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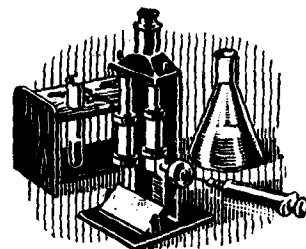
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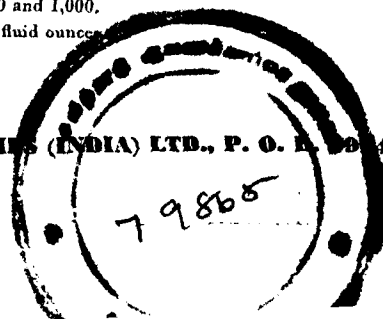
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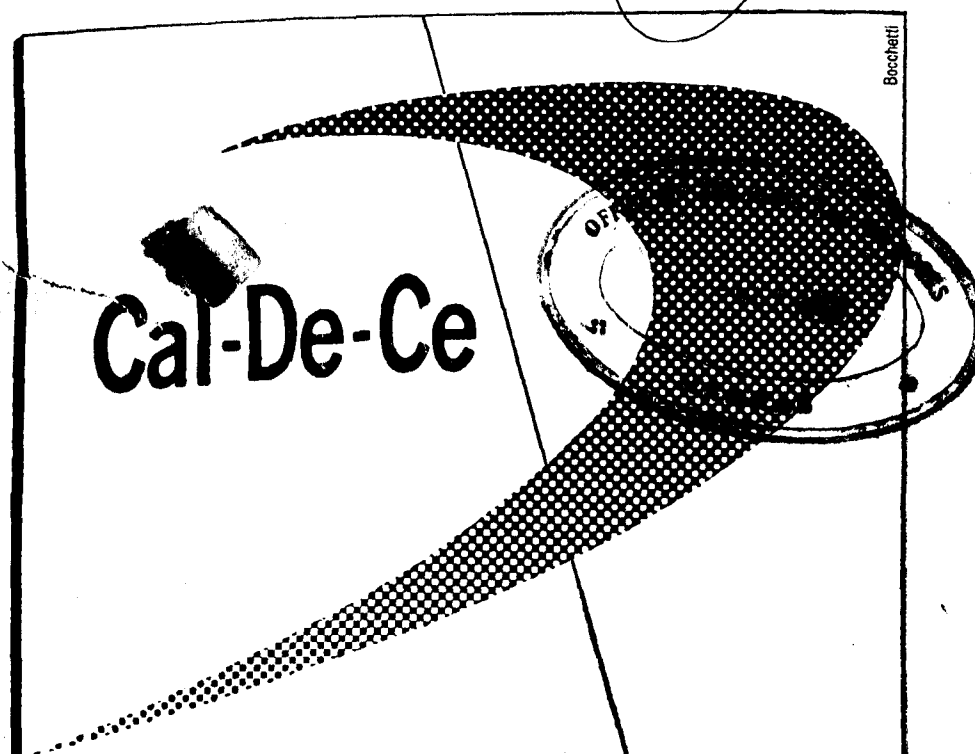
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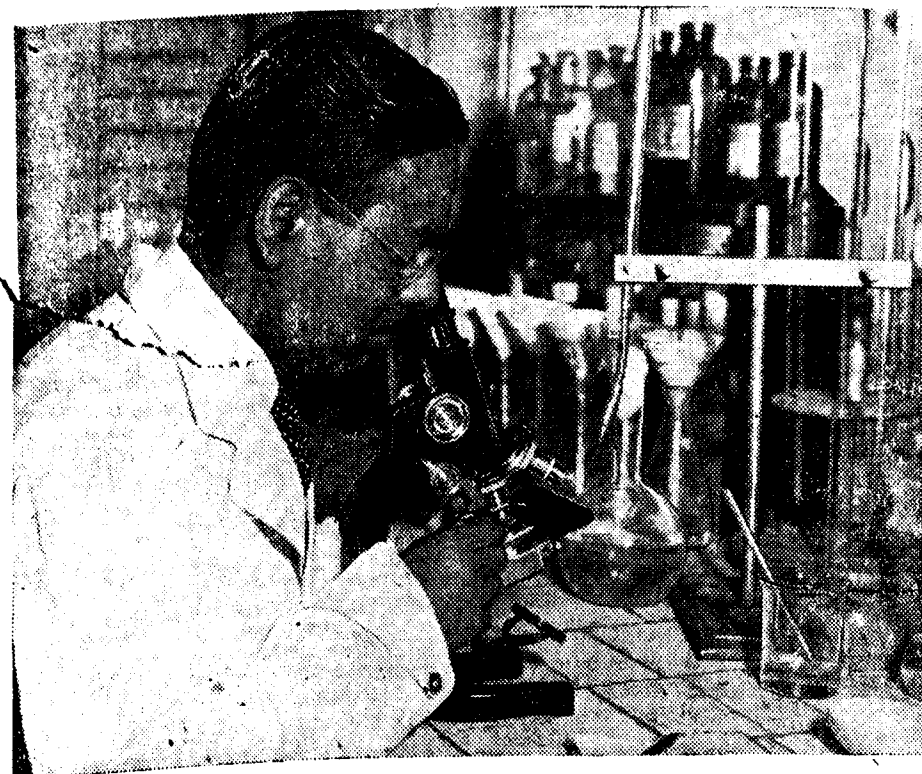
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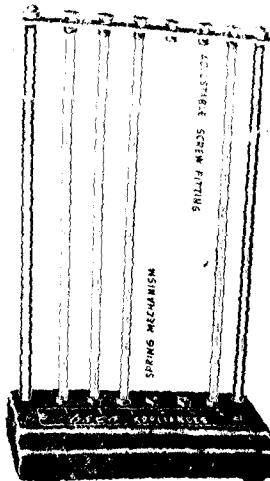
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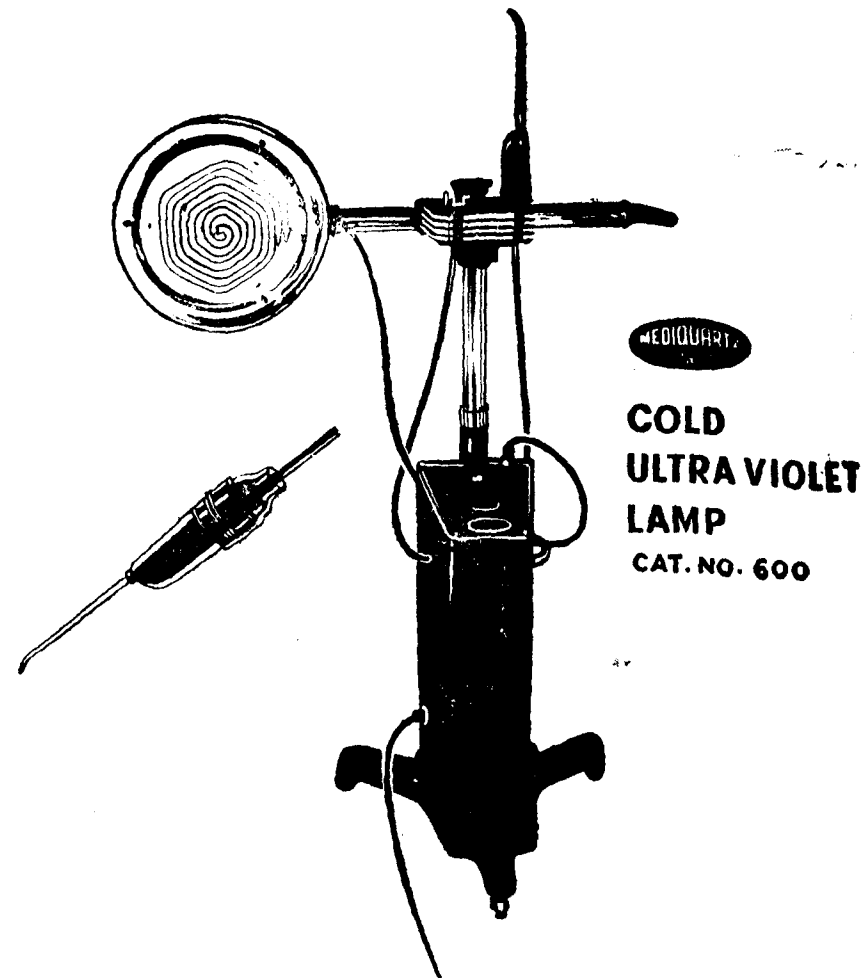
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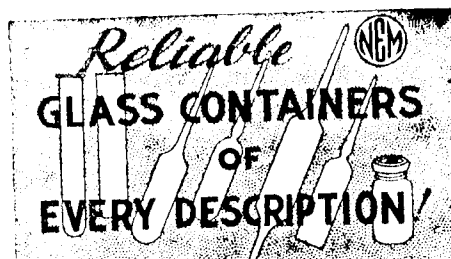
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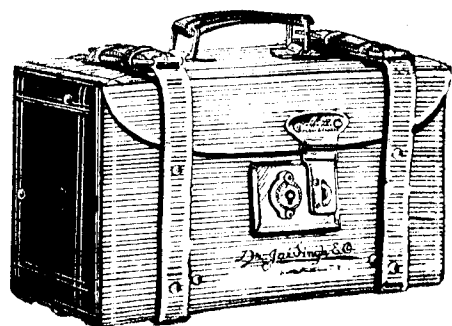
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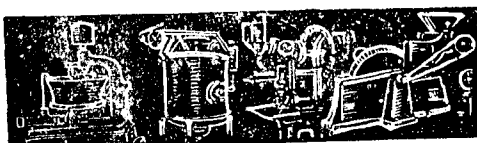
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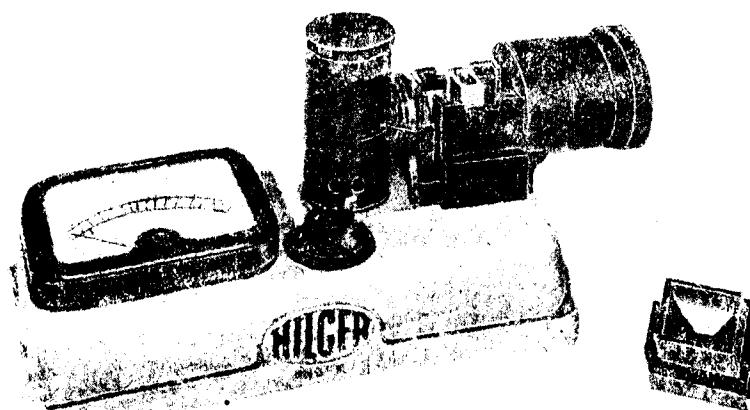
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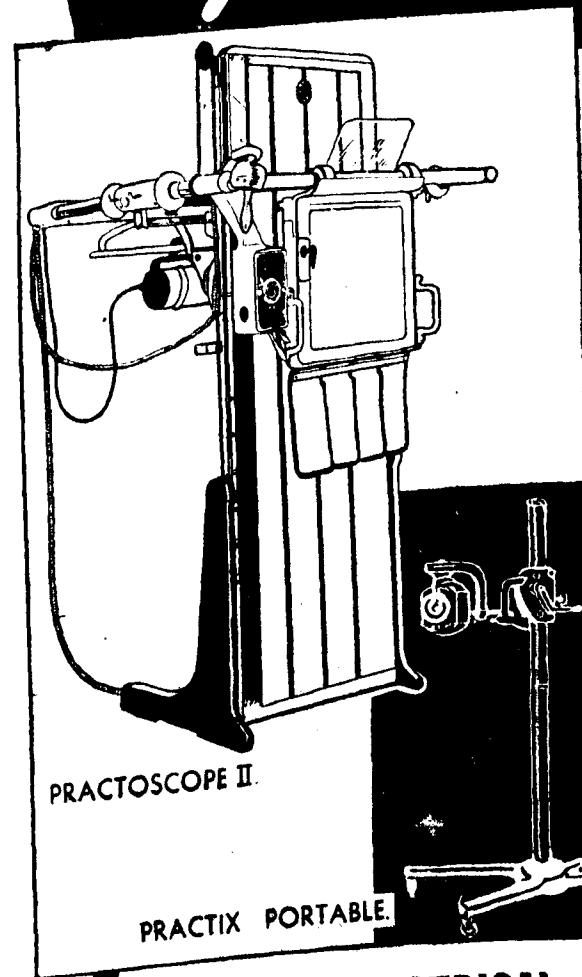
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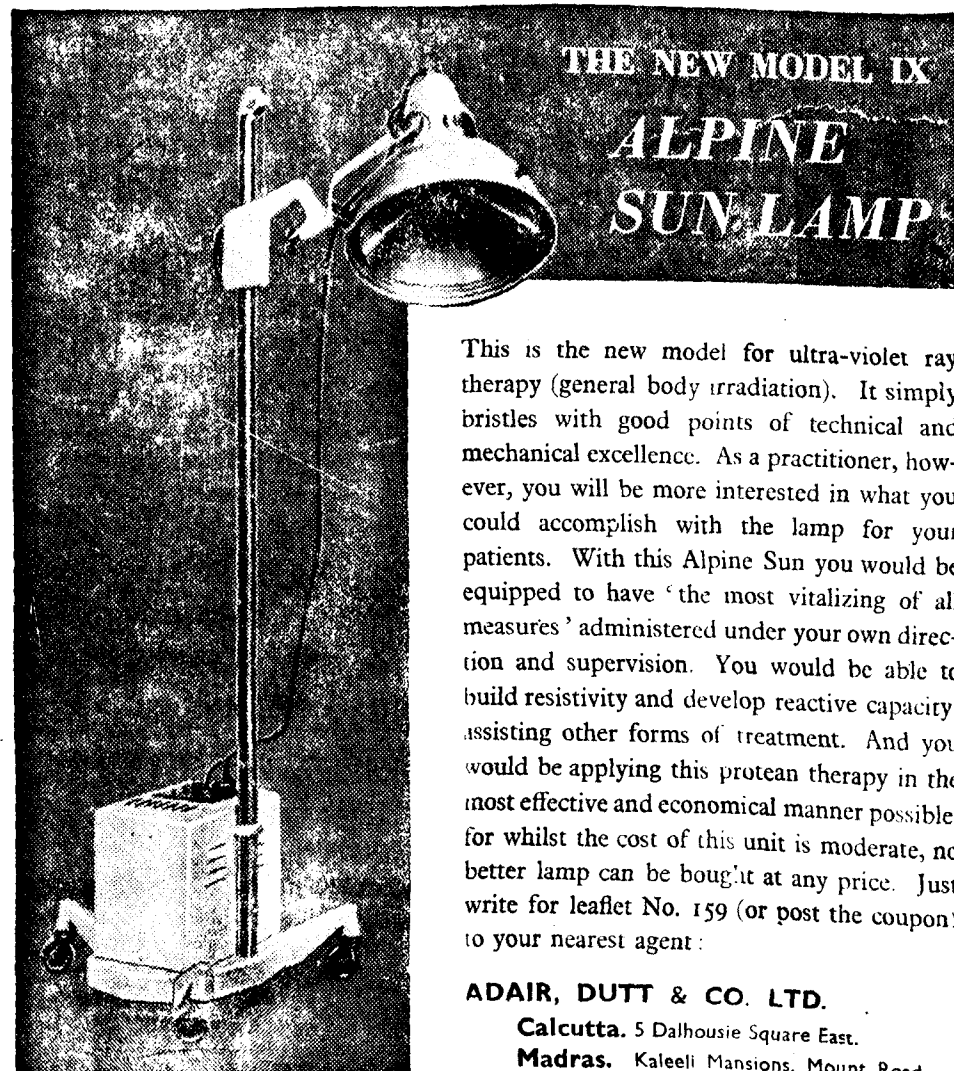
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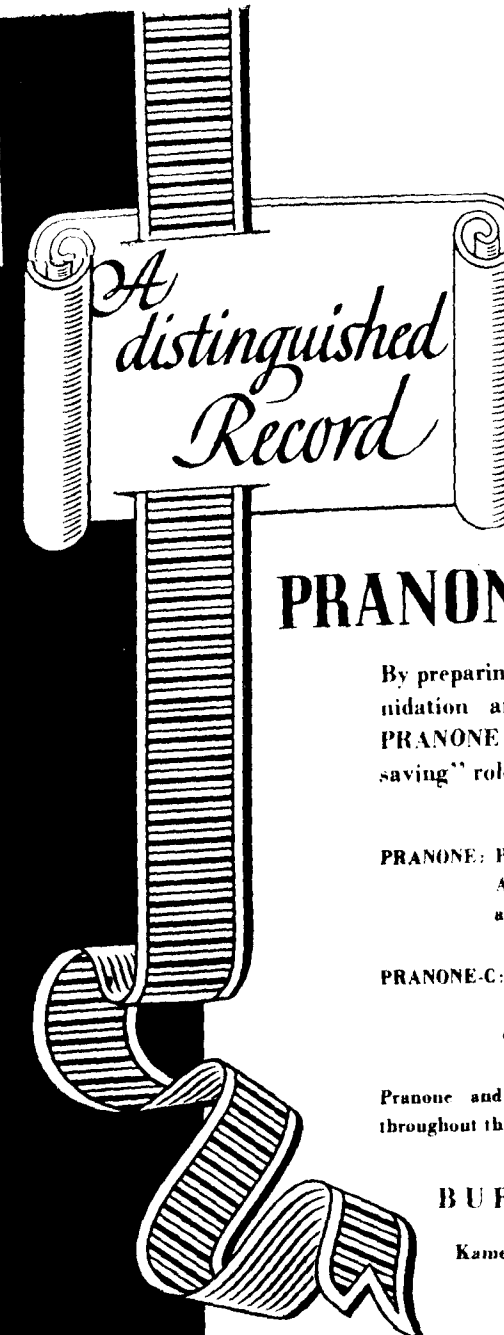


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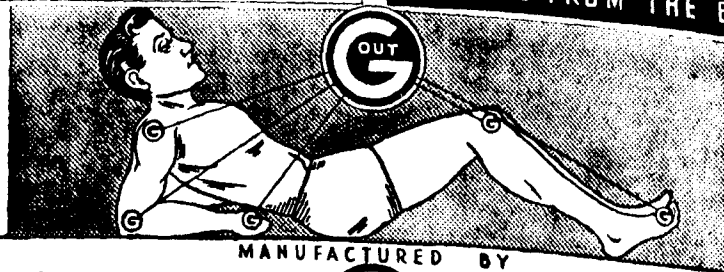
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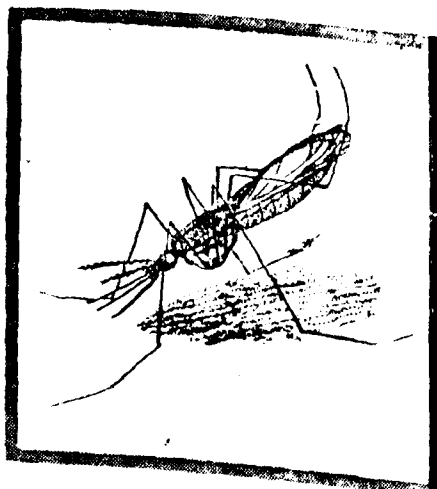
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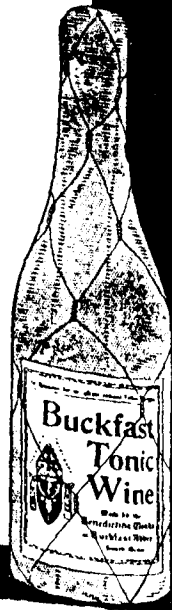
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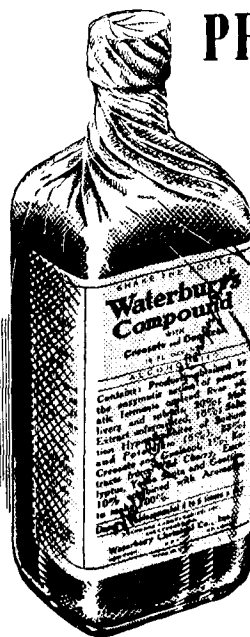


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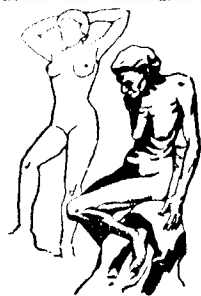
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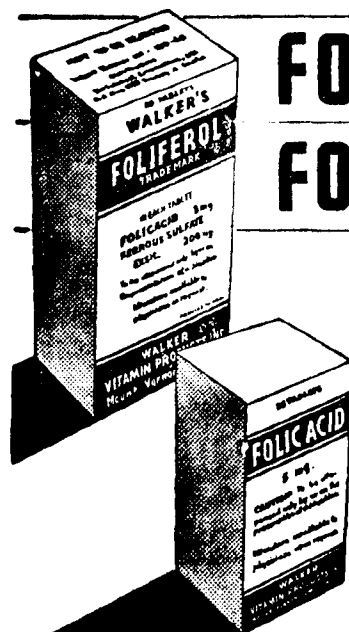
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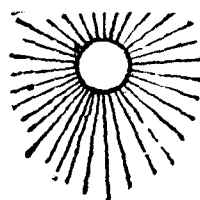
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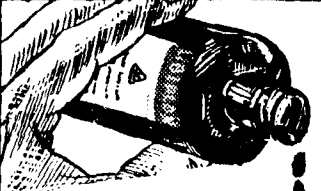
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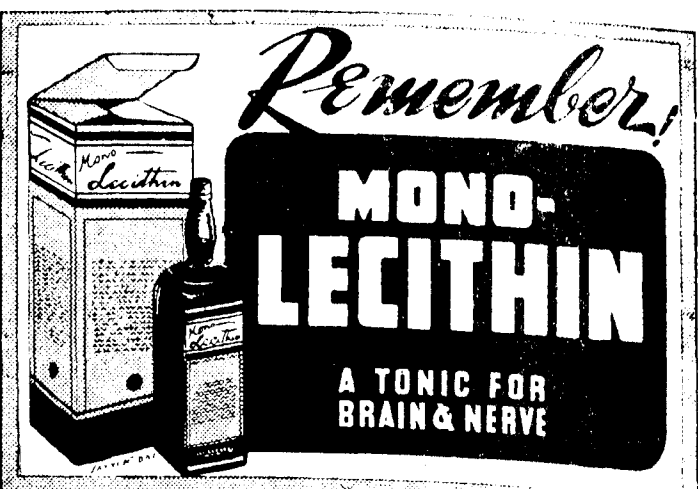
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Original Articles

Laboratory Diagnosis in Clinical Medicine*

PROF. R. K. GOYAL, D.Sc. (Paris), Ph. D. (Edin.),
M.R.C.P. (Lond.), M.B.C.S. (Eng.), M.B., B.S. (Punjab), F.R.S. (Edin.),
Professor of Pathology and Bacteriology,
Gajra-Raja Medical College, Gwalior.

PART I.

Introduction

IN these days of specialization it very often happens that a highly efficient clinician possesses at best an indifferent knowledge of clinical pathology and bacteriology and is not always in a position to order the exact laboratory examination required. A number of clinicians are also unable to interpret correctly the laboratory report and expect the pathologist not only to detail the laboratory findings but also to give the diagnosis. The pathologist should never attempt to diagnose diseases on the basis of laboratory investigations as he generally does not possess all the relevant data connected with the patient, and it is logically the duty of the clinician to diagnose the disease with the assistance of the pathologist and the radiologist, if necessary.

It is desirable for a country like India to have a large number of small laboratories carrying out routine investigations from the point of view of clinical pathology and bacteriology as well as chemical pathology. A pamphlet giving details of such routine investigations is likely to be of great value to laboratory workers as well as to medical students.

This paper is divided into two parts, Part I deals with the interpretation of laboratory reports and Part II gives the details of the techniques employed in laboratory investigations.

* Specially contributed to THE ANTISEPTIC.

Examination of Urine

Significance of albuminuria :

1. Orthostatic or functional albuminuria intermittent in character, not accompanied by the presence of blood-cells, pus, casts, or oedema, or any other sign of disease.

2. Bright's disease, acute febrile infections, passive congestion due to cardiac disease and as a sequel to many acute diseases.

3. Injury by chemical poisons such as mercury and lead.

4. Albuminuria in pregnancy :

(a) Simple albuminuria without manifestation of disease, observed in 3-5 per cent of all cases of pregnancy.

(b) Pre-eclamptic albuminuria—excess of leucocytes, red blood-cells and probably casts often present, oedema, raised blood pressure, and retinitis are often found.

5. Accidental and extra-renal contamination with discharge from the prepuce or vagina, inflammation of urethra, prostate or bladder, prostatic massage and catheterization of the ureters.

6. Bence Jones albuminuria :—This occurs in cases of tumours of the bone-marrow, myeloma, more rarely secondary carcinoma (precipitated by heat, redissolves on boiling).

Sugar in urine.—Causes :

1. Normal glycosuria (negligible).

2. Alimentary glycosuria in cases of excessive ingestion of glucose, when blood sugar rises above 180 mgm. per 100 ccm. (140-200 mgm.—renal threshold).

3. Nervous glycosuria—violent emotional disturbance e.g. fear, anger or pain, may result in temporary glycosuria—rarely observed in normal people.

4. Renal glycosuria—renal threshold set at a lower level than normal—say 140 mgm. (can be diagnosed by the glucose tolerance test).

5. Glycosuria in diabetes, intermittent in mild cases, positive 1-2 hours after a carbohydrate food.

Cells and casts

Epithelium :—A few epithelial cells are found in all specimens of urine. The epithelial cells may be squamous, transitional, small round or polygonal in morphology. The squamous type of epithelial cells are only of pathological significance if present in great excess. The transitional epithelium is normally found in the urine collected after prostatic massage and by ureteric catheterization. The small round or polygonal cells are rarely met in normal

They are found in renal disease such as acute nephritis, chronic venous congestion and chronic parenchymatous nephritis.

Pus cells :—Urine normally contains a few leucocytes. Bacterial infections of the urinary passages are as a rule accompanied by the excretion of large numbers of leucocytes or pus cells.

Red blood corpuscles :—Except when due to contamination from menstrual discharges, blood in the urine is always of pathological significance. An examination for traces of blood insufficient to alter the colour or appearance of the urine is of particular importance in suspected calculus disease, in renal tuberculosis or neoplasm, in papilloma of the bladder, and in infection with schistosoma hæmatobium.

Casts :—When casts are present in large numbers and associated with albuminuria they are an indication of renal disease. The finding of an occasional cast unassociated with albuminuria is not to be regarded as of any significance. Casts are classified according to their microscopic appearances into hyaline, granular, waxy, fatty, epithelial, blood and leucocyte casts.

Hyaline casts are transparent, refractile tubular structures, very delicate and clear in outline. They are of less pathological significance than other varieties. A few may be found in the urine of normal people, the number being increased after exercise and in a concentrated specimen of urine.

Granular casts are similar in appearance to hyaline casts except for the fact that the cast is dotted with granular albuminous matter. They are less commonly found in healthy individuals but occur in the later stages of acute nephritis and in subacute nephritis.

Waxy or colloid casts are more highly refractive in appearance than hyaline casts and have a peculiar dull opaque lustre. Waxy casts are never found in normal urine and are most often associated with the degenerative lesion of the kidney known as nephrosis. They are always an indication of serious renal disease.

Fatty casts are a variety of the previously described casts in which fat globules and granules are present. Fatty casts are not common. They are never found in normal urine but are mostly likely to be present in the later stages of subacute nephritis.

Epithelial, blood, and leucocytic casts have a central core usually provided by a hyaline or granular cast around which the cells adhere. As a rule epithelial and blood casts are an indication of the more acute phases of renal disease such as acute glomerular nephritis.

Common urinary crystals

Uric acid :—Uric acid crystals are most commonly found in concentrated and acid urine. They often occur in febrile diseases

and may be found in healthy individuals after profuse sweating and violent exercise. They are common in leukaemia.

Amorphous urates are commonly found in concentrated urine slightly acid in reaction. They are common in case of heart failure, in febrile diseases and in healthy individuals after severe exercise or as a consequence of a high protein diet with restricted fluids.

The crystalline form is composed of ammonium urate or sodium urate. Ammonium urate is usually found in alkaline urine, especially in conditions of ammoniacal fermentation.

Calcium oxalate:—Calcium oxalate is a very common urinary deposit, being found often in the urine of normal people. It is most likely to appear in slightly acid urine. The excretion of crystals of calcium oxalate is described clinically as oxaluria. Oxaluria is frequently observed in normal people after the ingestion of certain fruits and vegetables particularly rhubarb and spinach and there is no reason to suppose that this is in any way a sign of disordered function. But if the oxaluria is accompanied by slight hæmaturia and excess of mucus the condition is worthy of more attention.

Phosphates:—Phosphatic deposits are found in amorphous and crystalline forms. The commonest variety is an amorphous deposit of calcium and magnesium phosphate. This is found in alkaline urine.

Crystalline phosphatic deposits are found in three varieties:

(a) Triple phosphate crystals—composed of ammonium magnesium phosphates, occur most commonly in alkaline urine which has undergone ammoniacal fermentation.

(b) Stellar phosphate crystals are composed of di-calcium phosphate and occur in neutral or faintly alkaline urine. They gradually disappear from urine when ammoniacal fermentation sets in.

(c) Magnesium phosphate crystals are comparatively rare.

Bacteria and other parasites in urine

Specimens of urine for bacteriological examination need to be collected with special care and to be examined as soon as possible. Catheterization is not always necessary. A mid-stream specimen, collected after cleaning of the urethral orifice, is generally quite satisfactory for bacteriological tests if examined within a few hours. Catheterization is more often required with female patients, in fact, if urine from women is found to contain a few pus-cells and bacteria, a catheter specimen is generally necessary in order to decide, whether or not an infection is present.

Staphylococcal infections may occur in the kidney, bladder, prostate, urethra and epididymis. Such infections may be associated

with calculus disease and provide evidence both of the existence and nature of a stone.

It is common to find *streptococci* in the urine in mixed infections, especially in severe urinary sepsis associated with carcinoma of the bladder or vesico-colic fistula. Pure streptococcal infections of the urine are comparatively rare. Pyæmic abscess of the kidney and chronic urethritis and prostatitis are the streptococcal infections most likely to be encountered.

Pneumococci may be found occasionally in cultures from the genito-urinary tract. They may be excreted in the early states of lobar pneumonia.

Gonococcal infections are far too well known to require detailed description.

Infection with *B. coli* is one of the commonest of urinary diseases. It may give rise to lesions in the kidney, bladder, prostate or epididymis.

Proteus infection

Infection with proteus more commonly occurs in the bladder than in the kidney, and is generally of a more severe character than *B. coli* infection.

B. pyocyaneus is found in the urine under similar circumstances to proteus.

Typhoid and paratyphoid infection

During an attack of typhoid or paratyphoid fever it is very common for patients to excrete the causal bacilli in the urine, especially in the latter part of the pyrexial stage or in early convalescence. A small proportion of patients become permanent urinary carriers. It is obvious therefore that the urine of all enteric convalescents should be regarded as a possible source of infection.

Tuberculosis of the urinary organs may be part of a generalized infection or be limited only to the urinary tract or to one part thereof. The special points to which attention should be directed in urine tests in tuberculosis are the presence of blood, the presence of pus, and the search for tubercle bacilli. Films made from a 24-hour specimen of urine may show the presence of tubercle bacilli when these have not been found in a single sample.

Anærobic infections of the urinary tract are very rare. In long-standing cases of cystitis and pyonephrosis, anærobic bacteria may sometimes be found in the urine, but in these cases the infection is mixed and large numbers of anaerobic bacteria are also present.

An infectious form of jaundice known as Weil's disease is due to infection with a spirochæte, *leptospira ictero-hæmorrhagiae*.

The spirochaetes invade the blood-stream and may be excreted in the urine.

Renal function tests

Specific gravity.—The normal range of the specific gravity of the urine lies between 1010 and 1030. In nephritis with severe impairment of renal function the specific gravity is constantly low and about 1010.

Urea concentration test.—There should be 2.5 per cent or more of urea concentration. If it is less than 2% defective renal function may be diagnosed provided the total volume of urine voided at each hour is less than 100 ccs. and the bladder has been emptied completely.

In the hydræmic type of nephritis with chloride retention, the kidneys may respond well to the urea concentration test in spite of the fact that the urine contains albumin and casts. Similarly in infections of the kidney and in unilateral renal tumour the urea concentration of the bladder urine may appear satisfactory despite serious renal disease.

Chloride concentration test.—Normally after administration of 4 gms. of Potassium Chloride, the salt concentration of the urine exceeds 0.8 per cent. The response of the kidney to the chloride concentration test corresponds closely to its behaviour with the urea concentration test, though some difference may be found in cases of parenchymatous nephritis with œdema.

Blood Urea

The level of the blood urea lies between 20 and 40 mgm. per cent in childhood, youth and middle age. It may rise to 40 or 50 mgm. after the age of 60 years in healthy people and in the later months of pregnancy it may fall to slightly below 25 mgms. per cent. In uræmia, blood urea has been reduced considerably by restriction of proteins. The same fall occurs in cases of enlarged prostate with severe anorexia.

The level of the blood urea is raised in the later stages of all degenerative conditions of the kidney such as chronic nephritis, bilateral polycystic disease or gross destruction of the kidneys by tuberculosis or calculi. It is raised also in acute nephritis, but not as a rule in subacute nephritis or nephrosis. A rise in the level of blood urea invariably accompanies prolonged obstructive lesions affecting both kidneys, such as enlarged prostate or bilateral calculi. Pyelitis and pyelonephritis do not lead to urea retention in the blood, but bilateral pyonephrosis may be expected to raise the blood urea.

Any condition associated with severe anhydræmia such as excessive sweating or diarrhœa may raise the blood urea. This is specially likely to occur in children. Chronic venous congestion and

heart failure have a similar effect. Finally the severe and prolonged vomiting of high intestinal obstruction may be associated with a rise in the blood urea to 300–400 mgm.

The following table would be of use in this connection :

Urea Normal	20 to 50 mgm. per 100 cc. of blood	
Pathological-retention		
Chronic nephritis :	Moderate 50 to 80	„
„	Terminal up to 600	„
Heart failure	up to 70	„
Increased production		
Infections (pneumonia)	up to 70	„
Intestinal obstruction—acute	up to 250	„

The high values found in infections and acute intestinal obstruction are probably due to toxic protein break-down.

Blood-cholesterol

High blood-cholesterol values are found in nephrosis constantly and in many cases of diabetes mellitus, hypothyroidism, pregnancy, nephritis, tuberculosis, cholelithiasis and numerous other conditions, and their differential diagnostic value is not great. It is of interest to note that low values for blood cholesterol are the rule in pernicious anæmia and hyperthyroidism.

At present the main diagnostic value of cholesterol estimation lies in the differentiation between renal and cardiac œdema, particularly in chronic cases, there being no excess of cholesterol in cases of cardiac œdema.

The following table summarizes the actual values observed in different conditions :—

Normal average	.. 180 mgm. per 100 cc. of plasma
Range	.. 160 to 210 mgm.
Diabetes Mellitus	.. up to 1600
Nephrosis	.. 600
Pernicious anemia	.. 75
Liver disease	.. 130
Obstructive jaundice	.. up to 700
Hypothyroidism	.. 550
Hyperthyroidism	.. 100
Late pregnancy	.. 240 mgm.
Idiopathic	.. 400

In nephritis the blood cholesterol is higher the more cases approach the type called nephrosis, with œdema and little or no impairment of nitrogen excretion, while in cases of the type with no œdema and impaired excretion of nitrogen, normal figures are found.

High blood cholesterol is sometimes found in patients with no apparent cause. Subcutaneous nodules containing cholesterol may occur in such patients and in diabetes with high blood cholesterol.

Blood Calcium

In nitrogen retention due to renal failure, decrease in the serum calcium is to be expected. A similar hypocalcaemia has been observed in nephrosis without nitrogen retention. The calcium present in citrated plasma or serum is normally 9–11 mgm. per 100 ccm. The quantity is increased in cases of parathyroid tumour or after administration of parathyroid extract, and in some cases of gout. It is decreased in advanced cases of chronic interstitial nephritis, renal infantilism, and in tetany following parathyroidectomy.

The following table summarizes the relevant data :—

Calcium :—

Normal	9.6 to 12 mgm. per 100 cc. of plasma.
Nephritis with low proteins	.. 7 mgm.
„ with phosphate retention	.. 6 „
After parathyroidectomy	.. 5 „
Failure of absorption	.. 7 „
Rickets	.. 7 „
Osteitis fibrosa	.. 15 „

Blood phosphates :—In renal disease accompanied by nitrogen retention there is usually a parallel retention of inorganic phosphates in the blood. Normally the plasma contains 3–5 mgm. of PO_4 per 100 ccm. This does not include the organic phosphates present in the blood, chiefly in the red cells and in the form of lecithin or phosphate esters. The retention of phosphates in renal disease is one of the factors which cause the acidosis of nephritis, a rise in the inorganic phosphates to 10 mgm. is a very grave prognostic sign.

Blood sugar :—

Normal fasting	.. 90–120 mgm. per 100 cc. of blood
After meals	.. 120–150 mgm. „ „

Pathological :—

Mild diabetes	.. 150–300 mgm.
Severe diabetes	.. upto 800 mgm.

Hypoglycaemia after insulin. Below 60 mgm. and down to 30 mgm.

In normal persons glucose does not appear in the urine in appreciable amounts until the blood sugar rises above the renal threshold. In 70 per cent of people this lies between 145 and 195 mgm. per 100 cc. When the threshold is low, glucose appears in the urine, although the blood sugar is not excessively high.

Cerebro-spinal Fluid

In order to do a cell-count of cerebro-spinal fluid, shake the fluid thoroughly and mix with an equal amount of 2 per cent. acetic acid. The object of the acetic acid is to hæmolyse the red cells, which are always present in small numbers; if the fluid is grossly contaminated with blood, accurate cell count and protein estimation are not possible. The number of cells does not exceed 5 per cubic millimetre in normal fluids.

The cells present in these fluids are practically of only two kinds—small lymphocytes and polymorphonuclear neutrophils, endothelial cells in excess are almost never seen. If small lymphocytes are present, the fluid is probably either syphilitic or tuberculous, but a moderate excess of lymphocytes occurs in other conditions, of which the most important are encephalitis lethargica and poliomyelitis. Polymorphonuclears mean septic meningitis, of which the most common variety is due to the meningococcus, the next common to the streptococcus or pneumococcus.

The protein content is increased in all forms of meningitis and the increase usually corresponds roughly with the number of cells present. In cases of spinal or cerebral tumour, or with meningeal adhesions, the protein content is high and there is no cellular increase.

Protein in cerebro-spinal fluid :

Normal	0.020 to 0.035 per cent.
Meningitis	.. 0.05 to 0.20
Disseminated sclerosis	.. 0.03 to 0.06
General paralysis	.. 0.04 to 0.10
Tabes	.. 0.03 to 0.06
Syphilitic meningitis	.. 0.05 to 0.20
Xanthochromia	.. up to 2

The glucose in the cerebro-spinal fluid normally lies between 0.06 to 0.09 per cent. Low concentrations are found in hypoglycaemia produced by insulin, also in acute and particularly in tuberculous meningitis (0.01 to 0.05 per cent). The chloride in the cerebro-spinal fluid normally lies between (0.71 and 0.75). Low figures may be found associated with low plasma chloride, for example, in chronic nephritis (though not invariably) and in acute intestinal obstruction, also in acute and, particularly, tuberculous meningitis, in which the concentration may fall as low as 0.58 (reckoned as sodium chloride). This reduction in meningitis may be of value in diagnosis. High values may be found in cases of nephritis with nitrogen retention.

Gastric Acidity

The results of titration for acidity may be stated either in terms of number of c.cs. of N/10 NaOH required to neutralize 100 c.c. of

the stomach contents or indirectly in terms of HCl. The necessity for calculations may be avoided if one remembers that provided 10 c.c. of stomach contents have been taken and that decinormal soda is used for titration, the number of c.c.s. of Soda required $\times 0.0365 =$ HCl. per 100 parts.

Very wide variations are found among persons with no other evidence of gastric disturbance. Some 4 per cent have no free hydrochloric acid, in about 15 per cent the level is below 0.03 gram per 100 c.c. and in another 15 per cent over 0.20. The average is about 0.13 gram per 100 c.c. In some instances high hydrochloric acid in the absence of an organic lesion may give rise to pain which is relieved by alkalis, but in many cases it is found in persons who have no such symptoms. When no free HCl. is present, a condition which is often familial, the passage of food through the stomach is rapid and an Ewald test meal may have to be withdrawn in half an hour or less, the secretion is very scanty and the total acidity in the neighbourhood of 10; pepsin is usually absent. The normal figures for an Ewald meal are from 25° to 50° free HCl. and from 50° to 80° total acid.

In some 75 per cent of cases of carcinoma of the stomach free hydrochloric is absent; the gastric juice is nevertheless acid. The free HCl. may be absent within a very few weeks or even a few days of the onset of symptoms. In the rare form of carcinoma which appears in a patient who gives a long and definite history of previous attacks of gastric ulcer, free HCl. may persist. The total acidity is practically always low in gastric carcinoma.

Free HCl. is almost invariably absent from test meals from patients with pernicious anæmia: the fluid is actually neutral. The amount of gastric secretion is scanty and pepsin is present in very small amounts. In the microcytic anæmias of women achlorhydria frequently occurs and hypochlorhydria is the rule. Achlorhydria may also occur in prolonged secondary anæmia.

Free HCl. is almost invariably absent after a successful gastro-jejuno-stomy has been performed, apparently because of the neutralization of the gastric juice by admixture with pancreatic secretion. This neutralization is a most important effect of the operation for the cure of duodenal ulcer. In cases in which free HCl. persists after operation peptic jejunal ulcers may follow.

In cases of pyloric and duodenal ulceration it is rare for the free HCl. to be less than 0.15 gram per 100 c.c., about 0.25 gram is usual and the figure may rise to 0.33 gram. After a considerable hæmatemesis the acidity commonly falls very low and in some cases free HCl. may disappear.

In cases of ulceration of the body of the stomach, free HCl. may be high or low. Free HCl. is frequently absent in chronic

gastritis, cholelithiasis, rheumatoid arthritis and chronic appendicitis. On the other hand, levels of not less than 0.2 gram per 100 c.c. may be found in some cases of the last two conditions and in neurasthenia, nervous dyspepsia and tabes dorsalis.

Blood Counts—Total and Differential

Any increase in the total leucocytes above 10,000 per c.mm. is known as leucocytosis, a decrease below 4,000 per c.mm. is known as leucopenia. The limits of a normal leucocyte count are from 4,000 to 11,000 per c.mm.

Leucocytosis.—Physiological.

(a) *Slight*:—*Causes*:—Cold bath, walking against a cold wind, physical exercise, mental exercise, emotion and exposure to ultra-violet rays, protein meal, afternoon rise.

(b) *Marked* (up to 25,000 per c.mm.)—

Causes:—Violent exercise, high temperatures of the hypertherm, severe pain, great emotion, epileptiform seizure, new-born infant, pregnancy, parturition.

Pathological leucocytosis (up to 80,000 per c.mm.)

Diseases:—Diphtheria, small-pox (early stages), chicken-pox (early stages), cholera, rheumatic fever, anthrax, acute tuberculosis, empyema, appendicitis, pneumonia, typhoid fever with perforation, otitis, osteomyelitis, pyelitis, salpingitis, furunculosis, septicæmia, pyæmia, broncho-pneumonia, cholecystitis, meningitis, erysipelas, peritonitis, tonsillitis, and endocarditis.

Non-ineffective conditions:—Hæmorrhage into the peritoneum, dura mater etc., trauma, malignant disease, terminal states and cachexia, leukæmia.

Intoxications and drugs:—Acute yellow atrophy, acute gout, cirrhosis of liver, intestinal obstruction, eclampsia, uræmia and diabetic coma. Poisoning with carbon-monoxide, mercury or lead, administration of ether, chloroform, salicylates, quinine, camphor, digitalis, adrenalin, salvarsan, nucleic acid etc.

Irritants—such as turpentine, castor oil or calcium chloride (moderate doses).

Serum-sickness, liver extract injection, excessive vomiting and diarrhoea, paroxysmal tachycardia, coronary thrombosis.

Lymphocytosis.—The normal absolute number of lymphocytes varies from 1,500 to 4,500 per c.mm.

Physiological:—High altitudes, infancy.

Pathological:—Whooping-cough, glandular fever, infectious mono-nucleosis, healing chronic tuberculosis, influenza, measles, German measles, mumps, malaria, undulant fever, typhoid and paratyphoid, later states of small-pox and chicken-pox, syphilis, especially congenital syphilis, typhus, postinfluenzal.

2. Leukæmia, lymphosarcoma.

3. Hyperthyroidism, hypothyroidism, obesity, diabetes, Banti's disease, mycosis fungoides, emotional states, excessive ultra violet irradiation.

Lymphopenia. — *Causes*: Acute infection, exhaustion, excessive X-ray irradiation, vitamin deficiency, terminal phase of uræmia.

Monocytosis (above 500 per c.mm.)

Causes:—Glandular fever, malaria, kala-azar, monocytic leukaemia, endocarditis lenta, tuberculosis, typhoid fever, Hodgkin's disease (occasionally), sympathetic ophthalmia, ulcerative colitis.

Eosinophilia (150–400 c.mm. normal).

Causes:—1. Intestinal helminthic infection, allergic diseases (bronchial asthma, food sensitivity, hay fever, urticaria, Henoch's purpura), foreign protein injection, angioneurotic oedema, skin diseases (pemphigus, dermatitis herpetiformis, scabies, psoriasis, eczema and prurigo), convalescent phase of acute fevers.

2. Pulmonary conditions—Löeffler's syndrome and tropical eosinophilia.

3. Leukaemia and erythraemia, Hodgkin's disease (15 per cent cases).

Basophilia (0–100 c.mm. normal).

Causes:—Chronic myeloid leukaemia, polycythæmia vera and cirrhosis of liver.

Leucopenia (below 4,000 per c.mm.)

Causes:—1. Starvation or malnutrition especially in cachectic and debilitated states.

2. *Infections*:—Influenza, measles, German measles, mumps, small-pox in the post-febrile period, typhoid, paratyphoid fevers, chronic tuberculosis, undulant fever, malaria, dengue, kala-azar, histo-plasmosis, relapsing fever and severe toxæmia in overwhelming infection.

3. *Intoxications and poisonings*:—Although such conditions usually produce a leucocytosis, where the poisoning is more severe, a leucopenia occurs. Severe intoxications with arsenic, antimony, benzol compounds, sulphonamides, mercury, lead, mustard gas, as well as excessive X-ray and radium exposures.

4. *Blood diseases and diseases of the bone-marrow*:—Aplastic anæmia, Banti's disease, Gaucher's disease, aleukæmic leukaemia, extensive metastases in bones.

5. *Miscellaneous conditions*:—Acute hæmorrhagic shock, Widal's hæmoclastic crisis, anaphylactic shock.

Wassermann Test

The Wassermann reaction is positive in a number of diseases besides syphilis, hence it is essential to determine if the patient is syphilis in order to give a correct interpretation of a positive Wassermann test. The Wassermann test is positive in a variable manner in the following diseases:

Frequently positive in:—Leprosy, yaws, malaria, dengue, trypanosomiasis, pinta and bejal.

Less frequently positive in:—Septicæmia, endocarditis, pneumonia, tuberculosis, relapsing fever, spotted fever, typhus fever, scarlet fever, infectious mono-nucleosis, pernicious anæmia, leukaemia, sarthranatosis, severe jaundice, lymphogranuloma inguinale, kala-azar, asthma and other allergic conditions, lecithinophile hepatogastro-intestinal syndrome.

Very infrequently positive in:—Bacterial growth in serum, excess of fat and digestive products in serum, passive transfer from the mother.

The Wassermann test may give a weak or even negative reaction in early and late cases of syphilis.

Van den Bergh Reaction

Excess of bilirubin in the blood may result from excessive formation owing to increased break-down of red corpuscles, or from interference with excretion owing to damage to the liver or obstruction to the bile passages. There is some difference in the form in which bilirubin circulates in these two conditions. When the excess of bilirubin is due to obstruction, the bilirubin in the plasma will react at once with Diazo-benzene-sulphonic acid (direct Van den Bergh reaction) but when the excess results from excessive break-down, the reaction is delayed or does not take place unless the proteins of the plasma have been precipitated by alcohol (delayed or indirect reaction). This difference in the state of the bilirubin appears to result from passage through the liver cells and, therefore, with liver damage, a mixture of the two forms of reaction may be expected (biphasic reaction).

The presence or absence of excess of bilirubin in the plasma may be of value in differentiating hæmolytic from secondary anæmias, while the state of the bilirubin in the plasma may help in the diagnosis of the cause of obscure cases of jaundice. Excess of bilirubin can be seen in the plasma or serum before yellow pigmentation of the sclerotics is noticeable, and before bile pigments can be detected in the urine.

The following table gives some useful data:—

Bilirubin.	Amount per 100 cc. plasma.	Van den Bergh reaction.
Normal	.. 0.0 to 0.5 mgm.	Indirect.
Pernicious anæmia	.. 0.7 to 7.0 mgm.	Indirect or delayed.
Hæmolytic jaundice	.. 2 to 6 mgm.	Direct.
Simple obstructive jaundice	.. up to 50 mgm.	Delayed or indirect.
Pneumonia	.. up to 4 mgm.	Indirect or biphasic.
Cirrhosis of liver	.. 0.4 to 6 mgm.	

Icterus Index

This measures the intensity of the yellow colour of the serum. As this colour depends largely upon bilirubin, the test is of some

practical value in giving a simple approximate estimation of the bilirubin present. Hæmoglobin, hæmatin, carotene and other lipochromes, if present, cause gross errors. A normal icterus index excludes hyper-bilirubinaemia. However, high values are much less dependable and should always be confirmed by the Van den Bergh test.

Normal serum gives readings from 4 to 7 (maximum of 9). Readings from 10 to 16 may indicate latent jaundice, while higher readings are obtained with evident jaundice (up to 100 or more).

Urobilinogen and Urobilin

They are present in small amounts in normal urine. They are absent in cases with complete obstruction of the common bile duct. They are increased in conditions associated with increased blood destruction, or with diffuse liver injury. In freshly voided urine pigments are largely or entirely in the form of urobilinogen, which is rapidly converted into urobilin by light and acid.

Rate of Sedimentation of Red Corpuscles

An increased rate of sedimentation is found when the fibrinogen or serum globulin is raised. It is probably due to a qualitative rather than a quantitative change in the globulin. The rate of sinking is also increased when the number of red corpuscles is reduced. The rate of sedimentation is measured by the height of the column of plasma left clear in a given time by the sinking of the red corpuscles.

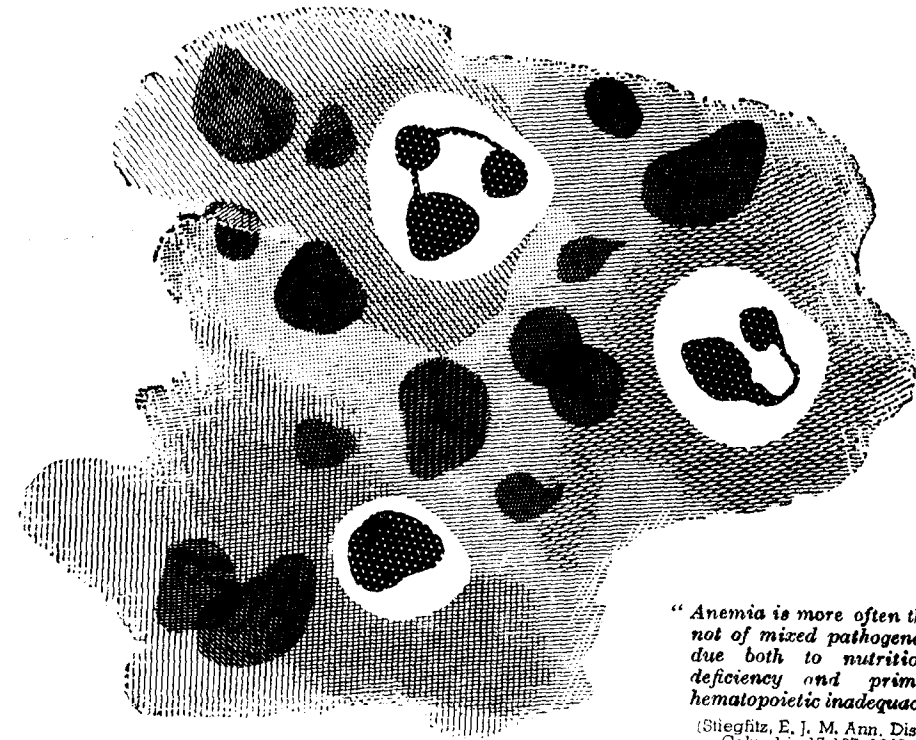
When the Wintrobe's method is used the rate of sedimentation of the blood of normal persons is 0 to 10 mm. with an average of 2 mm. in males and 5 mm. in females. In pregnancy a great increase in the rate is noticeable as the corpuscles sink about 30 mm. in the first 15 minutes and settle completely in about 45 minutes. In malignant disease and active tuberculosis the level sinks about 40 mm. in 15 minutes and even more rapid sedimentation may occur in acute infections and nephrosis.

An accelerated sedimentation rate has also been observed in active chronic infections including syphilis, internal hæmorrhages, any condition associated with necrosis or extensive destruction of tissue including coronary thrombosis and anæmias of all types.

It is not, as a rule, increased in relatively inactive, chronic focal infections, in benign tumours, in unruptured ectopic pregnancy, nor in purely psycho-neurotic states.

In general there is a rough parallelism between the activity of an infection and the degree of acceleration of the rate, particularly of the fall during the first 15 to 30 minutes. The rate of sedimentation is of great value in judging the progress of a disease such as tuberculosis.

(To be continued).



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Cardiac Failure or Circulatory Failure*

K. NARAYANAMURTHY, M.D.,
Madras.

We often speak of a cardiac failure as right sided or left sided or a decompensated heart, or congestive failure. All these terms are not now so commonly used to explain the true conception of the principles that underlie a heart failure. In majority of cases it is very difficult to recognise the beginning of a failure and to distinguish between the various degrees of failure which are likely to occur later. This leads us to concede that there are two varieties of circulatory failure viz., (1) Cardiac or central and (2) peripheral or vascular. In the former category the fault exclusively lies in the heart itself which is the central pump: when it fails to propel adequate quantity of blood into the general circulation we call it a central failure. The defect may be in the failure of tone of the cardiac muscle; (2) failure of contractibility causing circulatory inadequacy; and (3) changes in the primitive musculature, causing irregularities in rhythm. The degree of degeneration of the cardiac muscle is difficult to estimate clinically. We can have only guidance on this point by (1) Exercise which can be done without distress such as pain, palpitation and dyspnoea; (2) dilatation present; (3) character of the heart sounds; (4) the blood pressure; and (5) electro-cardiogram. In the second variety there is a considerable amount of dilatation of the vascular bed especially of the splanchnic area, which results in the diminution of the venous flow of the blood to the heart. Here the mistake exclusively lies with the peripheral blood vessels and not certainly in the heart. Very often it is not possible to mark a clear distinction between these two types of failure. It is for this reason we experience considerable amount of difficulty in the approach of not only making a diagnosis but also in the treatment part of it. That is why we come across several disappointments in the management of a case of cardiac failure.

If the diagnosis of a central or peripheral failure is correctly arrived at there is a possibility of recovery by giving appropriate lines of treatment. We have a number of drugs—practically all that we use in the treatment of cardiac conditions can be utilised as appropriate drugs in the treatment of central failure, but we do not know as yet any single drug that can be used with the same amount of confidence as in the case of peripheral failure. It must be said that it is very often difficult to diagnose a central failure where we have a number of drugs to treat, but it is very easy to diagnose a peripheral failure fairly a number of hours earlier if not days, but we have no drug to treat such an eventuality. Though in cases of diphtheria, pneumonia, influenza—not often

* Specially contributed to THE ANTISEPTIC.

recognised by us—and in coronary infarction, the primary cause of failure is central in origin, it may sometimes be combined with peripheral failure. That is why cardiac specialists are embarrassed in diagnosis and also in the treatment. In this paper I confine my observations to the symptoms of heart failure leaving the prognosis and treatment part of it to a later paper.

It should be remembered the essential cause of cardiac failure lies in the heart muscle itself. On account of the degenerative changes in the heart muscle it is incapable of meeting the demands of the body.

Valvular defects, as in double mitral, aortic regurgitation, aortic stenosis etc., cause a mechanical impediment to the heart muscle in its proper function. In all cases of chronic valvular disease it is possible the myocardium may be affected or the blood vessels as in hypertension to a varying degree.

What we mean by heart failure is that the heart muscle is incapable of maintaining an efficient circulation whatever might be the cause. The 'reserve' force of the heart becomes very much less as a result of which a person who was capable of a certain amount of physical exertion now unable to do so without feeling breathlessness. If this diminution of the cardiac output is not diagnosed fairly early the 'rest force' of the heart also becomes diminished and the symptoms like breathlessness and cough are evident during rest. In addition to this objective sign like pulsation in the jugulars, œdema of the feet and other dependent portions of the body are evident. The onset of cardiac failure is rapid or sometimes it may be gradual. Breathlessness with increased respiratory rate, an undue fatigue, palpitation, pain or discomfort over the precordial region, giddiness which may be slight or transient, sometimes severe enough to make the patient fall and faintness due to loss of consciousness are evident in case of a cardiac failure. Here it must be said that all these symptoms may be due to extrinsic causes and that is one of the reasons why certain of these heart attacks might cause alarming symptoms, though they may be innocent. On account of the heart not capable of maintaining an efficient circulation, symptoms due to cerebral anaemia such as fatigue, sleeplessness, giddiness, fainting, lack of power of concentration, headache, delirium, nervousness, loss of emotional control and mental irritability almost resembling insanity occur. The breath-paroxysmal dyspnoea which is termed cardiac asthma is often noticed. They are really paroxysms of auricular fibrillation or auricular flutter. The attack of suffocation prevents the patient from sleeping and he feels a sense of oppression in the chest, substernal discomfort in the chest, followed by cough frothy expectoration which may sometimes be blood-stained. The patient feels a sense of impending dissolution. On examination we find bubbling

rales at the base of the lungs—pulmonary œdema—first sound being feeble or completely been replaced by a soft murmur or resembling the first sound at the mitral area and an accentuated second sound over the pulmonary area. The patient sweats and cyanosis may be evident. This lasts a few minutes to a few hours and the attack may subside. Sudden death in the vast majority of cases is due to the left ventricle getting into a fibrillation which brings about a standstill of the heart, even without any previous manifestation of heart failure.

A gallop rhythm presystolic in time, pulses alternans a tic-tac rhythm, a triple rhythm viz., protodiastolic, presystolic and systolic may frequently be found. In protodiastolic rhythm the extra sound occurs immediately after the second sound and coincides with the normal third sound. It is found in chronic mitral disease, especially stenosis, when the auricular fibrillation is present. Sometimes we find in young subjects with normal hearts this protodiastolic rhythm and rarely pulmonary stenosis and broncho-pulmonary conditions. The two varieties of normal and abnormal protodiastolic rhythm may be diagnosed by auscultation of the heart when the patient is in a recumbent posture, and (2) an X-ray examination which might reveal the prominence of the pulmonary cone. It is not present in individuals after 25 years and seldom seen in persons above 35 years of age. The prognosis is not so serious as in the case of presystolic form of triple rhythm. In this form the extra sound immediately precedes the first sound—it is synchronous with the auricular systole. The electro-cardiogram reveals increase of the 'P—R' interval which may be evident especially in aortic incompetence, aortic stenosis, coronary disease and in left-sided failure with chronic myocarditis. It cannot co-exist with auricular fibrillation. We also notice a bundle-branch block in the electrocardiogram. The prognosis in a great majority of those cases is very serious. There may be quadruple rhythm—a first sound, a second sound, a protodiastolic extra sound and a presystolic extra sound. This extra sound is found between the first and the second normal sounds which is found most intense at the apex. This is really of no clinical significance. There may be cough with expectoration, cough and hæmoptysis are common in left ventricle failure and also in mitral stenosis. Cheyne-Stokes respiration sometimes occurs in chronic heart disease when there is left ventricular failure. If it is found to occur during sleep and if it is slight during the night it is not so serious as when it occurs during the day.

Digestive symptoms on account of the chronic venous congestion or due to deficient blood supply are often present. There is loss of appetite, flatulence, sense of fulness and pain in the epigastrium, nausea, vomiting, constipation and rarely diarrhoea. The liver is palpable having a sharp edge and a smooth surface. It is tender to the touch and rarely pulsates. Ascites may be present associated not always with jaundice.

The pulsation of the jugular veins in the neck associated with cyanosis and clubbing of the fingers may be present in congenital morbus-cordis or in chronic cardiac cases. The cyanosis that occurs in other conditions such as broncho-pulmonary affections, sulphæmoglobinæmia, in patent ventricular septum, patency of interauricular septum etc. have to be diagnosed from the real cardiac failure. Oedema which is often a late manifestation of cardiac failure is likely to occur in mitral and chronic myocardial lesions than in aortic lesions. Oedema, which is cardiac in origin, commences in the most dependent parts of the body, and by the evening a certain amount of the thighs, the genitalia, the trunk, and, in the later stages, it becomes generalised. Ascites, hydrothorax, and hydro-pericardium may be found. We notice also oedema in varicose veins in the legs, in severe as terminal event in great wasting disorders, in venous thrombosis, pressure due to tumours, hydrostatic oedema—which is common only seen in people of middle age etc. must be differentiated from the oedema of cardiac failure. If the heart disease occurs in the earlier years of life the individual growth is arrested. Embolism as well as arterial and venous thrombosis may indicate a cardiac failure, hæmoptysis, with liver enlargement and ascites, are common. Embolism, generally cerebral, produces hæmoptysis. The urine is high coloured, scanty and may exhibit excess of urates, albumen, and sometimes blood.

The clinical manifestations of a heart failure, for convenience, may be divided into 4 degrees—slight, moderate, severe and very severe or extreme. In the slight degree—shortness of breath, palpitation, fatigue, precordial pain on exertion are found.

In the second degree, shortness of breath, palpitation on walking, slight oedema of the lower extremities, increased pulse rate, slight enlargement of the liver etc.

In the third degree, shortness of breath, palpitation on slight exertion, considerable oedema, considerable degree of enlargement of liver, tic tac rhythm, triple rhythm—protodiastolic in time.

In the last degree, there is continuous shortness of breath, general anasarca, severe pulmonary oedema, murmur presystolic in time—triple rhythm, pulses alternans in the absence of increased pulse rate.

Peripheral Failure

In this we notice giddiness, faintness or actual syncope, shock, and circulatory collapse. In all these we find there is a remarkable fall of blood pressure. These symptoms are very seldom seen in the primary or cardiac failure. There are four varieties of syncope: (1) Cardiovascular autonomic dystonia or postural; (2) vasovagal or Nothnagel syndrome, (3) carotid sinus and (4) that

which occurs in shock and circulatory collapse. The first is more frequent in aged persons when the pulse becomes feeble and imperceptible. Vasovagal attacks are common among the young in whom the condition of health is not satisfactory. These are generally referable to the stomach, the circulatory and respiratory systems. These attacks are produced by emotion such as the sight of blood, a prick by a needle, or sudden fright, or by a stuffy atmosphere. They rarely get this symptom when they lie down in bed. The blood pressure falls, the pulse is not felt, there is pallor, cyanosis, profound fatigue, respiration becomes slow and may almost cease, dilatation of the pupil, nausea, vomiting and slight convulsions of the arms, face and trunk may be noticed. The patient may fall down and the attack may last upto 10 minutes and rarely to half an hour. But usually the patient takes several hours to recover. Carotid sinus syncope is due to the hyper-sensitivity of the carotid sinus which is usually functional. But it may also occur by a tumour, pressing over that area. The symptoms in this condition resemble those of vasovagal attacks. Circulatory collapse and shock may be found in some acute infective diseases such as diphtheria, pneumonia, pneumothorax, coronary thrombosis, pulmonary embolism, biliary colic, rupture of any abdominal viscera, severe vomiting and diarrhoea after severe injuries, surgical operations or profuse loss of blood. The patient becomes pale, there may be some cyanosis, coldness of the body surface and there may be profuse cold and clammy sweats. The mind is generally clear but there may be restlessness and apathy. The pulse is weak and almost imperceptible. The face is blue, the eyeballs sunken, and there is an anxious expression. The systolic and diastolic blood pressure almost falls. The first sound at the apex is weakened almost, may approximate the second sound and at some times not audible. The venous pressure is decreased. There is nausea and vomiting. There may be diarrhoea, and on account of the enormous loss of fluid by sweat the output of urine is diminished. It is very difficult to draw a clear distinction between shock and collapse.

It may be said that there is a great difference between the central and peripheral failure. Usually, it is found in practice that they both overlap each other and in such of those cases great care must be bestowed on the diagnosis for proper treatment. Serious cardiac affection, like coronary thrombosis, when correctly diagnosed and treated, holds out a good prospect, while wrongly diagnosed leads to the incarceration of patients in bed for weeks and months.

We know now that heart failure is not a disease *sera generis* and it is common all over the world. On account of the 'debilitating conditions' and proverbial poverty that occur in India—nutritional anæmias etc., as they throw considerable strain on the

damaged myocardium—possibly heart failure is met with in our country more often than in other countries.

If we put it figuratively, a cardiac patient toils along with "Breathlessness on exertion" as his companion on the rough road of life on which they meet with sudden death and if they don't meet with it they reach two destinations one discomfort and the other disability with pain. The discomfort is associated with oedema occurring off and on and the other disability with pain is associated with angina, cardiac infarction, cardiac asthma, pulmonary oedema, which all occur together or separately finally giving the individual an eternal rest in death—as a result of heart failure. No other organ in the body is so truthful as the heart. It starts functioning from the intra-uterine life of the foetus and finally gets its repose in death. A man can go on without limbs, eyes, teeth etc., but cannot go on without a heart.

Mechanism and Diagnosis of Bronchial Asthma*

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BRONCHIAL asthma is a very common condition. Every doctor realises that this condition, if not treated radically in early stages, may be crippling in its effects. Yet I have observed that the general practitioners always have a hopeful attitude when dealing with a case of bronchial asthma. As this condition, in its early stages, is readily amenable to symptomatic treatment, the etiological factor remains undiscovered, and the condition drags on for years, during which irreparable damage occurs to the respiratory organs. Thus a proper understanding of its mechanism is essential to the early radical treatment.

Definition.—It is a condition of breathlessness brought on by narrowing of the lumen of the bronchioles. There is thus interference with normal respiratory mechanism, which is responsible for the air hunger.

Anatomy and physiology.—A bronchiole has three coats. (1) An inner coat of mucous membrane. The mucous membrane contains plenty of mucous glands, and is lined by columnar ciliated epithelium; (2) a submucous coat, which contains blood vessels, and nerves; and (3) an outer coat in which are elastic tissue, and broken plates of cartilage, interspersed with plain bronchial muscles.

*Specially contributed to THE ANTISEPTIC.

The nerves supplying the bronchioles are the vagus and the sympathetic. The sympathetic fibres are from 3rd and 4th dorsal region. Both the nerves supply branches to the mucous membrane, blood vessels, and to the plain bronchial muscles. The action of the vagus and sympathetic nerves are antagonistic, *i.e.* the action of vagus stimulation, and sympathetic inhibition are the same, and *vice versa*. The vagus nerve is more powerful in its action than the sympathetic.

When the vagus is stimulated: (1) the mucous membrane starts secreting mucous; and (2) the blood vessels dilate. This dilatation brings in its wake diapedesis of blood corpuscles, and exudation of blood plasma into the submucous coat; and (3) contraction of the plain bronchial musculature. This reduces the lumen of the bronchioles.

Mechanism of asthma.—The main cause of asthma is the narrowing of the lumen of the bronchioles. This narrowing of the bronchiolar lumen can be brought about in three ways:—

- (1) Stimulation of the vagus;
- (2) Allergic action on the bronchioles; and,
- (3) Inflammation of the bronchiolar wall.

These three groups will now be considered in detail.

Vagal stimulation.—The vagus nucleus lies in the medulla oblongata. From here the vagus nerve runs into the posterior cranial fossa, passes out of the skull through the jugular foramen, runs down the neck and chest, and through the oesophageal orifice into the abdomen, and ends in coeliac ganglion. It supplies branches to meninges, external ear, pharynx, nose, larynx, trachea, bronchioles, lungs, pleura, diaphragm, heart, pericardium, oesophagus, stomach, intestine and other abdominal organs.

Thus reflexly from these various organs, the vagus nerve can be irritated, and that irritation may manifest itself by bronchial spasm. To cite a few examples of this vastly diverse group. It is well known that external ear conditions, *e.g.*, wax in the ear of children give rise to troublesome irritating cough, and even bronchial spasm. Conditions like dyspepsia, diarrhoea, and constipation in children often produce asthmatic attacks. This is because the central nervous system of children is very unstable, and gets easily upset. Even in adults, distention of stomach, intestinal worms, and uterine disorders, have been responsible for asthma.

Brodie and Dixon¹ (1903) have shown that stimulating the nasal mucosa produces a reflex bronchial spasm. In fact in some patients nasal operations have been the precipitating factor in asthma.

Besides this the vagus nerve through intricate nerve communications in the central nervous system is in direct contact with special

sensations of sight, smell, hearing, etc. Excessive emotional upsets due to fatigue, worry, fear, can precipitate asthmatic attacks. A woman having had an attack of asthma after smelling some flowers, got an asthmatic attack on another occasion on seeing from a distance a bunch of flowers made of cloth. A young boy of nervous temperament got an attack of asthma every time before examination. I once scolded a medical student for writing imperfect medical notes, and within a short time, in front of the whole batch he suddenly developed breathlessness and other symptoms of vaso-vagal collapse.

It is thus clear that reflex vagal irritation can cause asthma. Certain individuals are endowed with a very unstable nervous system, just as some inherit poor arteries. This may be hereditary. It is believed that "An unduly irritable bronchial centre" is transmitted by heredity (Hurst 1914). Subjects of this group are very nervous and of irritable temperament, and get easily upset. In them minor provocations, or somatic, or psychic trauma is sufficient to produce an asthmatic attack.

Allergy.—Various harmless common substances are known to produce undesirable bodily reactions in certain individuals. This allergy, or "Chemical idiosyncrasy" is an inborn tendency of certain individuals. This allergic sensitivity may be local and limited to certain system or organ, *e.g.*, skin, gastro-intestinal tract, respiratory tract, etc., or it may be general, when the symptoms of allergy may manifest as those of anaphylactic shock. Asthmatics are well known to show anaphylactic tendencies. On careful inquiry they often give history of urticaria, dermatographia, eczema, eosinophilia, migraine, etc.

The site of action of allergy is not clearly defined. Whether the allergin acts locally on bronchioles, or indirectly through the vagus is not certain. Eppinger and Hess (1917) believe that "The foreign protein or toxin produces the asthmatic attack by inducing the vago-tonicity." On the other hand, Brodie and Dixon² (1903), by their experimental observations, have established the action as one of bronchial spasm. It is now generally agreed that "The broncho-constrictor fibres or the vagus are the channels by which impulses discharging the asthmatic paroxysm reach the bronchi." (Price, 1941).

The exciting allergic agents can be classified as follows:—

1. *Inhalation*:—(a) Vegetable—Pollen of various grasses and flowers. Dust from certain cereals, moulds, etc. Room dust may be an important factor.

(b) Animal—Emanations from horses, sheep, cows, pigs, goats, rabbits, cats, dogs, birds, etc. Here again the infection may be *via* the substances in the dust.

(c) Mites.

2. *Ingestion*:—(a) Vegetable—Various vegetable proteins, *e.g.*, various grasses, wheat, maize, rice, barley, oats, leguminous foods, peas, beans, lentils, spinach, fruits and nuts.

(b) Animal—Various animal proteins, *e.g.*, meat, milk, eggs, oysters, lobsters, crabs, and other fishes.

(c) ~~Drugs~~—Aspirin, phenolphthalein, quinine, etc.

3. *Injection*:—Sera, vaccines, acetyl, choline, prostigmin, muscarine, pilocarpine, etc.

4. *Metabolic*:—Abnormal products of digestion in stomach, or intestine. Faulty metabolism in liver or tissues.

5. *Bacterial*:—Variety of organisms, streptococci being the most common. Their usual abode in the body are the septic foci, *e.g.*, sinuses of the nose, tonsils, nasal polypi, etc.

6. *Unknown*:—There are many cases where the cause remains unknown. There are some very curious peculiarities known about asthma. Some patients are better in dry, others in damp, climates. Cold air may induce an asthmatic attack, while warm air brings about relief. As regards locality, a person may get an attack in one locality and not in another adjoining locality. Even houses predispose to asthmatic attacks.

It is important to remember in regard to allergy, that certain individuals have an asthma-diathesis. By this is meant "A congenital and often inherited constitutional abnormality" (Hurst 1921), in the vagal constituent of the bronchial nervous system, which predominates over its sympathetic partner. In the presence of this diathesis, certain allergic substances, or reflex or psychical stimuli, which have no effect on the normal individuals, give rise to vagal discharges. These vagal discharges then produce spasm of the bronchial muscles, hypersecretion of mucous, and bronchial congestion.

Inflammation of the bronchiolar wall.—Repeated attacks of cold, frequent attacks of bronchitis, too often lead to asthma. Chronic bronchitis is a very important cause of asthma. The relation of asthma to bronchitis is three fold:

(1) Asthma commonly follows a primary bronchitis.

(2) Recurrent attacks of severe bronchitis are followed by attacks of asthma. These attacks of asthma come on only when the bronchitis is at its worst, and clear up again when the bronchitis clears up.

(3) True allergic asthma may lead to secondary bronchitis.

Pathology:—Asthmatic attacks are due to narrowing of the bronchiolar lumen. Inspiration being an active process, air can force through the contracted bronchioles, but finds difficulty in coming out. Thus is produced the expiratory dyspnoea characteristic of bronchial asthma.

Repeated bronchiolar spasm brings about hypertrophy of the plain muscles of the bronchioles, and this has been observed post-mortem in cases of allergic and vagotonic asthmatics who have died of some other diseases. Later, due to lowered vitality of the mucous membrane of the bronchioles, secondary invasion by bacteria takes place. This bacterial invasion is facilitated by the slightly hypertrophied state of the mucous membrane, which is pouring out excessive mucous. This mucous is the suitable medium for bacterial growth. Thus is produced a secondary bronchitis.

Whether the bronchitis is primary or secondary, it has a tendency to become chronic, if it is not treated properly in the early stages. The mucous membrane gets hypertrophied. The elastic and plain muscular tissues of the bronchioles is at first hypertrophied, later replaced by fibrous tissue. The fibrous tissue contracts, leading to further narrowing of the bronchiolar lumen. Thus there is permanent damage to the bronchial wall, which is not amenable to radical treatment, and even symptomatic relief becomes problematic.

These changes in the bronchioles lead to secondary emphysema of the lungs. Most of the German writers were of the opinion that all cases of chronic bronchitis and emphysema, occurring in earlier years, had an asthmatic basis. The emphysema and chronic bronchitis lead later on to varicose, beaded, or fusiform dilatations of the bronchial tubes, and in some cases even larger branches may show dilatation.

Effect on the heart:—Most of the writers have noted enlargement and hypertrophy of the right ventricle, in cases where chronic bronchitis and emphysema are present. Andrel has quoted a maxim (as old as Hippocrates) that "Asthma is a sign of long life."

In cases who have died of asthma, post-mortem has revealed that the death is due to complete obstruction of the bronchial tubes (already in a state of spasm) by tenacious mucous. In these cases no definite lesions were noted in the heart. Hypertrophy of the right ventricle was the only abnormality present.

Symptoms and signs:—The signs and symptoms are well known, and need no elaboration.

Classification:—Asthmas can be classified into—

- (1) Allergic—(a) Simple; and (b) tropical eosinophilia.
- (2) Non-allergic—(a) Psychic; and (b) bronchitic.
- (3) Status asthmaticus.

Tropical eosinophilia.—What exactly is the role of eosinophilia in allergy is not yet definitely known. Eosinophilia occurs in various infections, e.g., in infections with round worms, bilharzia worms, trichina spiralis, and filariasis. Certain neoplasms show eosinophilia, e.g., Hodgkin's disease. It is also noted in periarteritis

nodosa. I have noted marked eosinophilia in two cases of relapsing fever. Similarly asthma and other pulmonary infections may show eosinophilia.

Of the few cases of tropical eosinophilia observed by me, all did not show the picture of asthma. Besides asthma, other manifestations noted were those of acute bronchitis, broncho-pneumonia, and even of lobar pneumonia.

Status asthmaticus.—This is a clinical entity by itself. History runs over a number of years. At first the attacks are infrequent, and response to treatment prompt. After some years, the attacks come on more frequently, and ordinary remedies, which gave relief at first, now show no or very poor response.

The patient depicts a picture of misery. His countenance shows extreme anxiety and distress. He can hardly talk a few words. Such an attack may last for several hours, with little or no remission. The patient may become extremely ill and may die, unless the attack subsides. His chest is emphysematous, and shows typical signs, e.g., wheezing rhonchi, and rales all over the chest, with markedly prolonged expiration. In between the attacks, the patient is perfectly normal. In short, status asthmaticus is nothing but chronic asthma in those patients in whom irreparable damage has occurred to bronchiolar tissues. They are late results of asthma, either of allergic or non-allergic origin.

As for the other groups there is nothing very distinctive clinically. But a few points are noteworthy.

- (1) Bronchitic asthma is more frequent in children, usually from two to five years.
- (2) Allergic asthma often shows other allergic manifestations.
- (3) Psychic asthma should be suspected in overcautious, over-anxious, oversensitive type of asthmatics. These patients are of an intelligent nature. They show ready willingness to co-operate.

No definite statistics are available regarding the ratio of the various types of asthma. Various authors put down from 60 to 90% cases as allergic. In my own series of cases, 46 in all, the classification into various groups is as follows:

Psychic type	..	2 cases (4.3%)	} Non-allergic (10.5%).
Bronchitic type	..	3 .. (6.2%)	
Tropical eosinophilia	2	.. (4.3%)	} Allergic (89.5%).
Allergic type	..	39 .. (85.2%)	

ETIOLOGY.—A few remarks about etiology.

- (1) *Age*:—At all ages. About one third of all cases of asthma have their onset during the first ten years of life.

(2) *Sex*:—Males are three times more frequently affected than females.

(3) *Hereditary predispositions*:—Family history of allergy (asthma, eczema and migraine) is obtained in 70% of cases. In 50% unilateral. Transmission occurs twice as often through the females, as through the males.

INVESTIGATIONS.—It is very important to find out the etiology of asthma, as only then can a radical treatment be instituted.

(1) *W.B.C. total and differential* should be done in all cases of asthma. It is necessary to find out whether an asthma is allergic, or non-allergic. There are not many reliable means of finding this out. A routine white cell count—total and differential—often helps us. In tropical eosinophilia presenting a picture of asthma, the total white cell count varies from 15,000 to 60,000, and eosinophils vary from 15% to 80%. In simple allergic asthma the white cell count often remains normal, while the eosinophil count varies from 5 to 15%. In any differential count, if the eosinophil count is more than 5%, I take it as an indication of underlying allergic etiology. Between 3 and 5% the condition is doubtful.

(2) *Stool examination*:—If eosinophilia is found, the next step is to exclude other causes of eosinophilia. Stool examination to exclude parasites is necessary, especially ascaris infection.

(3) *Sputum examination*:—Sputum shows, Charcot-Leyden crystals and Curchman's spirals. It is said that the presence of eosinophil cells, more concentrated in plugs and spirals, is diagnostic of allergic cases. But in my own series of cases, except in those of tropical eosinophilia which showed clinical picture of asthma, I have not noted eosinophil cells in the sputum of allergic cases.

Few workers from Ceylon (Carter, Wedd, and D'Abbrera-1944) have shown the presence of mites (acarina) in the human sputum.

(4) *Protein sensitivity tests*:—Once allergic etiology is established, the next important thing is to find out the particular foreign substance to which a patient is sensitive. Protein sensitivity tests are not of much value. After middle age positive results are very rare. Very frequently tests are negative to all obtainable proteins. Many patients respond negatively to allergens to which they are known to be sensitive, and positively to foods which they can eat with impunity. Besides, in some patients, positive results are obtained with more than one protein. Hence a positive reaction is no proof that it is the etiologic factor.

Reviewing the literature, one finds that earlier in this century a lot of work was done in connection with protein sensitivity tests.

Almost all the workers gave varying proportion of positive sensitivity figures in their cases. The figures ranged from 50 to 90% in different hands. But now-a-days, knowing the limitations of these sensitivity tests, their importance has receded.

Technique:—There are two good and simple methods of doing the protein sensitivity tests. The non-hairy skin of the front of the forearm is cleaned as for an injection. It is then utilised for the skin tests.

(a) *Cutaneous or scratch test*—A freshly prepared solution, or a powder is put on the skin, and the skin is scratched through it.

(b) *Intra-cutaneous test*—0.05 c.c. of a freshly prepared sterile solution is injected into the skin so as to raise a small cutaneous wheal.

With both these methods a positive reaction consists of an urticarial wheal, $\frac{1}{4}$ inch in diameter, appearing within 20 to 30 minutes. Intra-cutaneous method is the more sensitive of the two.

(5) *Septic focus investigation*:—Whether an asthma is allergic, or non-allergic (bronchitic), the next step in investigation is the finding of a septic focus. This may entail a thorough overhauling of the patient, and the result may be disappointing.

Start the search for septic focus by examining the nose, mouth, and naso-pharynx. Look for swollen turbinates, deflected nasal septum, nasal polypus, sinus infections, septic conditions of the mouth, pyorrhœa alveolaris, teeth cavities, or deep seated pulp infections of the teeth, and septic tonsils. Ear examination for external ear conditions, otitis media, and mastoid cells infection.

Gastro-intestinal conditions, e.g. auto-intoxication from chronic gastritis, chronic cholecystitis, or cholelithiasis, chronic colitis, and ulcerative colitis.

Chest conditions, e.g. bronchiectasis, chronic pulmonary tuberculosis, fibroid phthisis, or fibrosis of the lungs.

Genito-urinary conditions, e.g. pyelitis, pyelo-nephritis, cystitis, prostatitis, urethritis, infections of the seminal vesicles in the male, and the vagina, uterus, and Fallopian tubes in the females. Many of these investigations may need expert examination by specialists, and radiological and pathological aid. In many chronic cases of asthma, this thorough search may fail completely to reveal this septic focus.

A word of caution is needed here:—In our enthusiasm for finding and eliminating a septic focus, too much is often expected from the patient. He is very often needlessly sent to different consultants, or is put to unnecessary trouble by getting the various pathological or radiological investigations, which frequently do

not prove of any use. Too much stress is sometimes put on a supposed septic focus, and many unnecessary operations have been performed, in the hope of affording a cure to the patient. Every doctor, with some professional experience, is well aware of such chronic cases, who have faithfully followed the advice of their doctors, suffering financial and bodily torture, without feeling any benefit to themselves. *Thus great judgement is often needed before suggesting some investigation, or operation.*

(6) *Wassermann reaction of the blood*:—This often proves useful, as an underlying syphilitic infection is frequently the cause of asthma.

DIAGNOSIS.—During an attack the disease can be easily diagnosed. In between the attacks, the history is more important. A history of attacks of paroxysmal dyspnoea is suggestive. A wheezing rhonchi is diagnostic of bronchial asthma.

In the diagnosis it is more important to know the cause of the asthma. For this a careful painstaking history, a good thorough examination of the patient, and diagnostic investigations, both radiological and pathological, are essential.

DIFFERENTIAL DIAGNOSIS.—Other causes of breathlessness, e.g. cardiac and renal asthmas, are to be differentiated. I have seen them very frequently being mistaken and treated as bronchial asthma.

SUMMARY.—1. Mechanism of asthma is discussed.

2. Methods of diagnosis are discussed, and it is shown how difficult it is to know the real etiologic factor.

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Relation of Vitamin A to the White Blood Cell Count

Hassan and his collaborators studied the blood of 60 normal male students to ascertain the relationship between the Vitamin A content of the blood, and the total and differential leucocyte counts. They found that with an increase in the Vitamin A content there was a decrease in the total number of leucocytes. Lymphocytes and monocytes increased and neutrophils and eosinophils decreased. Vitamin A deficiency was found to induce physiological leucocytosis. There appears therefore to be some relationship between Vitamin A, and the general defence mechanism of the human body.—(*Ind. Jour. Med., Res.*, 36: 1-72, 1948.)

T.N.S.R.

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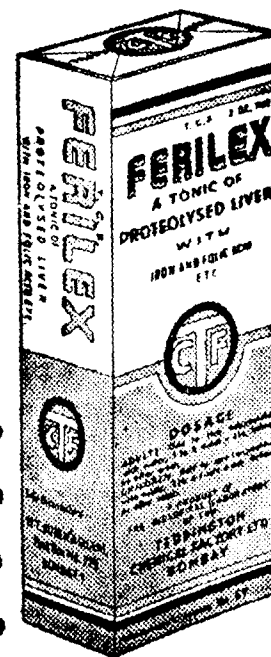
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Chrysotherapy in Rheumatoid Arthritis*

S. B. SINGH,

B.M.O., Canning College Dispensary, Lucknow University.

CAN some cases of rheumatoid arthritis be a manifestation of a mild form of tuberculosis? This most important question, raised by Poncet and his pupils, has been frequently answered in the affirmative in different countries, especially in France and Italy; whereas, in other countries, this affinity was denied (*Medical Annual*, 1933). However, it was the similarity between tuberculosis and rheumatoid arthritis which led Jacques Forestier (1928) to try the effects of gold salts in the latter disease (Chopra). Whitla says that, apart from gold, drugs are of little effect in influencing the course of the disease.

There is an ever-increasing trend in favour of gold salts in the treatment of rheumatoid arthritis. The toxicity of these products is the only deterrent to their wide-spread use (Bethea and Whitla). Cecil believes that gold salts are the best medical agents available for rheumatoid arthritis. Gold is in fact the one drug which seems to have a definite effect on the course of the disease.

Treatment of rheumatoid arthritis is purely empirical and not always successful even in the most favourable cases. Successful treatment depends largely on the promptness and vigour with which it is instituted. Early diagnosis is therefore essential for success. Darley says that, when diagnosis of arthritis is reasonably certain, therapy must be guided by the local joint condition and the patient's physical, emotional, economic and environmental situation. He adds further that gold is gaining favour as a means of checking the inflammatory process and the constitutional manifestations of rheumatic arthritis. It is not a cure but does arrest the arthritic process in a sufficient percentage of cases to warrant its use.

According to him, use of gold is justified only in cases of active rheumatoid arthritis in which the diagnosis has been definitely established. Gold is not indicated in far advanced cases in which the arthritic process has "burned" itself out or in very acute cases in which the constitutional reaction is severe. Hench believes that use of gold salts is entirely justified in progressive rheumatoid arthritis unrelieved by a reasonable but not too long period of treatment by older and safer methods and when the patient clearly understands and accepts the risk and when the physician is able to give the treatment with the necessary clinical and laboratory safeguards.

Preparations of gold employed.—To begin with these were Alloerysine (Sodium aurothiopropanol), and Sanocrysin (Sodium aurothiosulphate), the former intramuscularly and the latter intravenously. Slot and Deville (1934) used other organic preparations.

* Specially contributed to THE ANTISEPTIC

These were Solganal A, containing about 36.5% of gold and used as an intravenous injection; Solganal B and Solganal B Oleosum, both used intramuscularly, Solganal B Oleosum being an organic gold compound in an oily base. The use of an oily suspension increases the degree of tolerance resulting in gradual absorption with an even prolonged effect.

Allocripsine (Sodium aurothio propanol Sulphonate):—A course of it begins with three injections of 0.05 gm. each at intervals of 5 days, and if no reaction occurs, further injections of 0.1 gm. each should be continued weekly until a total amount of 1.5 or 2 gm. is given. Such a course takes about three months and no more than 2 gm. should be given in all and the daily dose should never exceed 0.2 gm. After the first course, the patient should rest for 6 to 8 weeks and then a second course should begin. The treatment is then continued by a series of 10 to 20 weekly injections of 0.1 to 0.05 gm. of the drug for one or two years.

Sanocrysin (Sodium aurothiosulphate):—It is administered intravenously. The initial dose is 0.25 or 0.35 gm. The second dose should never exceed 0.65 to 0.75 gm. and is given six days after the first dose. The third and the fourth doses being 0.75 gm. each, the former given on the 12th day and the latter on the 19th day after the initial dose.

Slot and Deville (1934) used other organic gold preparations, that is, Solganal A, Solganal B and Solganal B Oleosum, the last in the majority of cases and with encouraging results. The initial dose of Solganal B is 0.01 or 0.05 gm. and is given every 4th day, the increase depending on the individual condition. With Solganal B Oleosum increase may be 0.1, 0.2, 0.3, 0.4 and 0.5 respectively, provided no reaction occurs. The average total dose is 2.5 gm. per series of injections, but generally is between 1.0 and 1.5 gm. Glucose was given by mouth to avoid toxic symptoms during and even some time after the treatment. The cases were simultaneously treated with other anti-rheumatic drugs, colonic injections with normal saline and diet therapy. The majority of cases improved, though pyrexia and jaundice appeared as complications in a few cases.

At the present day the forms of gold most popular are aurothiomalate of Sodium and aurothioglucose (Solganal B Oleosum). According to Cecil of Cornell University, the dose of gold salts has been controversial, the amounts advocated by most American authorities for intramuscular administration are 10 mg. for the first, 25 mg. for the second and 50 mg. for the third and succeeding doses, at intervals of one week. Some authors advise repetition of each of the small doses once before proceeding to the maximum dose. The total amount administered in one course should be 750 to 1000 mg. To permit the body to excrete some of the accumulated gold, it is

customary to administer gold in several series or courses, with intervals of 4 to 8 weeks between. Darley's preparation of choice is aurothioglucose suspended in oil (Solganal B Oleosum). Doses are given at weekly intervals. For adults, the first four are 5 mg. each; the next two, 10 mg. and the remainder 25 mg. each. If there be no toxicity, the total course lasts 30 weeks. Gold therapy is given up if there is no improvement towards the end of the course of treatment. Improvement should, according to Darley, occur after the first ten or fifteen doses and is indicated by falling sedimentation rate, lessening of pain, subsidence of swelling and stiffness and gain in weight and strength.

The dose has been gradually reduced, so that until recently the standard maximal individual dose, according to Hench, was generally 100 mg. gold salts (about 50 mg. gold); while according to Smyth and Freyberg, the optimal maximum dose was 50 mg. gold salts.

Precautions.—The patient receiving gold should be in bed throughout the treatment. He should be kept under careful observation and the use of the drug discontinued, if there be any question of toxic reaction, and once a reaction has occurred, it is best not to resume gold therapy. Treatment should be discontinued, if there is persistent anorexia or if diarrhoea occurs. The urine should be examined for evidence of renal irritation before injection of gold, and, if it is present, treatment should be discontinued. A complete blood count once a week is done to detect gold toxicity to bone marrow. Since various schemes have all proved ineffective in preventing toxicity and since there is no method of preventing reactions, their early recognition is important. Questioning regarding untoward symptoms and complete blood and urine examinations are indicated before each dose. Treatment of reactions is at best only symptomatic and supportive in general and must be energetic if the condition is acute. It is therefore important that the interested physician must know management of toxic reactions.

When gold therapy is used, the sedimentation rate of red cells should be determined about every two or three months. Patients who react well to gold therapy usually have a rapid drop in sedimentation rate during the first course of treatment. Those who receive marked benefit from gold salts usually show striking improvement with the first course of therapy. If there is no improvement after two courses, further administration is contra-indicated.

Toxic reactions and limitations.—Certain dangers militate strongly against wide acceptance of gold therapy. A few early reactions represent hypersensitivity to gold, but most reactions occur after a few hundred mg. of gold salts have been given and represent metallic protoplasmic poisoning. According to Darley,

toxic reactions occur in 5 to 10 per cent and are apt to subside slowly over a period of several weeks. The incidence of toxic reactions has varied from as low as 8% to as high as 75%, the average being 40%, and serious in 5%.

On the basis of 3,000 cases (2,000 treated in America and 1,000 in other countries), Hench observes that usually the reactions were mild and transient, but in 3 to 11 per cent of cases (average 5%), they were serious (*i.e.* exfoliative dermatitis, agranulocytosis, thrombopenic purpura, acute entero-colitis or hepatitis), but were non-fatal. A few fatal reactions have occurred. Recent mortality rates have been between 0.38 and 0.43 per cent. In the experience of Cecil, approximately 25 per cent of cases have a rash or some other form of gold intoxication such as, stomatitis or gastro-enteritis. There have been a few cases of acute nephritis and rare instances of toxic hepatitis. Depression of bone marrow, with consequent development of hæmorrhagic purpura, aplastic anæmia or agranulocytosis has been noted in a number of cases. For this reason, when gold salts are given a blood count and urinalysis should be made at least once a month and the skin carefully watched for drug rashes.

A highly disappointing limitation in gold therapy is the comparatively high percentage of relapses. It is 50 to 75 per cent according to Cecil who holds that a maintenance dose of 25 to 30 mg. gold salts, every 2 to 3 weeks and eventually only once a month, certainly reduces this percentage. Summing up the literature of the day, Bethea concludes that a patient has about a 50 per cent chance of having no toxic reaction, a 45 per cent chance of having a serious but non-fatal toxic reaction and about one chance in 250 (*i.e.* 0.4%) of having a fatal reaction (unless this percentage is modified by early recognition of these reactions and their treatment with BAL).

Treatment of toxic effects from gold-therapy.—BAL (British anti-lewisite) (chemically, 2-3, Dimercaptopropano) is the answer to some of the gold therapy problems. Although BAL cannot be called an ideal antidote, because it has its own toxic effects, it does clear the toxic symptoms arising from treating any disease with gold preparations and itself possesses in barbiturates a cure for its own toxic effects which, however, are luckily transient and may be disregarded in view of the resultant benefit.

BAL is given intramuscularly. A 10% solution in peanut oil with 20% Benzyl Benzoate is made 1.4 to 2.0 c.c. (140 to 200 mg.) BAL is administered intramuscularly, every 6 hrs., for 48 hours and then twice-a-day for eight days. This suffices ordinarily. But in severe complications, six injections a day at four hourly intervals are given for the first 48 hours, then 4 injections on the third day and thereafter twice-a-day for ten days or until recovery.

Variations in results with gold therapy.—Good results with gold therapy in 35% of patients have been reported by Ragan and Tyson, but other rheumatologists have observed notable improvement in only 15 to 20 per cent. According to Hench (1947), most of the reports on the thousands of patients with rheumatoid arthritis treated with gold salts during the last 20 years, have favoured chrysotherapy, though drawbacks were admitted: not all patients having been benefited, many relapses and toxic reactions having been frequent and occasionally fatal. In general, 50% of rheumatoid arthritis appears to be arrested by gold therapy, another 25% shows improvement, 10 to 15% does not respond, 10 to 15% seems to grow worse and toxicity appears in 10% or less. Relapses after initial response to treatment take place in 10 to 20 per cent.

In reports of results in 2,000 cases in the United States of America and 1,000 in other countries, during the period from 1942 to 1947, the results have varied widely; but in general, about 50 to 60 per cent of patients become symptom-free or were notably relieved. Particularly interesting was a 3 to 5 year follow-up study of 142 patients by Ragan and Tyson who found that 11 per cent were not improved, 76 per cent were improved and in remaining 13 per cent complete remissions were obtained. There were 6% 5-year-cures. Relapses occurred in 75%, but 80% of these who relapsed improved under further treatment.

In summing up the recent literature up to 1947, it is concluded by Bethea that a patient treated with gold salts has about a 10 to 15 per cent chance of obtaining complete remission lasting from several months to an undetermined number of years, about a 50% additional chance of being notably improved and about a 35% chance of obtaining no significant relief.

References:

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2. R. N. Chopra.—A Hand Book of Tropical Therapeutics.
3. Medical Annual, 1933.
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5. Bethea, O. W., (1947)—Year-book of General Therapeutics.

Labyrinthine Disturbances due to Streptomycin

Moindiot *et al.* report the occurrence of vestibular and cochlear disturbances in 16 children and 34 adults treated with Streptomycin. The daily dose varied from 4 to 50 grains and treatment ranged from 12 days to 8 months. Vertigo appeared in 17 of the 34 adults on the 20th day of treatment; instability, mild nausea and a rotatory sensation occurred but no vomiting. These symptoms disappeared in 2 to 3 weeks, even when treatment was being continued. In spite of the vestibular dysfunction which persisted long after treatment was stopped, the disturbance of the equilibrium (inability of patients to maintain their equilibrium when standing on one leg) subsided in the great majority of the patients. It is suggested that the toxic effect of the drug is exerted on the central vestibular nuclei. Vestibular dysfunction seems to depend on the individual susceptibility of the patient.—(*Jour. Med. Lyons*, 29: 20-9, 1948).

T. N. S. S.

The Median Nerve Test in Leprosy*

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WHILE examining out-patients in the Leprosy Department of Stanley Hospital, Madras, I have found that in certain types of early leprosy, such as those with active or multiple or incipient or prelepromatous or maculo-lepromatous lesions on any part of the body, the median nerves medial to the cubital fossa were bilaterally tender. Not infrequently the radial nerves at a point lateral to the cubital fossa were also found to be bilaterally tender. At this stage the ulnar nerves are not usually tender or thickened, but in the later stages of lepromatous leprosy with infiltrations and nodules, the median nerves are not usually acutely tender, but the ulnar nerves are tender and/or thickened.

Thickening of cutaneous nerves in early cases of leprosy, particularly in tuberculoids, are not uncommonly met with the supra-orbital, the supra- and infra-trochlears, the great auricular and its branches, the cervical cutaneous, the supra-clavicular, the medial branch of the ulnar, the saphenous and its infrapatellar branch, the sural and the superficial peroneal. This thickening seems to indicate a localisation of the disease and is generally associated with anaesthesia over the area of nerve distribution. But tenderness of nerves with or without thickening is an indication of activity of the disease. The bilateral tenderness of the median and radial nerves when there is no lesion over their area of distribution, seems to imply that the disease is active, progressive and probably systemic; in other words, that the disease is taking a serious turn and therefore the treatment has to be undertaken with great care lest the disease is further activated. The bilateral tenderness of the median nerves may therefore be considered as an invaluable aid to determine whether the disease is tending to take a serious turn to lepromatous leprosy.

This sign is not characteristic of leprosy only, for, among some patients with signs and symptoms simulating leprosy, sent to me from the hospital wards and other departments for opinion, I was able to elicit this sign. The following are few of such cases met with:—

- (1) A case of radial paralysis of right hand only—no evidence of leprosy—had bilateral tenderness of median and radial.
- (2) An advanced case of peripheral neuritis.
- (3) A boy of 12 with hyperpigmentation of palms and soles (Leucomelanoderma)—Wassermann strong, positive.

* Specially contributed to THE ANTISEPTIC.

(4) Some well established cases of typhoid in third week.

(5) A case of lobar pneumonia examined the day after the crisis, but, in this case, only the median nerves were bilaterally tender.

The pressure to be exerted to elicit this tenderness should not be too great amounting to traumatic pressure. It is best elicited with the forearm in an extended position and then at first gentle pressure is made with the thumb and slowly increased. The Leprosy Department of the School of Tropical Medicine, Calcutta, has also reported that this bilateral tenderness of the median nerves has been observed in a certain proportion of early cases of leprosy, as well as in some well established lepromatous cases. Further investigations are being made to find out its significance. This bilateral tenderness seems to be suggestive of some factor, may be B₁ deficiency, which probably comes into play in those diseases which have a proclivity to attack the peripheral nerves, chiefly median and radial.

It is worthwhile to apply this test in the routine examination of cases, particularly fevers, and to note which cases respond to this test, and whether there is any correlation between it and the other tests, as Widal, Wassermann. Medical practitioners are requested to investigate its significance and to express their views about it through this journal.

Intravenous 'Paludrine' in the Treatment of Malaria

Chaudhuri and Chakravarti of the Calcutta School of Tropical Medicine, report on the successful use of paludrine acetate (soluble proguanil) in the treatment of 11 cases of malaria, eight of whom had *P. falciparum* infection, two had *P. vivax* and one had mixed infection. Four patients were gravely ill, four had heavy infection, and three ordinary malaria.

The paludrine acetate used was supplied in 5 cc. ampoules each containing 100 mg. of the drug. Starting with 25 mg. the dose was gradually and cautiously increased to 400 mg. in a single injection. In 4 cases it was given both orally and intravenously.

[Note.—Paludrine acetate has since been replaced by Paludrine lactate which is more soluble and less irritant. An ampoule of 2 cc. contains 100 mg. of the drug. It has been given by the authors in subsequent cases (to be reported upon later) intramuscularly with satisfactory results.]

At first, the dose was 100 to 200 mg. repeated 2 to 3 times a day at 4 hourly intervals; later it was 300 mg. and ultimately 400 mg. These patients, therefore, received by injection 300 to 600 mg. The majority of patients had either cerebral or gastro-intestinal complications and were not suitable for oral medication. A dosage of 200 to 400 mg. controlled the temperature and cleared the peripheral blood of asexual parasites in 2 to 3 days. The injections were well tolerated.—(Br. Med. Jour., 15.1.49.)

Dives into Ancient Hindu Medicine

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(Continued from page 281 of Apr. '49 issue)

VI. Aasavaas and Arishtas.

Aasavaas and Arishtas are spiritous liquors holding drug-principles in them. Aasavaas are prepared by steeping coarsely powdered drugs in a mixture of water, honey and treacle¹ and allowing all to ferment under the influence of Woodfordia Floribunda² (*Dhadhaki*) for a period of one to six months in air-tight earthen jars. Arishtas are prepared in a similar way; but, a part of the drug is made into decoction which takes the place of the water in the Aasava; the other drugs in coarse powder are steeped in the mixture of the decoction honey and treacle, and fermentation is allowed to take place as before. It will be observed that there is practically no difference between the Aasavaas and the Arishtas; while in the former a kind of *sita-kashaya* (cold infusion) with drugs in tact is used, in the latter a decoction of some drugs and coarse powder of others are used. Rationally, then, there can be no difference between the two in their medicinal properties; and that the two names are used indiscriminately—aasavaas being called sometimes as arishtas, and arishtas as aasavaas³.

Preparation of Aasavaas:—The drugs, finely cut or smashed, are steeped in pure cold water in suitable earthen jars, and, according to the practice of some, kept undisturbed for 24-48 hours (preferably in sun and dew). Honey and treacle or its like are added (along with grapes and flowers of Bassia Latifolia, when desired). The ferment *Dhadhaki*—Woodfordia Floribunda—is thrown into the mixture, and the mixture is stirred well. The jar is bunged air-tight and buried two feet in earth or *Dhaunya* which usually is paddy. Fermentation is allowed to proceed for the

1. Treacle is sweetening agent and a fermentable product. Its place can be and is sometimes taken by jaggery, coarse sugar, crystalline sugar or candy. Treacle, jaggery (and coarse sugar too to some extent) give a high colour to the preparations while the last two give it a light colour. It is also said that preparations containing treacle and honey lessen constipation but are prone to cause "bolly-ache" in some. Grapes and flowers of Bassia Latifolia are added by some to fortify treacle etc. both as sweetening and fermenting agents.
2. In preparations where fermentation is otherwise easy, Woodfordia Floribunda is not added. *Dhadhaki* probably takes the place of modern yeast.
3. "Draveshu chira-kalastham dravyam yath-santhitham bhavel |
Aasava-arishtha-bhedaisthu-prochyathe bhesajochiham |
Yada-pakwa-oushadha-ambubhyaam sidham madyam sa aasava |
Arishta: kwatha-sadhya: syath-thayor-manam palonmitham." —Sarangadhara.

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stated period (and for at least three months in un-specified cases). The jar, after that, is scooped out and opened; the scum, if any, is removed, and the contents are filtered. To the filtrate are added pieces of strychnos potatorum in sufficient quantity to remove the un-dissolved particles therein; the particles, sticking to the pieces and sinking to the bottom with the pieces, firmly settle then as sediment or dregs. When this process is complete, the clear fluid is drawn off and strained through flannel (if found necessary).

Preparation of Arishtas:—The decoction is first prepared by boiling the stated drugs in stated amount of water and reducing the contents to the stated quantity⁴. When cool⁵ the decoction is filtered into the earthen jar containing the other drugs in coarse powder. Honey, treacle etc. are added, and further operations are continued as in the preparation of aasavaas.

Unspecified Aasavaas and Arishtas:—The proportions of ingredients here are to be as under: Fluid, one drona (256 palas); Treacle, one thula (100 palas); honey, half the amount of treacle (50 palas); Drugs, one-tenth part.⁶ The verses are not clear on the drugs. *Prakshepam*, according to Apte's Sanskrit-English Dictionary, means "throw; cast; the sum deposited by every member of a Commercial Corporation." The word gives no difficulty in Aasava when all the drugs are steeped in water. In Arishta, however, part of the drugs is used for decoction and only a part of them is cast (or becomes subject to *prakshepam*) in the fluid; doubts, hence, arise if the proportion of one-tenth part mentioned above refers to the whole of the drugs used in the preparation or only to that amount that is used for *prakshepam* or steeping in at the end. A second uncertainty is about the drugs being one-tenth part of WHAT; are the drugs to be one-tenth of the fluid or the treacle or the honey or the entire mixture of fluids? The matter is left here for elucidation by someone well-versed; and the following table (*vide page 678*) may help the student to form his own conclusions.

Note:—In the Aasava-arishtas enumerated above, it is noteworthy that while all the aasavaas except loha-asava contain grapes, only four among the arishtas—Draksha, Kutaja, Abhaya and Dasamoola—contain raisins; and, the raisins in the arishta are tacked on to the drugs for decoction in the respective prescriptions.

4. In unspecified cases, raisins and flowers of Bassia Latifolia (if elected to be used) may, with advantage, be tacked on to the drugs for decoction and boiled along with them.
5. Honey, in Ayurveda, is incompatible with things hot or warm; it should always be added when the thing it is to be added to is cool.
6. "Anuktha-maana-arishieshu drava-drone thulaam gulam |
Kshoudram kshipet gulad-ardham prakshepam dasama-amakam." —Sarangadhara.

	Name of Asava	Drugs	Water	Drugs: Water	Reduced to	Treacle	Honey	Treacle & Honey	"Dhaa-dhaki"	Grapes
1	Punarnava-asava	20 palas	2 Dronas 512 palas	1 in 25 roughly	—	1 Thula or 100 palas	50 palas	150 palas	16 palas	20 palas
2	Usoera-asava	21 palas	"	1 in 24 roughly	—	"	"	"	"	"
3	Aravinda-asava	23 palas	"	1 in 22 roughly	—	"	"	"	"	"
4	Patraanga	20 palas	"	1 in 25 roughly	—	"	"	"	"	"
5	Pippali	12 palas	"	1 in 43 roughly	—	300 palas	nil	300 palas	10 palas	6 palas
6	Loha-asava	44 palas	"	1 in 12 roughly	—	100 palas	64 palas	164 palas	20 palas	nil

	Name of Arishta	Drugs for Decoction	Water	Drugs: Water	Reduced to	Treacle	Honey	Treacle & Honey	"Dhaa-dhaki"	Drugs Steeped
1	Draksha	50 palas	512 palas	1 in 10 roughly	128 palas	200 palas	nil	200 palas	nil	8 palas
2	Asoka	50 palas	512 palas	1 in 10 roughly	128 palas	100 palas	nil	100 palas	8 palas	5 palas
3	Amritha	100 palas	512 palas	1 in 5 roughly	128 palas	100 palas	nil	100 palas	nil	12½ palas
4	Bala	100 palas	512 palas	1 in 5 roughly	128 palas	150 palas	nil	150 palas	8 palas	5 palas
5	Kutaja	85 palas	512 palas	1 in 6 roughly	128 palas	50 palas	nil	50 palas	10 palas	nil
6	Abhaya	85 palas	512 palas	1 in 6 roughly	128 palas	50 palas	nil	50 palas	1 pala	10 palas
7	Khadira	66 palas	1024 palas	2 in 31 roughly	128 palas	50 palas	100 palas	150 palas	10 palas	5 palas
8	Dasamoola	80 palas	576 palas	1 in 7 roughly	144 palas	100 palas	8 palas	108 palas	7½ palas	5 palas
9	Vilanga	20 palas	1024 palas	1 in 51 roughly	128 palas	nil	150 palas	150 palas	10 palas	nil
10	Deva-draavya	120 palas	1024 palas	2 in 17 roughly	128 palas	nil	150 palas	150 palas	8 palas	10 palas

Some Spiritous Liquors in Ayurveda

A. Seedhu (or Seeththu) is got through the fermentation of grapes and other sweet fruits. *Aasavaas* of this are called *Seetha-rasa-seedhu*, while their *Arishtas* are termed *Pakwa-rasa-seedhu*. (Seetha—cold; Pakwa—cooked)⁷.

7. "Jneya: seetharasa: seethur-apakwa-madhura-oashadhai:
Sidha: pakwa-rasa: seeththu: sam-pakwa-madhura-draavyai:"

B. Vaaruni is got through the fermentation of juices from the palmyra tree, the date-palm and the like⁸.

C. Suktha is the product obtained when crushed tubers, roots, fruits, barks etc. of plants are steeped along with fats and saline substances in water or other fluids and kept sufficiently long. The Suktha wherein solution of sugar or jaggery has been used is called Guda-suktha; Ikshu-suktha is the product got where juice of sugarcane has been used, while Mridweeka-suktha is the one obtained where the fluid has been grape juice^{9 and 10}.

D. Chukram is the term applied to the product got when, in the process of fermentation, honey and other sweet things entirely lose their sweetness and become fully sour¹¹.

The author, in his Chakradatta, gives the following recipe for *Swalpa-chukra* (Chukra the minor, i.e. chukra with few ingredients): In a clean earthen jar, jaggery one part, honey two parts, kanchi-kam (see below) four parts, and whey eight parts are put. The jar is buried in paddy for three days. The product in the jar on the fourth day is Suktam or Chukram. (For *Brihat-chukra*, the reader is referred to *Grahnia-adhikara* in Chakradatta)¹².

It will be observed in the above that the author of Chakradatta makes no distinction between Suktam and Chukram!

E. Grain-liquors:—If Seedhu may be compared to wine or 'fruit-wine,' Vaaruni to sweet toddy, and Chukram to vinegar, the grain-liquors may be compared to malt-liquors like gin or beer.

(1) *Sura*:—This is from the grain paddy. When well-boiled rice is steeped in water for sufficiently long, it undergoes fermentation producing what is termed *Sura*. *Sura*, if the vessel is kept undisturbed, settles in layers of different density, the density of the layers increasing from above downwards. The top-most

8. "Yath-thantha-kkarjoora-rasai: saadhitham saa hi vaaruni."
9. "Kanda-moola-ppalaadeeni sa-sneha-lavanaani cha
Yathra drave-abhisheeyathe thal suktham-abhidheeyathe
(Gulaambura sa-thailena kanda-saaka-ppala's-thithaiha
Saadhitham chi-amlatham yaatham guda-suktam prachakshathe
Kram-ema-ikshu-sooktham syann-mridweeka-sambhavam thithatha." —Sarangadhara.
10. The following on spiritous liquors is from Bela-thanthra:
"Paanasam drankha-maadhookam kkaarjooram
Thaalam-aikshakam maadhweekam tamka-maadweekam
Maireyam naalikerajam samaanani vijaneeyan
Madhyam ekadasaivathu dwaadasanthu
Sura-madhyam sarvashaam-adhamam smritam "
11. "Vi-nishtam-amlatham yaatham madhu vaa madhura-drava:
Vi-nishta: sanditho yaathu thaah-chukram-abhidheeyathe." —Sarangadhara.
12. "Yam-masthu-adi suchow bhaande sa-guda-kshoudra-kaanchikam
Dhanya-vasow thri-raathrastham suktam chukram thad-uchyanthe
Dwijunam guda-madhu-saranala-masthu kramaaditha."

layer, called Prasanna, is limpid and whey-like. The successive layers from Prasanna are the *Prasanna*, the *Kaadambari*, the *Jagala*, and the *Medaka*. Sura, when deprived of all its water through boiling, leaves the amorphous residue *Sura-bijam*, also called *Kinnucam*.¹³

(2) *Thushaambu* is the product of fermentation got through steeping in water for sufficiently long Yava (Indian Barley) that has been split; the Yava here is freed of chaff, but is fully preserved of its bran.¹⁴

[Thushambu resembles Thaandula-ambu which is the product obtained by steeping for sufficiently long rice grain with bran in water.]

(3) *Sow-veeram*:—This is practically the *Sura* with Yava instead of rice, and holds the same relation to Sura as Thusaambu to Thaandulaambu. It is got by steeping in water well-boiled Yava that has been freed of chaff and bran.¹⁵

(4) *Saandaaki* is the product obtained when a mixture of mustard, garden radish, and grain (Yava or Paddy) is boiled in water and allowed to ferment.¹⁶

(5) *Kaanchikam* is the sour liquid got through fermentation of gruels prepared with pulses and grains of "inferior" order—e.g. paddy grown in rainy season which ferments easily.¹⁷

Kaanchikam or Dhaanya-amlam is a speciality in Ayurvedic Pharmacopœia. Different authors give different recipes for this. In fact, Thaandula-ambu and Thusha-ambu, Sura and Sowveera, and Saandaaki—all are varieties of Kaanchikam. The following are from Chakradatta and Yogaratnaakara respectively.

(a) *Kaanchika*, as in CHAKRADATTA:—

"*Vaatasya dadyaad-yava-sakthukaanaam prithak prithak
thu-aadaka-sammitham |
Madhya-pramaanaani cha moolakaani
dadyaach-chathu-shashti-sukalpithaani |
Drone-ambasa : plaavya ghate sudhouthe dadyaad-idam
bhesaja-jaata-yuktam |
Kshaara-dwayam thumburu-basthagandha-dhaneeyakam
syad vada-saindhavam |*

13. "*Pari pakwai-anvi-samgh dha samulppannam suram jagu :
Sura-manda : prasanna syaal thathiha : kaadambaree ghanaa
Thal adho jagalo jneyo medako jagalat ghanaa:
Pakwa-1sow Hritha-suara : synal suraa-beejam-cha kinnwakam.*"

14. "*Thushaambu san thitham jneyam aamair-vidalithair-yavai.*"

15. "*Yavassu-nis thushai : pakwai : sowveeram samhatham bhavel.*"

16. "*Saandaakee sandhitha jneya moolakai : sarshapa-aadhibhi.*"

17. "*Kul-misha dhaanya mundaadi santhitham kaanchikam vidu.*" — Sarangadhara.

*Souvarchalam Hingu-sivaatikaam-cha chavyam-cha
dadyaad-dwipala-pramaanam |
Imaani cha-anyaani palon-mithani vi-jarjjaree-krithya
ghate kshipel-cha
Krishnaam ajaajeem-upakunchikaam cha thaththa-asureem
kaavavi-chithrakam-cha |
Paksha-sthithihoyam bala-varna-deha vaya :
karotheeva bala-pradascha |
Kaan jeerayaameethi yatha : pravrittha sthal kaanchikethi
pravradanti thajnaa :
Vaataamayaan-aasu nihanthi sarvaan
sam-sevya-maanam vividhuan naraanaam : | "*

The following are put in a perfectly clean jar.—(1) Parched yava, four prasthams; (2) yava-meal, four prasthams; (3) medium-sized garden-radish, 64 palams; (4) water, 16 prasthams; (5) yavakshaar (impure Pot. Carbonate), Sarjjikakshaar (impure Sod. Carbonate), Tumburu (?), Bastagandha (?), Dhaneeyakam (Coriander), Vitlavana, Saindhava salt, Sowarcha lavana, Hingu (asafoetida), Sivaatikaam (?), and Charyam (root of wild pepper), each two palams; (6) Krishna (long pepper), Ajaaji (white cumin), Upakunchika (Black cumin), Asur (mustard), Kaaravi (sweet fennel), and Chithraka (Plumbago Zeylanica), each one palam. The jar is bunged air tight, and buried in paddy for a fortnight. After this, the Kaanchikam can be drawn off.

(b) *Kaanchika*, as in "Yogaradnaakara".

"*Aseethi-vaata-rogeshu dhaanya-amlam vini-vesayel
Subhemuhoortha nakshatre swa-grihe kalasam nyasel
Thasmin-ethaamscha sambhaaraan nikshipen-mathiman bhishak
Thandulasya dasa-prasthaan priththukasya thaththaiva-cha
Kulaththasya thaththa laajaam-chathwaarimsath-idi-ecritam
Aadakam kamgu-beejaanaam kodravasya cha thandulaal
Chathush-prastham kshipel paschaal dwi-prastham naagavasya thu
Kamsam dantasataanaam-thu deepyakaal kuduba-ashtakam
Ushnodakasya dwi-sata-prastham kshipthwa thiras-kriyaam
Thasya thad-vadanam badhwaa parithognim pravarthayel
Aa-sapta-divasaad-evam-ashtama divase puna :
Dhanya-amlam-asmaad-aakrishya punar-ushnodakam kshipel
Aathuram snigdha-sarvaamgam sadaa thathra-avagaakayel
Maathraanaam shal-sahasraanee parama : kaala uchyaathe
Loko-upakaaram-anwichchan vakshel baktya thu kumbhikaam
Poojayed-vidhivad-viprair-ayam vidhir-anuththama : "*

The following are bundled separately.—(1) Rice (preferably the variety called *shashtikam*, 10 prasthams; (2) beaten rice (rice moistened, parched and flattened), 10 prasthams; (3) horse gram,

10 prasthams; (4) parched paddy with chaff etc. removed, 40 prasthams; (5) Deccan grass (*Tinai* in Tamil), 4 prasthams; (6) Kodrava (*Varagu* in Tamil), 4 prasthams; (7) dried ginger in pieces, 2 prasthams; (8) limes cut into four, 4 prasthams; and (9) Ajowan, 2 prasthams. A clean and convenient spot in the physician's house is chosen, and a proper and well cleansed pot is placed over the spot at an auspicious moment. 200 prasthams of hot water are poured into the pot, and the bundles are put separately and successively beginning with that of rice and ending with that of Ajowan. The mouth of the pot is covered with cloth and bunged with mud. Fire is set all round the pot so that the pot becomes uniformly surrounded by fire and heat. This is maintained for 7 days; and on the eighth day, the kaanchikam becomes available for use. From the eighth day onwards, any amount of dhaanya-amla can be drawn off; but, every time a quantity is drawn off, an equal measure of hot water should be added immediately to the contents in the pot. The fire should never be allowed to extinguish; it must be smouldering always.

Dhaanya-amlam is, par excellence, the drug in Ayurveda for the 80 disorders born of vitiated vaata (i.e. neuro-circulatory disturbances). The patient or the part is bathed with the prescribed Sneha, and immersed or laid in Kaanchikam for a period never to exceed 6,000 mathraas—a mathraa being roughly a second.

Kaanchikam is costly and laborious to prepare; only a handful can afford to spend over it and for it. It behoves, therefore, that it should receive the care, love and devotion for all rare things, and be regarded as a "public-utility-preparation" serving all the needy alike. The ancients, in their pristine wisdom, have hallowed it and prescribed special prayers for it to be offered during the twilight and with light and incense before the *Kumbha*. One such prayer is "O, Lord Dhanwantari, thou that art of Lord Vishnu and hath risen out of the Ocean of milk, I invoke thee, the abode of generosity, to be by the side of this kumbha, blessing and guarding it."¹⁸ Also, they, the simple folk that they have been, have directed that, at least once every month "holy" people should be invited to offer special worships to the *kumbha* (the pot). The scientific wiseacres of today may deride such prayers of their unsophisticated ancestors; but, the over-ridings of the Divine Artist are beyond the ken of even these.

VII. Bhasmaas and Kshaaraas (Ashes)

The ashes of vegetable origin are given the appellation of kshaaraas, while those of animal or mineral origin go by the name of Bhasmaas. Strictly speaking, both are burnt substances, and so

18. "Paya : poyodher-udithas-sa-dev, dhanwantarir-vishnukala
-avatheeru : dine dine asmin mama divya-bhande
dayaa-nidhi sannidhim-aathanothu"

are Bhasmaas; in fact, the *Vibhuti*, the holy ash got through burning cow-dung, is known as bhasmam and not kshaaram. Perhaps, the fact that the ashes of vegetable drugs are all (with rare exceptions) alkaline and corrosive like Yavakshaar and Sarjiikakshaar induced the ancients to call vegetable ashes too as kshaaraas.

Preparation of mineral or animal 'ash' is a laborious process; and the former entails *Sodhana* (purification of ore), *Maarana* (destruction of deleterious elements therein), etc. Each mineral (*Dhaatu*) and 'sub-mineral' (*Upa-dhaatu*) has its own processes of *Sodhanam maaranam*, etc.; and without a consideration of these, no discussion on mineral ashes will be complete, and moment now is not propitious for that. Hence, kshaaraas alone will be dealt with here, and other ashes reserved for full discussion in the volume *Pharmaco-graphy*.

Kshaaraas :—The part of the plant whose ash is required is dried, comminuted and burnt to ash (as advised in the prescription, in specified cases). It is put in a mud vessel and mixed with four times its water; the mixture is well agitated and set aside undisturbed for one full night; next morning, the clear portion is strained and evaporated over a slow fire. The evaporation is carried on to completion leaving a dull white powder behind when the kshaara is required for sprinkling over wounds or for use as adjuvant to other medicines per os. Where, however, the drug is required for use per os per se or for brushing or painting over wounds, growths, etc., the evaporation is partial and is stopped when the fluid has acquired a creamy or semi-fluid or the required consistency.¹

The above is as found in Saarangadhara Samhita.

Chakrapaani Kavi, however, makes some differences in the preparation of Kshaaraas for internal use. He advises mixing of the burnt ash in six times water instead of four, although he admits that four times are suggested by others. Also, he advocates the filtration of the ash-cum-water mixture twenty-one times in succession before putting the product to evaporation, instead of keeping the mixture undisturbed for over-night and straining the clear fluid next morning for evaporation.²

1. "Kshaara-dravyasya kaashitaani sushkaani-agninow pradeepayel
Neethuwa shad-bhasma mril-paathre kshipithwa neere chathur-gune
Nir-mardhya dhaarayel-raathrow praathar-achcham jalam nayel
Than-neeram kwaathayed-vahnaw yaavad-sarvam visushyathi
Thatha : paathrath-sam-ullikkya kshaaro graahya : sithaprabha :
Ohoornaabha : prathisaarya : syaal peya ; syaal kwaathihaval-sithitha :
Ithi kshaara-dwavam dheemaan yukta-kaaryeshu yojayel."
2. "Naati saandro naati thanu : kshaara-paaka udaahrida :
Durnaamaka-aadow nirdhishta : kshaaro-ayam prathisaarana :
Paseyo yasthu gulma-aadow tham vaaraan-eka-vimsathim
Sraavayel shad-gune thoye ke-shid-aahus-chathurgune."

Kshaaraas intended for use as cauterising agents are first tested for their potency. If the drug, when applied to bark-free stalk of castor-plant, produces 'eschar' on the spot within 100 seconds (?)—'vaak-sathaal'—, the kshaara can be considered as *uththamam* or excellent³.

* * *

VIII. Arkaas and Draavakaas (Distillation Products)

These seem to hold but a minor place in Ayurveda proper, as renowned and popular books on Ayurveda contain no or but sparse information on them.

The following are verbatim reproductions from Dr. Nadkarni's *Indian Materia Medica*:—

Arkaas are made by soaking drugs in water for 24 to 48 hours and then extracting their essence by distillation; the essence thus obtained is *Arka*.

Draavakas or distilled mineral acids—several formulae are given in different works for their preparation. A number of mineral substances or salts are heated in a retort and the distilled fluid collected in a glass receiver. The acids are tested by their property of dissolving a Cowrie or shell thrown into them."

For full information on these, the keen reader is referred to the book *Arka-prakaasam* and like ones.

3. "..... *Eranda-ju-nalam-esha dahathi kahaaro varo vaak-sathuul*
Praayus-thri-bhaaga-sithe-asmin-achcha-paichchilyarakathai
Sam juyate thadu eravyum kahaara-ambo graahyamishyathe."

—Chakradatta, Arso-adhikara.

(To be continued)

Pneumonic Plague

Huang *et al.* report the case of a laboratory worker suffering from proven primary pneumonic plague who recovered after treatment with Sulphadiazine, Sulphamerazine, Penicillin and Streptomycin. He had a stormy and protracted convalescence. Sulphadiazine was given to 15 contacts (6 gm daily for an adult) prophylactically for a week and 3 gm. daily to 49 others who were less intimate contacts. None of these 64 contracted the infection.—(*Amer. Jour. Trop. Med.*, 28, May 1948.)

Pneumonic Plague

Favarell, Carriere, and Chartes, have described 3 cases from Madagascar in 2 of which Sulphapyridine effected a cure; in the 3rd case of a boy aged 9, whose father and mother succumbed to pneumonic plague, Sulphapyridine had been given prophylactically to the boy in doses of 2 gm. daily for 6 days before the onset of symptoms; with the development of fever pulmonary symptoms and the demonstration of plague bacilli in the sputum, 2.5 gm. of Sulphathiazole was given daily for 9 days. The boy became normal in 9 days and was convalescent.—(*Bull. Soc. Path. Exot.* 41: 7-7.48.)

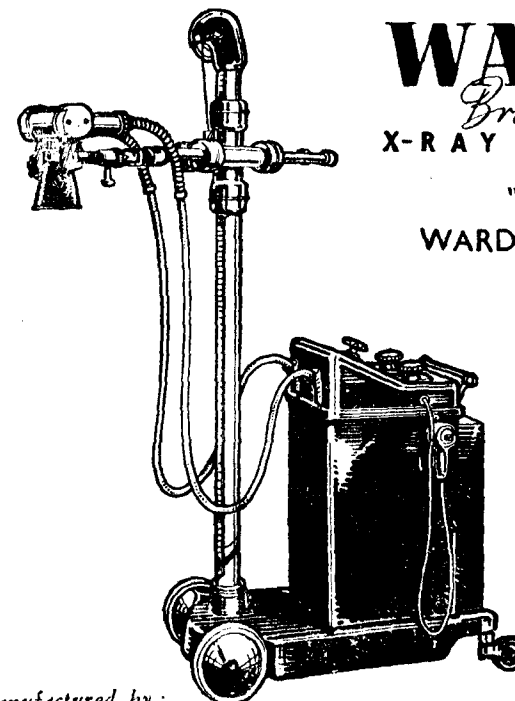
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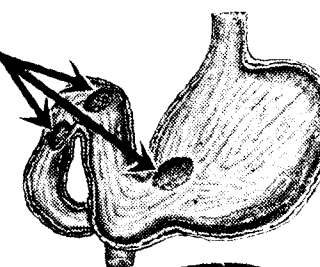
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Streptomycin and its Uses in T.B.

DURGADAS GHOSHE, L.M.F., I.T.M. (cal.),

M.O. Namring T.E., P.O. Rangli, Rangliah, Darjeeling.

STREPTOMYCIN is not the complete treatment of tuberculosis, nor is it useful in all types of cases. Why not is a very difficult question to answer. Some cases of pulmonary Tuberculosis respond to treatment very quickly and others do not and it is generally seen that cases having temp. ranging from 102°—103°F came down with the first ten day's of injections, and symptomatic improvement as evinced by decline of temperature, lessening of cough, improvement of appetite and increase in weight were seen in almost all the cases with the first month of treatment.

But for the actual cure of the disease it should be continued for several months at least, not less than 42–180 days.

As only a part of treatment, at a certain stage, and must be regarded as ancillary to bed rest, Sanatorium regime and in some cases of collapse therapy and not a substitute for them.

Dose :—One grm. of Streptomycin dissolved in 6 cc. of distilled water with 2 c.c. of 1 % Procaine in divided doses was given morning and evening with reversible toxic reactions which may be as follows—vestibular disturbances, skin rashes, loss of appetite.

Neurotoxic or vestibular disturbances.—Giddiness, nausea, vomiting, tinnitus, deafness and complete deafness if the drug is not stopped. (If reduction in hearing develops, Streptomycin should be discontinued immediately unless the infection is customarily fatal in the absence of antibacterial therapy). Heat sensation in the head according to the dose of the drug moderate or severe.

Usual method of administration is the intramuscular route, being the most common, but intrathecal, intraperitoneal; aerosol therapy methods also can be done in suitable cases. Fifteen minutes after a single intravenous injection of 600 mg. the concentration in the blood is from 30–35 micrograms per cubic centimetre. The concentration decreases over a period of six hours to about 5 micrograms per cubic centimetre. Oral administration of 1 grm. daily does not produce significant concentrations in the blood due to poor absorption. There is good retention in C.S. fluid given intrathecally. By nebulization daily doses of 500 mg. do not result in significant concentration in the blood.

The *distribution of the drug* is abundant in the blood serum, kidney, and gall bladder, not negligible in liver and spleen. Also transmitted through the placenta into the fetal circulation. Major part of the excretion is through the kidneys. Renal toxicity may occur particularly in cases which had renal disease prior to Streptomycin therapy. Leucopenia, neutropenia, and agranulocytosis should be kept in mind.

Indications.—According to Fabber and Eagle, the indications for Streptomycin are—(1) Laryngeal; and (2) endobronchial tuberculosis; (3) miliary tuberculosis; (4) tuberculous meningitis; (5) osseous tuberculosis; (6) tuberculous lymphadenitis; (7) sinuses; (8) fistulae; (9) exudative tuberculosis; (10) pulmonary lesions not responsive to conservative therapy; (11) in genito urinary tuberculosis; and (12) tuberculous enteritis and peritonitis. Streptomycin may have a place in treatment as a palliative medication.

Marked fall in temperature may be seen in neglected cases but generally the temperature declines to normal within the first ten days of injections. There was no change in rate of pulse in average cases in the early stage of treatment and the abnormally high rate of respiration can be seen in some cases to be normal within a very short time.

Hæmoptysis was seen unchanged and its usual treatment with calcium, serum, congo-red, AP etc. should be undertaken.

The drugfastness of the microbacterium is variable in different cases of T.B. and it is very difficult yet to give a real conclusion unless thousand cases have been kept under observation for a long time to add valuable information to our knowledge of Streptomycin in therapy in pulmonary and other tuberculosis.

Pulmonary tuberculosis.—Marked resolution generally occurs in soft, nodular, and exudative type of infiltration of recent origin. Consolidations and confluent infiltrations did not show the same response. Fibrocaseous lesions did not show any regressions. Small cavities did close but cavities of large size were not affected. Walls appeared to be thinner and most of them did not show any fluid level as they did before treatment. (*Indian Medical Journal*).

Diarrhoeas.—Several times due to tuberculous intestinal ulcers and enteritis, diarrhoea was seen to be stopped within the first 10 gm. of injections. Tenderness in the right iliac region due to caecal infection was less and palpable intestinal glands were not reduced in size, nor very effective in glandular T.B. cervical and axillary such as serujullacs where the drug perhaps cannot reach sufficiently to act on the organisms.

Tuberculous meningitis and miliary tuberculosis.—A high dose and prolonged treatment are recommended and the route should be both intramuscular and intrathecal as it does pass the blood brain barrier, and 0.5 gm. should be given intramuscularly 4 times for its response to treatment. Intrathecal injection should not exceed 20–50 mg. and concentration of drug should not be greater than 50 mg. per c.c. Severe neurological reactions are occasionally observed after intrathecal administration. A dose of 20–50 mg. in 5 to 10 c.c. of Isotonic Chloride solution every 24 to 48 hours is usually adequate. Since the drug is absorbed slowly from the subarachnoid space any excessive pressure within the subarachnoid space should be released and injected slowly.

For intrapleural and intraperitoneal injections 0.5 to 1.0 gm. may be dissolved in 20 to 50 c.c. of Isotonic Sodium Chloride solution. For nebulization and inhalation 25 to 50 mg. per c.c. of Isotonic Sod. Chloride solution may be used. For 50 mg. per c.c., 1 gm. should be dissolved in 20 c.c. of the solvent.

Laryngeal tuberculosis.—Apart from the usual intramuscular injections, aerosol therapy or simple throat sprayer can be tried. 1 gm. of Streptomycin dissolved in 8 c.c. of water can be sprayed by the patient himself or herself while the patient should be kept on breathing normally. Whole of the 8 c.c. can be sprayed within 24 hours or as advised before. Streptomycin resistant tubercle bacilli develop from 6 to 12 weeks after the commencement of treatment and the resistance may be enormous and is not lost by subsequent generations of bacteria, should always be kept in mind, in breaking and continuing the medicine in cases of T.B.

Commercially absolute purity, minimum pain on injections, uniform potency and decreased toxicity should be ascertained for these reasons. Dihydro-Streptomycin Sulphate is a highly purified product chemically derived from Streptomycin by addition of hydrogen in the sulphate form, less irritating and less painful and toxic.

Demerits of Streptomycin uses should always be seriously considered in all severe cases of T.B.

(1) A definitely toxic drug.

(2) When the disease progress is severe, the prognosis grave and use of Streptomycin indicated, the toxicity hazard is not comparatively great but when the disease progress can be controlled by other means or when Streptomycin has shown little promise in similar cases, the toxicity hazard cannot be taken lightly.

(3) With prolonged course of Streptomycin strains of tubercle bacilli frequently appear which are resistant to Streptomycin. This resistance to Streptomycin is prolonged; a return of sensitivity has not been reported (Fabber and Eagle).

(4) Long continued, costly treatment.

(5) Merely an adjunct to treatment of tuberculosis and patient given all the supportive measures of modern tuberculous therapy.

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Practical Points in Poisons

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I. Special Antidotes

For carbolic acid	= Epsom or Glauber's salt.
For arsenic	= Sodium Thiosulphate (I.V.)
For Morphine	= (a) Pot. Permanganas grain 10 to a pint of water (for washing out stomach). (b) Atropine Sulphate.
For Strychnine	= Sodium Amytal.
.. Cyanide salts	= Methylene Blue 50% of a 1% solution (I.V.)
.. Mercury	= Sodium Thiosulphate (I.V.) and by mouth white of egg and milk.
.. Wood charcoal	= Iodides, Apomorphine.
.. Phosphorus	= Copper Sulphate.
.. Mushrooms	= Atropine Sulphate.

II. Diagnosis Note on Poisoning

- (1) Drugs generally given for homicide :—Arsenic, antimony, aconite, opium and strychnine.
- (2) Drugs generally used for suicide :—Carbolic acid, oxalic acid, prussic acid and opium.
- (3) Drugs used as abortifacients :—Ergot, quinine, Spanish fly, oleander, colocynth.
- (4) Smell of drugs on breath :—Prussic acid, laudanum, carbolic acid, ammonia, chloroform.
- (5) Mouth bleached :—Carbolic acid and ammonia.
- (6) Patient vomiting :—Arsenic, antimony, digitalis, aconite, ammonia and phosphorus.
- (7) Patient purged :—Digitalis, arsenic and corrosive sublimate.
- (8) Cramps :—Lead.
- (9) Patient paralysed :—Aconite, conium, gelsmium and physostigmine.
- (10) Pupil dilated :—Belladonna, hyoscyamus, opium (late stage), alcohol, aconite and chloroform.
- (11) Pupils contracted :—Opium (early stage), physostigmine, chloral, carbolic acid.
- (12) Patient collapsed :—Carbolic acid, aconite, arsenic, antipyrin.
- (13) Patient comatose :—Opium, alcohol, chloral, chloroform.

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(14) Patient cyanosed :—Acetanilid, carbolic acid.

(15) Patient delirious :—Belladonna, hyoscyamus, alcohol and cannabis indica.

(16) Patient tetanised :—Strychnine, antimony and arsenic.

(17) Skin moist :—Opium, aconite, alcohol and tobacco.

(18) Skin dry :—Belladonna, hyoscyamus and stramonium.

Treatment

EMETICS.—When an emetic is ordered, one of the following should be given unless a special emetic is mentioned.

(1) Mustard and water :—A tablespoonful of mustard stirred up in 2/3 of a tumbler of tepid water.

(2) Sulphate of Zinc :—gr. 30 dissolved in half a tumbler of warm water.

(3) Warm water given copiously.

(4) Common salt :—Two tablespoonful in 8 oz. of tepid water.

(5) Tr. Ipecacunha (Vin Ipa) ½ to 1 dr.

Demulcent Drinks

When demulcent drinks are ordered one or more of the following may be used : (1) Albumin water or white of egg ; (2) barley water ; (3) milk ; (4) compound almond mixture ; and (5) olive oil.

Special Methods of Treatment

1. *Carbolic acid or Phenol* :—It burns the mouth and the entire gastro-intestinal tract. It is quickly absorbed and produces striking cyanosis. Respiratory failure causes death. In less severe cases, rapid pulse, clammy perspiration and the general picture of shock are noticed.

TREATMENT.—Epsom or Glauber's salts are the antidotes. Brandy and Ether may be used freely for the stimulating effect.

2. *Arsenic* :—White arsenic-symptoms produced rapidly within half to one hour. Initial symptoms are dryness and constriction of the throat, difficulty in swallowing and discomfort in the epigastrium, nausea and vomiting. Flocculent diarrhoea, suppression of urine, dizziness, headache and pain in the muscles follow. Collapse and coma precede death which may occur in 24 hours or at least in two or three days. The antidote for arsenic by mouth is the Hydroxide of Iron which may be given in unlimited quantities. It oxidises the arsenous acid into an arsenic compound and forms iron arsenate which is not so readily absorbable and, if absorbed, is not so easily ionised and therefore less poisonous. It

must be removed by repeated lavage. When arsenical dermatitis sets in Sodium Thiosulphate should be employed as follows :

3 gram Sodi. Thiosulphate in 10 cc. . .	1st day
45 " " " I.V. . .	2nd "
6 " " " " " " " " " " " "	3rd "
9 " " " " " " " " " " " "	4th "
1.2 " " " " " " " " " " " "	6th "
1.8 " " " " " " " " " " " "	8th "

1 gram is used by mouth daily throughout in the course of treatment by injection.

3. *Mercury*:—Milk and eggs are immediate antidotes followed by stomach tube or emetic immediately. Mag. Carb. by mouth. Sodium Thiosulphate I.V. injection (gr. 15 in 10 c.c. distilled water).

4. *Morphine*:—Opium poisoning calls for lavage of the stomach with Pot. Permanganate 1 in 1000 solution of grs. 10 to a pint of water. Physiological antidote is Atropine. The great danger is that the patient will fall asleep and that respiratory failure will set in. Coffee should be freely administered. The fatal dose is 1 gr. The fatal period is from 1 to 12 hours. If respiration could be maintained for 24 hours recovery usually ensues.

5. *Strychnine*:—Sodium Amytal is the drug which has most success as an antidote. Given I.V. in doses depending on the amount of strychnine, suffering and the severity of convulsion etc. Initial I.V. dose is $7\frac{1}{2}$ gr. in nearly any strength of solution and always indicated as a preliminary. One patient is recorded of having received 1.8 grain of the drug in a period of two hours with recovery.

6. *Cyanide salts*:—Hydrocyanic acid (Prussic acid) is one of the most rapidly acting poisons. It has been fatal in a dose of 1 gr. The usual fatal minimum dose is however 2.4 grains. It forms after absorption with hæmoglobin a stable compound cyno-hæmoglobin, which action deprives the tissue of oxygen. Treatment must be prompt. Methylene Blue (Methylthionine Chloride U.S.A.) lately introduced for that purpose appears to be an effective antidote. 50 c.c. of a 1% solution I.V. is the method suggested.

7. *Phosphorus*:—Gastric lavage with Pot. Permanganate solution as in Morphia poisoning should be promptly administered. Pot. Permanganate oxidises phosphoric acid which is non-toxic. H_2O_2 and Copper Sulphate in dilute solutions are also used. Fats should not be included in the articles of dietary for several days even after recovery.

Cases and Comments

A Case of Bronchitis

D. V. GOYAL, L.S.M.F.,
Madras.

IN November last when I was at Ambala (East Punjab), I had a chance to see an interesting case.

A young man, P., aged 25, had been suffering for the past eight days with the following complaints :

(a) Cough:—With yellowish white expectoration troubling both in the morning and evening.

(b) Fever:—Intermittent, temperature 100° — 101° F. in the morning, shooting up to 103° — 104° F. in the evening.

(c) Loss of appetite:—With no inclination towards any food.

On examination:—The patient was well built with good physique though reduced a great deal in the last few days. Pulse rate was 95–100 p.m. ; Respiration rate was 20 p.m.

On inspection:—The chest showed deformity and full movements in all areas were present. Expansion of the chest was normal.

On percussion:—Small areas of consolidation scattered over the lower lobes could be elicited with the stethoscope and crepitations were detected in the dull areas. Other systems seemed to be normal.

History.—The patient was a chronic sufferer of rhinitis. He was accustomed to have a walk in the evening and morning. In the beginning of the second week of November when the season has a sudden change, he did not take care to dress properly. One fine morning when he returned from walk he got an acute attack of rhinitis with slight malaise. In the evening he had high fever (104° F) with a little shivering. Cough also appeared the same night. He had some country treatment that night. Next morning the patient was examined by a doctor. Blood slide for D.L.C. was taken with the following results: Poly. 70% ; Lymphos. 25% ; Eosinophils 9% ; and Large monos 1%.

The case was taken up as pneumonia (lobular). So treatment was started accordingly :

(a) Subcutaneous Penicillin-Sodium 1 lac. in morning with repetition in the evening.

(b) Sulphadiazine 4 tablets stat and 2 tablets four-hourly.

(c) Milk of Magnesia 2 teaspoonfuls at night to avoid gastric dyspepsia.

(d) Saline stimulating expectorant. Mixture $\mathfrak{z}$ i t.d.s. with omission of Pot. Iodide which may increase rhinitis.

(e) Tr. Benzoin Co inhalations and Lin. Saponis at times.

(f) Complete rest in bed.

(g) Diet limited to milk and water at repeated intervals.

The treatment was continued for seven days without any amelioration of the symptoms. Sputum was tested with the following results: Pus cells + +; Streptococci +; no influenza bacillus or acid fast bacillus.

DIAGNOSIS:—Lobular congestion affecting very small areas and specially the lower lobes suggested to me that the case was one of acute bronchitis. Blood test and sputum test also helped me to exclude influenza and tuberculosis as the causes of consolidation.

TREATMENT.—The patient was treated with the following mixture—Pot. Iodide to stimulate the expectoration and to help the absorption of the exudate.

R Sod. Chloride	.. gr. v
Pot Citras	.. gr. x
Pot Iodide	.. gr. vi
Tr. Senegae	.. ℥. xi
Syrup Cosylan	.. $\mathfrak{z}$ i
(P.D. or B.D.H.)	
Aqua ad	.. $\mathfrak{z}$ i Sig. T.D.S.

He was asked to take to his usual diet gradually and was allowed to take vegetable soup and fruit juice so as to avoid starvation.

The next day the patient showed marked improvement. The patient was feeling very well. The temperature in the morning was 100°F. and went up to 102°F. in the evening. The main complaint was cough which troubled him even at night, so he was given Glycodin Terp-Vasaka to sip at night. Within a week the temperature came down to normal and the patient was able to walk about. No crepitations could be found but cough still persisted with continued loss of appetite Elixir Neogadine (plain) $\mathfrak{z}$ i plus Syrup Cosylan (P.D.) 1 dr. with an equal quantity of water was prescribed to be taken after every principal meal and the patient was asked to come to the clinic for next report.

By the beginning of December the patient had regained his full appetite with much gain in health without any expectoration but the cough still persisted. Lungs showed no signs of abnormality on

examination. He was given Kepler's Cod-liver Oil with Malt and Hypophosphite (B.W.) $\mathfrak{z}$ i b.d. daily and was asked to have a change for some hot and dry climate.

Recently I received a letter from him that he is alright and even his trouble of rhinitis has also stopped to trouble him.

A Case of Liver Abscess Successfully Operated Under Local Anaesthesia

CHINTA HARAN GHOSE, L.M.P., L.T.M.,
Retired M.O., P.O. Powakhali, Dt. Purnea (Bihar).

A MAHOMEDAN, male, aged about 50 years, a well-to-do cultivator, was seen by me at his rural residence for the following complaints:—

- (1) Pain and swelling in the right hypochondrium.
- (2) Fever low and persistent.
- (3) Discomfort and sleeplessness.

On examination I found a tense and diffused swelling in the region of the left lobe of the liver. The swelling was painful and caused discomfort. He had a dry sallow appearance with much emaciation; eyes sunken into the sockets but no jaundice. Tongue was dry and coated. Constipation and loss of appetite were complained of. Heart sounds were feeble, pulse small and quick, normal lung sounds. Temperature 101°F. No history of dysentery, not addicted to alcohol, no history of syphilis and no trauma.

Pain is reported to have started about a month ago in the right side just below the right costal arch. It gradually and steadily increased. Soon there was swelling with slow and persistent rise of temperature. At this stage he was treated by a quack who diagnosed it as a case of pneumonia and treated accordingly but with no effect. He then came to me for treatment.

I then examined the case thoroughly and provisionally diagnosed it as a case of Liver Abscess till the liver was punctured and pus drawn out. Next day I explored the abscess cavity with a stout hypodermic needle and drew out one 10 c.c. syringe of chocolate coloured pus thus establishing the diagnosis of the case as one of amoebic liver abscess. 30 c.c. of pus was drawn out and $\frac{1}{2}$ gr. of Emetine injected both locally into the abscess cavity and subcutaneously by way of treatment. In the evening another $\frac{1}{2}$ gr. of Emetine was injected subcutaneously but with no effect. Next day I repeated Emetine injection, also Soluseptasine 5 c.c. I.M.

with no response. As medical treatment failed operation was the only course open. I proposed operation and acquainted the patient about the possible dangers of the operation which might culminate even in death. Next day he left my place (Powakhali) obviously to avoid operation. After about a month he came back and *surrendered* himself for operation. By this time the abscess assumed a huge and serious form and bulged at one point.

I decided to open the abscess under chloroform anaesthesia and prepared the patient accordingly. Next day I examined his heart and lungs immediately before the operation and found both the lungs extensively affected with bronchitis. The idea of chloroform anaesthesia was therefore abandoned as the above condition contra-indicated chloroform anaesthesia. This could not desist me from performing the operation. I then decided to perform the operation under local anaesthesia by Procaine and Adrenalin Chloride injection. I put the patient on the operation table. Procaine and Adrenalin Chloride were injected deep into the skin in two parallel lines and longitudinally and tested the line of anaesthesia by pricking the skin along the proposed line of incision with suture needle. When the skin was fully senseless to the needle pricks I gave a longitudinal incision about 2 inches long, about one inch below the right costal arch, and $1\frac{1}{2}$ in. to the right of the linea alba, cut through the abdominal muscles and reached the adherent peritoneum covering the liver. This was cut through and abscess cavity was reached. I then introduced my left forefinger into the abscess cavity but could not come in contact with the solid liver surface and the finger was floating in the liquefied pus. Simultaneously profuse quantity of chocolate coloured pus gushed out. I checked the free flow of the pus by pulling on some sterilised cotton to prevent shock due to sudden removal of pressure. I then inserted a drainage tube, and covered the external wound with moist Acriflavine gauze, put sufficient sterilised cotton and dressed, no lotion being used to wash out the abscess cavity. When the operation was going on the patient complained of no pain. He was then talking with the dispensary medical officer as usual. The said medical officer was watching the patient. The operation was speedily done and the whole thing was finished in less than 10 minutes. I then injected Pituitrin and Atropine to prevent shock.

Next day soiled dressings were removed and replaced by new ones after thorough washing of the skin contaminated by the discharge with Acriflavine lotion. Abscess cavity was not washed with any antiseptic lotion during the whole period of treatment. He was given Soluseptasine and Emetine injections for the first three days. Discharge was less but fever persisted. On the 4th day he was given Penicillin (I. M.) injection (Crystalline Penicillin "G," Glaxo) twice daily for 3 days in 50,000 units doses. Also the same dose was injected locally into the abscess cavity through the drainage

tube. Simultaneously Emetine locally and subcutaneously was continued for the same period in $\frac{1}{4}$ gr. doses twice daily. Temperature came down and the discharge of pus almost stopped. But a bile-like discharge continued for some days. As healing was in progress, the drainage tube was pushed out which was cut short from time to time and ultimately removed. The wound was then put under pressure bandage and left unchanged for 3 days. When the dressing was changed after 3 days the wound closed up nearly completely but for a fine hole which again completely closed after one day's pressure bandage and the wound healed up quite satisfactorily.

N.B. : The operation was done in the local D.B. Dispensary (at Powakhali) with the help and full co-operation of the Relieving M. O., Dr. Monoranjan Das. The patient was provided with a bed in the dispensary for which Dr. Das deserves thanks.

Admitted on 7-6-49. Discharged cured on 19-7-49.

Note :—In liver abscess and in suppurative appendicitis, generally there is adhesion of peritoneum and the pus is, as it were, confined in a closed sac. So the risk of fatal peritonitis is less or none at all. The operation is not as dangerous as it appears to be.

A Case of Quinine Idiosyncrasy

M. A. P. GOONETILLEKE,

A.I.C., *Govt. Dispensary, Dodangaslande, Ceylon.*

ELARIS FERNANDO, 35 years, male, domestic servant, Sinhalese, Roman Catholic, came to me one morning and gave the following history.

History :—He went on a pilgrimage and returned home three days ago. He was feeling feverish on his way back, but no treatment was tried. The day after he arrived home, he developed a high temperature accompanied by chills and rigors. There was no cold or cough, but nausea, constipation and severe headache were present. No history of malaria for a number of years.

Examination :—Temperature— 101°F ; Pulse—120; Volume and tension—good; Heart—N.A.D.; Lungs—clear; Liver—not palpable; Spleen—not palpable:

This was no doubt a case of malaria and the following mixture was prescribed.

$\mathcal{R}$ Quinine Bisulphate	gr. vii ss
Pot. Bromide	gr. x
Tr Zingib	℥ x
Syr Tolu	℥ ss
Aqua ad	℥ i Sig. $\mathcal{Z}$ i. T.D.S.

A dose of this mixture appears to have been taken at about 11-30 a.m. By 12 noon, the patient was brought back to the dispensary with the history of itching and swelling of the body.

On examination he was found to be almost unrecognisable. The whole body was swollen and covered with red urticarial patches. He complained of severe itching and burning sensation. He vomited twice after coming to the dispensary.

History revealed that immediately after taking the dose of the mixture a slight itching sensation was felt over the face which gradually aggravated. Very soon the face swelled up and the swelling extended downwards involving the whole body. The patient was in agony and his state was very pathetic.

He was immediately given an injection of Liq. Adrenalin Hydrochloride m. xv and two tablets of Anthisan.

The effect of these was marvellous. Within 15 minutes he was completely rid of his urticarial rash.

He was kept in the dispensary till 2 p.m. and was again given a dose of the mixture. He developed the urticaria almost immediately and was treated in the same way.

By this time it was plain that the patient was having Quinine idiosyncrasy. Hence the Quinine mixture was discontinued and he was put on a course of Mepacrine, 1½ tablets twice a day after food.

There were no untoward symptoms with this treatment and his temperature came down within two days.

This patient assured me that he had taken Quinine before this, but such a thing had never taken place.

This was the second case of Quinine idiosyncrasy I have met with. The first was my own servant, who could not stand even a grain of Quinine. It was a pathetic sight to see him suffer in those days after taking Quinine. Unfortunately, Quinine was the only anti-malarial drug available then.

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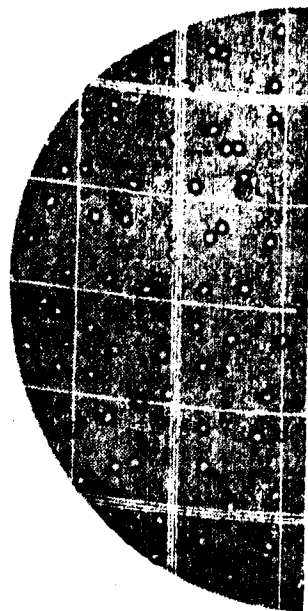
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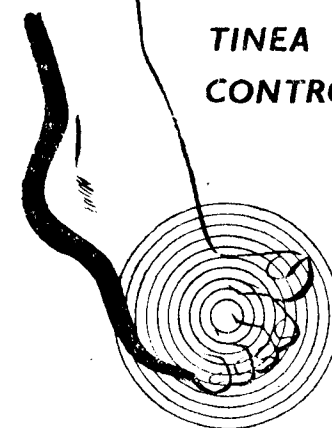
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Adulteration of Foodstuffs

It was an interesting and animated debate that took place in the Constituent Assembly the other day on the amendment moved by PANDIT LAKSHMIKANTA MAITRA that the subject of "Adulteration of Foodstuffs and other goods" be placed in the concurrent list. The object of getting rich quick has unfortunately permeated too deep into the hearts of many people that the evil of adulteration has assumed alarming proportions, never before heard of or known in this country, seriously endangering the health of the nation which is already at its lowest ebb owing to controls, increased cost of living and the rise in prices of absolute necessities of life. No wonder this matter was raised in the Constituent Assembly and was supported by many members.

Mr. BRIJESHWAR PRASAD who moved first his amendment seeking to provide for the control, superintendence and direction of the Union Government over this power of legislation which now vested in the States (Provinces), drew a very lurid picture of the extent to which the evil has spread. It was not confined to one Province alone. "There is not a single commodity," he said, "which is not adulterated. Mustard oil is mixed with mobile oil. In the milk that people buy, there is more water than milk. The evil is growing daily. If the Government is the servant of the people, as it proclaims to be, then it must serve the people and put down this evil with a strong hand."

PANDIT LAKSHMIKANTA MAITRA who moved the amendment above mentioned next recalled the labours of the Central Advisory Board of Health in the past several years and the Expert Committee which it had appointed to go into the question of adulteration. He said that the evil had assumed a dangerous proportion

and the State should take immediate note of it. Otherwise it would kill the whole nation. The Central Government's power in regard to standardisation of goods would not meet the situation.

It is not that there are no Acts on the Statute Book of Provincial Governments to prevent adulteration of foodstuffs. But the provisions of the law are not strictly enforced, and, even when prosecutions are launched under the Act, the offenders are let off lightly with small fines that they are not deterred from committing the crime again and again. As was pointed out in the course of the debate, each Provincial Government has its own peculiar standard. That is why the Expert Committee came to the unanimous conclusion that adulteration of certain foodstuffs which had by the Provinces or States alone. PANDIT MAITRA quoted the example of ghee, mustard oil, coconut and other edible oils, which were produced in different Provinces and sent to other areas. That was why the Expert Committee was convinced that without a comprehensive Central Legislation it would be an impossible task to tackle the problem of adulteration which had assumed the proportion of a scandal.

It is not surprising that Dr. P. S. DESHMUKH who supported the amendment charged the Government with callousness in this all-important matter. While they had been bringing forward and carrying through the Legislature hundreds of Bills they had not as yet come forward, he said, with any Bill dealing with this matter which affected the health of the nation and affected the nation more seriously than any other factor. He cited an instance in the Central Provinces where a merchant was melting thousands of tons of gur in big pans and mixing it up with twenty per cent of an adulterant. This kind of adulteration went on for six years and it was only when the potter supplying the adulterant did not get paid for it that the merchant was dragged to the court. The summary of the proceedings of the Assembly published in the Press does not give us the information how this merchant was dealt with by the court.

It is not only supplementary foods that are adulterated in this manner. Even in regard to staple foods, adulteration is going on, on a very large scale, that the Government of Madras had to issue warning to rice mill owners against the risk they run by resorting to such cruel tactics. The adulteration that is taking place in regard to ghee, oils, milk, etc. is something abominable. The public not only do not get the real value for the money they spend, but are also purchasing diseases in the process.

We thought that after this scathing exposure the Government of India would have come forward to accept PANDIT MAITRA'S

amendment with pleasure and with the realisation of the responsibility that was theirs. But the spokesman on behalf of the Drafting Committee came forward to defend the position taken up by the Committee on the basis of the "man on the spot" theory. It was desirable, he said, to lay the burden of responsibility squarely on the shoulders of the Provincial Governments who could deal with the problem on the spot. It is not that the burden of responsibility does not now lie on the shoulders of the Provincial Governments. As we have said above there are Prevention of Adulteration Acts on the Statute Book. It is because they have failed, and failed miserably too, to invoke the provisions of the Act to prevent the evil spreading like a contagion all round that the Expert Committee had recommended comprehensive Central Legislation and the members of the Constituent Assembly pressed that the subject should be placed in the concurrent list.

But for the intervention of the President, Dr. RAJENDRA PRASAD, it is very doubtful if Dr. AMBEDKAR would have later come forward to accept PANDIT MAITRA'S amendment. He asked the Drafting Committee whether it was not possible to defer consideration of the entry so that the Committee might consult the Health Ministry and Provincial Governments, adding significantly, "there is considerable feeling on this matter in the House. I myself agree with it." And this personal opinion of his, coming with the weight and authority that attach to his views not only as a great leader but as the President of the Assembly should have forced the Drafting Committee to accept the amendment.

We congratulate the Constituent Assembly and the Drafting Committee on the wise decision they have now taken. But we want the Government of India and the Legislative Assembly to realise the responsibilities that now devolve on the Centre. It is not that the Central Government had so far no adequate powers to prevent adulteration and safeguard the health of the people. But as once complained by the Hon'ble RAJ KUMARI AMRIT KAUR, the Health Ministry is allowed precious little that it is not in a position to tackle any of the serious problems of public health with which it is faced. The mere placing of the prevention of adulteration of foodstuffs in the concurrent list will not solve the problem. The Central Government must bring forward a Bill without delay to put a stop to adulteration of foodstuffs and deal with a strong hand those who resort to this malpractice. Then and only then would they have discharged the responsibility that devolved on them and have safeguarded the health of the people under their care.

The Provinces and States have also no reason to grumble that the Central Government has assumed concurrent powers. This should only strengthen their hands and make them enforce the existing Act rigorously with a view to put down adulteration and

bring to book public enemies. We trust that after the exposures in the Constituent Assembly, effective steps will be taken to prevent this crime against humanity.

State Insurance for Employees

"BETTER late than never", and in this sense we welcome the pilot scheme in Health Insurance proposed to be launched by the recently established Employees' State Insurance Corporation in New Delhi by January, 1950. Dr. C. L. KATIAL, who has had considerable experience in this work in England has been specially invited to come over to India and take up the Presidentship of the newly established Corporation.

India is getting industrialised day by day, and, when the many schemes that have already been drawn up or are in the offing are undertaken, our country will become a highly industrialised country and the number of workers employed in these industries would rise very high. There is enough of man-power in the country and raw materials are available in plenty. Only they have not been tapped to the extent they should have been, due to the step-motherly treatment accorded by the rulers in the past, in the view that such development would seriously retard the growth and development of the industries in their own country.

Such a fear has not only been found to be ill-founded but has also been disastrous, has been proved to the hilt in the course of the Second World War. Had India been developed to the fullest possible extent in the industrial field it is very doubtful if the war would have extended to South East Asia, and, even if it had, the war would have continued so long as it had done. Several industries had to be started in a haphazard manner during the course of the war, and many of them have become now stabilised.

With the present and prospective schemes for expansion, the number of people employed in industries would increase manyfold and the problem of State Insurance for the workers would assume serious proportions. Many other countries in the West have taken up the problem in right earnest and have adopted necessary measures to tackle them. It is time that a beginning is made in India, and we are glad such a beginning is made in New Delhi first, extended in six months to Kanpur and East Punjab, and extended further in course of time, benefiting about two and half million workers.

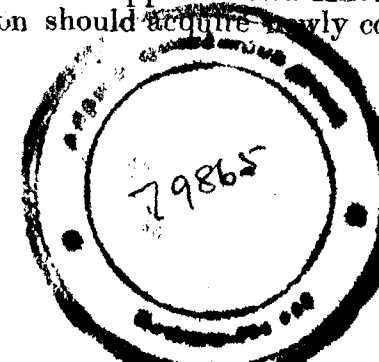
The Medical Benefit Council of the Corporation which met recently under the Presidentship of Dr. KATIAL and which was

attended by the Surgeons-General from many Provinces, considered the Report of the Sub-Committee appointed in April last to go into the question of medical benefit and maintenance of equipment of State Insurance Dispensaries and other cognate matters. The Sub-Committee had recommended that free medical attention through in-patient, out-patient and domiciliary services be given to insured persons.

This medical benefit, in the first instance, would consist of out-patient treatment and treatment in their homes. In the early stages, certain number of beds in the existing General Hospitals would be reserved for insured workers in highly industrialised areas. The medical care available, apart from hospital services, would include skilled supervision at childbirth, simple ante-natal and post-natal care, immunisation against common infectious diseases and free supplies at dispensaries of medical advice, drugs and dressings. In sparse areas medical services to the insured persons would be provided by a part-time doctor or through mobile dispensaries. There would also be preventive services including vaccination and inoculation against typhoid group of diseases and tetanus.

With scarcity of housing accommodation staring us in the face, a beginning can be made in regard to serious cases requiring in-patient treatment only by the reservation of some beds in the present General Hospitals. But we think it would be well not to mix these with the rest in those hospitals, lest others feel that there are two kinds of treatment in those places. Such an impression, if allowed to be fostered, would react on their health, however great the claim of the insured employee is for special consideration and treatment.

The number of working days lost now due to preventable illness is very large, seriously affecting production and thereby helping to maintain prices at the present high level to the detriment of their fellow citizens. Any attempt therefore to ensure the health of the worker, or, if he is ill, to make him fit to resume work without avoidable delay, is therefore to be heartily welcomed. The extension of the scheme to other areas depends largely on the availability of housing accommodation and we endorse the appeal of Dr. KATIAL that Employees' Insurance Corporation should acquire newly constructed buildings for the purpose.



SURGERY

Treatment of acute peritonitis.—(W. A. Altemeier, *Jour. Amer. Med. Assoc.*, 5-2-49). A review of the clinical experience obtained with nearly 600 cases of acute septic peritonitis secondary to acute appendicitis, perforated peptic ulcers, and penetrating wounds of the abdomen, indicates that successful management depends chiefly on early diagnosis and good surgical intervention, and also on chemotherapy as supportive treatment. A comparison of the results obtained in 685 similar cases treated without chemotherapy with those in cases in which chemotherapy was used reveals that the mortality has been reduced 60 per cent, and that this reduction is due to the effects of chemotherapy. The most effective chemotherapy is obtained by the combined parenteral administration of sulphadiazine and penicillin or that of penicillin, sulphadiazine and streptomycin. When used alone in the high dosage of 100 to 500 thousand units every 2 or 3 hours, penicillin was very effective in the great majority of cases, with the exception of those caused in part by virulent gram negative bacteria. The unexpected effectiveness of penicillin in acute septic peritonitis is the result of a conversion of a severe synergistic and mixed infection into a simpler one caused predominantly by gram-negative organisms, usually of low virulence. The combined use of sulphadiazine and/or streptomycin with penicillin is recommended by the author for further control of this etiologic component of peritonitis. T.N.S.R.

A new method of prostatectomy.—(*Jour. Am. Med. Assoc.*, 139, 4: 248: 22-1-1949).

Prof. Cavina of Florence (Italy) in his address before the Medico-Surgical Association of Romagna discussed Millin's new method of prostatectomy and described his own modification of the same which made for greater ease and simplicity. After preparatory treatment with penicillin, calcium and vitamin K, and after general anaesthesia

with ether, the patient is placed in the Trendelenburg position. A short median supra pubic incision in the abdominal wall is made, the rectus muscles are separated with a gosset bistoury and the bladder and the prostatic capsule are exposed. The latter is then incised with a median incision and the surgeon then proceeds to enucleate the adenoma with the index or middle finger of the right hand while the index and middle finger of the left hand are introduced into the rectum as in Freyer's operation. The adenoma may be removed with a suitable forceps. From the meatus of the urethra across the prostatic cavity one introduces into the bladder a large Nelaton's catheter. The breach of the prostatic capsule is then sutured as well as the abdominal wall after previously dusting of the Retzius' cavity with sulphonamide powder. The advantages of this new method are minimum shock, little haemorrhage, and little post-operative suffering. Prof. Cavina has operated on 30 patients by this method in the Giovanni de Dio hospital at Florence, with highly satisfactory results.

T.N.S.R.

Strangulated femoral hernia.—The purpose of this report by Jarboe and Pratt is to report on the contents of the sac found in 104 cases of strangulated femoral hernia and to report the pathologic condition of the strangulated viscera as related to the clinical manifestations, prognosis and fatality rate.

The femoral ring is the site of strangulation in femoral hernias because its rigid walls are formed by Poupart's, Gimbernat's and Cooper's ligaments. These ligaments form a fibrous aperture which is very tenacious when any abdominal or pelvic viscus protrudes into it.

It was observed that the viscera most often incarcerated in strangulated femoral hernias were portions of small intestine and omentum, and that the pathologic condition of these structures had a direct and influential effect on the

prognosis. This intimate association is demonstrated in tables.

A table which records the relationship of the contents of the hernial sac to the fatality rate reveals that in 64 of the 104 cases parts of the small intestine were found in the hernial sac and that 23 or 36 per cent of these 64 patients died, whereas of the 40 patients in whom viscera other than the small intestine were lodged in the femoral ring only one died (2.5 per cent).

Another table demonstrates the extremely high fatality rate associated with the presence of gangrenous portions of small intestine in the hernial sac. There were 19 deaths among the 26 patients in whom the strangulated segment of the small intestine was gangrenous, that is, among this group the fatality rate was 73.1 per cent.

The contents of the hernial sac found in 104 strangulated femoral hernias indicates that a definite relationship exists between the origin and pathologic condition of the affected viscera and the fatality rate.

The authors feel that the prognosis should be determined after evaluation of the pathologic condition of the strangulated viscera when they are visualised and treated at operation. In cases of strangulation of parts of the small intestine, when surgery has been delayed and irreversible pathologic changes have taken place, the prognosis is very grave for a person of any age. Since physical resistance in the aged is materially lowered, such persons are benefited most by early surgical treatment but the prognosis is influenced more by the condition of the strangulated viscera than by the factor of age alone.

An intimate relationship has been found to exist between the character of the contents of the hernial sac and the clinical manifestations. Careful evaluation of these components is essential for the rendering of a dependable prognosis.

(Jarboe, J. P., and Pratt, J. H.: *American Journal of Surgery*, 77: 172—180 February, 1949. The authors are connected with the Mayo Clinic, Rochester, Minn).—*Surgical News Letter, United States Information Service.*

OBSTETRICS AND GYNÆCOLOGY

New pregnancy test.—(S. S. Garrett, *Amer. Jour. Surgery*, 76, p. 261, Sep. 1948.)

In the course of his treatment of cases of amenorrhea with oestosterone Garrett devised a new pregnancy test, as a result of his observations on the effect of the drug on the mechanism of the ovarian function. He used this test on 250 cases with uniformly accurate result. The test consists of an intramuscular injection of three doses of oestrone in oil (1 mg. each) over a period of 5 days in certain selected patients. Failure to menstruate within 24 hours after the last injection is taken as a definite and positive test for pregnancy. The test is inexpensive and simple, easy to perform in the doctor's clinic, without the use of any animals or the assistance from any laboratory. Its chief drawback is that it is not applicable to patients who are bleeding. The test has so far been used only in 4 cases of women approaching

the menopause, and therefore caution is necessary in the interpretation of results in such cases. It may perhaps be possible to assess these results when more data are available. T.N.S.R.

Erythrocyte sedimentation rate in pregnancy.—(*Jour. Obst. Gynaec. Br. Emp.*, Manchester, 55, p. 464-70, Aug. 1948).

Dr. Obermer records the results of 283 determinations he made of E.S.R. in 88 healthy pregnant women and in one in whom the foetus had died *in utero* about the eighth month. He found the E.S.R. velocity increased slightly as early as the fifth or sixth week; from the eighth week there is a regular steady increase in the velocity up to a maximum at the 40th week. After labour commences the E.S.R. has a steep fall and in 4 weeks after confinement it reaches almost normal

values. The non-pregnant state is reached in about 2 months after delivery. Curves plotted from the values obtained for E.S.R. in the one case where foetal death *in utero* had occurred, showed a steep decline in the velocity at the 37th week, reaching almost the normal level for the non-pregnant state. There were no clinical grounds for suspecting any abnormality in this pregnancy.

T.N.S.R.

Thoracoplasty and pregnancy.—(*Jour. Obst. Gynaec. Br. Emp.*, 55, Aug. 1948).

Dr. J. P. McIntyre studied the effects of thoracoplasty on pregnancy and delivery in 12 women (seven multi and five primiparas). A two stage, seven rib thoracoplasty had been performed on each patient included in this study. The interval between thoracoplasty and pregnancy varied from one to ten years. The average duration of labour was 15 hours. Instrumental delivery had to be resorted to in 6 of the 12 patients. Slight dyspnoea was noticed in 2 at the end of the 2nd stage of labour. The placenta was expelled in all cases complete and without interference and no excessive bleeding or sepsis occurred. Observations on these 12 patients showed that the occurrence of distressing dyspnoea during labour after thoracoplasty is largely hypothetic and that interruption of pregnancy is not indicated in these women. Advice relating to pregnancy in patients with thoracoplasty should necessarily be given with great reservation. Sufficient time should elapse to permit thorough healing of the lesions before pregnancy could be allowed to take place. Given a patient with tuberculous lung lesions, anatomically collapsed by successful thoracoplasty, co-existing pregnancy may not be a great strain on the maternal organism.

T.N.S.R.

Results and causes of failure of radiation therapy in carcinoma of the cervix.—Henry S. Kaplan, Hugh M. Wilson and Arthur H. Morse, Yale University School of Medicine, New Haven, Conn. *Surg. Gynec. & Obst.*, 86: 332-40, March 1948.

The best treatment for cancer of the cervix remains undetermined and can only be decided by analysing five year survival rates of comparable series of patients. Statistics are not yet available for this however. A report is presented of survival rates, complications and possible causes of failure in a series of 88 patients with cancer of the cervix treated by radiation therapy. This group were all ward patients whose work-up programme and treatment were practically the same and whose follow-up data were most complete. The symptomatology is reviewed. Statistics of the five year survival rate in these cases showed that results obtained in stages I and II were about the same as elsewhere but those in stages III and IV were not nearly so good as others recently reported. The method of treatment used was closely reviewed for weaknesses. It was found that a comparatively long interval between external radiation and the insertion of radium was disadvantageous because of vaginal stenosis a few weeks after radiation and disappearance of the tissue reaction to preliminary roentgenotherapy. This delay increased the difficulty of applying radium and decreased the biologic response to radium. Conversely, too short an interval between these treatments will increase the incidence of radiation injuries. An interval must therefore be selected which will give reasonable safety with maximum effectiveness. Few complications in this series could properly be charged to radiation injury. The radium dosage used was 6,000 mg. hours equally divided between tandem and colpostat. This seemed both safe and adequate for early cases but better results would perhaps be obtained in stages II and III by doses of 7,000 to 8,000 mg. hours. The different methods of radium treatment are discussed. Good results were obtained by application of radium to the vaginal vault through the medium of hard rubber ovoids, the so-called Manchester technic, combined with an intrauterine tandem. Favourable results have recently been obtained by another combined method in which the intrauterine radium tandem is used but the vaginal applicators are replaced by

intravaginal roentgenotherapy. Radical surgery in the form of Wertheim's panhysterectomy is discussed. Iliae lymphadenectomy has been recently recommended because it is now rather simple and permits removal of pelvic lymph nodes about as well as the more severe Wertheim operation.

Comparison of the five year survival rates and the incidence of lymph node involvement in cervical carcinoma showed that the former averaged about 70 per cent in stage I cases, about 45 per cent in stage II and 25 per cent in stage III. Regional metastases increased up to 35 to 50 per cent when there was direct invasion of one or both parametria, showing that most fatalities from cancer of the cervix result from uncontrolled lymph node involvement. These studies indicate that the interval between roentgen and radium therapy should be decreased to two weeks or less, that more radium should be given to advanced cases, that the Manchester method of radium application should be more generally utilized and that extra-peritoneal lymphadenectomy should be used after irradiation of stages I, II and III to prevent metastases in patients with involvement of the regional lymph nodes.—*Quarterly Review of Obstetrics and Gynecology*.

Intracranial hemorrhage of the newborn.—Colman Saunders, National Maternity Hospital and Combe Lying-In Hospital, Dublin, Ireland. *J. Obst. & Gynaec., Brit. Emp.*, 55: 55-61, February 1948.

Intracranial hemorrhage may cause about 25 per cent of deaths among full term infants and perhaps 30 to 40 per cent of fatalities among premature infants but little advance has been made in its prophylaxis. Vitamin K was not as valuable prophylactically as anticipated. The occurrence of most of these deaths in the first twenty-four hours indicates that trauma is the predominant factor and asphyxia is second. The amount of trauma varies. A tentorial tear with bleeding above or below is the most frequent (96 per cent of this series). The part played by asphyxia in causing intracranial hemorrhage is uncertain but

the evidence indicates that it is a factor. Types of labor more commonly associated with intracranial hemorrhage are precipitate labor, a prolonged second stage, breech presentation, internal version and extraction and difficult forceps cases. Tentorial tears may be caused by antero-posterior or lateral pressure on the head, direct traction on the scalp and traction on the body in a breech presentation. Diagnosis of intracranial hemorrhage in the newborn is difficult because entirely objective. Every case of asphyxia should be treated as an intracranial hemorrhage until proved otherwise. The history is often helpful. The infant may be drowsy, comatose and refuse to suck or it may be restless and irritable with a so-called cephalic cry and terrified expression. Convulsions are common and always indicative of intracranial damage. The Moro or startle reflex is a valuable diagnostic aid. It is best produced by grasping the infant's legs at the ankles and giving a small sudden jerk. A normal child quickly abducts and adducts the arms as though to encircle an object but the reflex is greatly diminished or absent in intracranial hemorrhage. It may also be absent in intracranial edema but rapidly returns with subsidence of the edema. Lumbar puncture is considered inadvisable for diagnosis until other possibilities such as atelectasis, tetany, etc. are excluded. It may be misleading, the fluid being clear in the presence of hemorrhage. It may also cause further hemorrhage. An intermediate course is preferred. It is done to relieve pressure in cases with definite evidence of intracranial hemorrhage and a bulging fontanel but conservative treatment is employed when there is evidence of intracranial hemorrhage without marked pressure. Treatment consists of complete rest and avoidance of any violent resuscitative efforts. Vitamin K is administered and sedatives freely given. Oxygen or oxygen and carbon dioxide may be needed as a respiratory stimulant. Ordinary feeding is stopped and small amounts of fluids or food given by lavage. Riesenfeld considers the up right position important for eight or ten days.—*Quarterly Review of Obstetrics and Gynaecology*.

Early rising after delivery.

Edward L. Cornell and Joseph J. Mullen, Neurotin Hospital Chicago, Ill. *Am. J. Obst. & Gynec.*, 55: 768-77, May 1948.

This study is based on 1,000 deliveries and represents a typical cross section of postpartum cases (53 low cervical cesarean sections are included). Eighty per cent were allowed up before the fifth postpartum day, the remainder on the sixth day. These patients were closely observed and at any sign of complications, were returned to bed rest or limited movement. Five patients experienced various degrees of breakdown in the episiotomy wound, a secondary repair being required in 2 cases. Vertigo, although not uncommon on the first day out of bed, seldom

occurs thereafter. In 2 cases syncope followed vertigo; 1 case was unexplained but there was probably a psychic factor in the other case. No thrombophlebitis was detected in any case during hospitalization but one case of low cervical cesarean section out of bed on day 7 and discharged on day 12, was readmitted for severe thrombophlebitis on day 30. Patients who develop fever of unknown origin are returned to bed rest until the temperature is normal for a twenty-four hour period. There were no cases of uterine prolapse and early rising increased the rate of uterine involution. Cesarean section patients were usually given bathroom privileges on the first postoperative day.—*Quarterly Review of Obstetrics and Gynecology*.

EYE, EAR, NOSE & THROAT

The rupture of the zonule in intra-capsular cataract extraction—a new method.—*Brit. J. Ophth.*, 33: 3-21, Jan., 1949. D. B. Kirby writes:—

The rupture of the zonula fibers is essential in the intracapsular extraction of the lens and the finding of many resistant zonules is accounted for by the number of immature cataracts extracted. The point of rupture of the fiber is just at its junction with the capsule which accounts for the absence of retinal separation. A common error of judgement is to attempt to rupture all zonules by the same method instead of adapting a suitable method to each eye. For rupturing the fibers indirectly, Kirby prefers a combination of the three methods, pressure from without, traction on the capsule, and rotation of the lens in the capsule. After making the section, he uses enough pressure on a point within the corneal limbus at the 6, 8 and 4-o'clock positions to indent the cornea 2 to 3 mm. and to make the wound gape. Pressure outside the limbus is unnecessary, ineffectual and dangerous. Almost simultaneously with the point pressure and while the wound is gaping, the capsule forceps is applied to the anterior capsule just below the equator. He much prefers the forceps to the loop, the pointed hook, the elec-

tro-coagulation electrode and the various types of orisiphakes. Kirby describes his new forceps which is a combination of the Kalt, Arruga and Verhoeff forceps and has a cylindrical handle. His traction is actually a rotation from side to side and he often uses two forceps rather than one. Direct rupture of the zonule is indicated when the zonule is unduly resilient, when patient and surgeon are both relaxed, and when the zonule can be made tense by elevating the lens at least 3 mm. He has used direct rupture for 10 years with excellent results.—*American Journal of Ophthalmology*.

The present state of the intra-capsular cataract operation.—Knapp, Arnold, *Tr. Ophth. Soc. U. Kingdom* 66: 133-178, 1946.

The history of intracapsular cataract surgery is reviewed from 1903 when Henry Smith developed the intracapsular technique in India to the present time.

Three features of the anatomy of the zonule are important in the understanding of the technique of the intracapsular extraction; they are the origin of the fibers, the independence of the zonular membrane from the hyaloid part of the

vitreous, and the insertion of the zonular fibers in the lense capsule. The knowledge of the slight variation in the thickness of different parts of the lens capsule and of the consistency of the lens makes it possible to decide where the capsule should be grasped. The lens can usually be more easily dislodged tangentially, but the thickness, tenseness and elasticity of the lens capsule are important. The older the patient (usually over 60 years of age) the easier the subluxation, and intracapsular extraction is not suitable in persons under 45 years of age.

Smith brings about subluxation of the lens by pressure straight back on the lower half of the cornea, which causes the head to present, and pressure downward on the lowest peripheral point causes the lens to somersault. A slight turning of the lens around in the transverse axis aids in breaking the zonular fibers. The suction cup devised by Halen and popularised by Barraquer also turns the lens on the transverse axis. Verhoeff subluxates by torsion and traction. Kalt introduced the use of capsular forceps and now the use of different types of forceps with some counter pressure is almost universal. The capsule forceps must be small and firm, the speculum light in weight and easily removable with one hand. Akinesia and retrobulbar injection which temporarily weakens the extraocular muscles, especially the inferior rectus, simplify the operation. The use of corneoscleral sutures ensures a quick restoration of the anterior chamber. There should be no traction of the flap and no constriction of the tissues. The injection of air into the anterior chamber counteracts any tendency to anterior adhesion of the hyaloid membrane or of the iris.

The complications of intracapsular operation depend on the proper selection of the cataract. The intumescent cataract with swollen cortex is tense and most difficult to hold with blunt forceps; in its removal the suction procedure is helpful. Many nuclear cataracts, posterior cortical cataracts and sclerosed lenses are the most favorable for the intracapsular procedure. The loss of vitreous is the most important

complication and may occur directly after the incision, during subluxation or after delivery of the lens. The loss of vitreous or injury to it is often followed by a pupil which is drawn up because of cicatricial contraction of the vitreous and adhesions of the iris. Rupture of the capsule during extraction of the cataract may be serious if the hyaloid membrane is also ruptured. Iridocyclitis occurs after intracapsular extractions but is usually very mild. In the more severe types it may be followed by deep vascularization of the cornea and glaucoma. Glaucoma is usually secondary to anterior adhesions of the capsule to the incision or it may follow an anterior adhesion of the hyaloid membrane to the posterior surface of the cornea or to the incision. Trauma to the corneal endothelium may cause the hyaloid membrane or iris to become adherent before the anterior chamber is established. The injection of air or salt solution into the anterior chamber at the close of the operation will help prevent an anterior adhesion of the vitreous body. Detachment of the retina usually seems to be associated with low grade cyclitis. Complications of the intracapsular operation are difficult to treat satisfactorily. The future of the operation lies in the reduction of the difficulties by improvement in the methods of operating.—*American Journal of Ophthalmology*.

Local streptomycin therapy for suppurative otitis.—H. W. Harris and associates (*Archives of Otolaryngology*, 49: 69, Jan. 1949) report the use of local instillations of streptomycin in the treatment of suppurative otitis media in 18 patients, in 9 of whom both ears were involved, making a total of 27 suppurating ears. In all these cases there was a persistent aural discharge, cultures of which showed organisms sensitive to streptomycin. The concentrations of streptomycin most frequently used were 5,000 to 12,000 units per cc.; both lower and higher concentrations were used occasionally. Penicillin in a concentration of 500 units per cc. was incorporated in the solution for the treatment of 4 ears in 3 of which staphylococci had been found in the cultures. Only

one patient (with bronchiectasis) was given streptomycin by intramuscular injection in addition to the local treatment. About 1 cc. of the solution was instilled into the ear canal at each treatment; treatments were given from six to eight times a day in the hospital, and usually four times daily for patients treated at home. The discharge stopped in 15 of the 27 ears treated by this method, and did not recur in a period of one to six months. There was temporary improvement in 7 ears. Streptomycin-resistant strains of organisms appeared during treatment in a large percentage of cases, although the concentration of streptomycin in the solutions used was at least twenty times as great as the minimum concentration required to inhibit the organisms before treatment. But the fact that good results were obtained in over half the ears treated when other methods of treatment had failed indicated that local streptomycin therapy is of definite value in certain cases of suppurative otitis media. It is suggested that the effectiveness of local streptomycin therapy in suppurative otitis may be increased by increasing the concentration of the streptomycin solution employed and perhaps by more frequent treatments; by lavage or irrigation with alkaline solutions prior to the streptomycin instillations (since the activity of streptomycin is reduced in an acid medium); by combination with other antibiotics or chemotherapeutic agents; by methods to increase the diffusion of the solution instilled through the middle ear cavity; and by systemic therapy with streptomycin and other antibacterial agents in addition to local therapy. Surgical procedures to clear away necrotic debris and eradicate foci may also be necessary in some cases.—*Medical Times*.

The etiology, symptomatology and treatment of juvenile glaucoma.—O. H. Ellis (*American Journal of Ophthalmology*, 31: 1589, Dec. 1948) reports that he has treated 5 patients with glaucoma between the ages of fifteen and twenty-six years; 2 of these cases have been previously reported; 3 cases are reported in this article. In one of these cases the patient was a man

twenty-six years of age, who had had four attacks of acute glaucoma in the right eye; an extensive goniotomy was done in this eye, and a prophylactic goniotomy was done in the left eye; the patient has a corrected vision of 20/20 in each eye with full visual fields. In the other 2 cases, the patients were girls aged fifteen and nineteen years respectively. In these cases normal intra-ocular tension has been maintained with relief of all symptoms by the use of prostigmine. In the 2 cases of juvenile glaucoma previously reported bilateral goniotomies were done with good results. There was no history of glaucoma in the family in any of these cases. From a study of these cases, the author concludes that the cause of juvenile glaucoma is the presence of a remnant of pectinate ligament blocking and covering over the angle of the anterior chamber—the same tissue that causes congenital glaucoma; it can be seen with the gonioscopic lens. Early diagnosis of juvenile glaucoma is important. The most frequent symptom is severe headache, recurring frequently, present especially in the morning and not relieved by aspirin; another characteristic symptom is seeing coloured rings around lights, at first at intervals, then constantly; finally a sense of fullness or pain in the eye develops. Study of the visual fields often show contraction of the fields similar to that observed in adult glaucoma. The intra-ocular pressure is rarely constantly elevated, but shows considerable fluctuation. The diagnosis must be finally established by the use of the gonioscopic lens.—*Medical Times*.

Vitamin A therapy in deafness.—Writing in the *Eye, Ear, Nose and Throat Monthly*, Dr. M. Joseph Lobol, M.D., presents a new therapeutic approach to the problem of deafness. He gives the result of study on 300 patients with progressive deafness and treating them with an injectable vitamin A preparation. This preparation consists of olive oil terpins and the A factor of a concentration of 50,000 units per c.c. This is usually given by the intramuscular route, twice weekly for an initial period

of six weeks. If at the end of this time favourable results are observed, treatment was continued for an additional 20 to 22 weeks. Patients who failed to respond to the therapy after five weeks were not given any further treatment. Marked results were obtained in hard

of hearing patients treated for more than five months. Tinnitus was relieved in many patients in less than two months. It was found that prompt response for vitamin A therapy is very marked in the catarrhal form of deafness.

MEDICINE AND THERAPEUTICS

Streptomycin in the treatment of tuberculosis.—(*Lancet*, 12-2-49. An official memorandum by the Special Committee of the Standing Advisory Committee on Tuberculosis to the Ministry of Health).

The indiscriminate use of Streptomycin carries with it definite dangers both to the individual and to the community. The tendency to "try" this drug in every form of tuberculosis is to be condemned. In the present state of knowledge the following general criteria should be observed for selecting cases.

Pulmonary tuberculosis in which the lesions requiring treatment are of recent development progressive and unlikely to benefit from conventional methods (such as rest in bed and/or collapse therapy) alone. This definition includes rapidly advancing pulmonary tuberculosis in which immediate collapse therapy would be dangerous or impracticable and also acute "Spreads" including those after collapse therapy. Old chronic fibroid or fibro-caseous lesions, apparently terminal conditions, or minimal early lesions with favourable prognosis should be excluded. Streptomycin should not be used as the only therapeutic measure; its major role is to make possible the use of collapse therapy, which otherwise may have to be delayed or never performed.

Miliary and meningeal tuberculosis.—Streptomycin is the most effective drug discovered so far to treat these cases successfully. A daily intramuscular dose of 20 mg. per pound of body weight up to a maximum of 2 grammes is recommended. This should be maintained for 4 months. Further courses may be required. In addition for meningitis 50-100 mg. of the drug dissolved in 5-10 c.c. saline should be

injected intrathecally. Everyday for 3 weeks and then every other day for 2 weeks.

In tuberculosis conditions other than miliary or meningeal 2.0 grammes daily was at first the usual dose for all adult patients but this had toxic effects. 1.0 g. a day has been found to be less toxic. 10 mg. per pound of body weight per day is suggested and the dosage should be lower for patients over 45 years. The administration of the daily dose in 1 or 2 injections is recommended. The maximal effect is reached in one or 2 months. It is useless and may be dangerous to treat these cases with streptomycin continuously for over 3 months. It is also probable that treatment for less than one month is ineffective.

Ulcerative tuberculosis of larynx, trachea, bronchi, tongue and pharynx.—Streptomycin is of proved value in these conditions. In laryngitis and tracheo-bronchitis diagnosis should be confirmed by laryngoscopy or bronchoscopy wherever possible and the examination should be repeated during and at the end of the treatment, to assess the effects of the streptomycin therapy. Only intramuscular route is to be preferred.

Thoracic surgery.—Prophylactic use of streptomycin to "cover" routine thoracoplastics is not recommended. The value of the drug injected locally into infected sub-scapular spaces after thoracoplasty is established.

Cutaneous sinuses and fistula.—The drug is often useful for healing in these conditions.

Tuberculosis of the alimentary tract and peritoneum.—Favourable results have been reported in these conditions: (Please see page 711 for information on this subject).

Genito-urinary tuberculosis.—Streptomycin may prove of benefit in some types of this condition.

Bone and joint tuberculosis.—The value of the drug in this condition has not been established though a few encouraging results have been reported.

Note.—Streptomycin is generally supplied in one of three forms—as the hydrochloride, as the sulphate or as a calcium chloride double salt. It is recommended that any change-over from Sulphate or Cal. Chlor. Salt, to another in intrathecal (and probably intramuscular) administration should be avoided. If a change has to be made, several weeks should be allowed to elapse between the last injection of the one salt and the first injection of the other.

T.N.S.R.

Successful treatment of pneumonia with intramuscular aqueous penicillin once a day.—(W. Weiss and I. Steinberg, *Am. J. Med. Sci.*, 217, 1: 86-91, Jan. 1949).

The authors describe the treatment of 30 cases of pneumonia in man, with a single dose of 300,000 units aqueous penicillin-G, intramuscularly once a day until the temperature remains normal for 3 days. 15 males and 15 females ranging in age from 16 to 72 years, who were admitted to the Sydenham Hospital, New York, in the 1st to 8th day of illness (the onset of the pneumonic process was taken as occurring with the chill and pleural pain), are included in this study. On the basis of clinical and X-ray findings and complications, the degree of illness was graded as severe in 8, moderate in 14 and mild in 8 cases. Sputum studies showed pneumococci in 26, blood culture was positive in one; one lobe was involved in 25 cases, 2 in two and 3 in one case. One daily injection was continued (300,000 units given on admission, and at noon on each day) for 4 to 13 days, usually only 5 to 7 days. Two cases received 4 and five cases more than 7 injections. Patients were kept in hospital until X-ray picture showed complete resolution or until delayed resolution was adequately studied.

The results compared very favourably with those obtained by various other investigators in the treatment of pneumococcal pneumonia by the repeated injection of smaller doses at frequent intervals.

The mortality rate was 6.6% compared with 7.0% in the multiple daily dose series of 178 cases.

This report confirms in human the experimental impression that the effect of penicillin is not dependent on the continuous maintenance of a penicillin level in the blood stream. It appears that the anti-bacterial effect of the drug outlasts the demonstrable blood level by many hours. This lessens the need for special forms of penicillin having the property of delayed absorption. Intramuscular doses of 300,000 units aqueous penicillin-G, in man have been shown by McDermott and his associates (*J. Clin. Invest.*, 25: 190, 1946) to give demonstrable blood levels lasting 4 to 8 hours. Tucker and Eagle (*Am. Jour. Med.*, 4: 343, 1948) have confirmed this finding, for doses of 360,000 units. Parker and Mash (*Jour. Bact.*, 52: 181, 1946) showed that there is a period (after the administration of penicillin) during which the surviving organisms do not begin to multiply after the drug is withdrawn. Another factor is the tissue storage of penicillin, as demonstrated by Tillett and his collaborators in 1944. The blood level is not the only guide for treatment. Dosage must be based on therapeutic effect. The protective mechanism of the host is yet another factor to be taken into account.

T.N.S.R.

Para-aminosalicylic acid (PAS) in chronic pulmonary tuberculosis.—Drs. Eastlake and Borach writing in the *Diseases of Chest* observe as follows:

The serum of patients and normal individuals after ingestion of 2 to 4 gms. of para-aminosalicylic acid produces a definite inhibition of the growth of the tubercle bacillus in Dubos media. This has been demonstrated with the H37Rv strain as well as with strains recovered from patients with active pulmonary tuberculosis.

Para-aminosalicylic acid was administered to twelve patients with advanced chronic pulmonary tuberculosis. The dosage employed was 10 to 11 gms. daily in courses of three weeks, with a free interval of one week. In seven patients in whom the symptoms of active infection were sufficiently marked to permit judgment of the effectiveness of drug, observable clinical improvement took place in all. In three patients recurrence of symptoms on cessation of treatment took place. Three patients who suffered from fever and signs of acute exudative tuberculosis were much improved and in good general condition after treatment, but without arrest of their disease.

The characteristic effects of administration of para-aminosalicylic acid were prompt reduction in cough and expectoration, and decrease in fever when present. These favourable results were generally manifested within the first week of treatment. The side effects of the drug were intermittent nausea, vomiting and looseness of the bowels. In three of the twelve patients these symptoms were sufficiently severe to force discontinuance of the drug.

The favourable clinical results of treatment with para-aminosalicylic acid in chronic pulmonary tuberculosis suggests that this drug is a useful agent in the treatment of acute exacerbation of the disease.

One hundred cases of military meningeal tuberculosis treated with streptomycin.—P. A. Bunn (*American Journal of the Medical Sciences*, 216:286, Sept. 1948) reports 100 cases of acute military tuberculosis and/or meningitis treated with streptomycin in 47 Veterans Administration Hospitals. Of 22 patients with military tuberculosis without meningitis, 16 or 73 per cent survived. Of the patients with tuberculosis meningitis 37 per cent survived, the highest survival rate being in the group with meningitis alone, without military tuberculosis (16 survivors in 43 cases). There were 40 survivors in the entire series of 100 cases at the time that this report was submitted. Eight months later, 24 of these

40 patients were known to be living; 15 had died, and one was lost to follow-up. Three of these deaths occurred in patients treated originally for military tuberculosis; all of these patients subsequently developed meningitis; 7 deaths occurred in those originally treated for meningitis; in 5 death was due to relapse or progression of the original disease, and in 3 to progressive pulmonary disease (without evidence of meningeal disease). There were 5 deaths in the group with combined military and meningeal disease, all due to meningitis, 3 showing no pulmonary military tuberculosis at autopsy. Of the 24 patients surviving, 8 have active foci of tuberculosis other than the disseminated form; 1 is undergoing a fourth course of treatment for meningeal relapse, after "cure" of the military lesions for which he was first treated. There are 15 patients who are either free from tuberculosis or show arrested lesions; only one of these patients was treated originally for combined military and meningeal tuberculosis, 7 were treated originally for military tuberculosis and 7 for meningeal tuberculosis. Various dosages of streptomycin were used in this series of cases; a daily dosage of 1.8 gm. by intramuscular injection and daily intrathecal injection of 0.1 gm. in cases of meningeal disease were suggested, but this schedule was not used in all cases. Of the 24 surviving patients, 10 have moderate to severe impairment of labyrinthine function, one is deaf, and 3 have neurologic sequelae.—*Medical Times*.

Granuloma inguinale treated with streptomycin.—(*Ohio. St. Med. Jour.*, 44, Aug 1948; *Jour. Amer. Med. Assoc.*, 137: 7-8-1948).

(1) Streptomycin was given intramuscularly every 3 hours; the dosage was 250 mg. till a total of 20 to 60 grammes had been given. All the patients showed dramatic improvement with complete healing of the lesions; there were no recurrences over an observation period of 4 to 5 months. Two of the patients had received antimony preparations over a considerable period without any benefit.

(2) At the Chicago intensive treatment centre 122 patients with granuloma inguinale were treated. 85 had lesions in the genitalia; the inguinal folds were involved in 20 and the perineum, pubic region and thighs in 12. Of 64 adequately treated with antimonials, lesions healed in 57 and 7 failed to respond. In 11 cases streptomycin was given, with excellent results in 8. These had received only a total of 3.1 to 12.2 gm. of streptomycin, given in 3 hourly injections (for 15 to 30 days) of 250 mg. each. Four of these were of very long standing and had resisted antimony treatment (one of 24 years, one of 2 years, one of 10 years and one of 28 years' standing). In the 3 cases of initial failure 4 to 5 gm. had been given 3 hourly in 18 to 41 days. The first relapse occurred after 7 months and was successfully treated with streptomycin. The two other cases that relapsed after 3 weeks and 9 months respectively were successfully treated with antimonials.

T.N.S.R.

Folic acid. M. C. G. Isaacs, M.Sc., M.D., M.R.C.P. (From *The Manchester University Medical School Gazette*).

In any text-book dealing with the vitamins, there is a large chart or table giving the individual factors that make up the vitamin B complex: tucked away in an unimportant corner is folic acid. This minor factor has become one of the therapeutic "finds" of recent years, and is probably of wider application than any other member of the complex—thiamine, riboflavin, pyridoxine, niacin, and others—whose constitution is known. The story of the discovery of folic acid's importance is a fascinating chapter of medical research, but discouraging for researchers, since, once again, it has been a side-issue and not the main object of the research that proved most important.

Folic acid was first obtained in an almost pure form in 1941 by extraction from spinach leaves. It was found to be a growth factor for the metabolism of certain bacteria *Lactobacillus casei* and *Streptococcus faecalis* R—so that it was also named the "*L. Casei* factor." It is widely distributed in

nature and can be synthesized by the bacterial flora of the mammalian intestine. In order to study the effects of its absence from the diet, rats were not only given a specially purified diet, but were also given doses of sulphasuxidine to sterilize the colon. Rats treated in this way grew only slowly, and they developed a profound leucopenia; the bone-marrow lost its cellular appearance, the granular white cells being notably diminished; anaemia was only slight. When folic acid was given to the rats, remission was rapid and the bone-marrow returned to normal. This suggested that folic acid might have a place in the treatment of human granulocytopenia. The problem was tackled by typical American "high-pressure" tactics. A team of fourteen biochemists set to work and soon produced all the answers. It was found that folic acid could be isolated from liver and yeast, and that there were a whole group of compounds of very similar nature that had been given different names because they had been separately isolated: vitamin M—important in the nutrition of monkeys, vitamins B₆, B₁₀ and B₁₁ influencing chicken nutrition, the "liver eluate factor," and the "*L. casei* factor." The folic acid from liver was isolated, its constitutional formula shown to be that of a pteroyl glutamic acid, and finally it was synthesized; this synthetic pteroyl glutamic acid is the folic acid now available and the one that was used in the original therapeutic trials.

For the next stage, the investigators cast round for a nutritional disease in humans that was accompanied by a low granular white cell count; such a condition occurs in the nutritional anaemias among the improperly fed populations in the Southern States of the U.S.A. A team of physicians, radiologists, biochemists and assistants started work on the effects of folic acid in these patients. Folic acid produced the expected remission in the granulocytopenia, but it also had a much more startling effect on the accompanying anaemia, when the anaemia was of macrocytic (high colour-index) type. The investigation was switched to the treatment of macrocytic anaemias and it was found that folic acid, in quite small doses (20-100 mg.

daily), produced a dramatic recovery in nutritional anaemias of this type. Now sprue is a nutritional disorder accompanied by a macrocytic anaemia, and it has been known for some time that the bone-marrow is often megaloblastic, resembling that of pernicious anaemia. The investigation team therefore moved over to Cuba, where sprue is endemic and really severe cases occur. Folic acid was given to typical selected patients, and here, again there was a surprise, for the folic acid treatment not only relieved the anaemia as anticipated, but the diarrhoea with fatty stools that is typical of sprue was also relieved, though the fat content of the stools remained high. A combination of folic acid with appropriate diet relieved every patient in a remarkably short time. The final stage was to try folic acid in true cases of pernicious anaemia, that is, on patients with a macrocytic anaemia, megaloblastic bone-marrow and achlorhydria. Folic acid produced a remission with typical change-over of the bone-marrow picture from megaloblastic to normoblastic, reticulocyte response, and a rise of red cells and haemoglobin comparable with the rest results from liver treatment. These findings have been confirmed in our clinic at the Manchester Royal Infirmary. It was not surprising that soon physicians were giving folic acid to any patient with anaemia that was proving refractory to the normal treatment; there have been more failures than successes, and it is now becoming clear that folic acid is only therapeutically active in patients whose bone-marrow is megaloblastic, i.e., in Group 1 of the clinical classification of anaemias that I described in 1941.

There is thus no doubt that folic acid is a very valuable addition to the drugs available for the treatment of anaemia, and possibly of alimentary tract disorders associated with defective fat absorption. What is it? What is its dose and how is it given? Which patients are suitable for treatment, and are there any contra-indications? How does it act? There is information about all these points.

Folic acid is chemically N-[4-(2-amino-4-hydroxy-6-pteridyl) methyl-amino-

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benzoyl] glutamic acid. It has been synthesized by a somewhat complicated process whose yield is about 15 per cent; it is, therefore, as yet, expensive. It is an orange-yellow powder that is quite stable and can be dispensed in tablet form. For most purposes it is given by mouth, initially 20 mg. daily, and later the dose can be reduced in many pernicious anaemia patients to 5 mg. a day or even less. For sprue, 20 mg. a day is again sufficient. If a very rapid response is needed, or if the patient is vomiting, it can be given intravenously dissolved in alkaline sodium phosphate solution; it is the practice to give a single dose of 50-100 mg. dissolved in say, 100 c.cm. of fluid and the solution given in an intravenous drip apparatus; such a technique produces a reticulocyte response within 48 hours.

Before folic acid is given to a patient, it is essential that a firm diagnosis of pernicious anaemia, or other megaloblastic anaemia, should have been made. The patient should be thoroughly examined and special attention paid to the central nervous system; a full blood count, a sternal marrow puncture, and fractional gastric analysis, are minimum requirements. It must be remembered that these investigations have to be done *before* folic acid is given, since once given, all the relevant clues will have disappeared in 2-4 days, and no diagnosis can then be made. If the patient has the typical clinical picture of pernicious, or allied, anaemia, has a macrocytic anaemia and megaloblastic marrow, then folic acid can be given and a good response anticipated; patients with free HCl in the gastric juice may not do so well. In sprue the diet must be controlled as well; a high protein low-fat diet is needed. Folic acid is being tried for the treatment of coeliac disease in children; the results have so far been variable; only a minority of patients develop a megaloblastic anaemia and this may explain the variations observed. No cases of sensitivity causing unpleasant reactions after folic acid have been reported. The only contra-indication—and it is an important one—is evidence of postero-lateral spinal cord tract involvement; there is

evidence from America that when patients with this combined sclerosis have been treated with folic acid, the U.N.S. lesions have been unaffected, or have actually worsened, even though the anaemia has responded satisfactorily. These observations await confirmation and, of course, follow-up studies lasting many months are needed; but at least one of our patients has shown this intensification of symptoms of postero-lateral sclerosis while on folic acid treatment. For the time being, such patients should be given liver or desiccated stomach treatment.

The mode of action of folic acid is not certain. It is clear that folic acid is not the anti-anaemic principle present in liver extracts; completely colourless active liver extracts have been obtained, and liver extracts containing hardly any folic acid are still active. Studies of the action of folic acid in bacterial metabolism suggest that it acts as an enzyme or coenzyme and so allows the production of the anti-anaemic principle to proceed in the absence of the natural enzyme haemopoietin.

Folic acid has certainly come to stay: at present, treatment with folic acid is about ten times as expensive as treatment with intramuscular liver extract, but this margin may well be reduced. The convenience, one or two pills with breakfast—not necessarily every day—will make them popular with patients who do not like injections, and save the doctor much time in giving them. Tablets are much pleasanter than a dose of desiccated stomach powder, but it should be remembered that a patient having the latter gets an ounce of almost pure protein added to his rations every day. Progress with folic acid should be watched, but like all other really active drugs, it must be used properly and never distributed indiscriminately before diagnosis has been made.—*General Practitioner*.

Principles in the use of diuretics.

—By Arthur J. Merrill, M.D. (From *The American Practitioner*, Dec. 1947).

Cardiac oedema is generally conceded to be associated with retention of salt and water. The "backward" failure

proponents believe that the fluid is forced into the tissues by a high venous pressure. The "forward failure" concept seems to be receiving increasing support. Experimental evidence for this briefly consists of the following: When the cardiac output becomes inadequate for tissue demands, the efferent arterioles of the kidney contract in response to an unknown stimulus to divert blood to tissues whose metabolic needs are greater than those of the kidney. The efferent arteriolar constriction produces a rise in filtration pressure, which maintains a relatively normal filtration rate until the renal plasma flow falls below 200 per cent per minute. Below this point the filtration rate falls rather rapidly. Thus, the filtration of salt and water is greatly reduced, whereas re-absorption continues to be almost complete, which results in a retention of salt and water with oedema formation. This retention seems to occur in resting cardiac subjects at filtration rates of below 70 cc. per minute. This concept seems to explain more adequately than the backward failure theory the great importance of salt restriction in the oedematous cardiac patient.

Diuretics, in order to reduce oedema, must cause increased elimination of sodium as well as water. Schemm and Wolff contend that water, when given with a limited salt intake, will itself produce a sodium diuresis. Studies in our laboratories and work by Crutchfield do not bear this out. Further work is being done to attempt to account for these discrepancies.

Diuretics are indicated in congestive heart failure when, despite the use of digitalis, limited activity and limited salt intake, the patient has (1) attacks of paroxysmal nocturnal dyspnoea, (2) dyspnoea on moderate exertion, (3) enlargement of the liver due to congestion, or (4) distended neck veins. One does not wait until pitting oedema is evident, for the patient may accumulate as much as 15 pounds of excess fluid before clinical oedema appears. After dehydration of the patient to the condition of maximum comfort, his weight should be determined. A gain above this basic weight of more than three

pounds of fluid is usually an indication for administering another dose of the diuretic.

The chief contraindications to the use of diuretics are renal insufficiency and acute glomerulonephritis. Large amounts (3 to 4 plus) of albumin are often seen in heart failure and do not preclude the use of these drugs. A moderate elevation of the blood NPN to as high as 70 to 80 mg. per cent may be seen in uncomplicated severe heart failure due to reduced filtration by the kidneys. Levels above this demand extreme caution in the use of mercurials, acid-forming salts, potassium salts, and urea. A normal PSP test does not always mean that these substances may be given with impunity, since the PSP test depends on the function of the proximal convoluted tubules while non-protein nitrogen excretion is related to filtration. In patients with thickening of Bowman's capsule caused by glomerulonephritis, filtration may be greatly reduced in the presence of a normal PSP test. The insulin clearance is the most accurate index of such a state, but is not practical for clinical use. The urea clearance test is quite satisfactory, but for practical purposes severe limitation of filtration is indicated by an elevation of a single blood NPN or urea level.

Acid salts are useful, particularly in patients with mild oedema, because alone they may suffice to control the oedema. The exact mechanism of their action is uncertain. Some believe that the acidifying action causes water shifts which favour elimination; others think that the function of the kidneys in maintaining acid-base equilibrium causes the renal tubules to reject the excess anions which carry fixed base and water with them into the urine. Ammonium chloride is one of those most commonly employed. The ammonium radical forms urea, leaving the chloride ion free to carry away sodium. It is given to the point of gastric tolerance, usually about 4 to 6 Gm. daily, either continuously or with intermissions of two to three days weekly to avoid gastric irritation. It is also given prior to injections of mercurial diuretics, as the diuresis from the two combined is greater

than a simple additive one from each alone.

Osmotic diuretics act by simply carrying off an obligatory amount of water with them, and since they increase sodium excretion relatively little, they are not extremely effective. Fifty per cent glucose intravenously may be used when more active diuretics are contraindicated. Sucrose seems to be toxic to the convoluted tubules and is no longer used. Urea is quite useful in patients with moderate failure and relatively good renal filtration. It is given orally in grapejuice in doses of 30 Gm. three times a day. Its disagreeable taste prevents a wider usage.

The xanthine group acts by decreasing re-absorption of salt and water by the tubules. Theophylline is by far the most potent and is the best of oral diuretics, with the possible exception of mercurials, but, unfortunately, it causes marked gastric irritation, and can rarely be given for more than two or three successive days. Its salt aminophylline is not very effective. It may be alternated with an acidifying salt. Doses of 0.3 to 0.6 Gm. three times a day may be given. Theobromine is less active, but is better tolerated, and may be given in larger doses of 0.6 Gm. three to five times a day. Caffein is not a very potent diuretic. The salts of this group containing sodium would seem to defeat their own purpose, since it is sodium that we are trying to eliminate.

Mercurial diuretics have added many years of comfort to lives of thousands of cardiacs. They are by far the most potent of diuretics. While some evidence has been presented to indicate that they cause a release of bound water and produce a diuresis in this way, the overwhelming weight of evidence shows that their primary action is to decrease re-absorption of salt and water by the proximal convoluted tubules. Normally about 99 per cent of the filtered salt and water is re-absorbed by the tubules. If only 98 per cent is re-absorbed the urine output will be doubled. Thus, only a slight effect upon the tubules by the mercury is required for a good diuresis. The diuresis begins one to three hours after

administration of the mercurial, reaches its peak at about six hours, and is over in 12 to 24 hours. A slight increase in urine flow is evident in some patients during the second 24 hours. There seems to be little choice between the three commonly used mercurials, mercuripurin, salyrgan with theophylline, and mercurhydrin. All contain theophylline, which seems to help prevent sloughs if the material leaks into the tissues. It also makes intramuscular administration less painful. Some evidence has been presented to show that mercurhydrin is less toxic than the other two, but this is not striking. Some of the reactions are those of general mercury poisoning with diarrhoea (sometimes bloody), abdominal cramp, etc. Muscular cramps occasionally occur, supposedly due to chloride deficiency, and may be prevented by simultaneously giving ammonium chloride. In rare cases sudden death has been reported presumably due to ventricular fibrillation. Another serious reaction is a shock-like syndrome thought to be produced by sodium depletion. This is relieved by sodium chloride administration. In three instances the author has repeatedly seen a reaction to mercurhydrin, which is apparently benign. The patient becomes acutely dyspnoeic and remains so for about three to five minutes. In one of these subjects the reaction has become less pronounced with subsequent injections, and has almost disappeared. In the other two it was necessary to discontinue the drug. Salyrgan and mercuripurin failed to produce the reaction in these same individuals. Reducing the rate of administration seemed to decrease the severity of the response. It does not occur with intramuscular use. Mercurhydrin seems to cause less pain by the intramuscular route than the other two, some patients complaining not at all, though some complain bitterly. Mercuripurin suppositories, now called mercurxanthin, produce a very satisfactory diuresis, though not as great as via the intramuscular and intravenous routes. Severe rectal irritation and pain occur in some subjects, but others tolerate the suppositories quite well. Oral tablets of salyrgan with theophylline are available, to be given five at a time

about once or twice a week. The diuresis is not nearly so marked as that produced by other routes, but some fluid is lost by the diarrhoea, which frequently occurs with the tablets. Many patients have some diarrhoea and abdominal cramps.

The dosage of mercurial diuretic is 0.5 to 2.0 cc. and some have given as much as 3 cc. without untoward reaction. This may be repeated as often as twice a week indefinitely without giving any evidence of renal damage at autopsy. I have seen one patient who received 2 cc. daily for three weeks without evidence of renal irritation, but this is not recommended. The frequency of administration is best guided by the patient's weight as outlined above. Mercurial diuretics may produce irreversible fatal uraemia in patients with impaired kidney function, since a high concentration of mercury may become fixed in the tubules of the remaining active nephrons. No patient with malignant hypertension should receive these drugs without obtaining a blood NPN determination. No patient of any type should receive a mercurial if the NPN is above 100 mg. per cent. Blood NPN concentrations of as high as 60 mg. per cent are not uncommon in heart failure, but levels above this should make one proceed with great caution and with very small doses of mercurials, checking the NPN after each dose at first. Benign hypertension and marked albuminuria are no contraindications to the use of these drugs. Patients with the nephrotic syndrome of glomerulonephritis and patients with cirrhosis of the liver are sometimes benefited by mercurial diuresis.

Digitalis is a diuretic when it raises the cardiac output. Slight rises in renal plasma flow secondary to the rise in cardiac output may greatly increase the filtration rate. Furthermore, the increase in urine output may be far out of proportion to the increase in filtration rate, as a greater percentage of the filtrate seems to be rejected. The most spectacular results occur with auricular fibrillation in which the cardiac output may be almost doubled.

With the use of diuretics the signs of congestive heart failure may disappear

and the profound weakness associated with inadequate blood flow to the muscles may become pronounced because of the increased activity permitted by the relief from dyspnoea. Diuretics aside from digitalis-like preparations do not seem to influence the cardiac output and do not prevent these symptoms. *General Practitioner.*

Tonsillectomy and poliomyelitis. (*Jour. Am. Med. Assoc.*, 139:4 p. 249.)

A recent survey conducted by a Committee of American Laryngological, Rhinological and Otolological Society on Poliomyelitis, and Tonsillectomy revealed that when a bulbar poliomyelitis followed a tonsillectomy, it was coincidental and not the result of the operation. But Dr. Harold K. Faber of the San Francisco University Hospital has challenged this "dangerous conclusion" in the light of evidence totally disregarded by the Committee. The following points from the data furnished by the above "Cunning Committee" are reassessed by Dr. Faber:

(1) 24 cases of poliomyelitis in the reported series did follow tonsillectomy in 1947 and 62 in 1946. Six (25 per cent) of these were bulbar in 1947 and 18 (29 per cent) in 1946, while the incidence of bulbar poliomyelitis in all cases of poliomyelitis covered by the survey was 11.6 per cent in 1947 and 15.4 per cent 1946.

(2) Since the compilation in 1942 by Aycock and the Editorial of 21-3-1942 in the *J.A.M.A.*, (118. 980: 1942) tonsillectomy has come to be generally avoided during epidemics of poliomyelitis and to a similar extent even during the summer and fall months, because of the widespread fear of the epidemic flaring up.

(3) The proof of a relationship between tonsillectomy and poliomyelitis is positive in respect to the high proportion of bulbar cases and cannot be blindly disregarded.

Aycock's summary shows 170 cases in which poliomyelitis followed within 30 days of tonsillectomy (this coincides with the upper limit of the incubation period) of which 71 per cent were bulbar,

whereas in the 30 to 60 day period only 20 per cent were bulbar. Since the Aycock compilation, numerous other reports of post-tonsillectomy-poliomyelitis, have appeared (including the 86 of the Cuning Committee reports and the 17 of Anderson's paper published in the *Journal of Pediatrics*, 27: 68, 1945). All the post-tonsillectomy cases in Anderson's series were bulbar. *The incidence of poliomyelitis in children in whom tonsillectomy had recently been done was 26 times and of bulbar poliomyelitis 16 times greater than in the general child population.* The Cuning Committee survey is therefore in Faber's opinion a dangerous one particularly as it is in a sense the official report of a responsible and influential body of professional people. "That Cuning himself still had doubts of the correctness of his views is indicated by a remark at the end of his conclusion, to the effect that he does not advocate the indiscriminate removal of tonsils during any severe epidemic and has no desire to place our children's lives in jeopardy through injudicious surgery."

T.N.S.R.

[*Note*.—This above abstract is of topical and special interest in view of the reported epidemic of poliomyelitis in the City of Bombay in July–August 1949.]

T.N.S.R.

Vitamins and anti-vitamins.— (*Nature*, 4130, p. 985-986).

Sir Edward Mellanby, the originator of the modern concept of anti-vitamins, presided over a meeting of the Nutrition Society, at the Royal Society of Arts on 23rd Oct 1948, when an interesting discussion took place on *Anti-vitamins in Food*. Mellanby pointed out that the anti-vitamin or toxamin theory was only 20 years old. His work on substances in cereals which antagonise vitamin D and on phytic acid as an anti-calcifying factor had placed the existence of anti-vitamins beyond dispute. Mellanby's work on the rachito-genetic properties of cereal products indicated that some foods contain substances other than their major components which antagonise the action of vitamins. Flides and Woods showed that the sulphonamide drugs owe their bacteriostatic action to their

biological competition with para-amino-benzoic acid, a compound which has since been recognized to be a vitamin. The sulphonamides may therefore be regarded as artificial anti-vitamins. On the same principle, analogues inhibiting the action of most of the other vitamins have now been synthesized. Dr. E. Kodicek has enumerated five different ways in which the action of B₁ vitamin may be inhibited including (1) Failure of intestinal absorption; (2) destruction or inactivation of the vitamin by bacteria or toxin; (3) inhibition of bacteria producing the vitamin by antagonists or the absence of an essential metabolite; (4) displacement of the vitamin from the tissues by analogues, and (5) exposure of the organism to special stresses such as infections, pregnancy and a bad balance of the major components of the diet.

Several natural toxins have been reported including the thiaminase in live yeast which destroys B₁, a factor in maize which antagonises nicotinic amide and the avidin of raw egg white which inactivates biotin. Phytic acid, a phosphorylated derivative of inositol is the best known anti-calcifying agent in food stuffs. McCance and his colleagues found in human feeding experiments that the absorption of calcium was less efficient with whole meal bread than with white bread as the main article of diet. The

addition of calcium to national diet was, therefore, undertaken to compensate for the increased amounts of phytic acid which resulted from the higher extraction-rate.

The demands of the organism for carotene or vitamin A are influenced by many factors. In the human subject the habitual use of medicinal paraffin may seriously interfere with the absorption of carotene. Early work of Mellanby showed that certain cereal products and also ergot of rye may emphasize the effect of vitamin A deficiency in causing nervous lesions, which his later work has associated with disordered growth of bone. Massive over dosage with vitamin D causes excessive calcification and doses approaching the toxic level have been found to be valuable in the treatment of cutaneous tuberculosis. The liability to the effects of vitamin E deficiency is increased by the ingestion of readily oxidisable fats and decreased by adequate dietary supplies of the lipotropic factors which assist the transference and mobilization of fats. The results of several animal experiments relating to the effects of the deficiency of vitamins K, D, E and A, were also presented at this interesting discussion which sounds a note of warning to vitamin enthusiasts and faddists.

T.N.S.R.

Letters to the Editor

The following are summaries of a few of the letters received by the Editor:

1. Dr. Pratap Singh, Ophthalmic Surgeon, I/c, Medical Officer, Chandavir Hospital, P. O. Bijolian, U. S. Rajasthan.

Protests against the action of the Government in asking young doctors to go to the villages on Rs. 60 p.m. He is of the opinion that the ambitions of the energetic doctor should not be wasted in the villages. He is of the opinion that retired doctors who have not much work to do may be sent to the rural areas on Rs. 60 p.m. plus free lodging and food.

2. Dr. Dwijendranath Banerji, Medical Officer, D. B. Dispensary, Beliaberah (Midnapore).

Writing on doctor's fees deplores the habit of lay papers to criticise doctor's fees and appeals to them from the humanitarian point of view to work for the people. The doctor is of the opinion that the average income of

the Indian doctors is nothing enviable and he is of the opinion that the plight of the village doctor is indeed very bad. In many cases the doctor in the villages has to cycle down or walk on foot long distances over very bad roads, in sun and rain, for a paltry sum of Rs. 2 which is much less than what an unskilled labourer demands for his work now-a-days. He welcomes the suggestion that Government should undertake and manage medical and health activities in the villages.

3. A writer who prefers to be anonymous in a letter to the Editor, deplores the plight of the Rural Medical Practitioner working in Local Boards. He calls upon all Rural Medical Practitioners to stand united for their reasonable rights. He is of the opinion that the remuneration paid by Local Boards to medical practitioners is very meagre. While the cost of living has increased, the emoluments of the Rural Medical Practi-

tioners have not correspondingly increased. He deplores the fact that all Rural Medical Practitioners are treated very badly by the Local Boards. He compares the rates of pay paid to peons, conductors and cleaners in these days with those paid to the Rural Medical Practitioners.

4. Capt. N. Phani, M.B., Palpa P. C. Hospital, Blairaba, E.O. Nawatnwa Bazar P.O. (Gorakhpur) O. T. Ry.

Protests against what he considers inconsiderate remarks made by Dr. P. A. S. Raghavan in his Presidential Address at the South Indian Provincial Medical Conference. He is of the opinion that considering everything foreign as good and everything Indian as bad which was the case during foreign rule should not exist now-a-days. He criticises that Allopathic doctors are agents of foreign firms. He is of the opinion that one of the causes of indigenous medicine not flourishing is because it has not got State support. He goes on to describe how even today in many places patients crowd in the consulting rooms and dispensaries of eminent Ayurvedic physicians and concludes that in proper hands Ayurvedic medicine can compete with Allopathic medicine. He appeals to all patriots to uphold the traditions of Ayurveda and its culture.

5. Dr. N. Law, P. O. Ranchi, B. N. Ry.

Puts in a strong plea for the recognition by the Governments of the day, of the licentiates. In his opinion, it takes full four years after matriculation for a person to get his L. M. P. diploma. The study is hard and the course difficult. The responsibility it involves is great as its sister courses. The expenditure is equally great. He, therefore, deplores and cannot understand why the L. M. P. course is under-estimated. He, therefore requests our National Government to give the L. M. P. course its deserved respect.

6. Dr. R. D. Gupta, Rampur Maniaran, Dt. Saharanpur, U. P.

Writes that there are cases of catarrh and common cold with constant sneezing, flow and irritation of the nose. By repeatedly handling the nose, inflammation of the mucous membrane and nasal cavity takes place, with the result that even the slightest touching of the nose affords great pain. In addition to catarrh mixture the doctor has tried successfully nasal drops prepared by himself containing:

Menthol	0.5%	} Used in an automizer
Ol. Limon	0.5%	
Paraffin Liq.	1 oz.	

In his opinion this stops sneezing and flow of the nose etc. but does not properly help lessening of the inflammation and pain. He wants the readers of the ANTISEPTIC to help him on this point.

7. Dr. C. Kumaraswamy, L.M.P., Walajpet, North Arcot Dt.

Writes about the complaint of Dr. H. R. Hodge that practitioners of Indian medicine compete with Allopathic practitioners and use Western medicine without knowing fully what they are and how they act. According to Dr. Kumaraswamy, compounders have been registered by the Central Board of Indian Medicine and they are responsible for the troubles mentioned by Dr. Hodge. He complains that this quackery is due to the encouragement given by Allopathic practitioners. He urges on his compeers not to encourage quackery. He appeals that Inspecting officials appointed under the Drug Control Order, must prohibit the supply of Western Medicine to these quacks.

8. Research Committee, Mangalwarpeth, Kolhapur, would like to know the effects after baking, frying and steaming on the elements such as proteins, fats, carbohydrates etc. in principal food articles such as fruits, vegetables, milk, eggs etc. They also would like to know what action it has with respect to digestibility, absorbability and assimilability. For the present they would like to know the effects on Cholam (JUAR). A reply on this point may be sent direct to Research Institute, (Practical Branch) Mangalwarpeth, Kolhapur

News and Notes

1. The Fourth S.I. Provincial Medical Conference will be held from October 8th to 10th in the spacious premises of the Annamalai University. An exhibition of pharmaceutical preparations and scientific appliances is also arranged along with the Conference.

The delegate's and visitor's fee is fixed at Rs. 5. Accommodation for ladies is being arranged in the Ladies Hostel of the Annamalai University. An influential Reception Committee has been formed with Dr. Nataraja Jatavallabhar as Chairman, Drs. S. Chandrasekaran and Jagannadham as General Secretaries and Dr. V. Viswanathan as Treasurer. Members of the medical profession are requested to contribute scientific papers to make the Scientific Section interesting.

Those who want further information with regard to the scientific section or the exhibition are requested to get in touch with the General Secretary, Dr. S. Chandrasekaran, Cuddalore N.T.

2. Nuclear Physics Applied to Medicine. —The first 30,000,000-volt machine for the generation of high voltage X-rays has lately been installed at the Royal Cancer Hospital in London. The London synchrotron is part of the equipment of a new laboratory for the application of nuclear physics to medicine. It is expected that the next year will be devoted to preliminary measurement and experiment. It is reasoned that the greater the energy of X-rays the greater is the depth

within the body at which the maximum dosage is produced. This, however, is normally limited by the risk of surface burns on the skin. The London synchrotron has an unusual international history. It was in 1945 that the idea on which it is based was suggested by Prof. V. Veksler in Russia and Dr. E. M. McMillan in the U.S.A. In Great Britain, Prof. M. L. Oliphant was working on a similar project and it was in 1946 when electrons were accelerated to a mere 8,000,000 volts of energy in the first experimental machine of this type. By the summer of 1947 a team of medical research workers was carrying out tests with a 12,000,000-volt machine at the Malyern station. The Cancer Hospital synchrotron is housed in a basement laboratory. The first stage of the investigation will be to define the physical limits of the X-ray beam. At the same time, a careful investigation will be necessary to check the initial protection which has been provided. Then, in the final stage, the effects of the rays will be studied on a number of organisms, animals and different kinds of tissues.

3. Vaidyaratna Capt. G. Srinivasamurthi's 'Statue' Fund: It has been decided at the 18th Provincial L.I.M. Conference held at Madras, to take steps to present a Statue of Vaidyaratna Capt. G. Srinivasamurthi, B.A., M.B. & C.M., Retd. Principal, Govt. School of Indian Medicine, on the occasion of the Silver Jubilee celebrations in January 1950. Capt. Srinivasamurthi was practically the father of the Institution and credit to its present position must naturally go to the doctor. It is estimated that the cost of the Statue will be about Rs. 15,000. We appeal to every alumnus and every member of the institution and other admirers of Capt. G. Srinivasamurthi to liberally contribute to this fund to enable the Committee to place an order for the Statue. Cheques and remittances may be sent to Dr. Janani V. Badarinarayana, L.I.M., 2, Brodies Road, Mylapore, Madras.

4. At the meeting of the Staff Association, R. A. College, Hardwar, held on 29th June 1949, a resolution expressing their deep sense of sorrow at the untimely demise of the late Shri M. K. Mukherjee, ex-President, of A.I.A. Congress, President, A.I.A. Vidyapeetha and the Principal of Rishikul Ayurvedic College, Hardwar, was passed.

5. Dental Surgeries on Wheels:—Kent County Council is the first County in England to have Dental Surgery on Wheels, visiting villages in the County and treating school children and expectant mothers. These Dental Surgeries on wheels have been fitted with most modern equipment, operated by special portable generators. They also carry

X-ray apparatus, anæsthetic appliances, and gas and electrical drills. The crew of each will consist of one dentist and his assistant.

—Br. Inf. Services—B. F. 1073

6. Teaching Surgery Through Television:—With the installation shortly of special television apparatus, Guy's Hospital, London, will become the first institution in the world, to have permanent television as an aid to teaching medicine and the first in Europe to televise operations. As the skilled fingers of surgeons operate, their every move will be watched by students sitting outside the operating theatre. A camera will be suspended over the operation table and worked by an engineer from a remote control box. The camera will have three lenses, enabling close ups and long shots to be taken as well as a full view.—Br. Inf. Services—B. F. 707

7. £ 10,000,000/- endowed for Research-working of Nuffield Foundation. Many millions of pounds have already been given by Lord Nuffield for philanthropic and scientific purposes, including a £ 2,000,000 (Rs. 2.66 crores) grant to Oxford University. In all, Lord Nuffield has made gifts totalling about £ 16,000,000 (Rs. 21.30 crores). The object of most of these benefactions is the advancement of health and the prevention of relief of sickness and the care and comfort of the aged poor. During the first four years, the Foundation made enormous grants for the study of chronic rheumatism, for industrial health research, for research purposes in nuclear physics and radiation, for travelling fellowships and scholarships for teachers and other deserving objects.—Br. Inf. Services—B. F. 231.

(We wish the philanthropists and millionaires in our country to follow this splendid example of Lord Nuffield).—Eds. ANTISEPTIC.

8. Flying Squad to Fight Infant Mortality: A flying squad of nurses and doctors who can be sent anywhere at a moment's notice has been organised in London in the fight against gastro-enteritis—a children's disease which, if not quickly combated, may lead to very high death rates. Gastro-enteritis is one of the most dangerous of children's diseases. The danger spot lies in dehydration of the infant body, which, if allowed to progress, becomes irreversible.

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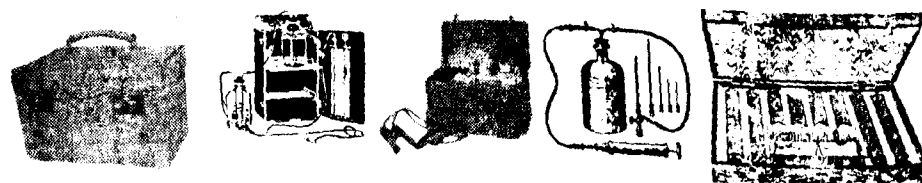
This mobile unit or flying squad not only carries out the modern treatment but in the minimum of time, can instruct doctors and nurses at the various hospitals on the best methods of treating and combating gastro-enteritis.—Br. Inf. Services—B. F. 290.

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"The Medical Review," Vol. VI, No. I.
"The Lancet," No. 3923, Vol. CLV.
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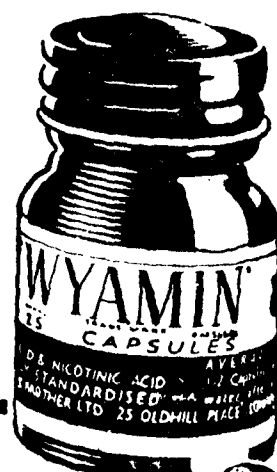
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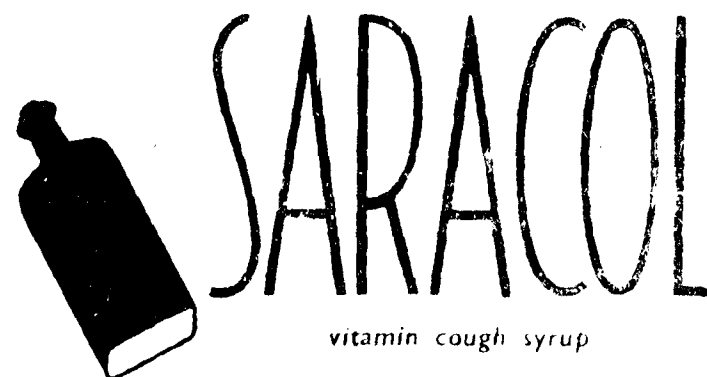


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Each fluid ounce of the preparation contains:

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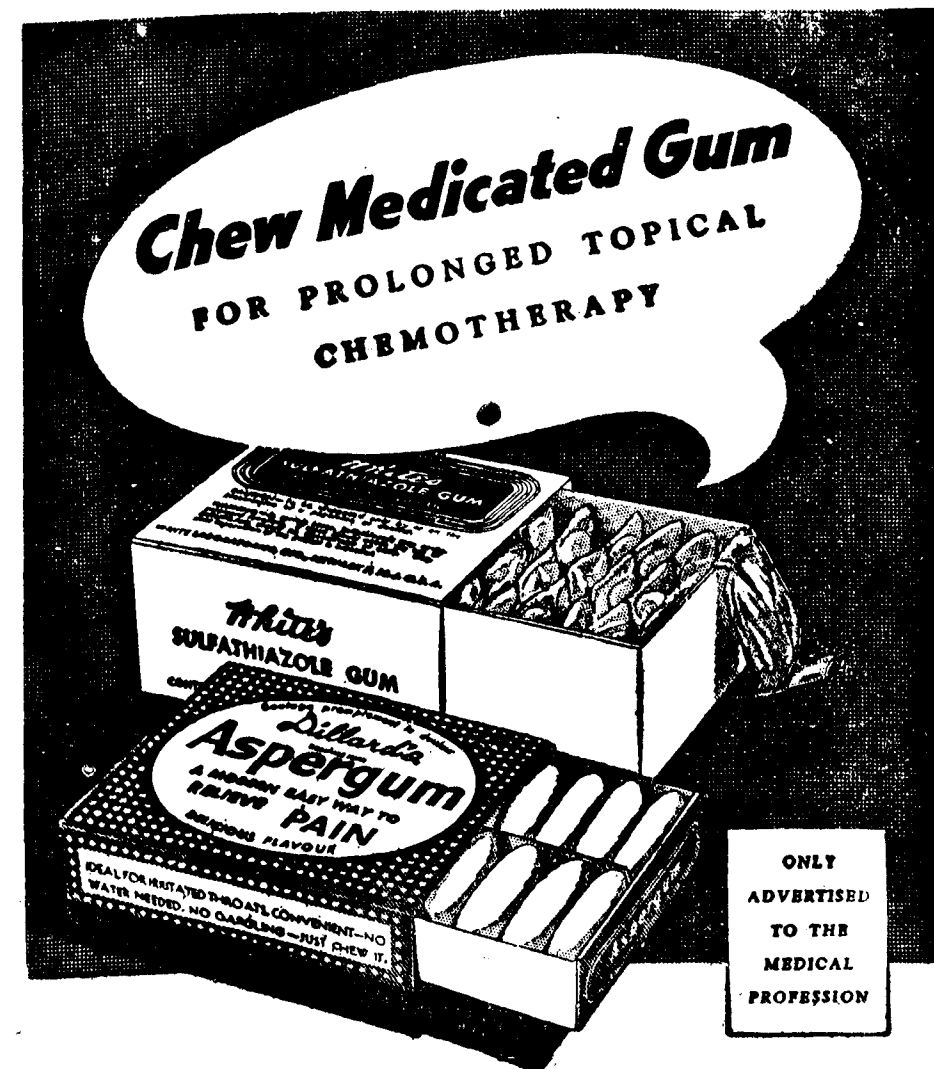
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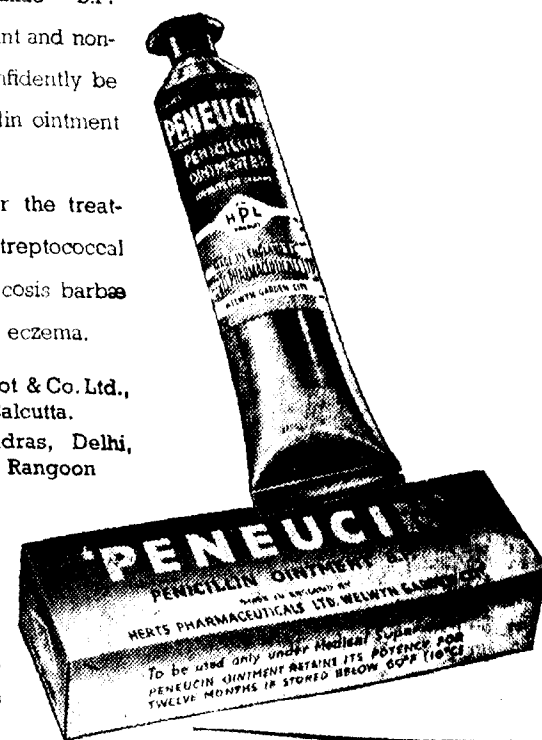
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In 'PENEUCIN' brand of penicillin ointment, the penicillin is compounded with 'Eucerin' Anhydrous, our well-known brand of Ung. Alcoh. Lanae B.P. 'PENEUCIN' is a stable, elegant and non-irritant ointment that can confidently be prescribed whenever penicillin ointment is required.

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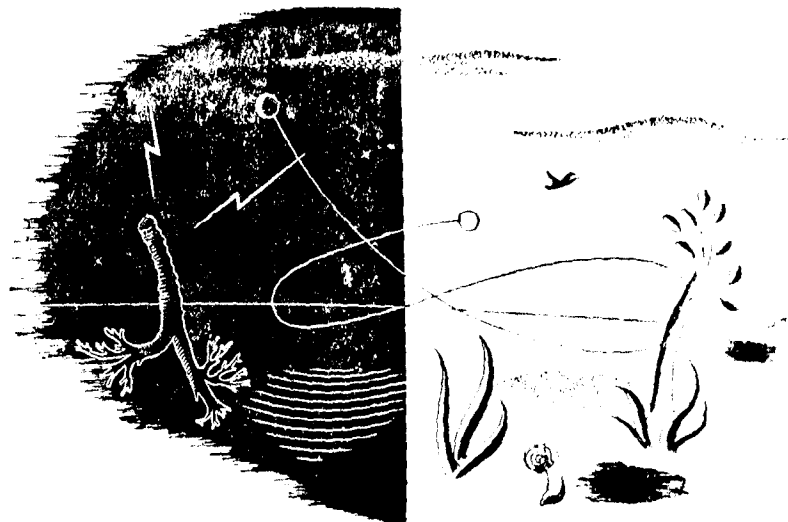


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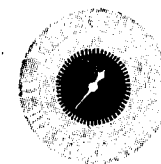
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seconds become MINUTES.



During wakefulness associated
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Sound sleep can be attained in fifteen to
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relax smooth muscle spasm
rapidly!

Smooth muscle spasm, the immediate cause of pain in intermittent claudication and URETERAL AND RENAL COLIC, may be relaxed rapidly by intramuscular injection with 'Depropanex' deproteinized pancreatic extract. This antispasmodic agent is effective also in the treatment of spastic ureteritis and severe PRIMARY DYSMENORRHEA.

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DEPROTEINATED PANCREATIC EXTRACT

Sharp & Dohme
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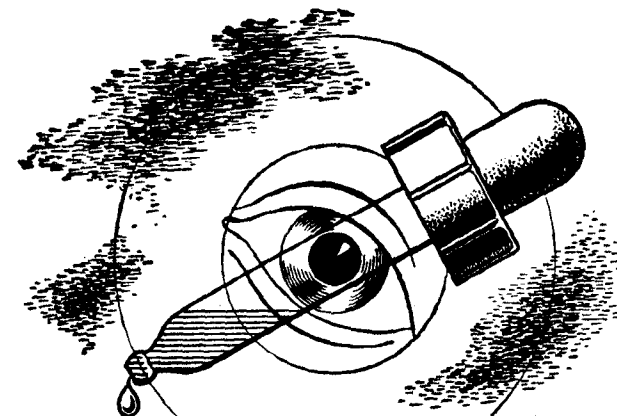
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For
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it is essential that local chemotherapeutic agents used in ophthalmology should be non-irritant. 'ALBUCID' Soluble Eye Preparations are adjusted to pH 7.4 and are thus free from irritating alkalinity. Effective penetration of stable bacteriostatic into the tissues is achieved and an adequate concentration can be maintained. Some of the main indications of 'ALBUCID' Soluble Ophthalmic Preparations are listed above.

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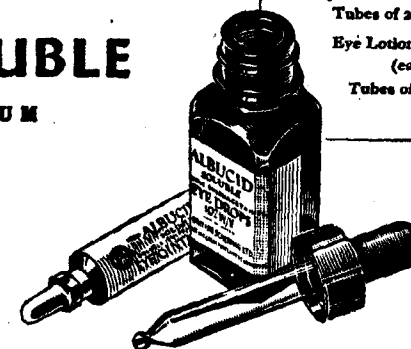
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- Eye Drops, 10% and 30%
Bottles of 12.5 c.c.
- Eye Ointment, 2½% and 6%
Tubes of 4 gm. (approx.)
- Eye Ointment, 2½%
Tubes of 25 gm.
- Eye Lotion Tablets
(each 0.25 gm.)
Tubes of 12 and 50
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LIVER-IRON-VITAMINS

Injectable.

HEMAREX provides the physician with a means of supplying iron, liver and the essential B vitamins in a form which possesses the advantages of potency plus a high degree of gastric tolerance.

Each 2cc. contains a sterile aqueous solution of 100 mg. of liver injection 10 U.S.P. Units (injectable), unitage of liver determined prior to compounding, iron (as colloidal iron oxychloride), 19 mg., niacinamide 50 mg., riboflavin 0.3 mg., pyridoxine hydrochloride 0.3 mg., and phenol 0.5% as a preservative.

HEMAREX is supplied in 2cc. ampuls in boxes of 12 and 100. Also 10cc. and 5cc. multiple dose, rubber capped vials.



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Capsule.

BETAPAN Capsule is recommended for treatment of Secondary, Nutritional and other Microcytic Anaemias. Each capsule represents: Liver Extract (1-20) 7 grs. (derived from 140 grains fresh liver), Ferrous Sulphate Exsic. 1 1/2 grs., Vitamin B1 60 I.U., Vitamin B2 100 Micrograms, with other factors of B-Complex as found in fresh liver.



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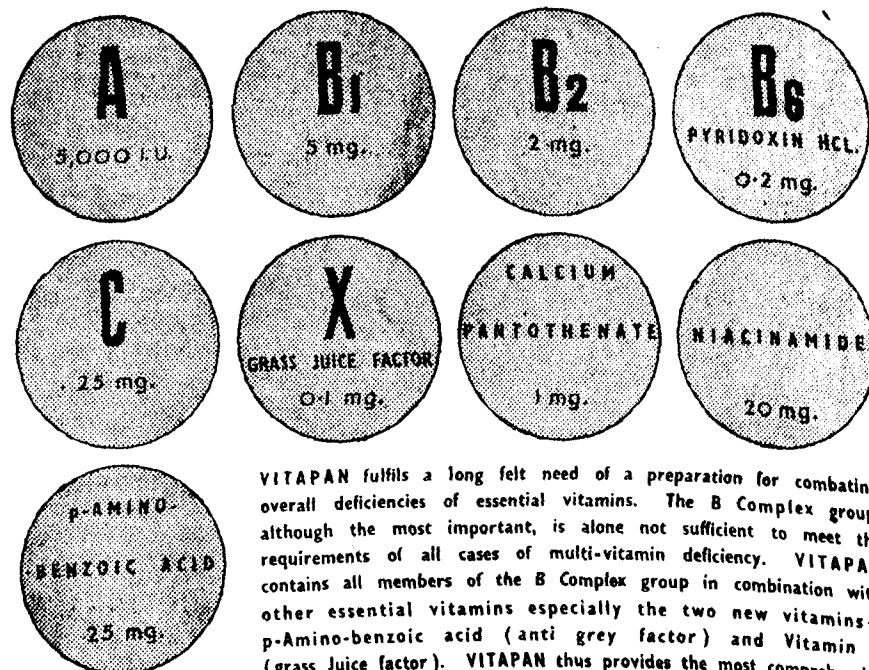
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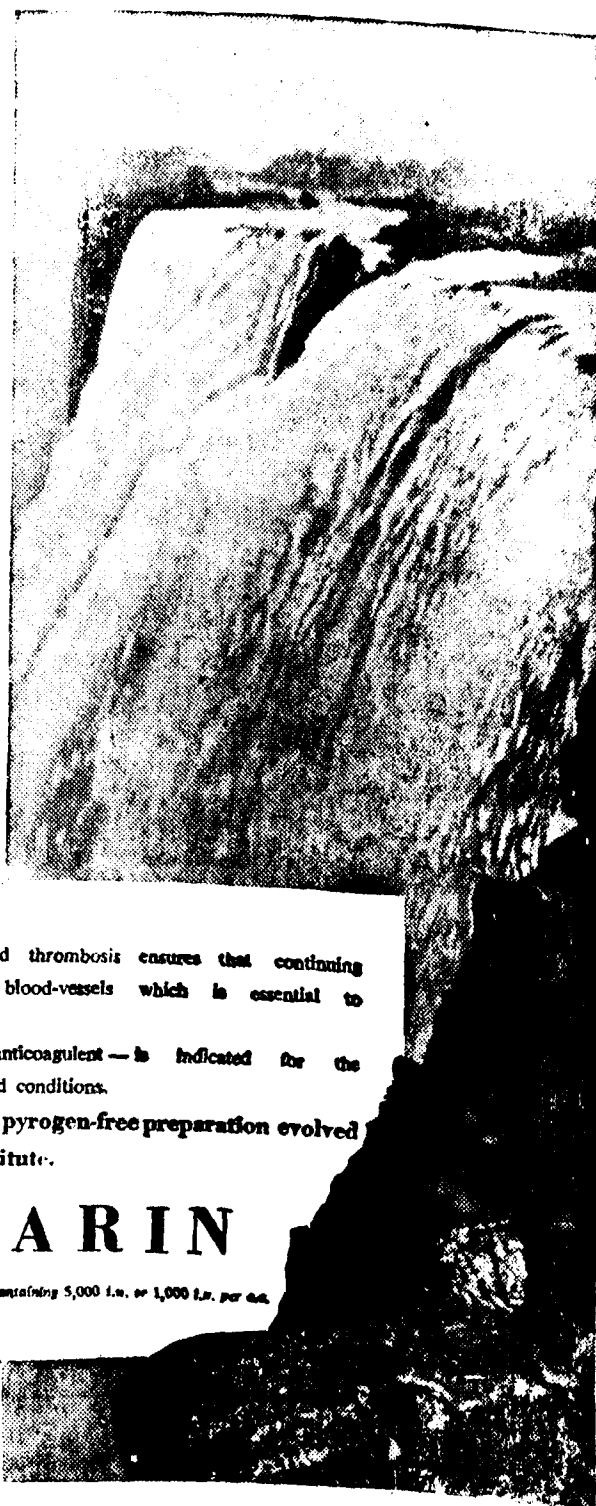
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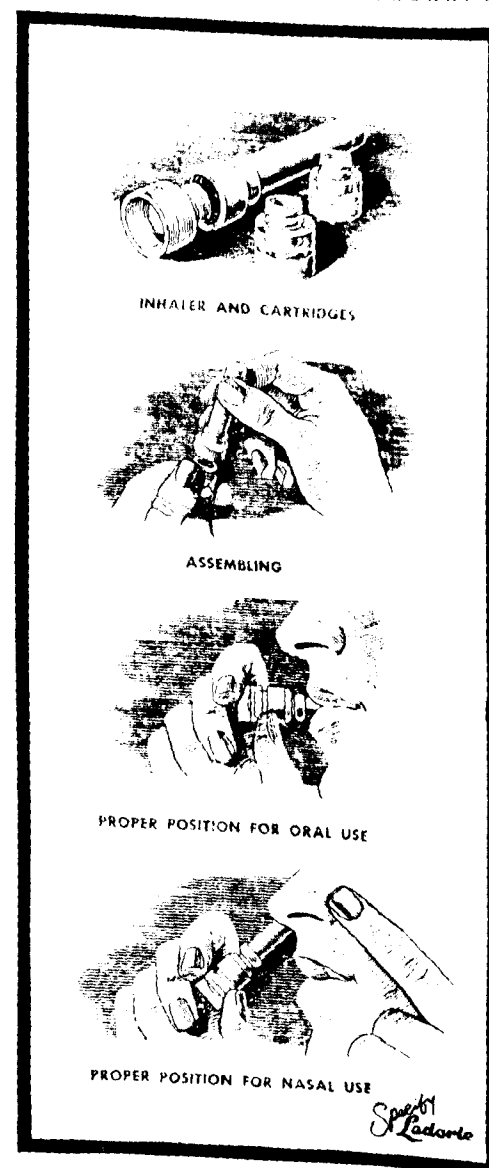
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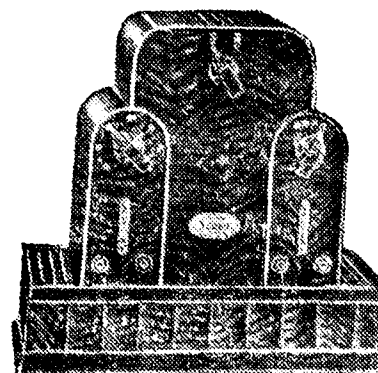
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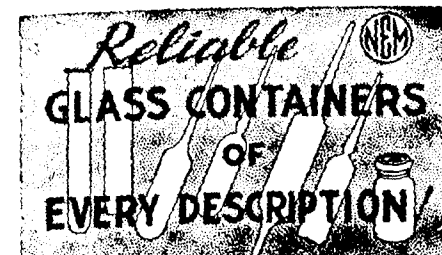
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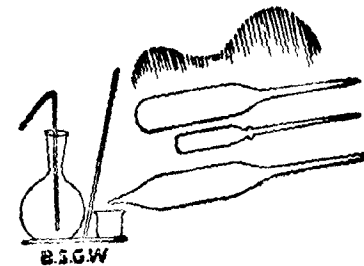
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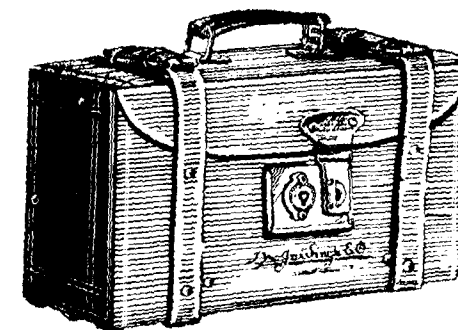
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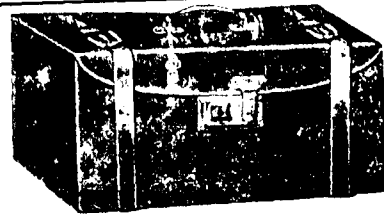
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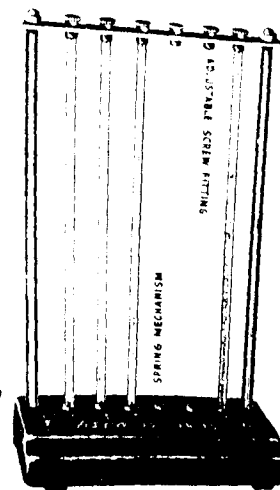
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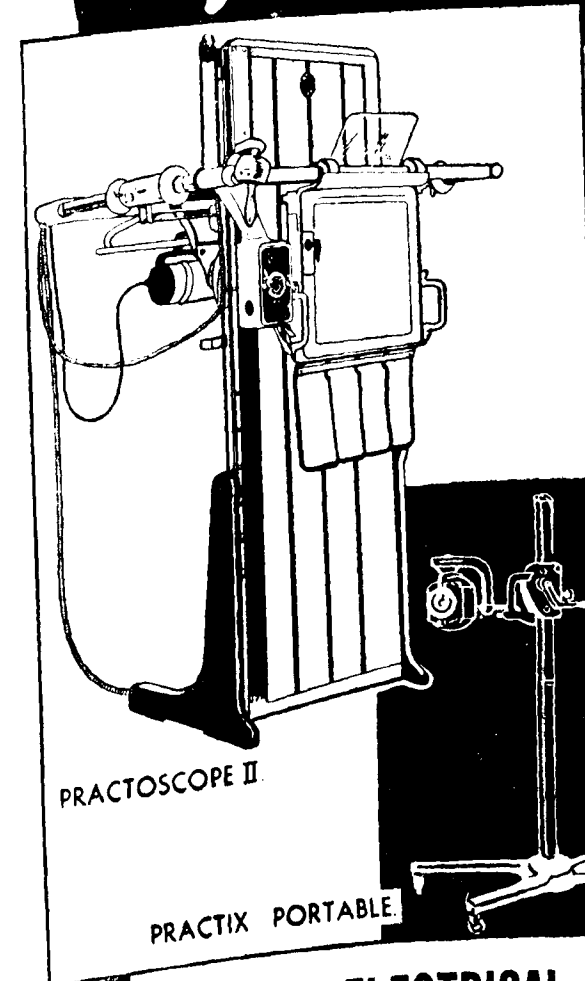
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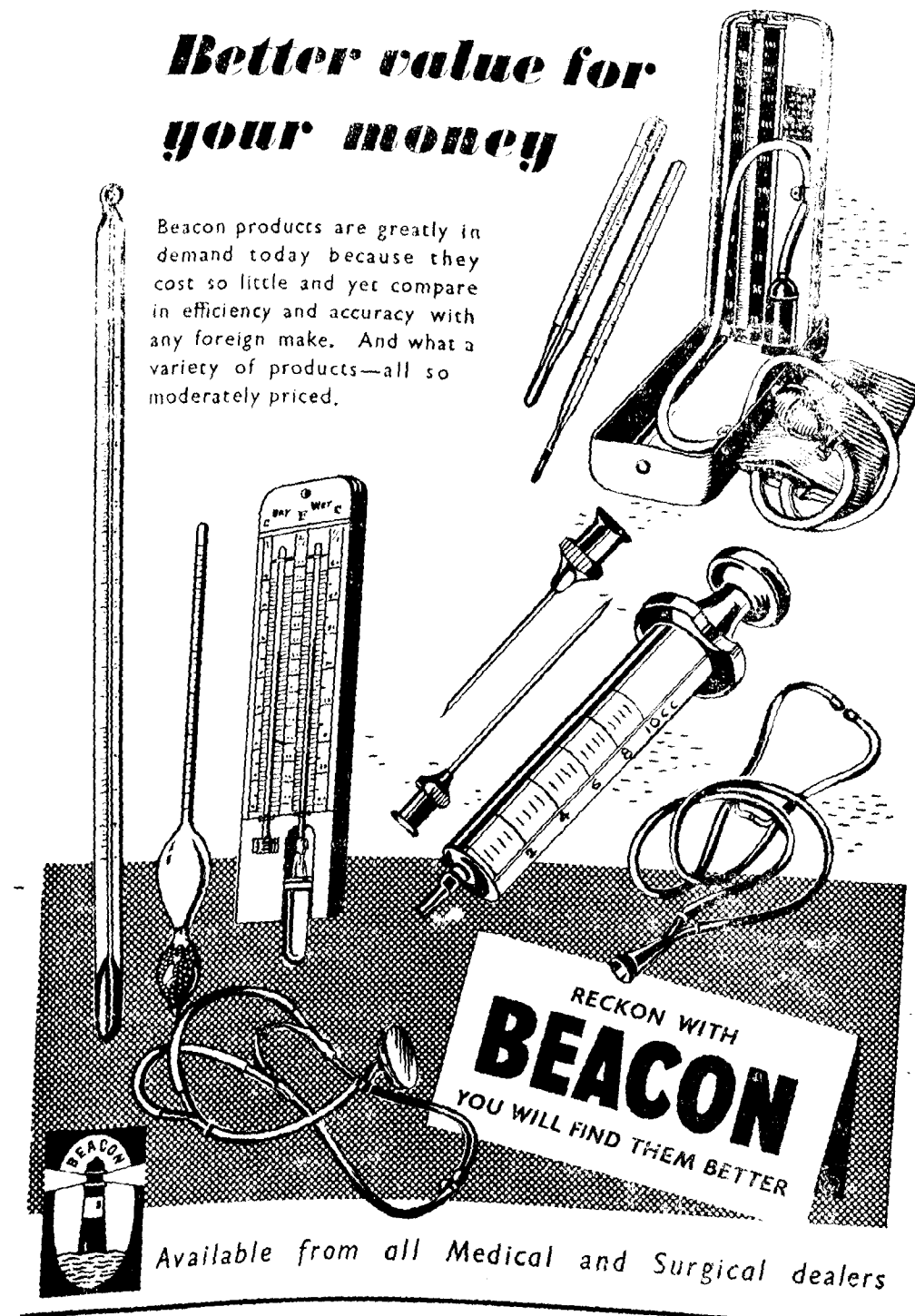
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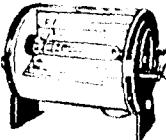
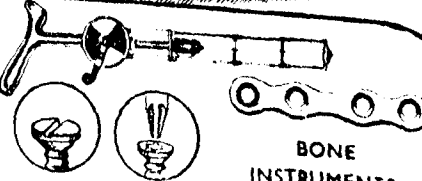
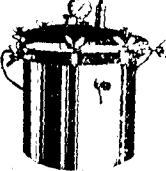
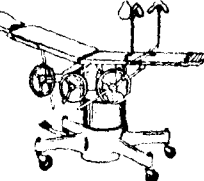
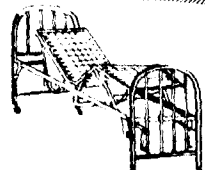
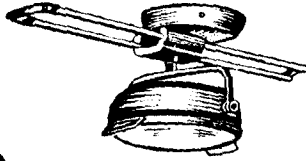
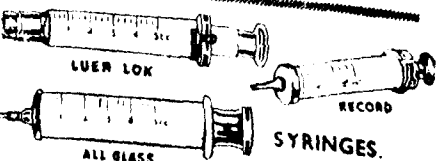
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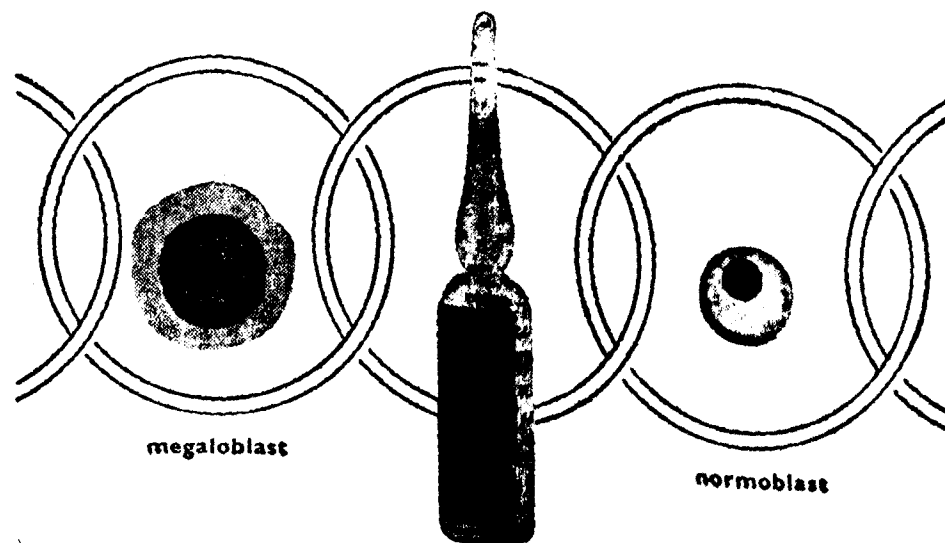
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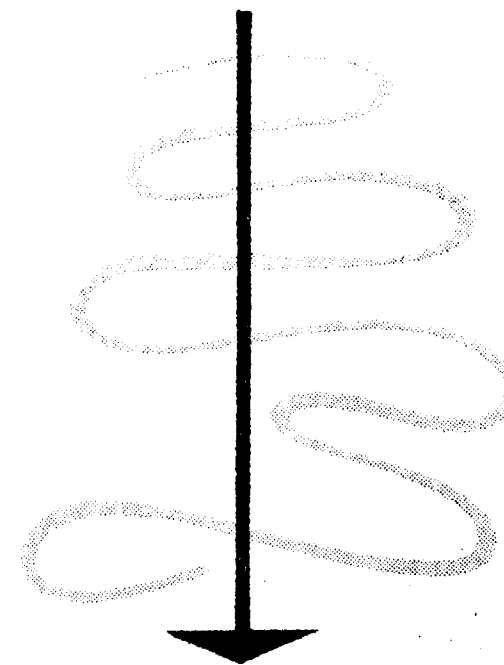
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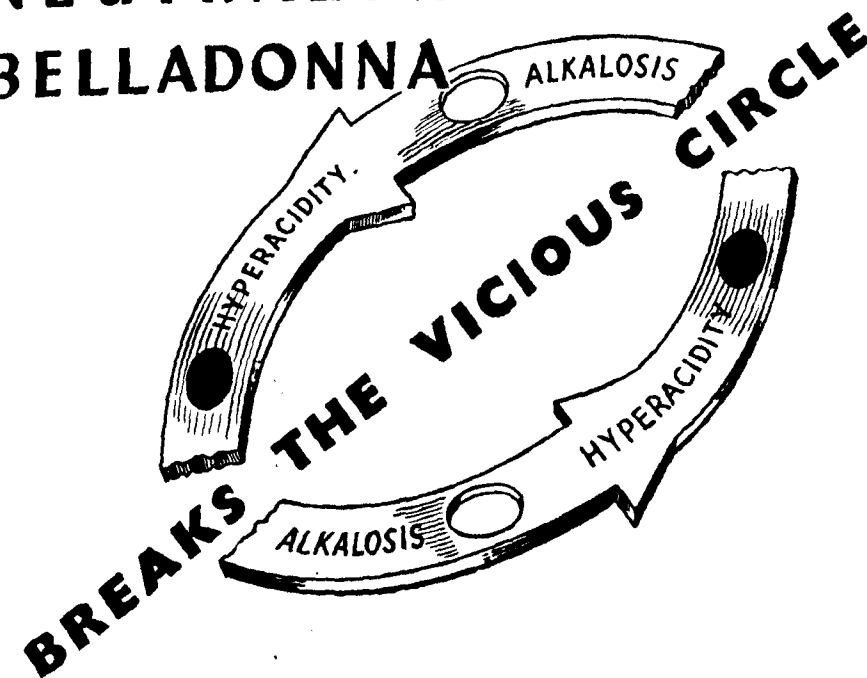
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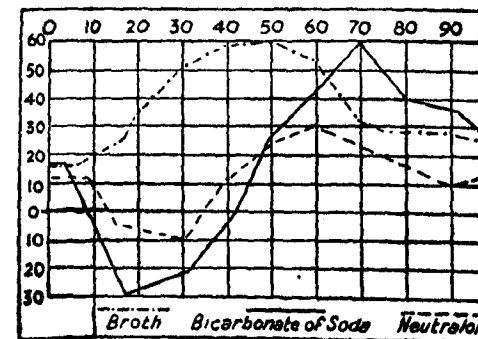
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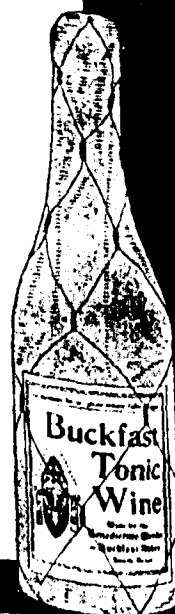
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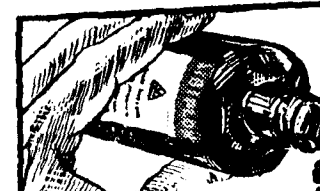
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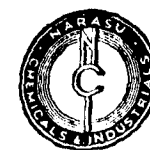
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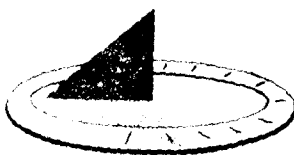
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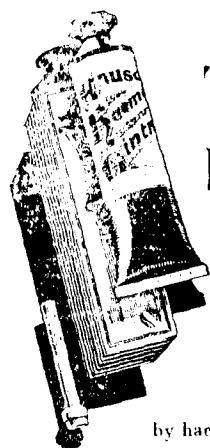
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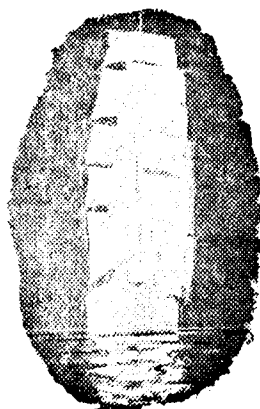
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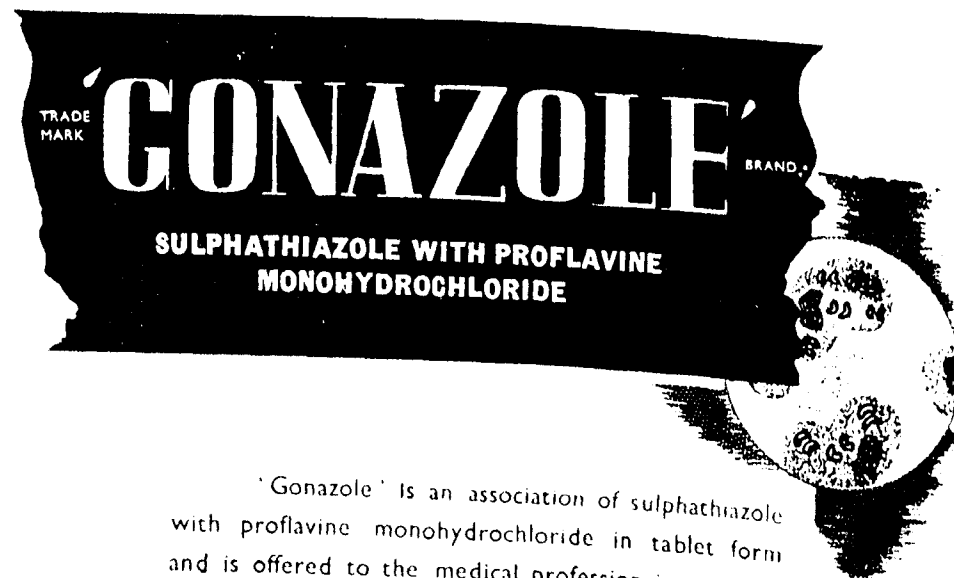
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OCTOBER, 1949

No. 10.

Original Articles

Prognosis in Heart Disease*

R. SUBRAMANIAM, B.Sc., M.D., M.B.C.P. (Lond),
Physician, Government General Hospital, Madras.

THE subject is a wide one and is rapidly advancing with surgery encroaching into the field.

Prognosis in any field of medicine has been a rock on which the greatest of physicians have been ship-wrecked and more so in the field of cardiology. Even so, prognosis in heart disease is of very great importance.

Family history is of great importance. It is essential to assess whether other members of the family have had the same malady and with what result. If there is a tendency for early death in other members, then the chances are not very good. I know of a rheumatic family in which one member has aortic regurgitation of rheumatic origin. His nephew of about the same age, died very young of rheumatic mitral insufficiency. His niece had rheumatic mitral lesion with auricular fibrillation. In this family, I take it that the death of one member in a group is not of serious significance in so far as the deceased did not have proper medical aid from the start. Hence the family history may only give an idea of the general trend of events likely to occur, and individuals are liable for wide variations in a group. In assessing individual patients, due consideration should be given to variations from the group.

The next factor to be considered is the age of the patient at which the cardiac affection is manifested. As a general rule, the

*Specially contributed to THE ANTISEPTIC.

younger the age at which symptoms occur, the worse the prognosis. Most congenital lesions carry a poor prognosis. Congenital defects vary widely. The lowest in the scale is the acardiac monster which is incompatible with life and there are other endless variations. The variations commonly met with are patent interventricular septal defect, patent inter-auricular septal defect, patent ductus arteriosus, dextroposition of the aorta, pulmonary stenosis, mitral stenosis and petit aorta with gross pulmonaire of French writers (*i.e.* small aorta with big pulmonary artery). The defects may occur either as an isolated phenomenon or in various combinations. For instance, it is only quite recently that I had a case verified at autopsy, in which the heart specimen showed patent interventricular septum, patent inter-auricular septum defect, patent ductus arteriosus, tricuspid insufficiency, mitral stenosis with insufficiency, small aorta and a very grossly dilated pulmonary artery. Multiple defects are very often noted at autopsy and some of them are not even thought of by the clinician. For example, in the case just cited, in the wards we only thought of patent interventricular septal defect and mitral stenosis with regurgitation.

For our purpose, congenital heart defects may be divided into two broad groups: (1) cyanotic and (2) the acyanotic. In the cyanotic group admixture of venous blood occurs in the arterial stream. These are the so-called blue babies. These cases have a bad prognosis. The other group, *i.e.* the acyanotic group in which arteriovenous shunt occurs, cyanosis occurs only with the onset of congestive failure. A good example of this is the patent ductus arteriosus. In this group the prognosis is very much better and the real danger is only the possibility of supervention of bacterial endocarditis. Bacterial endocarditis occurs not only in septal defects but also when semilunar valves instead of being tricuspid happen to be bicuspid.

Generally the prognosis is better as age advances in the acquired heart lesions, because the coronary vessels tend to form greater and greater anastomosis. Coronary thrombosis in the aged is far less serious in view of this and seldom proves fatal. Given the choice, under identical conditions, I prefer to look after an old man with coronary thrombosis than a younger man. In the younger age group coronary vessels should be considered as endarteries. At this stage it is worthwhile to recall that the arterial supply of the brain and the heart are different from the rest of the viscera. In these two organs, the vessels form a cage as it were and vessels pierce at various angles. This arrangement causes a maximum damage to the endocardium and minimum damage to the myocardium. In the other viscera, the vessels reach a hilum and branch out towards the periphery, so that the maximum damage is at the periphery and the minimum damage at the centre. The importance of this method of blood supply is that the signs and symptoms are minimal

and the sequelae may be far out of proportion to the clinical manifestation.

The next factor to be considered is the sex of the patient. For some reason or other, girls have a better prognosis than the boys until the time of puberty. From this time onwards, throughout the child-bearing age, the prognosis is bad in woman and if she has survived the child-bearing period and menopause has set in, once again she has a better prognosis than man. This is partly due to the fact that man has by nature to bear the burden of life and has to lead a more strenuous life, being exposed to greater hardships. The more sheltered life of the woman is a great asset provided she can avoid pregnancy. Some of the women with valvular defect do very well and may go through more than one confinement admirably. All the same, it is a wise precaution to help these patients with a low forceps and cut short the labour time. It is said mitral stenosis patients may be permitted three confinements, mitral regurgitation two or three, aortic stenosis one with safety and aortic regurgitation none. Occasionally, however, I have seen these rules neglected and patients being none the worse for it. I have also seen a mitral stenosis patient dying during the first confinement even before the labour was over. It is not so much the valvular lesion that matters as the myocardial efficiency. Unless one has a good idea of the patient's condition before pregnancy occurred, it is not possible to have a proper assay during advanced pregnancy to correctly judge the heart condition.

I have known a female patient admitted for mitral stenosis in a fully decompensated state. She responded very well to treatment. She was already the mother of two children and so she was sterilised and a subtotal thyroidectomy was done. She lived completely without any further deterioration for six years and then broke down and lived for a year after that. This case illustrated very well how the woman was helped along by two procedures, namely, an artificially induced menopausal state preventing conception, and a subtotal thyroidectomy lowering the B.M.R. and thus taking away a part of the burden on the heart.

The occupation of the patient is a very important consideration in arriving at the prognosis. If a heart patient is a manual labourer and has to earn a living by the sweat of his brow, the prognosis is definitely bad. In this connection, I will just narrate two cases which will better illustrate what I have got to say. The first case is that of a clerk in a Panchayat Board Office. His office and residence were in the same building, and, except on very rare occasions, he would do his work at his own leisure. This man was admitted with well-advanced congestive failure signs and symptoms. He responded very well to treatment and he was under observation for one year without developing failure. He used to report once a month and kept up well. Unfortunately, this man had an attack

of intercurrent infection and he died suddenly. The second case is that of a road cooly of the Madras Corporation. This man had also mitral stenosis and a systolic murmur on the right side of the sternum in the third right intercostal space. We thought this might be due to patent interventricular septal defect very high up. This murmur used to become very evident when the congestive failure was prominent and the murmur would be poorly marked when the congestive failure symptoms cleared up. We screened the patient but could not find any hilar density to account for patent inter-auricular septal defect, but we could only make out a mitral configuration with a poorly marked aorta. This patient used to respond to treatment and get over the congestive failure symptoms and would be back in the hospital within a week of discharge as bad as ever. So we decided to keep him after the fourth admission for some time and we kept him for nearly two months after he got over the failure symptoms. Then due to heavy admission, we had to discharge him. He came back in ten days' time in a very advanced state of failure, gasping for breath, and this time he succumbed. These two cases show very well how, when a patient can work quietly and lead a sedentary life he is able to go on, and, on the other hand, a manual worker could hardly manage for a week without slipping back into a desperate state. The manual labourer fortunately died in the hospital and a partial postmortem was conducted. This showed that the patient had advanced rheumatic mitral stenosis and no septal defect but a very small aorta and a very large pulmonary artery. Evidently what had happened in this case was that the left ventricle by losing its tone during attacks of failure was slightly dilated and the blood flow through the narrowed aorta was producing the murmur. The murmur over the third right intercostal space of course did not disappear at any time, as the congenital anomaly was always present. It is a pity we have no convalescent homes to take care of these congestive failure cases and rheumatic children once the acute phase is controlled in the hospital.

Even a sedentary worker, if he had to stick to a regime and has to hurry through his work, he is bound to be worse off, than the man who can choose his own time to attend to the office and can dispose without having to finish a particular quota in a given time. In other words, the better the social status of the man, the greater the possibility of his being able to lead a quieter life and so have a much longer span of life. Cardiac patients need ample rest not only at week ends but also they need longer rest period by night, i.e., they should be in bed for longer number of hours than others, though they need not actually sleep for all the hours that they are in bed. Also they should take one hour's rest at least after the noon lunch. They should also not hurry through their lunch. All these are possible only for a small group of people carrying on independent business.

After having considered these general features about the case, we must consider the general health of the patient subsequent to the commencement of the cardiac affection. Supposing two children 'A' and 'B' are attacked by rheumatic fever and both of them develop rheumatic mitral stenosis. 'A' may be enjoying very good health and a pre-systolic murmur in the mitral area may be the solitary abnormality. On the other hand, in the child 'B,' apart from mitral stenosis the child may frequently be falling sick, probably several relapses of rheumatic fever may occur and the general condition of the child may be far from satisfactory. Obviously the prognosis is definitely better in the child 'A' rather than in the child 'B.' Now and then one comes across even very old people with a mitral stenosis in the course of routine medical examination, showing thereby that the cardiac affection is not by itself a very serious thing, provided the general health is kept up and the myocardial tone is good.

Now we must consider the cardiac affection itself. The ætiology of the affection is of vital importance. If we come across a case of congestive failure, we have to determine the aetiology of the failure. If the failure is due to valvular lesion, then it is not so serious as failure subsequent to coronary thrombosis. In the latter case, recovery is very poor and even if at all it occurs, it takes a long time and even then it is not so complete as in other cases. Myocardial fibrosis due to syphilis is of much graver prognosis than that due to rheumatic infection. The damage done due to syphilis is irreversible and no amount of anti-syphilitic drugs can undo the mischief. In rheumatic myocardial fibrosis, on the other hand, bed rest ensures an amount of recovery which does not occur in other diseases. The same applies to the ætiological factor in other cardiac affections.

The same ætiological factor might affect differently in different persons and so the variety of the lesion is an important point to be considered. For instance, rheumatic fever might cause in one patient mitral stenosis, in another aortic regurgitation, in another pericarditis or rarely myocarditis. As you know, the prognosis is very much better in mitral stenosis than in the others provided it is not a progressive lesion. If, after the cardiac lesion is noted, the patient is not incapacitated by the same, it is of a better prognosis in so far as it shows that the myocardium is compensating in spite of the deficiencies. If the cardiac lesion is a recent one, then it carries a graver prognosis than a longstanding cardiac lesion. The longstanding one indicates that the lesion irrespective of the cause is not active, whereas we cannot be certain that the lesion is quiescent when it is of recent origin. As we know sometimes, mitral stenosis is progressive. The stenosis gradually gets worse and may become so great as to cause the auricular ventricular conduction time to be prolonged and thus resulting in the first degree of block.

This first degree of block can only be made out by electrocardiograph. In some others, mitral stenosis may not show such bad prognosis. So, in a recent case, we do not know whether we are dealing with a progressive lesion or a relatively stationary one. Also if a lesion has lasted for a time and the patient is able to get along, it shows that the heart condition is not enough to cause anxiety.

The most important advance in prognosis was made by Sir Thomas Lewis permitting a man with a systolic murmur to go up in the Everest expedition. He ignored the systolic murmur in view of the tone of the myocardium being good and subsequent course of events proved the soundness of his judgement. So when the myocardial condition is good, the murmur can be ignored. The integrity of the myocardium should be assessed before judging the prognosis. Even while auscultating, the concentration should be on the first sound in the mitral area and the murmur should be ignored. Sir Thomas's attitude fortunately acted as a good corrective and physicians have subsequently learned not to be led away by the murmur. Murmurs are pointers about the defects and no more. Their presence is not an indication for giving a grave prognosis. If a man who can do a normal physical exertion without being incapacitated in any way, has a murmur, whether loud or soft, he has a better prognosis compared to the man with no murmur but who gets breathless on exertion even if moderate. So the factor of the greatest importance is the integrity or the capacity of the myocardium to carry on even in the presence of defects and infection.

Cases with cardiac failure always carry a worse prognosis, the degree of failure indicating the prognosis as more or less serious. If a patient cannot exert and is breathless even at bed rest, the prognosis is much worse compared to the man who gets breathless only on exertion. Also the cases which show signs of failure like œdema of the legs or dependent parts, enlarged tender liver, congestive rales at the bases, prominent jugulars always carry a worse prognosis compared to the case in which these are at a minimum or not present. In other words, where the signs of congestive failure are patent, the prognosis is worse than where they are latent or not prominent. The day a man gets an attack of breathlessness doing a normal piece of work which he has done all along without getting breathless, it indicates a case of failure and one should not wait for the florid signs of failure to manifest. In the words of a famous cardiologist, a hill has appeared in the path of the patient where there was no hill previously. Everybody gets breathless on going up the hill. This patient has been doing the work all these days without getting breathless. Now on a particular day doing the same piece of work, he gets breathless as if he has climbed a hill, i. e., a hill has appeared in his path, which was not present on all

previous occasions. On that day he should be considered as a case of congestive heart failure even though all other signs are lacking.

All heart cases carry the risk of sudden death but particular types carry it to a greater extent than others. Particularly cases of aortic regurgitation, more often due to syphilis rather than due to rheumatic infection, coronary artery thrombosis, when occurring in the younger age group and often during the first attack. When death occurs due to coronary thrombosis or occlusion, at the first attack probably it is due to the shock being very great. Also in the younger age and in the first attack anastomoses is lacking. Given a case, it is impossible to say what will be the prognosis. I have seen cases in which the pain was quickly alleviated by administration of Morphine very soon after the onset of the attack, and yet the patient died in half an hour. I have also seen cases in which the heart was going fast with a feeble pulse and yet the patient recovered. If the patient does not die very soon after the attack and has pulled through the first 24 or 48 hours, then there are certain guides: If the blood pressure remains steady or does not fall down rapidly, the chances are that the case will recover. The systolic pressure should not go down below 60 m.m. The pulse rate which went up very markedly usually settles down in 24 or 48 hours. It should be less than 100. The fever that invariably occurs should cease by the third or fourth day of the illness. The longer the fever lasts, the worse the prognosis. If the temperature does not clear up in a week, an inter-current complication should be thought of. At this stage I will recall a case. A gentleman of 59 years of age had an attack of coronary thrombosis. He returned home and developed fever by the evening. The diagnosis of coronary thrombosis was missed because of the temperature. He was treated as a case of influenza, but the temperature was persisting, with an evening rise. When the temperature had lasted for a week, he was brought to my notice. In view of the typical history, coronary thrombosis was thought of and Morphine given and later sedatives. The pain ceased and the temperature also ceased for a day but began to recur. At this stage, physical examination was completely negative. In view of the temperature persisting an X-ray chest was taken to eliminate an incipient tuberculosis. X-ray showed a small radiating shadow in one hilum. In view of this, patient was put on Streptomycin. As the patient was getting Streptomycin, a check-up picture taken ten days later showed the original patch had cleared and a fresh one had appeared. This put us on guard. It is very unlikely of a tuberculous patch clearing under treatment and a fresh one appearing, while the patient is on Streptomycin all the time. All the time temperature persisted. In view of the transient shadows appearing and gradually fading, the case was diagnosed as one of pulmonary thrombosis. This case shows the need for looking for complications when temperature persists in a case diagnosed as coronary thrombosis. In cases of angina also sudden death might occur

in one of anginal attacks. The other common condition is senile aortic stenosis. Sudden death usually does not occur in hypertensive individuals and rheumatic carditis, as a rule.

Then, while assessing the prognosis, the general health of the patient should be considered. The prognosis is better when the general condition is good and free from complications of the other systems as well. Even if complications are present, if it is a complication that can be controlled or conquered without seriously interfering with the cardiac condition, it carries a better prognosis. Say, if a heart patient suffers from ankylostome anaemia as well, then we are dealing with a condition that can easily be controlled. On the other hand, if the patient is suffering from tuberculosis of the lungs, then we are dealing with a complication that can be terminated at our will, but all the same it carries with it some risk as the bleeding may be profuse or uterine inertia with shock might occur. Thus the existence of complications add to the risk already present and their nature should be analysed. Presence of diabetes mellitus definitely worsens the prognosis in coronary thrombosis. The mental outlook of the patient is also to be considered. Generally the placid individual who does not have an anxious and worrying temperament and does not court trouble half-way does well. Of course, a large share of the patient's attitude depends on his attending physician. While it is necessary to be guarded in giving the prognosis, still the physician, in order to safeguard himself, should not alarm the patient or his relations. Even, if at any time, the physician feels the patient's condition is taking a serious turn, he should inform one of the trustworthy near relations and continue to have an encouraging word for the patient and cheer him up. Several times I have seen patients unduly depressed because of the grave prognosis pronounced by some consultant and their having survived for a number of years subsequently with encouraging words.

Finally the most important thing to consider is the response to treatment. If two identical cases are taken, one case responds very well and the other does not. Then there are patients with failure who cannot stand Digitalis in any form and in any dose. These cases are definitely worse off since they cannot have the benefit of a valuable cardiac drug. It is the experience of everyone of us to come across patients who cannot stand mercurial diuretics. Sudden death occurs in some patients with the administration of mercurials, particularly when given intravenously. It is very difficult to say as to who is likely to manifest this idiosyncrasy, and when a cardiac patient gets a mercurial diuretic for the first time it is better to give a small dose intramuscularly and if no reaction is noted, then only to give the same intravenously. Then there are a few cardiac conditions which are amenable to treatment completely—the so-called reversible cardiac conditions. Heart failure occurring as a result

of thyrotoxicosis or in severe anæmias are easily treated unless the patient is in extremis. So the prognosis is greatly altered on the response to treatment and also on how long the progress attained is maintained. I have seen cases of congestive failure secondary to mitral stenosis respond very well on the first occasion; not so well on the subsequent occasions, and, when the patient had three or four serious failures, the response is very poor. This is particularly noticeable with the mercurials. When first given, the diuresis is very good and gives remarkable relief to the patient. But as time goes on, the relief is not quite so good. A time may come when a patient who used to pass 100 to 120 ozs. of urine with one injection of a mercurial diuretic passes only 20 or 30 ozs. of urine or even less and the relief obtained is negligible. The poor response to mercurials indicates a grave prognosis. In the case of aortic stenosis and regurgitation, failure is a late event and its occurrence is a bad sign. It is remarkable how many useful years these patients can live; but once the failure sets in, the end is not far off. Failure after an attack of coronary thrombosis is intractable and the response to treatment is extraordinarily poor. I had a patient who, in spite of digitalis, diuretics and bed rest, developed bilateral pleural effusion. The pleural effusion was aspirated mechanically several times and the failure was got over only at the end of three months, whereas failure with a valvular lesion clears in less than ten days with bed rest, digitalis and diuretics.

Finally, as I said at the beginning, prognosis in any serious disease is risky and in heart cases it is more so. It has been the experience of every practising physician to have talked to a patient and cheered him up and before he has even left the ward to hear that the patient has fallen back dead. Sudden deaths, though rare, are dramatic and stick to the mind. They are not common but far more common in heart disease than in all other diseases put together, and if there is any disease in which death occurs abruptly, it is in heart disease.

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Inoculability of Leprosy

Porrit and Olsen report two cases (from the U.S.A. Marine Corps) who were both tattooed on the same day by the same tattooer, in June 1943, when both the soldiers and the tattooer were drunk. In 1946 (2½ years later) both the soldiers developed discoloured anaesthetic patches round the new tattoo marks (an earlier tattoo mark in one of them was not involved). Portions of the lesions were removed and inoculated into guinea-pigs with negative results for tuberculosis. Sections of the tissues from both men showed typical tuberculoid changes. Scattered acid fast bacilli were found in both. The second patient developed similar patches on his other arm subsequently to those at the site of the tattoo mark. Sections of both were sent to the Garville Leprosarium, the authorities of which came to an unequivocal diagnosis of "typical tuberculoid leprosy." (This is a very important contribution to the disputed question as to the inoculability of leprosy).—(*Amer. Jour. Path.*, 23 : 5, 805-17). T.N.S.R.

Chloromycetin in Typhoid Fever*

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THOUGH a preventable disease, typhoid fever is still prevalent rather widely in Madras, at all times of the year. In the past, some attempts have been made to discover an effective therapeutic agent to produce a specific bactericidal action on the causative organism of enteric group of fevers, but, till now, these attempts have been futile. Now Chloromycetin has come to us, as a ray of light, illuminating the path of cure for typhoid fever.

History.—The discovery of Chloromycetin was due to laborious investigations by several teams of scientists. Several thousands of soil cultures were prepared and studied by Burkholder of Yale University in 1946. From a large number of such cultures exhibiting antibiotic properties, Ehrlich and his associates, working in P. D. Research Laboratory, in 1947, were able to isolate a special variety of *Streptomyces* named "*Streptomyces Venezuela*" and his associates working at the University of Illinois, isolated the same antibiotic substance from a different soil sample. This is what is now called Chloromycetin (Chloramphenicol, P. D. & Co.) and is a pure crystalline antibiotic, having specific therapeutic activities and can also be prepared synthetically.

Antibiotic activity.—Chloromycetin has been found to be effective against an impressive range of micro-organisms both gram positive and gram negative. The crystalline form inhibits bacteria when tested in dilutions as high as 0.35 microgram per c.c., a higher effective dilution than that of the earlier antibiotics. Chloromycetin shows considerably more activity in broth cultures against several gram negative organisms, than against gram positive and acid fast species. It is 7 to 36 times as active as Penicillin, against *aureus*. Chloromycetin shows remarkable activity against *staphylococcus* *Borralia recurrentis*. It has negligible effect against protozoa.

Chemotherapy.—Enteric infections notably typhoid fever, and other *Salmonella* infections, and diseases in which the rickettsias are etiological factors are among the conditions which yield to Chloromycetin therapy. *B. Typhosus* is inhibited by Chloromycetin in a blood concentration of 1 gamma per c.c. Experimental studies among other bacterial, rickettsial, and viral infections including atypical pneumonia, psittacosis and lymphogranuloma venereum, indicate the possibility of adapting these findings to clinical treatment.

Tolerance studies in man.—Smadel and his associates suggested first, that Chloromycetin might be of value in human medicine on account of its apparently low toxicity, ready absorption from the alimentary tract and its beneficial effects, even when given relatively late in experimental disease. As a preliminary to a proposed study of its therapeutic value, Smadel and his associates administered, single or repeated oral doses to a group of physician volunteers. After a single dose of 2 gms., appreciable amounts of the drug were demonstrable within 30 minutes in both blood and urine. The titers decreased to zero by the end of 8 hours.

After this preliminary work on normal volunteers, a group of five patients with Typhus fever were treated by Smadel *et al*, with remarkable and complete recovery.

Typhoid fever.—During the investigation in Kuala Lumpur, Malaya, the opportunity was present to test the effectiveness of Chloromycetin by Woodward *et al*, in the severe type of typhoid fever frequently encountered in that area. Ten cases were selected for treatment, with eight cases serving as non-treated controls. Diagnosis was confirmed by cultures found positive for *B. Typhosus*, before instituting Chloromycetin therapy. Patients were given 50 mgm. per kilo of body weight initially. Then 0.25 gms. was given every two hours until temperature returned to normal, and the same dose then repeated at 3 to 4 hours intervals during the following five days. In typhoid fever, response to Chloromycetin is not so abrupt as in typhus fever and treatment should be continued for at least 8 days; 3 to 6 gms. should be given on the first day and a total of 18 gms., or more during the period of therapy. The present dosage regimens are based on experience gained thus far in the process of clinical investigation and further evaluation will serve more fully to delineate this phase. Possibility of relapse is increased by the use of smaller dosage. Optimal dosage given at intervals around the clock is necessary to get the full therapeutic effect. In typhoid fever the evaluation of therapeutic benefit, or cure, is all the more difficult because its clinical course, duration and complications are so variable and unpredictable.

Earlier contributions about Chloromycetin therapy by Indian workers.—R. J. Vakil of Bombay, tried Chloromycetin in eleven cases of typhoid fever and two cases of para-typhoid A. fever and one of para-typhoid B. fever, all bacteriologically confirmed for diagnosis. There were 11 males and 3 females in his series, ages ranging between 13 and 47 years. In 2 cases, the treatment was commenced within the first week, in 6, on the second week and in 6, on the third week or later on. Treatment was oral and was carried out on the lines recommended by Woodward *et al*, i.e., an initial dose of 50 mgm. per kilo of body weight followed by a dose of 0.25 gms. every 2 hours, during the period of pyrexia, and a

* Specially contributed to THE ANTISEPTIC

subsequent dose of 0.25 gm. every 3 or 4 hours, for five more days. Due to limited stocks and the prohibitive cost he modified the dose. In 6 cases the temperature touched normal in 48 hours, in 9 cases within 72 hours, and in 11 cases within 96 hours. In 3 cases, persistent normal temperature showing the end of pyrexial period was recorded in 72 hours, or 3 days. In 10 cases, by the 6th day, the temperature was normal. Vakil stated that Chloromycetin had a definite temperature lowering action and he noticed that the drop of temperature to normal level appeared to occur, in step ladder fashion in two stages. There was a tendency for the characteristic sinus bradycardia of typhoid to lessen, or disappear during therapy, so much so, the pulse rate never altered with the fall of temperature, i.e., it remained constant. Mild leucopenia occurred in his cases after the administration of Chloromycetin. Toxic reactions were few. Tendency to excessive sweating, or perspiration occurred in 14 of his cases. Vakil noted the following side effects: loss of appetite (4 cases), diarrhoea (3 cases), oliguria (4 cases), abdominal discomfort (2 cases), nausea (1 case), distension of abdomen (1 case), extra systole (1 case), albuminuria (1 case), giddiness (1 case) and marked leucopenia (1 case). Classical complications of typhoid were few. Only two cases showed a relapse. Deaths were none. He found that the administration of Chloromycetin was a fairly safe measure and encouraged a fuller and extended trial.

M. J. Shah tried the action of Chloromycetin in 26 cases, at Sir Harkisondas Hospital, Bombay, and also in 7 cases in his private practice. In 12 cases, the fever was of 10 days' or lesser duration, in 18 cases it was just over 10 days' duration and in 3 cases, complications were present. All the cases were clinically diagnosed and confirmed in the majority, by widal and culture. The usual procedure of treatment recommended by Woodward *et al.* was carried out. In two cases, it was modified by the omission of the initial large dose. All the cases swallowed the capsules, except two cases in comatose state, in whom the contents of the capsules were dissolved in the nasal feeds. Average total dosage of Chloromycetin was 21 gms., while the average as recommended in routine cases and followed, was 18 gms. 21 cases recovered, 4 relapsed and 4 died. In 17, of the 25 recovered cases, the temperature came down to normal and remained so by lysis, in 4 cases, it came down by crisis and in one by pseudocrisis. Sudden collapse was noted in 3 cases. In the majority of cases, the temperature came down to normal in 72 to 96 hours. In 3 of the relapses Chloromycetin was administered again in the suggested standard dosage. Shah stated that Chloromycetin was the most effective known agent for therapeutic use in typhoid fever today, as 25 out of 33 cases in his series completely recovered. Besides controlling the temperature, by lysis in the majority of cases, the drug also controlled the toxic state, but the effect on the toxicity seemed to

be slower than that on the fever. Results were very encouraging, when the drug was used early in the disease, i.e., before the 10th day. Shah gave a nice explanation why the drug should be administered fairly early in the disease. In typhoid fever, during the first 9 to 10 days, there was only swelling of the lymph follicles and Peyer's patches in the small intestine and the toxic effects on various viscera were usually not marked. If the typhoid bacilli were successfully attacked by Chloromycetin during that period, the swollen lymphoid tissue would undergo resolution. There would be no slough formation, no ulcer formation due to the separation of sloughs, and hence no risks of complications like hæmorrhage or perforation. Administration of Chloromycetin after the 10th day, would control the fever but risks of complications remained, because of the sloughing and ulceration. Chloromycetin after the onset of complications cannot help local lesions, but might prove of indirect value, by controlling the toxæmia and fever. Shah also noted certain toxic effects like vomiting, diarrhoea, retention of urine, sudden circulatory failure, cerebral syncope after the administration of Chloromycetin.

Our Case Report.—R. Narayanaswami, aged 25, was admitted into our wards on 29th July 1949 in a moribund state of continued fever of 25 days' duration. His previous history revealed that he had typhoid fever when he was ten years old.

Starting insidiously with malaise on the 2nd July, headache on the 3rd and temperature on the 5th, the fever kept up steadily between 100.5°F and 102°F till the 19th July. On the 20th July, i.e., 15 days after the onset of illness, the fever started coming down to 100°F. Widal test was returned as positive for B. Para-typhosus 1 in 80 and negative for B. typhosus. On the 21st July his temperature reached a minimum of 99.6°F, but on the next day it shot up to 104°F in the afternoon. Urine examination that day revealed massive albuminuria, with the presence of red cells, pus cells and granular casts in the deposit. He had no œdema whatsoever. From the 25th onwards, the patient had about eight diarrhoeic motions daily and the fever ranged between 103°F and 105°F. On the 28th he became very toxic and delirious, being not at all conscious of his surroundings.

His condition deteriorated still further on the 29th. He was unconscious and had low muttering delirium. His tongue was dry and brown. Dullness with diminished breath sounds over the right base, with diffuse fine rhonchi all over the lung fields, were noted. His heart sounds were feeble and pulse was rapid and low tensioned. Urine was dark and scanty. Being in a moribund state, he was admitted into the General Hospital Wards on the evening of 29th July.

On admission, the patient was found to be completely unconscious, but with a slow muttering delirium. He had no diarrhoea.

His jaws were clenched and convulsive twitchings of his limbs were present. He had bilateral exophthalmos, and had marked respiratory distress but was not cyanosed. His temperature was 103° F. No oedema was present. In fact, he was greatly emaciated and anæmic. His tongue was brown and markedly dry. He had a rapid pulse with poor volume and tension. Heart boundaries were normal but the sounds were feeble. Examination of his lungs revealed a markedly poor air entry on the right base, with a correspondingly diminished vocal fremitus and resonance. The rest of the lung fields exhibited plenty of fine cooing rhonchi. Spleen was just palpable and soft. Liver could not be felt. Pupils were dilated and sluggish in reaction.

After admission, 1,00,000 units of Penicillin was administered parenterally 4th hourly, while 50 c.c. of 25% glucose, 100 mgm. Vitamin C, and 100 mgm. of Nicotinamide, were given by veinoclysis. Fast nasal oxygen was instituted. Continuous rectal saline, two pints at a time, was started. As he was unconscious he was nourished on fluids by nasal drip. Examination of his urine showed slight albumen but no red cells or casts in the deposit. Total white cells were 6,200 per cmm., with relative polymorpho-nuclear increase. His blood pressure was 110 mm. 86 mm.

On the 30th of July, the temperature rose up to 105° F. His general condition was the same as before, but his abdomen was slightly distended. A daily total dosage of 5,00,000 units of Penicillin was maintained. Intravenous 50 c.c. glucose with 100 mgm. Vitamin C and 100 mgm. Nicotinamide was given twice daily. Oxygen, rectal saline, nasal feedings, supplemented with Vitamin C and Nicotinic Acid orally were continued. Bed X-ray of the chest, revealed nothing abnormal in the lungs, or the heart.

Though the patient's condition was the same on the 31st, he became worse on the 1st of August. His widal was returned as B. typhosus was grown in blood and urine cultures. The previous day's treatment was continued.

On August 2nd the patient was still toxic and stuporous. Marked lung signs were present on the left side, though the impairment of resonance with diminished air entry, on the right base, was comparatively less that day. His temperature remained steady at 103° F. His heart sounds were still feeble and his pressure leaned towards hypotension 106 mm/84 mm. Hence in addition to the routine treatment, Percorten 5 mgm. and 1/120th gr. Strychnine Hydrochloride were ordered to be given, each, on alternate day. Liquid Coramine 10 drops in glucose water was frequently administered as a stimulant.

On the 3rd August the patient being in the same toxic state as before, plenty of fluid intake, about 7 pints orally by nasal feeds,

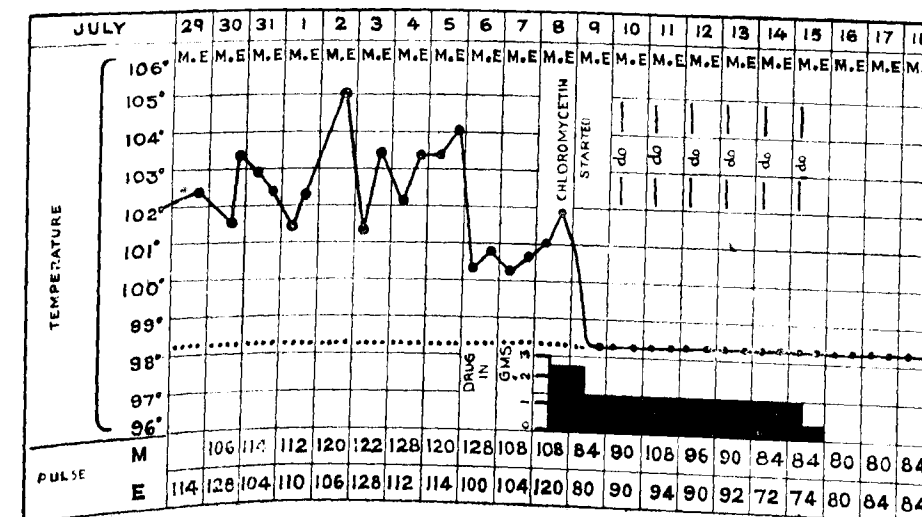
and 2 pints rectally, was maintained. Thiamine Hydrochloride 100 mgm. was given parenterally as a cardiac tonic. On the 4th, the heart sounds were better heard. B.P. was 104 mm/82 mm. and the urine was clear. The temperature ranged between 102° F. and 104° F. on the 5th August, and the patient's condition apparently looked better with improved tone of his heart sounds, but he was so delirious and boisterous at night, that he had to be quietened with Dial injection. Plenty of fluids were still given.

On the 6th and 7th August, the temperature showed a tendency to decline slightly and his heart sounds were better heard and his mental state seemed to improve. But his toxic state still persisted and he had diarrhoea in addition.

On the 8th, though the temperature was at 102° F, he was still toxic and delirious and had severe diarrhoea. We decided to try the new remedy *Chloromycetin* on him. With great difficulty, four bottles, each containing 12 capsules of P. D. & Co.'s Chloromycetin (0.25 gm.) were procured and at 8 p.m. the contents of two capsules i.e., 0.5 gm. total, was administered through the nasal feeding and one capsule of 0.25 gm. was given every two hours from 10 p.m. onwards. All the general, supportive and stimulant treatment was continued. The temperature which was 102° F at the time of start of the Chloromycetin treatment fell to normal during the night within 12 hours, i.e., by the time 2 grms. were over the temperature had touched normal—(see Chart.)

CLINICAL CHART OF TEMPERATURE, Etc.

Name: R. Narayanaswamy. Age: 25 years. Disease Result



On the 9th, though the temperature was normal he was still toxic and was in a dazed state. From 8 a.m. onwards one capsule of 0.25 gm. Chloromycetin was given regularly every 4 hours, till the total of 12 grams was reached. During the treatment with this drug, careful watch was kept over the patient's cardio-vascular system, especially for peripheral failure and collapse. His blood pressure was 105 mm/80 mm. All the supportive treatment was continued.

On the 12th August, though the patient was in a dazed state he obeyed simple requests and answered direct questions. Nasal feeding and rectal saline were stopped. He had no fever afterwards. His exophthalmos disappeared and from the 14th August onwards he made an uneventful recovery, went through the regular typhoid convalescence and was discharged eventually on the 24th August 1949.

Conclusions and summary.—1. A review of the history, chemotherapy, antibiotic activity and action of Chloromycetin in typhoid fever, is presented.

2. A short account is given about the work done on Chloromycetin therapy in India by Vakil and Shah.

3. A report of our case is appended. In our case, Chloromycetin was tried in the end as a desperate measure and the temperature came down by crisis. Our initial dose was small and the treatment extended nearly a week. No collapse or failure was noted. We agree with Shah in that the toxic symptoms continued even after the fall of temperature. Sinus bradycardia was still present with the fall of temperature in our case.

Acknowledgment.—I am much indebted to my Chief, Lt. Col. C. K. Prasada Rao, M.D., Physician, Government General Hospital, Madras, for his valuable help in the preparation of this paper. My grateful thanks are also due to the Superintendent, Government General Hospital, and the Surgeon-General with the Government of Madras, for their kind permission to publish this case. I would be failing in my duty if I do not thank my housemen Drs. Chandrasekhar (Appu), Narayanan and Ranjan David who, in their enthusiasm and zeal in the ward work, gave me all co-operation.

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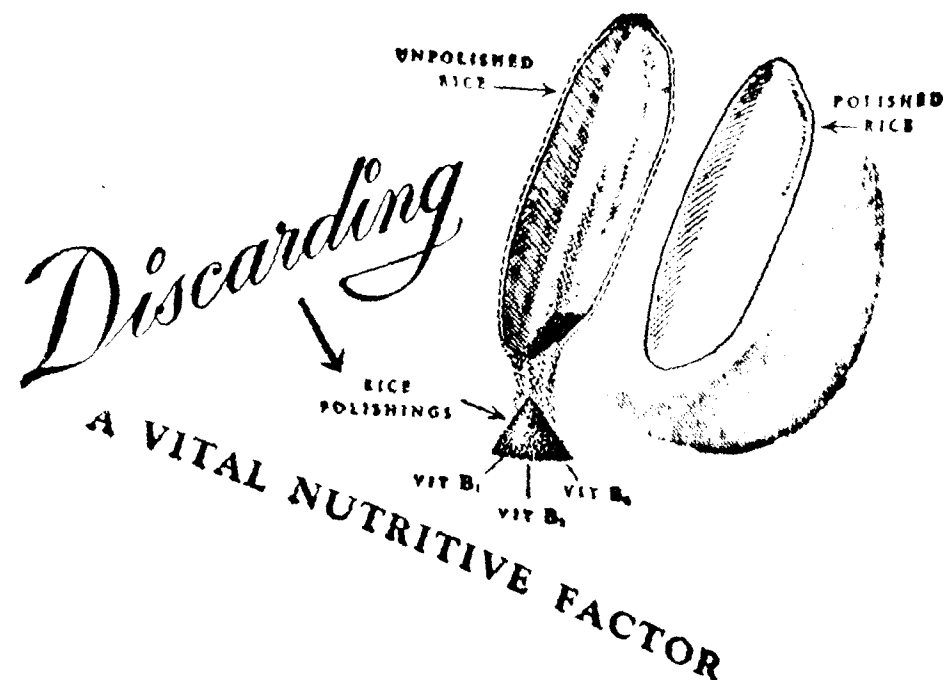
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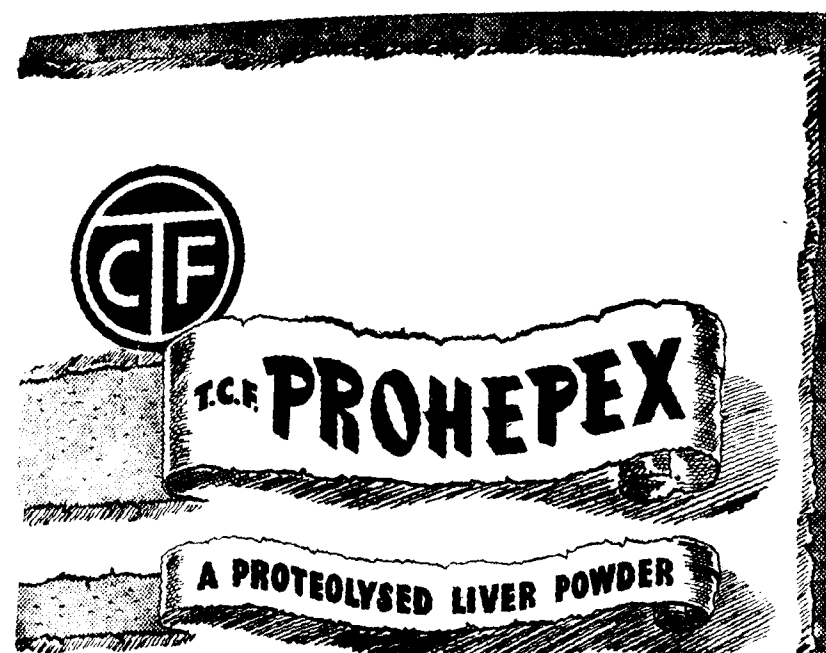
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Allergic Disorders*

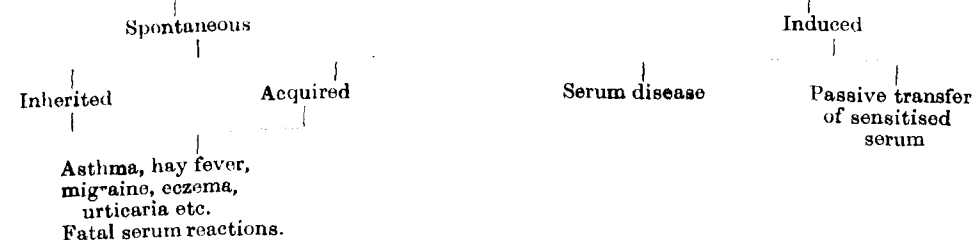
A. V. S. SARMA, M.B., B.S., D.C.M. (Eng.), F.D.S. (Lond.),
Hony. Physician, Government Royapettah Hospital, Madras.

General considerations.—G. W. Bray has given a classification of the allergic disorders which the writer chooses to follow.

HYPERSENSITIVENESS

In animals: Anaphylaxis
(Antigen-antibody reaction)

In man: Allergy
(Allergen-allergen reaction)
Immunological reaction



In France ANAPHYLAXIS is still the term used to designate clinical allergy, although ALLERGIE also appears in the French literature. Elsewhere allergy has in great measure replaced it. Von Pirquet introduced the word allergy ("altered reactivity") and also misused the same, but "a word once given to the world and accepted by it ceases to be the property of the donor." The immunological role of allergy is much debated and questioned in tuberculosis, particularly by Rich and his followers.

Allergy denotes peculiar reactions. One to ten per cent of human beings are allergic. Allergy is a clinical concept as opposed to anaphylaxis which is concerned in the laboratory; is inherited from parents; and is against various substances (bacterial and non-bacterial). Its duration in the young is long. The allergic reaction (first or repeated) is due to allergins of unknown immunological nature provoked by allergens (protein or non-protein). A sensitising dose and time interval are not always essential. The sensitivity is highly developed in some tissues and entirely absent in others. The reactions are mainly in the skin, respiratory and gastro-intestinal tissues. Non-bacterial allergy manifests as vagus syndrome and bacterial allergy manifests in the sympathetic systems associated with destruction and new formation of tissues.

"Allergy is constitutional or systemic phenomenon, predominantly functional, only secondarily organic which may occur in any individual suffering from any other specific malady and which not infrequently colors the picture of the disease."

—Vaughan and Black.

* Specially contributed to THE ANTISEPTIC.

[737]

The Allergic Diseases

The features are :—(1) Heredity is marked in over fifty per cent of cases. (Note twins with asthma; photographs of the twins *Fig. 1*).

(2) Characteristics: Onset may be congenital or acquired. Duration generally lasting. Cause may be antigenic, non-antigenic substance, protein, non-protein or even physical. Degree of sensitisation varies. Specificity is present. Multiplicity of sensitivity is the rule. Passive transfer of sensitivity is possible.

(3) Manifestations are characteristic.

(4) Other allergic manifestations are present.

(5) Positive skin tests.

(6) Pathology: The symptoms of allergy can be explained by smooth muscle spasm and increased capillary permeability. The allergic inflammation is exemplified in respiratory tissue.

(7) Eosinophilia in blood and tissues present.

(8) Positive relief to Adrenalin, Ephedrine, and Aminophylline; and Anti-Histamine drugs.

(9) Portals of sensitisation: active or passive.

Active :—Heredity and tissue trauma present. (1) Placental; (2) Ingestion (Foods and drugs-phenyl group); (3) Inhalation; (4) Infection (bacteria and parasites); and (5) Contact.

Passive :—(1) Placental; (2) Passive transfer.

The psychological factor in allergy is one of the non-specific catalysing factors. It is important as can be seen from the following.

An elderly woman suffered from mucus colitis (asthma of colon) till his son got married as per her wish. Another elderly woman got rid of the disease only when no more sympathy was possible in the family, as after her husband's death. An asthmatic never had spasms while in the hospital; but when he joined his office had relapses very frequently. Similarly another asthmatic got into spasms whenever there was a domestic quarrel between his wife and his mother. A young woman developed status asthmaticus on the eve of joining her new and second husband; and the apparent conflict is obvious.

The case sheet of an allergic patient is a speciality and includes special attention to heredity, influence of external factors, foods, previous illness (measles, bronchopneumonias, other allergic conditions), radiology of bone ends, chest and sinuses, gastric conditions and skin reactions, and a thorough blood examination (Wassermann reaction and Eosinophilia).

The chief characteristics of the allergic disorders are : (1) Periodicity; (2) positive personal or family history; (3) specific reactions to skin and mucous membrane reactions; (4) eosinophilia; (5) hypochlorhydria, (6) response to Adrenalin, Ephedrine or Aminophylline or the recent Anti-Histamine preparations like Anthisan (M & B) or Antistine (Ciba).

Regarding prolonged observations of allergic diseases under his in-patient care during the last 27 months, the writer could collect the following data from the Government Royapettah Hospital, Madras. The out-patient department is very rich in these allergic conditions, but only severe cases are admitted and therefore the list is by no means representative of the actual incidence, but only a record of the experiences of a worker in a responsible institution.

Status asthmaticus 4. Bronchial Asthma 36. Allergic Rhinitis 1. Food Allergy 5. Eosinophilic Lung 7. Drug Allergy 1. Allergic reaction after scorpion sting 1. Urticaria 1. (Cases of urticaria are too numerous for in-patient care; the one admitted was for associated abdominal colic).

Individual Manifestations of Allergy

Asthma.—Attacks of paroxysmal dyspnoea, principally due to spasm of bronchial muscles and oversecretion of mucus.

The age incidence, sex incidence, hereditary predisposition and an expressly assigned cause of onset (pertussis, measles, pneumonia, influenza, severe bronchitis) are all well-known.

The types of asthmatic attack in childhood differ from those in adult life.

Types of asthma in childhood:—Acute bronchitic type in infancy—resembles a mild pneumonia. Fever, sneezing, dyspnoea, cough, and cyanosis may all be present. Expiration is prolonged with sonorous and sibilant rales. Instances are not wanting in daily practice to exemplify this. In breast-fed babies spasms occur because the mother has taken fish, prawns, tomatoes, onion curry, mangoes, anacardium fruit, jack fruit, oranges, and fried curries.

The hereditary type:—The child might have had eczema earlier. Typical spasm and heredity are present. A child came up to the children's clinic, Government Rayapettah Hospital, with severe infantile eczema. Age 1 year. Male. Father (asthmatic) and mother are first cousins. Eczema cleared on treatment but in 6 months time the little baby came up to the clinic with typical asthmatic spasms.

Eczema-asthma-prurigo syndrome:—This is a variant of the former. Predominant in males. There is a family of a father with psoriasis and 2 sons (psoriasis), and 2 daughters (asthmatic). There

is persistent skin rashes in flexures in one daughter who has regular spasms of asthma. The writer has observed children having eczema, later developing cirrhosis of liver, and when cirrhosis is arrested, developing asthma. Allergic history is positive. (Fig. 2.)

The lung damage type: Bronchitis with superposed spasm present and appearing in older children, may be recurrent bronchitis. Many children convalescing from pertussis, measles, and bronchopneumonia, develop persistent bronchitis and never seem to get rid of the same. Iodides and Belladonna do well.

Coryzal asthma (True seasonal asthma): Is due to pollens, odours and emanations.

Types of asthma in adults: (1) Cases with intervals between spasms; and (2) cases with no freedom from spasms. Pulmonary inflammation may be present or absent.

Allergic catarrh is characterised by positive history, no free interval and relief from anti-allergic drugs, while infectious catarrhs are sharp and clear off generally.

SYMPTOMS:—Prodromata are coryza and oppressive feeling in chest or digestive upsets.

Obstructive and paroxysmal dyspnoea, pulmonary distension, cough with or without expectoration, and sometimes cyanosis and pulsus paradoxus.

Allergic bronchitis may dominate as a result of bacterial allergy and vaccine therapy is indicated then. Morning coryza, sneezing, or cough may also be allergic.

The time of onset, effect of locality, influence of season, inter-paroxysmal intervals (sometimes regular) are all characteristic caprices of asthma which may in some patients be associated with skin conditions, sick headaches and enuresis in children.

The allergic individual is generally mentally above normal and may later in life develop a defection. Constitution may be obese or thin. Peculiar or characteristic chest deformities are noticed in the asthmatics. (Figs. 3 and 3-a) Kyphosis and square shoulders are seen.

PATHOLOGY:—Sudden death might occur in an asthmatic spasm (vagal inhibition, sudden failure of myocardium, or asphyxia due to mechanical obstruction of the bronchi by viscid secretions).

An attack of asthma is explained as due to (1) Spasm of bronchial muscles; (2) oedema of lining membrane of walls; and (3) obstruction of bronchial lumen by exudate.

Thickening and hyalinisation of basement membrane of bronchi, hypertrophy of the bronchial musculature, eosinophilia of the tissues, sacculatation of the epithelial layer of the bronchi, bronchial

Allergic Disorders

L. V. S. Sanyal



FIG. 1.
Asthmatic child.

(Vide page 748)



FIG. 2.
Asthmatic, after recovering from catarrhic liver. (Note marking of enlarged liver).
(Vide page 749)



FIG. 3.
Chronic Asthmatics.

(Vide page 749)



FIG. 3-a.



FIG. 6.
Urticaria (patient sensitive to fish)
(Vide page 745)

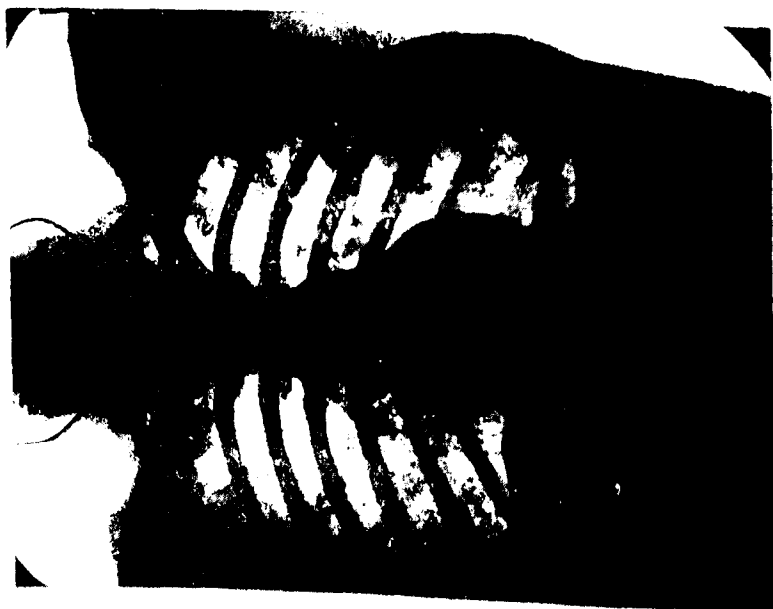


Fig. 3. Hyperinflation of the lungs in asthma. The lungs are hyperinflated and the diaphragm is flattened.



Fig. 4. Hyperinflation of the lungs in asthma. The lungs are hyperinflated and the diaphragm is flattened.

stenosis, hypertrophy and metaplasia of the epithelium form the local pathology. Emphysema is marked in old asthmatics.

Asthmatic sputum is thick and viscid in the absence of secondary infection with which it becomes thin and muco-purulent. Charcot-Leyden crystals, Laennec's perles, Curschmann's spirals are known to be present. The bacteriology of sputum reveals generally streptococci, *m. catarrhalis* pneumococci and staphylococci. Stools tend to acid side.

X-RAY CHEST:—Generally marked hilar shadows and peribronchial fibrosis are seen and emphysematous change in elderly patients. (*X-ray picture: Fig. 4*).

Complications in asthma are allergic (urticaria, eczema, hay fever, migraine) and non-allergic (over-distension of the lungs, chronic bronchitis, atelectasis, subcutaneous emphysema, lowering of vital capacity). Thoracic deformities are kyphosis of the upper spine, increase in the A-P diameter of the chest, pigeon-breast, and elevation of the clavicles.

Cardiovascular changes are rather few and very late results. Right-sided heart failure is the rule in emphysematous cases.

DIAGNOSIS.—Diagnosis during an attack is easy. The posture of the patient, hyper-resonant chest, expiratory dyspnoea with wheezing, sibilant and sonorous rhonchi, and later secretory changes confirm the diagnosis. In the free interval diagnosis is difficult. The study is for the general allergic state.

Differential diagnosis lies in separating asthma from laryngeal, tracheal and bronchial, and pulmonary conditions; cardiovascular conditions like failure of heart, coronary sclerosis, paroxysmal auricular flutter or fibrillation; renal causes; and nervous manifestations like functional air hunger, hysteric polynœa, respiratory syndrome following encephalitis. A blood and sputum eosinophilia is pathognomonic of bronchial asthma, pulmonary distomiasis, hydatid disease, and the new tropical eosinophilia excepted.

PROGNOSIS.—Longevity is good. The writer does not agree with Bray who says that asthmatics are free from peptic ulcers, diabetes, and in children acute rheumatic fever. Male children might clear at puberty. Status asthmaticus occurs in older age. Menopause benefits asthma in some women. Early treatment especially in allergies is beneficial.

“The shorter the duration of the disease and the length of the attacks the better the prognosis. The greater the frequency and less the complete recovery between attacks the worse the prognosis. The finding of a definite exciting cause either by protein skin tests or elimination diets improves the prognosis.”

—Bray.

Increasing complications and lung damage are adverse factors. Unsuccessful surgery and poor therapeutic response are obviously unhelpful.

TREATMENT:—Treatment of an attack: Adrenalin 1 to 5 minims of 1 in 1000 solution subcutaneously early enough in an attack is very effective, but not in established cases. Adrenalin 1% aqueous spray or oral Ephedrine Hydrochloride gr. $\frac{3}{4}$ or 1 c.c. s.c.i. of Evat. mine are also advocated. Inhalation of Amyl Nitrite (5 m) or hot water bottle to feet or hot coffee with Liq. Ext. Grindelia m. 20 every 20 minutes for 3 doses are also used for relief. 250 c.c. of 20% Dextrose with gr. $\frac{1}{4}$ Aminophylline I.V. is excellent; later Aminophylline gr. $1\frac{1}{2}$ to 3 orally t.d.s. is maintained. Stramonium cigarettes or use of Morphine or Cocaine are deprecated. Bronchitic element is benefited by Penicillin inhalation. For status asthmaticus 2 minims Adrenalin every 5 minutes for $\frac{1}{2}$ hr. is advocated but has not often succeeded. Adrenalin in oil is said to be better. 90% oxygen or a mixture of 20% oxygen and 80% Helium given for 45 min. by a B.L.B. mask at the rate of 6 to 9 litres a minute is good in severe cases and relieves respiratory effort considerably. An alternative is intravenous Aminophylline. For sleep Phenobarbitonum gr. $\frac{1}{4}$ to 1 or particularly Belladonna at bed time. Lumbar puncture is dramatically useful in a few cases.

An enema or purgative and institution of light liquid diet are necessary. A mixture containing Iodides and Stramonium is useful after the acute phase.

The writer depends upon intravenous Aminophylline with Dextrose solution for status asthmaticus. Anti-Histamine drugs did not impress the writer except in a few patients with dietetic excitants (fish, prawns, fried puddings). Treatment between attacks lies in the following:—

- (1) Avoidance of removal of the specific cause.
- (2) Desensitisation—(a) specific; (b) non-specific.
- (3) Avoidance or removal of contributory factors: (a) General hygiene; (b) diet; (c) reflex irritation, focal sepsis; (d) endocrine system (Thyroid given if B.M.R. is low); and (e) psychopathic states.
- (4) Symptomatic—Drugs and surgery.
- (5) Physical therapy.
- (6) Remedial exercises.

If the F.T.M. reveals low acidity, dilute Hydrochloric Acid and Pepsin or Caripeptic liquid (Upjohn) are advised. A course of Iodides and Belladonna as also Vaccine therapy is instituted. The writer has used recently Nicotinic Acid and Ephedrine together with very gratifying results. Nicotinic acid is anti-allergic and vasodilator, and also stimulates gastric secretion, thus counteracting the

evil of Ephedrine. Further the mental symptoms of anxiety, depression, irritability and loss of memory encountered in asthmatics are also relieved, probably by improving the oxidative catalysis of brain cells.

DIET:—Avoidance of exciting foods; and in general spiced foods, fried preparations, excess of fat, citrus fruits, milk or butter-milk at bed time. Early and light meal at night and avoidance of sleep by day are good dicta.

Eosinophilic lung.—This condition is characterised by vague or marked symptoms resembling pulmonary tuberculosis. X-rays of chest reveal diffuse mottling of lung fields, and blood films show marked eosinophilia. What is behind the disease we do not know. Is it mites? Allergy? Response to arsenic is excellent. The author has used daily intramuscular injections of Emetine Hydrochloride, as in amoebiasis, in eosinophilic lung with equal success. The idea is from a worker in America. (*X-ray picture: Fig. 5*).

Hay or Pollen allergy is due to hypersensitiveness to the pollen of Timothy and other grasses and amenable to desensitisation.

Nasal allergy.—The condition, more common in females, is characterised by paroxysmal attacks of sneezing and running of water from the nose with partial or complete stoppage and loss of sense of smell, but it tends to recur all round the year. This is related to asthma.

ETIOLOGY.—Inhalant factors (dusts). Occupational. Physical (climatic). Foods (egg, milk, ice creams, chocolate, potatoes, beans, onions, tomatoes, fish and beans). Bacteria. Endocrine factors (hypoparathyroidism). Drugs (Aspirin). The diagnosis is from recurrent colds, sinus infections, cerebrospinal rhinorrhea, syphilis, and intranasal abnormalities. Smear test is valuable, also intra-mucosal test. Treatment is as for allergy. Best examples are the persons with rhinorrhoea in busy market places; the cooks; and the sewer workers.

Eczema in infants and adults.—Neurodermatitis—Contact dermatitis.—Eczema and urticaria are common in patients with hay fever or asthma, and their family members (Cooke and Vander Veer). Infantile eczema is due frequently to food sensitisation.

Then the characteristics of allergic skin conditions are: (1) Itching; (2) paroxysmal nature, with typical localisations; and (3) appearance at allergic age period (first decade).

The specific sensitising substances are variable, first foods and later inhalants.

Eczema:—"Eczema has been defined (Goldsmith) as an inflammation of the skin in which the epidermis is predominantly involved, characterised in the early stages clinically by clusters of tiny vesicles and histologically by epidermal spongiosis, that is, inter- and intra-cellular oedema."—A. C. Roxburgh.

The hyperaemic stage, the exudative stage, and the desquamative stage are all known. Sensitivity resides in the epidermis. The course is acute, subacute or chronic with exacerbations and the lesions are distributed characteristically with itching, burning and tingling. Male children suffer more. Face, head, trunk and extremities are affected. Heredity is marked.

In the etiology of eczema, of teething, focal infection, endocrine disturbances or metabolic alteration, dietary factors (in children), and protein sensitisation, one or more may be operative. Diagnosis is by skin testing and elimination diet. MacLeod and Muende adduce antigen-antibody reaction in the epidermis in the eczematous reaction.

Infantile eczema has to be differentiated from impetigo contagiosa, seborrheic dermatitis, congenital syphilitic skin lesions, intertrigo, sweat or napkin rashes, or scabies (wherein allergy has a role). Sudden death has occurred in a few eczematous children. Natural cure is common by the end of the second year. Sometimes flexural eczema is persistent and asthma may supervene in many children later on. This is eczema-prurigo-asthma syndrome. Such children are liable to diarrhoea and bronchitis. The writer has seen a few of these children develop cyclic diarrhoea and hepatic cirrhosis and this aspect has been elaborated under cirrhosis in children. (Sarma, A. V. S., 1946).

Treatment is induction of restful sleep, soothing lotions and desensitisation and use of Anti-Histamine drugs (Anthisan M & B or Antistine 'Ciba') which are really useful here.

Pruritus :—This is shown to be due to hypersensitisation to foods (wheat, Bengal gram, gingelly or pea-nut oil or its hydro-genated products, fish, prawns, tomatoes, onions, watermelon, jack and mango furits).

Contact dermatitis :—External irritants (occupational) cause this.

Occupational dermatitis has for its excitants in the profession. Plant, pollen, perfume, and medicament dermatitis are common experiences.

Urticaria :—Urticaria is an oedematous condition of the skin characterised by the formation of wheals and accompanied by itching and stinging. Urticaria of adults, papular urticaria of infants and angio-neurotic oedema are known.

Normal and allergic persons are victims of urticaria. The rash commoner on the trunk than on the face affects only the superficial layers of the skin. The mucous membranes of the mouth, larynx, pharynx, and alimentary canal (gives rise to colic) are also occasionally involved. Pruritus is present, as also flatulence and constipation.

Acute and chronic cases are recognised. In the urticarial wheal is seen the triple response of Lewis.

Causes are light, heat, cold, trauma, animal factors (insects, bugs, pediculi or leeches, stings by bees etc., the hair of caterpillars, woollen clothing, animal contacts), and vegetable agents. Ingestant factors are absorbed from the gut (grapes, mangoes, jack fruit, water melon, drugs, parasites). Two asthmatics eat mangoes in summer and develop severe urticaria. A clerk anxious to evade the office to see a Test Cricket Match, developed severe urticaria to serve his purpose with medical opinion. Septic foci are causes. Injected materials (arsenic, bismuth, insulin, liver extract) are noteworthy; a patient receiving liver extract developed intense urticaria after 6th or 7th doses. It was prevented when the same extract was given mixed with Adrenalin. Endocrine dysfunction (puberty, menses, menopause) and psychological states (anxiety states, fear, anger) are guilty in some susceptible individuals. (Fig. 6).

Treatment of an acute attack is successful with Adrenalin, Ephedrine and Anti-Histamine drugs are excellent choice here. A brisk purgative or emetic and a soothing skin lotion are needed. Vitamins K and C, as also Nicotinic Acid, are very useful. Interval treatment is based on the exciting factors. Rash after serum injection can be prevented by previous or simultaneous administration of Anti-Histamine drugs. Autohæmo- or sero-therapy is also used.

Angio-neurotic oedema involves the subcutaneous tissue and is treated as above.

Papular urticaria is common in children and ceases at puberty. This is an allergic condition depending on fats, and common foods like fish, egg, potato, oats, chocolate, and has to be differentiated from varicella, scabies, Hebra's prurigo, insect bites, sweat rashes, lichen planus, papular eczema, and papular erythema multiforme. Treatment lies in adjustment of diet and use of lotio calminæ and internally calcium and vitamin C.

Allergic purpura :—This is due to changes in the blood capillaries, and is characterised by skin lesions, gastro-intestinal crises, painful and swollen joints, and hæmorrhages. Dietary agents and drugs are exciting factors. Vitamin P is specially mentioned, but C and K as also calcium are really more useful.

Erythema multiforme (allergic to diet or streptococcal infection) and erythema nodosum (streptococcal or tubercular allergy or rheumatic indication) are also conditions demanding detailed study. Dermatitis herpetiformis and acne vulgaris are sometimes allergic in nature. Psoriasis is also allergic in a few cases.

Migraine :—The allergic basis of migraine is supported by periodicity, marked heredity, and its association or alternation with the other allergic manifestations. The writer has collected

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data which lead far into various diseases. The following are noteworthy:—

1. An adult now 45 years old. Subject to migraine as a school boy. April 1934: Developed glycosuria. 5-4-1939: Blood Kahn and Wassermann: negative. May 1939: Developed hypertension (150/80). E. C. G. showed left ventricular preponderance. B. M. R. was plus 19½%. 22-9-1939: Fasting blood sugar 179 mgm. percent. 2-8-1939: X-rays skull—shows thickening of inner table. No evidence of increased intra-cranial tension. Pituitary fossa of normal dimensions. Body of sphenoid thickened. Right frontal sinus shows comparative opacity. Left, middle, and inferior turbinates show thickened and hypertrophied condition. To date, the patient is alive and a confirmed diabetic, but a subject with depression and anxiety neurosis. Three of his children developed infantile hepatic cirrhosis; two died, and the other has overcome the disease and is now a college student. (*vide* Infantile Hepatic Cirrhosis, Jan. --July, THE ANTISEPTIC, 1946).

2. An adult (36 years old now), one of nine and subject to migraine and asthma. Migraine is common to all his brothers (2) and sisters (6). He developed recently a pneumonitis (SENSITIVE TO NICOTINE?) of the left lower lobe (confirmed by X-rays) and hæmoptysis and fever and loss of weight and paroxysmal cough. Blood Wassermann and Kahn negative, but a smear showed marked eosinophilia. The patient improved on intravenous calcium and abstinence from tobacco.

A child of a brother of this patient developed hepatic cirrhosis, now cured but having asthma.

Cyclic vomiting is exchanged for migraine in later childhood; and migraine may be exchanged for epilepsy. The writer has interesting cases on these points.

The writer had for a period of five years a rich Marwari boy who is subject to epileptic seizures. He gets bouts of vomiting especially when indulging in sweets. Vomiting ceases only to exchange for Grand Mal. X-rays skull: nil particular. Blood sugar normal (done to eliminate insular hyperactivity). Blood Kahn and Wassermann negative.

In another, a female, migraine has developed later in a child who was prone to idiopathic convulsions of infancy, and now has today typical rheumatic mitral stenosis with failure.

Associated with migraine, cyclic vomiting, asthma, epilepsy, diarrhoea, and which is generally precipitated by excess of fat in the diet. This is notorious in paving the way for hepatic cirrhosis.

Allergic headaches (not migraine) are generally common as a result of food allergy. Whether epilepsy has allergic basis or not

is debated. Serous meningitis (Quinke), functional paralysis, Meniere's syndrome, disturbances of sleep, are described as having an allergic basis.

The writer offers an allergic syndrome for scrutiny and study and is characterised by a triad: HYPOTENSION, INSOMNIA, AND MORNING HEAD-HEAVINESS. The victims of this syndrome have the three symptoms, and are upset by purgatives, fatty foods, and indulgence in sex act. Examples:—(1) An adult male 41 years old. Developed asthma on two occasions with long freedom. Has the triad of trouble. Temporary response to intravenous glucose is good.

(2) An adult of 57 years. Male. Subject to asthma, arthritis, and fits of sneezing. A great intellect like No. 1. Hypotension and giddiness are constant.

(3) An adult male of 50 years. Insomnia dominates the picture. Has low B.P. and heaviness of head in the mornings. Very intelligent and active man. Personal and family allergy to foods is present.

All the three are free from syphilis, diabetes mellitus and any other severe malady that could account for the symptomatology.

Food allergy:—Reactions arising from the ingestion of wholesome and normally well tolerated foods. The "fortunate" food allergies are sensitive to fish, prawns, cucumber, water-melon, strawberries, tomato, onion, and cabbage; and the "unfortunate" major group of allergies are always in our hands for dealing. The major group generally is sensitive to common foods, wheat, milk, bean, egg, potato, mango, jack fruit, citrus fruit, gingelly oil, peanut oil. The symptoms are contact reactions (gastro-intestinal) or general reactions (nasal, neuro-psychiatric, cutaneous, and genito-urinary). Oil baths produce asthmatic spasms in some; and in some others fever and joint pains. Skin reactions, elimination diets, and leucopenic index are helpful in diagnosis. Two patients were admitted into the Royapettah Hospital with signs and symptoms suggestive of acute abdomen. Both of them were sensitive to prawns and the abdominal colic was associated with only slight tachycardia (90 per minute) and the history was very indicative. Treatment with Atropine was cure in itself. Another patient sensitive to fish and prawns was admitted in the night with dyspnoea and restlessness suggesting "coronary thrombosis." The history was clear that such foods upset him likewise always and this time markedly so. Orally Anthisan tablets, and a purgative and rest, cured the patient.

Treatment consists in avoiding exciting agents. Specific desensitisation non-specific desensitisation, and symptomatic relief with Adrenalin, Ephedrine are to be remembered. Anti-Histamine drugs are useful.

Oral and peptic ulcers claim an allergic basis. —Arthus Phenomenon.

Muco-membranous colitis is a condition characterised by the periodic expulsion of large quantities of mucus from the bowel, and is a functional disturbance. Food sensitivity and mental worry are important causes. Two cases were quoted under psychological basis.

Hepatic cirrhosis:—The writer has a strong argument to show that an allergic diathesis is present in adults and children developing cirrhosis of liver which is an ultimate picture. Arthus phenomenon helped to clarify the pathological position.

Bacterial allergy:—This is very important in adult medicine. Allergy in nephritis, rheumatic diseases, rheumatoid arthritis, scarlet fever, and tuberculosis is always a discussed topic. The writer believes that in whooping cough, definitely bacterial allergy plays a decisive part. Is this Arthus phenomenon? Shwartzman phenomenon is a hæmorrhagic necrosis at the prepared area in response to bacterial toxins, and this appears to be an important element in cases of hæmorrhagic bronchopneumonia.

Physical allergy:—Asthma, coryza, urticaria, are solely brought about by such physical agents as heat, cold, light, or mechanical irritation, which act through the skin or mucous membranes. Reaction may be contact reaction or general. Hypersensitivity to light includes urticaria solaræ, hydroa aestivale (Hydroa vacciniforme, summer prurigo).

Drug allergy:—Aspirin, Sulpha group of drugs, Quinine, Copaiba, Iodides, Turpentine, Argyrol, Mercurochrome, Arsenic etc. are known. Purpura follows the use of Sedormid or Nirvanol, Luminal or Quinine. Insulin, thyroid, pituitary, ovarian hormone, aneurin, liver extract are also offenders. Agranulocytosis has resulted from use of Pyramidon, Thiouracil, Sulpha drugs and certain others.

A patient after taking advertised tablets for purgative developed acute gastro enteritis; this was sensitivity to Phenolphthalein. The same drug produced in another susceptible person intractable dermatitis resembling psoriasis.

Serum disease, fatal reactions, and artificial sensitisation.—A second dose of serum following a first dose after 10 to 14 days interval (or a primary dose in a sensitive subject) may result in fatality. Large and frequent doses of serum may produce serum disease in non-allergics. Reactions are common only with equine serum.

Serum disease has fever, vomiting, eruption, joint pains and tenderness, œdema, lymph node enlargement and sometimes neurological complications. Treatment consists in use of Adrenalin, Ephe-drine, and Aspirin. Anti-Histamine drugs save the patients well

Fatal accidents in serum therapy are prevented by quick desensitisation or prophylactic use of Adrenalin or better still *Anti-Histamine drugs*, *Anthisan (M & B)*, or *Antistine (Ciba)*.

Allergic manifestations of systemic nature are angina pectoris. Thrombo-angiitis obliterans, periarteritis nodosa, paroxysmal tachycardia, essential hypertension, and a few obscure circulatory phenomena.

The allergic joint including intermittent hydrarthrosis (giant urticaria) are also described. Spring catarrh in the eye is allergic. Eyes are sensitive in some to wheat. Parotid swellings, dysmenorrhœa, enuresis, testicular swellings are known to be allergic.

Hypersensitiveness to insects, parasites, moulds and fungi.

Bee stings. Wasp stings. Mosquito bites. Flea bites. Bed bug bites. Sand fly bites. Insect emanations. Ascariasis. Hydatid disease. Bilharziasis. Oxyuris. Malaria: A patient with chronic malaria has asthmatic spasms corresponding to the malarial paroxysm.

Moulds, fungi and yeast can also excite reactions.

Fatal reactions (allergic) to scorpion bite were faced by the writer. Prevention is by use of anti-scorpion serum prepared by King Institute, Guindy, Madras. The victims are generally children, while adults are not exempt, and when the reaction has set in, treatment is generally of no avail. Recently the writer had an adult with the reaction and was treated with serum (though late) and as for circulatory failure, and was discharged cured.

(The writer is indebted to Dr. P. R. Kalyanasundaram, M.B., B.S., Superintendent, Government Royapettah Hospital, Madras, for all the help and encouragement received in the preparation of this communication).

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Laboratory Diagnosis in Clinical Medicine

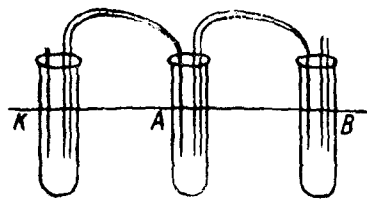
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(Continued from page 654 of Sep. '49 issue).

Blood Urea

Principle:—The urea in the blood is converted into ammonium carbonate by the action of the enzyme of soya-bean, which acts specifically on urea. The ammonia so formed is drawn off by a current of air and absorbed in a known volume of N/100 HCl. The HCl left unneutralized is titrated with N/100 NaOH. The amount of HCl neutralized by ammonia is then given by difference, from this the amount of ammonia given off, and hence the urea in the blood, are calculated.



and place in the water-bath occasionally.

Procedure:—Out of the three tubes used, in the central tube put 2 c.c. of oxalated blood, add a knife-point of soya-bean or one urease tablet, finely ground with water, 2 c.c. of 0.6 per cent acid Potassium Phosphate, and 2 drops of caprylic alcohol. Shake at 50°C, for half an hour, shaking

Put 10 c.c. of N/100 HCl and 1 drop of Methyl-red solution in the right hand tube and about 10 c.c. of dilute Sulphuric Acid in the left hand tube, the latter being attached to a water-pump. Run a gentle current of air through. Remove stopper from the central tube and add 8 c.c. of saturated solution of Potassium Carbonate. Replace stopper, run current of air gently for five minutes, then run as fast as possible without the fluids bubbling over, leave for 1 hour, with the air current running.

Detach from water pump (do not turn off the tap before detaching). Titrate the acid left in the right hand tube with N/100 NaOH. If x c.c. of NaOH are used the urea in the blood equals $(10-x) \times 15$ mgm. per 100 c.c.

Blood Cholesterol

Principle:—The proteins of the blood or plasma are precipitated and the fatty substances are extracted by the alcohol-ether mixture. The cholesterol is dissolved in chloroform, which will not dissolve soaps, and the colour produced by adding Acetic Anhydride and Sulphuric Acid compared with that obtained from a standard solution of Cholesterol. It is essential that all glassware used should be quite dry.

Procedure:—Run 2 c.c. of blood or plasma into a 50 c.c. graduated flask, about two-thirds full of alcohol-ether mixture,

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shaking the flask gently meanwhile. Hold the flask by hand in boiling water until the alcohol-ether mixture boils, beware of pumping. Cool and make up to 50 c.c. Filter, covering the funnel with a Petri dish to check evaporation. Evaporate 10 c.c. of the filtrate on a water-bath. Extract with 2 c.c. of chloroform and pour into a 10 c.c. graduated cylinder, which must be quite dry, wash out the dish twice with about 1 c.c. of chloroform and make the contents of the cylinder up to 5 c.c. with Chloroform. Measure 5 c.c. of the standard solution of Cholesterol into another cylinder. To each cylinder add 1 c.c. of Acetic Anhydride and 0.1 c.c. of pure concentrated Sulphuric Acid, stopper and mix.

Stand for 15 minutes at 20° to 22°C in the same light as that by which the colorimeter readings will be made. Compare in the colorimeter with the standard at 20 mm. If the reading of the unknown is x, the blood (or plasma) contains 4,000/x mgms. of Cholesterol per 100 c.c.

Blood-Calcium

1. **Principle:**—The calcium is precipitated as Calcium oxalate.

2. The precipitate is separated by centrifuging. As calcium oxalate is slightly soluble in water, a saturated solution of calcium oxalate is used for washing; in order to assist the solution of any protein which may contaminate the precipitate, ammonia is added to the solution.

3. The precipitate of calcium oxalate is dissolved in Sulphuric Acid and titrated hot with N/100 Potassium Permanganate.

Procedure:—In a 15 c.c. graduated centrifuge-tube put 2 c.c. of clear serum, add 2 c.c. of water and 1 c.c. of 4% Ammonium Oxalate solution. Mix thoroughly and let stand 30 minutes or longer. Mix, and centrifuge at high speed for 5 minutes. Decant the supernatant fluid carefully, taking care not to agitate precipitate, put the inverted tube on a pad of filter paper and drain 5 minutes. Wipe the mouth of the tube dry, wash down the walls of the tube with 3 c.c. of 2% ammonia solution; and resuspend the sediment. Centrifuge and drain as described. Add 2 c.c. of normal Sulphuric Acid so as to break up and dissolve the precipitate, and heat in a boiling water bath for a minute. Keep the solution at 70°C or higher, and titrate with a 0.01 N solution of Potassium Permanganate until a definite pink colour lasting one minute is obtained. The strength of the Permanganate solution should be checked with freshly prepared N/100 Sodium Oxalate.

Calculations:—1 c.c. of .01N KMnO₄ solution 0.2 mgm. Calcium. Burette reading $\times (0.2 \times 100/2)$ mgm. calcium per 100 c.c. serum.

Blood Sugar

The glucose in blood is destroyed on standing. Estimation should therefore be started without delay.

Principle:—The proteins of the blood are precipitated with Tungstic Acid and filtered off. An alkaline copper solution is reduced by the reducing substances in the filtrate. Phosphomolybdic Acid added to the cuprous oxide so formed gives a strong blue colour. The colour is compared in a colorimeter with that obtained by using a standard solution of glucose in place of the blood filtrate, and the amount of glucose in the blood calculated from the relative depths of colour.

Procedure:—Measure 2 c.c. of blood, as soon as possible after withdrawal, into a 50 c.c. bottle. Add 14 c.c. of distilled water and after each; add 2 c.c. of $2/3$ N Sulphuric Acid, shaking gently during the addition; then stopper and shake violently; the fluid should not froth. Stand fifteen minutes, during which time the fluid should go brown. Filter, covering the funnel with a Petri dish, the filtrate must be clear and colourless.

Measure 2 c.c. of filtrate into a blood sugar tube, into two blood sugar tubes measure 2 c.c. of standards I and II. To all these tubes add 2 c.c. of alkaline copper solution. Place in a boiling water-bath for six minutes.

Cool the tubes in water for one minute and add to each 2 c.c. of Phosphomolybdic Acid solution. When bubbles cease to appear, dilute each to 25 c.c. Invert several times to mix.

Pour the fluid under test into one cup of the colorimeter, into the other pour the fluid from one of the standards, whichever appears to match the unknown more closely. Set the standard at 20 mm.; if a match is obtained with the unknown at x ; the concentration of sugar in the blood is:—

$2000/x$ mgm. per 100 c.c.—If standard I was used for comparison.

$4000/x$ mgm. per 100 c.c.—If standard II was used for comparison.

Standard I:—Measure 1 c.c. of the 1 per cent stock solution of glucose into a 100 c.c. flask, dilute to 100 c.c. with saturated Benzoic Acid solution.

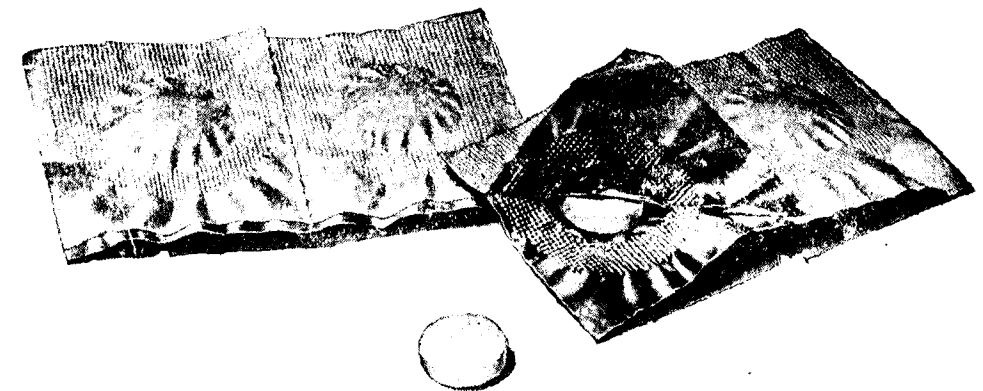
Standard II:—Make up in the same way with 2.0 c.c. of 1 per cent stock solution in 100 c.c.

Van Den Bergh

Required:—(a) Sulphanilic acid solution—dissolve 1 gram of Sulphanilic acid in water with 15 c.c. of concentrated hydrochloric acid and make up to 1 litre.

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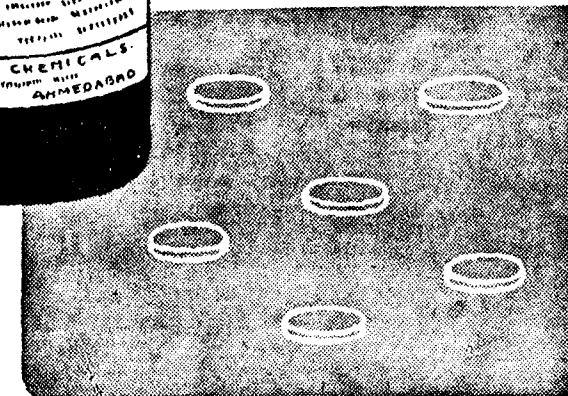
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(b) Sodium Nitrite solution—dissolve 0.5 gram of Sodium Nitrite in 100 c.c. of water.

Before a test, mix 25 c.c. of solution (a) with 0.75 c.c. of solution (b) to form diazo-benzene-sulphonic acid (Ehrlich's diazo reagent).

Principle :—Bilirubin gives a cherry-red azo compound with diazo-benzene-sulphonic acid. If the diazo reagent is added directly to the plasma the colour is obtained when the bilirubin is in one state only (direct reaction), if the proteins are precipitated with alcohol the colour is obtained whatever the state of the bilirubin.

Procedure :—To 1 c.c. of plasma in a centrifuge tube add 0.5 c.c. of diazo reagent. If a reddish colour appears within a minute the reaction is direct and immediate. If a colour appears and slowly deepens the reaction is delayed direct. After 15 minutes add 2.5 c.c. of absolute alcohol and 1 c.c. of saturated ammonium sulphate, mixing after each centrifuge. If a colour was not obtained before the addition of alcohol and a cherry-red colour now appears in the supernatant fluid, the reaction is indirect. If a quantitative estimation is required pipette off the clear supernatant layer into one cup of the clear colorimeter and compare with the cobalt-sulphate solution. Read with the standard at 10 mm. If the reading of the unknown is x, the plasma contains $10/x \times 2.5$ mgm. of bilirubin per 100 c.c.

Glucose Tolerance Test

This test is not indicated if the fasting blood sugar is above 150 mgm. per 100 c.c.

Procedure :—In the morning the patient who has taken nothing but water since the previous evening is bled for sugar estimation, then drinks 50 grams of glucose, dissolved in 100 c.c. of water and flavoured with the juice of one lemon. He is bled again after $\frac{1}{2}$ hour and after 2 hours. With children the dose of glucose should be reduced to 0.5 gram per pound of body weight.

Fractional Test-Meal

Paint the throat of the patient with 1 per cent Cocaine solution and after five minutes pass the Ryle's tube, the end of the tube is lubricated with Paraffin or Glycerine. The end of the tube should reach the fundus of the stomach. The mark on the tube gives an approximate indication of when this point is reached. The contents of the stomach should be emptied by using a 10 c.c. syringe attached to the tube. One hundred c.c. of 7 per cent alcohol are then administered through the tube. About 10 c.c. of gastric contents are withdrawn every half-hour to a period of $2\frac{1}{2}$ to 3 hours.

The gastric contents are examined for :—

- | | |
|--------------------|-------------|
| (a) Free HCl. | (d) Starch. |
| (b) Total acidity. | (e) Bile. |
| (c) Occult blood. | (f) Mucus. |

Free hydrochloric acid :—Measure 10 c.c. of the filtrate from gastric contents into a conical flask, add 30 c.c. of distilled water and 2 drops of Topfer's solution. If the fluid turns red, titrate with N/10 Sodium Hydroxide until the colour turns canary yellow without a trace of orange. If x c.c. of Sodium Hydroxide are required, 100 c.c. of the test meal contain $10x \times 0.00365$ gram of Hydrochloric Acid.

Total acidity :—To the contents of the flask used for estimating free Hydrochloric Acid add 5 drops of Phenolphthalein solution and continue the titration until a definite permanent pink tinge is produced.

If y c.c. of Sodium Hydroxide are used for the titration of all the acids in 10 c.c. of the filtrate, that is, that used for determining the free acid, plus that used for determining the total acidity, $10y$ will be required for 100 c.c., $10y$ is called the total acidity of the filtrate.

Occult blood :—Prepare a saturated solution of Benzidine in 2 c.c. of glacial Acetic Acid, and add an equal amount of 3 per cent Hydrogen Peroxide. Then add 1 c.c. of the gastric juice, and mix well by shaking. A blue or bluish-green colour indicates the presence of blood. Traces of blood may be present due to trauma caused by the passage of the stomach tube.

Starch :—To a few drops of unfiltered gastric contents add a dilute solution of Iodine (N/50 or even more dilute) drop by drop, and shake. A blue colour demonstrates starch, which may be in solution or enclosed in some of the white lumps in suspension.

Bile :—A yellowish or greenish tint indicates regurgitated bile, small amounts are normal. If in doubt, perform Hay's Sulphur test. Sprinkle Sulphur dust on the surface of a specimen of filtered gastric juice. If bile salts are present, the sulphur will sink to the bottom.

Mucus :—Excess of mucus can be detected by naked eye examination.

Chlorides in Blood or C.S.F.

Principle :—Chloride is precipitated as silver chloride by the addition of a known amount of Silver Nitrate. At the same time the proteins are destroyed by heating with Nitric Acid. As Silver Nitrate is present in excess no chlorine escapes.

The Silver Nitrate remaining is titrated with Thiocyanate, the amount precipitated can be calculated and hence the amount of chloride present.

Procedure :—Measure 1 c.c. of plasma into a pyrex test tube, and add 10 c.c. of Silver Nitrate solution and 40 drops of Nitric Acid. Place in a boiling water-bath and leave until the precipitate of protein has dissolved, and that of silver chloride has

formed a large grained deposit at the bottom of the tube. If whole blood is used, 2 c.c. of Nitric Acid should be used and the heating continued until the brown colour disappears. Cool, and if the volume is under about 5 c.c. make it up to this with distilled water, add a knife-point of iron-alum and titrate with Thiocyanate until a faint red colour appears, which lasts on shaking. If x c.c. of Thiocyanate are required, $10-10x/y$ c.c. of Silver Nitrate solution must have been precipitated by the chlorides in the plasma, so that 1 c.c. of plasma contains $(10-10x/y) \times 0.00117$ gram of chloride, that is, there are $(10-10x/y) \times 1.17 \times 100$ mgm. of chloride reckoned as NaCl per 100 c.c. of plasma.

Examination of Urine

1. **Albumin.—1. The heat test :—**The urine, if cloudy, must be filtered, and if alkaline must be rendered acid to litmus with dilute acetic acid (1 in 40). Fill a clean test-tube two-thirds full with the clear acid urine. Slowly heat to boiling point over a small Bunsen flame the top inch of the fluid, rotating the tube but not shaking it. If a cloud, however faint, appears in the heated portion, add a few drops of the dilute acetic acid. If the cloud persists, albumin is present; if the turbidity vanishes again, it was due to phosphates. If the urine remains clear after heating, add a little acetic acid, and heat again, a turbidity due to albumin is sometimes found. The tube must be examined against a dark back-ground.

2. **Sulphosalicylic Acid test :—**Take 2 inches of urine in a test tube, if alkaline, make slightly acid with acetic acid and add 10 drops of 20 per cent Sulphosalicylic Acid. If albumin is present a cloud appears.

3. **The Nitric Acid test (Heller's test) :—**This test, though consecrated by tradition, cannot be recommended. It is not very delicate and is given by several substances other than coagulable proteins.

In routine examination, when albumin is found in urine, it is advisable to make a rough estimate of the amount present as follows :—

If a cloud only appears, such as would cause no appreciable bulk of precipitate at the bottom of the test tube, note that a faint trace, a trace, or a heavy cloud of albumin is present, according to the opacity which is present after boiling and acidifying. If a precipitate is found in bulk, boil the whole contents of the test-tube and allow to stand. When the precipitate has all collected at the bottom, hold an ordinary tape measure against the test tube and read the level of the precipitate as a fraction of the depth of the column of urine, as one-sixth, one-fourth, one-third, etc. If the fraction is one-half, the urine contains about 1 per cent of

albumin, if one-eighth, about 0.2 per cent and if a cloud, only about 0.02 per cent.

II. Reducing substances.—*Fehling's method*:—Preparation of solution:—

(a) Dissolve 36.64 grams of powdered crystalline copper sulphate in warm water, cool and make up to 300 c.c.

(b) Dissolve 180 grams of Sodium Potassium tartrate in 300 c.c. of hot water; filter, add 70 grams of pure caustic soda, cool and make up to 500 c.c.

For use, mix equal volumes of (a) and (b).

To test urine (if albumin is present in the urine, coagulate this by boiling, filter, and test the filtrate) take an inch of urine in one test-tube and an equal volume of Fehling's solution in another. Boil both, add the Fehling's solution to the urine, and mix well. Allow the mixture to stand till cool before deciding that the reaction is negative. With abundant reducing substances, e.g., over 1 per cent of glucose, a reddish or yellow precipitate of cuprous oxide appears at once, while if the amount of reducing substance is small, e.g., under 0.5 per cent of glucose, nothing but a greenish deposit may appear on standing.

Benedict's test:—Preparation of solution:—

(a) Dissolve 173 grams of Sodium Citrate and 100 grams of Anhydrous Sodium Carbonate in 600 c.c. of water; filter into litre cylinder and dilute to 850 c.c.

(b) Dissolve 17.3 grams powdered crystalline copper sulphate in 100 c.c. of water and dilute to 150 c.c., add this solution to the citrate-carbonate solution in a beaker, with constant stirring.

To 5 c.c. of this solution, in a wide test-tube, add 10 drops of the urine, free of albumin, to be tested; boil for 2 minutes and allow to cool. If a reducing substance is present a precipitate will appear, depending as with Fehling's solution, on the amount of this substance. This test is more delicate than Fehling's.

This reducing substance may or may not be glucose in every case. The following are the chief reducing substances other than glucose that may be found in urine:

(a) Fructose.

(b) Galactose.

(c) Lactose.

(d) Pentose.

(e) Glycuronic acid.

(f) Homogentisic acid.

III. Acetone bodies.—*Rothera's test*:—Take solid Ammonium Sulphate in a test-tube to the depth of about 2 inches, fill the test-tube about two-thirds full of urine, shake well to saturate the urine with Ammonium Sulphate, add 3 drops of a strong freshly prepared solution of Sodium Nitro-prusside and $\frac{1}{4}$ inch of a strong Ammonia solution, mixing after each. If acetone or aceto-acetic acid

is present a Permanganate colour develops, reaching its maximum in about 15 minutes. Neither urine nor Nitro-prusside solution should be heated.

The use of fresh Nitro-prusside solution daily is wasteful; a permanent reagent may be made as follows:—

Dissolve 71 grams of Ammonium Nitrate and 5 grams of Sodium Nitro-prusside in water and make up to 200 c.c., keep in an amber bottle in the dark. For use add 1 c.c. of this solution to 3 c.c. of urine, mix and add about 0.5 c.c. of strong Ammonia solution. The colour resulting is less intense and develops more slowly than that produced in the usual way. The reagent will keep for several months.

For acetone alone the Iodoform reaction may be used. To 2 inches of urine in a test-tube add about $\frac{1}{4}$ inch of strong caustic soda, filter if cloudy, and to the filtrate add 1 c.c. of Gram's Iodine. If acetone is present, the smell of Iodoform may be detected or Iodoform may precipitate. This is a very delicate test for acetone in water, but in urine the smell may be obscured by other smells and the precipitate held up by colloids, so that the test is not very satisfactory.

For the detection of aceto-acetic acid, the Ferric Chloride reaction is commonly used. To 1 inch of urine in a test tube add Ferric Chloride (10 per cent) solution drop by drop; a precipitate usually forms which clears up on adding more Ferric Chloride solution. If aceto-acetic acid is present in quantities over 0.2 per cent, a brownish-red colour appears. The reaction is not specific and is given by Salicylic Acid, Rhubarb and Senna and their derivatives.

IV. Bile pigments.—If a test-tube containing bile-pigments in urine is shaken, a greenish-yellow froth appears on top.

Gmelin's test:—Filter several cubic centimeters of urine through a clean filter paper. When all the urine has passed through, dip a glass-rod in yellow Nitric Acid and apply to the filter paper. A spreading wing of colours appears round the area touched by the acid. The colours are from within outwards yellow, red, violet and green. The test is not positive unless the green colour is certainly seen.

Should the bile pigments be present in small amount a positive reaction may be obtained if a considerable bulk of urine is filtered two or three times through the paper.

Iodine test:—Take 2 c.c. of urine in a test-tube. Carefully pour on the top of the urine 1 c.c. of Tincture of Iodine. A green ring appears at the junction of the liquids.

V. Bile salts.—*Hay's test*:—Sprinkle a little flowers of Sulphur on the surface of the urine. If bile salts are present the Sulphur sinks, owing to the reduction of the surface tension of the urine, otherwise the Sulphur floats.

VI. Blood.—For red blood corpuscles and hæmoglobin the guaiacal reaction or a similar test is commonly used.

The Guaiac test:—Boil 1 inch of urine in a test-tube. Allow to cool. Add 2 or 3 drops of Tincture of Guaiacum. A white precipitate forms. Pour gently down the side of the tube 1 inch of ozonic ether. Allow to stand. A blue ring appears at the junction of urine and ether if blood is present. This is a fairly delicate test for the presence of blood or hæmoglobin. It is given also by the urine of a patient who is taking iodides.

Spectroscopic test:—The urine should be examined spectroscopically to determine the presence of hæmoglobin or methæmoglobin.

Intravenous Administration of Atabrine in Falciparum Malaria

The intravenous use of Mepacrine is usually considered dangerous by several physicians. Machella and his collaborators have recorded their experience based on the results of treatment of 125 Chinese soldiers treated in Burma during an active malaria season (June to Oct.) No conscious side-effects. All were blood films positive.

Intravenous mepacrine solution was made by dissolving 0.4 to 1.0 gm. of the dihydrochloride in distilled water (0.2 gm. in 10 c.c.) and this solution was added to 1000 c.c. of normal saline and given by intravenous drip over a period of 3-4 hours.

Quinine the dihydrochloride 1.2 to 2 gm. was dissolved in 30 to 50 c.c. of distilled water and the solution was added to 1000 c.c. Normal Saline and given by the intravenous drip.

S.N. 6911 bisulphide (Quinoline derivative) 0.64 gm. was dissolved in 1000 c.c. Normal Saline and given by intravenous drip.

Atabrine by mouth. 0.2 gm. of the dihydrochlor. was given every six hours for 5 doses and 0.1 gm. thrice daily for 6 days.

Daily thick smears were examined till negative for parasites.

The results were as follows:—(1) *Mepacrine (oral)* 20 cases. Average duration of fever after treatment began was 45.6 hours. Blood films were negative 3 days after treatment. All cases responded.

(2) *Quinine (Intravenous)* 10 cases:—Average duration of fever 45.6 hours in cases where one injection terminated the fever. Blood negative in 3.2 days. In 5 patients one injection failed to terminate the attack and a second was given. A single infusion of 2.0 gm. was given to 3 patients. Alarming symptoms of collapse were noticed and so was discontinued.

(3) *Atabrine (Mepacrine) (0.4 gm. intravenously)* 10 cases:—Average duration of fever 21.8 hours. Blood negative after 2.3 days.

(4) *Atabrine (0.6 gm. intravenously)* 20 cases:—Average duration of fever 29.1 hours. Blood negative 2.3 days. No toxic results; all cases responded.

(5) *Atabrine (0.8 gm. intravenously)* 20 cases:—Duration of fever 24.2 hours. Films Negative after 2 days. All responded; no toxic results.

(6) *Atabrine 1.0 gm. intravenously* 25 cases:—Average duration 31.6 hours. Blood negative 2.2 days. All responded. In 2 cases mental symptoms occurred. In one of these the injection was given in 50 minutes, and that was the reason for the reaction.

(7) *S.N. 6911 (0.64 gm. intravenously)* 20 cases:—Duration 41 hours; blood negative 2.5 days, no toxic signs and all responded. 8 of the 125 cases. The authors emphasise the need for administering the drop in not less than 3 to 4 hours.—(Bull. U.S. Army. Med. Corps-7).

T.N.S.B.

Temperature Chart—Its Diagnostic Value*

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CLINICAL THERMOMETER is one of the simplest instrument in the armamentarium of the modern physician. Though simplest it gives us most valuable information over which rests the diagnosis of many important diseases such as typhoid, malaria and tuberculosis. The very nature of the chart leads us to differential diagnosis and by careful study of the chart we may be able to come to a conclusion as to the nature of the diseases.

What should be recorded in our temperature chart?—To be useful the chart must have some of the salient points besides the temperature. It should have the record of the following, at least, in addition to the name, age, sex, address and date etc.

1. *Date* of the day on which the temperature is being noted.
2. *Day* of the disease which gives us an idea about coming events:

(a) Change in the nature of temperature: (i) begins to fall by lysis in third week of typhoid; (ii) comes down by crisis on 9th day of pneumonia.

(b) Appearance of rash.—We expect the appearance of rash on certain fixed days in certain diseases: (i) *Veriola*—1st day; (ii) *Scarlet fever*—2nd day; and (iii) *Pox*—3rd day.

3. *Pulse*:—No temperature chart can be called complete without the record of pulse. It is slow in the beginning of certain diseases and becomes rapid in later half. In other diseases the pulse has a constant relation with the temperature.

4. *Motions*:—One cannot neglect the number of motions the patient has. It may be a case of simple bacillary dysentery in which the chart will show an acute onset of high temperature with high number of motions.

5. *Urine*:—The quantity and the number of times it is passed.

6. *Respiration*:—This should be noted as the pulse and respiration rates give us valuable information in cases of respiratory diseases.

7. *Rash*:—The time of appearance and its nature.

8. *Laboratory findings*:—(If possible) such as the results of examinations of (a) Sputum; (b) Blood; (c) Stool; (d) Urine; and (e) Serum reaction.

9. *Special symptoms*:—For example (a) Mental condition of the patient; and (b) Intestinal hæmorrhage in typhoid cases.

*Specially contributed to THE ANTISEPTIC.

FIG. 1. A typical Temperature Chart showing the above points. Above we have given the points that should come in the temperature chart as shown in Fig. 1. (*vide chart*). Now let us study a given

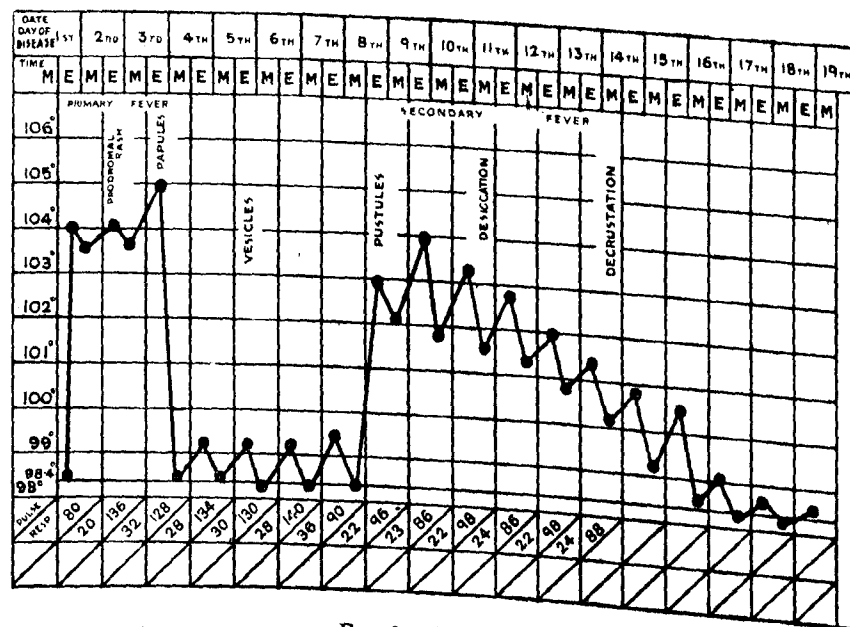


FIG. 1. Small-pox.

chart as regards the fever. By simply looking at the chart we can find out the mode of onset whether it is: (a) sudden or (b) gradual.

Nature of fever:—This will lead us to exclude the other types. If the chart shows that the temperature is remittent type we concentrate towards: (a) Typhoid; (b) Pneumonia and Kala-Azar group, and leave out (i) Malaria; (ii) Filaria; (iii) Localised pyo-mittent fever.

Duration of fever also gives us a clue as to the nature of the disease. Certain fevers are of: (a) *short durations* such as Malaria, fever due to dysentery. They are there for a day or two. In dysentery the chart will show sudden onset, short duration and number of motions. Others are of (b) *long duration*. In this group we can put in pneumonia, dengue and small-pox, while the (c) *very long duration* group will have (i) pulmonary tuberculosis; (ii) kala-azar; (iii) unresolved pneumonia; (iv) typhoid; (v) hepatic abscess; (vi) B-coli infection of urinary tract; and (vii) nervous pyrexia and heat fever of children.

In new born children sometimes the heat regulating centre does not work properly and the temperature is kept at a high level, specially during hot weather, and comes down gradually as the rains set in.

4. **Height of temperature:**—The occurrence of high temperature in the beginning of the illness is a characteristic of all fevers with sudden onset. Hyperpyrexia is observed in the following:—(a) malaria—specially the benign tertian type; (b) pneumonia; (c) cerebral meningitis; (d) septicæmia; and (e) heat stroke.

Let us now take up a few typical Temperature Charts of certain diseases.

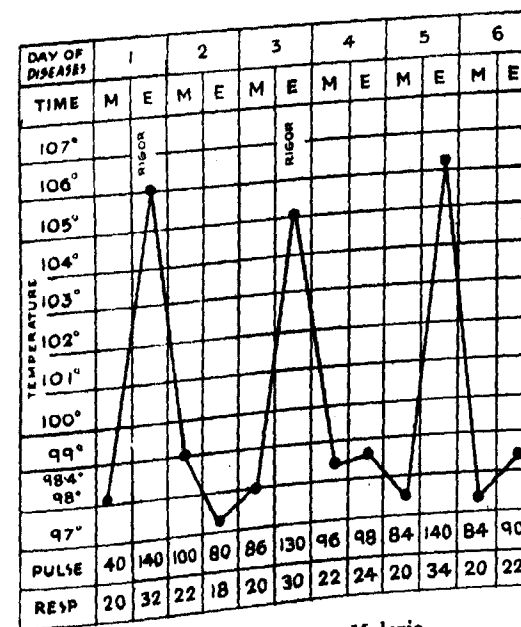


FIG. 2. Benign Tertian Malaria.

Malaria. FIG. 2.—The onset is sudden there is rigor in the onset and the height of temperature is rather high. The chart shows that the fever is that of an intermittent type. The rise of temperature being on alternate days. If a careful examination of a blood slide in a series is done, it will be seen that the malarial parasites are present and that the liberation of the parasites from the R.B.C. corresponds with the rigor that the patient feels.

DURATION.—The paroxysm lasts for five to

six hours and the fever may continue for 15 days or so if untreated. The enlarged spleen and the anæmia will also help the diagnosis.

Pneumonia. FIG. 3.—We see that the temperature has suddenly shot up to 104°F. It is maintained in between 103°F and 104°F for seven days and comes down to 99°F. by crisis and in a day or two it comes down to normal. The pulse and respiration ratio as shown in the chart is 2:1 instead of 4:1. The height of temperature is 104°F. These facts guide us towards the diagnosis of pneumonia. Moreover the signs in the lungs will confirm the diagnosis. (*vide chart Fig. 3, page 762*).

Dysentery. FIG. 4.—Shows a very simple chart. There is only one day fever. If we fail to look up at the number of motions written in the chart it would be rather difficult to come to a conclusion. The number of motions here give us a clue to the diagnosis; together with this the nature of stool gives us the right diagnosis. Microscopic examination of the stool will however clinch the diagnosis. (*vide chart Fig. 4, page 762*).

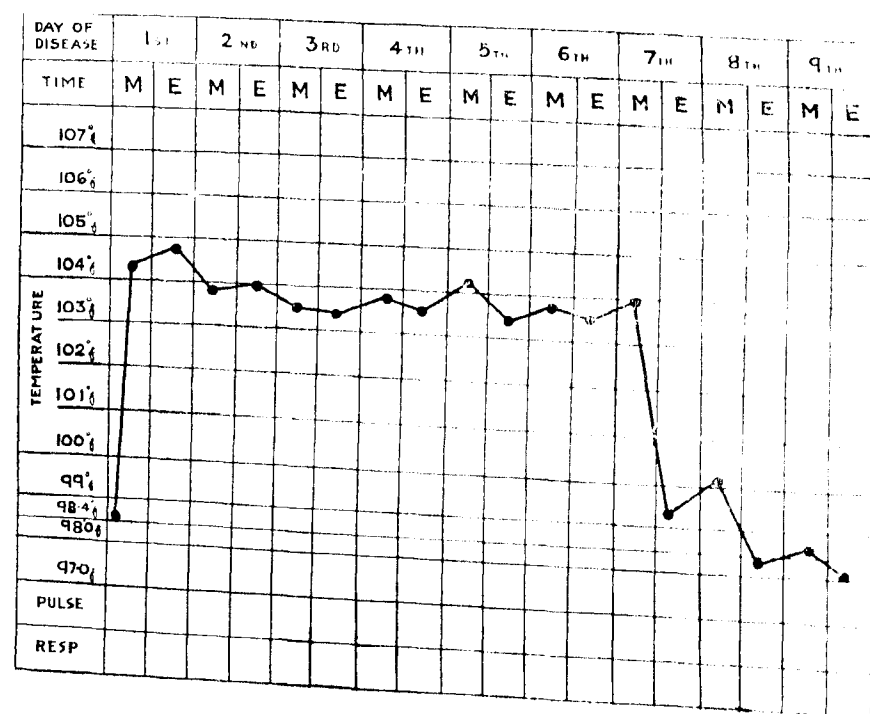


Fig. 3. Pneumonia.

Small-pox. Fig. 5 (*vide chart, page 763*).—The various points of the charts are very distinctly shown in this.—The sudden onset, the height of temperature, the continuous temperature, for 3 or 4 days with slight remissions. Prodromal rash-erythematous on the second day. Macules appear on the 3rd or 4th day and change to papules within 24 hours by which time the eruption is complete. On 5th to 6th day the papules change to vesicles. The temperature has come down during this period only to shoot up again when the vesicles change into pustules. Temperature rising upto 103°F or 104°F during this

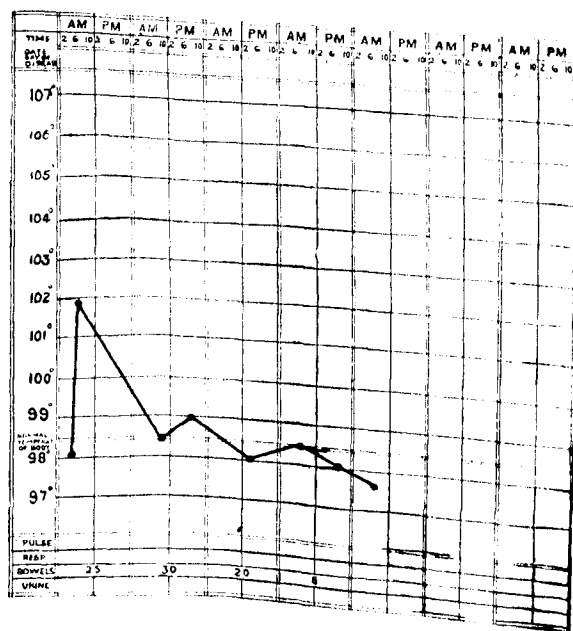


Fig. 4. Bacillary.

gradually goes down with the drying up of the pustules and forming crusts by 11th or 12th day (desiccation). By the 14th to 15th day the process of separation of crusts-decrustation has begun and the temperature comes to 100°F or so.

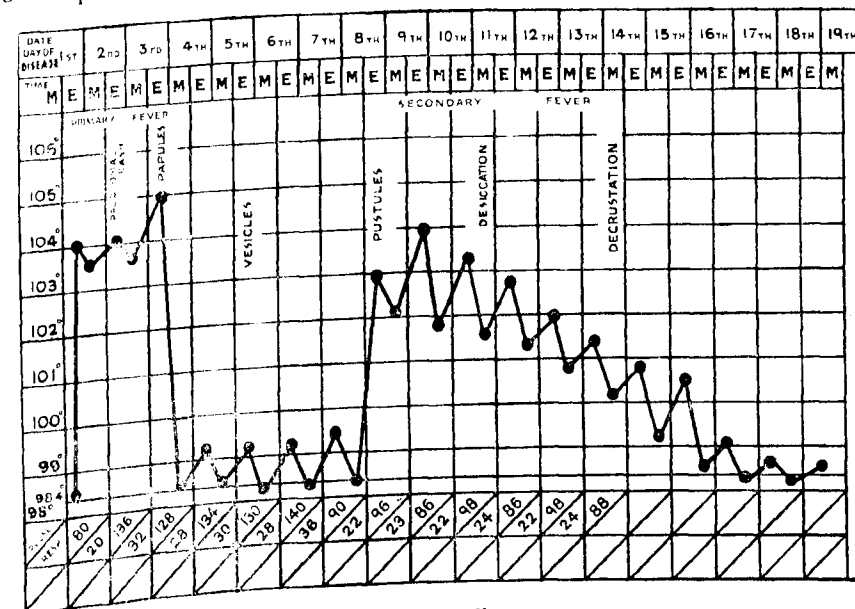


Fig. 5. Smallpox.

If we keep a close record of the events occurring we will be able to predict some of the coming events and their time as to when they should be expected.

Measles. Fig. 6.—The chart begins with a high temperature which has suddenly gone up to 103°F or so. Coryza—running of the nose and eyes with a flushed face accompanying the high temperature there is irritating cough. On second and third day the temperature has come down by two degrees or so. On the fourth day

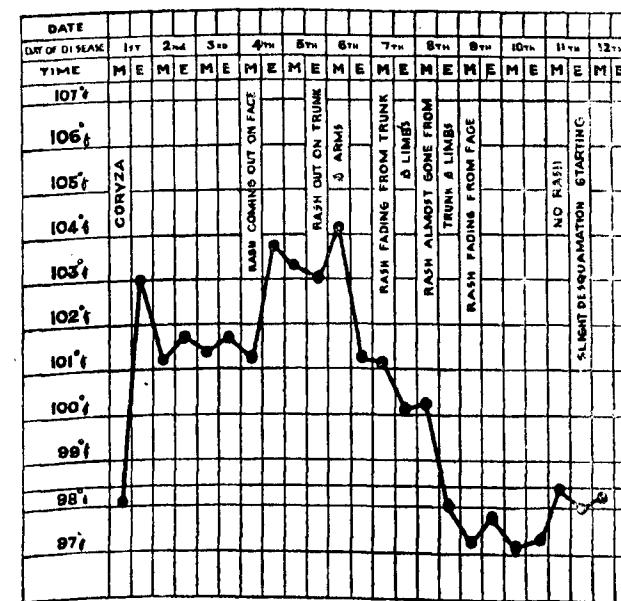
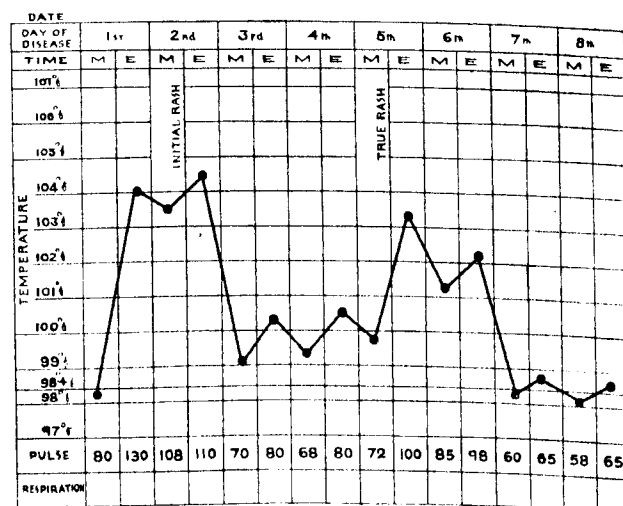


Fig. 6. Measles.

rash is seen appearing on face. The temperature has shot up to 104°F . Fifth day there is a slight fall in temperature only to shoot up to 104.2°F or so with rash appearing on the trunk and limbs. Symptoms are aggravated with the approach of rash on trunk and limbs. By 7th day the rash has faded and by 8th day it is almost gone. 9th day shows that the rash on the face has begun fading. The temperature during these days is gradually coming down. By 11th day there is no rash and the range of temperature is within the normal. Slight desquamation has started by 11th day.

Dengue. Fig. 7.—The saddle back type temperature chart of dengue fever is well-known. There is sudden onset of fever which



Leucopenia
Progressive Relative Bradycardia
FIG. 7. Dengue.

risers of temperature with the stage of remission in between give the appearance of the "Saddle Back." This is the commonest variety of the temperature curves seen in dengue.

Typhus. FIG. 8.—The onset is usually sudden with a rapid rise of temperature, chill, rigor and severe headache, vomiting, flushing of face and marked prostration. The temperature reaches its height (104° — 106°F) on the 3rd day, when the patient may be restless, delirious or even comatose.

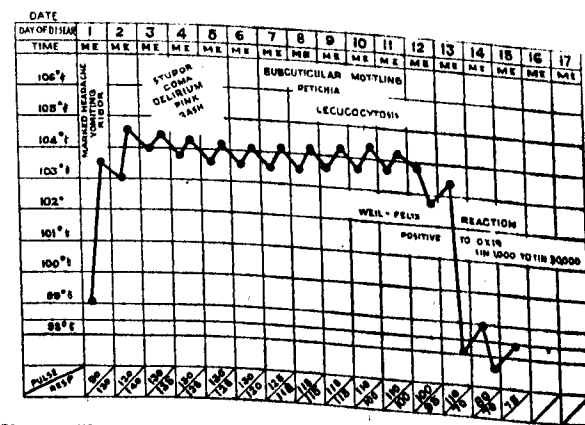


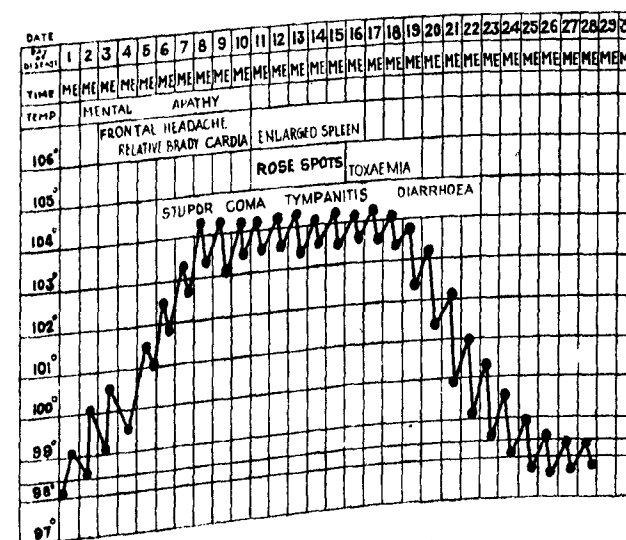
FIG. 8. Temperature Chart in a typical case of louse typhus.

Tongue is coated with brownish white fur. Pulse is rapid in proportion to the temperature.

Rash:—The rash appears on the 4th or 5th day on chest, abdomen, back, limbs and in axilla. The face, neck, palms and soles are exempted. The rash is subcuticular mottling of a pinkish colour. By 7th day it becomes deep pink. By 10th day the rash becomes deep brown. By 12th to 14th day it begins to disappear leaving a brown stain.

Blood:—Shows a moderate leucocytosis 10,000 to 14,000 per cm. with an increase of polymorphonuclears. The serum of typhus fever patients taken after the fifth day of disease agglutinates B proteus OX19 in high titre (Weil-Felix reaction).

Crisis appears on the tenth to the fourteenth day followed by disappearance of the rash in about a week leaving brownish stains in the skin.



Widal's Reaction +
Faecal Culture +
Urine Culture +

FIG. 9. Temperature Chart of a typical typhoid case.

Fever—Temperature rises steadily by steps (ladder like rise) being a degree or more higher on each successive day.

Tongue is coated with a whitish fur in the middle, the tip and the margins are red and clear.

Anorexia is present.

Gurgling in the right iliac fossa. Abdomen is tumid and slightly tender. Bowels are usually constipated.

Pulse is slow in proportion to the height of temperature.

Blood slight polymorphonuclear leucocytosis (8,000 to 10,000).

Typhoid. FIG. 9.—The onset is usually insidious. A sudden onset is not uncommon. The common premonitory symptoms are frontal headache, lassitude, anorexia and bronchitis, vague abdominal discomfort. The clinical symptoms that may be expected week by week are:

FIRST WEEK.—Patient is somewhat restless, cheeks are flushed and eyes bright.

Spleen is slightly enlarged towards the end of the week (sixth to seventh day).

Headache diminishes towards the beginning of second week.

Rose spots appear in successive crops over the abdomen, the sides of the chest and back between 7th and 10th day.

SECOND WEEK—General:—The toxæmia has set in and the patient is stuporose and does not respond to questions.

Temperature reaches its fastigium and becomes steady varying between 103°F and 105°F . Tongue is coated with yellowish white fur at the centre and the margins and tip are red. The lips, teeth and gums may be dry and covered with sordes. The lips, teeth tumid, there is tympanitis. Pulse is soft and rapid 120 to 140 per minute. Blood shows moderate anæmia and leucopenia with relative lymphocytosis. Spleen is larger and definitely palpable. Hæmorrhage from bowels and perforation may occur by the end of 2nd week.

THIRD WEEK—General symptoms may not increase in mild cases, though in severe cases the patient may pass into typhoid state.

Temperature begins to remit and begins to decline gradually (lysis) towards the end of this week.

Pulse is rapid. Symptoms gradually subside and patient becomes a convalescent.

Special diagnostic methods:—(1) Blood culture very important for diagnosis in the first week, when other materials, urine and stool culture and Widal test would not be of much use.

(2) Urine and stool culture—Positive results are usually obtained during the 1st to 4th week.

(3) Widal's test is usually positive in most cases after the 8th or 9th day of typhoid fever.

Kala-azar. FIG. 10 (*vide chart, page 767*).—The most characteristic type is remittent or even intermittent pyrexia with double or triple rise in 24 hours. The temperature subsides in the early morning and remains low until about midday. It rises in the afternoon, subsiding again in the evening. At about 8 or 9 p.m. it rises again or the second rise may be delayed until midnight and the characteristic feature of the fever is that the patient with a temperature of 102°F or more may be doing his work in the ordinary way and be quite unaware of the pyrexia.

Tongue is usually clean, appetite good but digestion poor.

Spleen is enlarged and soft.

Liver is enlarged and soft.

Pulse is rather rapid even when there is no temperature.

Blood pressure is low about 100 mm. of Hg. Marked pulsation in the neck.

Blood count etc.: R.B.C. $2\frac{1}{2}$ to $3\frac{1}{2}$ millions; hæmoglobin 40%; sedimentation rate of R.B.C. is increased; and progressive leucopenia.

Skin is rough and dry, pigmented in patches.

Laboratory findings:—

Blood: (a) Progressive and marked leucopenia; (b) characteristic differential count:

(i) diminution of polymorphs below 50 %;

(ii) relative increase of lymphos and mononuclears.

(c) proportion of white to red cells is 1 to 1,500; and (d) detection of parasites in the peripheral blood.

(e) proportion of white to red cells is 1 to 1,500; and (d) detection of parasites in the peripheral blood.

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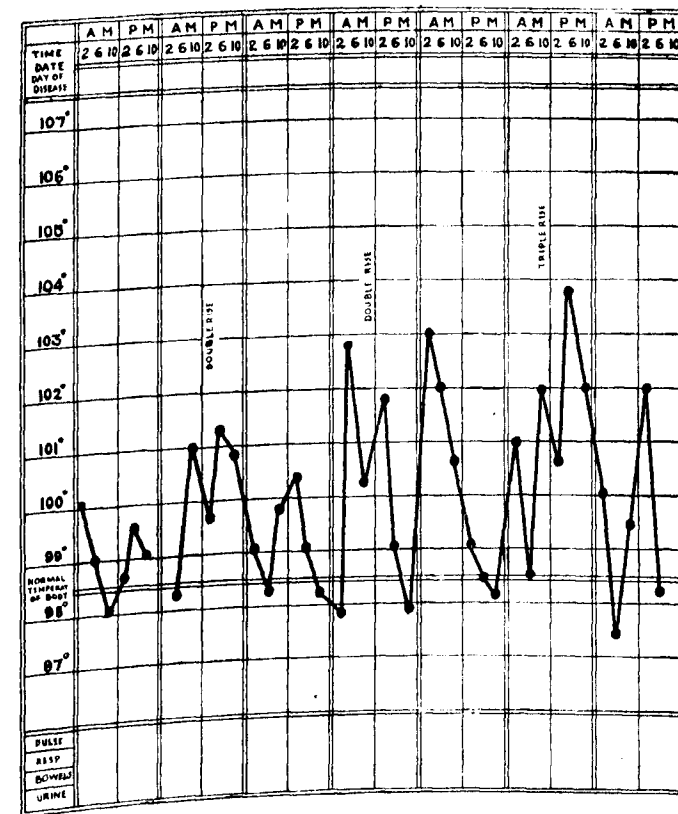


FIG. 10. Four hourly Temperature Chart of Kala-Azar showing double or triple rise in twenty-four hours.

Serum tests:—(a) Napier's aldehyde test is positive. (b) Chopra's antimony test is positive.

Culture:—Blood culture in N.N.N. media is positive for L.D. bodies.

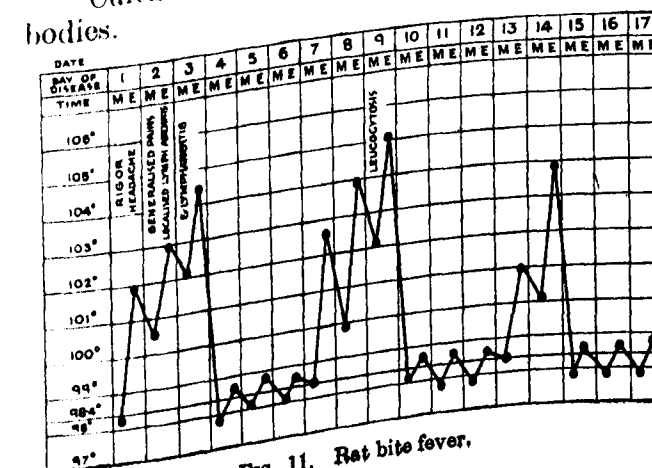


FIG. 11. Rat bite fever.

Sternal puncture:—Smears from sternal puncture are positive for L.D. bodies.

Rat bite fever. FIG. 11.—Sudden rise of temperature with rigors, headache and generalised pains and a rapid pulse.

The site of bite which might have healed by now again shows signs of inflammation—formation of few vesicles and even ulceration. Localised lymphangitis and lymphadenitis may be present.

Temperature rises to 103°F to 104°F. Assumes a remittent character lasting for 3 to 6 days. Comes down by crisis with marked sweating. Sometimes there is a rash—urticarial or maculopapular—on trunk, limbs and face. This may be with every spell of temperature or during one spell only.

The crisis is followed by an afebrile period of 2 to 8 days when all symptoms disappear except weakness. Then there is another bout of fever for a few days to be succeeded by another afebrile interval. The cycle of febrile and afebrile period lasting usually for 5-6 days repeats itself for weeks or months till the attacks are milder and spontaneous cure ensues.

Blood examination:—(1) Leucocytosis with increase of polymorph and eosinophilia; (2) anaemia; (3) spirillum may be detected in the stained blood smear; and (4) W.R. may be positive.

Pulse:—Closely follows the curve of temperature, febrile and slow during afebrile period.

Tuberculosis. Fig. 12.—The temperature chart is typically a hectic one. One cannot predict what will be the temperature of next day. All sorts of rises and falls with irregularities can be found. Yet it can be said that the 4 p.m. temperature rises invariably.

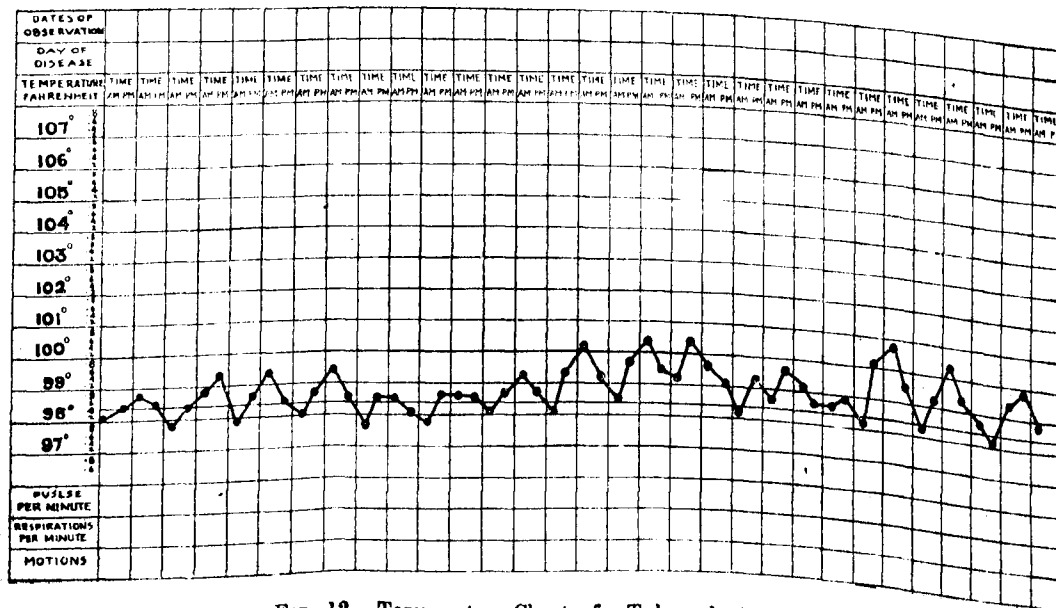


FIG. 12. Temperature Chart of a Tuberculosis Case.

Pulse is quick. Digestion—Loss of appetite. Weight—Loss of weight. Night sweats—The patient complains of night sweats.

Laboratory findings:—Sputum is positive for acid-fast bacilli.

X-ray shows generalised mottling or cavity formation at a particular spot.

General emaciation—Patient is very much emaciated.

Hæmoptysis—There may be spitting of blood with the phlegm.

Conclusion.—1. The essential points to be given in a temperature chart are discussed.

2. It is shown how an up-to-date temperature chart can be of aid to diagnosis.

3. Typical temperature charts of certain diseases with their typical symptoms are given.

References:

1. Price—Practice of Medicine.
2. Savil—A System of Clinical Medicine.
3. Banerjee and Bhattacharya—A Handbook of Tropical Diseases.
4. Napier—The Principles and Practice of Tropical Medicine.
5. Class Notes by Prof. R. N. Chaudhary, School of Tropical Medicine, Calcutta.

Therapeutic Value of Chaulmoogra in Leprosy

Schujman records his 17 years' experience of treating lepromatous cases with large doses of chaulmoogra oil, until the patients have remained free from symptoms for some time. Reported failures, he considers, are due to deficient doses and duration of treatment. Untreated lepromatous cases become worse and irregularly treated ones show an unfavourable evolution. Previously untreated cases nearly always improve, often very rapidly. Cases not benefited by small doses benefit by larger ones and may become bacteriologically negative. Schujman believes strongly, in injecting the maximum amount that can be tolerated in the minimum time, as the best method for ensuring success. All his patients—hospitalized—who became clinically and bacteriologically negative had received a total dosage of 1500 c.c. of Ethyl Esters, preferably with 4% Creosote. Of those who showed only slight improvement, only 27% had received the full dose. The injections are made intramuscularly and intradermally. With the full dosage (30 c.c. weekly) 40% of all treated lepromatous cases have been rendered clinically and bacteriologically negative and another 40% have become definitely improved.—(*Internat. Jour. Leprosy*, 15: 2, 135-45).

T.N.S.B.

Syphilis Interfering in the Treatment of Kala-Azar

Four cases of infantile Kala-azar which resisted treatment with Tartar Emetic, were found by the Carnevale to have a concomitant syphilitic infection. These cases were then treated specifically for syphilis with a course of salvarsan. Prompt response was thereafter obtained to antimony treatment. A syphilitic infection should therefore be suspected and looked for, in all cases of kala-azar which resist treatment with tartar emetic.—(*Carnevale A. Pediatrics*, 51: 145-55).

T.N.S.B.

Migraine Ophthalmoplegia (Unilateral)

D. N. GOEL, D.O.,

(Eye Specialist), Sri Maharaja Gulab Singh Hospital, Jammu, Kashmir.

I am enclosing herewith two photographs of a case of migraine ophthalmoplegia for requesting the medical profession to suggest the line of treatment for checking and preventing the attacks.

The case was a young boy of 18 years. He has been getting the attacks of severe pain in the right eye, headache, vomiting and gradual ophthalmoplegia. The recovery used to occur after two to four weeks.

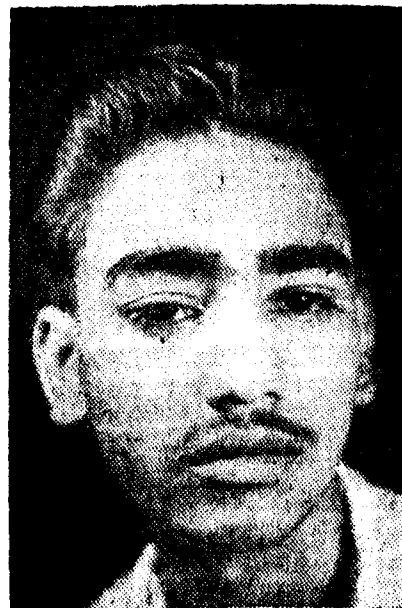
He got about fourteen attacks at different intervals and almost all resulted in recovery.

The first attack started when he was of six years of age.

The last attack for which he attended the "Eye Clinic" of Sri Maharaja Gulab Singh Hospital was in the month of May 1949.

Family history:—Father: living—normal. Mother: living—normal. Brothers: six—all living and normal. One died due to boil when he was 8 days old. Sisters: six—all normal.

Migraine Ophthalmoplegia



S. K., 18 years (30.8.49).

Past illness:—Suffered from plague when he was of 5 years of age.

[770]

Description of the last attack:—He got sudden and severe pain in the right eye and had vomiting—headache on the right neck, right head and the occipital region.

During the attack he could not take anything and vomited out whatever he took by mouth.

Gradually his right eye closed (ptosis) which persisted along with the pain.

The ptosis gradually subsided along with the subsidence of the pain. On examination of the right eye there was ptosis. The eye was deviated outwards. Pupil was dilated—not reacting to light. Distant vision was normal, near vision was much affected. The medial were clear—fundus was normal. No colour vision changes. Eyeball was immobile. There was diplopia on using both eyes. His blood for Kahn's test was negative repeatedly, urine contained albumin. Gradually the pain subsided and ptosis passed off. The eye gradually assumed normal position but the pupil remained dilated and not reactive. This condition is now persistent.

The attack was the severest of all the previous attacks as the previous attacks used to subside within a month.

His left eye was normal.

His other systems were normal.

The photographs were taken when he started improvement from ptosis.

There was never any response to any of the analgesic drugs, salicylates and luminal.

Bromides by mouth and Vitamin B₁ parenterally and Vitamin B complex by mouth gave little or no response. Codein was also tried for the pain but with little advantage.

Gower's mixture was also prescribed for sometime but could serve no useful purpose. Rest to the eyes was given by dark glasses.

Streptomycin in Human Plague

Karamchandani and Rao treated during an epidemic, five moribund plague patients with intramuscular injections of streptomycin only. The dose was 0.125 gramme every 3 hours and 24 to 30 such were given. Improvement resulted with a total dose of 1.5 grammes, no toxic effects were noted; all the patients recovered and are quite well. Only 5 cases in a moribund condition, (the epidemic of 152 cases with 66 deaths) could be treated as the streptomycin supply was enough only for five.

[This communication was sent to the *Lancet* by Lt. Col. P. V. Karamchandani, during the last quarter of 1947 and was published in Jan. 1948. There have been subsequent reports by others on the successful use of this drug in plague but Karamchandani lays the prior claim to this finding.]—(*Lancet*, 1948, Jan. 3, 22.)

T.N.S.B.

A Case of Puerperal Septicaemia

SHARENDRA NATH BAGCHI, M.B., B.M.S. (std.),
Calcutta.

Case I.—Mrs. N. Pal, H. F., 33 of Calcutta, a multipara with 8 children, was delivered of a female child in which her placenta had to be removed bi-manually under chloroform anaesthesia about $\frac{1}{2}$ hour after the third stage of labour was completed. The doctor sterilised his hands with soap and water and applied Tinct. Iodine on his hands up to the elbow when he performed the operation of the removal of placenta of his patient without using a pair of sterilised gloves. On the 2nd day following the operation of headache and backache and felt nausea and vomiting, loss of appetite and was constipated. As her lochia was scanty, dirty-brown and foul-smelling instead of pure blood with some shreds of decidua as the normal cases, I took a blood slide and decided on no M.P., total W.B.C. count. The blood slide revealed culture showed streptococcus B-Haemolyticus. Her pulse was 130 per min., respiration 36 p.m., urine showed no sugar or albumin, reaction was acid and other systems were N. A. D. A diagnosis of puerperal septicaemia was given.

TREATMENT.—Rest in bed, a nurse to watch over the patient and her baby was given to a wet-nurse for care and management.

Mag Sulph $\dots 3$ iv
Aqua Chloroform $\dots 3$ i given stat

With a sterile catheter and a glass syringe the following mixture was introduced into the uterine cavity. Tinct. Iodine *Mellis* Rectified 1 part, Glycerine pure 9 parts. Of the above mixture one drachm introduced daily for 4 days, Penicillin $\frac{1}{2}$ lac units in pyrogen-free redistilled water injected intramuscularly every 4 hourly for five days along with Irgafen (N, 3, 4 Dimethyl Benzoyl Sulphanilamide) tablets 1 gm. every 8 hourly given for 4 days. Alkaline mixture 3 i t.d.s. given for 4 days. Glucose-D, barley water and green coconut water are given to the patient. Diet—milk, bovril, fruit juices, biscuits. From 5th day lochia improved and became watery and was not foul smelling.

	Morning	Evening	Morning	Evening
1st day	Temp. 104°F	Temp. 105°F	P. 130 Resp. 36	P. 140 Resp. 40
2nd day	" 102°F	" 103°F	" 120 " 36	" 128 " 36
3rd day	" 101°F	" 102°F	" 110 " 28	" 120 " 32
4th day	" 98.5°F	" 99°F	" 100 " 26	" 108 " 28
5th day	" 97°F	" 97.6°F	" 90 " 22	" 90 " 22

Patient was cured.

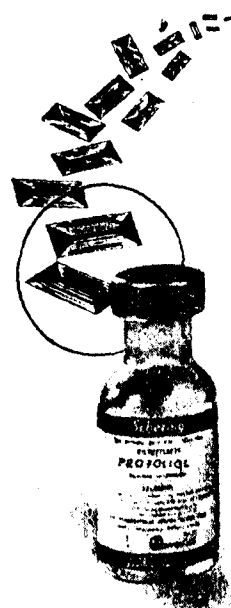
Reference:

O. St. John Moses—Manual of Obstetrics, 1920.

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Some Clinical Findings:

Mono-articular Arthritis of right shoulder joint.

Male—age 51, started getting mild pain in right shoulder six months back. With salicylate and iodide for nearly a month there was no response. Lactoprotein Iodide injections with salicylate by mouth did not help. Dr. and Dr. advised respectively Novocaine and Percaine injection locally or other mixtures but these could not help. Lastly under general anaesthesia his joint was mobilised which after nearly three months of his illness has been much limited in movement. The matter went from bad to worse.

As a last resource, he was put on Tablet RHEUMOPHAN. Gradually, improvement occurred. The pain has been much less, movements are better and his sleep has been regained. Even now the treatment is continued as the restrictions in movements are still continued and slight pain is existing. No injections given during this last treatment.

Osteo-arthritis of spine.

Male—age 45 Merchant. The patient complained of pain in spine and kyphosis—duration 4 years. On X-Ray examination previously diagnosed as Osteo-arthritis of spine.

Treated with RHEUMOPHAN COMPOUND tablets and with three months treatment his pain completely disappeared. He was able to move about and do ordinary work. It is now about a year that the treatment is stopped. He is now in Burma and doing his normal work.

Osteo-arthritis of knee-joint.

Male—age 60—Farming. Patient complained of pain in the knee joints and left shoulder joint since last six months. Had been treated with several medicines but very slight improvement.

Treatment with RHEUMOPHAN COMPOUND—I am not in a position to give X-Ray and other laboratory findings. However you will be glad to know that the patient who was using your medicine has remarkably benefited by it.

Neuralgia.

Female—age 70. Getting tingling, numbness and shooting pains all over the body, more on the chest for the last six months, the patient was shouting day and night. High blood pressure 180/110. Urine normal—put on treatment for high blood pressure and a course of vitamin B₁ and Iodide and salicylate injections—the B.P. came down to normal but the pains did not subside.

She was put on Tablet RHEUMOPHAN COMPOUND and in about three weeks' time all her complaints of Neuralgia pains disappeared and she was able to sleep. Treatment was continued for three months. It is now a long time since she has left treatment and still she is keeping well without a relapse.

Stiffness and pains in joints & muscles after an injury.

Female—53 years. Fell down on slippery ground. Got pain in the knee joint and wrist (left)—could not get up—severe pinching pain in the legs and hand—Allopathic and Ayurvedic drugs given (With Tablet RHEUMOPHAN COMPOUND) I treated my mother. Now she moves her hands and legs. Relieved of pain in the whole body—taken her bath regularly—can sit in bed and stand by the help of the wall for two minutes.

Literature from Masters of Tablet LEPTADEN and MYRON
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Eclampsia

HARENDRA NATH BAGCHI, M.B., B.M.S., (Rtd.),
Calcutta.

Case No. 1:—Mrs. A.M.F., 24, of Mymensingh, a primipara, complained of persistent headache, vertigo and epigastric pain. She also complained of nausea and vomiting whenever she rises from her bed. There was solid oedema of lower extremities and urine diminished in quantity. On the top of it she had an epileptic-like fit lasting about 2 minutes, a typical clonic and tonic convulsions, biting of tongue, foam issued from her mouth with open eyes fixed in vacant stare, pupils dilated, twitchings at the alae nasi followed by unconsciousness and on regaining consciousness forgetfulness of immediate past events. There was no epileptic cry. On examination:—Woman of medium height, good musculature, pale and oedema of lower extremities noticed. Uterus enlarged to the size of 9 months' pregnancy, fetal movements present and fetal heart sounds audible with changes in the breast, urine 3 oz. acid, albumin present, sugar nil, specific gravity 1010, hyaline casts present in plenty, percentage of urea decreased. Patient constipated, temperature 98°F., pulse 100 p.m., high bounding respiration 24 p.m. Blood pressure Systolic 160 } m.m. of Hg.
Diastolic 80 }

DIAGNOSIS:—Eclamptic seizure.

TREATMENT:—Patient put to bed, a nurse appointed to look after her, a folded towel kept ready to be put between the teeth during a fit, Calomel gr. iii stat and an enema given after 2 hours consisting of Glycerine—3i, oil olive 3viii, warm Normal Saline solution to 2 pints. After evacuation of bowels the following mixture introduced through a rectal tube slowly.

R Sodi Bromide	.. gr. xxx
Chloral Hydrat	.. gr. xv
Aqua Distil	.. 3 iv

No food given by mouth except dub water iced and glucose D solution (3ii to 1 oz. of distilled water) small sips frequently.

The 2nd fit occurred after 4 hours' duration, ½ minute, and patient was not unconscious.

Normal Saline Solution	.. 1 pint.
Sodi Bicarb	.. 3 ip fit.

Fit. Lotion was introduced in her rectum slowly.

Patient was given hot bath for 10 minutes, a poultice of Antiphlogistine to her loins and hot water bag kept there.

Morphia	.. gr. i
Atropine Sulph.	.. gr. 1/150

injected hypodermically. A third fit after 8 hours lasting ½ minute. Urine measured in 12 hours 15 oz. Stools watery, yellow twice in 2 hours.

Half pint of Normal Saline Cum Sodii Bicarb given subcutaneously:

Morphine Sulph.
Atropine Sulph.

gr. $\frac{1}{150}$ } injected hypodermically.

No fit, patient had labour pain followed by delivery of a dead fetus on the 3rd day. Patient recovered and no complications thereafter reported. The term eclampsia signifies a set of tonic and clonic convulsions associated with the pregnant condition and occurring during advanced pregnancy, labour or the puerperium. The convulsions are not identical with hysterical fits but resemble the convulsions of epilepsy. Eclampsia rarely occurs before the twenty-first week of pregnancy and more common during the last 2 or 3 months of gestation. It may manifest itself a few days after parturition.

ETIOLOGY:—Cause is not definitely known. A close connection has been observed between renal inefficiency and hepatic affection. The result is that there accumulates in the blood of women, the victims of eclampsia some toxin which the organs are unable to dispose of or neutralise.

B. Hicks showed cases in which the albumin made its appearance only subsequent to the convulsions.

Kussmaul suggests convulsions closely associated with anaemia of the brain. The following facts have been noticed:—

(1) Albuminuria is always found to be present before or after a fit.

(2) Disease of the kidney is associated with the condition in some cases.

(3) Eclampsia is more common in primipara.

(4) More common in tropics.

(5) Danger of the mother and the child is greater in proportion to the number of fits.

(6) Protracted labour is a predisposing cause.

(7) The liver changes (hepatitis and presence of thrombosis in the portal vessels) are probably the primary lesion in eclampsia.

Signs and symptoms in a case of eclampsia of moderate severity:—Warning symptoms of eclampsia are headache often on one side of head and face during pregnancy, attacks of vertigo, sudden loss of memory, drowsiness nervous irritability, insomnia, flashes of light before the eyes, dimness of vision (signs of optic neuritis?) and a humming in the ears. Oedema of the subcutaneous tissues of the upper limbs and face or of the lower extremities appears.

The onset of the convulsive attack is sudden and the fit epileptiform but *no initial cry*. There occurs a short stage of tonic

convulsions and the expression of the patient's face is fixed and eyes staring and the patient becomes *unconscious* and *stiff* and falls to the ground if not supported. Her pupils become contracted, eye-balls turn upwards, jaws are fixed and the tongue is thrust out of the mouth and bitten. Then clonic spasm starts, muscles twitch, eye-lids open and close, eye-balls move in their sockets and the pupils dilate. Then the face muscles begin to twitch and commence to jerk. The face becomes livid, neck veins swell and the muscular jerkings spread to body and lower limbs. The breathing becomes stertorous and action of the heart intermittent. Respiration at first temporarily arrested and then becomes irregular and the bladder and rectum evacuate spontaneously. The hands and the bladder and rectum begin to jerk, while the body is becomes clenched and the arms begin to jerk, while the body is thrown into a state of opisthotonos and the body is bathed in cold sweats. The patient during this time is *totally unconscious*. This condition lasts about 3 or 4 minutes. The rigidity commences to disappear after the cessation of the attack and the patient passes into a state of coma when an attack is subsiding consciousness is gradually restored and pupillary reflexes appear. During the attack urine becomes scanty and loaded with albumin. The coma is probably due to congestion of the brain and when the patient recovers she seldom recollects past events. The termination may be:—

(1) The condition may pass off after labour and the patient recover.

(2) Post partum hæmorrhage may take place.

(3) Cerebral hemiplegia may follow the recovery or some distinct impairment of intellect.

PROGNOSIS.—The prognosis is grave when it appears during pregnancy and gravest during labour. Maternal mortality is from 20 to 30 p.c. and the infantile mortality from 25 to 50 p.c.

Immediate cause of death:—(1) Asphyxia due to prolonged tonic spasms; (2) Asphyxia combined with exhaustion; (3) Anæmia of the brain; (4) Failure of the heart; and (5) Pulmonary oedema and pneumonia.

TREATMENT.—**Prophylaxis:**—In cases of pregnancy where albumin is discovered the possibility of the occurrence of convulsions should be kept in mind. The patient must be warned against all kinds of excitement, her diet regulated and milk diet is advocated, iron tonics pushed, her skin kept active and bowels stimulated.

Curative:—To reduce high blood pressure: (1) blood-letting, about a pint of blood; (2) administration of hydrogogue purgatives Pulv. Jalap and Calomel. Blood-letting not advocated now-a-days.

1. **Arrest the fits:**—(a) By favouring elimination; (b) by putting an end to pregnancy where possible. The rapid dilatation of the cervix, followed by rapid artificial delivery is condemned as

shock and sepsis may follow; and (c) by administering sedative mixtures *e.g.* Morphia, Chloral and Bromides.

2. *Ward off further attacks*:—(a) By reducing the metabolic processes—keeping patient on a starvation diet; (b) by washing out the stomach; and (c) by keeping the bowels free.

Symptomatic treatment:—(a) Support the heart; (b) watch the depression in the respiratory function (i) Digitalin and Strychnine, (ii) Oxygen inhalation, Atropine, artificial respiration.

3. *Favouring elimination of the toxic material*:—(a) Keeping bowels empty; (b) promoting sweating; (c) securing free flow of urine by injecting subcutaneously 1 pint of the following Saline Cum Sodi Bicarb Solution.

Sodi Chloride	3 i	} Not recommended in cases of oedema.
Sodi Bicarb	3 ii	
Aqua Distill	1 pint	

Linseed meal poultices applied to the loins and dry cupping advocated.

4. *Terminating the pregnancy*:—Prognosis improves when the patient is safely delivered.

5. *Administering medicines*:—(1) Chloroform is in disfavour in toxæmic conditions; (2) Morphine is recommended, for in diminishing cerebral irritability and in reducing metabolism it helps to ward off the fits; it does not depress the heart and has no effect on the kidneys—initial dose is $\frac{1}{2}$ gr. 2nd and subsequent doses gr. $\frac{1}{4}$ every 4 (four) hourly; (3) Chloral and Bromides are given in eclampsia for the fits; and (4) no medicine by mouth to unconscious patient.

Diet.—Patient starved during fits up to 2 days, then give small feeds of milk food: rectal saline 6 oz. every 6 hours advocated.

6. *Gastric lavage*:—By a stomach tube.

7. *Keeping bowels free*:—By a low diet and rectal enema. Bowels should be worked by purgatives. Keep 8 oz. of Saline Cum Sodi Bicarb solution to rectum after rectal douchings.

8. *Supporting the heart*.

Digitalin and Strychnine administered when the action of the heart is weak. Coramine and Cardiozal may be of use.

9. *For respiratory depression*:—Oxygen inhalation and Atropine injected, also Coramine and artificial respiration.

10. Employ experienced nurses with hospital experience.

After the attack the patient must be kept in bed on a mil. diet for about 2 weeks and urine examined for albumin.

11. Diaphoresis by hot air baths and Pilocarpine. A careful watch should be kept for oedema of the lungs.

References:

1. Sajou's Analytic Cyclopaedia of Practical Medicine, 7th Edition Revised (Vol. 7).
2. O. St. John Moses—Manual of Obstetrics, 1920.

43, Baghbazar street,
Calcutta.

Clinical Evaluation of Penicillin Therapy in Syphilis

S. N. TRIPATHI, L.M.P.,

Ex-Pathologist, Raghunath Group of Hospitals, P.O., Ambikapur, Surguja (C.P.)

PENICILLIN although a potent anti-syphilitic drug cannot for the present replace the time-honoured Arseno-Bismuth therapy.

My experience with Penicillin in the treatment of syphilis is not upto my satisfaction unless and until adjuvant treatment with combined Arseno-Bismuth therapy is also done. I quote below some cases in which although prolonged and massive treatment with Penicillin was given the results were not gratifying and the clinical improvement produced was not satisfactory.

CASE 1:—A male, aged about 60, suffering from polyarthritis.

Quantity of Penicillin injected:—2 mega units of crystalline Penicillin 'G', 3 mega units of Penicillin in oil and beewax.

Result of treatment:—Clinical improvement noticed was not very marked. According to the version of the patient there was some amelioration in the pain of the bigger joints only. I was called to see the patient after some time of the Penicillin treatment, and was requested to take up the case under my treatment in the month of Oct. 1948.

On examination I found: Patient suffering from polyarthritis. Very much reduced and bedridden. Knee, ankle, elbow, wrist and phalangeal joints badly affected. Duration of illness: About 2 months. History of V. D.: Denied.

Clinical investigation:—Urine examination showed no pathogenic abnormality. Serological test (Kahn's) + + + (strong positive).

TREATMENT:—T.A.B. Vaccine 25 millions organisms injected intravenously which produced hyperpyrexia as a result of which swelling and pain in the bigger joints were markedly diminished.

Mapharside: Dose 0.04 gms. given in the 1st injection. Later on 0.06 gms. given (six injections).

Stabismol: 1 c.c. every 4th day. T.C.F. whole liver extract injection with Vitamin B complex etc. every 4th day. Mixture containing sufficient quantity of Pot. Iodide was also given.

Result of the above treatment:—Patient felt much better only after the 1st doses of Mapharside and Stabismol. He was able to leave his bed and walk with the support of a stick after the 4th injections of Mapharside and Stabismol. And by the 7th injection of Mapharside the patient was apparently cured and after a few days of the last Mapharside injection he was able to move about and walk without the support of a stick even for a long distance and after some time he began to attend to his work in his village.

CASE 2:—A young man (Government servant), aged about 33 years. Suffering from polyarthritis with marked pyrexia. Quantity of Penicillin injected:—30 lacs units freeze dried Penicillin (Glaxo).

Result of Penicillin treatment:—According to the patient's version Penicillin in his case was simply a waste. I also hold the

same view. Later on when Penicillin did not do him any good he sought admission in the Sadar Hospital where he was treated for rheumatic fever with adequate doses of Sodium Salicylas and Soda Iodide injections without any effect. Having been disgusted with his life he came out of the Hospital and later on he sent for me in Nov. '48.

On examination I found: He was suffering from polyarthrititis. Knee, ankle, elbow and wrist joints greatly affected. He was almost reduced to skeleton and was completely bedridden so much so that he could not cover and uncover himself and was unable to change his sides even. Duration of present illness—3 months. History of V.D.—present.

Treatment already done. In addition to the treatment already mentioned he was given only one injection of N.A.B. 0.15 gm. about three months back after which he left the place for Ambikapur for the sake of treatment. Rheuminol two injections were given before starting anti-syphilitic injection and before serological test was done.

Clinical investigations:—

Total leucocyte count		..	9,800 per c.mm.
Diff. : Leucocyte count		..	%
Neutrophile polymorphonuclear	67%	..	6,566 "
Lymphocytes large and small	28%	..	2,744 "
Large hyaline mononuclears	1%	..	98 "
Eosinophile polymorphonuclears	4%	..	392 "
Total	100%	..	9,800 "

Hæmoglobin 9.4 grams per 100 c.c. of blood.
Total R.B.C. count 3.4 mil. per cmm.

B.S.R.—84 at the end of one hour (Westergreen). Serological test (Kahn's test) + + +. Clinical and microscopic examinations of urine revealed no abnormality.

TREATMENT.—T.A.B. Vaccine 25 millions organisms injected intravenously and this injection was again repeated and the dose was doubled this time. This time the reaction was very severe, the temperature shooting up to 105.8°F. Patient got delirium and hence as the relatives of the patient got very much frightened I had to stop further injections.

Sulfarsenol injected every fifth day I.V. in gradually increasing doses. Bismecol 1 c.c. I.M. every fourth day. A mixture containing sufficient quantity of Pot. Iodide was also prescribed. Ferilex (T.C.F.) was prescribed as a tonic.

Result of above treatment:—In all seven injections of Sulfarsenol and six injections of Bismecol were given which apparently cured the patient. Patient began to show distinct improvement only after the first injections of Sulfarsenol and Bismecol and there was more and more clinical improvement with each successive injection. B.S.R. was reduced to 22 mm. at the end of one hour; as this was

still higher I gave a few injections of Anti-rheumatic Vaccine and only two injections of S.B.T. after which the patient left the place to resume his duties in the interior and has been maintaining sound health till now.

CASE 3.—A male, aged 55, suffering from paresis of the left hand and optic neuritis of the left eye. Duration of illness: Several years. He was treated before inadequately with Arsenicals. About two years back he was injected three mega units of Penicillin Sodium Salt three hourly. The clinical improvement noticed in this case was not very satisfactory. Serological test done after six months of the Penicillin treatment was + + +. About six months back he was again injected 5 mega units of Crystalline Penicillin G (Sodium Salt) which too has produced only subcurative effect. Serological test recently done is + +.

CASE 4.—A male, aged about 25, had suffered before from syphilis for which he was treated by some quack. Later on he contracted gonorrhœa for which he was injected 3 mega units of freeze-dried Penicillin. About one and half months back he came to me with his wife with multiple vague complaints. He was more worried about his wife who was getting palpitation of the heart and had severe frequent headache. Having taken the history of the cases I decided to do serological test (Kahn's test) which was + + + in the case of the wife and + + in the case of the husband. The husband just after a couple of days fell ill and suffered from typhoid and hence his treatment could not be started. I gave only two injections of Bismostab to the wife. This has relieved her much. Her further treatment could not be continued owing to the serious illness of the husband.

Comments and suggestions.—The status of Penicillin as a sole curative agent in the treatment of syphilis has not yet been established and hence it would be unwise on our part to depend on Penicillin alone.

Penicillin although is very effective in the early stage of the acquired disease, it would not be desirable to rely on Penicillin alone and hence adjuvant treatment with arsenicals and a heavy metal is always advisable. In combined infections of "G and S" Penicillin should only be used if the particular gonorrhœal infection has proved resistant to Sulphonamides. In patients who have been treated for gonorrhœa with Penicillin repeated serological tests are necessary for the detection of the concomitant infection of syphilis because the dosage of Penicillin required to cure gonorrhœa is subcurative in syphilis but is sufficient to delay or prevent the development of the primary sore thus masking the infection until the occurrence of the secondary eruptions or positive serological findings. Now since an equally good and effective antibiotic viz. Aureomycin has been discovered and in the near future may be available in adequate quantities it would be worthwhile to use this in gonorrhœa, more so in cases where there is any reason to suspect that concurrent infection

with syphilis is also probable as this product has been found to be equally efficacious in gonorrhoea as is Penicillin but so far has not been reported to have any action on *Spirocheta Pallida*.

In primary syphilis I personally reserve the use of Penicillin for combating the secondary infection in which I have found it to be extremely effective and for this purpose only a few injections are required.

Penicillin no doubt has proved to be of great value in many cases of neuro-syphilis but in meningo-vascular disease where there has been no preparatory treatment with Bismuth severe Herxheimer reactions have been reported. In tabes dorsalis and G. P. I. no dramatic clinical improvement usually occurs.

In cardiovascular syphilis it may produce serious Herxheimer reactions and hence it is necessary to remember that whereas in early syphilis a Herxheimer reaction following the exhibition of this powerful anti-spirochetal substance can be ignored, in late and latent syphilis it can be a very real danger. Hidden lesions may be present in the heart and great vessels and for this reason the treatment of late and latent syphilis should always be cautiously commenced. It is a good rule in these cases not to use Penicillin or Arsenicals without preparatory treatment with Bismuth.

The clinical and serological response of infantile congenital syphilis to Penicillin is although as satisfactory as it is in the early stage of the disease it would not be desirable to depend on Penicillin alone and hence adjuvant treatment with Arseno-Bismuth therapy is always preferred. Herxheimer reactions may be dangerous in undernourished and marasmic infants in which treatment should always be cautiously started.

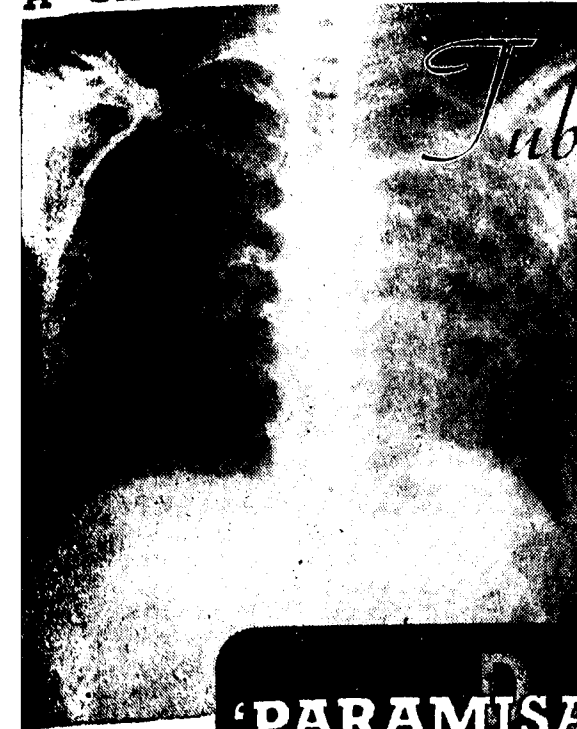
Considering the above facts and figures I am of the decisive opinion that Penicillin should not be used indiscriminately and haphazardly in the treatment of syphilis. Arseno-Bismuth therapy should be given the first preference unless there are contraindications to it. Arseno-Bismuth therapy should be the treatment of choice specially in poor patients to whom treatment with Penicillin can be done in experiment. Adjuvant treatment with Penicillin will be a costly cases of patients who can afford it and who are very much after it.

Arseno-Bismuth therapy has been now made more safe since the introduction of the powerful detoxicant *viz.*, B.A.L.

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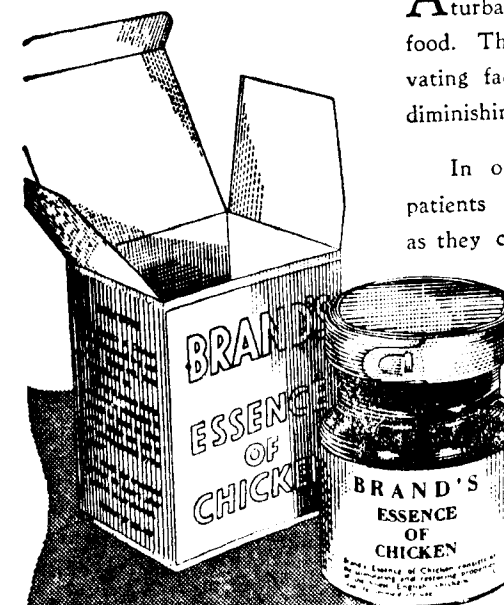
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The Health Minister's Panacea

IT was a fervent appeal that the Hon'ble Dr. T. S. S. RAJAN, Minister for Public Health, made, while presiding over the annual prize-giving ceremony of the Madras Medical College held last month, that the medical graduates should utilise the knowledge they have gained for the service of the poor and the afflicted. It is not that the small number of medical graduates turned out from the all too few colleges in this Presidency are insensible to their responsibilities. If they are not able to serve the poor and the ailing as well as they desire and would, the fault lies elsewhere and unless something is done to keep the wolf from their doors by improving their position and their pay and prospects, to enable them to make available to the needy public the knowledge they have gained, there appears to be very little prospect of improvement in the present position.

It has been a long-standing complaint that while the need for medical personnel had increased enormously with the advance of so-called civilization and the increase in the number of nameable and unnameable diseases afflicting the people, there are not a sufficient number of medical colleges in India to prepare the personnel required to meet the ever-growing needs. This matter received considerable attention at the hands of the Bore Committee, and, while their recommendations have not gone far enough, the Governments in the land are unable to give effect even to those recommendations on the so-called ground of paucity of funds, least realising that the more the facilities provided for medical relief and the improvement of public health, the greater will be the well-being and prosperity of the country, social, industrial and economical. In our own Province steps were taken to start two more medical colleges, but the attempt was given up owing to lack of funds.

The position comes to this: the need for medicos is very great; and the establishment of more medical colleges is beyond the range of possibility at present owing to financial considerations. How then to solve the problem? The Hon'ble the Minister for Public Health suggests two ways to solve the problem. One is a more generous valuation by the examiners, bearing in mind the needs of the people; the other is the chalking out of a more simple and practical system of medical education by which they could turn out capable and qualified doctors in sufficient numbers to meet at least to a certain extent the present needs.

The first suggestion owes its origin to the figures furnished by the Principal, of the large number of failures in the final examination. According to the statement of the Principal only 42% of those who sat for the final examination came out successful. Influenced perhaps by the view given expression to by the Universities Commission very recently, that examinations are hardly a satisfactory test of merit, the Hon'ble the Minister appears to have jumped to the conclusion that the medical examiners are hard to please and therefore an appeal to them is called for to make them more generous in the valuation of papers than is the case at present. Whether examinations are a satisfactory test of the merit of candidates is a very large question for policy-framers to decide, that we think it unnecessary to enter into a discussion of the subject. But in the consideration of the subject in the present state of things, we will be failing in our duty if we ignore certain factors which are not a little responsible for the present sorry state of things.

The failure of 58% of the examinees in the final examination is certainly deplorable from the point of view of the Government and from the point of view of the students also. Medical education is a very costly one; the Government spend, according to the Minister, a sum of Rs. 1,000 a year on each student in a Medical College; and the amount spent by the student also is considerable; and the failure of a large number of students every year affects the finances of the Government and of the parents of the students alike, very much. But who is responsible for this state of things? Can the Government absolve itself of all responsibility in this matter? While admissions are regulated by the meticulous balancing not only of communal but also of regional claims, thereby depriving students of real merit from entering into the portals of the medical college, and throwing open the gates to students merely because they belong to a particular community or region, irrespective of their merits, how can one expect a result different from the one we now have? Recently at least 20% of the seats in these colleges were reserved for students of merit, but that order has been withdrawn and communal and regional considerations have been allowed to dominate in the selection. So long as this pernicious principle is followed, we are afraid any improvement is out of the question, and any

appeal to the examiners to lower the standards, so long as examinations continue to be the test for merit, would hardly be helping the cause of educational standards or discipline. It will also lower the prestige our medical colleges have for imparting instruction of a high order.

We are surprised that the second suggestion for the evolving of a simple and practical system of medical education should have emanated from one who was in our province responsible for the abolition of the shorter L.M.P. course and the elimination of the caste system in the profession. Just as English education was originally introduced in this country to prepare people for clerkships under the East India Company at a comparatively low administrative cost to the Company, so also the shorter L.M.P. course was instituted by their successors to make available medical men at a low cost to make a show of catering to the needs of the people. What all serious problems it had created and how much the medical profession as a whole had to fight to get rid of this caste system are matters of recent history into which it is unnecessary for us to enter for our purpose. Suffice it to say, that the adoption of any plan on the lines recommended by the Hon'ble the Minister will only create a less qualified class of medical men and women and revive again the caste system which it had taken decades of agitation to bring an end to. And such a lesser course of education cannot but lower the standard.

True it is, as the Hon'ble the Minister has stated, that there is a great lack of medical personnel to cater to the needs of the people; equally true it is that there are not sufficient men to tackle even sporadic epidemics like cholera which are taking a heavy toll every year. While therefore we are wholeheartedly with the Hon'ble Minister in his view that the medical personnel should be considerably increased, the remedy adopted should not be worse than the disease itself. It is all very well to say that, left to himself, he would take in every willing person who would offer to serve the country in this behalf. But he is not left to himself and the very fact that the doors of medical colleges are closed against students of real merit who are willing to serve the country is itself proof positive that there is something rotten in the present arrangement which requires to be mended, if not ended altogether. Let the Hon'ble Minister use all his eloquence and influence to bear upon his colleagues in the cabinet against the present communal and regional arrangements, and the battle will be half won.

We are not of those who hold the view that the present long course of five and a half years for medical education is sacrosanct and should not be touched. Opinion is slowly and steadily veering to the view that such a long course is uncalled for and unnecessary, that it is quite possible to reduce the course considerably to the great advantage of the State and of the student by cutting down

certain amount of ritual intended to impress the public without in any way impairing the efficiency of the training. It is in these lines that the medical profession is thinking, and a close collaboration between the profession and the State will be of great help in evolving a curriculum of studies which, while cutting down certain subjects and thereby shortening the course, will still maintain the efficiency of the profession unimpaired.

By the time this issue is published, the South Indian Medical Association would have met in Conference. We trust the Conference will consider the suggestions put forward by the Hon'ble Minister in all their bearings and make known to the Government its considered views in the matter. Of one thing we are certain. There can be no revival of the caste system in the profession, and this we are sure the Conference will make clear to the Government. We await the proceedings of the Conference with considerable interest.

Dental Education and Treatment

THAT Dental Health is a *sine quo non* for general health has now come to be accepted as an axiom in all countries. That is why the importance of oral hygiene for maintaining intact the health of the individual and the need for the provision of adequate dental health services have received considerable attention in the advanced countries of the West. There is at least one dentist for every 3,000 of the population there. But in India the matter has upto very recently received scant attention. Therefore the Bhore Committee put forward in its Report a programme of development and made pointed reference to the fact that although in all progressive countries the dental care of the population is an essential part of any comprehensive scheme of public health, in India the matter has not received that attention it deserves. The Committee recommended a considerable expansion of training facilities in this field; the regulation of dental training and of the practice of the profession through the establishment of Dental Councils at the Centre and in the Provinces; and the organisation of dental services for the people in the urban and rural areas.

The Government of India have placed on the Statute Book in 1948, The Dentists' Act, throwing open the right of entry to the Register to all persons of Indian domicile who have been practising dentistry as their principal means of livelihood for a period of two years during the five years before the commencement of the Act, at the same time prohibiting the registration thereafter of unqualified persons and also making it impossible for unregistered practitioners to practice dentistry in the country. When those who, though unqualified, secure registration under the saving clause on the ground of having practised the profession for two years in the

five years previous to the passing of the Act, are eliminated, dental service in the country will contain only of qualified people. But if the country is to have an adequate number of qualified dentists, every effort will have to be made to expand greatly the existing training facilities which are altogether insufficient now.

But as pointed out by Dr. C. S. RAMAN, Honorary Secretary of the Madras Provincial Branch of the All-India Dental Association, while the production of a large number of workers in dentistry is a prerequisite to the creation of machinery for the regulation of the profession, the establishment of adequate dental services for the people is dependent on the expansion of training facilities.

If India is to be placed on a par with other countries in the West in the matter of dental treatment, if the schemes of public health and medical relief are to be made as comprehensive as they are in the West, at least 80,000 dentists will be required for the whole of the Indian Continent. Simply to place an Act on the Statute Book for the regulation of the profession of dentistry without making proper and adequate arrangements for the training of people in dentistry will be just like putting the cart before the horse. Therefore a number of dental colleges have to be established all over the country. The Bhore Committee have recommended that Dental Colleges should be established at Calcutta, Bombay, Madras, Lucknow and Patna, as well as one in Delhi in association with the proposed All-India Medical Institute. But just like all schemes helpful to the people are put off for lack of funds, so also this proposal has been held in abeyance.

But we are glad that the Government of Madras have recently appointed a Dental Surgeon to the General Hospital and Professor of Dentistry in the Medical College. At the instance of the Government we understand he has submitted a scheme for the establishment of a proper Dental College and hospital. We are also glad that the Hon'ble. Dr. RAJAN, Minister for Public Health, is fully alive to the importance of this problem and is doing his best to solve it. The Government should realise that dental equipments are different from the ordinary equipments used in hospitals and dental education and relief cannot be adequately given in Medical Colleges as they are today. A separate College and Hospital are necessary if dental education is to be given on sound and proper lines and dental relief is to be well provided for. May be that the Government cannot in the present circumstances afford to set apart large sums for these purposes, but if proper appeal is made to the richer classes of people we are sure money will come in. If they are impressed on the need for the establishment of the College and the Hospital, they will loosen their purse strings for this noble cause. We appeal to the Hon. the Minister for Public Health to take up this matter in right earnest and usher in a new era.

Gleanings From Medical Press

MEDICINE AND THERAPEUTICS

Dosage and potency of liver extracts used in the treatment of pernicious anaemia. (*Lancet*, 22-1-'49, 5-2-'49, 12-2-'49, 19-2-'49, *Lancet*, 1947: 2, 468).

In his Oliver Sharpley lecture before the Royal College of Physicians in March 1948, Dr. J. F. Wilkinson of the Manchester University dealt with this subject at great length. Dr. McSorley raised the question again in the *Lancet* for 22-1-'49. Dr. Wilkinson, in a letter to the *Lancet*, dated 5-2-'49, has very useful remarks and suggestions to offer.

Adequate dosage of liver extract depends on several factors but in particular on the degree of anaemia, the presence of complications, and haemopoietic potency and strength of the liver extract used. He stresses the need for full and adequate dosage to be given and maintained till the blood count is normal. The initial dosage will vary from 2 c.c. daily to 4 c.c. twice or thrice a week with subsequent reduction to the maintenance levels of 2 to 4 c.c. weekly or fortnightly. He does not recommend intervals longer than 2 weeks between individual doses. "These doses are much greater than pre-war ones," says Dr. Wilkinson, "for several important reasons." For example, many batches of commercial liver extract supplied for use, cannot have been properly and individually tested clinically before issue. From time to time he has recently come across patients with quite uncomplicated pernicious anaemia who have failed to respond to massive doses of "Liver Extract". This occurs much more with highly purified liver preparations. It is not proper for manufacturers to assume that because one batch is active the next and subsequent one will also be equally so even when the same conditions of manufacture have prevailed; every batch must be tested or else bulked with others to be tested in large batches.

From the early days of the war up to the present time another startling factor has operated. Pre-war liver extracts for the treatment of pernicious anaemia were

made from fresh or frozen, but healthy animal livers, and the manufacturer had a good starting material. The best liver extracts were, in fact, made from fresh calf liver; and fresh livers from other young herbivorous animals were particularly potent. Now the livers are disposed according to the directions of the British Ministry of Food, and so they are not available for preparing highly active extracts to save the lives of patients with pernicious anaemia. The manufacturers have, therefore, to use so-called "pharmaceutical livers" i.e., livers unfit for human consumption that have been rejected in the slaughter houses on account of disease. The more obvious diseased parts are no doubt excised before use but the starting product is nevertheless faulty and contains much less or little healthy normal liver tissue and therefore less of the active principle. Many may contain none at all, of this active principle even before extraction begins. Subpotent liver extracts are thus produced and so greater doses are required for treatment.

Dr. James Innes of the Edinburgh University Department of Medicine relates his experience at the blood clinic in the Royal Infirmary. He agrees with Dr. McSorley that 4 c.c. of a reliable liver extract is the minimum monthly dosage required for maintenance of satisfactory blood levels. Some elderly patients, however, do better with 2 c.c. at fortnightly intervals. It is not sufficient to prescribe a monthly dose of 2 c.c. as recommended by manufacturers, for with this dosage blood examination shows subnormal levels.

Dr. G. E. Shaw of the Evans Biological Institute, Cheshire, agrees with the Editorial in the *Lancet* of 22-1-'49, that it is desirable to have some unitage system for describing antipernicious anaemia potency and that the units should preferably be referable at any time to a standard clinical potency and should be correlated with microbiological activity (Dorner Assay of Vit. B₁₂ or some modification thereof.)

Dr. D. L. Mollin of the London Postgraduate Medical School, says that in his experience, it is exceptional with the liver extracts now available in England, to observe the optimal rise of red cells continue for 14 days after a single injection of liver extract in the amount recommended by the manufacturers or for the bone marrow to be completely normoblastic at the end of this time. He gives an example:—"The red cell count of a patient with pernicious anaemia treated for almost a full year 1946-47 with 2 c.c. a fortnight of a concentrated British Extract failed to rise above 4 million per c.mm. Later when she relapsed, a concentrated American Extract in the same dosage as before, restored the normal values in 3 months. The potency of liver extracts should be more critically assessed."

Drs. Callender and Wills of the Oxford Radcliffe Infirmary, used Anahaemin (which they themselves tested for potency before use) in 65 cases and found 2 c.c. produced maximal rise of red cells over a period of 2 or 3 weeks in a patient in relapse. More liver may be required for maintenance than for the induction of a remission.

Dr. Dyke of the Wolverhampton Royal Hospital, disagrees with Dr. Wilkinson about the intervals between doses, in the treatment of pernicious anaemia, (should not be greater than 2 weeks). Four to six weeks intervals or even more would be proper according to Dr. Dyke. The dangers inherent in the presence upon the market of liver extracts, either totally inactive or of suboptimal potency have long been manifest and can only be met by the assay of every batch of liver before it is put into use. He does not use in his clinic any preparation not previously subjected to such assay. He also advocates the use of the cruder preparations and avoidance of highly refined ones.

Drs. E. Lester Smith and W. J. Cuthbertson of the Glaxo Laboratories Ltd., confirm Dr. Wilkinson's finding that the liver available for extraction varies in quality and is sometimes poor in antipernicious anaemia factor, but are not inclined to accept the latter's sweeping condemnation of the liver extracts in

the market. They share Dr. Wilkinson's view that every batch should be clinically tested before release. They also state that the batches of liver extracts produced by them are all individually tested both clinically and microbiologically before issue. They had to discard several sub-potent batches and during a shortage of clinical cases had to withdraw their preparation temporarily from the market.

Dr. Jens Dedichen, Chief of the Medical Faculty in the University of Oslo (Norway), states that he had made extensive observations and tests with Liver Extracts for the last 3 years and found that hog-liver gave active extracts only in the summer and autumn months, while liver from pigs given "winter-food" was quite inactive or pretty nearly so. The usual raw material the author and his co-workers have been using is calf-liver, and they found no such variation in calf-liver as they found in hog-liver. Experience in Norway with Darner assay for antipernicious anaemia potency was similar to that reported by Dr. Shaw in *Lancet* of 5-2-'49.

[Note.—The above notes and comments by different and independent medical men, would seem to point to the great need for caution amongst our doctors in India, relating to the choice and use of liver extracts for the treatment of anaemia.] T.N.S.R.

Ascorbic acid in subacute bacterial endocarditis.—(D. Aleksandrow-Polsk, Tyg. Lek., 1948, *Abst. World Med.*, 5: 3, March 1949).

Ascorbic acid hastens the proliferation of fibroblasts and is indispensable for production of connective tissue fibres and formation of scars. In the opinion of the author, the recurrence of symptoms despite intensive treatment with penicillin, is perhaps due to a delayed and insufficient healing of the endocardial lesions, which forms a source of re-infection. The serum of 15 patients was estimated for ascorbic acid during and after penicillin treatment, and an attempt was made to determine if intensive saturation with ascorbic acid would affect the course of the disease.

In all these 15 patients, the amount of ascorbic acid was much lower (0.16 to 0.3 mg. per 100 c.c.) than in healthy controls (0.41 to 0.62 mg. per 100 c.c.). This was not due either to the effect of the fever or the chemical action of the penicillin. The low level of ascorbic acid was not related to the degree of cardiovascular failure. In patients who after penicillin treatment had no fever for 40 days, the level of ascorbic acid gradually increased and a normal saturation reaction with a positive balance was restored. The author, therefore, suggests that large doses of ascorbic acid should be given to all patients with subacute bacterial endocarditis during and after penicillin treatment. He advises 300 mg. every day during the first month and 100 mg. thereafter every day.

T.N.S.R.

Streptomycin in the treatment of tubercular enteritis.—(*Am. J. Med. Sci.*, 217: 1, 28-47, Jan. 1949).

A co-operative study involving 15 hospitals, several doctors, numerous other hospital personnel has been summarised by Mason, Kridelbaugh, Crouch and Ward at the request of the Veterans' Administration's Central Office Committee on Streptomycin. This investigation was launched in June 1946 and was at first directed towards the effect of streptomycin on pulmonary tuberculosis. It was, however, soon extended to other forms of tuberculosis. It was resolved by the Veterans' Administration's Third Conference in May 1947, that the experience of all hospitals be collected and the results published in a single paper. The large volume of information gathered is sufficiently complete to permit certain conclusions of value being drawn.

Selection of cases.—The protocol governing the treatment of tuberculous enteritis permitted the use of streptomycin in cases with proved pulmonary or extrapulmonary tuberculosis, who had gastro-intestinal symptomatology with radiological evidence of an intestinal lesion which could be reasonably considered tuberculous. Several other ulcerative lesions as added complications responded favourably to the

treatment. 33 cases (all male), of the ages of 20 to 58 were studied. All had far advanced pulmonary tuberculosis, and cavities were present in 21 cases. Positive cultures of sputum or gastric washings were reported in all cases. Intestinal symptoms were present in 12 for 4 to 6 months prior to streptomycin treatment. All the 33 cases had diarrhoea or constipation, alone or alternating, abdominal pain, soreness or loss of appetite, nausea, vomiting. In 4 tuberculous appendices had been removed. Two had rectal fistula and 2 had tuberculous laryngitis as well.

Technique.—The daily dose varied from 1 to 2 grammes and within this range no difference in response was noted when the intramuscular route was used. 1 gramme was later recommended in 2 doses, and the incidence of vertigo was reduced considerably. Oral treatment 2 gm. per day in 5 doses was used in treating 2 patients for 66 and 120 days respectively. Four to six weeks elapsed before complete relief was obtained as compared with one week when intramuscular treatment was used. The oral route has the advantage of avoiding toxic manifestations and the development of resistant organisms. 19 patients completed courses of 60 to 120 days. 14 day courses were used in 6 cases to provide symptomatic relief. 8 cases were still under treatment having received 81 to 86 days of the streptomycin.

Clinical results.—These have been uniformly good and shown relief of symptoms of tuberculous enteritis in this series. After the initiation of the treatment by the intramuscular route improvement resulted almost immediately and maximum benefit was reported in one week in 20 cases. Four investigators said that in 3 days all abdominal pain was completely relieved. "Relief only slight on oral therapy but complete no intramuscular" was the verdict of one based on 4 cases. Tremendous increase of appetite was recorded in nearly all cases after one week.

Radiological findings.—In 17 cases the authors examined skiagrams and discussed the findings with the Radiologists in the hospital. All these cases

showed abnormalities before treatment; and in six the findings returned to normal after treatment. In 8 cases definite improvement was seen but there was still some residual deformity.

The use of streptomycin *completely alters for the first time in the history of tuberculosis* the dread outlook of "*phthisical persons who develop diarrhoea*."

T.N.S.R.

Roentgen rays in treatment of infections.—(*Radiology*, Syracuse, N.Y., 51: Sep. 1948).

Kelly *et al.* consider that adequate dosage of roentgen ray, living tissues, and a suitable time factor are the three essential things to obtain satisfactory results and beneficial effects in treating infections by radiological means. Amputation of an arm or leg is unnecessary for the successful treatment of gas-gangrene if X-ray treatment is promptly given. Removal of tissues during the acute toxic phase of the disease is also unnecessary. Due to the latent period of the effect of the X-rays, from 18 to 24 hours treatment should be started early. Antibiotics will not, in their opinion, replace X-rays in the treatment and prevention of acute infections, but they may be also used simultaneously with no ill effects, and perhaps with some amount of benefit. *Sulphanilamide* drugs should *not be given* in conjunction with X-ray treatment. "No person should be allowed to die of bacterial toxemia without the benefit of irradiation treatment" is the conclusion of the authors.

T.N.S.R.

Sensitivity of mercurial diuretics.—Gottlieb shows that mercurial diuretics may act as allergens, giving rise to a variety of clinical manifestations and even fatalities. These potent drugs may also produce untoward reactions by any one of several other mechanisms, of which may be mentioned primary toxicity of the mercury, especially on the heart, disturbances of plasma volume and fluid balance, electrolyte imbalance, "speed shock," overdigitalization and renal complications. Hence, it is incumbent on the physicians to study each such case as carefully as possible in an

effort to determine the cause. From the allergic standpoint, patch-abrasion and patch tests would appear to be the most informative. Fortunately, the over-all incidence of untoward reactions to these valuable drugs appears to be low. A case is reported in which generalized urticaria followed the fifth and sixth injections of mercuriophylline. The causation of the eruption was confirmed by a positive patch-abrasion (scratch-patch test). The minute amount of the drug absorbed from the test site evoked a focal reaction in the form of further urticaria. It is recommended that the scratch-patch test be more extensively employed in suspected drug allergy.

—J.A.M.A.

Sensitization to petrolatum in ointment bases.—Levin and Moss direct attention to the fact that many of the patients with chronic dermatitis presenting themselves for allergic study have been under treatment for months or years. These patients have had many ointments prescribed, the base of which almost invariably contain petrolatum. Sensitivity may be acquired more readily through the broken than through the normal skin. The conditions for the acquisition of secondary sensitivities in cases of dermatitis are ideal; *i.e.*, the repeated and frequent application of a substance to the broken skin over a long period of time. It is not surprising, therefore, that under these conditions sensitivity to medicaments is frequently encountered. Six cases of definite sensitivity to petrolatum or related substances are reported. Patch tests on normal skin for purified petrolatum rarely elicit positive reactions. Usage tests are a valuable means of making a diagnosis of petrolatum sensitivity. The results may be positive on affected sites and later negative on the same healed sites. In view of the widespread use of ointment bases containing petrolatum and/or mineral oil for the local treatment of the allergic dermatoses, the possibility of sensitization to these items should be kept in mind. Recently the authors have encountered 4 additional cases of definite sensitivity to petrolatum. These patients were all negative to patch tests and positive to the usage tests carried

out on the affected sites. *J.A.M.A.*, March 1949.

Streptomycin in granuloma inguinale.—Chen and his co-workers treated 100 patients, 65 men and 35 women, with laboratory proved granuloma inguinale, by intramuscular administration of streptomycin dissolved in distilled water containing 20 to 30 per cent of a 1 per cent solution of procaine hydrochloride. Sixty-seven of the patients had been treated before with some forms of antimonial preparations, but had not been benefited permanently or at all. The remaining 33 patients had not received any specific medication prior to admission. All the patients were arbitrarily divided into three groups regardless of their duration of disease and previous medication. Thirty-two patients were treated six to sixty-two days with a daily dose of 0.3 to 2.0 Gm. of streptomycin. Sixty-one patients were treated with a uniform dosage schedule of 4 Gm. per day for five days. The remaining 7 patients, who had extensive lesions, were treated with 4 Gm. per day for six to ten days. In all cases the antibiotic was administered in divided doses every four hours. Results showed that streptomycin so far constitutes the best treatment for granuloma inguinale. It is effective even in small dosage, though recurrences are more frequent and there is danger of creating streptomycin resistance, as in 2 of the authors' cases. The optimum daily dosage probably is 4 Gm. in divided doses. For average cases of granuloma inguinale a five day course is generally sufficient to effect a cure with disappearance of Donovan bodies and complete healing of the lesions with depigmented cicatrization in one to two weeks. For patients with extensive lesions or in whom clinical and microscopic responses are not so prompt, a ten day course is probably to be preferred. *J.A.M.A.*, March 1949.

Laboratory diagnosis of chancroid disease.—Kuhns and his co-workers show that hitherto the diagnosis of chancroid was usually made by a process of exclusion, which consisted of repeated dark field examinations for spirochetes. If results were negative, diagnosis of

chancroid or lymphogranuloma venereum was made, based on clinical manifestation. Chancroid disease was a considerable problem in the occupational troops in the Philippines. A study of contacts reported by Trussell of the Medical Laboratory in Manila indicated that many of the women demonstrated chancroid lesions which were negative for spirochetes on dark field examination; findings on direct smear were typical of *Hemophilus ducreyi*. Other investigators confirmed the role of *H. ducreyi*. Various mediums have been used for the culture of the organisms from chancroid lesions. At the laboratory with which the authors are connected it was found that tissue impressions on glass slides properly made and stained from chancroid ulcers consistently demonstrated morphologically characteristic *H. ducreyi* organisms. Of 40 penile ulcers suspected to be chancroid, 21 were found positive by direct smear; in 34 of the 40 cases cultures were made, and 14 of these were positive for chancroid. It is their experience that the impression smear and culture are reliable. The method of preparation of the lesion for making the smear is essential for success in making a good impression slide and for obtaining cultures of the organisms. The authors confirm the value of the tryptose phosphate agar medium. An apparent mutation form of *H. ducreyi*, hitherto undescribed, has been reported. *J.A.M.A.*

Treatment of cutaneous anthrax with antibiotics.—The favourable results of treatment with antibiotics of cutaneous anthrax was recently published in a paper by Reilly and Beeson. Five cases of cutaneous anthrax are briefly described. Four patients were given daily 300,000 units of penicillin and 6 Gm. of sulfadiazine for five days; the fifth received daily 2 Gm. of streptomycin for five days. No fatalities occurred; improvement was noted within 24 hours, and thereafter progress was rapid. Review of the literature shows that doses of only 100,000 to 200,000 units of penicillin daily for three days have sterilized the lesions of cutaneous anthrax. Longer treatment is, however, considered safer.

Four cases were infected by direct contact with animals, but the wife of one of the cases, who had dressed the anthrax lesions of her husband and who developed an anthrax lesion two days later, is the first case report of transmission of anthrax from one human being to another by direct contact. She had not come in contact with the animal the men had skinned, nor had she come in contact with the instruments used.

Although sulfadiazine was combined with penicillin and both were considered effective in this disease, it was felt that the sulfadiazine could have been dispensed with. A choice between penicillin and streptomycin for therapy would at present favour penicillin for it has more effectively suppressed cultural growth of anthrax, it is more widely distributed, and costs less. However, as streptomycin is studied more in connection with anthrax, it may prove the better drug. It was recommended that a sensitivity test with the two antibiotics when there is doubt, as for instance, when a patient has cutaneous anthrax and septicemia.

The dosage of streptomycin used in the treatment of cutaneous anthrax was in keeping with the normal amount recommended for its proper usage. The patient's toxicity and the malignant appearance of the lesions improved greatly within the first 24 hours, the fever disappeared after 36 hours and axillary lymphadenopathy was barely palpable, the areas being no longer tender, by the sixth day of treatment with streptomycin. The renal lesion slowly improved and was not aggravated. It was thought possible that the dosage (an initial dose of 500 mg. intramuscularly and 250 mg. every three hours for five days) and the duration of its administration could have been decreased. This is the first case report of the successful use of streptomycin for human cutaneous anthrax.

(Reilly, W. A., M.D., Little Rock, Arkansas, and Beeson, C. R., M.D., *Antibiotic Therapy for Cutaneous Anthrax. Archives of Internal Medicine*, 82: 529-533, December 1948.—*Medical News Letter, United States Information Library*).

SURGERY

Sulphonamides in the treatment of war wounds and burns.—(C. L. Fox, Jr. *Smithsonian Institution Report*, 1943.)

When a person is struck by a bullet or shell fragments or his clothes may be set on fire or he is flung off his ship into water covered with burning oil, treatment has to be given for shock and control of bleeding promptly as a first aid measure. Recent research has made a very important contribution in this direction. Patients in shock from burns should not be "kept warm" or have "heat applied" with hot water bottles or hot blankets. Extensive and careful studies have shown that room temperature 70°—75°F is best and that extremes of heat and cold are definitely harmful. After shock has been treated infection should be prevented. Practically all burns are potentially infected; bacteria will frequently be present though no infection will develop unless they gain the upper hand.

In the Napoleonic wars only one out of 25 soldiers was killed in battle, but one of every two (50 per cent) died of infections in hospitals. In World War I gunshot wounds of the abdomen resulted in 60 to 80% mortality; at Pearl Harbour the mortality from these cases was less than one per cent and in the Solomon Islands less than 3 per cent. Medical officers are now agreed that the Sulphonamide drugs were in a large measure responsible for this wonderful result. How do these drugs accomplish the miracles has been and is being intensively and extensively studied by medical research workers all over the world. It has been found that bacteria are not killed by the drug but their rate of multiplication is retarded and bacteriostasis results. This bacteriostatic effect does not, however, begin immediately but only after several hours, during which the bacteria in the drug environment grew just as well as the control bacteria. This delay in action, probably

represents the time required for conversion of the drug by oxidation to an active principle.

A recent discovery by Wood offers another explanation *viz.*, that para-aminobenzoic acid almost specially nullifies the action of sulphanilamide, one part of P.A.B. can nullify 5000 parts of the drug; other inhibitors of sulphanilamide, are pus, peptone, devitalized tissue and certain bacterial extracts. Careful study has, however, shown that these substances generally improve the growth of bacteria throwing more strain on the drug in grappling with the vigorously growing organisms.

During this time chemists have been synthesizing other sulpha-compounds. Sulphapyridine was the first important improvement and proved effective in the treatment of pneumonia. Sulphathiazole was next synthesized, and proved to be more efficient and less toxic. Sulphadiazine which followed, is as effective as sulphathiazole, still less toxic and better tolerated by most people. These drugs are given by the mouth and distributed in the body through the blood-stream. A localized infection in one part of the body say, a limb or the abdomen, can be treated by placing the drug directly in the wound in contact with the zone of infection. A high concentration of the drug is thus attained in the dangerously infected site. There are certain practical difficulties in this method. The drugs do not dissolve well and tend to "cake", or lump when poured into wounds.

The researches carried out in the laboratories of the College of Physicians and Surgeons at the Columbia University, showed that the order of increasing therapeutic potency—Sulphanilamide, Sulphapyridine, Sulphathiazole, and Sulphadiazine—was a consequence of a single physico-chemical characteristic: *increasing ionization of these weak acids*. The more effective ones were likewise more effective in overcoming the locking (nullifying) effect of PAB. Acidity reduced their activity against bacteria and also reduced the solubility of the drugs.

Bedside observations showed that infected wounds usually became extre-

mely acidic. This acidity interfered with the action of the drugs and also tended to destroy the leucocytes. Unless the leucocytes are preserved from destruction it is not possible to get rid of the Sulpha-treated organisms or eradicate invading ones. Clinical trials were then started using soluble sodium salts of Sulphathiazole and Sulphadiazine and the interference of wound acidity in wound infections was soon controlled. It is important to realize that this is achieved without any tissue damage—such as often occurs when antiseptics such as Iodine are used. The greatest virtue then of the Sulpha drugs is their selective action against bacteria without any damage to the tissue cells.

With the above background before us, it is easy to predict that *burns would be specially suited for local Sulphonamide therapy*.

Burns represent destruction of the skin, (man's natural barrier against infection), and also of the mechanical covering of the underlying tissues. The heat of the burn causes swelling of the tissues and also water logging, with plasma; loss of the skin covering permits the escape of this fluid, aggravating the shock. Tannic acid has been used for years to coagulate the burned skin and produce a hard stiff adherent covering—the Eschar. When neutralised and used on the burns, tannic acid prevents swelling and helps to form a soft pliable covering which prevents loss of the tissue fluids. Pain is immediately alleviated. This neutralised tannic acid works very well with the sodium salts of the sulpha drugs and effectively prevents infection. The mixture is prepared in ointment form, applied over the burns and covered with a light protective bandage. No additional drug by the mouth is necessary, as the soluble sulpha-drug is placed in intimate contact with the zone of potential infection. The dressing ointment is then found to have dissolved covering eschar. Gauze impregnated with vaseline and Sulphathiazole is then placed on the area and allowed to maintain the concentration of the drugs. When the skin is completely destroyed, skin from another part of the body is

grafted on and Sulpha-drug dressing is given to the grafted area. T.N.S.R.

Urethrostomy drainage of the bladder.—(*Proc. Royal Soc. Med.*, 41: 777-780).

Dr. J. G. Sandrey, F.R.C.S., in his very lucid paper presented to the Section of Urology of the Royal Society of Medicine, discusses the methods of bladder drainage in common use, and his personal experience of the drainage of the bladder by urethrostomy, which has been carried out for many years at St. Peter's Hospital in the treatment of urethral stricture. This procedure has been extensively used in the U.S.A., but has not been popular with urologists in Britain. Young and his associates recommended this procedure (in U.S.A.) for diverting urine when operating for hypospadias and other deformities of the penis. The majority of British Plastic Surgeons have since been following his example. Not being satisfied with the routine methods of bladder drainage by cystostomy or urethral catheter, Sandrey explored the possibilities of urethrostomy early in 1946 and has employed it till now in 379 cases both in pre-operative and post-operative drainage. The technique adopted was that described by Barney (of U.S.A.) in 1934 and is referred to as the "closed method." A rubber catheter is passed along the urethra to its full length and the distal end is securely clamped with a curved artery forceps (Moynihan or Wells pattern). The forceps is advanced until its beak reaches the bulbous urethra and by rotating the handle through 180°, the point is made to present in the perineum where it is cut down on.

The end of the catheter is then disengaged from the forceps and the instrument withdrawn through the incision to the correct length for bladder drainage. This method is speedy, simple and requires no special equipment or gadgets. In patients who are very ill, it can be readily performed in bed under local anaesthesia. The "open method" is employed in (about 20% cases) cases where the external meatus is stenosed or the urethra congenitally

small. An incision is made in the perineum, on the curve of a metal bougie dividing the skin, fascia, and bulbocavernosus muscle, the spongy tissue and the urethral mucosa. The edges of the mucosa should be carefully identified and grasped with fine tissue forceps before trying to pass the catheter; otherwise the urethra may invaginate preventing the passage of the instrument. The 'open' method is really much more difficult than the closed, as it requires wider dissection in a highly vascular area. It should *not* be done under local anaesthesia.

The best position for the urethrostomy opening is in the scrotal raphe about an inch in front of the perineo-scrotal angle. This is important as if it is made too far back, it is less accessible for nursing, and anal contamination will occur. If too far forward, the catheter may kink excessively at the subpubic angle and if comes out replacement will be difficult.

For post-operative drainage after prostatectomy, partial cystectomy, etc., a whistle-tip resectoscope catheter with at least three lateral eyes, has been found to be the most satisfactory. The perineal urethra readily accommodates 22 and 24 ch. sizes and where haemorrhage is difficult of control 26 ch. is used.

The best position for nursing the patient is with the tube draining over the thigh and the scrotum supported by an elastoplast bandage. Dr. Sandrey's findings based on his experience with nearly 400 cases, are summarised below:

(1) Suprapubic cystostomy offers no real solution to the relief of bladder obstructions. It is mechanically unsound, being associated with fatal urinary infections. In the treatment of paralysed bladder, however, suprapubic drainage is still the most satisfactory method.

(2) Having regard to traumatic urethritis the indiscriminate use of the tied-in catheter to drain the bladder for over 2 or 3 days is condemned. Less than 10 per cent of the urethra examined tolerated a catheter of 22 ch. or larger size for longer than 7 days. This small group may be suitable for this form of drainage.

(3) Urethrostomy drainage, though not ideal, goes a long way towards solving many of the defects of the other methods. Preoperatively, it is the most efficient, simple, and safe method for bladder drainage. Post-operatively it confers an increased margin of safety on complete closure after operation on the bladder or prostate. Drainage can be maintained indefinitely and so ensure sound healing of the abdominal wound in the most unfavourable conditions.

T.N.S.R.

Hot or cold plaster casts with temperature control.—Charles R. Sullivan, Lieut. (J.G.) (M.C.), U.S.N., (From "U.S. Navy Hospital Corps Quarterly," Feb., 1949).

The time spent wearing a plaster of paris cast—often extending over a period of months—is most uncomfortable. Particularly is this true during the summer months or in the tropics.

Plaster forearm, hand, or foot casts are uncomfortable but not unbearable for the few weeks usually necessary for immobilization of the affected extremity. However, body casts and hip spicas, which cover considerably more body area, interfere to a great extent with the normal cooling mechanism. And a longer period of immobilization is usually necessary.

If by simple means a patient can be made more comfortable during his convalescence, it is worthwhile to devise such a method. For this purpose the advantages of a water-cooled cast are obvious.

The construction of the water-cooled cast entails no more difficulty than one that is not. For example, in a full body cast, the foundation is applied in the usual manner. Then gauze (six-inch) impregnated with plaster is wrapped around the body until four or five thicknesses have been applied to the entire area to be covered. While still wet, this is carefully moulded to the body contours. Now rubber tubing is wrapped loosely (to avoid denting the cast) around the cast, allowing two or three inches of space between the turns.

Holding the tube in place, additional plaster is applied on the basic cast until it is of desired strength. Care must be

taken not to pull the plaster too tight in order not to close the lumen of the tube. At least five inches of the ends of the tube are left exposed at the top and bottom of the cast.

Required lengths of tubing can be attached to the tubing in the cast for intake of supply of water and drainage. The intake end is connected to a cold water tap, or a refrigerated drinking fountain. Ordinarily, tap water is satisfactory. The comfort of the patient is enhanced considerably.

When it is desired to apply heat, as to a fractured femur site, it is a simple matter to use warm water instead of cold. Tubing is applied only over the affected area. A water temperature of 98 deg. to 100 deg. F. is adequate.

Case Report:—G.S.G., aged 21, was admitted to the hospital on July 24, 1948, following an injury to his neck sustained while diving in shallow water. Roentgram of the spine showed a fracture of the body and laminae of the fifth cervical vertebra. Treatment consisted of reduction of the fracture by means of a halter traction over a period of three days.

Then a water-cooled hyperextension body and neck cast was applied. Water was not introduced into the tubing for three days—until the cast was thoroughly dry. During this period, the patient was very uncomfortable owing to the extremely hot weather. He complained of intense pruritis over the trunk and neck. On the third day, the tubing was connected to a refrigerated drinking fountain and the cooling effect was observed in about 30 seconds.

He was much more comfortable and his pruritis subsided. It was found to be unnecessary to continuously run cold water through the tubing of the cast. Stopcocks were secured to the ends of the tubing and the cold water contained within kept the patient cool for several hours.—*General Practitioner*, July 15, 1949.

Verrucae plantaris: a resume.—(Howard K. Schwarzfeld, M.D., Newark, N.J.).

Verrucae plantaris are common warts modified by their location in the thick

skin of the soles. They are horny, translucent or opaque yellowish papules usually set deeply into the skin. The ordinarily inconsequential common wart is transformed into a lesion of immoderate annoyance because of its location and resistance to therapy.

Verrucae are due to a filterable virus. They are infectious as demonstrated by scratching the skin with material from a wart.

Verrucae tend to occur in younger individuals perhaps because of their increased susceptibility to all infections of the skin. Structural deviations of the feet as pes planus, and to a lesser extent, pes cavus, are notoriously more susceptible to plantar verrucae over points of pressure. In structurally normal feet, trauma may play an important role as shown by the increased incidence (about five to one) of plantar warts in the female. These occur on the anterior portion of the foot since (because of high heels) there is four times more pressure on the toes and metatarsal heads.

Histologic examination shows the same microscopic features as a verruca vulgaris but with changes due to pressure. The epidermis is thickened with outstanding increase of horny layers that seem to press in on the epidermis. The prickle cells show perinuclear vacuolation and may contain inclusion bodies.

Plantar warts can be confused with corns and calluses as well as such rarer lesions as arsenical keratosis, congenital keratosis palmaris and plantaris, keratosis blennorrhagica and climacteric hyperkeratosis. The callus is an area of increased horniness of the skin that may be sharply localized or diffuse. The papillary skin lines show no break in continuity. Calluses are often symptomatic and occur over areas of friction and pressure. A corn is a further development of a callus having a central conical horny mass with the base externally and its apex pressing against cutaneous nerve endings. The corn is composed of nucleated horn cells in a laminated arrangement. In the intertriginous areas, corns may be soft, due to maceration. They are caused by pressure and are not related to dermatophytosis.

The average plantar wart if of any duration is usually in the skin rather

than on it. It is often a deep lesion and at its depth it may be 5 to 8 millimeters below the skin surface. Plantar warts are painful and frequently are found in pressure areas as under the metatarsal heads, heels, toes, etc. They are encircled by a zone of callus, separated by a sharp border. The papillary skin lines swing widely around or abruptly end at the margin of the wart.

These verrucae are of three major sub-varieties:

1. The *mosaic* variety is composed of multiple "pressed-together" or agglutinated verruca elements. They are dry horny masses, frequently asymptomatic, and have an irregular border. They often occur under pressure points. The closely packed-together wart elements are revealed by paring.

2. The *mother-daughter* subvariety can occur in any location on the sole and consists of an older central large element with smaller, younger satellites.

3. The *single* type is the common painful variety situated under a pressure point as a metatarsal head, sharply delineated and having a zone of surrounding callus. An extremely painful corn upon paring occasionally shows the capillary tips of a verruca. Such a verruca in a corn is an unusual type and, where found, is on the fifth toe.

The less there is known about a disease, the more the number of treatments recommended. For verrucae, an encyclopedic list of treatments is available and the numerous remedies prove that none is sovereign. Caustic destructive therapy whether by acids, radiation or electro-surgery is the basis for most treatment methods.

X-ray is one of the preferred modes because it is painless and easy; the skin is not broken; and requires no dressings or loss of time. The requisite special equipment and training necessary for this treatment, limit its use to properly qualified therapists. Sequelae to radiation therapy are rare (if the proper precautions are observed) if any wart previously treated by radiation is excluded from further exposure. This, despite the fact that the doctor may know the previously given radiation dose, he does not know the shielding technic observed by the previous operator.

Verrucae are radiosensitive but acquire radio-resistance after successive exposures and therefore Montgomery, A.H., and Montgomery, R.M. use an initial intensive dose to a very closely shielded pared lesion. Each wart is treated separately. They use unfiltered rays of 85 kilovolts. The initial dose is determined by the wart's size, depth, and horny coverings. Children and diabetics receive smaller doses. The initial dose is followed each ten days by a saturation dose that is 80 per cent of the previous dose. Close shielding is important. The usual regime consists of three or four treatments with a cure rate of 90 per cent.

An infrequently used treatment is the injection of sclerosing and other solutions directly into the base of the wart. One to three minims of solution are used for each wart. This is often successful but is painful and dangerous. Cameron cites the severe local and systemic reactions from local injection and warns against them.

Surgical excision is easy, requires a minimum of equipment, and is fairly efficient. The greatest drawback is that recurrences are common. Postoperative pain and disability may be severe drawbacks. Karp by her electro surgical method reports cures in 94 per cent of cases. Under novocaine, a twisted electrode loop is thrust deep into the center of the lesion and rotated 180 degrees. The electrode is removed and only a linear incision remains. There is no bleeding due to vessel coagulation by the current. The crust is removed in about 10 to 14 days and healing is complete in another two weeks. The twisted electrode loop is very important for the usual plantar verruca is deeply situated and the loop must be plunged to the depth of the wart to be effective and any superficial treatment will certainly fail.

Blank with his "phenol and nitric acid method" uses 60 per cent salicylic acid ointment, close paring, and a ring of felt. He is enthusiastic about this but in other hands it has been less effective. It is difficult to use because of the numerous reapplications necessary.

The mosaic type of plantar wart is radio-resistant and the acid treatment is the choice. A 40 per cent salicylic acid

plaster is applied after painting with caustic monochloroacetic acid. Or a piece of elastoplast can be applied directly to the lesion after painting with caustic.

Kurtin and Yontef employ the escharotic action of podophyllin. After paring the lesion down until bleeding occurs, they paint the surrounding skin with collodion and then apply podophyllin suspension to the wart concavity. A flamed strip of adhesive tape is applied to seal in the solution and to cause maceration. A week later, the procedure is repeated. After several repetitions a painless hole occurs which heals in cleanly.

Numerous other modes of treatment are advanced by enthusiastic claimants who report effective results. Among these may be mentioned daily formalin soaks, carbon dioxide snow with heavy pressure, psychotherapy, Biberstein's wart vaccine, prolonged elastoplast application, and systemic medication such as Bismuth.

No matter what method is used, the proportion of recurrence will be high unless orthopedic care and correction are given along with treatment directly to the verruca. Women must wear low heeled shoes for most of the day and should reserve high heels for rare important occasions. Pes planus requires proper supports and correction. A metatarsal bar placed one-half inch posterior to the widest part of the shoe and projecting one-fourth of an inch is a good method for relieving pressure from the anterior part of the foot. Close and meticulous attention to details is necessary no matter what form of therapy is used in order to give the patient full and permanent relief.—*Journal of Medical Society of New Jersey*.

Facial palsy.—(Br. Med. Jour., 25-12-1948, p. 1117).

Osborne of Liverpool addressing a meeting of the Liverpool Medical Institution said that whenever a case of facial palsy had to be treated, whatever the cause of the paralysis was, if signs of recovery were not manifest in 4 months, support to the paralysed side should be provided by means of fascia lata slings. The view that plastic

surgery was only of value when the paralysis was deemed permanent was incorrect. Nerve-grafting and other procedures were spoilt by the stretching of muscles which occurred during the

long interval (two years possibly) before return of function. Whatever operation was contemplated on the nerve itself, the early provision of fascia lata sling would do nothing but good. T.N.S.R.

OBSTETRICS and GYNÆCOLOGY

Effect of malaria on pregnancy.—(*Jour. Canadian Med. Assocn.*, 57: 4, 332-337).

Dr. Kulesar of the Ravelin Provincial Hospital studied the effect of malaria on the course of pregnancy in 360 cases admitted into the hospital during one full year. Kwangsi is perhaps the worst malarial district in China, where nearly 50 per cent of the population suffer from malaria. Of the 360 cases 59 (17 per cent) showed a positive blood smear on examination while 173 gave a definite past history of malaria on admission. 8 patients were treated on clinical signs alone. Of 59 who were positive on blood examination, 19 had premature labour. 9 had threatened or incomplete abortion, i.e., 48 per cent of abnormal cases compared with 18 per cent of normals in the malaria free group (120 cases).

A past history of malaria has no adverse effect on the course of pregnancy except as regards the possibility of a recurrence. Manson-Bahr considers that malaria *per se* is a far more potent oxytocic than any drug. He however recommends care in giving Quinine as large doses may induce miscarriage. Acton however differs from this view.

Mepacrine was found to be well tolerated by pregnant women in relatively large doses and is replacing quinine in pregnancy. Dr. Kulscar treated 30 cases with quinine and 20 with mepacrine. 50% of cases treated with mepacrine were controlled after one febrile attack but only 22 per cent of those treated with quinine were so controlled. No untoward results were experienced with either drug. It is however considered that paludrine will be the drug of choice in the near future. T.N.S.R.

Vaginal hysterectomy in treatment of prolapse.—Palmer. A.C., *Proc. Roy. Soc. Med.*, Oct. 1948. So long ago as 1920, it was considered

desirable that some forms of prolapse may require the removal of the uterus, in the attempt to build the pelvic diaphragm. It was, however, not adopted as a standard procedure and even today experienced obstetricians and gynaecologists are not agreed on the best method of obtaining abiding results. Dr. Palmer, in a discussion on the subject before the Royal Society, furnishes an answer to the question, "What is the position of hysterectomy in the surgical treatment of prolapse?", in three words (1) Never, (2) sometimes, (3) always. *Never* applies when the prolapse is confined to the walls of the vagina: *sometimes* when the cervix is in the first stage of procidentia, i.e., when it has fallen to round about the vulval level and *always* when the cervix is outside the vulva.

The variations in treatment depend on the condition of the utero-sacral ligament. If the cervix has reached the vulva by stretching and *without damage* to the above ligament, hysterectomy is contraindicated. If, on the other hand, the cervix has brought the ligament with it to the vulva, so that the index finger can easily be looked round the ligament, then hysterectomy is indicated. When the cervix is well outside the vulva, the ligament would almost certainly have been brought down with it and hysterectomy must be performed. Removal of the uterus means that the utero-sacral ligaments are in fact shortened and when approximated in the midline stabilize the vault of the vagina. In his hands, infolding the ligaments below and in front of the cervical stump does *not* stabilize the vault of the vagina and too many cases have come back in a year or two with the vagina again everted. Hysterectomy was very successful and only three out of 300 cases have returned with an enterocele and one with a cystocele and these 4 were due to errors of judgment. T.N.S.R.

Vaginal hysterectomy in treatment of prolapse. (Hawkins, J., *Proc. Roy. Soc. Med.*, 676-77).

Dr. Hawkins who followed Dr. Palmer stated that in removing the uterus at or near the menopause, one frequently eradicates an organ which may be causing symptoms of excessive bleeding which may harbour fibroids or even a carcinoma. The chief advantage of the operation is that when the uterus has been removed a very efficient fascial shelf can be reconstructed from the total depth of the blood ligament down to the utero-sacral ligament. By sewing the round ligaments together and anchoring them to the triangular ligament, they form under the bladder neck a kind of Millin's sling made of round ligament. It exerts a most beneficial influence upon stress incontinence. This operation is easier to perform than Fothergill's technique.

The main and most powerful criticism levelled in the past against hysterectomy has been its liability to produce a subsequent hernia of Douglas's pouch. In the small series dealt with personally by Hawkins he found one such hernia which was immediately operated on. He has since altered the technique of the operation and pays special attention to the suturing of the uterosacral ligaments across Douglas's pouch. He believes that with improved technique and experience hernia here should cease to occur. He finally entered a strong plea for the bold surgical handling of all women, with prolapse regardless of age.

This is a most distressing and disabling condition. The day of pill and pessary gynaecology has passed, and he has not used rings or Napier's pessaries for over two years. There is no more pathetic spectacle than the woman who has worn a ring for 20 or 30 years with stress incontinence, which a ring never cures, and pessary ulceration and vaginitis as also the possibility of a cancer developing from the ring. The successfully operated prolapse patient is a grateful and rejuvenated person. T.N.S.R.

Endometriosis—its surgical significance.—Reviewing the most widely accepted hypotheses concerning the etiology of endometriosis, Kelly and

Schlademan show that no theory explains all the different manifestations of endometriosis.

During 1,991 consecutive abdominal gynaecologic operations at Scott and White Clinic, 179 (8.9 per cent) histologically verified cases of endometriosis were found; and these, together with follow-up study, form the basis for this paper. The average age of these patients was 38 years, the youngest 20, and the oldest 65 years.

Endometriosis presents a rather typical gross appearance, best referred to as "puckered" areas, especially in the advanced stages. Early manifestations are likewise characteristic in their "blueberry spot" form. When these cysts perforate or are ruptured, a black, tarry, irritating fluid may be scattered over the contiguous organs. Just as the cysts react to ovarian stimulation at the menstrual phases much the same as normally situated endometrium, so at the menopause, whether natural or artificial, endometrial lesions will recede.

Adhesions due to endometriosis are frequent and when once seen should rarely be confused with those caused by other pelvic inflammatory disease. The adhesions of the latter are generally broad and labile and easily torn while those of the former are tough and solid with a tendency to strand formation. Fixed retroversion of the uterus should always cause the surgeon to suspect endometriosis.

Endometriosis, in spite of its prevalence and importance, has a low diagnostic index. In this series the diagnosis was correctly made in 14.5 per cent. The clinical diagnosis is difficult because of the variable history, symptoms, and physical findings. There is one symptom, however, toward which particular attention was directed in this review. This is the factor of increasing or progressive dysmenorrhea. With a complaint of painful periods it should be determined whether or not the dysmenorrhea has been present since menarche or has been acquired following normal menstrual cycles. In most cases of endometriosis, the latter situation will have prevailed. If the patient further states that her dysmenorrhea, acquired or not, has progressively

become more intense with each succeeding cycle, the diagnosis rests with endometriosis.

Thirty-two of the 179 patients had lesions of the rectosigmoid, but only one-half of these gave a history of gastro-intestinal disturbance. One of these, however, had a partial intestinal obstruction while the remainder complained of painful defecation, abdominal distention or flatulence immediately prior to or during the menstrual period.

The peritoneal surface of the uterus, the wall of the uterus and the cul-de-sac were the most frequent sites of endometriosis.

The extent and location of lesions and the age of the patient are the most decisive factors in the choice of the operation. Although most surgeons are in agreement that it is worthwhile to spend 30 minutes or more excising individual lesions rather than perform more radical procedures, the authors say they had to do radical procedures in 132 of their patients (hysterectomy in 130 and

bilateral oophorectomy in two). Thirty-eight of the women could be regarded as relatively fertile after surgical treatment, that is, pregnancy was both theoretically and practically possible. The average age of this group of patients was 31 years. It has been possible to follow these patients a minimum of two years and a maximum of six years. Though a longer follow-up interval would be desirable, the results during this time serve to re-emphasize the value of conservative surgery whenever possible. Eleven pregnancies have occurred in these 38 patients and all occurred within four years following surgery. Two patients have been pregnant twice during this time, neither of whom had previously been pregnant.

(Kelly, F. J., Amarillo, Texas, and Schlademan, K. R., *Surgery, Gynaecology and Obstetrics*, 88: 230-236, February, 1949. The authors are connected with the Departments of Surgery and Pathology, Scott and White Clinic, Temple, Texas.)—*Surgical News Letter—United States Information Library.*

DERMATOLOGY

Vitamin B₂ (Riboflavin) in infantile eczema and Besnier's prurigo.—(*Ann. Derm. Syph.*, 8: March, 1948). Five cases of infantile eczema, and 21 cases prurigo were treated with riboflavin. Infants of the ages 6 to 12 months, with eczema, received intramuscular injections of riboflavin (½ to one ampoule) every other day for 10 days. Others (ages 3 to 30 years) with prurigo had daily intravenous injections

of 2 ampoules of riboflavin or one ampoule of lactoflavine for 10 days. A second course was given, if required, after a month's rest.

Results in infants were more satisfactory in acute than in sub-acute eczema, but rapid disappearance of itching was a feature.

In prurigo, the results were not as good as in eczema. T.N.S.R.

Book Reviews

Epilepsy and Convulsive Disorders in Children.—By EDWARD M. BRIDGE, M.D., Research Professor of Pediatrics, School of Medicine, University of Buffalo; Director of Medicine, Children's Hospital of Buffalo; Research, Children's Hospital of Buffalo; Formerly Associate in Pediatrics, John Hopkins School of Medicine, and Physician in-charge, John Hopkins Epilepsy Clinic. First Edition, McGraw-Hill Book Company, Inc., New York, Toronto, London, 1949. [Price: \$ 8.50.]

Few diseases of childhood plague the physician more than epilepsy and convulsions. The author of this book is a distinguished member of that group of highly trained specialists who devote themselves entirely to the problems of infants and childhood. Dr. Bridge has had an unusual wealth

of source material on which to base this book, the major part of which comes from experiences accumulated in the epilepsy clinic at John Hopkins over a period of sixteen years. What he observed in the laboratory and in the clinic he has presented here in a remarkable volume which will be useful to all in the profession. The traditional outline has not been followed in the presentation. Emphasis has been placed first on the underlying forces that give rise to the symptoms. Later sections are devoted to the various manifestations of epilepsy. Pathology as such is not considered but is included in the chapter on brain injury, which also discusses the functional disturbances initiated by organic disease. Treatment is scattered throughout the presentation,

in particular as part of the discussion of the various manifestations. A pictorial table of contents has been prepared for each chapter. The book maintains practical usefulness in addition to its scholarly discussion of the physical and emotional background of epilepsy. It will fill a great need among the practitioners and will extend the benefits of modern treatment to many epileptic children.

S.R.

Handbook of Diseases of the Skin—By RICHARD L. SUTTON, M.D. and RICHARD L. SUTTON JR., M.D., with 1057 illustrations. St. Louis, The C. V. Mosby Company, 1949.

A book useful to medical students, practitioners, and specialists in dermatology. This is well-written, with a clean, concise presentation of the subject matter. The first few chapters cover the basic knowledge of the skin and its appendages with anatomy, embryology, physiology and pathology. Dermatoses are classified and discussed from an etiological standpoint. Important contributions to current dermatologic literature are made and are well documented by numerous bibliographic references. There are 1057 illustrations and these have been carefully selected, trimmed, placed and titled, their legends in appropriate instances. Smaller types are used to separate the more detailed material of less interest to the non-specialist from the text in general. Personal views on subjects, some controversial, are frequently expressed.

S.R.

Handbook of Materia Medica, Toxicology, and Pharmacology—By FORREST RAMON DAVISON, B.A., M.Sc., Ph.D., M.B., Consultant and Toxicologist, Minneapolis, Minnesota; Formerly Assistant Professor of Pharmacology in the School of Medicine, University of Arkansas, Little Rock; Medical Department, The Upjohn & Co., Kalamazoo, Mich.; Assistant Professor of

Pharmacology, University of Tennessee Medical School; Toxicologist to University Clinics, Memphis, Tennessee; Fourth Edition, 35 illustrations, including four in colour. St. Louis, The C. V. Mosby Company, 1949.

The aim of this book is to present the information about drugs, essential for the student of medicine and the practising physician. Guided chiefly by the needs of the medical student the author has presented a classification of drugs which should help to simplify, for the student, the rapidly accumulating mass of information concerning drugs found in medical literature. The site of action and the therapeutic activity of the various drugs have been used in the development of the classification presented. Prescription writing is given a place of prominence. Intelligent drug therapy is closely related to the writing of good prescription. Significant new remedies are discussed in this book. New information on hormones and also on vitamins is given. The whole book is revised.

S.R.

Emergencies in General Practice—By J. R. GOYAL, M.B., B.S., Printed by BAL KRISHNA, M.A., at the Yugantar Press, Mori Gate, Delhi. First Edition, 1949. 352 pages. Price Rs. 10-0.

The general practitioners in our country have a very difficult task to perform. They are often called in to see cases of very serious and diverse nature and at all hours in the day and night. They should therefore be familiar with all emergencies that can occur in the course of their practice. This is especially so in the case of general practitioners in the smaller towns and in the villages where specialists are not available. Dr. Goyal in his little book has made an attempt to present all these emergencies in a brief and concise manner so that the beginner and also the general practitioner may be benefited by the information that is given. It is a book which should be useful to all.

—P.K.R.

News and Notes

United Nations Educational, Scientific and Cultural Organization proposes to publish later in the year a Manual on the International Exchange of Publications.

It is intended to publish as an annex to this manual a classified list of institutions, including libraries, universities, scientific institutions, learned societies, etc. throughout the world, which are willing to exchange either their own publications or other publications which they have regularly at their disposal. In the course of its activities Unesco has been able to obtain a considerable amount of information concerning the availability of exchange material, but we feel that the information at our disposal is still far from complete. All institutions which have so far not sent to Unesco details of their exchange material in one form or another are therefore urged immediately to communicate the following information to the Unesco Clearing House for Publications, 19 Avenue Kleber, Paris, 16e:

(a) Name and full address of institution;
(b) Exact titles of publications offered. (In the case of duplicates offered for exchange purposes actual lists of duplicates are not required, but only a statement that lists of duplicates are available. Institutions possessing a catalogue of their own publications available for exchange are asked only to send a copy of the catalogue or to give a full bibliographical description of the catalogue.); (c) Institutions which wish to exchange their publications only under conditions, are asked to state what these conditions are.

Only information which reaches Unesco before October 1st, 1949 can be used in the Manual and it is therefore in the interest of all institutions concerned to communicate immediately with the above address. Informations not received before 1-10-49 will be published in the supplement which will appear next year.

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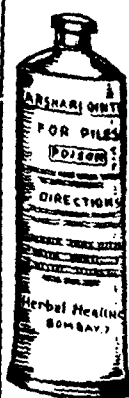
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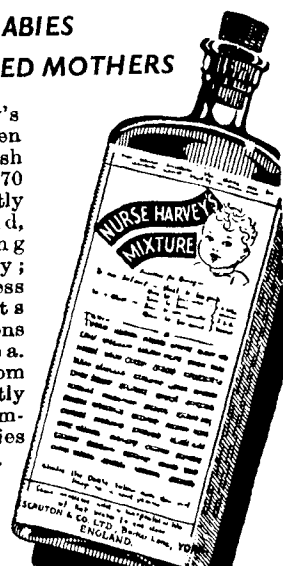
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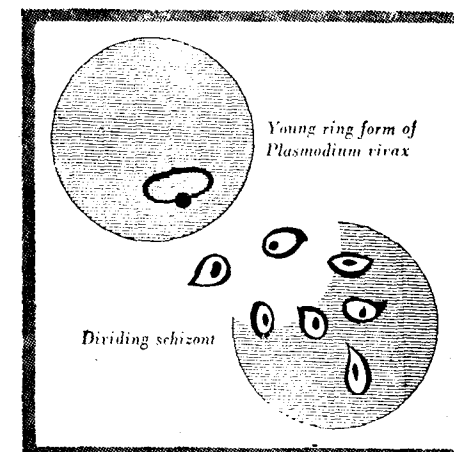
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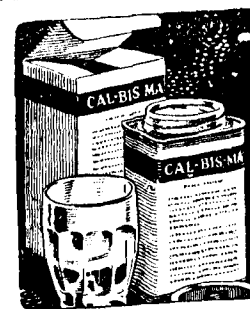


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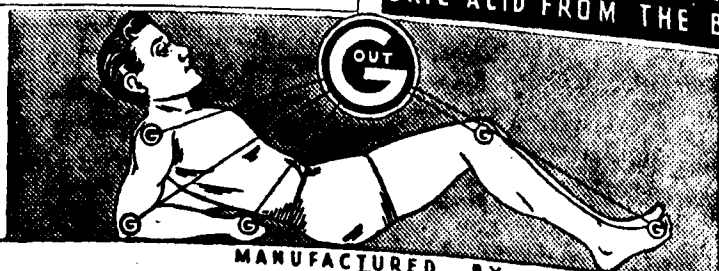
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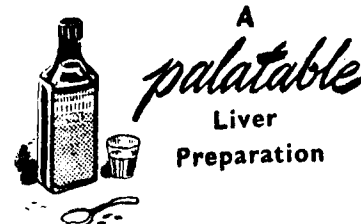
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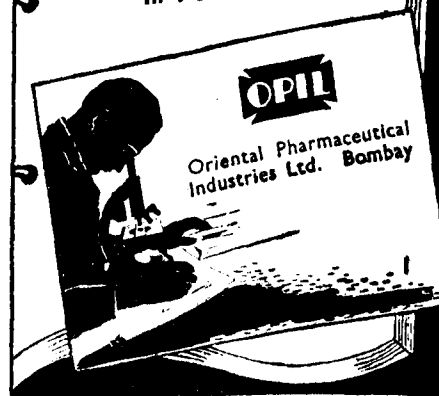
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Thiamine hydrochloride (Vitamin B ₁)	... 2.5 mgm.
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Ascorbic Acid (Vitamin C)	... 10 mgm.
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The preparation has been preserved in a most suitable pH to ensure maximum activity and stability of all the above ingredients.

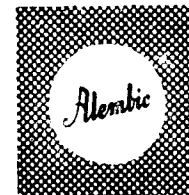
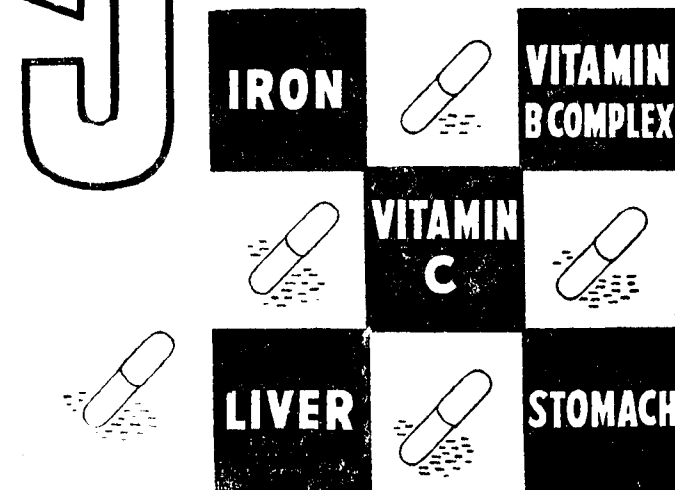
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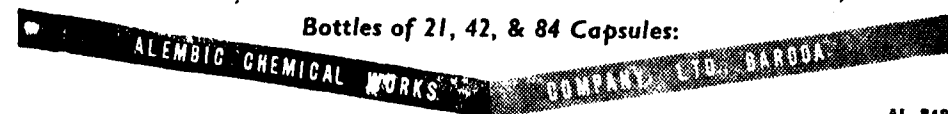
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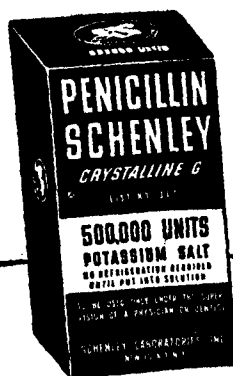
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100,000 Units	in 20 c.c. rubber-capped vials.
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Vitamin D	3,000 I.U.	Calcium Pantothenate	20 mgm.	Ferrous Chloride	50 mgm.
Vitamin B ₁	25 mgm.	Vitamin C	100 mgm.	Manganese Glycero-phosphate	10 mgm.
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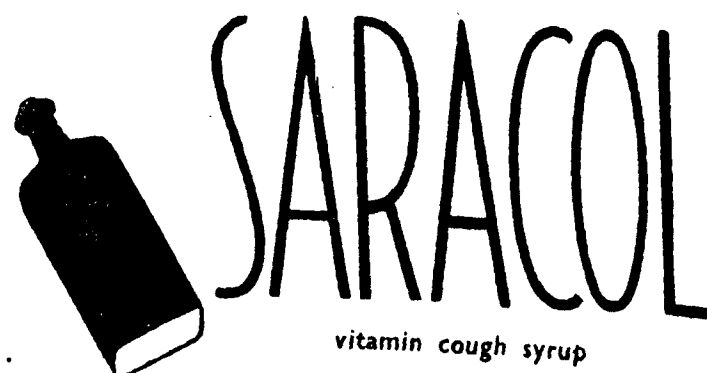
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Chloroform	.. 3.0	min.
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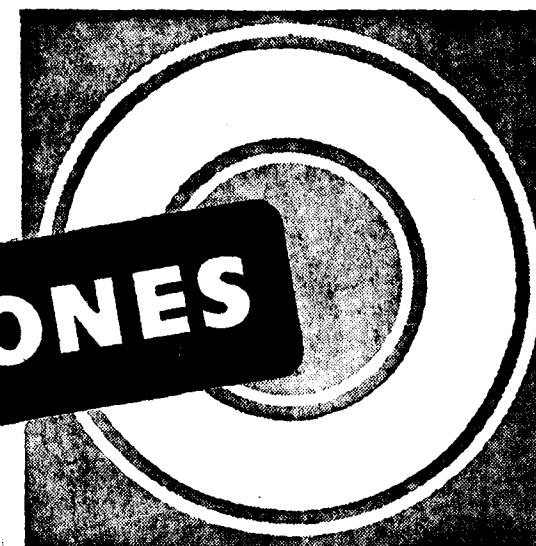
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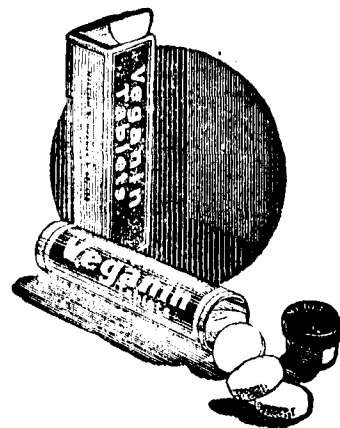
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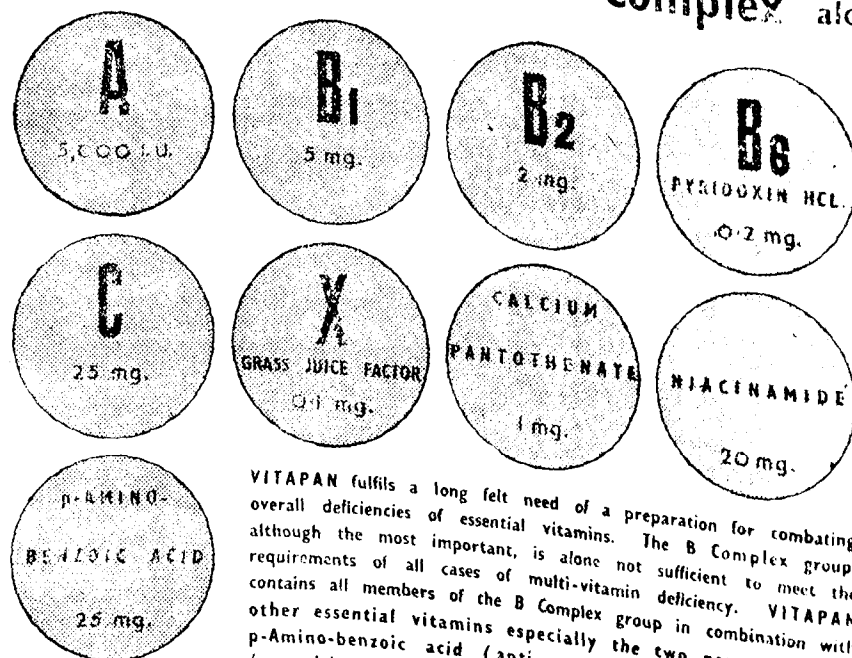
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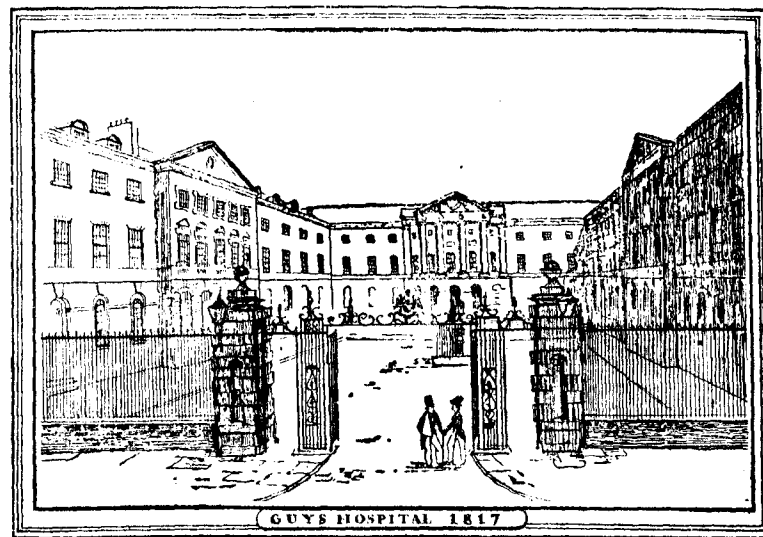
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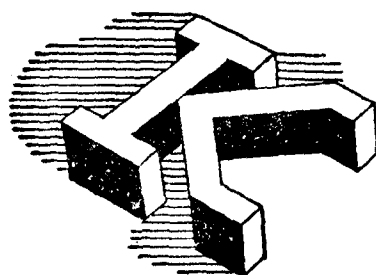
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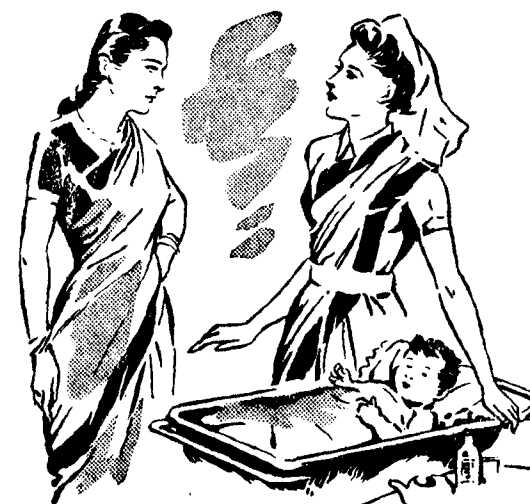
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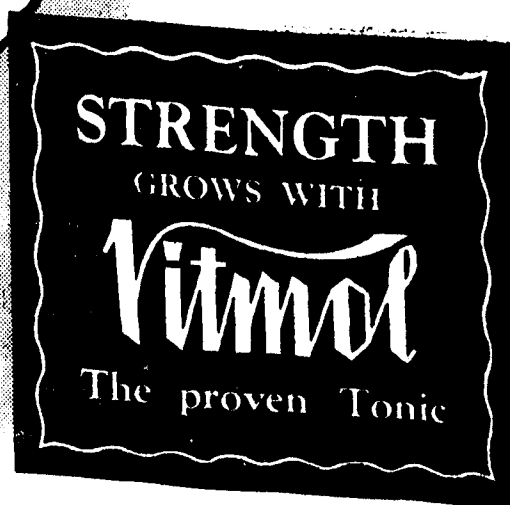
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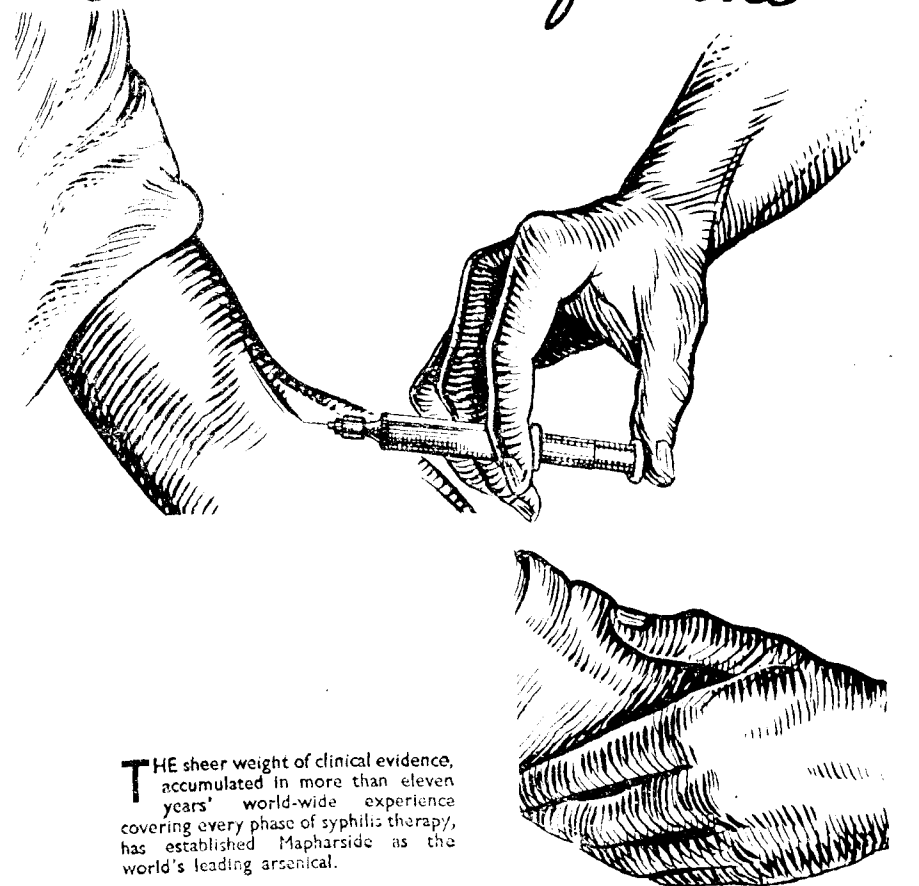


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