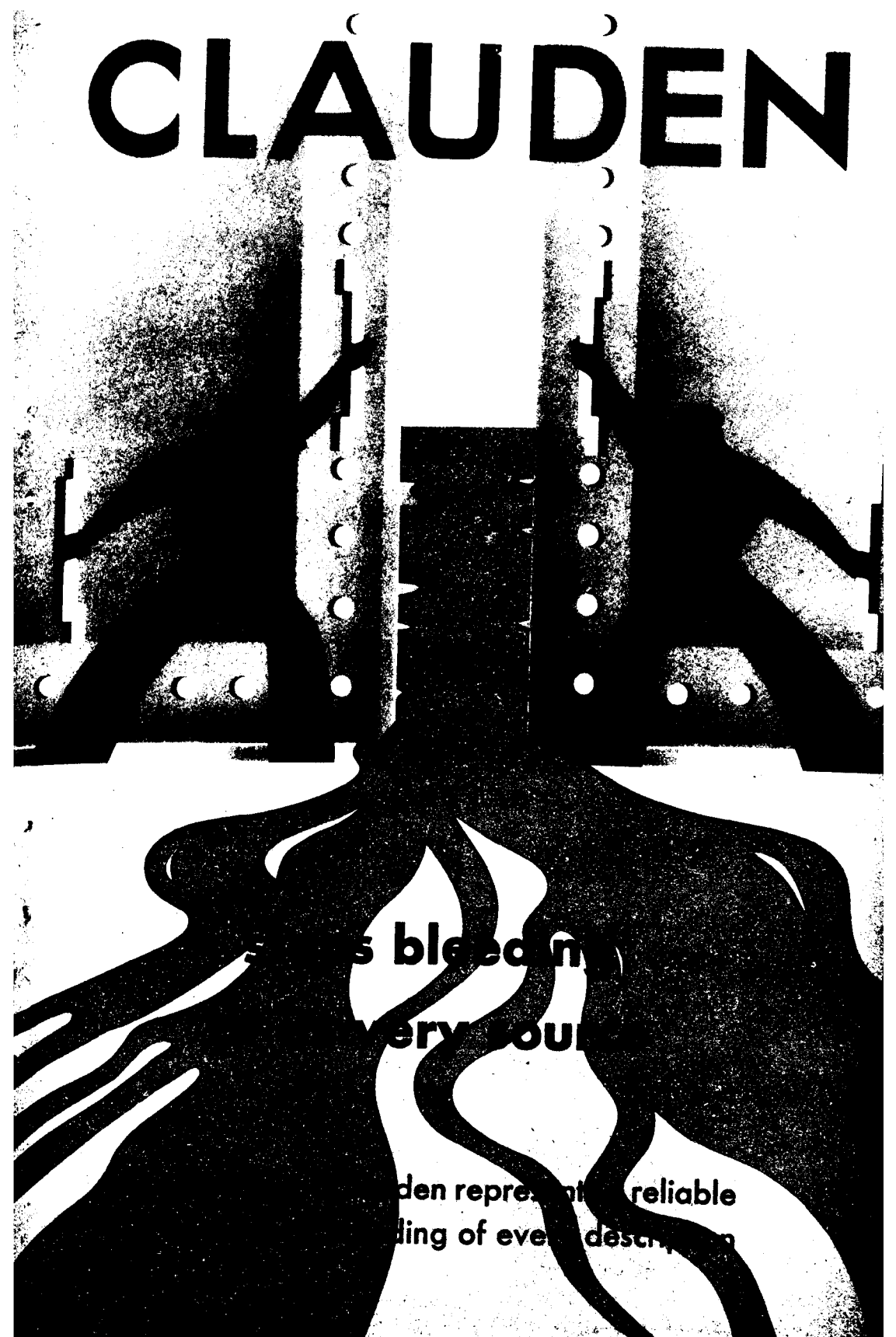
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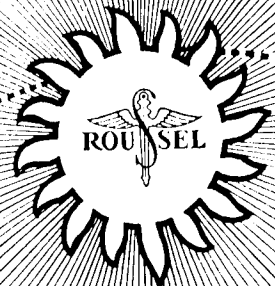
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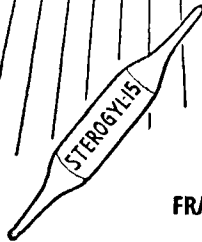
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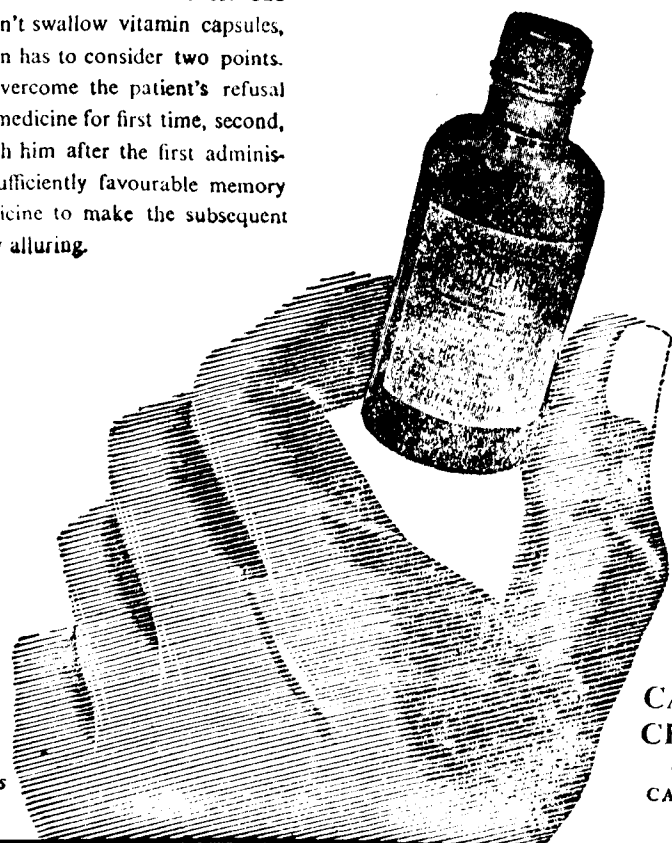
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Original Articles

PEPTIC ULCER*

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PEPTIC ULCER is denudation of tissue which occurs in parts of the alimentary canal which are bathed by gastric juice. Its most constant situation is in the duodenum and stomach. It also occurs in the lower part of the œsophagus when the cardiac sphincter allows the gastric juice to regurgitate constantly into it or in the stoma of gastro-jejunosomy. Sometimes it occurs in the lower part of the œsophagus or in the Meckel's diverticulum when their mucous membrane is heteroptic and resembles that of the stomach.

AETIOPATHOLOGY:—Normally, the gastric juice does not act upon the stomach or duodenum and produce an ulcer. Two factors are essential before an ulcer can form: *viz.*, (1) the presence of hydrochloric acid; and (2) the devitalisation of the mucous membrane of the stomach.

(1) "No acid; no ulcer." Peptic ulcer is practically unknown in cases of Addisonian anæmia in which there is histamine-fast achlorhydria. Histamine stimulates the secretion of hydrochloric-acid. Hay and his associates have experimentally produced ulcers in laboratory animals by daily injections of histamine in beeswax. Shay and his collaborators ligated the pylorus of fasting rats and ulcers occurred in their stomach within about nine hours. Mann-Williamson performed gastro-jejunosomy in dogs in such a way that the secretions of the pancreas and intestines and bile were diverted to the distal jejunum and ileum. The proximal portion of the jejunum which received the gastric juice

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which was not neutralised by the pancreas and intestinal secretions was the seat of ulcer. These experiments prove that excess of hydrochloric acid produced either by histamine injection or not neutralised by intestinal juices could produce ulcers.

(2) Devitalisation of the mucous membrane of the stomach, the other essential for the formation of ulcers, is brought about by several factors and in a given case not one, but many act conjointly. In many cases devitalisation and excess of acid secretion are due to the same aetiology.

One of the most important causes of devitalisation which also causes excess of acid secretion is the psychosomatic factor especially when it occurs in hypersensitive highly strung individuals. The stomach is supplied by two sets of nerves, *viz.*, the vagus and the sympathetic. The former is concerned with the functions of secretion and motility, and is the predominant one. The latter contracts the cardiac and pyloric sphincters. These nerves are governed by the diencephalon—the hypothalamus—which is controlled by the cerebral cortex. Stimuli originating in the cortex under stress, pass to the stomach *via* the hypothalamus and the vagi. In the case of other organs they pass *via* the suprarenals. Keller produced lesions in the hypothalamus in dogs; on account of these lesions ulcers and hæmorrhagic spots occurred in the gastric and intestinal mucosa. Vagotomy done prior to this operation protected the stomach and intestines from ulceration, showing that hypothalamic lesions produced ulceration of stomach and intestines *via* the vagus. Wolf and Wolf have demonstrated that the mucous membrane of the stomach becomes hyperæmic, secretes more hydrochloric acid and shows increased motility under the effect of emotion. Mittleman and Wolf showed that these changes lasted long after the emotions had subsided and continued even during sleep. Emotions therefore, play an important part in hypermotility and hypersecretion, the former causing traumatisation of the mucosa and the latter acting upon this traumatised area producing an ulcer.

In ancient times the Indian's temperament generally was philosophical. This has now given place to worry, resentment and frustration, on account of the keen struggle for existence. Cerebral stimuli due to these emotions are transmitted from the cortex, hypothalamus and vagi to the stomach and duodenum resulting in constant hypermotility and hypersecretion resulting in ulceration.

Another important factor in the production of devitalisation and hyperacidity is 'Food.' People who take sufficient quantities of food containing milk, eggs, fresh vegetables and fruits do not generally get ulcers, because the food contains sufficient amount of proteins and vitamins. Milk and eggs depress excess of acid production. A dietary which is lacking in these foods and which is

made up mostly of starches, combined with the use of an excess of spices and coffee is very conducive to the production of ulcer. Such a diet is found among the poorer people of South India, where the incidence of peptic ulcer is very common. Their food consists of starch—like rice in the Tamil and Telugu districts and tapioca in Malabar, Cochin and Travancore. Though most of them are not vegetarians they have not got adequate means to buy meat, fish, milk and eggs. The rice and tapioca diet is supplemented by *rasam* (pepper water,) pickles and *kuzhambu* (a thick syrup-like liquid made up of dhal, tamarind water, vegetables like brinjal, onions, chillies, condiments, and very little oil to fry the spices; the whole is very well boiled, long enough to destroy all vitamins). This preparation is irritating to the mucous membrane of the stomach and when taken constantly devitalises it. The irritation produces excess of acid; being almost devoid of proteins, the acid combining power of the proteins is lost. The deficiency of fat in this diet leads to the diminution of a normal hormone substance called enterogastrone produced by the upper intestinal mucosa, whose vitality it increases. Ivy *et al* injected enterogastrone intravenously in Mann-Williamson dogs and prevented the formation of jejunal ulcer. The animals also acquired some increased vitality of the mucous membrane as evidenced by the fact that they did not get ulcers for more than 4 months after stopping the injections. This shows that enterogastrone has got a preventive and vitalising effect on the mucous membrane of the intestines.

Dogra carried out experiments using South Indian diets on monkeys for 260 days but found that no ulcers were produced though the animals became emaciated and died of inanition; but these experiments cannot be regarded as conclusive, because the animals died of inanition most probably before the time required for the production of ulcer and also because in the production of ulcer, diet is not the sole factor. Want of proper mastication of coarse food, bacterial contamination of food either on account of staleness, pus from bad teeth, post-nasal dripping from sinusitis, chemical irritation of the mucous membrane following the excessive use of tobacco, coffee, or alcohol are all contributory factors in the aetiology of ulcer. In extensive burns, toxæmia and hypoproteinæmia, are known to cause acute ulcers.

Thus several factors conspire jointly to produce ulcers and no single factor by itself causes a chronic ulcer. The ulcer formed may be shallow and extend up to the submucosa only, when it is called an erosion, or it may extend more deeply. It may be acute or chronic. The acute ulcer has got a funnel shaped or terraced appearance and there is no induration at the base or the edges. It heals generally without leaving a scar. The chronic ulcer has got a rugged base containing muco-pus and an indurated undermined edge. The ruggedness and induration are due to fibrous tissue.

The great majority of gastric ulcers are found near about the lesser curvature of the stomach above the incisura angularis. The lesser curvature not only bears the mucosa producing acid but is also the 'magenstrasse,' or the direct pathway of foods and fluids entering the viscus. A few are found below the angulus, and about one per cent of them are found on the anterior wall of the pyloric end of the stomach. They rarely occur on the greater curvature. Ulcers in the latter two situations are generally malignant. The size varies between 2 to 4 cm. They are generally single, but may be multiple. An ulcer of the stomach and duodenum may be found in the same individual. Ulcers larger than 4 cm. in diameter are mostly malignant. Ulcers situated on the anterior wall are more liable to perforate than those on the posterior wall which generally become adherent to the pancreas, liver or gastro-hepatic omentum.

Duodenal ulcers are smaller than gastric ulcers and are about 0.4 cm. in diameter. They are situated in the first part of the duodenum called the bulb, either on the anterior or posterior wall. Just like the gastric ulcers they may be single or multiple and the ulcers situated on the posterior wall adhere to the same organs, as the gastric ulcers do.

A chronic ulcer whether of the stomach or duodenum may heal completely leaving a scar, it may recur, it may adhere to the neighbouring organs, it may contract producing stenosis especially at the pylorus, it may perforate, and it may erode a blood vessel causing hæmatemesis and melæna. A few gastric ulcers may become malignant. Malignancy of duodenal ulcers is however, unknown.

Incidence and pathogenesis.—Donab in the sixteenth century was the first to describe peptic ulcer, at an autopsy. Abercrombie in the 19th century gave a clear description of 'ulcer' and differentiated it from other gastric lesions. In India, peptic ulcer was considered to be rare until 1922 when Somervell, Bradfield and Pandalai showed, during their operations, that it was quite common in South India. Geographically peptic ulcer occurs all over the world. From autopsy findings, it has been estimated that about 1 in 10 is affected by the malady at some period of life. In the past decade it seems to have increased all over the world, and particularly in India, in response to the stress and strain of the present times and to the fast pace and increased tension of life. This may be partly due also to a failure to distinguish between recrudescence from new disease and improved facilities for diagnosis. The age group is between 20 and 40 years with a peak at about 30, though ulcers before the age of 20 and even during infancy and childhood are not unknown. Both gastric and duodenal ulcers are commoner in men than in women, the ratio being 4:1 for duodenal and about 3:1 for gastric ulcer. This relative greater incidence in the male sex may be due to the greater strain from work and its attendant worries, hurried meals and the greater use of tobacco by them. The relative

immunity of the female may be due in part to the influence of sex hormones, because peptic ulcer is found to be rare during pregnancy. The age of onset is usually earlier in duodenal ulcers than in gastric ulcers by at least 5 years. The duodenal ulcer is primarily a disease of the intelligentsia and businessmen who lead lives of high tension while gastric ulcer is common among the poorer folk, who suffer from malnutrition and mucosal irritation.

In India, Dogra quoted figures in 1939 for peptic ulcer in the different provinces. I do not want to quote them, because he himself says that these figures do not indicate the true incidence of the disease; but the figures for South India, show that it is commoner there than in the North. They also show that it is commoner in the provinces of Madras, Bihar, Bengal and Orissa where the staple diet is rice with a high carbohydrate and low protein content. According to him, the figure for Bombay is 7 per 100,000 of the population. Autopsy reports from the K.E.M. Hospital, Bombay between the years 1925 and 1946 show that the percentage is 3.3 (J. C. Patel). In the B. Y. L. Nair Hospital, Bombay 8,17,016 patients were examined during the 5 years from 1948 to 1952. Out of these 1,126 cases were diagnosed as peptic ulcer. This works out to 7.2%.

SIGNS AND SYMPTOMS:—Pain is the outstanding symptom of peptic ulcer in the great majority of cases. In some cases the pain is very little, depending most probably upon a high threshold of pain in the individual. In some such cases hæmorrhage or perforation may be the first symptom. The pain is due to the action of hydrochloric acid on the inflamed tissues round the ulcer, spasm and irregular peristalsis. In a given case, all or some of these factors are causative. Hydrochloric acid does not cause pain in the normal gastric mucous membrane. Most probably it has got an action upon the swollen tissues and that is why antacids relieve pain. In some cases atropine also has to be given. These are the cases where there is associated spasm and irregular peristalsis.

The pain may cause slight discomfort, dull aching, and burning or it may be colicky. The latter is specially found in pyloric stenosis. It is also crescendo in character. The site of the pain is in the epigastrium but may be found in the right or left hypochondriac regions or in the mid-abdomen in stomal ulcer. It has got a definite relation to the intake of food, and this is diagnostic. The pain increases some time after food. This interval may be short or long. It is generally short in gastric ulcer and when irritating foods are taken, and long in duodenal ulcer, so much so, that in some cases the pain occurs in the early hours of the morning, waking up the patient. This is the typical 'hunger pain' of duodenal ulcer. The pain is decreased by antacids, atropine and vomiting and increased by dilute hydrochloric acid. If in a proved case of peptic ulcer the pain is not relieved by antacids and atropine, the

case is one where adhesions have taken place or where there is stenosis.

An important characteristic of the pain, is its periodicity. There are periods of remission when the ulcer is quiescent and of exacerbations when there is recrudescence. Remissions are of long duration at first, but in the later stages, become short and bouts of pain become more frequent due to indiscretions in diet. In uncomplicated cases, the pain does not radiate, but when the ulcer is adherent to the pancreas or posterior abdominal wall it radiates around the right costal margin or towards the back. The appetite is generally good in early cases, and food may be declined only on account of the fear of pain. In long standing cases, the appetite gets impaired slightly on account of the attendant gastritis. The bowels are usually constipated. If there is diarrhoea, it is generally due to some other complication like amœbiasis, or if after a surgical procedure, to gastro-colic fistula. Vomiting is found during the acute phases and is sour. It is more frequent in gastric than in duodenal ulcer and is suggestive of a spasm at the pylorus. In pyloric stenosis it contains morsels of food taken during the previous days. Vomiting is a reflex act and relieves the pain as it empties the stomach of its acid contents.

Waterbrash is a symptom in which watery fluid is regurgitated from the stomach into the mouth and is due to reflex salivation and acid regurgitation. It is found only sometimes in peptic ulcer and is suggestive of an œsophagitis.

Nausea and flatulence are found in a few cases. The former is due to excessive secretion and the latter due to gastritis and aerophagia. They are more often found in gall bladder disease, than in peptic ulcer.

Anæmia and emaciation are found in long standing cases due to the loss of blood and lessened intake of food.

Physical signs.—These are few. The patient is generally of a spare build. There is tenderness in the epigastrium round about the middle line and this spot remains the same in the same patient. Slight rigidity of the recti is found when the ulcer is adherent to the pancreas which has got the same segmental nerve supply, namely, the sixth and seventh thoracic segments. Greater rigidity is found in subacute or chronic peritonitis when there is a small leak. In cases of pyloric stenosis of some duration peristaltic waves may be seen traversing from the left to the right of the epigastrium and a gastric splash may be obtained even 6 hours after a meal.

Special investigations.—These are: (a) radiology; (b) gastric analysis; (c) occult blood test; and (d) gastroscopy.

(a) *Radiology*.—This should always be resorted to whenever possible and consists of: (i) fluoroscopy and (ii) taking of X-ray films, both of which should be done on an empty stomach, food and drink

being withheld from the previous evening. By fluoroscopy we can learn the type of stomach whether hypersthenic, sthenic or hyposthenic, its position in the abdomen, its rugæ, whether there is any spasm and peristaltic movements. In adhesions, the stomach cannot be moved. The X-ray films are taken afterwards, especially if a pathological lesion is found or if the patient desires it. They are taken in the postero-anterior, oblique, lateral and recumbent positions. These are called serial grams. Yet another method in which the chances of missing a pathological lesion are few, is called spot-filming. It consists of taking a radiographic record of a fleeting fluoroscopic image. Here the radiologist, when he sees the desired image on the screen, releases the cassette carriage and after a fraction of a second depresses the foot switch thus exposing the film.

The direct radiological evidence of duodenal ulcer is a niche which is filled with the barium emulsion interrupting the normal outline. Indirect evidences are persistent deformity of the cap (*vide pictures 1 and 2, page 1349*), a spasmodic contraction opposite to the site of the ulcer resisting peristaltic waves and also the massage by the radiologist, and relieved only by atropine, tenderness over the ulcer area on palpation which may at times increase the peristaltic activity. All these indirect signs together afford fairly good evidence of ulcer. Persistent deformity alone may be due to scarring of an ulcer. Indirect evidence of duodenal ulcer may be found in the stomach also. They are excessive secretion and hyper-peristalsis. Whenever indirect evidences are found and ordinary radiography does not show an ulcer, spot-filming should be resorted to.

The direct evidence of gastric ulcer is a niche projecting outwards from the cavity of the stomach. Indirect evidences are a spasm of the greater curvature pointing towards the ulcer and thickened rugæ. The former may be due to inflammation elsewhere, *e.g.*, the gall-bladder and the latter due to hypertrophic gastritis.

In pyloric obstruction or spasm barium will be found in the stomach even after three hours, and there is hyperperistalsis (*vide picture 3, page 1349*).

(b) *Gastric analysis*.—For this purpose the fractional test meal is employed. The patient takes his last meal before 8 p.m. and takes no drugs. A Ryle's tube lubricated with liquid paraffin is passed through the nose into the stomach with the patient propped up in the sitting position. He is asked, not to swallow his saliva, but spit it in a kidney tray placed by his side. The fasting stomach contents are withdrawn completely, measured, the colour and odour noted and then examined for mucus, bile, blood, starch, food residue, red cells, pus cells, epithelial cells and malignant cells. The free and total acidity are measured and recorded in terms of N/10 Sodium hydroxide. He is then given a pint of thin arrowroot porridge (*conjee*). Specimens are withdrawn at intervals of 15 minutes

for 2½ hours and examined for total and free acidity, abnormal acids, mucus, blood, bile and starch. The patient may be asked to keep himself engaged in reading. The results obtained are charted. A small quantity of the first four specimens are examined with Topfer's reagent which produces a cherry-red colour, if free acidity is present. If free acid is not found, the patient's blood-pressure is taken and he is questioned for a history of allergy and asthma. If the blood-pressure is not high and there is no history of allergy, 0.25 mg. of histamine is given subcutaneously and further samples are taken until the 2½ hours' period is over.

Normally the fastening contents are not more than 25 cc. If the quantity is more than 50 cc., it is suggestive of hypersecretion, pylorospasm or pyloric obstruction. In cases of pyloric obstruction, the odour is foul and food taken on the previous days is found. The examination of the cellular elements should be done as soon as the fasting contents are withdrawn, but even then they might have been already destroyed by the hydrochloric acid, pepsin and duodenal juice. If the red cells are found in large numbers, it is suggestive of a bleeding ulcer or carcinoma. A small number, may be due to trauma caused by the Ryle's tube. A large number of pus cells is suggestive of carcinoma or severe gastritis. Carcinoma cells may sometimes be found, if one is lucky. A small amount of mucus is usually found; but if it is thick and ropy, it indicates gastritis. If bile is not found, pyloric obstruction should be suspected. If, in addition starch is found even after three hours, this suspicion is strengthened.

As regards the acid content, if lactic acid is found it is suggestive of obstruction. If no free hydrochloric acid is found in the first four specimens and acid appears after histamine injection, chronic gastritis is to be suspected (*vide Chart I, page 1351*). If acid does not appear at all, the patient is suffering from either Addisonian anaemia or combined degeneration of the cord. In cases of gastric ulcer, free acidity may be normal, slightly greater than normal or less than normal, except when the ulcer is situated in the pyloric region when, on account of pylorospasm, there will be hyperacidity. Subnormal acidity is suggestive of associated chronic gastritis. In cases of duodenal ulcer the acid curve is of the climbing type, due to the slow emptying on account of pylorospasm or hypersecretion of the stomach. There may be a slight climb-down after the highest point is reached, and this is due to rapid emptying. (*vide Chart II, page 1352*). Increased acidity which may also occur reflexly in gall bladder disease and in chronic appendicitis, is not of the climbing type.

(c) *Test for occult blood* :—This test indicates nothing more than that there is a bleeding somewhere in the gastro-intestinal tract which includes the mouth also. This may be due to ulcer, carcinoma, ankylostomiasis, or bleeding piles and gums. It may not be



PLATE 3. Barium is found in the stomach even after 4 hours' hyperperistalsis of the stomach is seen. In this case X-ray showed tenderness and persistent deformity of duodenal cap. The gastric analysis showed hyperacidity up to 65 munits.



PLATE 1
PLATES 1 and 2.—Per 3-5 cent deformity of duodenal cap with incisula deformity (arrow mark). This patient had typical hunger pain relieved by vomiting which was sour. The hyperchlorhydria with climbing curve (*vide Chart II, page 1352*) is from the same patient.



PLATE 5.—Gastric ulcer seen (greyish area) near the pyloric antrum. This patient got pain in the epigastrium about 1 year ago after eating pickles. It was relieved by milk. Gastric analysis showed hyperacidity rising upto 65 units. X-ray showed tortuous rugosity of the distal part of antrum with gastrosplasm. No ulcer crater seen. Duodenal cap normal.



PLATE 6.—Nodularity is seen. Patient, a woman aged 30 years complained of pain in the epigastrium having no relation to food. Haemetemesis, two kidney trays full. Had such a bout 1 year ago. Histamine fast achlorhydria. Liver function tests normal. Liver biopsy normal. No oesophageal varices. Blood examination showed negative tests for bleeding.



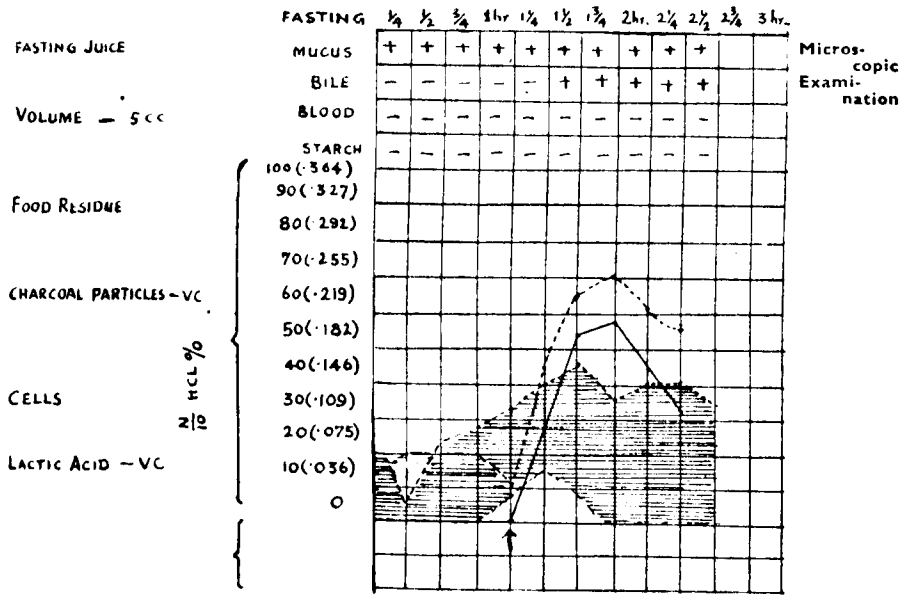
PLATE 4.—Gastric ulcer seen (greyish area). This patient was suffering from burning epigastric pain for 6 years. X-ray showed no gastric ulcer. Gastric analyses showed no acid in the fasting sample and next two samples; administration of coffee produced an acid rise of 50 units, in third sample and 55 units in the fifth sample; acid was normal from the sixth specimen onwards. Operation showed ulcer.

found in ulcers which bleed intermittently. So the stools have to be examined at least on three occasions. Before the test is performed, the patient should not be given haemoglobin or chlorophyll-containing substances like meat, leaves, fruits etc., for three days. The test is performed as follows:—To about 2 cc. of a saturated solution of benzidine in glacial acetic acid an equal quantity of 3% hydrogen peroxide is added and 2 or 3 drops of boiled and cooled faecal emulsion. A blue colour developing within 2 or 3 minutes indicates the presence of occult blood.

(d) *Gastrosocopy*:—By this means direct visualisation of the inside of the stomach is possible; a tube is inserted, the upper part of which is rigid and contains an eye-piece at the upper end and the lower part flexible, and made of rubber with a finger-like end which guides the instrument while it is passed and which also contain an electric bulb for lighting up the inside of the stomach. The flexible rubber part contains a number of lenses which reflect the image of the part which is viewed and also a device for inflating the stomach. The Herman Taylor's modification of Schindler's flexible gastroscope is the instrument which is generally used. The

CHART I

Achlorhydria with acid secretion after histamine (arrowmark). This patient had vague pains in the abdomen. Appetite was not good; hookworm ova were found in the stools.

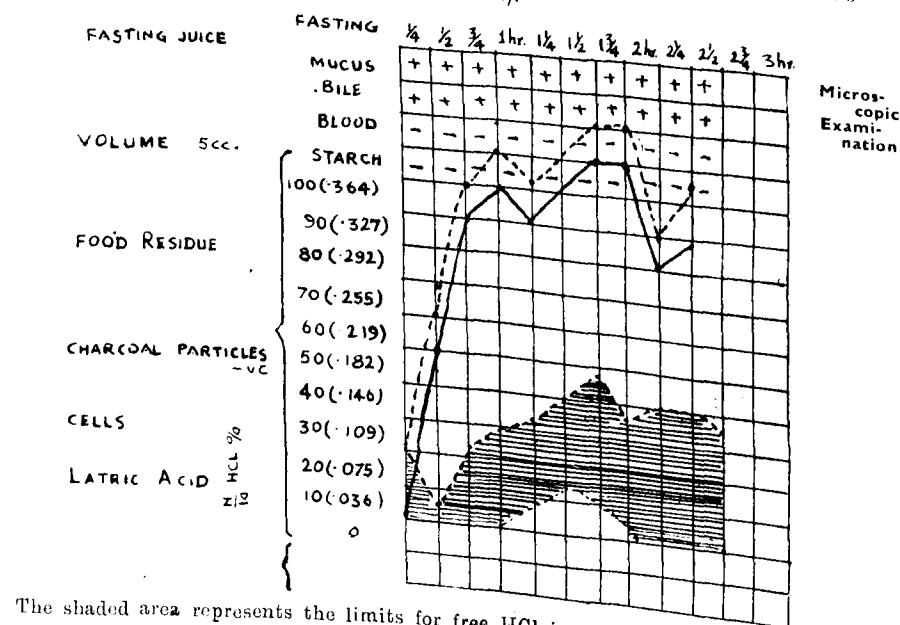


Represents free HCl
..... Represents total acidity.
Test meal employed:—500 cc. of oat-meal gruel without milk or sugar.
The meal was given after a fast of ——— hours.

patient is starved over-night, and the gastric contents are aspirated on the next morning. The examination is conducted in a dark-room. Artificial dentures if any are removed and the patient asked to gargle and swallow 2 cc. of 2% anethaine about 5 minutes before the passage of the instrument. This anaesthetises the throat and oesophagus. The patient is placed in the left lateral position, the head is supported by an assistant who also blindfolds him by covering his closed eyes with a towel. The knees are flexed over the abdomen. Another assistant holds the instrument which has been lubricated by K.Y. jelly, in a horizontal position and passes it on to the gastroscopist who supporting the instrument with his left hand passes it on to the stomach of the patient guided by the middle and fore-fingers of your right hand which are placed in the patient's mouth. The stomach is then lighted up, inflated and observed in different parts. A simple ulcer is seen as a depressed area greyish in colour (*vide plates 4 and 5, page 1350*) except when seen after a recent hæmorrhage when it is brownish. A malignant ulcer has got indurated edges and a dirty brown nodular base. Sometimes only nodularity is seen. This may be due to malignancy or polypi (*vide plate 6, page 1350*).

CHART II

Climbing curve with a slow fall after reaching highest point. This patient's X-ray showed duodenal ulcer. (*vide plates 1 and 2*).



The shaded area represents the limits for free HCl in 80% of normal people, and average rate of emptying (2-2 1/2 hours).

..... Represents free HCl
 Represents total acidity
 Test meal employed—500 cc. of oat-meal gruel without milk or sugar
 The meal was given after a fast of ——— hours.

Gastroscopy is of use in revealing shallow ulcers which are not revealed by radiology as the barium does not flow into them, in distinguishing to some extent between a simple and malignant ulcer, and in the follow-up of the healing of an ulcer. It has been found by gastroscopy that when an ulcer is symptomless it does not always mean that the ulcer has healed, but it is only in a quiescent stage.

Gastroscopy is of no use in the case of duodenal ulcers, because the gastroscope cannot pass through the pylorus into the duodenum. It cannot also visualise the fundus and the lesser curvature of pyloric antrum. It cannot be done in non-cooperating patients and in patients who have heart disease or curvatures of the spine. The other contra-indications are those in which a stomach tube should not be passed, *e.g.*, oesophageal disease, aneurysm of the aorta, and corrosive gastritis.

DIFFERENTIAL DIAGNOSIS:—Careful history taking and assessment of symptoms are more important in the diagnosis of peptic ulcer than physical examination which may not reveal anything of importance. Laboratory investigations should always be done whenever possible to confirm or disprove the provisional diagnosis made at the clinical bedside-examination; but too much stress should not be placed on them, to the exclusion of clinical diagnosis. A good rule which is very useful where laboratory methods are not available is, that if in a case suspected to be one of peptic ulcer, relief is not obtained after three weeks' treatment, by rest, ulcer-diet and antacids, the disease is not simple peptic ulcer.

The following diseases have to be distinguished from peptic ulcer:—

(1) *Chronic gastritis*:—A history of alcoholic excess may be elicited. There is loss of appetite, bad taste in the mouth, nausea and vomiting of mucus especially in the mornings. Hæmatemesis may occur if there is associated oesophageal varices due to cirrhosis of the liver. On examination there is a furred tongue, no definite tenderness in the epigastrium; an enlarged liver may be palpated in the right hypochondrium if there is early cirrhosis of liver.

(2) *Carcinoma of the stomach*:—When a person, especially a man on the wrong side of forty, who had a very good appetite complains of sudden loss of appetite, loss of weight, vague pains in the abdomen and pallor, malignant disease has to be thought of. Later, coffee grounds vomit and swelling in the abdomen may be found.

(3) *Nervous dyspepsia*:—Here the history is of a multiplicity of symptoms inconsistent in character. A psychological cause may be at the root of all the abdominal troubles, and when these are remedied the dyspepsia disappears automatically. The pain has got no relation to the food intake and there is ærophagy. On examination the tenderness is diffuse over the abdomen.

(4) *Chronic cholecystitis*:—A history of jaundice, or acute gall bladder disease is obtained in some cases. The pain is not so closely related to meals as in peptic ulcer. Flatulence is a marked symptom especially after ingestion of fatty foods, and vomiting is more common than in peptic ulcer. On examination the patient is generally a woman rather fatty for a peptic ulcer patient. The tenderness is to the right of the middle line, increased on deep breathing and a little rigidity of the rectus is found.

(5) *Chronic appendicitis*:—A history of acute appendicitis is found in a number of the cases. Constipation is marked and the pain is aggravated after purgatives; these should not be taken least an acute attack be provoked. On examination tenderness is found both in the epigastrium and in the right iliac fossa, generally more pronounced in the latter region.

(6) *Diseases of the colon*:—The history is of pain in the mornings relieved by defæcation. The tenderness which is found in the region of the descending colon is greater than in the epigastrium.

(7) *Ankylostomiasis and chronic dysentery*:—People who suffer from these diseases generally reside in places where promiscuous defæcation is the rule. In cases of chronic dysentery a history of acute dysentery is elicited in most cases. There is anaemia in ankylostomiasis. Stools examination after a saline purgative reveals the cysts of *entamoeba histolytica* or *giardia lamblia* in chronic dysentery and ova of hook-worm in ankylostomiasis.

(8) *Hiatus hernia*:—This is a rare disease and has to be thought of in cases of supposed peptic ulcer in which the ulcer is not revealed by X-rays and treatment is ineffective. This is a condition in which there is herniation of the stomach into the thorax through the oesophageal hiatus. The patient complains of pain in the epigastrium after food which increases after lying down, becoming less on standing up. There is also some dysphagia. Definite diagnosis can be arrived at only by radiology, with the patient lying down in the supine Trendelenburgh position and other postures.

(9) *Gastric allergy*:—In this condition certain kinds of food especially proteins, like milk and eggs produce pain, the stoppage of which causes remission of the pain. There may be an eosinophilia in the blood. The patient should be asked to maintain a diary to find out which foods cause pain.

(10) *Gastric syphilis and gastric tubercle*:—These are rare. Ulcer diet does not relieve the pain. In the former disease, the blood is strongly positive to Kahn test and antisyphilitic treatment gives relief. In the latter disease, there may be tuberculosis elsewhere in the body. Dihydro streptomycin parenterally produces prompt amelioration of the symptoms.

(11) *Pancreatic disease*:—This may be due to a lack of enzymes, or chronic pancreatitis, pancreatic calculi or cancer. Deficiency of enzymes causes vague pains in the epigastrium and these are relieved by supplying the enzymes. In chronic pancreatitis, there is a large quantity of neutral fats in the stools. In cancer, the pain is very severe and there is jaundice. In stones, there may be only very severe pain, and sometimes an excess of neutral fat in the stools. X-rays alone will help in diagnosis.

(12) *Ascariasis or round-worm infection*:—In this condition the pains are colicky and vomiting is generally present. A round worm might have been passed in the vomit or motions and microscopic examination of the stools reveal the ova of ascaris.

(13) *Dyspepsias due to diseases elsewhere in the body*:—Diseases of other organs, e.g., tuberculosis of the lungs on account of the toxæmia, congestive cardiac failure, nephritis and anaemia caused by circulatory disturbances may produce diseases of the stomach. Nervous diseases like tabetic crises and diseases of the spine by pressure on the spinal cord, may simulate the pain of peptic ulcer. So in all obscure cases having abdominal pain, the knee jerks, and pupils have to be examined. In tabetic crises the knee jerks are lost and there is Argyll Robertson's pupil and the blood is positive to the Wassermann test. In diseases of the spine there is tenderness in the dorso-lumbar region and X-rays show the lesion.

Complications.—These are: (a) hæmorrhage; (b) perforation; (c) stenosis; (d) alkalosis; and (e) malignancy in a very few gastric cases, especially when they are in the prepyloric area and greater curvature.

(a) *Hæmorrhage*:—Small bleedings and occult blood are the causes of anaemia. Large hæmorrhages occur in acute ulcers or in chronic penetrating ulcers eroding a vessel at the base. The symptoms are hæmatemesis and mæna which result in giddiness and faintness. In favourable circumstances a clot forms in the retracted ends of the vessel. In arteriosclerotic vessels retraction does not occur, hæmorrhage continues and operation has to be resorted to. Generally speaking, in people below 40, operation is not necessary if it is the first hæmorrhage.

(b) *Perforation*:—Occurs more in duodenal than in gastric ulcer, especially in those situated on the anterior wall. Excessive smoking is sometimes an exciting cause. There is severe pain in the epigastrium which extends to the hypogastrium. The face shows anxiety. The temperature is subnormal, the body is cold, the pulse rate rises, and the volume falls. This is the stage of collapse which is succeeded by the stage of reaction when the patient looks better, though he is actually getting worse. He lies down with the thighs flexed or sits up so as not to stretch the abdominal wall, which is tender and rigid. The respirations are shallow and

rapid. Rectal examination reveals tenderness and a swelling. This stage is followed by one of frank peritonitis in which the pulse is fast and feeble, the abdomen is distended and the facies is hypocritical due to dehydration.

(c) *Stenosis*:—In this condition there is severe colicky epigastric pain relieved by vomiting which brings out food taken on the previous day. There is wasting and constipation is obstinate. Visible peristalsis is seen and gastric splash elicited even six hours after meals. Alkalosis occurs if vomiting is severe. In pylorospasm visible peristalsis is generally not seen and the symptoms are relieved by atropine. In hour-glass stomach also peristalsis is not generally seen and complete relief is not obtained by vomiting.

(d) *Alkalosis*:—This complication occurs in prolonged treatment by antacids especially sodium bicarbonate. Incessant vomiting, anæmia and kidney disease predispose to it. There is first of all a distaste for food especially milk. Then there is nausea and vomiting. Nervous symptoms like headache, giddiness and irritability are present. Respiration is slow, pulse is rapid, the urine is alkaline and contains albumin. Tetany may be present in severe cases.

Prognosis.—This is good in persons who are cooperative and can follow the treatment faithfully. Economic factors sometimes stand in the way of proper treatment; but I think the poor man who cannot afford to spend much on milk and eggs can at least clean his teeth properly, forego his tobacco, *pansupari*, spices and strong coffee. I have found, that this is not done in very many cases.

If not properly treated, the ulcer becomes chronic and one of the complications mentioned is likely to occur. Operative procedure, *viz.*, partial gastrectomy by itself as treatment for ulcer is not always successful as the acidity cannot be reduced in about half the number of cases sufficient to prevent the occurrence of stomal ulcer. Dietetic treatment should be followed after operation to prevent stomal ulcer.

Treatment.—1. **PREVENTIVE TREATMENT**:—A life of contentment is essential. This does not mean that there should be no ambition in life; but if one endeavour fails after serious attempts the failure should be taken calmly, philosophically and sportingly. Constant excesses in food and drink should be avoided. The teeth should be looked after carefully and food masticated properly. Milk and eggs or at least one of them must be taken every day; spices and tobacco should not be taken in excess or on an empty stomach. In cases of hyperchlorhydria deep X-rays have sometimes been effective in reducing the acid and thereby preventing the formation of ulcer.

2. **CURATIVE TREATMENT**:—This may be (A) medical or, (B) surgical. Medical treatment will be described in detail and surgical treatment will only be briefly outlined in this paper.

(A) *Medical treatment*.—This should be resorted to in all cases of uncomplicated ulcer. Economic factors should not be considered a contra-indication for this treatment. As has already been mentioned about 50 per cent of the cases get stomal ulcer after operation if strict dietetic treatment is not followed. Medical treatment may be divided into: (i) general, (ii) dietetic, (iii) medicinal, and (iv) other measures.

(i) **GENERAL**:—The patient should have physical and mental rest for at least 4 weeks, and should be in bed without worrying about his profession or business avocations.

(ii) **DIETETIC**:—*First week*:—This consists of the administration of milk, 5 ozs. every two hours from 7 a.m. to 10 p.m. every day. If there is vomiting, the milk may be citrated by the addition of 1 grain of sodium citrate to an ounce of milk. A cup of milk is kept by the bedside to be taken if awakened at night.

Second week:—If the pain is still severe, unrelieved after a week by this diet and medicinal treatment, a milk drip is instituted. The patient is propped up in bed. A Ryle's tube lubricated with 2 per cent anethaine ointment, is passed through the nose and the stomach aspirated with the syringe attached to the tube. The Ryle's tube is then withdrawn a little, so that its end is just past the cardiac end of the stomach. To it, is attached at a height of about 2 feet a transfusion bottle containing a pint of milk to which an antacid like magnesium trisilicate has been added. The milk is allowed to drip into the stomach at the rate of 40 drops a minute. If the pain is relieved the milk is given as in the first week and as an additional diet thereto, an egg morning and evening is given as plain flip, or half boiled but never fried. Five ounces of filtered fruit juices are also given twice a day.

Third week:—If the pain has subsided, well boiled rice *comjee* or soft bread with butter, vegetable purée may be added to the diet of the second week.

Fourth week:—Double boiled rice and biscuits may be added to the diet and non-vegetarians may take fish which is boiled but not fried.

During the fifth and sixth weeks, the patient can sit up and be ambulant within the house.

Post-ulcer-regime:—After the sixth week he can begin his normal duties if the pain has completely subsided. The two-hourly milk feeds should not be forgotten and he should take a bottle of milk when he goes to work. An hour's rest at lunch time and a day's rest at the week-end, should be religiously observed. Weak coffee and tea are permitted. Spices are to be avoided for three months and later taken only in extreme moderation and never on an empty stomach. All food even if it is soft should be masticated properly. The teeth are to be cleaned morning and night and kept

in good order. Alcohol should be completely avoided; but if abstinence from it makes the patient miserable, a small quantity of light wine may be taken after meals from the sixth month onwards but only occasionally. Similarly smoking may be resorted to after six months, one cigarette or *beedi* after each meal. Seeds and skins of fruits should be scrupulously discarded. The life generally should be peaceful and one of moderation.

If there is recrudescence of pain, recourse should be had to the ulcer regime for at least a week or until the symptoms have again subsided completely and then normality should be resumed slowly. An ulcer takes a long time to heal and subsidence of symptoms simply means that the ulcer is in the healing stage.

(iii) MEDICINAL TREATMENT:—This consists of administration of antacids, antispasmodics, sedatives and vitamins.

Antacids:—A combination of the carbonates of bismuth, magnesium, and calcium (10 grains of each) is the cheapest and best. This powder is mixed with water and given every two hours six times in the day in between the milk feeds. Bismuth carbonate must be reduced if there is constipation. If it still persists, a mixture of half an ounce of liquid paraffin and olive oil is given at bed time. In addition to its laxative properties, olive oil depresses acid secretion and is nourishing also. If there is diarrhoea the magnesium carbonate is reduced. If it still persists a complication like amoebiasis or sprue must be looked for. Magnesium trisilicate may be given, a teaspoonful three times a day, in the post-ulcer regime. Sodium bicarbonate should *never* be given.

Antispasmodics:—These drugs should be given if there is pylorospasm with its attendant hyperacidity, pain and vomiting in spite of ulcer diet and antacids. The drugs which are commonly used are atropine, bellafoline and traserentin. The action of all these drugs is to relieve spasm, motility and gastric acidity. Traserentin has very little of the side-effects of atropine, *viz.*, mydriasis and drying-up of the throat. A tablet of bellafoline or traserentin or one-hundredth grain of atropine may be given three times a day. Centriline (Bristol Labs.) in doses of 0.5 mg. to 1 mg. three times a day is a new synthetic drug which is said to be one of the best antispasmodics without the side-effects of Banthine, another synthetic drug used for the same purpose. All tablets are to be crushed and given to swallow.

Sedatives:—These are useful particularly when the person is much worried either on account of the disease or for other reasons. Phenobarbitone $\frac{1}{2}$ to 1 grain three times a day should be given in addition to the antacids.

Vitamins:—These are strictly accessory foods, and should be given when there has been starvation on account of excessive vomiting or from the second week onwards even in ordinary cases

of peptic ulcer. The vitamin tablets which should be crushed and given are vitamin C and vitamin B complex. Hundred milligrams of vitamin C and 1 tablet of vitamin B complex should be given three times a day.

(iv) OTHER MEASURES:—Septic teeth and other septic foci in the tonsils and sinuses should be attended to. Artificial dentures may be used to make good the loss of teeth.

(B) *Surgical treatment* should be resorted to: (a) in chronic ulcers which have become adherent to the neighbouring organs; (b) in chronic gastric ulcers in the prepyloric region and greater curvature when the patient is above 40; these ulcers do not generally heal with medical treatment because that part of the stomach is never at rest but have a tendency to become malignant; (c) in cases of pyloric obstruction and hour-glass stomach; (d) in cases of perforation; (e) in cases of hæmatemesis or melæna which is recurrent and the patient is above 40 years of age. When the patient is below 40 and it is the first bleeding, rest in bed, ulcer-diet, and blood transfusions may be tried before resorting to operation.

The type of operation to be performed should be decided by the physician and the surgeon in mutual consultation. The patient is made fit for the operation by blood-transfusion, parenteral glucose, saline, plasma and vitamins. This is specially necessary before operation is done for complications. The operation of choice is partial gastrectomy which removes a large portion of the acid-producing areas of the stomach. If the patient is old and debilitated, gastro-enterostomy with vagotomy to reduce the acids should be done.

A few complications may occur after surgery, *e.g.*: (1) dumping syndrome; (2) stomal ulcer; and (3) gastro-jejuno-colic fistula.

(1) *Dumping syndrome*:—After intake of food the patient feels a sensation of warmth, pain in the epigastrium and giddiness and he perspires. There may be nausea, palpitation and a feeling of collapse. These symptoms are due to the rapid distention of the jejunum by the passage of food from the stomach into it through the stoma and also to hypoglycæmia following the hyperglycæmia due to rapid absorption of carbohydrates. Treatment consists of giving food in a solid form, fluids being taken only between meals. Atropine, one-hundredth grain, given half an hour before meals aborts the symptoms.

(2) *Stomal ulcer*:—This occurs after gastro-enterostomy either on account of the patient not following ulcer-diet or due to the septic foci not having been attended to. The symptoms are pain in the mid-abdomen and hæmatemesis with melæna. The ulcer-diet should be begun at once and if there is no relief after about three weeks, vagotomy must be performed.

(3) *Gastro-jejuno-colic fistula*:—This complication is attended by fatty diarrhoea and fecal eructations and vomiting. The treatment is surgical, *viz.*, partial gastrectomy.

Summary.—1. The aetiology of peptic ulcer is reviewed and special stress is laid on psychosomatic factors and irritating diet containing very little protein.

2. The increase in the incidence of peptic ulcer as revealed by hospital statistics is mentioned.

3. The importance of gastroscopy in the diagnosis and in the follow-up of the healing process in gastric ulcer is noted.

4. Due importance should be given to prophylactic treatment in peptic ulcer.

5. Medical treatment is the treatment of choice in simple uncomplicated peptic ulcer, except in prepyloric cases or ulcers on the greater curvature, when operation must be resorted to, because they do not heal at all by medical measures.

6. The continuation of the ulcer-diet after surgery is necessary to prevent stomal ulcer.

My thanks are due to the Bombay Municipal authorities for allowing me to do research in gastro-enterology and to Dr. C. A. Paes, M.B., B.S., for helping me in my work.

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PERFORATED PEPTIC ULCER

Noordijk made a survey under the auspices of the Netherlands Surgical Association, covering 33 clinics reporting 2550 cases of perforated peptic ulcers. 324 died giving a mortality rate of 13.4%. Interestingly enough, the mortality was highest during the last year of the war when all conditions were unfavourable for patients as sick as those with perforating ulcers. In the period from 1934 to 1939 the mortality of 608 patients was 15%. In the period from 1940 to 1945 the mortality for a group of 1133 patients was 17.4%. In the last period 1946-1949, the 810 patients considered, showed a mortality of 7%. The comparison primarily related to the efficacy of simple seroseros closure in contrast to primary resection. Obviously there is a fallacy in comparing only the mortality rates for the total groups treated by each method, as primary resection would be used only in patients who were in a fairly good condition, whereas patients who were desperately ill would be designated for simple closure only. When equivalent groups, *i.e.*, those in the same age bracket and with the same duration of perforation, were compared the mortality was essentially the same for the 2 types of procedures.

The leading cause of death was still peritonitis in the patients who died. In simple suture cases, a surprising number of patients succumbed to after-bleeding. The late results as judged by the recurrence of ulcer symptoms and the necessity for subsequent operation were very much better in patients treated by resection.—(*Internat. Abst. Surg.*, 99: 3: Sep. 1954, from *Arch. Chir. Nederl.*, 5: 262).

PROLAPSE OF THE RECTUM*

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PROLAPSE of the rectum is a protrusion of the rectal wall through the anus. Various classifications have been made both with regard to pathological anatomy as well as to the degree of prolapse but the simplest and the most convenient classification is to divide prolapse of the rectum into 'partial' *i.e.*, when the protrusion is limited to the mucous membrane of the rectum and 'complete' when all the layers of the rectal wall are involved. More accurate terms would be 'mucous membrane prolapse' and 'all-layer prolapse' which do not entertain the suggestion of the degrees or stages of prolapse implied by the words 'partial' and 'complete.'

Partial prolapse is usually found in children and less commonly in the aged and infirm. In children in whom it is more common in those under 5 than in others, the predisposing causes are the natural absence of the sacral curvature and an acquired reduction in the supporting fat of the ischio-rectal fossæ brought on by malnutrition and wasting diseases. A certain amount of protrusion of the mucous membrane during the act of defaecation is a normal phenomenon, but if this is repeated due to diarrhoea or dysentery and tenesmus and constant straining it is likely to progress into prolapse. Ill advised and frequent administration of purgatives and lack of proper training in bowel action are responsible for some cases of prolapse. In others, prolapse may be brought about by straining due to phimosis or whooping cough. When found in adults it is almost always a continuation of the condition from childhood or as a sequel to prolapsed piles or laying open a high anal fistula. Again some degree of partial prolapse may be found associated with the constant straining due to an enlarged prostate or a stricture of the urethra. In certain neurological conditions where the anal sphincter is relaxed, some partial prolapse is found.

In children, apart from the concomitant condition which caused the prolapse, there may not be any symptoms, the prolapse spontaneously reducing itself. Rarely there may be bleeding from the prolapse, particularly when it does not reduce spontaneously. In adults, the prolapse usually disappears after defaecation or when straining ceases, but in long standing cases with stretched-out relaxed sphincter the prolapse tends to remain outside and may give rise to bleeding from distended veins or discharge of mucus and pruritus. In other cases there may be irreducibility due to oedema, or

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ulceration may take place which has to be distinguished from malignancy by biopsy if necessary. One occasionally comes across a moribund case with shrunken gangrenous dirty mucous membrane protruding from the anus.

Complete prolapse.—As compared to the incidence of partial prolapse, complete prolapse is rare, although it is said to be not uncommon in the mentally deficient. Within the last 50 years, Moschowitz's theory propounded in 1912 that the condition is of the nature of a sliding hernia of the rectum through the pelvic diaphragm has generally been the accepted view. In children and young adults probably a congenital weakness or defect in the muscles of the pelvic diaphragm along with a low placed pouch of Douglas probably exists. When it occurs in middle age persons it may be due to a generally relaxed condition of the pelvic musculature due to sedentary habits and fatty infiltration, much in the same way that sliding or direct hernia occurs in the inguinal region. In women, injury to or too much stretching of the pelvic floor at previous child-births may be contributory also; in fact prolapse of the rectum may be associated with prolapse of the uterus. Again as in the case of any other hernia, increased pressure from above on a weakened pelvic floor due to straining or coughing of any kind will be an exciting cause.

In complete prolapse there is prolapse of two layers of all the coats of the rectum and when it is 2" or more in extent there is in front between the two layers the slid-down pouch of Douglas containing coils of the small intestines. The diagram, at page 1363, drawn after Roscoe R. Graham gives a good idea of the nature of the condition (The rare recto-sigmoid intussusception in which the sigmoid colon gets into the rectum as an intussusception is very rare, almost never protrudes through the anus and will not be referred to again). The anterior rectal wall just above the levator ani first gets pushed into the rectum and in course of time the whole mass prolapses outside the anus. All the supports of the rectum—the lateral ligaments and the peritoneal folds between the rectum, sacrum, bladder and vagina become stretched and elongated. The sphincters get stretched and lose the power of contraction.

In the beginning, the prolapse occurs only during defaecation or severe straining, reducing itself when this ceases. In long standing cases—these individuals seem to put up with their condition for a number of years—it occurs at the slightest provocation and has to be reduced. Control of defaecation on account of the relaxed sphincter becomes difficult. In addition to constant recurrence, there may be irreducibility on account of oedema, or there may be ulceration and bleeding or gangrene. Rupture of the sac with escape of small intestines and death has been reported. In complete prolapse in children the exciting cause is often trivial (*e.g.*, mild diarrhoea of a week's duration) and the prolapse is said to be sudden *i.e.*, it is fully present one day, not being present on the previous day.

Examination and diagnosis of prolapse.—If the prolapse is not obvious the child should be watched during the act of defaecation to see its extent and if possible palpate it. Later on the child should be examined on the table not only digitally, but also proctoscopically for any associated conditions like polyp. Specimens of mucus or faeces obtained proctoscopically should be examined for amoebae and cysts or ova if previous examination of stools was negative. In adults, the prolapse should be inspected while the patient strains bending forward holding on to a table, or better squats down and strains till the prolapse reaches its maximum. If possible the prolapse should be reduced and again allowed to come out slowly, its progress being noted.

Generally a prolapse of more than 1½" in length is likely to be a complete prolapse. In a partial prolapse mucosal folds, if any, radiate from a centrally placed lumen, whereas in a complete prolapse the mucous membrane is thrown into circular folds around an opening which looks backwards because the anterior wall is the longest.

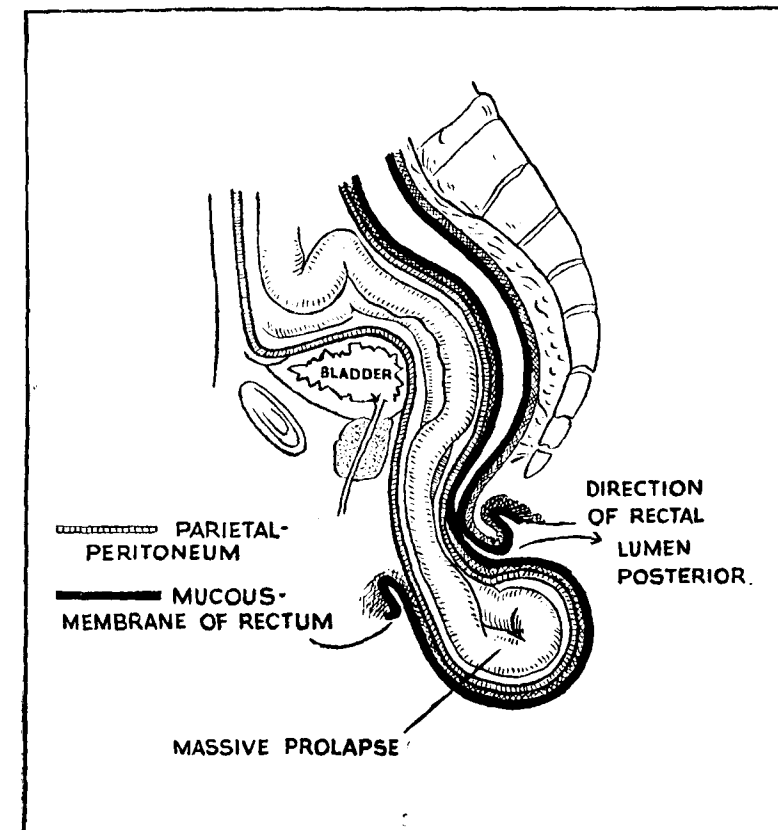


FIG. 1. Diagram showing the disposition of the peritoneum and the layers of the rectal wall in a case of complete prolapse of the rectum.

The extent and thickness of the prolapse should be gauged by palpation—any ulceration present should receive particular

attention to rule out malignancy, and gurgling of the intestines which may be present in the sac is felt for.

The patient is put on the table and the prolapse is reduced. In a complete prolapse pressure over the anterior anal margin or wall forwards with the examining fingers prevents the prolapse from coming down when the patient strains but similar pressure posteriorly does not prevent it. Moreover with the finger in the rectum and patient straining the anterior wall is felt to descend first. These findings and the fact that the anterior wall is the longest, confirm the view that the condition is a sliding hernia involving the anterior rectal wall.

The condition of the sphincter is noted—not only the resting tone but also its ability to contract—this latter is tested by asking the patient to try to 'hold back a liquid motion.' In women the condition of the uterus and its adnexa and supports has to be noted (after changing gloves), particular attention being paid to the perineum and the presence of a rectocele.

A routine proctoscopy and sigmoidoscopy are also done which will reveal not only the congested thickened rectal mucous membrane and the normal mucous membrane of the sigmoid but also any co-existing conditions *e.g.*, carcinoma in the bowel.

Treatment: Prolapse in children.—Among children an attempt is made to keep the prolapse reduced and many cases get cured once the bowels begin to act normally and irritating conditions like dysentery or worms are got rid of. As the child puts on weight, under a proper dietetic regimen and the encouragement of the bowel-habit, the prolapse becomes less and less frequent.

In other cases submucous injections of 5% carbolic acid in almond oil (the almond oil is autoclaved prior to addition of the required quantity of carbolic acid) are made with a view to producing adhesions between the mucous layer and the muscular coat of the rectum. The method is as follows:—

After a thorough bowel wash-out, the child is anaesthetised with ether and put into the lithotomy position. After cleansing the parts, if the prolapse is still out, the injection is given in the form of an interrupted circle all round the apex in the submucous layer through multiple punctures, 1 to 2 cc. being injected at each puncture. This procedure is repeated more proximally in a larger circle. 10 cc. to 15 cc. are usually required. After this the bowel is returned in. If the bowel is not prolapsed, then the injection has to be done with the help of a proctoscope which is passed as high as possible, the lax mucous membrane visualised and the injections are given in interrupted circles starting from above and working distally. After the injections, an 'internal splint' made up by winding $\frac{3}{4}$ " roller gauze (first fixed with a transfixion suture) over a $\frac{3}{8}$ "– $\frac{1}{2}$ " rubber tube to form a spindle and smeared with vaseline is introduced so that the

spindle lies above the level of the anal canal and kept in place by means of a safety pin and T. bandage (See Fig. 2 below). The 'internal splint' is kept in place for a week during which the child is put on liquid (non-residue) diet and a binding mixture containing bismuth carbonate and creta preparata (in older children and adults Tr. opii may be added).

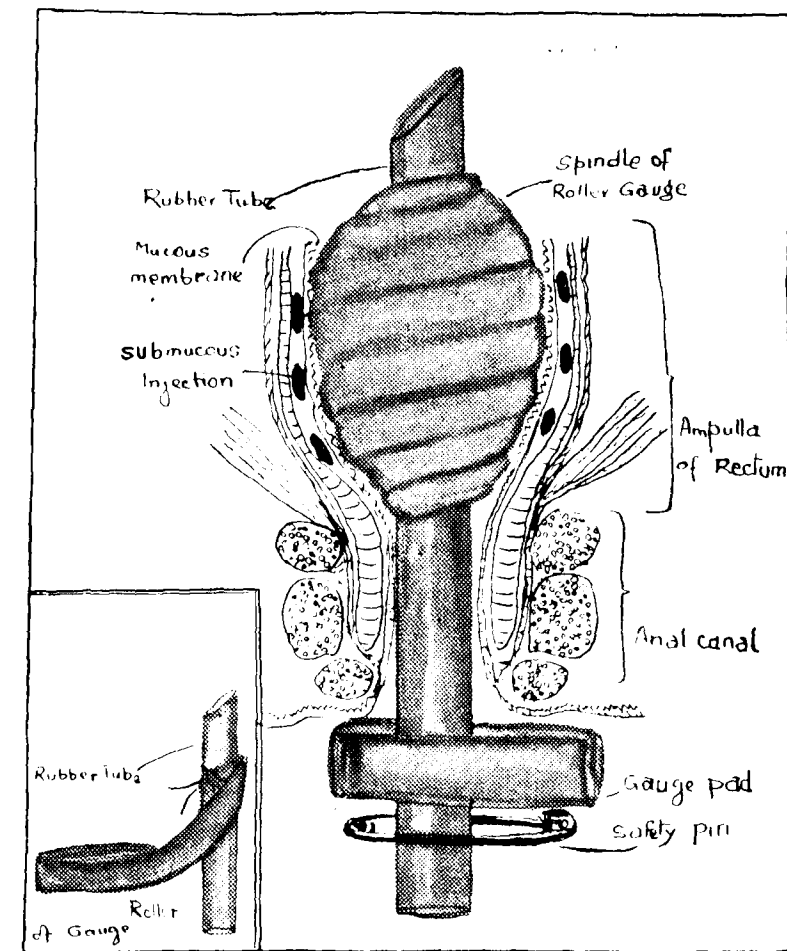


FIG. 2. Diagram showing the principle of 'internal splinting' in the treatment of partial prolapse of the rectum in children and adults. 'Internal splinting' is also used in cases of complete prolapse in children after perirectal alcohol injection. Insert shows roller gauze being wound round the rubber tube to form a spindle.

If it is thought that the prolapse is a complete one—it is sometimes difficult in children to determine whether the prolapse is partial or complete—the submucous oil injection is preceded by a perirectal injection of absolute alcohol or rectified spirit given as follows:—

Guided by the left index finger in the rectum, a fine 3" needle is inserted $\frac{1}{4}$ " outside the anus in the postero-lateral quadrant with all aseptic precautions and passed into the hollow of the sacrum to

about 2½". A syringe filled with 1.5 cc. of alcohol is fitted to the needle and the alcohol is injected slowly, part of it being injected as the needle is withdrawn but the injection should not be made too superficially.

At the end of a week, gingelly oil (sweet oil) or ground-nut oil 2 to 4 ounces is injected through the rubber-tube in the rectum followed an hour later by a soap and water enema. After this the child should be given liquid paraffin regularly for some days and a diet containing vegetables and fruit, such as banana to help to soften stools and relieve constipation.

It is surprising how quickly a lax sphincter in children regains its tone which progress can be further hastened by pricking the anal margin with a pin and making it contract several times a day.

Sometimes the injection needs repetition—the injection being made into any lax mucous membrane that may be seen but much smaller quantities of the carbolic oil are required and there is no need for the 'internal splinting' and binding of the bowels.

Treatment of partial prolapse in adults.—In adults the exciting cause of the prolapse, if there be any, should be removed. If the prolapse is one accompanying a third degree pile, operative treatment is the same as for piles. Otherwise submucous injection of carbolic in almond oil as described above gives good results. Should there be sphincter relaxation the patient is asked to do sphincter exercises two or three times daily—'holding back a liquid motion.' At each sitting the patient preferably lies down and ten to twelve good contractions of the sphincter are made with a pause after each relaxation. Intelligent co-operation of the patient is essential. In a college student who had a partial prolapse of 1½" for 12 years from child-hood with much sphincteric relaxation, the prolapse has not recurred for over 2 years and although the power of contraction of the sphincter has not fully recovered his resting tone is good.

Treatment of complete prolapse in adults.—This is operative, but the nature and scope of the operative procedure has not yet been standardised. Without going into technical details, it may be stated that out of many, the operation of rectosigmoidectomy in which the prolapsed rectum and as much of the sigmoid colon as can be drawn down after opening into the peritoneum, are amputated with end to end suture of the sigmoid colon to the anal canal, has been widely popular and fairly successful but there have been relapses and recently the unphysiological nature of the operation has been pointed out *viz.*, that it does away with an important receptor-organ like the rectum which initiates the reflex arcs on which sphincter tone and function depend. It seems therefore, more rational to treat this condition on the same lines as if it were a sliding hernia. This is achieved by Graham's operation in which, after

opening into the abdomen and with the patient in the Trendelenburg position the inner margins of the levator ani muscles are brought together with silk sutures in the middle line in front of the rectum. The lax peritoneum of the pelvic floor is then cut away and resutured so that the pouch of Douglas is done away with. The operation has to be followed by the usual sphincter exercises. On account of the technical difficulties of the operation in a recent case the edges of the levator ani were defined from below and brought together with strips of fascia lata woven into the muscles so as to form a good pelvic floor in front of the rectum. This fascial suturing of the levator ani was taken as far back as possible with an assistant's fingers in the rectum to control its extent. It is estimated that 23% of relapses after any operative procedure are during the first year but relapses have been reported even 10 years after operation. Fourteen months after the operation in this case of an agricultural labourer there is no sign of complete prolapse although the mucous membrane is peeping through a sphincter whose resting tone is still poor in spite of exercises. He may require circum-anal silver wiring (Thiersch's operation) or facia-lata insertion in which the silver wire or fascial strip is drawn tight till the anus just fails to admit the proximal interphalangeal joint of the index finger.

SURGERY FOR PATIENTS WITH DIABETES

With attention to anaesthesia, fluid and electrolyte balance, antibiotics and control of metabolism and nutrition, the patient with diabetes mellitus can safely be subjected to even major surgical procedures.

Careful preoperative preparation is essential to successful management of the diabetic patient. Close supervision is necessary to be sure that such details as insulin dosage are being fulfilled. If the surgery is elective, preparation with a high-protein and carbohydrate intake augmented with vitamins to replenish liver glycogen, tissue, and serum proteins may be valuable. Fluid and electrolyte losses should be corrected.

The cardiovascular status of the patient must be thoroughly evaluated before surgery. Treatment for heart failure, angina, or arrhythmias is given if appropriate.

For cardiac diabetic patients, parenteral fluids and feedings when needed are administered at a slow rate as concentrated solutions of dextrose or fructose. Slight hyperglycemia is advisable in known cardiac cases, a fasting blood sugar level between 150 and 180 mg. being maintained before and after operation. High levels of blood sugar should be reduced gradually. Sustained hyperglycemia above 200 mg. should not be permitted because of predisposition to infection, poor wound healing, body protein loss and ketosis.

Patients with occlusive vascular disease who have peripheral gangrene and infection should receive antibiotics preoperatively, and roentgenograms should be obtained to show any osteomyelitis and the extent of vessel calcification.

Insulin and fluid requirements must be tailored to meet the individual needs. However, some general rules may be followed. Depot insulin is administered subcutaneously on the day of operation at the time when parenteral feedings are

started to replace meals and is given in dosage equal to preoperative maintenance amounts. The operation should preferably be in the morning, the first infusion replacing breakfast carbohydrate. Individuals scheduled for afternoon surgery may be given a liquid breakfast and subsequent parenteral feedings.

The intravenous feedings replace only the carbohydrate portion of the diet, omitting the estimated available carbohydrate from fat and protein. The infusion period extends over twelve to sixteen hours and is terminated in the late evening, if possible. The volume and concentration of fluid depend on the patient's fluid requirements, tolerance based on age and cardiovascular status, and caloric requirements. Solutions given during and immediately after operation do not contain sodium chloride unless a cause for electrolyte loss is known. Vitamin B complex and ascorbic acid should be added.

Increased caloric intake and replacement of electrolytes are needed if parenteral feedings are required longer than forty-eight hours. Supplements of regular insulin may be needed post-operatively.

Diagnosis of an acute intra-abdominal condition in a diabetic may be difficult since acidosis occasionally causes abdominal pain localized or diffuse, with nausea, vomiting, constipation, leukocytosis, and tachycardia. With acidosis, polyuria and polydipsia are likely to precede the onset of abdominal symptoms. Persistence of pain after treatment with fluid, electrolytes, and insulin for several hours is indication for laparotomy.

The patient with acidosis who requires emergency surgery should be treated vigorously with regular insulin, fluids, electrolytes, and antibiotics. Operation should be delayed for several hours if possible to institute diabetic management. Subsequent stabilization of the diabetes is simplified by surgical correction of the complicating disease.

Diabetes does not call for a particular anaesthetic agent. Anaesthesia to give rapid and successful conduct of the operation is the chief aim. Special care should be given to prevent anoxia during general anaesthesia. During spinal anaesthesia, hypotension should be avoided by the use of pressor substances to prevent coronary or vascular occlusion. Standard preoperative medications are used except in some emergency operations on the diabetic with ketosis and dehydration.—(*J.A.M.A.*, 155: 621-26, 1954; *Mod. Med.*, 1-9-'54).

CORTISONE AND ACTH

In a review of three years' "experience of cortisone and ACTH at the London Hospital, during which over 100 patients were treated, Clifford Wilson sums up the present status of these two preparations as follows: "Cortisone and ACTH have a definite role as a substitution therapy in adrenal and pituitary insufficiency respectively. Cortisone is of undoubted value in certain infections of the eye and probably in some forms of dermatitis. Good results have been obtained in periarteritis nodosa, less satisfactory in disseminated lupus; continuous maintenance therapy may be necessary in these disorders. Red cell destruction may be controlled in acquired hæmolytic anæmia. Cortisone does not appear to influence the inflammatory process in progressive nephritis but may be of value in the treatment of resistant oedema. In severe acute rheumatic carditis which appears likely to prove fatal, the use of cortisone is justifiable, with proper precautions against fluid retention. In rheumatoid and related forms of arthritis, cortisone may undoubtedly benefit patients *during the early stages* by prevention of structural damage and by facilitating other remedial measures. Its continued administration in chronic active forms of the disease is problematical and will always need considerable supervision. In any case it can only be regarded as an addition to the various therapeutic measures which are already employed in chronic rheumatoid arthritis."—(*London Hos. Gaz.*, April 1954).

THE PROBLEM OF KALA-AZAR*

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KALA-AZAR is widely prevalent in India, especially in the province of Assam. For many years its aetiology has baffled the clinicians and research workers. The epidemiological features and transmission of the disease are problems which are still puzzling to the medical men. At one time it was thought that the disease was spread by mosquitoes and Sir Leonard Rogers and Sir Ronald Ross had even postulated that it was a severe form of malaria. Whilst physicians and research workers in the early part of this century, were searching for ways and means to combat it, kala-azar which had started in a mild form round Dum Dum and Burdwan in Bengal (hence the alternative names Dum Dum fever and Burdwan fever for kala-azar) began to spread rapidly like wild fire. The extent of the devastation caused will be realised from the words of Sir Upendranath Bramachari: "Before the days of the epidemic, the district of Burdwan was noted for its healthiness, and the town of Burdwan was regarded as a sanatorium. It was even customary for persons suffering from chronic malaria to go to Burdwan where cures from the disease were common. The census report for 1881 states that the district was considered one of the most salubrious in the province.....There is no portion of territory in Hindustan that can compare with it for productive agricultural value—in proportion to its size it appears like a garden surrounded by wilderness.".....The disease converted some parts of Burdwan and Hooghly into "a valley of the shadow of death" into which if any human being, whether robust or weak well-nourished or ill-nourished, entered, an attack of fever, was almost certain to overtake him; he should consider himself lucky if he escaped with his life intact. In his presidential address to the 25th Session of the Indian Science Congress held in Calcutta in 1938, Sir Upendranath referred to the epidemic in the following words. "In this epidemic it was noted that countries that smiled with peace, health and prosperity had been turned into hot-beds of disease, misery and death, and that the fell disease had mocked every human effort and absorbed in its powerful grasp, day by day and inch by inch every blessed spot which was once used to be prized for its salubrity. This was the Burdwan fever.....In more recent times the epidemic of the disease in Nowgong District in Assam produced such an appalling mortality that there was a decrease of 31.5% in the population of the place in the decade 1891-1900." This then, was the problem which confronted the clinician, the research worker, the entomologist and others during the early years of this century.

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Some of the salient features of this epidemic have been graphically described by Bramachari. Starting from Bengal it spread into the fertile Brahmaputra valley, in three invasions within the last 50 years. The waves of epidemic were characterised by the fact that every ten years of spread when the disease was carried further into the interior, were followed by a quiescent period of another ten years. During this quiescent period there would be a definite fall in the incidence of kala-azar, but the disease did not disappear entirely from the area. These waves of the epidemic have carried the disease into Assam as far as Sibsagar. An epidemic was due in the last few years, but adequate preventive measures and the discovery of an effective remedy for the disease arrested the further spread of the disease eastwards. It is surprising to note that while the spread was rapid into the Brahmaputra valley, its spread westwards up into the Gangetic valley did not take place with the same rapidity and to the same extent.

In Assam, many other peculiar features are noted in the distribution of the disease. Kala-azar was found to be confined more to the rural areas, than to towns. Even in the towns certain towns are more affected than others. For example, Jorhat, is less affected than Sibsagar, which is situated further east. It was also noted that while the incidence was falling in one village, it was on the increase in another village just a few miles away. Damp, old, low, one-storied houses surrounded by green vegetation and puddles offered ideal places for the occurrence of the disease. Destroying or even burning down a house and re-erecting it at the same site did not result in the eradication of the disease. Re-erection of the house a few hundred yards away from the original site resulted in the disappearance of the disease. This shows clearly that kala-azar is more a site-disease than a house disease. The presence of cows in the house, is a blessing as sand-flies prefer cow's blood to human. Other animals also like fowls, ducks, goats, etc. attract sand-flies, but are dangerous as the sandflies seldom feed on their blood, but merely take shelter on their bodies.

Kala-azar occurs along the East Coast of India right up to Madras, although not to the same extent as in Bengal, Assam and Bihar. In the city of Madras the disease has a peculiar distributional feature in that it very seldom occurs north of the River Cooum, while the incidence of the disease is fairly high, south of the river, especially in the crowded slum areas. Why the disease should have taken a foot-hold round Madras where the climate is not ideal for its occurrence is a problem which still remains to be tackled.

The question of transmission of kala-azar is a problem by itself. While one school of workers believe that kala-azar is transmitted by sand-flies, another school believes that the evidence for such transmission is not conclusive. Controversy has long raged over this question and quite recently a series of letters appeared in

the *Indian Medical Gazette* on this subject. Eminent specialists in Tropical Medicine took part in the controversy. At the beginning it was thought that bed-bugs, the flea and other blood-sucking insects were responsible for the spread of the disease. The finding of parasites in the excreta was one of the reasons which lent support to this view. Later this theory was abandoned and sandflies, the distribution of which corresponded closely to the distribution of kala-azar, were believed to be the insect vector. Moreover, it was found that after the ingestion of a blood-meal the Leishman bodies instead of going down the intestinal tract to be excreted, were going forwards towards the mouth and the proboscis of the insect. But, in spite of all efforts, the insect was found to be incapable of transmitting the disease. Recently research workers hit upon the solution and found that the taking of a second blood meal after the first one arrested the growth of Leptomonads and the insect was then incapable of transmitting the disease. Instead of this second blood-meal, if these sandflies were fed on fruit and plant juices, the multiplication of the Leptomonads went on and the insects were then capable of transmitting the disease. Success has been obtained experimentally in transmitting the disease to a few but not all the volunteers. The transmission of kala-azar by sandflies therefore depended on whether it had fed on blood after the first blood meal or on fruit juice. It is questioned by some workers whether it is possible for an insect whose natural instinct is for blood, to change its habits suddenly and become a "vegetarian." The transmission of kala-azar thus remains, one of the problems which has not been solved conclusively and satisfactorily.

The relationship between kala-azar and oriental sore, the other manifestation of Leishmaniasis, is yet another interesting problem. Kala-azar and oriental sore are very rarely present in the same area in India. Oriental sore is present in the drier regions of Bihar, the Punjab and Delhi, while kala-azar is present in the wetter regions of the country like Bengal and Assam. As we proceed eastwards from Calcutta we find kala-azar on the increase, while proceeding westwards oriental sore takes the place of kala-azar. This peculiarity in distribution—kala-azar in moist places and oriental sore in the dry areas—has not been observed to the same extent in other parts of the world, for example, Asia Minor, Crete, Greece and other Mediterranean countries where kala-azar and oriental sore are known to exist side by side.

Another difference between kala-azar in India and in the Mediterranean countries is that the disease is very rare among the older people in the latter region where children under five and infants are mostly affected. In India the disease is not known among infants and very young children. Older children and adults are found to be more often affected. The view is held that the dog is a probable carrier of the disease in the Mediterranean countries,

because whilst the disease is not known among the dogs in India, kala-azar affects dog in the Mediterranean countries.

This disease which offers so many problems to the scientist, was eventually controlled by the discovery of Ureastibamine which was found to be specific against the infection. The following memorable words of Sir Upendra Bramachari will be read with respect and admiration. "I recall with joy that memorable night in Calcutta Campbell Hospital at Sealdah after a very hard day's work, when I found at about 10 o'clock in a little room with a smoky dimly burning lantern that the results of my experiments were up to my expectations. But I did not know then that Providence had put into my hand a wondrous thing and that the little thing would save the lives of millions of my fellow-men.....Gentlemen, antimony is a wonderful element. It was supposed to do so much harm that graduates in medicine in the University of Heidelberg had to swear never to use antimony. And today it had been found to be a specific in one of the most terrible of tropical diseases." But with the introduction of antimonial drugs in the treatment of kala-azar, we are faced with another problem *viz.*, the development of a resistant type of kala-azar and of post-kala-azar dermal-leishmaniasis. This was first recorded in 1922 after the introduction of Urea-stibamine, when it was noticed that cases of kala-azar which were imperfectly treated with urea-stibamine developed certain peculiar skin-eruptions. The enemy in the process of destruction tries to retreat from the internal organs to some other part of the body. L.D. bodies seek shelter under the skin and escape destruction in the imperfectly treated cases of kala-azar. Later when the treatment is discontinued they begin to multiply at the site and cause various skin manifestations. Post-kala-azar dermal-leishmaniasis is an extremely difficult condition to treat.

Kala-azar presents therefore, several interesting problems. The epidemiological features, and spread of the disease, its transmission, the difference between kala-azar and oriental sore and between the disease as it occurs in India and as found in the Mediterranean countries, and the development of chronic, resistant types of the disease along with post-kala-azar dermal leishmaniasis are all problems which have still to be studied and understood. Much has been done in India to solve the problem and we owe a great deal to the Calcutta School of Tropical Medicine and to Sir Upendranath Bramachari and others for their valuable contributions in enabling us to fight the disease. But a wide field is still left unexplored; many problems still face the medical profession and it is hoped that the problem of kala-azar will receive the enthusiastic and concentrated attention of research workers with a view to its complete eradication from our midst in the not distant future.

HYPERIDROSIS IN PULMONARY TUBERCULOSIS*

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HYPERIDROSIS is a condition where there is excessive perspiration due to the over-activity of the sweat-glands. It may be localised or general. The localised condition occurs in certain emotional states or in an imbalance of the nervous system, whereas general hyperidrosis is commonly associated with tuberculosis, malaria, acute rheumatic fever, septic fevers and with low vascular tension.

Hyperidrosis in tuberculosis is a toxæmic symptom, due to a stimulation of the sweat glands by tuberculin that is produced in the body by disintegration of the tubercle bacilli. The peculiarity of hyperidrosis in pulmonary tuberculosis is that the sweating starts sometime after the patient has gone to sleep. The time varies with individual patients; usually the patient starts sweating about 1½ to 2 hours after he has fallen asleep. Hence it is called "night-sweats"; but the sweats occur even in the day time if the patient goes to sleep. So, in fact they are slumber-sweats. In far advanced cases, these sweats occur both when the patient is asleep and awake.

Hyperidrosis is met with in varying degrees of severity from a slight excess of normal sweat-gland activity to a condition in which drenching perspiration soaks through his clothing and the patient has to change his clothes. Sweating may occur at any stage of the disease. Usually it is not a feature in early tuberculosis. It is often seen after cavities have formed but it is almost a persistent and distressing feature in advanced febrile cases and in exhausted states. No doubt, the sweating increases with a sticky atmosphere, over-clothing, bad ventilation etc. It appears first on the forehead and then in other parts of the body.

Frequency of sweating causes unhealthy skin and various forms of dermatitis by constant soaking. Saprophytic organisms grow freely, decomposition takes place and an offensive odour develops. A peculiar odour is sometimes associated with the sweat of tuberculous patients, akin to the smell of a ripe guava fruit—sweet but repulsive. Such odour can be recognised even at a distance of 5 or 10 feet from the sick bed. While examining a patient if you get such a smell, you may be almost certain that it is a case of fairly advanced pulmonary tuberculosis. A faecal odour without any evidence of diarrhoea or soiling of bed clothes indicates impending death in tuberculosis.

*Specially contributed to the ANTISEPTIC.

Sweating is not of much importance in prognosis. The extent of sweating gives no indication of the extent of the disease. But persistent night or slumber sweats are unfavourable. Abrupt cessation of sweating, in a person who is accustomed to sweat is however, a bad prognostic sign and he does not usually live for more than a week.

TREATMENT:—1. *General*:—Rest both physical and mental is important. Food must be rich in vitamins. Toxicity must be controlled with chemotherapy or other measures. Sweating usually disappears if the toxicity is controlled.

2. *Personal hygiene*:—The clothing should be light; the skin must be kept clean by frequent sponging particularly at bed time and dusting with a powder containing 3% salicylic acid. A change of clothing should follow sweatings.

3. *Hygiene of the surroundings*:—The sick-room must be kept clean and well-ventilated, with doors and windows always open but avoiding exposure to draught.

4. *Drugs*:—If the sweating still persists, drugs are usually tried: Belladonna with bromides, zinc oxide (gr. 3) with Ext. Belladonna sicca gr. 1, Atropine, Picrotoxin, Agaricin, Strychnine and Calcium.

5. *Special methods*:—Try to wake the patient up a few minutes before the sweating is due, give him a cup of milk, or a few biscuits and ask him to go to bed again. This often helps to stop the sweating.

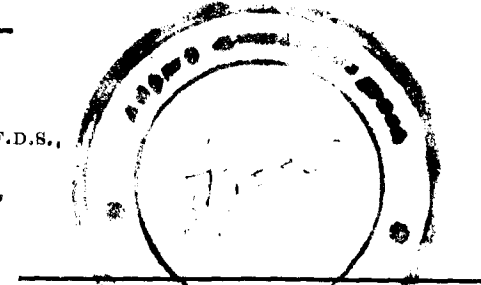
Soft porous rush mats called Paterson's slumber-mats placed between the bed and the bed-sheet are known to lessen sweating and give comfort to patients.

TREATMENT OF HYPERIDROSIS

Satisfactory results are reported by E. L. Reder from the use of methyl phenobarbitone ('phemitone'; 'prominal') in the treatment of 'those cases of excessive sweating where emotional factors play an important role'. This conclusion is based upon the results obtained in a series of 99 patients with hyperidrosis 'selected for their psychoneuroses of the anxiety and tension states. In every case one of the outstanding symptoms was excessive sweating.' The dose was 32 mg. by mouth thrice daily after meals. "Excellent control" of the hyperidrosis was obtained in 91 cases, "doubtful results" in one, no change in six, and increased sweating in one. The rationale suggested for this action of methylphenobarbitone is that "it produces more tranquility and less necrosis in anxiety states than other sedatives" and that it is able to "reduce adequately the activity of the diencephalon when employed in an amount below the hypnotic or anæsthetic level."—(*New York State Jour. of Med.*, May 15, 1954 in *Practitioner*, August '54).

LUPUS ERYTHEMATOSUS*

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THIS is an inflammatory affection of the skin characterised by redness, scaling and atrophic-scarring, with two syndromes:—
A. The chronic discoid lupus erythematosus. B. Acute or sub-acute disseminated lupus erythematosus.

A. Chronic discoid lupus erythematosus.—This disease is most common in women and occurs between the ages of 20 and 45.

ÆTIOLOGY:—The ætiology of this condition is not definitely known but the attack may be precipitated by exposure to U.V. rays and to the sun. Focal sepsis is supposed to be a predisposing factor. Some of the patients show evidence of tubercular infection of the lungs or glands and give a positive tuberculin test. It has however, been proved that tuberculosis is no longer considered the ætiological factor.

SIGNS AND SYMPTOMS:—The disease starts insidiously with small red scaly patches, usually symmetrical over the upper cheeks and nose and extend slowly tending to be red and scaly at the margins, healing at the centre and leaving a thin atrophic scar. The lesions may also occur over the vermillion border of the lips, over the pinna and on the scalp.

Examination with a hand lens will usually show a characteristic stippling produced by the filling of the sweat and sebaceous glands with little horny plugs; whereas the lesions on the scalp are very atrophic and depressed with total destruction of the hair follicles leading to alopecia. The lesion on the face takes a butterfly pattern and follicular plugging is very characteristic. On the earlobes the lesions are always destructive. On the hands, lupus erythematosus affects commonly the dorsal surfaces, and takes the form of oval patches or rings on the back of the hand.

DIFFERENTIAL DIAGNOSIS:—(1) *Lupus vulgaris*:—Occurs commonly on the face and neck, the characteristic feature being the lupus nodule, known as the apple-jelly nodule. On pressure with a watch-glass the lesion becomes white and the nodules appear as small semi-translucent spots. The stippled appearance with a central atrophic scar is absent in lupus vulgaris. (2) *Seborrhoea*:—A seborrhœic lesion on the face causes scaly patches which may be slightly red or may be yellowish and greasy-looking. (3) *Psoriasis*:—The lesions are covered by scales which become silvery on scraping.

*Specially contributed to the ANTISEPTIC.

There are similar lesions all over the extensor surfaces. (4) *Rosacea*:—This disease occurring usually above 30 years, more commonly in women is caused by an instability of the vasomotor system causing a paralytic distention of the superficial blood vessels of the skin of the face and a secondary hypertrophy of the sebaceous glands and sometimes superficial pustular lesions called rosacea. The condition is usually localised in the middle third of the face—a region called “the flush area.” The manifestations vary considerably. There may be simple erythema but usually there are other lesions as papules, pustules and telangectasia. (5) *Pityriasis simplex* or scaly impetigo is a condition with dry scaly patches with scales arranged more or less in parallel rows (like a ploughed field) often seen on children's faces. The condition is a chronic form of impetigo which is difficult to treat. (6) *Eczema*:—In infantile eczema the centre area of the face is spared. The affected skin is rough and red with minute patches, vesicular or weeping and crusted or dry. The eczematous patches occurring in adults may be erythematous, vesicular, weeping, crusted, dry and scaly and lichenified.

The lupus erythematosus lesion occurring on the hands may have to be differentiated from the following:—(a) *Chilblains* or erythema pernio. The lesions are dusky, red, round or oval intensely itchy and occur in cold damp weather. The size is variable. Occasionally they ulcerate. (b) *Erythema multiforme*:—This is a condition with irregular erythematous blotches occurring on the back of the hands and forearms. The lesions may be circinate as in the idiopathic type of Hebra or erythematous and oedematous as in the multiform erythema group or erythema and vesicular bullous lesions with constitutional symptoms as in Steven-Johnson's Type. (c) *Papillo-necrotic*:—Tuberculoids are small papular lesions which tend to appear in symmetrical crops on the extensors of arms, hands, shoulders, elbows, and buttocks, legs and feet. They occur in young adults. (d) *Psoriasis*:—(discussed under item 3 *supra*).

When lupus erythematosus lesions occur on the scalp producing alopecia the condition has to be distinguished from alopecia areata where the patches of alopecia are circumscribed. The affected area is completely denuded in alopecia areata with the possible exception of some ‘exclamation-point-hairs’ of the periphery; whereas in alopecia resulting as a sequel to lupus erythematosus there is destruction of tissue with resultant atrophy, permanent scarring and cicatrization.

B. Disseminated lupus erythematosus.—This form of lupus erythematosus occurs usually in young women about 20 years of age and the manifestations in the skin are only part of the involvement of the collagenous tissue in many other parts of the body.

SYMPTOMS:—The skin eruptions may resemble the chronic discoid form with erythematous lesions on the face and back of the

fingers and other parts of the body. Lesions usually take the form of a superficial bright red eruption. Occasionally the lesions appear like urticaria. Hemorrhagic or purpuric lesions may appear late in the disease. The general symptoms are:—(i) fever which may be slight at first but later may spike to high limits; (ii) lassitude and loss of weight; and (iii) anemia. In the year 1924, Libman and Sacks described a syndrome in which a multiplicity of skin and visceral lesions appeared in the form of lupus erythematosus, nephritis, endocarditis, enlargement of lymph nodes and of the spleen and pneumonia. In typical cases the initial skin-lesion is a butterfly erythematous patch across the bridge of the nose which develops rapidly. The course of the disease may be short and stormy with a fatal termination in six weeks, or it may be chronic with acute exacerbations. The aetiology is unknown but is considered to be a septic infection in a previously sensitised individual. The basic lesion is a fibrinoid degeneration of the collagen to which may be added a minor degree of reactive inflammation.

The principal lesions are:—(1) cardiac; (2) renal; (3) vascular; and (4) cutaneous.

In the heart the striking lesion may be flat warty vegetations. The valvular lesions are larger and flatter than those of the rheumatic and some of the subacute bacterial ones. The tricuspid valve is most commonly affected and occasionally the pulmonary. Microscopically the basic lesion is fibrinoid degeneration of the connective tissue of the endocardium, myocardium and pericardium. A characteristic lesion frequently present in the spleen, is periarterial fibrosis involving the arterioles and small arteries. This takes the form of concentric rings of collagen which may become hyalinized. Lesions in the kidney occur in the glomeruli. There is a thickening of the basement membrane of the capillaries giving a resemblance to a bent wire, “*wire-loop capillaries*.” In addition there is focal necrosis of the glomerular tuft. The vascular lesions are most commonly in the kidneys but may occur in any organ of the body.

The skin lesion:—There is fibrinoid degeneration in the upper layer of the corium involving the collagenous fibres and ground substance. Tilum suggests that the periarterial hyalinization in the kidneys and the hyperglobulinæmia are essential features.

The L. E. phenomenon:—In January 1948 Hargraves, Richmond and Morton first reported observations on certain bone-marrow elements of patients with disseminated lupus erythematosus to which they gave the name L. E. cells and tart cells. In February 1949 Lick and Sundberg found L. E. cells in the peripheral blood.

The following is a summary of the work done:—The tart cells and the L. E. cells were first described by Hargraves. The tart cell is a histocyte or monocytoïd reticuloendothelial cell which contains a second characteristic nucleus placed on the top of the

primary nucleus. This second nucleus stains a reddish purple with Wright's stain and has a distinct chromatin pattern. The critical L. E. cell is a mature neutrophilic leucocyte which contains within its cell membrane one or more masses of nuclear material. These masses called L. E. bodies may be round or oval and vary in size and take a bluish stain. The L. E. phenomenon includes also the formation of clumps and rosettes. The rosette consists of a central mass of bluish staining cell debris surrounded by phagocytes forming a ring around the central mass. The phagocytes appear to be engulfing the material in the centre.

Barnes *et al* mixed the plasma from heparinized peripheral blood from acute lupus erythematosus patients with heparinized normal sternal marrow and obtained L. E. cells consistently, confirming the work of Haserick and Bortz. This procedure carried out with plasma from two patients is considered to be a good method. They also succeeded in producing L. E. phenomenon by adding to the plasma of a patient with acute lupus erythematosus, centrifuged heparinized blood from a normal person. It was suggested that the anticoagulants are responsible for the production of the L. E. phenomenon *in vitro*, but the contrary has been proved to be true. Recently Barnes has noted oval intracellular bodies of a unique type, for the most part in the cytoplasm.

Treatment for lupus erythematosus.—The method of treatment used in lupus erythematosus varies with the type:

A. Discoid lupus erythematosus.—(a) *Preventive*:—Patients with lesions of this type should live a temperate life, avoid sunlight and light-sensitizing drugs *e.g.*, the sulpha group.

(b) *Systemic*:—(i) Bismuth salicylate in oil 0.2 g. once weekly intramuscularly; twelve injections constitute one course to be repeated after six weeks. (ii) Gold and Sodium-thio-sulphate is given intravenously or intramuscularly (depending on the type of preparation). Doses: 10 mg. increased to 15 mg., 20 mg. and 25 mg. finally which is continued. The injections are given once a week for 20 injections. The course may be repeated after six weeks. The precautions in both the above lines of treatment are to prevent renal damage and bleeding tendency. The urine should be examined for albumin before administration and concurrently Vitamin C may be given.

(c) *Local*:—A sun-screen type of ointment as sodium parabenzoic acid may be used for this purpose. Calamine liniment is also helpful. Cortisone ointment 1.5% has helped in a small percentage of cases.

B. Disseminated erythematosus.—Three types may occur, the chronic disseminated, the acute disseminated and the sub-acute disseminated types.

(a) *Preventive*:—In the chronic disseminated type, wide dissemination with fatal result is unusual. The chronic discoid form

may get disseminated. The most important sign is an increase in the blood sedimentation rate. The measures of rest, general supportive treatment, prevention of focal sepsis and protection from sunlight are *sine qua non*. For acute and sub-acute disseminated types, the incidence may be decreased by observing the following rules: (i) avoid overfatigue; (ii) maintain good health; (iii) correct any focal sepsis; (iv) avoid sensitizing drugs, like the sulpha group; and (v) avoid exposure to the sun.

(b) *Active*:—(i) Rest in bed; (ii) correct the anaemia by transfusion and antianemics; (iii) correct inter-current infection like pneumonia with penicillin; (iv) use cortisone and A.C.T.H. which are the only drugs for emergency use in gravely ill cases of disseminated lupus erythematosus; cortisone should be used on this plan:—First day 100 mg. I.M. every eight hours for three doses; second to sixth day 100 mg. I.M. twice daily; seventh to 14th day 50 mg. I.M. twice daily. Subsequently daily 50 mg. I.M. once for a week and then gradually stop.

Inglemann advises ACTH to be given intravenously in a 12 to 16 hours' drip with 5% glucose in water, the initial dose not to exceed 25 mg. daily. After the first two or three days the intravenous method of administration may be replaced by long acting preparations, administered intramuscularly. A dose of approximately 40 mg. is given once daily. If it is desirable to continue the intravenous form of administration daily, doses of 5 to 10 mg. will probably suffice. Significant eosinophilia is a helpful guide to the minimal effective dose of ACTH.

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TREATMENT OF SYSTEMIC LUPUS ERYTHEMATOSUS

No other known remedies except cortisone and ACTH have any effect at all on the course of the disease. The immediate effect of the treatment is usually dramatic, even in patients who appear to be moribund: arthralgia, fever, pericardial and pleural effusions rapidly disappear. The characteristic laboratory findings, however, are not much altered: a high or relatively high E.S.R. usually persists, hyperglobulinæmia may be reduced during treatment but it may return during continued treatment. Lupus erythematosus cells can generally be found at one time or another. In cases with leucopenia, an increase in the white cell count occurs sometimes. Prunty *et al*, (1952) note that the renal lesion in systemic lupus erythematosus may be present at a relatively early stage of the disease. At this stage administration of ACTH or cortisone is likely to improve impairment of renal function; but as the disease progresses, in spite of courses of therapy, the renal changes become irreversible and the continued administration, of these hormones is likely to lead to exacerbation of the renal phenomena. Soffer and Bader (1952) also find that the renal lesion persists during both drug-induced and spontaneous remissions and is of grave prognostic significance.—(*Practitioner*, Aug. 1954).

TREATMENT OF CUTANEOUS LUPUS ERYTHEMATOSUS

Most of the remedies which have been in common use for years, e.g. quinine, gold compounds, acetarsol, oxophonarsine, have sometimes produced a well-marked temporary change for the better, but at least as often they have failed completely. Gold is probably the most effective of these remedies, but it is unsafe.

MEPACRINE:—Page (1951), while treating a patient with discoid lupus erythematosus for another disease with mepacrine, noted that the skin cleared up. Subsequently, in collaboration with F. R. Bottley, he treated 17 others; nine results were classed as excellent and six as good, two were slightly improved and one unchanged. These findings are supported by Nielsen (1954): in a series of 12 cases, disappearance of almost all symptoms occurred in two, considerable improvement in nine, no change for the better in one. Kierland points out that this method of treatment was actually begun in Russia by Prokoptchouk in 1941, followed by Sorinson, Popoff and Kutintseff, and Ottolenghi and Lodigiani in Italy. The results reported by these observers, and quoted in Kierland's article, are much the same as those in the later British, American and Scandinavian reports. It is thus clear that mepacrine, though very far from a perfect remedy, is of some value. The usual dosage is 100 mg. a day, and the treatment is pursued for from two to three months. In those cases which respond to treatment favourable effects are invariably apparent within about six weeks. As a general rule the labile erythematosus cases respond more rapidly and more completely than the fixed discoid scarring cases, in which, none the less, the effect of treatment is often surprisingly good. There are two objections to treatment with mepacrine: yellow discoloration of the skin, and occasional toxic reactions including a drug eruption of some severity—the well-known tropical lichenoid eruption of the war. Fortunately, this complication occurs comparatively rarely but it is probably not wise to administer mepacrine for more than three months at a time. During the last few years nearly every dermatologist in England has been using mepacrine in preference to any other remedy. It is not known why or how it acts in this disease, and experience has shown clearly enough that relapse is often to be expected in due course. On the other hand, it is also evident that mepacrine, if effective in the first instance, is likely to be helpful in the treatment of a relapse.

CHLOROQUINE:—Prakken and Molhuysen-van der Wallo (1954) used this drug on the grounds that it has much the same effect in malaria as mepacrine. They were satisfied that mepacrine was a real advance in the treatment of lupus erythematosus of the skin, but noted that it had its drawbacks, particularly the yellow colour that it provoked and its toxic sequelae. They tabulate the results of treatment of 25 cases with chloroquine as excellent in eight, good in nine, moderate in six, no result in two. Blurred vision due to disturbance of accommodation occurred as a complication in some patients. Harvey and Cochrane (1954) report that they have found chloroquine as effective as mepacrine and in a series of 30 patients report the results as excellent in nine, good in nine, slight in four, no change in eight. One patient had a hæmatomesis ten days after starting treatment, two others had difficulties in focusing near vision. Goldman *et al.* (1953) treated 21 cases, claiming great improvement in 14, improvement in two, and failure in five.

The dosage employed by Prakken and his colleague was 200 mg. daily, by Harvey and Cochrane 150 mg. daily for the first two weeks followed by 300 mg. for four weeks, then again 150 mg. daily for the next four weeks. Results so far seem similar to those from mepacrine, perhaps with a somewhat higher proportion of complete failure.

LOCAL TREATMENT:—Very fixed chronic discoid scarring cases, if not extensive, can be helped by repeated freezing with solid carbon dioxide for five to ten seconds. No other local remedy has much value. Opaque creams, such as 'cover mark', and other 'cover creams' are useful both cosmetically and in providing protection against the harmful effects of sunlight.—(*Practitioner*, Aug. 1954).

X-RAY EXAMINATION OF THE GASTRO-INTESTINAL TRACT*

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THE diagnostic value of radiography in the examination of the digestive tract is now universally recognised and increasingly used by the general practitioner as well as the surgical and medical specialists.

The concept of the conduct of this investigation varies greatly, being sometimes obscured by unnecessary detail, and at other times conducted in a perfunctory manner, without reference to the natural progress of events.

When asked to contribute a paper to the Jubilee number of this well known journal, I thought that I might give for the benefit of X-ray workers in general, a description of the method employed in my practice here, which is the outcome of many years' experience as Honorary Radiologist to the Presidency General Hospital and the School of Tropical Medicine, Calcutta for over 20 years in each institution, using all types of apparatus, from coil outfits with mechanical and electrolytic breaks to the modern multipurpose installations. This, I hope, would stimulate uniformity in the ranks of X-ray workers in this country, always bearing in mind that many of them have to work with indifferent apparatus. The first question then that arises is the apparatus, which in a vast country like India will necessarily vary widely in nature and design. The minimum general requirements to ensure efficiency, however, are detailed below:

Current.—200 to 400 milliamps. We use 200 as a routine, as being fast enough for all ordinary work, and not causing undue strain on the tubes and other delicate parts of the installation, e.g. for ordinary postero-anterior skiagrams in patients of average thickness 70 K.V.P., 200 milliamps, 0.12 seconds at a distance of 30 inches, with a fine Potter-Bucky grid. These figures, of course, must be altered according to the thickness of the patient. For serial pictures of the stomach and duodenum we use the "Explorator," an universal serial changer* which provides for the following:—(a) general screening with a fluorescent 14"x14"; (b) simple serial aimed radiographs up to 14"x14"; and (c) aimed serial films: Cassette frames are available to hold either two 13"x8" or one 15"x6" cassette, thus enabling up to 4 exposures either 9x12 cms. or 10x14 cms., to be taken in rapid succession, or at intervals as required, and (d) two aimed oesophageal radiographs size 12"x5",

*Manufactured by General Radiologist Ltd., London.

can be made on a 12"x10" film with the aid of a special frame. Further, varying pressure can be applied to selected areas to bring out special features such as ulcer-craters etc.

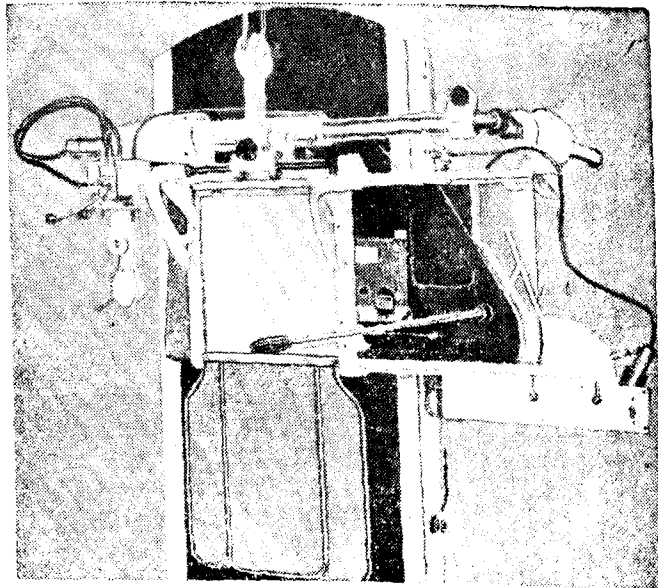
Full protection is provided by a detachable three-part lead rubber apron. This can be fitted to most types of apparatus with vertical or tilting couches. We use a tilting table made by Sieman's Ltd., which provides for radiography in the vertical, horizontal, intermediate and Trendelenburg positions.

The procedure adopted is as follows:—

General fluoroscopy.—The chest is always screened in the erect posture and note made of the size and form of the shadow of the heart and great vessels. Special attention should be paid to the possibility of early tuberculosis. On many occasions, I have had cases referred to me for examination of the stomach and duodenum when the patient was suffering from the gastro-intestinal symptoms of early tuberculous disease of the lungs. The movements of the diaphragm are carefully observed and a note made of the size and movements of the liver, which can be confirmed later when the duodenal cap is filled. A note is made of any abnormal collections of gas in any part of the abdomen including the hollow abdominal organs. A clue may thus be found to obstructions including intussusception, imperforate anus in children (by holding the patient's head downwards and noting the position of the highest point of a collection of gas in the rectum and its distance from the skin in the anal region).

Any abdominal condition noted on the screen should at the same time be recorded by making the necessary exposures or films.

In this connection one is sometimes asked to determine the site of acute intestinal obstruction including intussusception. The signs of both these conditions may be found in any advanced text book.



A universal apparatus for X-ray diagnosis by fluoroscopy and radiography (aimed and serial films)

In the average case, however, nothing abnormal may be noted up to this point and so one proceeds to give a *barium meal*. As regards the meal, the opaque medium usually employed nowadays is pure *barium sulphate*, free from any trace of the soluble salts of barium. Fatalities have, in the past occurred through accidental swallowing of barium sulphide, which of course had a strong smell. The writer once saved a patient's life, on accidentally entering the X-ray room, when an orderly was about to hand a glass of meal made with a fatal dose of this salt. The drug, supposed to be barium sulphate, had just been received from the chemist. The smell drew my attention to its nature before the patient swallowed it. [In our clinic here, for general purposes we use Horlick's shadow food which is pleasant to take, and meets all general requirements. For small intestine work, however, it is sometimes advisable to use preparations* specially designed for this examination]. The patient is placed standing behind the vertical screen and the chest is screened for any abnormalities of the heart or lungs and the movements of the diaphragm are observed in inspiration and expiration. For the size of the liver it is best to wait till the stomach gets filled, when the duodenal cap marks the lower border.

Oesophagus.—The patient is turned into the R.A.O. position and is given a small amount—about 2 ozs. of barium emulsion to swallow when told to do so. The progress of the barium through the oesophagus is watched carefully till the bolus enters the stomach. During this period, dilatation and stricture, delay or irregularity in outline should be looked for. The entry through the cardia should be carefully watched for signs of delay or obstruction, hiatus hernia, reflux oesophagitis, congenital short oesophagus should be noted and skigraphed if necessary, with the spot-film device.

Having completed the examination of the oesophagus, the patient is turned into the P.A. position and when the barium has reached the fundus of the stomach a skiagram is taken of the gastric mucosa, if necessary under compression. It sometimes happens that the meal sticks in the fundus. Massage and a few deep breaths will usually induce the viscus to open up. The rest of the meal is now given and filling watched on the screen. The points to be looked for are:—1. The general outline of the filled viscus. Is there any dilatation? and the level of the fundus—visceroptosis. 2. Does the outline show any irregularities, or coarse crenations?—chronic gastritis. 3. Are there any protrusions—diverticula or ulcers? If so does palpation elicit tenderness?

On conclusion of this survey three skiagrams are taken—postero-anterior, right anterior oblique and left anterior oblique. If any ulcer-crater or any filling defect is seen, it may be necessary to take serial skiagram with varying compression. In the case of gastric ulcer, diverticula or suspected malignant disease, skiagram

* Alubar is one such made by Wander.

on the horizontal couch, usually in the prone position may help. Having completed this survey of the stomach one turns to the duodenum. In the case of chronic ulcer, gross irregularity of the duodenal cap will have already been noticed. The pressure cone of the explorator is not introduced.

A careful screen examination is made with varying pressure over the duodenal cap and any suspicious areas in the stomach suggesting ulcer, neoplasm or diverticulum noted. Spot films may be taken of any abnormality for permanent record. Then eight serial films of the duodenal cap are taken, 4 on each 15"x6" strip of film, with varying pressure. It is our custom to take 4 P.A. views—two without using pressure and two under varying pressures on one 15"x6" strip and then two in the R.A.O. position, and two in the L.A.O. position. Finally the pressure cone is removed to one side and the stomach and duodenum are carefully palpated for mobility and tenderness. This is also a good time to observe the size and movements of the liver, as the filled duodenal cap marks the lower border, so that the whole liver can be brought under purview, shape, size, and movements. The second, third and fourth parts of the duodenum should also be examined for signs of ulcer or diverticulum and the passage of the opaque food noted. Tenderness and mobility of these parts should also be examined. All these procedures take about 30 minutes and this is a good time to get a picture of the jejunum. The patient is placed prone on the horizontal couch and a picture on a 15"x12" or 17"x14" film is taken. In the average case this should give a good view of the whole of the jejunum and proximal part of the ileum. If anything unusual, *e.g.*, a suggestion of sprue or other disease of the small intestine is observed. Further prone picture at 1 and 2 hours should be taken and the patient directed to return after 4 hours when an exposure to determine the emptying rate of the stomach and small intestine should be made. This concludes the morning examination and in the average case, if the stomach is empty, the patient should be directed to have a light lunch and return at 4 p.m., 7½ hours after ingestion of the meal. By this time the head of the meal should be at about the hepatic flexure of the colon, though there should still be a residue in the terminal part of the ileum, the position having been noted. The visible part of the ileum and proximal colon is carefully palpated and a note made of (a) deformity, mobility and tenderness and (b) a careful search for the appendix should be made if there is no history of its previous removal, and the trio of signs *viz.*, deformity, impaired mobility and tenderness, noted in the first instance. Also whether it crosses or is adherent to the terminal coil of the ileum. The visible part of the colon is then palpated and a note made of deformity, mobility or tenderness. At this stage a single P.A. skiagram is usually sufficient. Further skiagrams at 8 to 9 hours may be necessary—at least one at about this time should be taken to show progress of the meal in the large intestine.

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The patient is then told to have a light dinner, morning tea and toast, and return at 24 hours after ingestion of the meal. The greater part of the colon should now be visualised and a careful screen examination and palpation should be made. The following points should be noted: Progress of the meal—position of the head of the meal. In this connection the patient should be questioned about movements of the bowels, colour of the faeces—a whitish coloration indicating the passage of some of the barium. One or two films may be required to record the precise state of affairs. These should show the outlines of the bowel; any alteration in size—spasticity indicating chronic colitis, filling defects suggesting neoplasm, diverticulosis and so on. It may be necessary to make a 48 hour examination to determine the permanency or otherwise of abnormalities especially diverticulitis, but the 24 hour examination usually ends the series.

Barium enema.—The barium enema is primarily a method for examination of the large intestine by introducing a barium emulsion rectally and watching its progress as it fills the large intestine as far as the caecum, and sometimes the appendix, and passes into the terminal coil of the ileum. Films are taken at intervals during introduction, to show abnormalities observed under fluoroscopy. After the patient has evacuated as much of the barium as possible, further films are taken as indicated by fluoroscopy, and finally air sometimes may be injected to produce a double-contrast effect.

We proceed as follows:—Castor oil is given the night before the examination and on the morning prior to the examination the patient receives a thorough colonic irrigation to free the colon of faecal material. The barium emulsion is then introduced per rectum. For this purpose an irrigator can be marked in half pints and connected to 3 or 4 feet of rubber tubing with a suitable clip, which can be released and replaced as necessary and connected with a self-retaining rectal nozzle and provision is made for raising and lowering, mechanically or by hand. It will be found convenient to run in the emulsion half a pint at a time and to mark the skiagrams accordingly. Generally speaking a half pint fills the rectum and pelvic colon. The first film is taken and marked "half pint." Another half pint is then run in and reaches the splenic flexure. There is usually a little hold-up at this point. A third half-pint reaches and fills the hepatic flexure and a fourth, the caecum. If the small intestine is not entered at this point, a little more—4 ozs. or so assisted by gentle massage, will cause some filling of the terminal coil of the ileum.

As the examination proceeds note should be made of abnormalities, such as constriction, dilatation, stoppage, deformities, and a careful search for tenderness made and necessary skiagrams taken. The patient is then allowed to go to the bathroom to empty his bowels, or if he is unable to do this, he is given a bed-pan.

After this, one or two skiagrams marked "after evacuation" are taken.

If indicated, particularly if a neoplasm is seen, or suspected, air is now introduced per rectum, usually by means of a Higginson's rectal syringe with the valve removed, so that pressure and distension may be controlled in the case of excessive pain or discomfort. Further skiagrams are then taken with a view to demonstrating ulcers or tumours projecting into the lumen.

THE NEGLECT OF THE GASTRO-DUODENAL MUCOSA BY CLINICIANS AND RADIOLOGISTS

A radiological examination of the upper part of the alimentary tract should include skiagrams which show the mucosal pattern from the fundus of the stomach to the jejunum. If it does not do this, it may be worthless or even worse, misleading.

Fluoroscopy is not a satisfactory substitute for skiagrams. Skiagrams are necessary not only to permit leisurely study and to demonstrate the lesion to the clinician who will be expected to treat it, but also because skiagrams show lesions which remain undetected during fluoroscopy. The technical procedure for demonstrating the mucosal pattern should include the following: (1) posturing of the patient; (2) the use of the air naturally present in the stomach to provide a double-contrast air-barium relief picture; (3) compression with mechanical devices, dosed under fluoroscopic control; (4) multiple spot-films, with narrow coning not only at the tube end, but also at the film end of the incident ray, so that Bucky grids may not be needed. The mucosal-relief method and the half-full stomach technique should supplement, not exclude, each other. Of the two, the former is the more valuable, but the less used.

Just as the symptoms may be absent in some cases and characteristic in others, so also may the radiological, gastroscopic and test-meal findings, when taken by themselves, be characteristic in some cases, absent in others. Thus, while any one method taken alone may be most misleading, a great measure of diagnostic accuracy may be achieved by using to the full the data contributed by all four methods—the clinical, the radiological, the gastroscopic and the fractional test meal. In some cases, laparotomy, gastrotomy and histological examination may be needed.

Some of the radiological signs of chronic gastritis depend upon a disturbance of the living reactions (secretory, motor and vascular) of the stomach; others depend upon gross anatomical disruptions of the mucosa by erosions and small-celled infiltration and fibrosis.

The radiologist is understandably wary of "over-reading" his skiagrams; but he can remain on safe ground by objectively describing the deviations which he sees, whether of function or of structure (the increased irritability, the segmentation of rugæ, the osteophyte, the calcified vessel etc.), leaving to the clinician the task of evaluating the significance to the particular patient of these deviations. The radiologist is on safe ground if he reports—"there is such-and-such a deviation." But he is not on safe ground if he adds or implies: "It is of such-and-such significance." It is for the clinician to make these additions. If the referring clinician is unable to do this, the radiologist should not be trapped into the false position of undertaking a task which does not fittingly belong to him. Instead, a third opinion should be sought from a consultant clinician.—(*The Med. Jour. Austral.*, 25-9-1954).

PERTUSSIS (WHOOPIING COUGH)*

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Introduction.—Clinical diagnosis of pertussis is possible on the triad of suspicious cough, bloated eyelids, and soft, painless, and progressive splenomegaly. Pertussis is worse in children with allergic heredity and hæmoptysis has to be recognised as a common and serious complication particularly in them; it may herald also a flare up of latent tuberculosis. Hepatic cirrhosis appeared as sequel in a few children who suffered from enteric symptoms. Streptomycin therapy appears to be ideal.

Epidemic as well as sporadic pertussis picks out chiefly children under six, with a greater predilection for girls and is characterised by a respiratory catarrh and paroxysmal coughing ending in a whoop. It can be congenital, and I have seen pertussis in babies during the mother's puerperium. The statement that one attack protects from further infection does not entirely hold good in my experience. Measles, pertussis, and diphtheria occur in the same patient as inseparables.

Bacillus pertussis is the infecting organism, which is preferably isolated by the cough-plate method early in the paroxysmal stage. Pulmonary collapse and emphysema, and enlarged hilar glands are seen post-mortem in uncomplicated cases.

Infection spreads directly by droplets, although fomites and infected clothes may also spread it. Infectivity lasts for 6 weeks from the onset. Quarantine is for about 3 weeks.

SYMPTOMS:—The incubation period is from 1 to 3 weeks, with an optimum of 2 weeks. Symptoms in the catarrhal and paroxysmal stages are described.

The catarrhal stage (duration one week) consists of a feverish cold with gastric upset. Cough out of proportion to the catarrh, is worse at night, and tends to become paroxysmal in the absence of a complicating pneumonia.

The paroxysmal stage (duration 4 weeks), consists of repeated expiratory barks ending in a respiratory "whoop," and the child is seen congested and exhausted. Vomiting sets in and clinches the diagnosis. Paroxysms may be only a few or as many as forty per day; they get always worse at nights. The whoop is ill-developed in some infants and is absent in the presence of a complicating broncho-pneumonia.

Examination of the lungs may reveal a few scattered rhonchi. Sublingual ulcer, tachycardia and accentuated pulmonary second

* Specially contributed to the ANTISEPTIC.

sound, bloated face, cyanotic tendency, raised leucocyte count with marked relative lymphocyte preponderance, and retarded sedimentation rate are all characteristic. I attach great importance to a progressive soft painless splenomegaly as an important diagnostic aid in pertussis. Paroxysms increase, decrease, and ultimately disappear, except when the throat remains unhealthy with septic tonsils and adenoids, and maxillary sinusitis.

Complications.—1. Pulmonary:—Broncho-pneumonia, sometimes tuberculous, is always dreaded. Pulmonary collapse. Interstitial emphysema and pneumothorax. 2. Gastro-enteritis and emaciation. Vomiting may become marked and may be accompanied by diarrhoea, especially in summer. (The diarrhoea present during incubation—parenteral diarrhoea responds to vaccine therapy, while the later diarrhoea promptly responds to streptomycin therapy). 3. Nervous: Convulsions (encephalitis? air embolism?), paralysis and encephalitic sequelæ. 4. Development of herniæ, rectal prolapse, and various hæmorrhages (epistaxis, subconjunctival bleeding, and intracranial bleeding). I have observed persistent hæmoptysis which is probably due to a basic allergic explosion (hæmorrhagic necrosis at the prepared site) during the course of pertussis and this responds to parenteral use of vitamins C and K. Hæmoptysis indicating a flare-up of pulmonary tuberculosis during pertussis is diagnosed promptly by finding tubercle bacilli in the sputum or in gastric washings, and a positive Mantoux reaction. X-rays of the chest and progressive deterioration in the general condition may be 'not indicative.' 5. Nephritis (frank and interstitial) complicating pertussis has also been observed by me in a few children, and albuminuria clears leisurely and along with it, occurs improvement in the general condition.

Sequelæ are pulmonary and glandular tuberculosis, chronic bronchitis and asthma, emphysema, pulmonary fibrosis and bronchiectasis. Pigeon-breast chest may appear. Otitis media and maxillary sinusitis are chronic residues. Immediate relapses are rare. Second and subsequent attacks usually described to be rare, are however, in my experience and opinion very common. Diagnosis is in recognising the whoop (vagal irritation?), and using the bacteriological cough-plate method for confirmation—which gives an earlier result.

PROGNOSIS is always serious. Mortality is high in the younger infants and invariably due to complicating broncho-pneumonia, the incidence of which appears to be less in the tropics and particularly now after routine chemotherapy and the use of antibiotics. Children who recover may later develop pulmonary damage, an intractable marasmic condition and sometimes hepatic cirrhosis.

PROPHYLAXIS:—Protection to the new-born is achieved by administering pertussis vaccine to the pregnant mother. Hyper-immune sera and vaccine therapy are useful in prevention. Risk

of APM is reported after vaccine administration. Encephalopathy is a risk and I have had personal experience of it. How far the make-up of the child is responsible for the convulsions, is difficult to assess. Familial allergy is probably the potent basis, and in families with allergic stories of asthma, eczema, migraine, or epilepsy or eclampsia, it is wise not to exhibit vaccine therapy as a prophylactic and much less as a curative.

TREATMENT:—Isolation is necessary. Light clothing and frequent small feeds of milk and milk preparations, rice and pepper water (semi-solid), and thick *conjees* of wheat and rice powder sweetened with milk and sugar are advised. Chemotherapy with sulpha-drugs may form a routine as every pertussis patient has latent broncho-pneumonia. A polyvitamin supplement is needed.

During the catarrhal stage, expectorants, and during the paroxysmal period sedatives (phenobarbitonum sodium, gr. 1/24 per year of age; Tinct. Belladonna minim $\frac{1}{2}$ per year; papaverine; Tinct. Camphor Co 4 minims per year; Ephedrine gr. $\frac{1}{2}$ at 6 years age) are used. Vitamins C and K are useful. For mild infections, Vaccine GL 0.25 cc., 0.5 cc., 0.75 cc. at 3 or 4 day intervals is useful. This is however, useless in severe cases.

Recently antibiotics have come to be used and I have used the following:—For children with pyrexia, gastroenteritis and whoops, parenterally Streptopas—54% Dihydrostreptomycin and 46% PAS. The content of one bulb is dissolved in 4 cc. of double distilled water; 1 cc. of this is given I.M., daily for 4 to 8 days with good results and more prolonged therapeutic effect.

For children with pyrexia (broncho-pneumonia) and occasional whoops, (a) Chloromycetin palmitate was orally given for 6 days only; (b) Aureomycin soluble tablets were given for 6 days; (c) Terramycin oral suspension was given for 6 days. In all these groups immediate improvement was visible (in pyrexia and respiratory symptoms). Two to four days after cessation of the antibiotic, symptoms recurred demanding further treatment. Therefore, I gave to a batch of children Chloromycetin palmitate orally and concurrent vaccine GL (0.2 cc., 0.5 cc., 0.75 cc. on 2nd, 4th, and 7th day respectively) with greater and more lasting relief. This procedure is very efficacious and avoids prolonged antibiotic therapy and its untoward consequences. Convalescence must be slow, in open air places, and supported by a tonic. Colds and febrile illnesses should be avoided.

Conclusion.—Very short Streptomycin therapy is ideal as it is perhaps directly lethal to *B. pertussis*, and prevents early relapses.

Radiological observations.—1. Child, R., male, 10 months old. Admitted with fits and broncho-pneumonia during whooping cough. 20-11-1953: Chest A-P: mottling seen in hilar zones and right base; skull, nil particular. 27-11-1953: Chest A-P: mottling

persists even more over hila and right base inspite of the fact that penicillin and streptomycin were used, with great clinical improvement. (*vide pictures 1, 1-a and 1-b*).

2. K., female child, 7 years. Admitted with whoops of 3 months' duration and with subconjunctival hæmorrhage. 16-11-1953: Chest A-P: diffuse mottling present. 27-11-1953: Chest A-P: diffuse mottling with pneumonitis in the right upper zone. Clinical improvement was noted on dihydrostreptomycin. (*vide pictures 2 and 2-a*)

3. Child, S., female, 3 years. Admitted for broncho-pneumonia after pertussis. 4-12-1953: Chest A-P: dense homogeneous opacity in right midzone, strictly limited by the transverse fissure. Pneumonitis, right midzone. 7-12-1953: Chest A.P: picture suggestive of interlobar effusion. 14-12-1953: Chest/lateral view fluid level suggestive of interlobar effusion. All these findings seen in spite of the clinical improvement obtained with chloromycetin palmitate for four days prior to admission into hospital (2-12-1953). Discharged clinically improved on 1-1-1954 (Mantoux negative). (*vide pictures 3, 3-a, 3-b and 3-c*).

4. Child, P., male, 7 years. Admitted on 27-1-1954, and discharged on 16-2-1954. Duration of whooping cough 7 days on admission. Hæmoptysis for one day. Mantoux on 6-2-1954, positive. X-rays Chest A-P: 27-1-1954: mottling on right upper and left midzones suggestive of Koch's bacillus infection? 11-2-1954: dense shadows with mottling on right upper zone (tubercular). Treated with Calcium, Vitamins C and K, penicillin. This appears to be a flare-up of tuberculosis during pertussis. (*vide pictures 4 and 4-a*).

5. Child, S., male, 2½ years. Admitted on 11-12-1953 and discharged on 17-12-1953. Pertussis with broncho-pneumonia. Chest A-P: 15-12-1953: dense hilar shadows and mottling over right lower zone present. Treated with sulphathiozole and penicillin; a control to streptomycin-treated or chloromycetin-treated children. (*vide picture 5*).

Conclusion.—Immediate clinical improvement is not reflected in radiological findings which are of the nature of large shadows, fluid accumulations, and diffuse mottlings. Do these shadows indicate local *allergic explosions* like urticaria and angioneurotic œdema? If so, do *toxins* (?) released from infecting organisms (B. pertussis or others) under the antibiotic therapy produce these allergic reactions?

(I am thankful to Dr. M. A. Latif Khan, M.B., B.S., Superintendent, Government Royapettah Hospital, for his encouragement. To Dr. M. K. T. Rajan, B.A., M.B., B.S., D.M.R., our radiologist, who helped me enthusiastically in taking the pictures and in interpreting them, my special thanks are due. My Assistant Dr. S. Krishnan, M.B., B.S., took kind care of the routine and gave me much help).

(I am thankful to the Representative of Messrs. Ranbaxy & Co. Ltd., Madras for a free supply of Streptopas and to the Representative of Messrs. Parke, Davis & Co. Ltd., for a free supply of Chloromycetin palmitate for clinical trials).



FIG. 1. R., male child, 10 months Chest A.P. (20-11-'53).

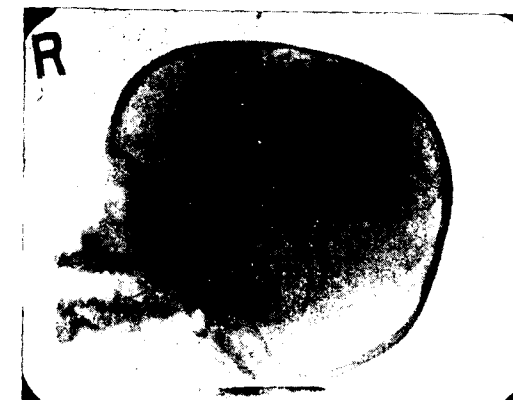


FIG. 1-a. R., Skull (20-11-'53).



FIG. 1-b. R., Chest A.P. (27-11-'53).



FIG. 2. K., Female 7 years. Chest A.P. (16-11-'53).

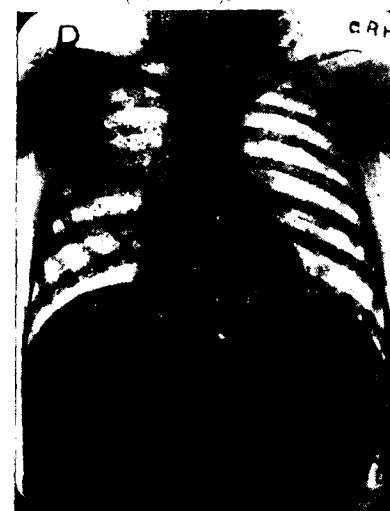


FIG. 2-a. K., Chest A.P. (27-11-'53).



FIG. 3. S., Female child, 3 yrs. Chest A.P. (4-12-'53).

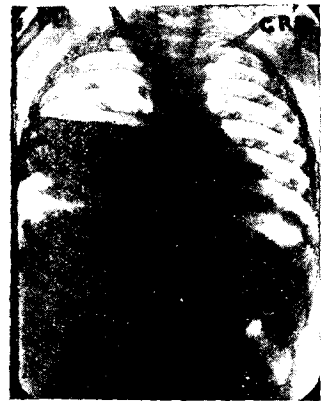


FIG. 3-a. S., Female child, 3 years.
Chest A.P. (7-12-'53).



FIG. 3-b. S., Female child, 3 years.
A.P. (14-12-'53).



FIG. 3-c. S., Female child, 3 years.
Lateral view (14-12-'53).

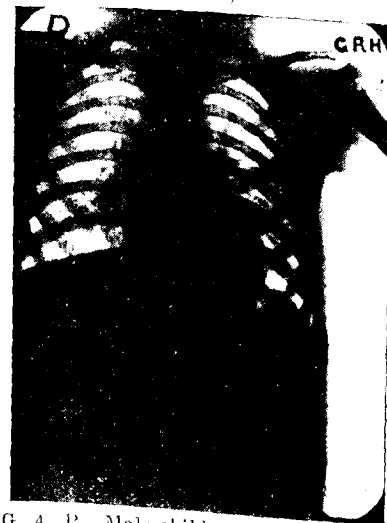


FIG. 4. P., Male child, 7 years. Chest A.P.
(27-1-'54).

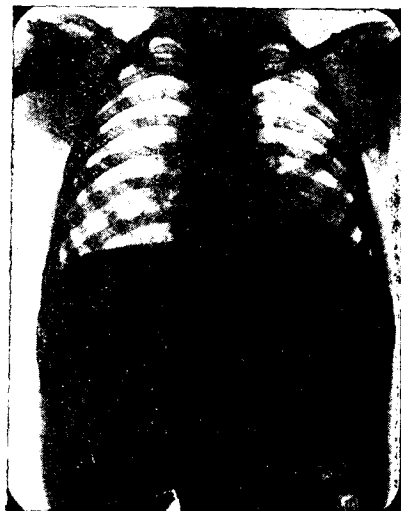


FIG. 4-a. P., Male child, 7 years.
Chest A.P. (11-2-'54).

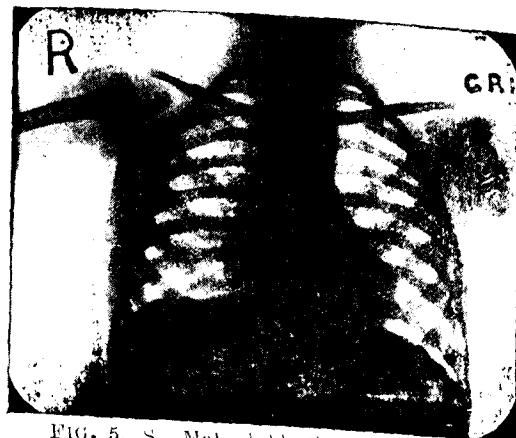


FIG. 5. S., Male child, 24 years. Chest A.P.
(15-12-'53).

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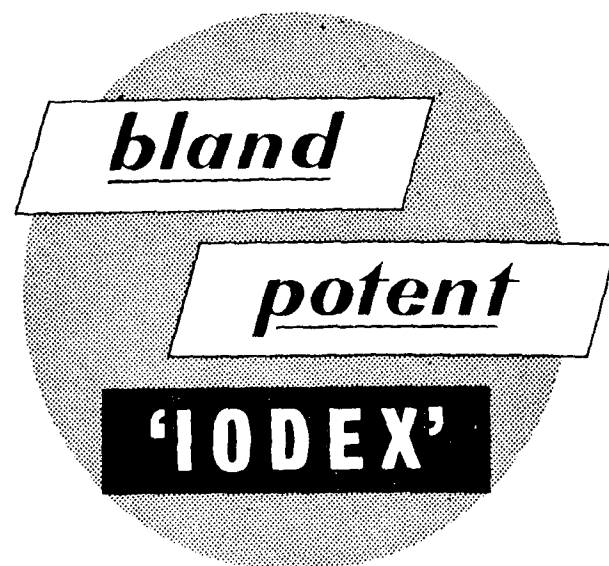
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THE ROLE OF GRANULES OF EOSINOPHILE LEUCOCYTES*

S. BALASUNDARAM,

Director, The Ehrlich Laboratory, Madras-14

THE origin of blood cells is still the subject of controversy and opinions differ. There are at least thirteen differing views. Several haematologists from Ehrlich (1898) down to Ferrata (1933) have expounded their own views. These various concepts may be classified as:—(1) Unitarian; (2) Dualistic; and (3) Trialism.

According to the Unitarian theory, the cells are derived from undifferentiated mesenchymal cells. The Dualistic theory considers that the sources are myelogenous and lymphatic. The 'Trialism' school of thought headed by Schilling, the Austrian haematologist and his followers, believes in the three points of development of white cells, *viz.*, myelogenous, lymphatic and from the histiocytes of the tissues. This theory has found acceptance with most haematologists of the present day. The difference of opinion that exists relates mainly to the development of the lymphocytes and monocytes. Many observers have agreed on the myeloblastic origin of the granulocytes; or at any rate of the granulocytes having a different origin to that of other leucocytes. Thus these cells form a class by themselves.

There are again differences of opinion as to the life-span of a leucocyte which is considered to be about two hours by some and two to three weeks by others. The precise mode of destruction of the white cells has not been established definitely. Under normal conditions the number of white cells in the body is remarkably constant except for post-prandial leucocytosis. Some observers feel the even this leucocytosis is not so pronounced after a meal consisting of non-albuminous food as after an albuminous diet. When a leucocytosis does occur, the bone-marrow, spleen and even the lymph glands show signs of hyper-function and haemopoietic activity. The slight increase noticed, after food, has been attributed by some to a daily physiological increase during afternoons irrespective of the food taken.

The sum total of these observations is that under physiological conditions the leucocytes have a particular constant pattern of life and do not show any evidence of having any special roles in the daily routine.

The white corpuscles are typical animal cells, and so have all their characteristic features, and contain the following substances generally found in such cells; *viz.*: proteins, fats, glycogen, purines,

* Specially contributed to the Golden Jubilee issue of the ANTISEPTIC

enzymes, phosphatides, cholesterol, inorganic salts and water. Nucleoproteins predominate in the compound proteins found in the leucocytes. Powerful proteolytic and glycolytic enzymes are also present. The proteolytic enzymes in leucocytes are believed to be of two kinds: one active in alkaline solution and present in the granular leucocytes and the other active in acid medium, and present in the monocytes.

When the leucocytes come in contact with water, their amoeboid movements soon stop; their cytoplasm gets swollen by the intake of fluids and the granules within the protoplasm show active Brownian movement. This experiment reveals that the leucocytes have superficial films or membranes which act as osmotic membranes. The addition of an excess of water bursts the leucocytes, which get disintegrated and set free the granules.

Substances insoluble in water and unaffected by the action of enzymes do not attract the leucocytes; but substances like glucose, and glycogen are positively *chemotactic* to the leucocytes. Acids produce a granular precipitate around the nucleus.

From these and other observations, it has been found that the enzyme activity in the leucocytes is an important feature of the cellular activity of the cells. The granules come into play, only when chemical activity is needed; otherwise in neutral substances like water, they float about without undergoing any change.

Though the functions of the leucocytes have been extensively studied, the part played by them in physiological conditions, has not been assessed finally or fully. Their role in pathological conditions has been recognised. Even during normal physiological states the leucocytes take charge of the abnormal or the pathogenic products found during the normal functioning of the system (Medlar).

Among the granulocytes, the neutrophile leucocytes have been observed to take part in the phagocytosis of most types of bacteria. Enzymes capable of liquefying and absorbing injured or dead cells and tissues are produced by these cells. The tendency in damaged tissues to repair themselves has been found by experiments on tissue cultures with extract of leucocytes, to be stimulated by the neutrophils.

We have very little information however, about the function of these eosinophils. They are rarely phagocytic. The large number of granules found in them are bright, crystal-like and are highly refractile. In the blood of horses these granules are larger and are more oval in shape. The refractive nature and their prominence leave us in doubt as to whether they are really solid particles or liquid or semi-liquid substances encapsulated by a permeable membranous structure. A study of the granules of the eosinophile leucocytes is really the study of the leucocytes themselves.

Conditions in which the eosinophile leucocytes are increased afford opportunities for us to study their role. Eosinophilia occurs in three important pathological conditions *viz.*: (1) Infections with animal parasites (helminths); (2) skin diseases depending more on the extent of the disease than on the type of skin involvement; and (3) allergic states due to foreign proteins or endogenous toxins or similar substances.

An increase may also occur in diseases of the hæmopoietic organs where there is a general increase of all the leucocytes. This, however, is beyond the scope of the present communication.

A significant fact in this connection is that eosinophils are definitely diminished in the acute stages of severe infections. We will now consider the eosinophilia met with in helminthic infections and in allergy due to proteins like histamine.

In the investigations he carried out, John Vaughn of the St. Bartholomew Hospital, London, prepared two extracts of *ascaris suum*: (i) one with a mixture of polypeptides and other protein break-down products; and (ii) the other with a poly-saccharide. He injected these two intra-peritoneally into guineapigs. The result of his study showed that the former (i) was a more powerful eosinophilogenic agent than the latter containing the poly-saccharide. He further observed that an anti-histaminic substance given before the poly-saccharide injection prevented the increase of eosinophils from *Ascaris* extract. Two important factors emerge from this study:— (1) That histamine is an active agent in producing at least some forms of eosinophilia; and (2) in cases of production of eosinophilia by other methods, like the *ascaris* extract, the presence of a polypeptide is essential to produce the desired results. In other words, the eosinophilic response depends on the presence of a protein substance.

Some experiments on the staining reactions of blood cells have been done by some observers and their findings are of great significance in this connection. Little, employed the differential destructive action of nitrous acid on primary amines and found that the eosinophilic granules were not affected by the nitrate treatment, even when prolonged for six hours. Whereas the same treatment within half-an-hour altered the staining reactions of nuclei of the other leucocytes, erythrocytes and other elements of blood. This experiment shows that the chemical nature of the granules of eosinophils is different from that of the other leucocytes and their granules. Whether the granules are solid particles or liquid or semi-solid substances encapsulated by permeable osmotic membranes, they seem to have a selective action on certain substances.

The granules are found to contain iron in sufficient quantity; but the part played by these granules in the iron metabolism of the system is not sufficiently known, to be able to assess the role of

THE TREATMENT OF INTRACRANIAL ARTERIAL ANEURYSMS

Intracranial aneurysms are very much "in the news" at the present time. The application of angiography to the diagnostic problems and the improvement in operating conditions made possible by anæsthetic advances have engendered a wave of enthusiasm and optimism in their treatment.

The vitally important factor from the point of view of treatment, however, was the determination of the exact site of the aneurysm and the state of the collateral circulation. Lack of this knowledge led to the adoption of non-specific forms of treatment, such as ligation of the carotid vessels in the neck. In a significant proportion of such cases hemiplegia followed, or the symptoms of the aneurysm, most commonly subarachnoid bleeding, failed to regress. Recent work has clearly shown that carotid ligation in relation to recent subarachnoid bleeding is productive of hemiplegia in a high proportion of cases (Brackett, 1953).

Cerebral angiography, unfortunately, was developed only slowly, and it was not until after the 1939-45 war that reliable methods of percutaneous arterial puncture of both carotid and vertebral arteries were devised. This new method, coupled with the use of relatively innocuous contrast media of the "diadone" group, enabled angiography to be carried out rapidly and safely.

The widespread use of angiography not only made available much information of use for treatment but improved the clinical diagnosis of the condition. Aneurysms began to be picked up in patients hitherto unsuspected of having such lesions, and as more and more information accumulated the clinical picture of aneurysms in various situations became clearer. As clinical diagnosis became more reliable it did not replace angiography, but, on the contrary, angiography became more and more essential, for it was the only method by which the collateral circulation could be demonstrated.

There is no standard treatment applied to intracranial aneurysms. Each case is an individual problem and treatment will be determined by the site and size of the aneurysm, whether it is bleeding or not, the state of the collateral circulation and the results of carotid compression tests. It will be the aim in every case to cure the aneurysm either by establishing thrombosis in the sac or removing it, and this must be accomplished with a minimum of physical disability.

Aneurysms of the carotid artery itself form the largest group; some are in the intracavernous portion, but many are on the intradural portion of the artery. They present as cases of subarachnoid hæmorrhage, or with involvement of the third and is usually a small one and arises from the posterior aspect of the vessel.

Aneurysms on the middle cerebral artery pose a more complex problem. They are usually situated on the trunk of the artery at the first bifurcation in the sylvian fissure. Cases present with focal fits and hemiplegia, usually due to intracerebral bleeding from the aneurysm. The aneurysm is situated away from the circle of Willis, and so no collateral vessels are available and, in addition, the sac usually has a short, wide neck and ligation of this is impracticable. Emergency measures may be necessary in these cases and exposure of the brain and removal of an intracerebral clot may be life-saving. The aneurysm can be easily cured by ligation of the middle cerebral artery and removal of the sac. Such a procedure however, inevitably results in a hemiplegia, and aphasia if the sac be left-sided. Whilst such a measure may be necessary finally to preserve life, initial attempts to reinforce the wall of the sac whilst leaving the lumen of the artery in tact are desirable. The results in this group are bad; severe physical disability is the rule.

Anterior cerebral aneurysms are often large and usually involve the junction of anterior cerebral and anterior communicating arteries. This involvement of the collateral trunk makes treatment extremely difficult.

Aneurysms on the posterior communicating, posterior cerebral and basilar arteries are rare, and there is insufficient information at present available to make any useful statement concerning their treatment and prognosis.—(Brodie Hughes, Ch.M., F.R.C.S., Neurosurgeon, Birmingham in *Med. Press*, Feb, 1954).

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Cases and Comments

THE LAURENCE-MOON-BIEDL SYNDROME

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Professor of Ophthalmology, S.C.B. Medical College, Cuttack

IN 1866, Laurence and Moon reported four cases of Retinitis pigmentosa in the same family, three males and one female, which showed certain atypical features. These cases presented a combination of dwarfism, hypogenital dystrophy, retinitis pigmentosa and mental retardation. Bardet in 1920 distinguished a form of hypophyseal obesity which seemed to be of congenital origin. This form was accompanied by malformation (polydactyly or syndactyly) and Retinitis pigmentosa. In 1922 Biedl showed two cases of adiposogenital dystrophy in the same family which were characterised by congenital anomalies:—polydactyly, atresia ani, retinitis pigmentosa, and mental sluggishness. These two cases were reported in greater detail by Raab in 1924. Cases with the syndrome incomplete has been previously reported, but to Bardet and Biedl belong the credit of describing the complete syndrome. The disease is therefore, known in the literature by several names, generally as the Laurence-Moon-Biedl Syndrome although it is also spoken of as the Laurence-Bardet or as the Laurence-Moon-Bardet-Biedl Syndrome (Elwyn). Complete reviews of the reported cases have been given by Reilly and Lissner (1932, 77 cases) Cockayne, Krestin and Sorsby (1935, 101 cases) Sorsby, Avery and Cockayne (1939, an additional 49 cases). In India cases have been reported by Kutumbiah and Abbu (1942); and Muthayya and Iyer, quoted by Kutumbiah and Abbu (1942), Gantayat and R. Rao (1952).

Clinical picture:—The complete syndrome includes: (1) obesity; (2) genital dystrophy; (3) retinitis pigmentosa; (4) polydactyly; (5) mental retardation; and (6) familial occurrence.

PATHOLOGY:—The association of obesity of the pituitary type with hypogenitalism suggests an underlying pituitary or hypothalamic defect. Mararon (1941) regards them all as one component-adiposogenital dystrophy. Reports of two post mortem examination have been added to the literature since 1938 by Anderson (1941) and Radner (1940). The post-mortem findings were:—the reproductive organs were rudimentary, the anterior lobe of the pituitary consisted chiefly of basophil and eosinophil with very few chromophobe cells, there was a colloid goitre and the brain and its blood vessels were normal (Anderson, 1941).

The following case came under observation on 12-3-1954 and was admitted into S.C.B. Medical College Hospital.

Case report.—T. D., aged 15 years, Hindu female was admitted into S.C.B. Medical College Hospital on 12-3-1954 for defective vision during the previous 8 to 10 months.

FAMILY HISTORY:—Her mother died when she was very young. She was the only child of her parents. Father is healthy. Mother is said to have had supernumerary digits and poor vision.

General features of the patient:—She is slow and lazy; has poor memory and cannot follow instructions or answer questions clearly. Has the voice of a female child. Special deposits of fat on the abdomen, thighs, buttocks and breasts. Secondary sexual characters not developed as evidenced by the absence of axillary hairs. Bridge of the nose somewhat depressed; has no cranial deformity; has supernumerary toes, on both sides of the feet; polydactyly; has rudimentary knobs on both the hands on the ulnar side; skiagram does not reveal extra digits in both hands. (*vide* photograph). Height-4 ft. 6½ inches. Weight-87 lbs.



Vision—Right eye can count fingers at less than ½ metre distance. Left eye can count fingers at less than one metre distance. Has nystagmus of the rotatory type in both eyes. Ophthalmoscopic appearance of the fundus and media:—both eyes are almost of the same appearance. Optic disc yellowish-white in colour. Blood vessels considerably narrowed. Retina atrophic, sclerosis of choroidal vessels present. Only one solitary black pigment present on the inner and lower quadrant of the fundus of the right eye. No other pigmentary disturbance found in both the fundi. Field of vision could not be recorded due

to want of co-operation and poor vision of the patient. General condition: Not anaemic, well nourished. Genitals: Excess of fat about the extremities and proximal portion of thighs, abdomen, buttocks, breasts (secondary sexual characters not well-developed); has infantile uterus. Cardio-vascular system: Pulse regular, 68 p.m. of normal volume and tension and no other abnormality in the cardiovascular system detected. Respiratory system: 18 per minute, regular, no abnormality seen. Alimentary system: normal. Laboratory findings: B. M. R.—31.5% W. R.—Negative. Urine—normal. Stool shows a few hookworm ova. Blood: total R.B.C. 4.02 million/cm. Total W.B.C. 8,500/cm. Hb-10.3 gram %. Differential

Count: Nentrophil—54%; Lympho—30%; Mono—2%; Eosinophil—14%; Baso—0%.

Comment.—The case shows the features of the Laurence-Moon-Biedl Syndrome with some variations in its seven components, *viz.*: obesity; genital dystrophy; nystagmus; mental deficiency; polydactyly; retinitis pigmentosa; familial occurrence.

The first 5 factors are fully developed. The most variant factor is the pigmentary disturbance, the case shows the least pigmentary disturbance, although all the features of retinal degeneration are present. The family history is not very clear due to incomplete data; she is the only child of her parents and the mother is dead. Excepting the last two features all the other features of Laurence-Moon-Biedl Syndrome are well-marked.

Summary.—1. The literature on the Laurence-Moon-Biedl Syndrome is reviewed. 2. Though the sex-ratio is greater in males than in females, the case reported here is that of a female. 3. The feature of pigmentary disturbance in the fundus is least marked.

Acknowledgement.—I am grateful to Dr. S. C. Das, M.B., Ph.D., E.R.S.E., Principal and Superintendent, S.C.B. Medical College and Hospital for his kind permission to publish the case and also to the departments of Physiology and Obstetrics and Gynaecology for their kind help and to my Junior Surgeon and House-Staff Dr. K. Misra, Dr. U. Naik and Dr. H. Sahu for their assistance.

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2. Burn, R. A. (1959)—The British Journal of Ophthalmology, 51: 65-88.
3. Gantayat, S. N. and Surya Prasad Rao, R. (1952)—Jour. I.M.A., 21: 8, May.

OXYGENATION OF RETINA

Abnormal electro-retinographic findings are usually obtained in patients with hypertension or arteriosclerosis, even though ophthalmologic examination is normal. Most of 15 patients with hypertension had super-normal responses, whereas 15 patients with arteriosclerosis had sub-normal or normal b-potentials. The hypertensive super-normal responses reflect irritability of the retinal neurones as a result of metabolic deficiencies. Subnormal responses of arteriosclerotic patients appear to reflect retinal oxygen insufficiency. Prolonged rest in the dark before electro-retinographic procedures appears to improve retinal oxygenation of arteriosclerotic individuals so that the subnormal b-potentials revert to normal.—(*Angiology*, 5: 49-58, 1954; *Mod. Med.*: Short Report: 1-9-54).

OCULAR LESIONS WITH INFECTIOUS MONONUCLEOSIS

These may include bilateral iridocyclitis and papilloedema. In some instances, conjunctivitis, peri-orbital oedema, optic neuritis, uveitis, and retinal disorders may be caused by the viral infection. The disturbances in the visual apparatus with the accompanying pain upon rotation of the eyes and backward pressure on the globe are of short duration and disappear without tissue destruction or permanent functional loss.—(*Arch. Ophthalmol.*, 51: 240/1954; *Mod. Med.*, Sep. 1954).

ANOMALIES OF FOETAL DEVELOPMENT HYDRENCEPHALOCLE (A Case Report)

S. RAMANATHAN, D.M.P.,
Kalyani Clinic, Vadugapalayam, Palladam

ON 16-10-54, at 4.30 p.m. a full term primipara, aged about 20 years, was brought to my clinic with the following history:—

According to calculations, her delivery was expected any moment; she complained of bleeding for about three hours, which recurred; no severe pain was experienced except back ache.

On admission she reported recurrence of bleeding which was considerable. Her general condition and pulse appeared satisfactory. She had no labour pains. Her blood-pressure was 110/80 mm. Hg. On auscultation, foetal heart sounds could not be heard.

The assistance of a midwife was sought and a P.V. was made. The os was three-fifths dilated and the membranes were not present. The presenting part was above the brim and movable and had not the normal configuration of the head. An ovoid tumour which was soft and pliable in consistency could be felt. It appeared very much like a placenta prævia (posterior).

A decision was taken to terminate labour as the patient was losing blood and the foetus appeared dead, as evidenced by the absence foetal heart beats etc.

The patient was given intravenous injections of glucose and Calcium gluconate 50% and 10% respectively; she was then anaesthetized, when it was possible to have a better and clearer idea of the presenting part. It was a cystic tumour, ovoid and outside the cranium.

The non-engagement of the presenting part and its persistence over the pelvic brim were probably due to the presence of the tumour. The same was punctured when fluid and brain substance were found to escape. This afforded a better opportunity for further manipulations. It was thus possible to define clearly the presenting part and spot out the mouth of the foetus. Traction was applied and the head delivered with difficulty.

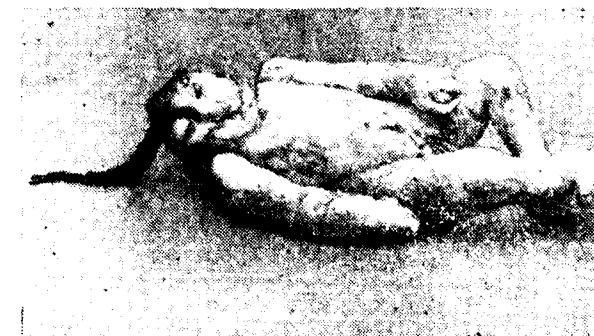
The delivery of the shoulders was still more difficult than of the head. Traction and fundal pressure having failed, the fingers of one hand were passed and the posterior axilla hooked. As it was not possible to effect delivery by this, the fingers of the other hand were also passed to hook the anterior axilla. Then traction was applied alternately with both hands. The shoulders having responded, the rest of the body was delivered. It was then

noticed, that the extreme size of the shoulders of the foetus was only a part of the general foetal enlargement. The third stage presented no difficulty but a tear of the perineum occurred during the delivery of the shoulders and body which was repaired by sutures. The patient was given prophylactic pituitary and penicillin injections to guard against post-partum hæmorrhage and sepsis.

Delivery was effected by 10.30 p.m. The child (a dead male child) presented a monstrous and frightful appearance and weighed 10 lbs! The patient was properly looked after during the puerperium, no complications occurred and she was discharged on the ninth day.

I am reporting this case as anomalies of foetal development are rare and a Hydrencephalocle is said to be rarer still. The diagnosis

about the presenting part, the possibilities of a posterior placenta prævia, delivery of the enlarged shoulders and body were some of the difficulties encountered in this case.



A few points about the foetus to be noted:

1. Tail-like projection seen at the anterior portion of head—the punctured hydrencephalocle.
2. Enlargement of shoulders—General enlargement of foetus.
3. Deformity of feet—Dorsiflexion at the ankle joints (bilateral).
4. Deformity of hands and fingers—Contraction of hands and fingers (bilateral).
5. Extraordinary growth of hair—Upper portion of forehead, side of neck, and back.
6. Appearance—Monstrous and frightful.

VULVAL LUMINESCENCE

In certain lower forms, the colour of the genital skin changes with hormonal function. To MacDonald and Margolese must go the credit of first describing the phenomenon of human-female-external-genital luminescence which, by their definition, is absorption of radiant energy with re-emission at wave frequencies characteristic of the absorbing substance. Several other articles have followed disagreeing with some details but generally confirming the original observations. A mercury arc ultraviolet lamp is used and colour variations are correlated with sex hormone metabolism. Margolese states that the perineal and vulvar surfaces exhibit a yellow colour prior to adolescence. At puberty the colour is green, but soon becomes purple. A darker purple is apparent during pregnancy. After the menopause the colour changes to green and finally back to yellow. Benson and Chappell have attempted to interrelate observations of vulvar fluorescence with normal pregnancy, the normal puerperium, chorionepithelioma, ectopic pregnancy, abortion, and hydatid mole.—(*Obst. Gynaecol. Qly. Rev.*, 11: 2, 1954).

PNEUMONIA *cum* PARATYPHOID

(A Case Report)

Mrs. V. C. ANGULI, B.Sc., M.D.,
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THE striking incidence of enteric fever in the City speaks for itself and of the yet primitive sanitary organisation we have. It is said that typhoid fever can probably be caused by the ingestion of relatively small numbers of bacilli whereas paratyphoid fever does not usually result unless a large number of bacilli is ingested. In other words, the incidence of paratyphoid fever may be taken as a better index (than that of typhoid) of the sanitary condition of a given population.

In these days of antibiotics, the mortality from the disease is almost *nil* while the complicating pneumonic consolidations are no more a bug-bear. The distressing factor appears to be the toxæmia, resulting in toxic inhibition of the marrow and sudden collapse due to myocardial failure. Perhaps, an early transfusion—preferably from an immunised individual may save the patient.

Case report—A young man, aged 25 years was admitted into the medical wards of the Government General Hospital, Madras in a moribund state. No history could be obtained except that he was having fever since ten days. The only positive finding on clinical examination was diminished air entry into the right lung.

The following laboratory investigations were done:—*Blood*:—Total W.B.C. count—4600/c.m.; Differential count—Polymorphs 66%; Lymphocytes 26%; Monocytes 5%; Eosinophils 30%. Blood culture—*B. typhosum* isolated on the second day after admission. Blood: Widal test negative. Blood-pressure on admission was 90/50; gradually fell to 60/50 on the last day.

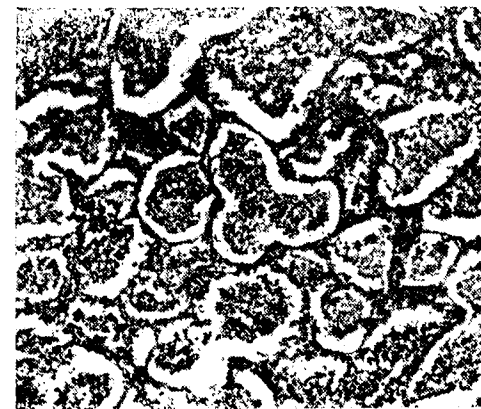
There were no abnormal findings in the other routine tests. The patient had a continuous rise of temperature—102°F to 104.3°F. Systemic penicillin 2.5 lakhs units twice daily was given. Fourth hourly coramine was also given. Despite excellent medical care, the patient died on the seventh day. A clinical diagnosis of pneumo-typhoid was made.

Autopsy notes.—Post-mortem examination showed no evidence of ulceration of the small gut. The Peyer's patches were not prominent. The spleen was moderately enlarged and contained a soft diffuent pulp. The myocardium was pale and flabby with dilatation of the right chambers. The right lung (weighing 20 oz.) was reddish grey, voluminous, perfectly solid in the lower lobe and friable. The pleural surface showed slight thickening and opacity over that

region. The cut surface presented the same reddish grey appearance and there was slight serous oozing. The left lung weighed 18 ounces and the lower lobe showed similar features. No gross pathology could be detected in the other viscera.

Summarising the above: a young man aged 25 years with a history of 17 days' fever, positive blood culture and clinical findings

was very rightly diagnosed as a case of pneumotyphoid. Autopsy revealed pneumonic consolidation of the lower lobes of both the lungs and there was no positive evidence of typhoid infection in the small intestines. Chronologically, it was logical to assume that pneumonia preceded typhoid considering the stage of grey hepatization of lungs (as confirmed by histopathological study) and the date of positive blood culture. No information was available regarding previous T.A.B. vaccination. However,



Microphotograph x 160.
The lungs: The alveoli are distended by a fibrinous exudate. There is a definite shrinking of the exudate giving rise to a halo all-round—Grey hepatization.

material taken from gall bladder, spleen and bone-marrow was cultured and "paratyphoid B" was grown.

COMMENT:—To distinguish between typhoid and paratyphoid fever is easy by blood-culture made early in the disease, while it may be difficult and sometimes impossible at a later stage when the diagnosis is attempted by means of agglutination tests alone.

With a positive blood-culture, the strain could be isolated in pure subculture and identified by morphological, cultural and biochemical characters. In the above instance, it is probable that the production of 'gas' in the fermentation sugars is either delayed or missed. The precise identification with a complete set of sugar media including xylose, arabinose and dulcitol is essential. The biochemical reactions are of value when considered with the antigenic structure. Again, it appears highly probable, that the common XII somatic antigen had played its part in the agglutination with the specific high titre antisera. In persons with a history of previous typhoid infection, it is wondered whether a mixed infection of *B. typhosum* and *B. paratyphosum* would alter the course of events giving rise to an initial positive blood culture of *B. typhosum* as if during a preliminary bacillæmia in the incubation period and the paratyphoid infection manifesting itself later. This is

just a surmise. The cause of death in this instance is definitely toxæmia and it seems reasonable to think that the therapy should aim at diluting and neutralising the circulatory toxins in addition to antibiotics and other symptomatic treatment.

My grateful thanks are due to Prof. D. Govinda Reddy, M.D., Principal and Professor of Pathology, Andhra Medical College, Vishakapatnam and to my colleagues in the Bacteriology Department, Stanley Medical College, Madras, for their valuable suggestions.

Reference :

1. Mackie, T. J. and McCartney, J. E. (1950)—Practical Bacteriology, Sixth edition, pp. 400-434.

TREATMENT OF INTERNAL HÆMORRHOIDS

Hæmorrhoids are particularly amenable to treatment, and if the surgeon fully understands their anatomy and pathology, he is in a position to offer a cure to most sufferers.

Hæmorrhoids are abnormal protrusions of the anal canal mucosa. Usually they prolapse in 3 main groups, one on the left lateral aspect of the anal canal, one on the right anterior, and the other on the right posterior. There is no satisfactory evidence to show that enlargement of the anal canal venous plexus is primarily responsible, and it is possible that the relatively constant situation of the hæmorrhoids is related to extension of tenial thickenings of the longitudinal muscle into the anal canal. Slight prolapse is not recognized as such by the patient, but the anal sphincter closes on the prolapsed mucosa to produce engorgement of their contained vessels with subsequent rupture and hæmorrhage. As the condition advances, the hæmorrhoids prolapse with defæcation and may require manual reposition. Although there is no evidence to show that hæmorrhoids are a symptom of carcinoma of the rectum, bleeding is common in both conditions and patients with hæmorrhoids may have an unsuspected carcinoma until discovered with the sigmoidoscope.

Some patients, on learning that nothing more serious than hæmorrhoids are responsible for the symptoms, are satisfied and desire no treatment. Injection therapy is useful for the patients with first degree hæmorrhoids; 5% phenol in almond oil has proved a successful agent. If care is taken with the injection complications such as pain, hæmorrhage, ulceration, and hæmaturia are rare, and long remissions are secured in patients with first degree hæmorrhoids. For the prolapsing second and third degree piles, the most satisfactory results follow the "ligature and excision" operation.

Complications following this operation are again rare. The incidence of secondary hæmorrhage has been minimized by the preoperative administration of intestinal sulphonamides. Stenosis is not uncommon after poor surgery and is often amenable to a plastic procedure involving excision of a wedge from the anal margin and the application of the primary split-skin graft.—(*Australian and New Zealand J. Surg.*, xxiii: 116-126, and *Qly. Rev. Surg. Obst. Gynec.*, June '54).

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THE MANUFACTURE AND SALE OF DRUGS IN INDIA

PHARMACEUTICAL ENQUIRY COMMITTEE'S REPORT (1954)

A PHARMACEUTICAL Enquiry Committee was set up by the Government of India in March 1953 with Major General BHATIA as President and Dr J. B. SHAH as its Secretary.

Writing in April 1953 and again in November of the same year, we stated that we were looking forward to the report of this Committee which was to go into the question as to why such large scale adulteration is being practised and to suggest ways and means to eliminate and effectively put an end to the causes that led to this evil which has grown to be a grave menace to the health of the people. The Committee's report which was submitted to Government in June 1954, was placed on the table of both the houses of the Central Legislature on the 29th September. This report states that out of 1603 pharmaceutical factories in India 1568 are small scale ones and many are located in insanitary places and their equipment is antiquated and inadequate. In November last we pointed out that it would be fatal for the future of the indigenous drug industry in India if steps were not immediately taken to ensure that only such concerns which had all the necessary mechanical devices, expert personnel and detailed processes in the manufacture of the drugs were encouraged. The Committee had come to this conclusion and said that early steps should be taken to cancel the licences and to close pharmaceutical establishments which did not meet the immediate requirements as regards premises, equipment and qualified staff laid down by the Committee. This would mean that the medical profession and the pharmaceutical industry should join together and set up testing laboratories for certifying the products made by the Industry and thus create public confidence in the drugs manufactured by them. The samples for testing should be taken at random and without

the knowledge of the manufacturer who should not know when and where the tests will be conducted. To make it more effective, surprise samples should be taken from retail shops, for as we have already pointed out, there is a considerable volume of trade in spurious and adulterated drugs by the retailers; this nefarious practice has become unfortunately much too common in many large cities and towns of India.

The Committee has suggested many amendments to the Drugs Act and the rules there under and also several measures to improve testing facilities. Regarding the scope of the Industry, the Committee is of the opinion that the pharmaceutical factories should not merely convert bulk pharmaceuticals into dosage form like tablets, capsules, compounded preparations, etc., but should also manufacture as many fine chemicals and drugs as practicable starting from the basic chemicals and intermediates. The Government should sponsor the manufacture of fine chemicals and drugs either in existing government factories or by encouraging private enterprise; and the manufacture and supply by the Government Medical Store Depots should be thoroughly reorganised on a commercial basis. The Quinine factories, the Committee considers, should be assisted by curtailing imports of Quinine and synthetic anti-malarials and by a levy of customs duty on these products. We heartily endorse this view.

The scope of the Penicillin factory at Pimpri, which was expected to go into production this year but has not done so yet, should in the Committee's opinion be extended to produce other antibiotics like Streptomycin, chemotherapeutic drugs, synthetic anti-malarials and also vitamins. 'No new foreign firms should be allowed to set up factories' says the report 'unless they undertake to manufacture new drugs or those which have not been produced in adequate quantities by other factories in India. The Government on their part should not allow a large number of firms to take up the manufacture of one and the same product; only then would unhealthy competition be held in check.'

The Committee suggests that those factories which do not come up to the required standards should be given a year in which to reform, under threat of having their licences cancelled, if they did not do so. On the question of patent laws, the Committee notes that most patents are held in foreign firms who charge heavily for their use. The Governments are asked to consider, if the International Patents Regulations should not be abrogated in the case of essential drugs and vitamins. Such abrogation was made during the last war when it was felt that such rules ran counter to humanitarian considerations.

The Committee has recommended that in the matter of control and the administration of the Drugs Act, a Central Drug Control Department should be set up under the Union Health Ministry, the

rules for the detection of spurious drugs should be further tightened up and the pay scales of Drugs Inspectors, Government chemists and other necessary personnel should be raised to assist in the development of the Industry on an ethical basis and carrying out the recommendations of the Committee.

The recent bill regarding the advertisement of the drugs gave powers to deal with false unjustifiable claims in advertisements. The declaration of an offence under the Drugs Act for trading in spurious drugs was fixed. The Committee has recommended a deterrent punishment including adequate fine; a specified period of rigorous imprisonment is made compulsory. Giving publicity to the name and address of the offender in the press and otherwise and also cancellation of his trading licence are considered essential to put a stop to the adulteration of drugs. This alone would constitute a salutary deterrent against malpractice. The making and possession of spurious drugs will then become an offence under the Drugs Act.

The Committee feels that a large number of new remedies have been introduced into medical practice in rapid succession and they are put on the market even before their effects are observed and assessed carefully. It is therefore, suggested that a panel of experts should be appointed to report periodically about the various drugs and their reports should be published. A system of fair-trade prices should be enforced, under which the consumer's price of all drugs and medicines is fixed by an organisation representing the Government and the trade. The Committee feels that dispensing of drugs should ordinarily be done by pharmacists. A person should be allowed to enrol either as a pharmacist or as a medical practitioner but not both.

Medical practitioners may however, dispense drugs to their own patients and employ qualified persons to do the actual dispensing. The Committee has also suggested that a Central Institute of Pharmacists should be set up and every state must have an institution to give training in pharmacy.

It is our considered opinion that the Committee in their valuable report have not omitted to deal with any aspect relating to the manufacture and distribution of drugs and to provide for a better and more efficient control and enforcement of the rules and regulations governing the same. It remains to be seen how the report is received by the Houses of Legislature and by the medical and pharmaceutical professions, both of whom should really welcome most if not all, the suggestions and recommendations contained in this exhaustive and well-considered report. The entire report should be carefully studied by every member of the medical and pharmaceutical professions, as it is not possible to notice editorially all the valuable suggestions contained in this bulky volume of 402 pages.

MEDICAL CONFERENCES (Nov. 1954)

The latter half of last month witnessed the holding of several conferences in different parts of India on subjects relating to Medicine and Public Health. It will not be possible in a short review to do full justice to all the important subjects discussed and the decisions reached at these Conferences. So only the salient features of all of them are presented in this short resume.

1. The All India B.C.G. Workers' Conference was held at Coimbatore on the 13th and 14th of November 1954. Shri Dr. P. V. BENJAMIN, Tuberculosis Adviser to the Government of India who presided on the occasion said that nearly 40 million persons had been tested and over 13 million people were given B.C.G. vaccination till the end of September this year in India. The target of the Government was to test and vaccinate all the susceptible population in the country below 25 years, (estimated at 170 millions) during the next five years. Speaking about the effectiveness of B.C.G. vaccination, Dr. BENJAMIN said that its efficacy could not be proved in advance, nor during the short period of three to four years it had been in progress in this country. For the present, they had to be satisfied with the findings of such studies as had been made in other countries of the world. The Madras State Health Minister stated at the Conference "It seems better to begin B.C.G. programme at the District Headquarters stations one after another, and to take up other towns later in the order of their size, population and importance and to tackle the villages finally. Referring to the new improved vaccines, Shri SHETTY said, the freeze-dried vaccine recently developed in the laboratory seemed to have come into use in France and Japan. Whether this would be a more stable product to use in warm climates and in the more remote villages of India which are legion and to which fresh liquid vaccine could not be delivered or used within the prescribed time-limit was a matter for careful investigation. Another new vaccine was Weil's vole-bacillus vaccine which is believed to give a more severe reaction but to confer greater and more lasting immunity. The comparative merits of the B.C.G. vaccine and this new vaccine are under trial. The Government of India also proposes to make large-scale trials on B.C.G. vaccine as a leprosy-preventive in the pilot projects. The mass B.C.G. campaign was inaugurated at Coimbatore on the 14th November by the Director of Medical Services, Madras.

2. Shri SANTILAL SHAI, Bombay's Health Minister inaugurating the Fifth Annual Session of the Indian Association of Pathologists appealed to the conference to direct their research so as to make the diagnosis of diseases easier and medical aid cheaper. He stressed the need to protect people against the generally prevalent diseases and thus increase the average span of life. Dr. V. S. MANGALIK in his presidential address called upon the

medical profession to accept the new emphasis on preventive medicine and methods combating mal-nutrition and infection. He also referred to the extreme paucity of trained teachers in medical colleges, the poor salaries they were paid and the enormous load of routine work which they had to do.

3. At a Conference of the Trained Nurses of India, Hyderabad State Branch held on the 15th of November 1954, Miss ADELAID HARRIS, the WHO Senior Nurse of the Niloufer Health Visitors' School at Delhi, stressed the need for post-graduate education to nurses. There was a discussion on the present conditions of the Nursing Service; plans for improvement and suggestions concerning the development of the nursing profession in the next Five-year plan were made. These will be presented to the National Trained Nurses' Association meeting to be held at Bangalore in December 1954.

4. The Fourth All India Conference of Moral and Social Hygiene concluded its two days' session on the 21st November at Chandigarh, the new capital of East Punjab and passed resolutions urging the Government of India to take immediate steps to pass into law the uniform Central Bill for the suppression of immoral traffic in women and children. An appeal was made to all Women Organizations in the country to take up seriously the work of eradicating immoral traffic and to chalk out a positive programme of action for this purpose. The Union Government was requested to appoint a Commission on juvenile delinquency to prepare a comprehensive model legislation and a manual of juvenile courts, probation, institutional care, and other services relating to the programme. State Governments were also requested to arrange for separate days for the trial of delinquent and non-delinquent children at the juvenile courts. The Conference demanded the total prohibition of the screening of undesirable films in view of the "baneful influence of cheap and vulgar films on young and impressionable minds."

5. Inaugurating the 12th Bombay Medical Congress at Bombay, Major-General K. R. SAHGAL, Director of Medical Services, Army Headquarters, New Delhi outlined a scheme for the organization of "mutually supporting health centres" with the village as the base and running parallel to the administrative set-up in the country for tackling India's rural health problem. He suggested the formation at the base of: (1) a village health-centre under sanitary cum-medical assistants to deal with sanitation, environmental hygiene, nutrition, treatment of common ailments, maternity and child welfare; and (2) well-organized main rural health centres with facilities for hospitalization and treatment of diseases including medical and surgical emergencies. "In its entirety," said Major-General SAHGAL "the organization will cover the whole State and would constitute the public sector of the country's health

organization in which will be dovetailed what I call the private sector consisting of charitable hospitals and institutions, consultants, specialists and general practitioners." He also suggested the introduction of a "modified voluntary sickness insurance scheme to augment finances for the running of the health scheme suggested by him." We are glad he fully realised that his somewhat novel proposition did not fit in with the ideal of a Welfare State. He would, however, like the people and the government to remember that idealism must yield to realism. He exhorted the medical profession to play an important role as an integral partner in the great nation building adventure that India has embarked upon.

6. Coming nearer home, the Madras State Medical Conference was held at Madhurai on the 20th of November and succeeding days, Dr. Y. P. VASUDEVAN presiding. Dr. R. M. ALAGAPPA CHETTIAR who inaugurated the Conference rightly laid stress on the necessity for more and more highly qualified medical men to man hospitals and colleges efficiently for which purpose they should be paid handsomely so as to enable them to devote themselves wholeheartedly to their work and do research in different branches of medicine. "They had a very high potential here in India" said he "and given the opportunity, I have not the slightest doubt that the medical profession in India would acquire still greater reputation in the world." Dr. VASUDEVAN, the President of the Madras State Branch of the Indian Medical Association said that the Employees' State Insurance Scheme offered the best solution to the problem of rural medical relief. Factors, like poor emoluments, want of suitable residential accommodation and educational facilities for children had so far been hindering the success of rural medical service schemes: the best solution to these difficult and vexing problems was offered by the Employees' State Insurance Scheme which had come to stay. Dr. VASUDEVAN appealed to all doctors to work unitedly for the success of this scheme. He dwelt also at length on several other pressing problems facing the medical profession and medical education at present.

The Conference adopted several important resolutions. Chief among them were: (1) the inclusion, in any scheme of protected water-supply to cities and towns, of the villages *en route*; (2) the inclusion of one representative of the Indian Medical Association on the Madras State Public Health Board; (3) the increase of emoluments to the medical and public health officers in Government services to conform with those of officers in other departments having similar responsibilities; (4) the starting of more regional laboratories at a very early date for food-testing and for diagnostic work; and (5) restrictive legislation to prevent quacks from practising medicine. The Chief Minister of Madras, Sri KAMARAJA NADAR also addressed the Conference and entered a strong plea that doctors and social workers should create a feeling among the

suffering masses that they (the people) were not friendless, and that even the creation of such a feeling among the masses would go a long way in the building-up of the nation on healthy lines.

7. The second South Indian Ophthalmic Conference was held at Madhurai on the 20th of November '54. Dr. E. V. SRINIVASAN of Madras presided. Dr. SRINIVASAN in the course of his presidential address, referred to the ophthalmic practice 40 or 50 years ago as compared to the present day and stressed the need for doctors maintaining good relationship with co-practitioners. He also dwelt at length on the treatment of various diseases and referred to the vast studies made in various cities of America in the diagnosis and treatment of eye diseases. He also urged the importance of maintaining high ethical standards such as he found, during his recent tours, to exist among the practitioners in Europe and America.

8. Inaugurating the 14th annual general meeting of the Indian Chemical Manufacturers' Association in Bombay on the 20th of November 1954 RAJKUMARI AMRIT KAUR, the Union Health Minister urged the Indian Chemical Manufacturers to start the production of basic chemicals and pharmaceuticals in India. "There is no reason" she said "why your Association should not examine the possibility of producing in India glandular products such as Insulin and vegetable products including the active alkaloids. The Government of India hopes to secure the services of an expert to advise on the production of coal-tar products and other chemicals. The private sector should take advantage of such developments and collaborate with the Government so that we may march rapidly towards self-sufficiency." We consider it very necessary that the Chemical Industry should create confidence in medical practitioners about the quality of its products and also keep the consumers well informed about its activities through a well-planned advertisement campaign.

INDIAN RED CROSS

THE Red Cross is a voluntary organisation established as far back as 1864 for rendering humanitarian service. More than 70 countries of the world have adopted its aims and established Red Cross Societies in their respective countries. In our own country, the Indian Red Cross Society was established in 1920 with branches in all the States. The President of the Indian Union is the President of the Indian Red Cross Society. The Society is a member of the League of Red Cross Societies and of the International Committee of the Red Cross at Geneva.

The Society works for the promotion of Health, prevention of disease and mitigation of suffering, irrespective of race, caste or creed, both in peace and in war. In the Madras state the Indian Red Cross Society has been doing very useful work, by giving grants to

Maternity and Child Welfare Centres and to hospitals for anti-venereal disease work. To people afflicted by famine, cyclone or floods, the Society sends donations in the shape of money, clothes and food stuffs. The Red Cross Societies of several foreign countries like the U.S.A., Canada, England, Australia, Russia, Burma, China, and Thailand sent gifts to India during the recent famine and keep sending large gifts of food-grains, milk-powder, multi-vitamin tablets and money. The Indian Red Cross Society renders similar relief to people in other parts of the world. The gifts received are distributed to the poor and undernourished including children, nursing and expectant mothers in distressed areas.

A Junior Red Cross is functioning in educational institutions where children are encouraged to practise ideals of health, service and friendship. Welfare workers teach chronic patients in hospitals diversional therapy like knitting, sewing etc.

The income of the State Branch is derived mainly from grants from Headquarters, interest on investments, subscriptions from members and donations from philanthropic persons. The Red Cross Society is thus an organization which does humanitarian and useful service and therefore, deserves support and encouragement from one and all. We heartily support the appeal made by the Indian Red Cross Society for funds to enable it to do its useful work for the relief of distress. There is no limit to the amount of money that may be paid to the Red Cross. The more the money that becomes available, the greater will be the services rendered.

ŒSTROGENS AND THE BUST

Ethinylœstradiol used systemically or locally is of no value for flat-chestedness due to failure of breast development in adolescence. Usually such hypomastia unaccompanied by other evidence of hypogonadism is due to target-organ failure—i.e., the absence of responsive mammary tissue or to inadequate response of such tissue. Insufficiency of adipose tissue in the breast is commonly found in these cases. Only when there is evidence of œstrogen deficiency is it at all likely that œstrogen treatment will have much effect. There is no direct evidence that the continuous application of stilbœstrol cream leads to malignant changes in the breasts, but the results of animal experiments leave no doubt that such changes are a possibility. The continued use of these creams is, highly imprudent, as well as unlikely to achieve any useful result.—(B.M.J., 25.9.54, p. 765).

The ANTISEPTIC will be published on the 8th instead of the 15th of every month, commencing from the issue of January 1955—Ed. "Antiseptic."

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SURGERY

Clinical surgery of the first 100 verified brain tumours.—(*Ind. J. Surg.*, 1953, 15: 173.—*Internat. Abst. Surg.*, June 1954).

Dr. Jacob Chandy, Neurosurgeon of the Christian Medical College Hospital in Vellore, reports briefly on the first 100 verified intracranial tumours which have been operated upon. He emphasizes that a large number of the patients were far advanced in their disease when they sought medical aid and that approximately 30 per cent of them were bed-ridden and in a semicomatose condition. The majority of them had evidence of increased intracranial tension with headaches, vomiting, and visual disturbances. Only a few patients displayed occasional epileptic convulsions. The author experienced great difficulty in localizing the tumours, even in advanced stages, on clinical examination alone. Ventriculography was the main diagnostic aid for localization and was found to be most reliable. The majority of patients were operated upon under general anaesthesia with intratracheal intubation.

This group included 12 astrocytomas and 11 glioblastomas, most of which were very extensive, often involving both hemispheres. The patients with glioblastomas were given deep X-ray therapy following operation. No attempt at complete removal of the tumours was made. There were 9 medulloblastomas, 2 of these in adults aged 17 and 28. The mortality rate for these three types of tumour was 45 per cent.

There was only 1 case of ependymoma and 2 cases of oligodendroglioma. Meningiomas formed 16 per cent of the tumours, and only 6 of the patients had definite evidence of bony changes in the skull; most of them had only minimal neurological dysfunctions, even though the tumours had attained considerable size. Neurofibromas of the acoustic nerves were found in 7 cases. One patient had bilateral tumours of the eighth nerve, associated with neuro-

fibroma of the sixth nerve and a basilar meningioma. In only 3 cases was there evidence in the roentgenogram of erosion of the acoustic meatus. The mortality rate in the meningioma-neurofibroma group was 23 per cent.

Pituitary tumours were present in 18 per cent of the patients; 4 of them were acidophilic, and one was basophilic in nature. Malignant changes were found in chromophobic adenoma. Headache was the most prominent feature in these cases, sometimes associated with ophthalmoplegias, bitemporal hemianopsias, and even total blindness. Enlarged pituitary fossa in the roentgen films was not found to the evidence of pituitary tumour. The operative risk was greatest in the eosinophilic adenomas. The mortality rate among patients with this type of tumour was 15 per cent.

A group of tumours of the third ventricle consisted of 9 craniopharyngiomas, 1 colloid cyst, and 3 astrocytomas. The operative mortality was high.

Cardiospasm.—(*Chirurg.*, 1953, 24: 529).

Dr. Saegesser differentiates between cardiospasm, as part of a diffuse oesophagospasm, and an isolated spasm of the cardia. This may be either idiopathic or symptomatic.

Cardiospasm is termed idiopathic when difficulties in deglutition are the only symptom and pains and heartburn are absent. The pathogenesis is unknown. A small hiatus hernia may play a causative role in occasional cases. A great number of cases are of psychosomatic origin and the patients recover when the underlying conflict is solved. Seventy-five per cent of patients with idiopathic cardiospasm are cured by conservative methods. These include: (1) psychotherapy; (2) medication with perlingual nitroglycerin or various drugs of the atropine group; (3) bilateral anaesthesia of the stellate ganglia every 2 or 3 days until 6 treatments have been given; (4) trans-

oesophageal mechanical dilatation after pre-medication with nitroglycerin.

If conservative methods fail, surgery is indicated. The operation of choice is cardiomyotomy, with use of the method of Heller. The cardia is approached either by the transthoracic or the abdominal route. After isolation of the lower oesophagus and the cardia, two longitudinal incisions of from 8 to 10 cm. in length are made through the muscular wall of the lower oesophagus and the cardia at the anterior and posterior aspect, leaving the mucosa in tact. In most cases of idiopathic cardiospasm, no pathological changes are found at operation: if, however, scar tissue is found in the area of the cardia, a fusiform flap of the wall should be excised.

In the symptomatic form of cardiospasm, the patient complains of severe pain in addition to difficulty in swallowing. The pain radiates to the back and to the left arm. This clinical picture always suggests peptic ulcer disease, of which the cardiospasm is only a symptom. Thorough investigation sometimes discloses the presence of small gastric or duodenal ulcer which had caused few or no pains. In other cases, only a peptic oesophagitis is present. If conservative therapy fails to cure the pains and dysphagia, surgery is indicated. For these cases, the author advocates resection of two thirds of the stomach by a Billroth I or II procedure in combination with the cardiomyotomy described.

A third form of cardiospasm can be caused by stenosis of the cardia after an ulcer in the cardiac region has healed and left constricting scar tissue. This stenosis is treated by mechanical dilatation; if unsuccessful, the Heller operation or an oesophago-gastrostomy is performed.—(*Internat. Abst. Surg.*, June 1954).

Rest and movement.—*J. Bone Surg.*, 1953, 35-B: 521).

Dr. Perkins enters the controversy of 'rest versus movement' as it applies in a number of varying conditions including infection, chronic arthritis, soft part injuries, and fractures. In discussing the treatment of tuberculous arthritis,

the author deplures the shortcomings of our present treatment but does not say that he advises full activity in the tuberculous patient. In osteoarthritis, rest does seem to benefit the joint. For rheumatoid arthritis the combination of splinting and active motion is outlined.

Soft tissue injuries are divided into muscle tears, sprains, and strains of joints. All are treated by active motion within certain limits. The author believes that sprains should be treated by early motion and strains by recumbency. He favours immediate active motion following dislocation of the humeral head as well. He states that if movements are unrestricted and encouraged from the start, a patient with an uncomplicated dislocation of the shoulder is using his arm naturally again within a week. Presumably this means it is possible to use the arm at near 180 degrees of abduction as well as in other positions.

The author cites numerous examples of good function following fractures in which no immobilization was used. He thinks that all structures in the body when divided tend to join and that when the repair tissue is under compression the uniting bond becomes bony. In fractures of the shaft, there are muscles attached to both fragments capable of compressing the bone ends against one another so that union will be by bone even in the absence of splintage. The author thinks that splintage is only essential for bony union when there are no muscles attached to the fragments, which are capable of impacting the bone ends. This is the missing factor in the fractures that are slow to unite, such as fractures of the carpal scaphoid, transcervical fractures of the neck of the femur, and fractures of the medial malleolus. Most authorities believe that there are factors other than this one which are responsible for slow union. It would seem that the same muscle-compressing forces act on an inter-trochanteric fracture of the femur as well as on a transcervical fracture. The one unites almost in spite of treatment, whereas the other unites slowly, if at all.

The author plainly indicates that he is interested in restoring function

rather than anatomical form. Other surgeons in the same field would seem to have this goal in mind also, even though they may use methods somewhat different from those advocated here.—(*Int. Abst. Surg.*, June 1954).

Treatment of keloids. (*New England J. Med.*, 250: 138, 1954).

As a result of their experience with the use of ACTH and cortisone in the treatment of keloids, Ronchese and Kern state that these agents are effective

only when given systemically after surgical removal of the keloid. Ineffective techniques included (1) local application of cortisone ointment, with or without surgical removal of the keloid; and (2) cortisone or ACTH by mouth or by injection, without concomitant surgery. In conclusion, the authors stated the opinion "that the treatment of choice is still surgery combined with radiation and that hormonal therapy, as described, should not be employed except in cases that have failed to respond to this method of treatment."

OBSTETRICS AND GYNAECOLOGY

Caesarean section. An evaluation of practice in the New York lying-in-hospital.—(*Obst. & Gyn.*, 1953, 2: 593).

The improvements in maternal care and the pre-operative and post-operative management of obstetric patients in recent years may well be resulting in a change of indications for caesarean section with emphasis more upon infant survival. With this in mind, the caesarean sections at the New York Lying-in-Hospital, were reviewed from 1932 to 1952. There were 65,880 deliveries with caesarean section in 3.7 per cent. The maternal mortality in this group was 0.7 per cent. In private patients the rate of caesarean section was 6 per cent, with a maternal mortality of 0.6 per cent. For the purpose of this study the last 3 years (1950 to 1952) were contrasted with the previous years in respect of incidence, changing indications, anaesthesia employed, operative type, and the maternal and infant results.

There appeared to be definite alterations in the indication for caesarean section. Among the group that decreased or disappeared from the list of indications in the 1950 to 1952 group were cephalopelvic disproportion and contracted pelvis, heart disease, tuberculosis, and prolonged labour. It was thought that the increased use of pitocin intravenously plus the control of intrauterine infection with antibiotics and the earlier institution of effective

means of treatment accounted for the absence of prolonged labour in the 1950 to 1952 group. The reduction in caesarean sections for cephalopelvic disproportion was thought to be due to the more liberal use of the trial or test of labour with the aid of antibiotics as an additional safety measure. There was a rise in the incidence of caesarean section for previous caesarean section from 19.7 to 26.5 per cent in the entire group. There was also an increased percentage in elderly primigravida and diabetics. There was no change in the incidence of placenta praevia and premature separation of the placenta as an indication for caesarean section.

The preferred anaesthesia has been local infiltration with procaine accompanied by inhalation anaesthesia or pentothal. The operative procedure most commonly used has been the low flap operation with a transverse elliptical incision. About 20 per cent of the procedures have been of the classical type; very few extraperitoneal and radical caesarean sections have been performed.

Thirty-eight per cent of the deaths were from peritonitis during the period from 1935 to 1939. There were no deaths from infection after this period. Hemorrhage was the next most important factor in the maternal deaths, occurring in 19 per cent of the patients. The remaining factors were divided between embolus, cancer, necrosis of the renal cortices, and heart disease.

The gross infant death rate has decreased despite the recent inclusion of a greater proportion of premature babies. The 1950 to 1952 infant death rate was 5.3 per cent as contrasted to the 6.3, 8.3 and 9.5 per cent rates for the 5 year periods going back to 1944, 1939 and 1933. When the infant death rates were considered in terms of the indications for caesarean section, it was noted that there was a definite reduction in infant deaths associated with premature separation of the placenta. Similarly, placenta praevia showed a decreased percentage of corrected infant deaths. Elderly primigravidas and previous caesarean sections had very low

infant mortalities. Fetal deaths rose percentagewise in relation to fetal distress from about 8.1 to 9.8 per cent for the 1950 to 1952 period. Deaths in connection with the diagnosis of diabetes dropped from 15.4 per cent prior to 1949 to 12.5 per cent for 1950 to 1952. If infant survival was compared on the basis of prematurity of the infant, it was noted that the child delivered by caesarean section had a slightly better chance of survival when its weight was below 2,500 gm. For term babies above 2,500 gm. in weight vaginal delivery offered a slightly better chance of survival. (*Int. Abst. Surg.*, July 1954).

DENTAL DISEASE

Aetiology of enamel caries. (A new concept). (Den Norske Tandlægeforenings Tidende.—*Ind. Dent. Rev.*, 23: 5-6, 1954).

The acidogenic theory of enamel caries is based chiefly on two observations: (1) the enamel is dissolved by the action of an acid of sufficient strength; (2) bacteria of the oral cavity, *Lactobacillus acidophilus* and many other species, are capable of fermenting carbohydrates containing food debris which collects on certain areas of the teeth, to produce an acid which is strong enough to decalcify the enamel.

Based on present knowledge, it is difficult to believe that enamel caries are solely of acidogenic origin.

At the turn of the century it was a common opinion that the enamel consists of mineral salts only. This view fits well into the acidogenic theory of enamel caries. Recent researches have shown that enamel also contains a certain amount of organic matter. The protein content is inconsiderable when expressed in weight per cent. The organic matter, being much lighter than the mineral salts, makes up a larger part of the enamel in volume per cent. As shown by the electron microscope, the organic matter is distributed throughout the whole enamel layer but is densest where it is most stainable.

In histological examination of decalcified enamel, the first signs of caries are

detectable before any changes occur at the enamel surface. Bacteria are often found in the organic tracts of such enamel. Chemical investigations show that the organic matrix contains proteins which may serve as nutriment for the bacteria.

Enamel caries starting in the organic matrix may be the result of bacterial metabolism breaking down the protein and, in so doing producing an acid which demineralizes the inorganic part of the tissues. There are also investigators who maintain that the proteolysis of the organic matrix will itself destroy the enamel.

The dental film with its contents of bacteria and nutriment is just as significant for a proteolytic theory as for an acidogenic theory of enamel caries. Some persons are practically immune to caries. In susceptible individuals, the susceptibility varies during different periods of life and certain groups of teeth are attacked more infrequently than others. Cases of unilateral caries are found where only one of two approximate tooth surfaces is attacked. The teeth apparently not only have a resistance to caries which varies from person to person but also during the life time and in different groups of teeth.

Primitive people who masticate coarse food and grind down their teeth rarely show dental caries regardless of the chemical nature of their food. It is possible that the vigorous use of the

teeth has in itself a protective effect against caries. This may act through stimulation of the circulation around

and in the teeth or through the self-cleaning effect which the mastication of the coarse food has on tooth surfaces.

MEDICINE AND THERAPEUTICS

Emotional stress before apoplexy.—(*Ann. Int. Med.*, 40: 49, 1954).

Ecker has presented twenty cases illustrating the association between cerebral apoplexy and preceding emotional stress. There were thirteen cases in which psychiatric studies revealed that the patients had great difficulty in dealing with aggressive and hostile feelings. There were fifteen cases in which a cerebral stroke apparently was precipitated by emotional stress. In eight cases both factors were found.

The author proposed that emotional stress provokes cerebral arterial spasm (a change that he has demonstrated in arteriograms). This spasm causes cerebral ischemia that in turn contributes to pathologic changes in the brain and its blood supply. He did not imply that emotional stress is the only factor implicated in apoplexy, but he expressed the belief that it is an important predisposing factor. He stated, "If this is true, cerebral vascular 'accidents' would not be entirely accidental."

Chair treatment of myocardial infarction.—(*Ann. Int. Med.*, 40: 49, 1954; *G. P.*, June '54).

Several years ago, Levine began advocating the "chair treatment" of myocardial infarction. Clinical experience has shown that patients with this disease often are more comfortable and do better in a chair than in bed. It is known that breathing is easier (due to reduction of pulmonary blood volume), and bowel and bladder complications are less frequent. Recently, Coe measured cardiac work in the recumbent and chair positions. His data indicated that the work of the heart is significantly less when the patient is sitting. The author warned that there are nevertheless some contraindications to the "chair treatment" of myocardial infarction—symptoms of cerebral ischemia when sitting, and shock. He warned also that one must be sure that the armchair does not unduly compress the

popliteal spaces and thereby predispose to venous thrombosis.

Prevention by testosterone of the adrenal atrophy produced by cortisone treatment.—(Turiaf, J., Zizine, L., and Jean, Y., *Presse Med.*, 61: 825, 1953. *Jour. Internat. Coll. Surg.*, May '54).

Cortisone administered to the rat produces atrophy of the adrenal cortex which is histologically identical to that following hypophysectomy. Testosterone, if administered simultaneously with cortisone, prevents the adrenocortical atrophy in the rat without, however, reducing the cholesterol reserve in the adrenal and without removing the "thymo-atrophiant" action and the anti-inflammatory properties of cortisone.

Cortico-atrophy from cortisone occurs in human therapy. A prompt breaking down of Thorn's test with ACTH and an anatomo-histological thinning of the adrenals take place.

Fluctuations in the adrenal functions were assessed in patients using the Thorn's test (4-hour test by intramuscular injection of 25 mg. of ACTH). The following observations were made: (1) Testosterone controlled the "cortico-atrophiant" potency in half of tested cases; (2) the protective effect of testosterone in doses of 25 mg. per week, caused no genital disturbance and evidence of hyperandrogeny; (3) histologic data on patients who died at the end of long courses of cortisone treatment were comparable with data from animal experimentation. The protective effect of testosterone on the adrenal cortices of animals or patients treated with cortisone is not affected by the interposition of the pituitary gland, but probably by direct action on the adrenal gland according to a process not yet defined; and (4) testosterone given with cortisone appears safe whenever the cortico-therapy cannot be discontinued for a sufficient period to permit spontaneous repair of the adrenal.

BOOK REVIEWS

BCG and Vole Vaccination—By K. NEVILLE IRVINE, M.A., D.M., B.C.H., M.R.C.S., L.R.C.P., pp. 96, 1954. Published by National Association for the Prevention of Tuberculosis, Tavistock House North, Tavistock Square, London W.C.1. England. Price 12/6.

This is a little handbook on BCG and Vole vaccination issued under the auspices of the National Association for the Prevention of Tuberculosis in Eng-

land. It gives a concise summary of what is known about BCG and Vole vaccination together with practical instructions for organization, tuberculin test and vaccination. The ordinary illustrations as well as the coloured plates are very instructive. We are sure that the book will stimulate interest in those who read it and thus help in the prevention of tuberculosis.

U.K.R.

BOOKS RECEIVED

The Surgical Appendix—By M. V. BHAIKAR, pp. 134. Published by Dr. J. C. Patelon behalf of the Editorial Board of the Indian Journal of Medical Sciences, Back Bay View, New Queen's Road, Bombay-4. Agents: M/s. Kothari Book Depot, Bombay-12. Price Rs. 5/- inland, Rs. 6/4/- overseas.

The Journal of Family Welfare—Vol. 1, No. 1, Whiteaway Building, Bombay-1.

Techniques in Clinical Chemistry—By Frederick N. Bullock, Pp. 171. Published by M/s. John Wright and Sons Ltd., Medical Publishers, 42/44, Triangle West, Bristol 8. [Price 10s 6d.]

NEWS AND NOTES

The Fifth All-India Leprosy Workers' Conference and The Second Biennial Meeting of The Indian Association of Leprologists

The Fifth All-India Leprosy Workers' Conference and the meetings of the Indian Association of Leprologists will be held at The United Club buildings in Jamshedpur (Behar) from the 3rd to the 5th February, 1955.

The first two days (3rd and 4th February) will be devoted to the meetings of the Indian Association of Leprologists, which will be open to non-medical workers. The general Conference

will be held on the succeeding two days (5th and 6th February).

Sri G. V. Mavalankar, Chairman of the Gandhi Memorial Leprosy Foundation and Speaker of the Loka Sabha, New Delhi, will preside over the General Sessions of the Conference on the 5th and 6th February, 1955.

The Conference is open to all medical, para-medical, and social workers, directly interested in leprosy or connected with any organization engaged in or furthering any anti-leprosy work.

For further particulars. Apply to Secretary, I.A.L., School of Trop. Med., Calcutta-12.

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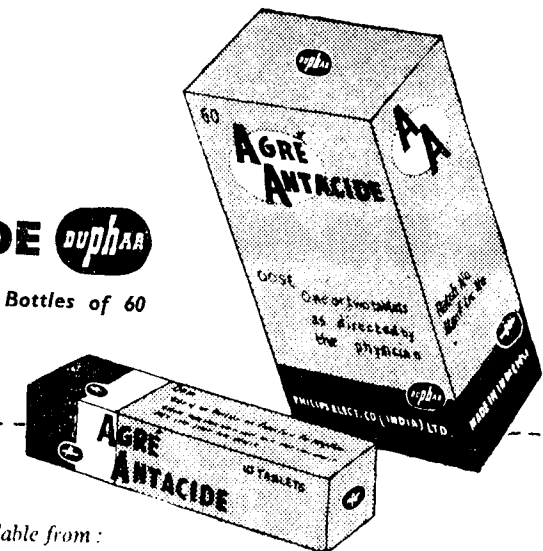
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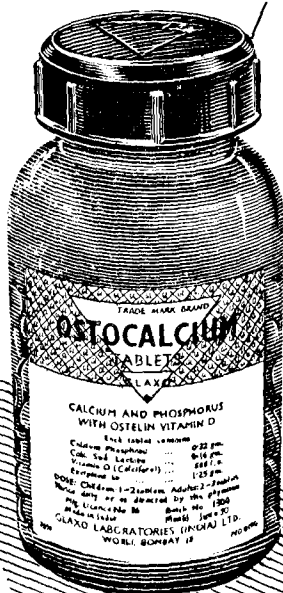
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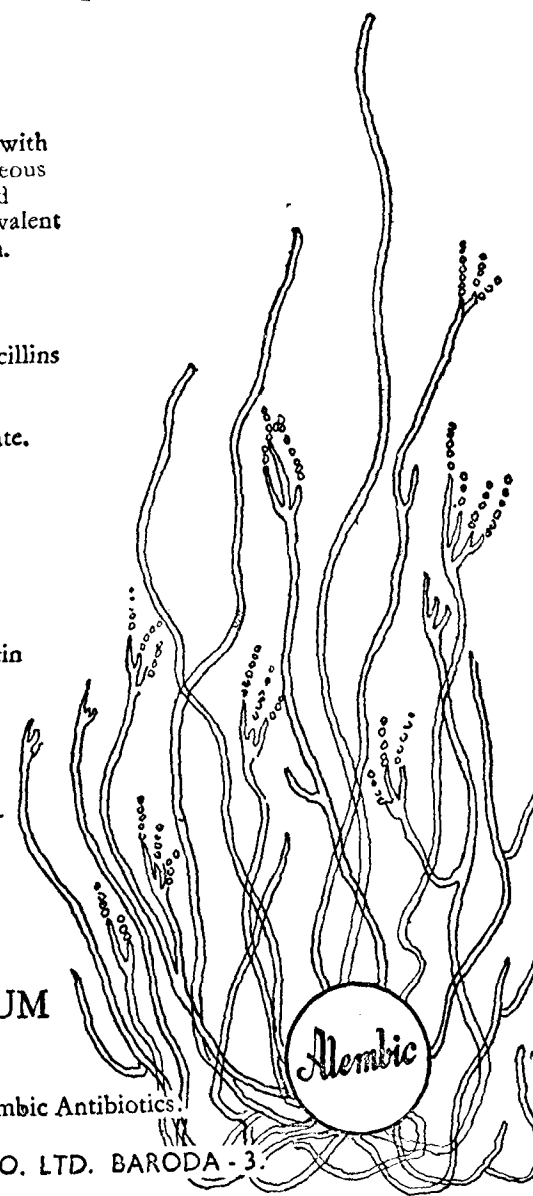
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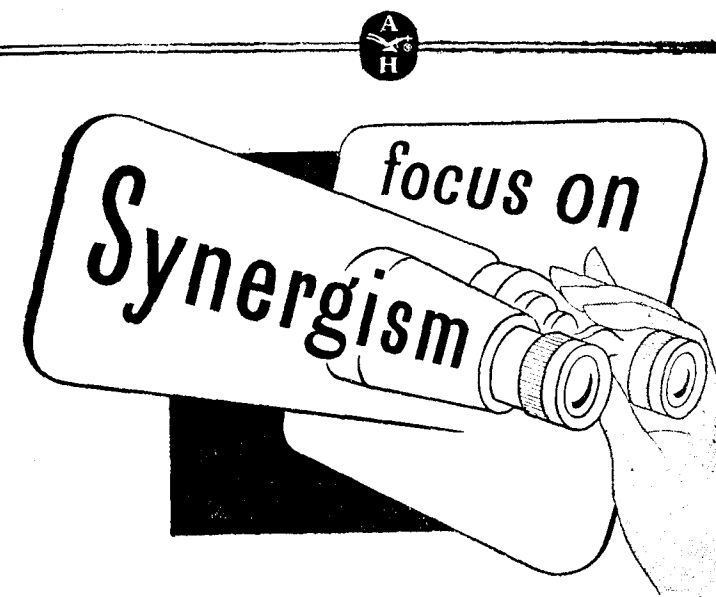
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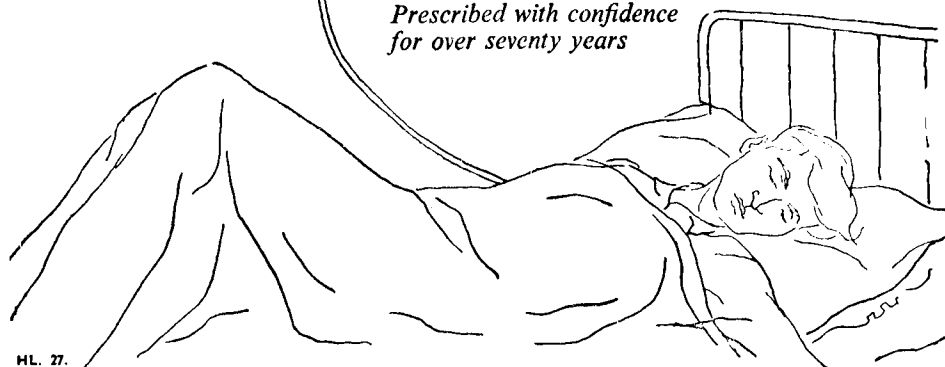
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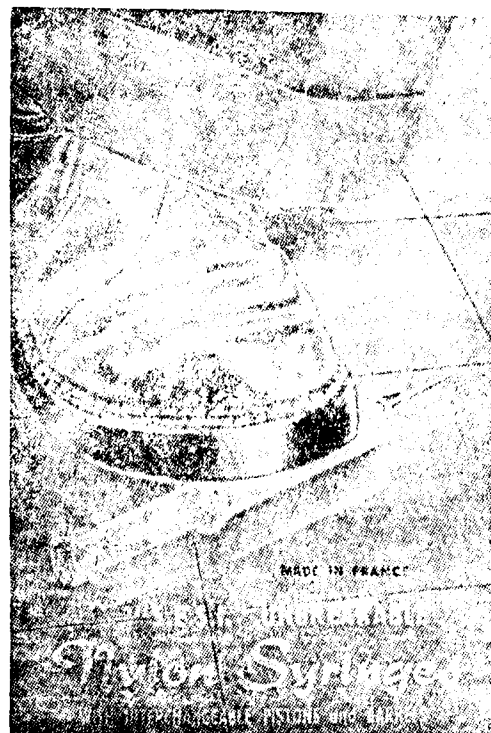
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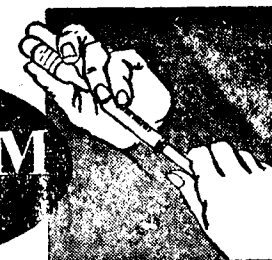
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	Folic Acid	... 1.5 mg.	Vitamin A	... 5000 I.U.
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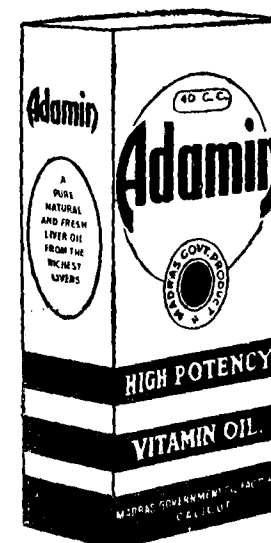
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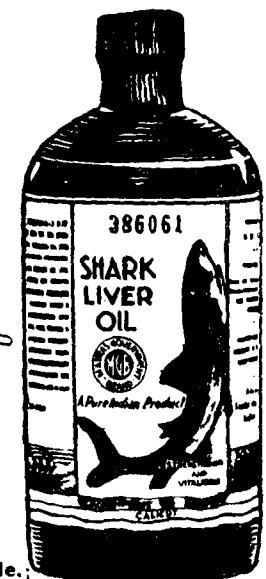


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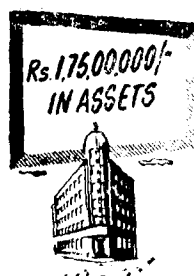
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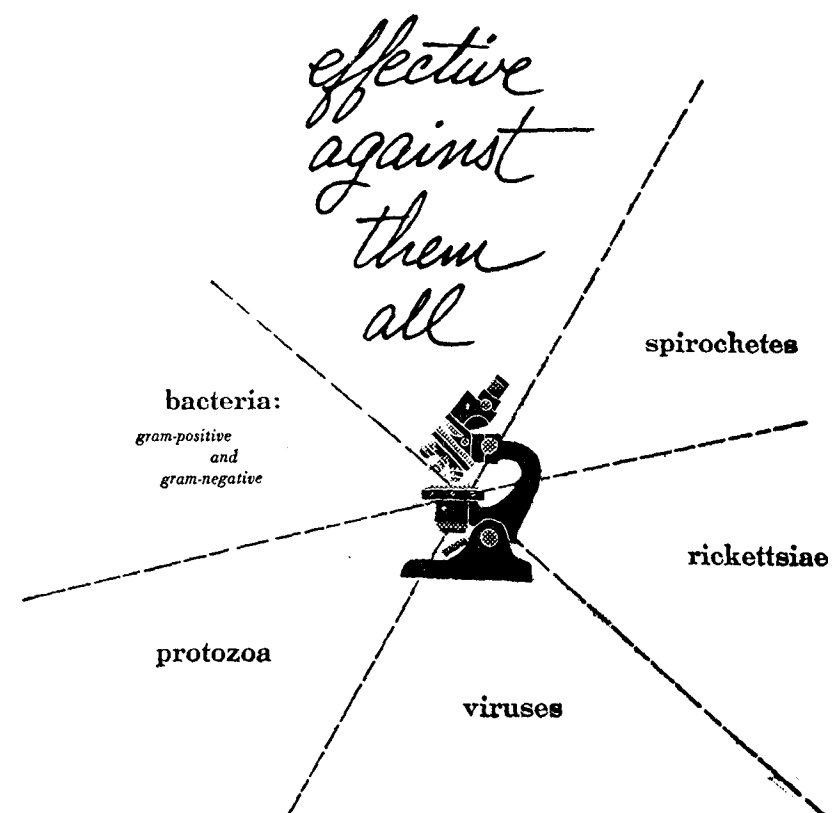
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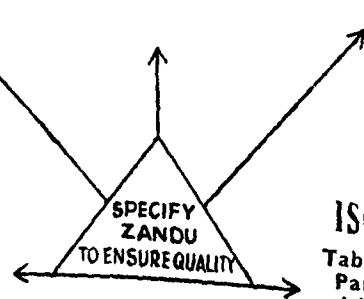
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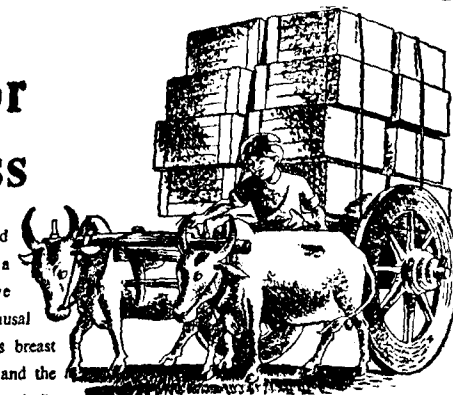
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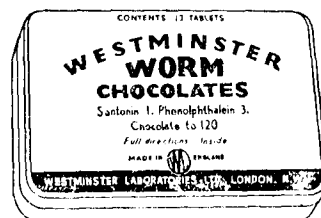
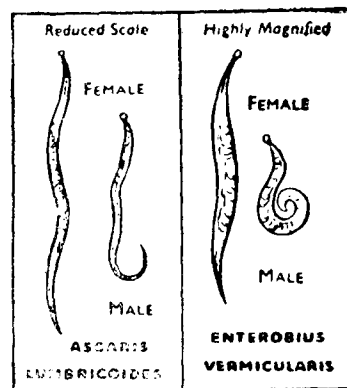
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References:-

1. "The Role of Ayurvedic Medicines in the Treatment of Diabetes Mellitus"—Indian Journal of Medical Sciences-Vol. 6, No. 2, Feb. 1952, pp-117-130.
2. "Diabetes Mellitus for the General Practitioner"—Medical Digest, July '53

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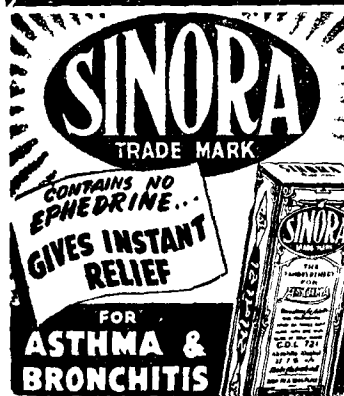
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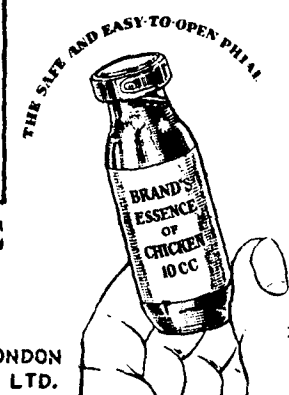
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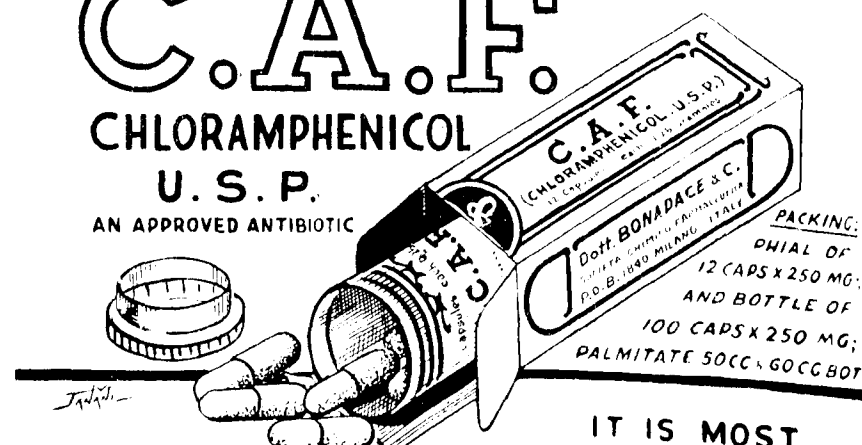
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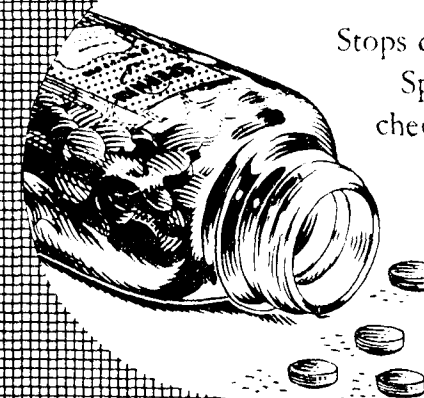
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HEALTH

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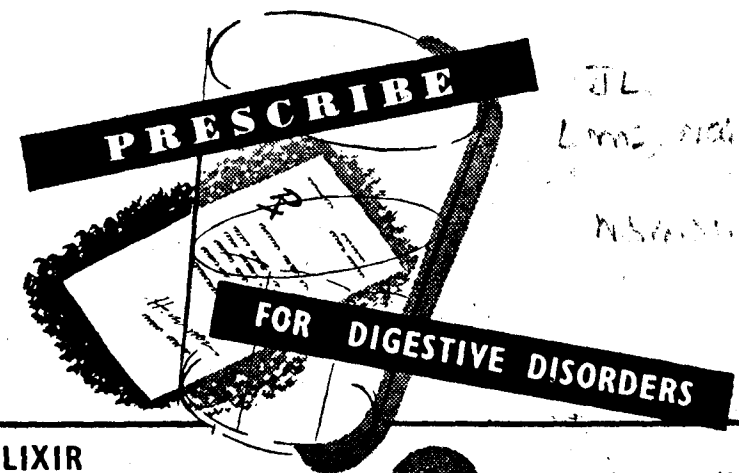
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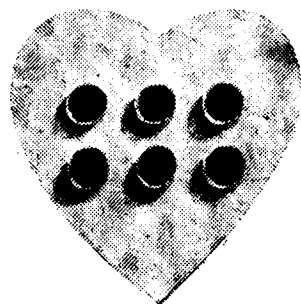
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