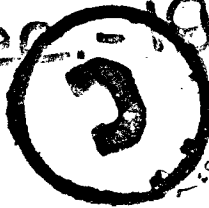


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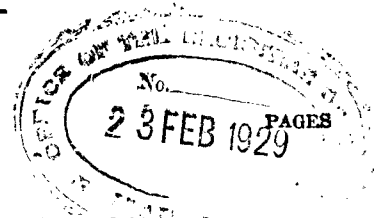
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EDITOR

V. R. GORAKSHAKAR, B. A., M. B., Ch. B., (Edin.)
 OSMANIA GENERAL HOSPITAL,
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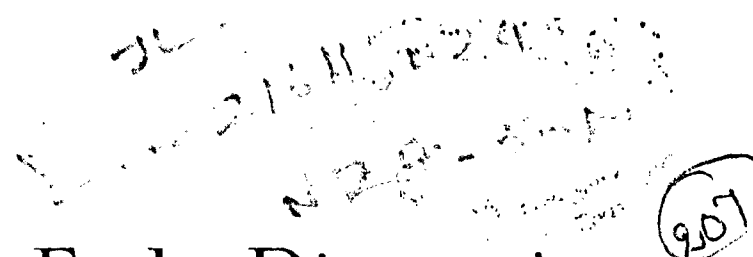
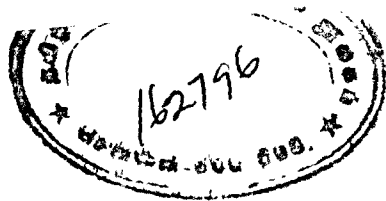
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Early Diagnosis of Leprosy

BY

DR. LOWE

in charge Leprosy Hospital, Dichpalli.

WITH modern developments of leprosy work, increased knowledge has been gained concerning the early stages of leprosy and we have now satisfactory treatment for these early cases. Leprosy diagnosis and treatment in the future will not be the work of a few specialists, it will be a part of the ordinary day's work of the ordinary doctor whether in Government service or in private practice. The leprosy problem cannot be tackled by specialists only, it must be tackled by the whole medical profession. Doctors will find leprosy work well worth while if they have the necessary knowledge. This knowledge is not difficult to acquire. There are books and pamphlets on the subject, the best book at present available being "Technical Lectures on Leprosy" prepared by Dr. Muir's department and published by the B. E. L. R. A. There are short courses of instruction in leprosy at the School of Tropical Medicine, Calcutta and also at the Leprosy Hospital, Dichpali.



A Ward in the Osmania General Hospital.

The success of a doctor's work in treatment of leprosy will depend very largely on his ability to diagnose really early cases of leprosy. Treating long standing cases who are often crippled and deformed and also often noninfective, is a waste of time and energy. We must concentrate on early cases. With this in mind I think we might profitably spend a little time discussing the early diagnosis of leprosy.

I wish to make quite clear at the start one important point. There is an idea which is commonly held that a diagnosis of leprosy can only be made by finding the lepra bacillus. This is not true in leprosy any more than it is true in tuberculosis. In many early stages of tuberculosis of the lungs no tubercle bacilli can be found in the sputum. If one waits until tubercle bacilli are found before a diagnosis is made then many early cases will be missed and the chances of recovery will be very much less. Exactly the same in leprosy. In the earliest stages no lepra bacilli can be found but there are other signs which make diagnosis possible. If one does not diagnose leprosy without finding the leprosy bacillus, then one will miss 50% of the early cases, and the patient's chances of recovery will be much diminished.

(a) *Latent period.* In some way which we cannot discuss now the lepra bacilli enter the body of a healthy person. For some time, often for several years, the disease lies latent this being the latent period before the onset of definitely leprosy symptoms. Towards the end of this latent period there may appear certain symptoms which while not diagnostic of leprosy are suggestive of the disease.

(i) A dry rhinitis with a scab formation and occasional cracks in the mucous membrane and slight bleeding especially in dry hot weather. Examination of the nasal mucous membrane will show no ulcers and no lepra bacilli, as are found in the later stages of the disease.

(ii) Abnormal sensations in the distribution of certain nerves. The nerves most commonly affected are the ulnar and superficial peroneal nerves. Occasionally the radial nerve may be affected. The abnormal sensations take the form of tingling and partial loss of sensation. Patients often say they feel as though ants were crawling over their limbs. These sensations often last for a few seconds or minutes only. What frequently happens is that the patient wakes up in the night and finds his hands and feet tingling and partly numbed. This passes off in a short time, he goes to sleep and the same thing may be repeated later on. Later these sensations sometimes come on during the day also, especially after severe exercise. Sometimes the sensation amounts to real pain, neuritis, and this is usually relieved by pressure. I have often known patients tie a handkerchief tight round the arm at night to relieve the neuritis. A patient told me only the other day that he slept in same bed as his brother and he put his arm under his brother's head so that the pressure would relieve the pain.

Examination of the patient will often show nothing further than these two symptoms neither of which alone is diagnostic of leprosy, but when these two

symptoms appear together in a patient who has lived in contact with lepers we are justified in making a provisional diagnosis of leprosy. The diagnosis can be verified by the administration of Potassium Iodide as described later. Sometimes these symptoms are accompanied by actual thickening and inflammation of the nerve and then the diagnosis is fairly certain.

(b) *A1 stage.* (early nerve leprosy.)

As the disease develops other more definite signs of nerve leprosy may appear in addition to the two symptoms already described.

(i) Patches of superficial anaesthesia (tested for by light touch test.).

(ii) Thickening of nerves especially common in ulnar nerves, superficial peroneal and great auricular nerves.

(iii) Depigmentation of patches of skin which may or may not be anaesthetic.

(iv) Anhydrosis of effected areas of skin.

Often all these signs are found and then the diagnosis is obvious. If only one or two are found are we justified in diagnosing leprosy? Superficial anaesthesia alone is diagnostic of leprosy in the absence of any other possible cause. Other causes of anaesthesia are very rare and anaesthesia alone means leprosy in practically every case. Thickening of nerves is also diagnostic of leprosy even if no other signs are found. Depigmented patches of skin alone are not diagnostic of leprosy. There are many other causes of this besides leprosy. If the patches are

typical in appearance and distributions and if there is a history of living in contact with lepers, a diagnosis is sometimes justifiable. Anhydrosis of skin is not diagnostic of leprosy in the absence of other signs of leprosy. Here again in any doubtful case the Potassium Iodide test for leprosy should be used.

(c) *B1 Stage.* *Early skin leprosy.*

Here the disease has developed and produced slight skin lesions in which lepra bacilli can be found. The symptoms already described may or may not be present. There are two ideas commonly held which are misleading and need to be mentioned here. The first is the idea that skin leprosy is a disease which is marked by the presence of nodules. It is quite true that in the later stages especially nodules are common but by far the commonest form of skin leprosy we see is the slight diffuse thickening of the skin with no nodule formation whatever. The second idea is that in skin leprosy the easiest and commonest place to find the lepra bacilli is in the nasal discharge. Here again in the more advanced cases it commonly is so, but a far commonest place to find lepra bacilli is the skin at the site of the thickenings and the commonest place of all to find them is in the lobe of the ear even if there is no apparent thickening. In our experience the examination of the nasal discharge is of comparatively little value in the diagnosis of leprosy. I have never known a case with lepra bacilli in the nasal discharge and none in the ear lobe. I have seen hundreds of cases with lepra bacilli in the ear lobe and none in the nasal discharge. Therefore

in diagnosis of leprosy ignore the nasal discharge and examine the ear lobe.

In the early stages of skin leprosy the commonest lesions are slight thickening of the skin usually over prominences and exposed parts and extensor surfaces. It is perhaps most commonly seen on the face and ears but is also common on the back, shoulders, elbows or buttocks, also on the chest and rarely on the abdomen. This thickening will not be found anaesthetic although adjacent areas of skin may be anaesthetic. It is important that this should be realised. Thickened areas of skin usually contain lepra bacilli but they are not anaesthetic. Anaesthetic areas of skin rarely contain lepra bacilli. For diagnosis clip off with sharp scissors a small piece of thickened skin, squeeze out thoroughly on the slide, fix by heat, stain, and examine for lepra bacilli.

Diagnosis in the later stages of leprosy is easy and we need not discuss it here.

ROUTINE EXAMINATION IN ALL CASES OF SUSPECTED LEPROSY.

1. Take careful history; enquire about contact with lepers. Enquire about dry rhinitis, formication and neuritis. Strip the patient and examine in good light. Look for depigmented patches and thickenings of skin. Examine for thickening of skin. Examine for thickening of nerves especially ulnar nerve, superficial peroneal nerve and great auricular nerve. Examine the whole body for any areas of diminished sensation. Take clipping from

ear lobe even if there is no apparent thickening. Take clipping from any other thickened areas of skin and examine for lepra bacilli.

If this is done ninety nine out of every hundred cases of early leprosy can be diagnosed.

VERIFICATION OF DIAGNOSIS IN DOUBTFUL CASES.

There remains a very small class of patients where the diagnosis is uncertain. There may be slight evidence of leprosy such as depigmentation but not sufficient evidence to make a diagnosis. In many of these cases the administration of Potassium Iodide will clear up the diagnosis. Potassium Iodide is given on the first day ten grains increasing the dose every day by ten grains until sixty grains is reached, then increasing the dose by twenty grains to 120 grains and then by forty to 240 grains. In this way a dose of 240 grains can be reached in two weeks. If any leprosy infection is present, before the maximum dose is reached it will be made apparent by the occurrence of leprosy reaction. This may be shown by one or more of the following signs. Fever, swelling and tenderness of nerve trunks and neuritis, the appearance of reactionary skin lesions which usually take the form of small circular thickenings of the skin or subcutaneous tissue. If in the skin, the reactionary lesions are red in colour. These signs of reaction often disappear in 24 hours so the patient must be examined carefully every day. The administration of Potassium Iodide will thus corroborate in

many cases the provisional diagnosis of leprosy made on evidence which alone is not conclusive.

If a patient is suspected of having leprosy and can take 240 grains of Potassium Iodide without any signs of reaction appearing then this is strong evidence that there is no leprosy infection present. But this is not conclusive and there is a further refinement of the Potassium Iodide test for leprosy namely the Potassium Iodide Sedimentation test advocated by Dr. Muir. I need not describe this test here. Nearly all cases of leprosy can be diagnosed by the means already described.

Leprosy is often compared with tuberculosis. Leprosy is certainly easier to diagnose, it is certainly easier to treat, and in most early cases the prognosis is good, far better than in tubercular infection. The medical profession as a whole will find that if they have the necessary knowledge leprosy diagnosis and treatment will provide a sphere of really fruitful service. We hope that in Hyderabad more and more medical men will take up this work and I can assure you that if the Dichpali staff can help in any way we shall do it gladly.

. . .

Development of the Vascular System

PART II

BY

H. HYDERALI KHAN, F. R. C. S. E.

Professor of Anatomy

The Prince of Wales Medical College, Patna

THE VEINS

WE have followed the development of the veins as far as the formation of the vitelline and the umbilical veins on the one hand and the precardinal and the postcardinal veins, and the duct of Cuvier on the other. The vitelline veins are spread out on the yolk sac and run along the sides of the vitelline ducts through the septum transversum to the sinus venosus. The septum transversum later on develops into the diaphragm and ventral mesogastrium into which the liver develops. As the liver grows the part of the vitelline veins which

passes through it gets broken up and opens into spaces called sinusoids (Minot.) The vitelline vein is thus divided into a vein leading into the liver, called vena advehens and a vein carrying blood away from the liver called vena revehens. The left vena revehens passes to the right to open into the right vena revehens which forms the upper part of the inferior vena cava.

The vitelline veins as they run on each side of that part of the primitive gut which is going to form the duodenum are connected by three transverse anastomoses. The upper and the lower anastomoses are situated ventrally and the middle one dorsally. Meanwhile a fresh vein called the superior mesenteric vein appears which carries blood from the intestines and joins the left vitelline vein just distal to the dorsal cross anastomosis. The splenic vein which brings in blood from the spleen also opens at the same spot. In this way the portal vein is evolved cephalic to the entry to the superior mesenteric vein, and is formed by the portion of the left vitelline between the superior mesenteric vein and the dorsal anastomosis, the dorsal anastomosis itself and the right vitelline vein cephalic to the dorsal anastomosis. The right branch of the portal vein is formed of the right vena advehens, and the left is formed of the cephalic (ventral) anastomosis and the right vena advehens.

The veins from the placenta fuse into a single trunk called the vena umbilicalis impar which runs through the umbilical cord. As it passes through the umbilical opening into the body, it divides into

a right and a left umbilical vein. The right umbilical vein disappears quite early. The left umbilical vein opens into the sinus venosus, then into the sinusoids of the liver as the latter develops, and lastly into the left vena advehens. Another channel called the ductus venosus springs between the left vena advehens and the right vena revehens. Blood from the placenta therefore passes by the left umbilical vein and the left vena advehens to the inferior vena cava by two routes, one through the liver and the other through the ductus venosus. After birth the left umbilical vein and the ductus venosus shrink and become the ligamentum teres of the liver and the ligamentum venosum respectively.

The precardinal vein which receives blood from the head, and the postcardinal vein from the trunk open by means of a common channel called the duct of Cuvier into the sinus venosus. The postcardinal vein which lies dorsal to the Wolffian body receives the external iliac and the hypogastric veins as well. A second group called the subcardinal veins makes its appearance ventral to the Wolffian body. The postcardinal and the subcardinal veins are joined together by a series of transverse channels; two of these main channels become the renal and the left common iliac veins.

The inferior vena cava is developed from five different parts, 1. The extrahepatic portion, of the right vena revehens, 2. The hepatic portion, by a caudal prolongation of the right vena revehens to the right subcardinal vein, 3. The prerenal portion,

As the cranium and the membranes of the brain develop from the mesoderm round the brain stem the plexus of veins is divided into a superficial portion which drains the surface of the head and a deep portion which drains the brain stem. Three secondary plexuses remain in connection with the dura mater. The channel by which the middle plexus joins the primary head vein undergoes atrophy but later develops into the superior petrosal sinus. A portion of venous plexus between the cavernous sinus and the internal jugular vein develops into the inferior petrosal sinus. The anterior and middle plexuses extend anteriorly to the developing hemisphere where they unite and form the sagittal plexus. A portion of the venous plexus hangs between the growing hemispheres in the region of the falx cerebri and develops into the superior sagittal sinus dorsally and the inferior sagittal sinus and the straight sinus ventrally in the lower free margin of falx cerebri. The superior sagittal sinus when completed opens usually in the right transverse sinus. The backward growth of the hemisphere carry the superior sagittal sinus and the straight sinus with them; these unite at the confluens sinuum. The posterior plexus becomes connected with the confluens sinuum, loses its connection with the transverse sinus and becomes the occipital sinus.

The external jugular vein drains the post auricular region, and is connected with the lingual and facial veins which open into the precardinal vein. Later on the facial veins also open into the external jugular vein. The cephalic vein which pours its

blood into the external jugular vein is later transferred to the axillary vein. The former arrangement occasionally persists and the cephalic vein passing superficial to the clavicle opens into the external jugular vein.

THE FOETAL CIRCULATION.

The foetal heart consists of two atria imperfectly separated from each other and communicating through the foramen ovale, and the two ventricles which are entirely separated from one another. The termination of the arch of the aorta is connected to the pulmonary artery by a channel called the ductus arteriosus. Blood is received into the right atrium from three sources, the superior vena cava, the coronary sinus, and the inferior vena cava. Blood from the superior vena cava is poured into the right auricle together with blood from the coronary sinus and passes into the right ventricle. Blood from the inferior vena cava passes into the right atrium and is directed by the valve of the inferior vena cava into the left atrium through the foramen ovale. From the left atrium the blood passes through the left atrio-ventricular orifice into the left ventricle, thence to the aorta the branches of which distribute the greater portion of the blood to the head and upper limbs, a small quantity of blood may find its way into the descending aorta. After circulating in the head and the upper limbs the blood is brought back by the superior vena cava into the right atrium, whence it passes into the pulmonary artery. Only a small

quantity however goes to the lungs as they are inactive, the greater portion of the blood passes into the arch of the aorta by the ductus arteriosus. A portion of the blood travels to the lower limbs but a large quantity is carried over to the placenta by the umbilical arteries; here exchange of gases takes place and the blood is carried to the foetus by umbilical veins which unite to form the vena umbilicalis impar which enters the body and divides into the two umbilical veins. The right umbilical vein disappears but the left persists till birth and becomes degenerated into the ligament teres after birth. The left umbilical vein pours its blood into the liver and is connected with the inferior vena cava by the ductus venosus. The inferior vena cava therefore receives blood from the liver which in turn is supplied by the portal vein and the left umbilical vein, and from the left umbilical vein directly through the ductus venosus. The blood from the lower extremity and the abdominal wall is also poured into the inferior vena cava.

It will thus be seen that there is an admixture of blood throughout the foetal circulation, first blood from the umbilical vein gets mixed in the inferior vena cava with blood from the inferior extremity, abdominal wall, and the liver; secondly there is a mixture of blood of the superior and the inferior vena cava in the right atrium, thirdly blood passing through the pulmonary veins from the inactive lungs mixes in the left atrium with blood from the right atrium, and fourthly blood passing through the aorta is mixed with blood from the pulmonary artery passing through the ductus arteriosus.

When the child is born it cries and takes a deep breath. Blood rushes from the pulmonary arteries into the expanding lungs. The placental circulation ends and the pulmonary circulation begins. Blood from the superior vena cava and the inferior vena cava enters the right auricle and passes down into the right ventricle which pumps the blood through the pulmonary arteries into the lungs which are now active and where exchange of gases takes place and is returned to the left auricle by the pulmonary veins. The blood next passes into the left ventricle which pumps it into the aorta which distributes it throughout the system. After it has circulated in the system it is returned to the right auricle by the superior and the inferior vena cava. The ductus arteriosus subsequently atrophies into a cord known as the ligamentum arteriosum. The foramen ovale soon closes, the left umbilical vein and the ductus venosus atrophy and become the round ligament of the liver and the ligamentum venosum respectively. The part of the hypogastric artery which extends from the bladder to the umbilicus also atrophies and is known as the lateral umbilical ligament.

Instructions for Surgical Note Taking

BY

S. A. RAHIM, B. A., M. B., D. O.,
*Civil Surgeon, Osmania General Hospital
Hyderabad, Deccan.*

THIS article is written to help students of the Osmania Hospital in methods of approaching surgical cases which will not only give them a good grounding of the elements of surgery but far more important still would make them learn how to handle cases in after life in practice. The writer has freely drawn from the notes by Dr. Handfield Jones of St. Mary's Hospital, Paddington and made alterations to suit local conditions.

General scheme for all cases.

1. *General statement* (inpatient's own words) of the cause of admission e.g. pain in the stomach,

difficulty in passing water, lump in the breast, broken leg etc.

2. *History of present condition.*

- (a) Accurate date of first start of the trouble.
- (b) How the disease started or nature of the accident.
- (c) The first symptom is important.
- (d) Obtain history of symptoms in their exact chronological order.
- (e) Details of previous treatment if any.
- (f) In case of an old case the out-patient or in-patient notes must be obtained.

3. *Past History of*

- (a) Venereal diseases.
- (b) Typhoid or Dysentery.
- (c) Infectious diseases.
- (d) Gout and Rheumatism.
- (e) Tuberculosis.
- (f) In women-menstrual history and past pregnancies.

4. *Family history of*

- (a) Syphilis.
- (b) Tuberculosis.
- (c) Cancer.
- (d) Congenital Deformities.
- (e) Haemophilia.

5. *Condition on admission.*

- (a) Describe general condition.
 - (i) Conscious or not.

- (ii) Collapsed, suffering from shock etc.,
- (iii) Note pulse, respirations and temperature.
- (b) Describe injury or disease.
- (c) Note the result of urine tests.
- (d) Copy in Radiologists report in cases of Skiagrams.
- (e) Copy in reports of any special Pathological or Bacteriological tests.

6. *The examination, diagnosis and special remarks by the Surgeon.*

7. *Treatment.*

- (a) If by apparatus note.
 - i. Nature of apparatus—splints, belts, jackets etc.,
 - ii. Nature of extension used (if any) and angle and direction of the pull.
- iii. Whether put up under anaesthesia or not.
- (b) If operative.
 - i. Operation to be described in detail,
 - ii. Nature of anaesthetic,
 - iii. Details of dressings or splints applied.

8. *After progress note.*

- i. Daily temperature—pulse—respirations.
- ii. Progress of wound, date of removal of stitches, nature of dressing,
- iii. If a drainage tube has been used, mention when it is shortened, removed and replaced, removed and smaller one substituted or removed altogether and so on,

- iv. Diet after operation and its variations,
- v. Any new symptoms that may occur or grave alteration in patient's condition e. g. pelvic or subphrenic abscesses, secondary haemorrhage etc.,
- vi. If these arise note their treatment as fully as for primary operation.

9. *Result.*

State condition on discharge. If sent out in splints or plaster note details and Surgeon's instructions.

SPECIAL SCHEME NO. 2.

Swellings of the Breast.

i. *Special points in the history,*

- (a) Age of patient,
- (b) Nature of onset,
whether pain or swelling first noticed or discharge from nipple.
- (c) Whether patient has been recently confined or is suckling.

(ii) *Examination.*

Always compare affected with non-affected breast.

(a) *The nipple.*

- 1. If retracted,
- 2. If on higher level than the other,
- 3. If ulcerated,
- 4. If there is a discharge—its nature, blood, serum, milk.

- (b) The areola.
 1. If redness or swelling,
 2. Any eczema or ulceration.
- (c) The Breast itself,
 1. The skin,
 - (a) If freely movable or fixed,
 - (b) If red or ulcerated,
 - (c) If oedematous—like the skin of an orange,
 - (d) Tenderness,
 - (e) Presence of small nodules in it.
 2. The gland substance.
 - (a) If larger or smaller than the other,
 - (b) Feel with flat of palmar surface of fingers for presence of lumps,
 - (c) Feel breast between the finger tips for thickenings or lump.
 3. If a tumour is felt.
 - (a) Is it freely movable within the breast,
 - (b) Is it movable only with the breast,
 - (c) Its characteristics,
 4. Examine mobility of breast.
 - (a) Is it attached to the pectoral muscles or serratus magnus,
 - (b) or to the skin,
 - (c) Lymphatic glands,
 1. The axillary groups of the affected side,
 2. The supra-clavicular group,
 - (d) Examine for secondary deposits,
 1. In the liver,

2. In the lungs,
3. In the vertebrae, sternum, humerus and femur.

SPECIAL SCHEME NO. 2.

Disease of Genito-urinary system.

- i. Special points in the history,
- ii. Nature and mode of onset of symptoms,
2. Pain—Its position and whether it radiates anywhere, if so where, its relation to micturition; if made worse by exercise or jolting.
3. Micturition—If frequency—note times during day or during the night, nature of stream, difficulties in micturition, if at the beginning middle or end of the act.
4. Urine—Has patient noticed anything wrong e. g. much sediment, blood stained—if blood has been noticed, is it well mixed with urine, or if pure blood, does it come at the beginning or end of the act. Have any calculi been passed.

I. *Examination.*

(a) Inspection.

1. Note any swelling in kidney region, lower part of abdomen or in connection with the penis or testicles.

2. Watch patient micturate and note any abnormalities.

(b) *Palpation.*

- (1) The kidneys—examine both sides and note.
 1. If they are palpable.
 2. If so, are they enlarged.
 3. If so, nature and direction of enlargement and mobility in respiration.
 4. Note if examination causes pain.
 5. Note relation of intestinal resonance to swelling.
 6. Examine for tenderness in angle between erector spinal and last rib.
 7. Examine along course of ureter for tenderness.
 8. Test for hyperaesthetic areas of skin.

II. *The Bladder.*

1. Test for swelling or tenderness about symphises pubis,
2. If there is a swelling percuss out its boundaries.

III. *The Penis.*

1. The meatus. Is it in its right position or does it open on the under or upper surface of penis.
2. The prepuce—any inflammation or growth.
3. The glans—any inflammation or growth.

4. The body of the penis—any swelling, ulceration, growth, if so their nature and characteristics.
5. The urethra, presence of a discharge.
6. Examine inguinal glands.

IV. *The testicles.*

1. Is the testis or epididymis affected or both.
2. Is there a hydrocele (trans-illuminate).
3. Scrotal skin. Is it adherent to testicle, if so is there a sinus, or signs of acute inflammation, or a large ulcer through which the testicle has herniated or fungated.
4. Is the van deference thickened or not.
5. Are the remaining structures of the cord thickened.
6. Compare the weight of the two testicles.
7. See if the testicular sensation is normal, increased, diminished or lost.
8. Then examine prostate.

V. *Prostate and seminal vesicles.*

1. If enlarged, note the size, shape, consistency of swelling and its position.
2. Examine median and lateral grooves.
3. Examine its mobility or if fixed to neighbouring structures.
4. Are the seminal vesicles enlarged.

(c) The Urine.

1. Note quantity passed in 24 hours.
2. Note all the usual tests.
3. Record results of chemical, microscopical and bacteriological examinations.

(d) X rays—examine plates carefully for abnormal shadows in the kidney, ureteric and bladder areas.

Clinical Notes.

AN UNCOMMON CASE OF POISONING FROM THE USE OF COCCULUS INDICUS.

BY

DR. S. MALLANNA, M. D., F. C. S., D. P. H.

THE seeds of cocculus indicus known as kak-mari locally are sold in the Bazaar and are used as a local application in the form of a paste for the treatment of scabies, ring-worm and for destroying lice. The seed also known as Levant nut contains about 50 per cent oil which is also used for similar purposes. In India it is occasionally used for destruction of cattle and fish. It is not known whether leaves and root are poisonous. Accidental case of death has taken place in a child after local application of an alcoholic tincture of the berries to the head. It is believed that the cases of poisoning by the internal administration of seeds is not known. Excise Inspector named Md. Umar of Aurangabad was given these seeds for purification of blood by a local Hakeem. He was given 5 doses of the medicine. The first dose caused his

rapid death. Each dose consisted of 10 grammes of seeds. The first dose was taken on 15th July 1928 at 4 p. m. after grinding the seeds. He went to the club where he vomitted at 6 p. m. a clear whitish fluid. Later he tried to vomit but nothing came out. Then he suddenly fell down, convulsed, became unconscious and died within 3 hours after taking the medicine. The convulsions were severe and lasted some time preceded by shriek. The post-mortem was not performed as it was believed at the time that he had an attack of cholera. *Cocculus indicus* is a highly poisonous drug and the poisonous effect is due to the presence of an active principle named Picrotoxin a powerful narcotic poison. The seeds contain 1.5 p. c. of picrotoxin. The public are not aware of its poisonous effects and the drug is easily procured in a Bazaar as there is no restriction as regards its sale. The seeds are brown, reniform in shape and so short that they look more or less round of about $\frac{1}{2}$ an inch or less in diameter and possess black wrinkled appearance on the surface. There is an elevated ridge on the surface running longitudinally from the hilum along its convex surface. The hilum is short and slightly concave and presents a minute prominence the reminence of the style and a scar. They are light and weigh about 5 to 6 grains in weight. Seeds are bitter. When the seed is opened it contains a mushroom shaped kernel surrounded by a cavity and attached to the shell at the hilum by a short pedicle. The kernel contains aleurine granules, oil globules and a gluconide. The powdered seeds can be distinguished by means of colour re-

actions peculiar to *cocculus indicus*. Pure sulphuric acid produces pink colour and addition of sulphuric acid and bichromate of potash produce pink, brown and green colour.

A CASE OF TRIGEMINAL NEURALGIA

A Hindu patient from Amrabad by name Runga Reddy was admitted in the Casualty Ward Osmania Hospital on 5th December 1928.

Patient's own statement of complaint:—

Pricking and darting pain on the lower part of the right side of the face.

History of present disease:—

Three months back patient suddenly felt severe darting pain starting from the right angle of the lower lip extending upwards and lateralwards to the cheek. For the first ten days these attacks were few. But later the number of attacks per day as well as the severity of pain increased. He had undergone native treatment but it was of no avail. At present he gets two or three attacks of this pricking pain in every five minutes and each attack lasts about a few seconds.

Personal History:—

Occasionally the patient drinks toddy and country liquor.

Family History:—

Nothing of importance.

Condition on Examination:—

The patient is a robust adult with prominent eyes. When the patient gets the paroxysm, twitching of the muscles of the face is noticed. During the attack the patient presses the affected side and grinds his teeth which relieves him of his pain. The affected side becomes slightly flushed. No lacrimation but there is increased secretion of saliva. There is no dental caries. On examination the pain was found radiating along the maxillary and mandibular divisions of the trigeminal nerves. The Superficial divisions like infra-orbital and mental nerves were tender to pressure.

Digestive system:—

Teeth clean; tongue normal; appetite is lessened and bowels are slightly constipated.

Other systems are normal.

Treatment:—

First day analgesic remedies were employed, the following prescription being given

1. Rx.

Pot. Bromide Dr. 1.

Liq. Morph. Hydrochl. Dr. II

Aqua Chloroform Ozs. VI

Into six doses one every 4th hour.

2. Blister applied to a spot on the affected side.

This treatment continued for five days and finding no relief neurectomy was performed on 9-12-28.

Operation:—

A curved incision about two inches in length along the lower border of the angle of the Right side of the mandible was made; muscle Masseter divided; periosteum separated; and on the ramus (right side) a circular piece $\frac{1}{2}$ " in diameter was removed by trephining; internal Pterygoid was exposed, and the most posterior structure on it, the inferior dental nerve, branch of the mandibular nerve was cut; Muscle Masseter was sutured; flaps of the wound brought together and sutured; tr. iodine painted and the wound was dressed and bandaged.

Result:—

No more paroxysms of the attack; the infra orbital is still tender to pressure.

These notes have been taken with the kind permission of Dr. S. A. Rahim, M. B., D. O., who operated upon this case.

A. Natesan Pillay,
House Surgeon,
Casualty Ward,
Osmania General
Hospital.

Abstracts.

BY

ROBERT J. ROWLETTE, M. D., F. R. C. P. I.

THE mucous membrane of the stomach secretes a juice which contains two substances of importance—pepsin and hydrochloric acid. Pepsin is an enzyme which acting only in an acid medium, digests proteins. In this respect it is important but its action is not essential in the digestion of food, as the digestion of the proteins is effected in the intestines by the action of trypsin. The main action of hydrochloric acid is bactericidal, but it also activates the pepsin, and is necessary for the production of secretion. It is present ordinarily in strength of from 0.1 to 0.2 per cent of the secretion rarely exceeding the latter figure. In normal circumstances gastric juice is secreted when the appetite for the food is roused, during the act of eating, and for a little time afterwards. During

ing, however, secretion is almost at a stand still, and the juice becomes nearly neutral in reaction.

If a portion of the mucous membrane of the stomach or of the duodenum close to the stomach loses its self protective power against the gastric juice it will be digested. If we assume that from some unknown cause (and the cause is unknown) a limited area of the epithelium loses its protective power against pepsin, then ulceration of that area is likely to occur. It is noteworthy that the appearance of gastric or duodenal ulcer is quite unlike that of an ulcer of the ileum or colon. In the latter situation severe inflammation and necrosis precede ulceration; in the former there is no evidence to suggest that there is any inflammation precedent to ulceration, and one never finds any necrotic material. It is noticeable that ulceration of this digestive type (now generally as peptic) is never found in area bathed in alkaline fluid. It is a striking fact that trypsin, an enzyme of much greater proteolytic power than pepsin, does not appear to have any capacity to produce any ulceration in the intestines. Acidity is found, therefore, to be essential for the production of peptic ulcers, although hydrochloric acid of itself, in any concentration that may be present, has certainly no digestive power.

We know nothing of the relation of the acid to the formation of ulcers. We know nothing of its power of preventing the healing of ulcers. But persistent acidity is a frequent accompaniment of ulceration, and that it is a frequent source of pain

when ulcers are present. It is the persistence of the acidity of the juice, rather than excess of acidity, which is the important abnormality.

Modern treatment:—Prof. Faber of Copenhagen has put forward the view that acid dyspepsia, gastritis, and gastric ulcer are three closely allied conditions, if not, indeed, different phases of the same condition. If we succeed in keeping the acidity of gastric juice at a persistently low level, or are able to keep the juice alkaline, gastric ulcer does not occur, and if it has already occurred, healing takes place. Details of treatment will depend largely on the special features of the individual case. If there has been severe haemorrhage great care is necessary. Of the treatment of haemorrhage itself, morphine hypodermically and the administration of normal serum appear to furnish us with our best defence. After haemorrhage one would hesitate to give any thing by mouth except ice or cold water. Thirst may be relieved by saline enema and if necessary saline enema may be given. Rectal feeding has been abandoned. Salt, sugar, and alcohol are the only substances absorbable by the rectum which tend to maintain strength.

Diet in the Acute Stage:—Except in cases of haemorrhage feeding by mouth may begin from the first. Give food which does not stimulate gastric secretion or the movements of the stomach. At first milk or milk and cream and albumin water are given in alternate feeds at two hourly intervals. The quantity of either given at a time varies from

two to four ounces, according to what the patient can comfortably take, and to the stage of the disease. With fluid food of this simple kind, associated with the drug treatment described below and of course, rest in bed, pain usually disappears in a few days. As soon as pain has disappeared beaten up eggs can be added to the diet (at first, and soon two or three in a day). In the second week bread-and-butter and biscuits, such as cracknels (which cannot be swallowed unless well chewed) may be added gradually. Acids, jellies, soups, and the meat extracts are better avoided; as they tend to excite gastric secretion.

In the third week light steamed fish and boiled chicken come into the diet. In the fourth week mutton and light meats can be taken, and thereafter the diet becomes very liberal.

General Treatment:—

In severe cases the patient is kept in bed until the end of fourth week, and return to normal is gradual. In duodenal ulcer one can usually proceed more rapidly as regards food than in gastric ulcer. Close attention to the condition of bowels is required. Phenolphthalein is convenient and pleasant in the early stages, and, in the later, olive oil, liquid paraffin, agar jelly that is to say lubricant aperients are better than irritating ones. Where a patient is receiving only liquid or soft food, the mouth should be carefully washed several times in the day with a mild antiseptic mouth wash. Dental help must be obtained where the condition of teeth requires it.

Drug treatment:—

For generations, Bismuth, soda, hydrocyanic acid, and morphine have been given on empirical grounds. Two conditions above all others tend to prevent ulcers from healing. One is the active movements of the stomach and the other is the presence of the acid gastric juice. Remembering that pepsin can only act in an acid medium, and that the hydrochloric acid may itself be directly irritating to an ulcer, we must endeavour to inhibit the activities of both these substances at one stroke that is by rendering the gastric juice alkaline. Bread soda has been a traditional remedy for generations for all kinds of dyspepsias, including no doubt, many cases unrecognized ulcerations. It has the advantage of being easily soluble and diffusible, but the corresponding disadvantage of being readily absorbable and therefore temporary in its action. Bismuth carbonate is more stable and persistent in its action but is rather constipating in its action. Intermediate between the two, and definitely laxative in action, is heavy magnesium carbonate. Sodium citrate is another convenient and pleasant form of administering alkali. By a combination of two or three of these salts, varied according to the needs of the case, we can generally keep the gastric juice under control. 20 grs. of sodium bicarbonate and 10 grs. of heavy magnesium carbonate will rarely be found too laxative. MacLean recommends a combination of one part of sodium bicarbonate and two parts each of magnesium carbonate and bismuth oxycarbonate. Belladonna has the effect of diminishing the secre-

tion and the movements of the stomach. It is only rarely that doses greater than five minims of tincture three or four times a day have to be given. In some cases in which alkalies did not give the desired relief belladonna was rapidly effective. Oils and fat have an inhibitory effect on the gastric secretion, but this effect is almost negligible unless they are taken on an empty stomach. A tablespoonful of olive oil with 20 grs. of heavy magnesia mixed up in it, taken midway between meals, will keep many people comfortable. Cream may be given as it is almost as useful. The time at which the medicine is administered is important. It is governed by the time of occurrence of pain and discomfort in relation to food. In cases of gastric ulcer in which the pain comes on twenty minutes or half an hour after food, the medicine should be given within a few minutes of taking food. In other cases of gastric ulcer and in nearly all cases of duodenal ulcer in which the pain appears some hours later the medicine should be given one or two hours after food, that is to say a quarter of an hour before the appearance of the pain. In cases in which the discomfort occurs during night, a fairly big dose of alkali should be given as the last thing before going to bed. One or two more powders with a supply of water, should be within reach of patient and when he awakes in the middle of night he should take one of these. On waking in the morning it is necessary to take another.

Alcohol should be strictly forbidden while there is any discomfort at all and afterwards strictest moderation should be observed. Only lightest wines

should be allowed. Spirits of all sorts must be abandoned. Tobacco smoking is as injurious as alcohol. Cigarette smoking appear to be more injurious to stomach than pipe or cigar smoking. Smoking does less harm if done immediately after a meal, and it should be altogether avoided when the stomach is empty.

Diet after the acute stage:—The patient should never be allowed to remain with an empty stomach or feel hungry. He may carry a few biscuits in his pocket and break his fast therewith. The following classes of food should be definitely excluded from the diet. All highly spiced foods; condiments such as mustard and pepper; pickles and acid fruits. All food which are likely to leave a solid residue persisting in the stomach. The worst of this kind are, raisins, currants and generally green vegetables. Hard food such as nuts and meat extracts and soups which stimulate the secretion of gastric juice.

On the other hand meat, both fat and lean, may be given in moderate quantity, cream and butter freely, bread-and-butter, toast, and biscuits in ordinary quantities. After gastric ulcer more caution is necessary than after duodenal ulcer. In every case individual attention must be given and each food treated as an experiment. At the end of two years from the cessation of symptoms treatment by medicines may cease and greater freedom in diet permitted.

Surgical Treatment:—It may be asked whether surgery is no longer to play any part in the treatment

BY

R. N. CHOPRA,

(*Indian Medical Gazette*)

63: 186—190 April.

The chief active constituents isolated from *Saussurea Lappa* (Kuth root) are an essential oil which forms 0.5 per cent of the root, traces of an alkaloid, a bitter substance of glucosidic nature and a resin. The most important of these from a medical point of view is the essential oil, though the alkaloid and the glucoside may undoubtedly help. It is rapidly absorbed and is excreted into lungs. It has an expectorant action and also produces relaxation of bronchial muscles. The author used an alcoholic extract of the root in his therapeutic trials. Following up the broncho-dilator and marked expectorant action produced by this drug, an extensive trial has been given to it in the treatment of bronchial asthma. Besides its depressant action on the vagal tone, it produces the relaxation of the involuntary muscles and relieves the congestion of the bronchial mucosa by its expectorant and liquifying action on the tenacious sputum. To cut short the paroxysm it is better to give the extract by itself but for long continued uses given, in the authors opinion, better as a mixture. The drug has no cumulative action. It is

better to give the mixture for ten days and stop it to see if the attacks recur. In many cases where the cause is not deep rooted, extract combined with general treatment cures the patient for a long time. The drug failed in cases where the causal factor was very potent, such as a lesion in the nose, enlarged glands in the chest or pathological changes in the gastro-intestinal tract.

CORRESPONDENCE.

To

The Editor,

Deccan Medical Journal,

Hyderabad, Deccan.

Sir,

I shall thank you very much if you will kindly publish the following few lines in your esteemed Journal.

At present the Medical Department gets only 16 copies of the Deccan Medical Journal and I think about a similar number of the copies of the Indian Medical Gazette. These are supposed to be sent to each Civil Surgeon in the District and he in his turn is supposed to send them to each of the Medical Officers in the Particular district under him. Granting that every Medical Officer takes care to return it within a fortnight, the last Medical Officer in the list will receive it nearly six months after the date of its Publication. All his enthusiasm in reading it generally fades away when he sees that it is as old as this. Moreover whenever he wants any of the old numbers for reference he is generally unable to get them with ease. The Deccan Medical Journal being only one of its kind, he generally looks to it for fresh news concerning medical affairs and particularly Hyderabad Medical Department. As every other Department such as the Educational etc., generally arranges for the supply of Newspapers etc.,

to its various institutions in the districts so also the Medical Department if it arranges for the supply of at least less costly Journal—D. M. J.—to all Dispensaries in the Districts, The Medical Officers in the Districts will feel very grateful to it. Having the Journals permanently in the Dispensaries will not only create interest in reading them but it will enable the Medical Officers to keep in touch with the latest developments in the Medical Science. I think that if the Hyderabad Medical Association represents the matter properly to the authorities concerned it can be arranged without great difficulty. After all spending annas eight per month on each Dispensary (the subscription of the Journal being only Rs. 6 per annum) is not of great consequence when compared to the great benefit it is going to give to the Medical officers directly and the patients indirectly. Thanking you once more,

News.

RAZA Khan M. B., Ch. B., Civil Surgeon Sangareddy has been posted to the Osmania General Hospital.

Abdul Jabbar M. B., B. S., Civil Surgeon Bidar on his return from Study leave has been posted to the X-ray department of the Osmania General Hospital.

S. A. Rahman M. B., Ch. B., B. Sc., D. T. M. & H., has been posted as Civil Surgeon Osmanabad owing to retirement on pension of—Balm L. R. C. P. & S.

R. K. Garde L. M. & S., Assistant Surgeon Raichur has been posted as Officiating Civil Surgeon at Sangareddy.

Syed Mohiuddeen L. M. P., Medical Officer Mahbubabad, has been transferred to Yelandu Dispensary.

D. Paramashivam L. M. P., District Sanitary Assistant Warrangal has been posted as Medical Officer at Mahbubabad.

B. S. Narayen L. M. P., Medical Officer, Mahbubabad is posted as District Sanitary Assistant at Warrangal.

S. Raghavender Rao, L. M. & S., Medical Officer, Rajura Manickgarh has been transferred to the Osmania Medical College as Senior Demonstrator in Physiology.

Vishwanatham Pillai L. M. P. has been posted temporarily as Medical Officer at Rajura Manickgarh.

R. G. Jatkari L. M. P., Medical Officer Bhokardan has been transferred to Purbhani Civil Hospital in place of L. S. Karajgikar L. M. P., transferred to Bhokardan.

Miss B. DeSouza L. M. P., has been transferred from Nanded to Mahbubnagar.

B. Sitapati Rao L. M. & S., Medical Officer Adilabad has been posted as Officiating Assistant Surgeon at Nizamabad in place of S. K. Jatkari L. M. & S., transferred to Nazam Sagar Project.

L. R. Deshpande L. M. & S., acting Medical Officer at Adilabad has been transferred to Nizamabad in place of Brahmandam L. M. P., but till the return of B. Sitapati Rao L. M. & S., he will continue to work at Adilabad.

Miss H. D. Charles L. M. P., has been appointed as Sub-Assistant-Surgeon at Chaderghat Dispensary.

Miss B. Taylor L. M. P., has been appointed as temporary Sub-Assistant-Surgeon at Sangareddy.

V. H. Atre L. M. P., Medical Officer Mahur is posted as Sub-Assistant-Surgeon at the Civil Hospital Nizamabad.

Hamidulla Khan has been posted as Medical Officer at Mahur.

Bhaskar Rao L. M. P., Medical Officer Chincholi, has been posted as Assistant to the Medical Officer at Central Jail Hospital Gulbarga.

Ram Rao Ghanekar, L. M. P., Assistant to the Medical Officer of C. J. Hospital Gulbarga, has been posted as Medical Officer at Chincholi.

Md. Mazhar Hussain L. M. P., has been transferred from Mominabad Dispensary to Siddipet Dispensary.

M. Sitaramiah L. M. & S., Medical Officer Palem has been posted as District Sanitary Assistant Adilabad.

Hanmanth Sham Rao L. M. P., District Sanitary Assistant Adilabad has been posted as Medical Officer at Palem.

P. G. Tole L. M. P., Medical Officer Chinnur has been transferred to Pakhal vice Sanjiv Rao L. M. P., transferred to Chinnur.

G. V. Deshmukh L. M. P., Medical Officer Kandhar, has been posted as Medical Officer at Makthal.

Kolandavelu L. M. P., Medical Officer Makthal has been transferred to Kandhar vice G. V. Deshmukh L. M. P.

G. B. Kulkarni L. M. P., has been posted as Medical Officer at Mominabad.

Dikshacari L. M. P., has been posted as House surgeon at Beed vice G. B. Kulkarni.

Md. Faruk L. M. P., Medical Officer Jangaon, has been posted as Medical Officer Sirsilla.

C. M. Hanumantha Rao L. M. P., Medical Officer Sirsilla has been posted to Jangoan vice Md. Faruk.

Bhoomanna, L. M. & S., has been posted to the Chemical Laboratory Hyderabad in place of Rangaswamy L. M. P., retired.

X. M. Pariera L. M. P., has been transferred from Chemical Laboratory to the Osmania General Hospital.

S. Mahmudul Hassan has been transferred from Patan to Kamareddy in place of Joseph Israel L. M. & S., transferred to the Chemical Laboratory.

Narsappa L. M. P. Medical Officer Otnur has been posted as Medical Officer at Huzurabad.

Manick Rao L. M. P., has been posted as Medical Officer at Otnur.

The Deccan Medical Journal December 1928.

MEDICAL RELIEF IN HYDERABAD.

THE construction of a large hospital like the Osmania Hospital which has been recently completed and which will be shortly equipped with the most modern appliances is a sufficient proof that the authorities are well aware of the medical needs of the population of the Hyderabad Dominions. Till recently the main hospital could accommodate only about 125 patients. The present building can accommodate 500 patients. The hospital is built on the most modern principles of sanitation and consists of a ground floor with two upper floors. There are 18 large airy wards of 24 beds each and private rooms for over 36 patients. Equipment under the direction of Major Khaja Moinuddin the present Director of the Medical Department is almost complete and it is expected the organization of the hospital will be undertaken shortly.

For our needs we not only require a Surgical side and a Medical side in the hospital but we require also well managed departments of the Eye, the Ear, Nose, Throat, the Dental Department, the Venereal diseases Department, the Maternity Department and the Skin and X'Ray Departments. Until about 1920 A. D. Surgery was the main work of the Hospital. Then Col. Drake-Brockman introduced the X rays and encouraged specialization in Eye Surgery by establishing a separate Eye Ward. Dr. Lankester and Col. Singh later on found that the Medical side was not enough paid attention to for we had till then mixed wards only and they insisted on specialization. Col. Singh helped by the then Secretary of the college Dr. Farhat Ali began by separating the Surgical from the purely Medical side and created purely Medical Wards. Thus came into existence by the side of the Surgeons, the Physicians of the Hospital; and this added to the efficiency of the college teaching also. We now want the other departments mentioned above. The X-ray Department has been recently equipped with a new X-ray plant and some additions will be made as suggested by the Medical Officer in-charge.

There is great need of radium therapy. We have seen marvellous effects of this on patients sent to the Radium Institute in the North—now located at Patna. We venture to suggest that our large hospital should have a well-equipped Radium Department.

We wrote in Vol. II No. 4 of this Journal that maternity hospitals for non-purdah women were

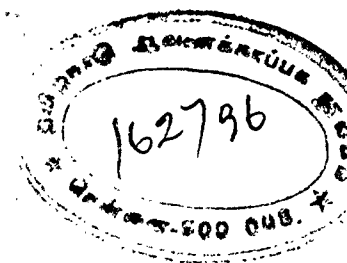
badly required and we are glad to have a block kept apart for this purpose in this Hospital. Airy rooms are essential and our wards are best suited for the care and treatment of lying-in women.

Our Hospital has been known for its Eye Work and as we have now an eye-specialist on the Staff, the Eye Department will be well organised.

The other departments of Ear, Nose, Throat, Dentistry, Venereal Diseases will require men specially trained for the purpose and it would be well if the Government Scholars now studying in England specialized themselves in these subjects.

Attempts have recently been made by major Khaja Moinuddin to start Tuberculous Hospitals but owing to want of suitable sites nothing definite has been done in that direction. Our large building has a good roof-terrace and it could be well utilized for Tuberculous patients until such time as a separate hospital is built for such cases. The Medical College Hospital of Calcutta has such Tuberculous Wards on the top terrace.

Large hospitals like ours supply good material for teaching and for research work. Our hospital requires a few additions here and there for this purpose. An out-patients' theatre, a clinical theatre with a Clinical Laboratory for students are absolutely essential. In the J. J. Hospital of Bombay, the General Hospital of Madras, the Medical College Hospital of Calcutta, rooms are set apart for Clinical work well equipped with chemicals and microscopes, etc. and facilities are thus allowed to students



of medicine to work freely. Similar facilities can be allowed to our students at this hospital.

The present laboratory when equipped will be of great help to the Physicians and Surgeons of the hospital. In addition to this the Physicians should be given rooms where they could do their own clinical and research work.

We have a good Europe-qualified and local Medical Staff and the hospital when fully equipped with the necessary appliances and additions as above indicated will be the best institution of its kind in the Deccan.

Post Graduate Notes.

HEART FAILURE AND ACTION AND USE OF DIGITALIS.

THE chief symptoms of heart failure or cardiac insufficiency are peripheral oedema, cyanosis, venous stasis, dyspnoea, enlarged liver, and diminished quantity of urine.

Heart Failure is that condition in which the heart is unable to maintain an efficient circulation during the efforts necessary for daily life of the individual. In studying heart failure, therefore, an enquiry must be made of the the circumstances which have impaired the functional efficiency of the heart muscle.

The energy of the heart consists of two elements:—

1. The actual energy
2. The reserve energy

When the reserve energy is diminished we get the first sign of heart failure (acceleration of heart-beat and shortness of breath on exertion). Later

the actual energy is involved and then we get early exhaustion on slight exertion, cyanosis and venous disturbances—congestion of the various veins (pulmonary, renal, hepatic, splenic, etc). Even at rest, we get rapid pulse and shortness of breath. Heart is increased in size, with diffused impulse. Basal systolic murmur we may hear. We have pulse irregularities such as extra-systoles and dropped beats and heart block, auricular fibrillation, tachy-cardia, pulsus alterans, etc.

Tests for functional efficiency of heart are:—

1. Pulse rate before and after walking
2. Duration of acceleration of pulse on exertion.
3. Blood pressure test
4. Sphygmography
5. Electro-cardiogram
6. Radiography

There is no mathematical formula to serve as a test. Many factors are involved: Excessive peripheral resistance, weakened energy of pericardium, partial or total derangement of its principal functions, etc. All parts are not involved to the same degree and in the same way.

SYMPTOMS OF RIGHT HEART FAILURE:—

Failure is usually slow and insidious; seldom sudden symptoms. There are three forms: acute, sub-acute, and progressive.

Acute:—Chiefly during pregnancy or after confinement especially in women with mitral stenosis. Gravidocardiac symptoms, at first, are respiratory oppression with rales in the chest. Then dyspnoea increases and becomes continuous—cyanosis of mucous membranes and lips and fingers and toes; engorged jugular veins, quick pulse, heart much dilated, sputum pink then red and then bloody clearly; death from cyanosis often takes place. Sometimes termination of pregnancy may put an end to symptoms.

Sub-acute:—This is seen in mitral disease, in chronic changes of lungs or pleura, in acute failure of left ventricle.

Progressive:—Symptoms are: dyspnoea on exertion; condition is not painful. It is relieved by rest and is not troubled during sleep. Bronchial congestion follows, cough, sluggish digestion, gastric fulness, cerebral torpor, all indicating visceral stasis. Besides this there may be palpitation also. Objective symptoms at this time are:—Cyanosis of lips and cheeks, oedema over ankles, towards evening especially, disappearing in the morning. Pulse is regular or may show extra systoles. If there is auricular inefficiency there may be constant arrhythmia. Arterial pressure gives no indications. Heart shows no organic lesions at that stage or there may be commencing dilatation. Apex beat will be displaced downwards and outwards. Then the disease progresses. Urgent symptoms and signs appear such as dyspnoea on slightest exertion, orthopnoea, con-

gestion of the base of the lungs, oedema of the extremities, cyanosis of the lips, prominence of the jugular veins, congestion of the liver, and urinary disturbances.

FAILURE OF THE LEFT VENTRICLE.

Causes:—Arterial hypertension (with or without chronic Bright's disease)

Extensive lesions of Aorta;

Adherent pericardium especially in children;

Exceptionally there are no organic causes, but excessive exertion or exhaustion may cause it; but, in all probability in such cases the heart has been damaged before.

Symptoms:—Without prodromata, acute oedema of lungs or angina pectoris, causing sudden death.

More often there are some previous signs such as painful dyspnoea, different from cardiac dyspnoea of effort where we have no pain; different also from angina pectoris where there is no dyspnoea, at least in the beginning. Walking and exertion cause it. Coitus, defecation may also cause it. At times other diseases bring on this condition.

Subjective Cardiac symptoms at this stage are:—

Palpitation, attacks of tachycardia, painful distension of epigastrium, extra—systolic irregularities.

Objective signs are:—

Accelerated heart beats.

Gallop rhythm.

Sudden and abnormal increase in arterial pressure at times.

Tachycardia: Beats are never more than 110 to 120 per minute but they are always above 80. This means that the ventricular hypertrophy is no longer capable of combating peripheral resistance; secondly there is the beginning of dilation; thirdly deficiency in the energy of beats is compensated by acceleration of beats.

Gallop rhythm means the same, namely hypertrophy complicated with dilatation.

Tachy cardia.	} Triad—recognize it to foresee imminence of left ventricular failure.
Extra systolic irregularities.	
Gallop rhythm.	

A lowering of pulse pressure does occur during the course of heart failure but we have a sudden rise at times at the approach of cardiac failure. The prodromal phase may last from few days to several months. It may at times be absent, and we may have sudden failure of left ventricle.

Cardiac Asthma:—In the evening or later after hours of quiet sleep. There is distress and respiratory anguish. Patient cannot lie down or rest in bed must sit up in chair. After several minutes or hours subsides like true Asthma to appear again the following night. Though appears slight it is a sign of failure of left heart.

Angina Pectoris:—More characteristic sign. It may appear at the same hour but comes on suddenly and immediately attains maximum intensity. Pain

behind sternum and in pre-cordial region radiates to shoulder, left arm, and left scapula. Sensation of the chest being crushed and back and arm being torn by claws. There is a sense of imminent death. Face is pale and anxious and there is an excessive sweating. Fatal syncope may end the same but recovery is always possible. The condition may be temporary or long lasting.

Oedema of Lungs:—It may be the end of Angina pectoris, but it may come on without preceding Angina. The sensation of fulness or constriction of chest is followed by incessant cough which ends in expectoration of salmon-coloured bloody and frothy liquid which is the objective sign of Oedema of Lungs. Fine rales may be heard from base upwards. Death by Asphyxia is common here as death by Syncope in Angina. At times death is deferred for several days when progressive failure, ordinary heart failure, succeeds acute left ventricle failure.

DIGITALIS.

Digitalis is very valuable in the treatment of heart disease. William Withering by pointing out its marvellous effects in cardiac dropsy about 1785 A. D. became one of the benefactors of humanity. It was introduced in France in 1804 A. D. by Bidault and Villiers.

The lamina of the leaf is alone used in therapeutics as its medicinal power is almost constant. Powdered leaves constitute the most important pharmaceutical form of the plant. On exposure to air and light it deteriorates in quality owing to an

oxydase in it and therefore it has to be renewed frequently. Powdered leaves, infusion or maceration, are superior to capsules and pills. Infusion is prepared in hot water at 70 degrees centigrade for half an hour and contains a little more of the active principles than does maceration which is prepared in cold water for 12 hours. Both are unsuitable for long use as they cause nausea, vomiting, and diarrhoea being irritant to digestive tract.

1½ grains of powdered Digitalis correspond to 57 drops of the Tincture. Tr. of Digitalis preserves its full activity for one year. Three glucosides form the active principles of the leaf. (i) Digitalin. (ii) Digitalein, (iii) Digitoxin. The last mentioned one plays an important part in the powder, the tincture and the extract. Digitalin is a crystallised glucoside insoluble even in boiling water; especially soluble in chloroform; the German product Schmiedeberg's Digitoxin is nothing but the French crystallised chloroformic Digitalin. It may be given in solution thus: 16 grains of crystallized Digitalin in 15 ounces of 95 per cent Alcohol with 13 ounces of Glycerine and distilled water up to 33 ounces. This solution keeps well. It is not given hypodermically. 10 to 25 drops of it may be given intravenously daily or every second day for 3 or 4 doses.

By using digitalin the useful action of other substances in the leaves is lost. The defects of the older preparations have been remedied by stabilizing the preparations (Oxydases should be got rid of by treating plant with alcohol vapour at low pressure)

and standardising them by biological tests. In place of these the fresh Tincture is very valuable.

Action of Digitalis: Francois Frank's researches have shown that Digitalis slows the cardiac rhythm and makes it regular. Prolonged action or increased dose brings on toxic symptoms of Arrhythmia which consisting at first of doubled and rhythmic systoles (bigeminy) becomes later irregular with pauses of unequal duration between the abortive or retarded systoles. Finally the heart undergoes a short paroxysm of rapid but dissociated shocks. These are followed by Fibrillary undulations and ultimately the heart stops in diastole. Thus there are two phases of digitalis poisoning. The first one is the useful phase in which the beats of the heart are better coordinated—the myocardium becomes more distensible—more elastic—the systolic contraction more energetic—the heart opposes less resistance to the column of blood during diastole. The second one is the noxious phase in which the functions of the heart are profoundly disturbed. The slowing of the beats involves the whole heart (auricles and ventricles) and is due to lengthening of the diastolic pauses and diminution of the number of excitations per minute. It is thus a sinus slowing. This arrhythmia is of nervous origin (vagus). Digitalis also has inhibitory action on the conduction by the A. V. bundle. We get digitalis Heart Block. Feeble contractions of auricle are thus prevented from reaching the ventricle—time of diastole is prolonged and the systole is more energetic. Thus we get the favourable effects of the drug.

Digitalis produces another form of arrhythmia consisting in coupled beats (bigeminy), at times trigeminy. It indicates that the limit of tolerance is reached. It is due to extrasystoles brought on by an increase in myocardial excitability and like digitalis block it is due to a lesion of the cardiac muscle and is an indication for discontinuing the drug.

Action of digitalis on contractility is not definitely known yet. Cushney and Mackenzie think Digitalis diminishes contractility because it provokes alteration of the pulse. The question is not yet solved.

Has Digitalis any action on the tonicity of the heart? Some deny the possession of tonicity by heart muscle. Starling did not think that the heart was endowed with tonicity but he does not deny to the myocardium the energy of contraction. There is no general agreement as regards the action of this drug on the tonicity of the Heart.

Digitalis has no control over failure due to acute distension of heart on account of loss of energy of contraction. By prolonging the diastole it may do harm by imposing excess of work on the heart.

Digitalis is to be withheld in disorders of conductivity and also extra systole although small doses have a sedative effect on the latter. Both effects are obtained in auricular fibrillation or complete arrhythmia. It depresses the activity of the sinus and blocks the weak contractions—only strong ones pass through. Thus ventricle is able to fill up more completely and to contract more energetically. It

also acts on peripheral circulation and is therefore efficacious in conditions in which it may not be formally indicated.

Accessory properties of Digitalis.

Artetial Pressure: Formerly it was believed that action of Digitalis was due to elevation of arterial pressure. Now it is shown by Potain and others that degree of diuresis has no relation to change in arterial pressure—that diuresis begins before there is any change in pressure—that sometimes it is only when diuresis decreases that a slight rise is noticed to begin. Therefore it can be given without danger to hypertensive subjects as well as to those whose pressure is normal or low.

Kidneys: In health it does not alter quantity of urine. Diuresis is due to vasodilatation of renal vessels and so the action is merely mechanical and it is always quite limited.

Peripheral circulation: Vasoconstriction action on peripheral circulation—very doubtful.

To summarise: Digitalis diuresis is due to direct action on heart. Edemas of cardiac patients are due to peripheral stasis essentially—to diminution of the energy of ventricular systole and to insufficient emptying of right heart. Digitalis lengthens the duration of diastole, enabling the heart to contract with more energy and this explains the diuresis.

Toxic and accumulative effects:—

Symptoms of excessive dose are:

Headache, Nocturnal delirium, Excessive slowing of pulse, violent and repeated vomiting of greenish glairy mucus.

In very severe cases Diarrhoea and suppression of urine. Usually only headache and nausea are present where poisoning is due to too prolonged use of relatively small doses.

Ed.

Extract from the Annual Report of the Osmania General Hospital for the year 1337 Fasli.

BY

R. N. COORLAWALLA, F. R. C. S., D. P. H.

*Superintendent of the Osmania General
Hospital.*

I have the honour to forward herewith report of the work performed at the Hospital during the year 1337 Fasli.

This is the 7th Report I have the honour to submit. During the early part of the year, a severe epidemic of Plague raged in the City of Hyderabad and its environments, and the City was depleted of its population. People who could afford left Hyde-

rabad City for the Districts and heavy masses of the population camped out of the city in the suburbs. During this period the number of out-patients had fallen to a low level and the in-patients also had decreased but as the epidemic subsided both the in-patients and out-patients treated was 1,66,344 and that of in-patients 3,655 and the number of major operations was 1,205. The average daily number of sick in-patients new and old being 181.77 and the mortality percentage of the total patients treated inclusive of cases admitted moribund during Plague epidemic period etc., being 8. 12 per cent, the lowest after a good number of years.

During the last six years' period, the first consideration and principle kept in view is that the function of a Hospital is the healing of the sick poor. Hospitals have of course, other functions e. g., the training of those who care for the sick; viz., Physicians, Surgeons, Nurses, Social Workers, etc., i. e. (Teaching function); the prevention of diseases (research function), and constructive health building; but healing the sick poor must always claim first attention, in other words, the primary responsibility of the Hospital is to the patients. It does not exist for Doctors, Nurses, and Social workers, but that each of these may most efficiently serve the patients.

The value of the Hospital as an educational centre to the public is becoming more and more apparent as its activities are broadened to bring it into closer contact with the community.

To meet these responsibilities it appears necessary that Hospital should provide at least the following:—

1. Trained and competent personnel for each activity, adequate in number to serve the patients when service is required.

2. All that is necessary in the way of Medical and Surgical equipment and accessories, so that the personnel may render their service with the maximum result.

3. An efficient organisation, so that every worker in the Hospital is able to render the greatest service to the patient at the time it is needed.

4. A Physical environment where all the elements of successful treatment and handling of patients can be applied, including proper food, etc., and element of cheerfulness.

5. An atmosphere conducive to recovery, which embraces such imponderables as kindness and courtesy, not only to the patients but to their relations and visitors, on the part of both the professional and the administrative staffs, quiet, comfort and cheerfulness.

6. A sound and up-to-date business organization.

From being places where physic, pills and ointment were the armamentarium of the Physician and the knife all the surgeon could offer, Hospitals have gradually developed into institutions for the pre-

vention and treatment of disease by organized co-operative methods.

The advance of knowledge, the discovery of precise and scientific methods, of diagnosis and treatment, the harnessing of many allied sciences to the art of Medicine and Surgery, have all placed heavier burdens upon them. Few people realize how many different individuals are concerned in discovering the nature of a patient's illness and assisting in its treatment. Physicians and Surgeons and specialists in diseases of the Skin, Ear, Throat, Nervous system etc., as well as those specially skilled in X-Rays, Radium and light treatment, may all be needed for precise diagnosis, or efficient treatment. In short, the Hospitals have long outgrown the purposes for which they were originally founded, they are no longer simply refuges for sick people; they have become centres for research and education as well as for treatment.

POPULARITY OF HOSPITALS.

With the increase in knowledge and the facilities afforded by the hospitals of today, it is not surprising that they are becoming immensely popular. This is evidenced by the larger and ever increasing number who now seek attendance and admission. At one time the Hospital was the place where one went as a last resort, and the expression. "I would rather die than go to a Hospital" was quite a common one. Possibly at the time, there was good reason

In the first place, the surroundings can never be as hygienic and convenient as they are in a modern hospital, secondly a private practitioner cannot be expected to possess all the equipments and apparatus for every variety of case; and thirdly, amongst the poor, there cannot be the constant care and supervision of a trained nurse, which is so often essential to recovery.

OUT-PATIENTS.

Years.	Mohammadans.	Hindus.	Christians.	Other castes.	Average daily sick new and old.	Mohammadans.	Hindus.	Christians.	Other castes	Average daily sick new and old.
1335 F.	1907	1163	113	60	153.07	32615	26908	37	14614	459.40
1336 F.	1996	1205	101	45	169.61	35225	26264	53	11031	493.43
1337 F.	2107	1202	96	93	181.77	33702	22834	64	11892	455.73

CLASSES OF DISEASES.

The following statement shows the total treated (among in and out-patients, of the most important diseases which came up for treatment during 1337 Fasli as compared with 1336 Fasli.

Classes of Diseases.	1336 Fasli Total treated.			1337 Fasli Total treated.		
	In- door.	Out- door.	Total.	In- door.	Out- door.	Total.
Other General diseases.	...	...	...	...	...	...
Malarial Fever,	217	41440	41667	279	39450	39729
Disease of the Digestive system.	199	6385	6584	182	6199	6381
Disease of the Respiratory system.	139	5370	5509	107	4779	4886
Disease of the Skin.	14	2194	2208	16	2497	2513
Disease of the Ear.	5	3189	3194	4	4177	4181

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Disease of the Generative system.	168	525	693	186	613	799
Disease of the Nervous system.	148	379	527	172	274	446
Disease of the Urinary system.	189	183	372	176	159	335
Disease of the connective tissue.	139	651	790	162	625	781
Injuries (General and Local)	145	494	639	187	324	511
Ulcers.	39	463	502	56	199	255
Dyspepsia.	30	5592	5622	27	5235	5262
Rheumatic Fever and Rheumatism.	25	1375	1404	35	1071	1106
Dysentery.	46	1535	1581	44	868	912
Tuberculous affection.	89	234	323	109	78	287
Syphilis.	42	382	424	38	221	259
Genorrhoea.	3	186	189	11	209	220

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REPORT OF SURGICAL WORK PERFORMED
IN THE OSMANIA GENERAL HOSPITAL.

DURING THE YEAR 1337 FASLI.

	Major.	Minor.
1335 Fasli	... 867	3355
1336 Fasli	... 1198	4214
1337 Fasli	... 1205	3532

Major 1205 and 3532 Minor operations were performed during the year.

The following list gives the Surgical diseases for which operatives interference was found necessary.

	Number.
Operation on Tumour	... 90
do. on Cyst	... 54
do. on Throat	... 838
do. Abscess	... 143
Removal of foreign body	... 5
Operation on Nerve	... 2
do. on Lymph, Glands	... 26
do. on Bone	... 313
Amputation	... 31
Operation on Skin and Sub-Tissue	.. 639
do. on Ear	
do. on Joints	... 69

Operation on Eye and its appendage	... 319
do. on Breast	... 13
do. on Chest	... 20
Dental Operation	... 661
Operation on face and accessory Sinus	... 17
Operation on Abdomen	... 230
do. on Rectum	... 67
do. on Bladder	... 392
do. on male generative organs	... 788
do. on female generative organs	... 18
Poison	... 2
	4737

Names of Surgeons.

Names of Surgeons.	Gastro Jejunostomy.	Appendicitis.	Hernia.	Liver Abscess.	Litholopaxy.	Sup. P. Lithotomy.	Fibroid Uterus.	Ovarian Tumour.	Amputation Upper.	Amputation Lower.	Operation Major bone.	Operation Major Joints.	Tumours.	Plastic Operation.
Superintendent.	49	30	28	14	11	4	1	7	5	7	4	4	108	11
2nd Surgeon.	17	13	22	18	2	8	4	4	5	6	3	...	87	4
Casualty Surgeon.	5	2	2	6	1	2	2	...	2	3	...	1	30	4
Total.	71	45	52	38	14	14	7	11	12	16	7	5	225	19

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Names of Surgeons.

Names of Surgeons.	Empyema.	Carbuncle.	Perineal Section.	Mastoid Abscess.	Pleurisy.	Fracture.	Fistula in-ano.	Pan. Ophthalmitis	T. B. Glands.	Operation on Throat.	Piles.	Prostatectomy.	Stricture.	Large Abscess.	Total.
Superintendent.	6	9	5	3	5	42	14	4	5	...	6	1	66	9	458
2nd Surgeon.	3	11	3	6	5	12	13	5	2	2	8	...	65	23	351
Casualty Surgeon.	4	4	1	1	...	3	6	1	7	...	...	1	8	7	99
Total.	9	24	9	10	10	57	33	10	14	2	14	2	139	39	908

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Names of Surgeons.	Grand Total.				
	Superintendent.	2nd Surgeon.	Casualty Surgeon.	Total.	Grand Total.
Cataract.	67	152	53	272	1205
Irridectomy.	1	1	5	7	169
Petirigium.	..	6	4	10	510
Entropion.	..	..	4	4	526
Keratitis.	..	..	2	2	526
Hypopoin.	..	..	1	1	526
Opacity of Cornea.	..	..	1	1	526
Total.	68	159	70	297	1205

The Hyderabad Medical Association.

THE membership is open to all persons holding medical and Surgical qualifications according to the Western System and the chief object of the Association is to promote Medical and Surgical knowledge by reading of papers, demonstration of cases and discussions, in monthly meetings.

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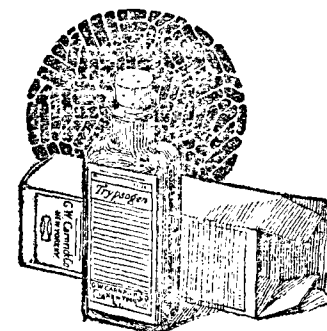
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The most effective treatment of this condition (Wiener Klinische Wochenschrift, Feb. 2, 1928) is rest in bed for from 8 to 14 days in a well-warmed room and with cutaneous counter-irritation.

Physicians find that applications of hot antiphlogistine placed over the lumbar region for two or three hours every day for about two weeks usually bring marked relief in these painful conditions. Warm full baths are to be avoided. A flannel binder should be worn during the attack and is recommended for a month afterward to prevent a recurrence of the attack!



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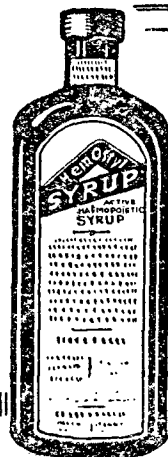
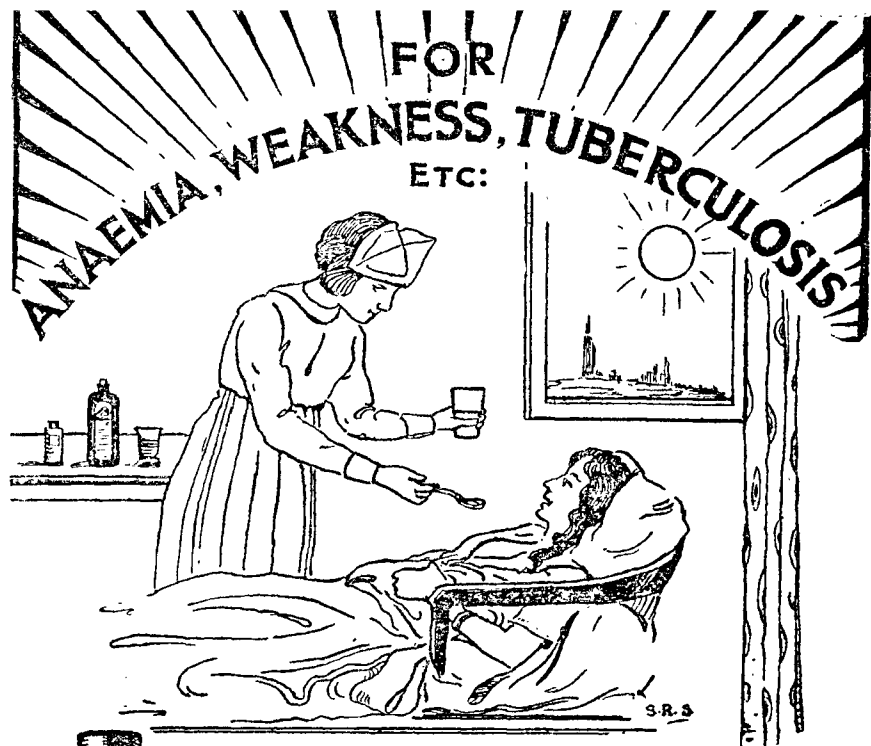
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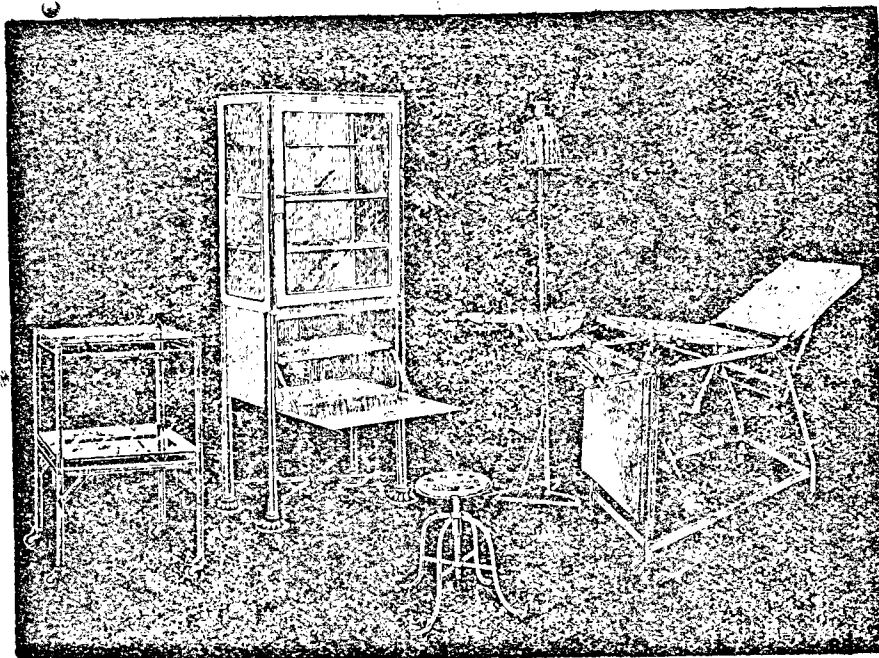
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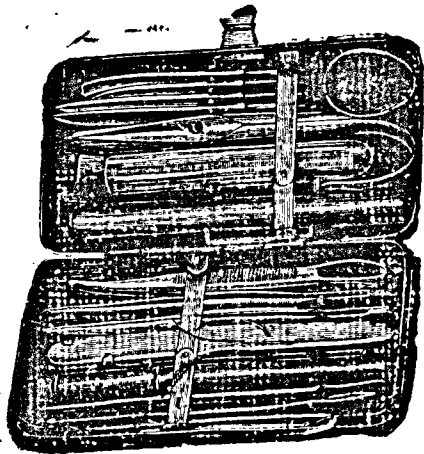
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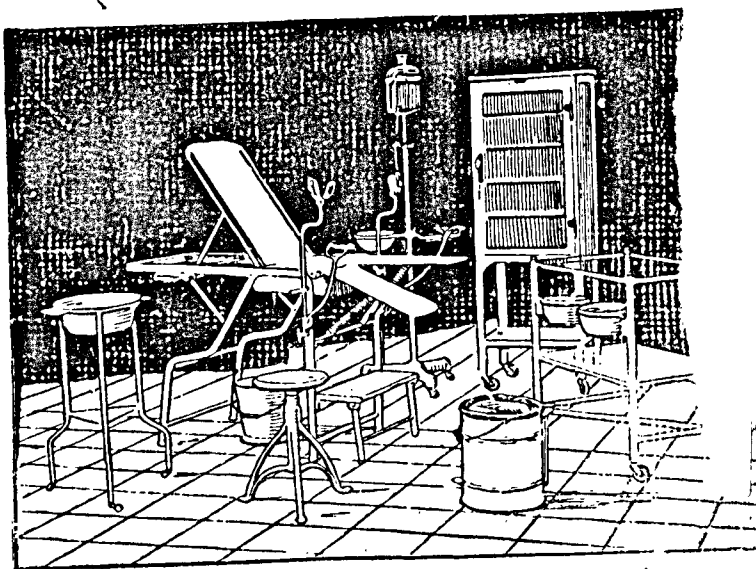
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