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MEDICO-SURGICAL SUGGESTIONS

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DISEASES OF THE PROSTATE GLAND AND THEIR TREATMENT

By MAJOR P. GOVINDA RAU, M.B.B.S., F.R.C.S. (E)

In the whole field of urology, diseases of the prostate are of special interest to the general practitioner, as the diagnosis of many affections of the prostate is within reach of the facilities available to him and treatment of some cases and alleviation of symptoms in other cases are also within his domain.

The prostate gland is the most important of the male accessory reproductive glands. It is a musculo-glandular organ lying at the neck of the bladder and enclosing within its structure the prostatic portion of the urethra and the ejaculatory ducts. Diseases of the prostate are fairly common, from the acute prostatitis both specific and nonspecific occurring in the young, to the benign and malignant enlargements of the prostate in the adult and the aged.

Anatomy and Physiology of the prostate.—The prostate is the size of an English walnut and weighs on an average about $4\frac{1}{2}$ drachms. Its measurements are on an average $1\frac{1}{4}$ " in length, 1" anteroposteriorly and $1\frac{1}{2}$ " transversely. The gland consists of five lobes—two lateral, a middle and an anterior and a posterior. There are two accessory structures (1) a group of tubules lying just under the mucosa at the bladder orifice and (2) the so called Home's gland located in the mucosa of the middle of the trigonum vesicae. The various lobes of the gland send their ducts to open into the posterior urethra.

The function of the prostate gland is the production of a chemical substance which dilutes the testicular and seminal vesicular secretions and separates and activates the spermatozoa. It also contains a specific enzyme which is said to coagulate the secretion of the seminal vesicles, thus favouring the retention of the seminal fluid within the female genital canal and thus promoting impregnation.

Prostatitis is a common disease. It is usually associated with inflammation of the posterior urethra, seminal vesicles, vesical neck, trigone or even the epididymes and should therefore be studied in relation to both the urinary and the genital tracts.

Acute Prostatitis is most frequently due to the *Gonococcus* when it comes under the attention of the venereologist, but there is also an acute

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prostatitis which is non-venereal and the most common organisms responsible for this are the staphylococcus albus and aureus, the streptococcus pyogenes and the colon bacillus. Contributory causes of prostatitis are masturbation over a protracted period, excessive sexual excitation without gratification, excessive sexual intercourse and coitus interruptus.

Infection reaches the prostate most commonly from the posterior urethra but it may be descending, as from the kidney, or blood borne, as from a primary focus in the sinuses, teeth or tonsils or again a complication of a condition like influenza.

An acute prostatitis may end in resolution, the formation of an abscess or chronic prostatitis.

Symptoms of acute prostatitis are those of the posterior urethritis with which it is most frequently associated—urgency, frequency, burning, pain during urination and there may be dribbling of urine. The prostate may swell up so much that there is retention of urine. There may be associated fever, and pain in the perineum, rectum, loins, penis or above the pubes may be present.

Diagnosis is easy. Rectal palpation reveals a hot enlarged gland and the second glass in the 2 glass test reveals a cloudy urine.

Treatment consists in rest in bed, avoidance of strain or sexual excitation, the application of heat in the form of hot sitz-baths or diathermy; bromide and diuretic mixtures may be given for relief of dysuria. Chemotherapy is of great value in shortening the acute stage of prostatic infections.

Chemotherapy is used in combating acute and chronic infections of the prostate as also in bladder and kidney affections occurring as a complication of enlargements of the prostate. It has been shown that sulphathiazole and sulphadiazine are of proved superior value in combating the common types of infection due to the Gonococcus, Staphylococcus aureus and albus, Bacillus coli and Bacillus Proteus. The sulfadruugs are concentrated in the urine 10 to 20 times more than the blood concentration and so heavy doses are not necessary.

The usual practice therefore is to give 2 gm. daily (in 4 doses) of sulfathiazole or sulfadiazine for from 6 to 10 days if tolerated well by the patient.

In cases of impending abscess, larger doses as 1 gm. 4 times a day may be found necessary. Penicillin is also useful in such infections. The method (at present adopted in New York Hospital) of giving Penicillin is to give 300,000 units twice a day until fever subsides and then one injection a day for two more days. This method is used in all cases where penicillin is indicated and saves a lot of trouble for patient, nurse and doctor and results have been very gratifying.

Chronic Prostatitis—Chronic inflammation of the prostate gland is a very common condition in adult males. When searching for the source of obscure infectious conditions, it is very important to examine this

gland. It is considered as second only to infected tonsils as a cause of arthritis. Chronic prostatitis is most frequently the sequel to an acute infection which may be caused by either the Gonococcus or other organisms. The nonspecific infection is common and may be direct extension from the urethra (as in non-specific posterior urethritis) or blood borne from focus in the tonsils, teeth or sinuses; or the aftermath of an acute systemic infection. The most common organisms in nonvenereal prostatitis are the colon bacillus and the staphylococcus, streptococcus and their subforms.

Symptoms—The most frequent complaints are of pain, a urethral discharge which may be profuse or merely the so called "morning drop" and some disturbance of sexual function, always accompanied by neuroaesthesia. The pain may be local or referred. Ordinarily it is located in the perineum, and may be described by the patient simply as a "heaviness" in the rectum. With this type there is generally a history of the passage of prostatic fluid on defecation. The pain may be referred down the back or inner sides of the legs, or into the groins, penis or sacrum. Frequent and painful urination, urgency, and difficulty are all common complaints, and result largely from involvement of the posterior urethra and bladder neck.

Diagnosis—The diagnosis of chronic prostatitis should be based on (1) Rectal palpation (2) repeated analyses of the voided urine (3) microscopic examination of the prostatic secretion and when necessary (4) urethroscopic examination of the posterior urethra and vesical neck. Rectal palpation may show no abnormality; ordinarily however there will be hard nodules and boggy spots between the areas of induration.

Microscopic examination of the prostatic fluid, expressed by massage is the only reliable method of diagnosing infection in the gland. Often the diagnosis must solely be on microscopic evidence of pus in the expressed secretions. Negative findings on one examination of the prostatic secretion are however, insufficient proof of the absence of infection, since pus in some instances does not make its appearance until after the prostate has been massaged from two to five times. The secretion expressed from the first massage may be from normal portions of the gland and two or more manipulations may be necessary to open the ducts from the diseased portions of the gland. Massage for diagnostic purposes must be carried out firmly enough to express the secretions but gently, as otherwise epididymitis may result.

Normal prostatic fluid is opalescent and viscid, and microscopically is seen to consist of corpora anylacea, lecithin globules and columnar epithelium. In chronic prostatitis the prostatic secretion is less opalescent and much of the lecithin is replaced by pus cells, often in clumps. Bacteria may be present in great numbers.

Urethroscopic examination may be necessary in some cases and the marked chronic inflammatory changes of the posterior urethra provides a clue to the underlying prostatitis.

Prognosis—The patient suffering from long standing prostatitis is not easily cured and so it is advisable to inform him at the outset of treatment.

Treatment—The main problem in the treatment of chronic prostatitis is the restitution of free drainage, since retention favours infection.

The most effective method of restoring the patency of prostatic and ejaculatory ducts is by a gradual, gentle but thorough dilatation of the prostatic urethra, and then only should massage be carried out. When carrying out dilatation the usual precepts about not using a sound below size 21 French or 12 English should be borne in mind. The dilatation is carried out once a week in the beginning and later once in two weeks and so on.

Prostatic Massage is carried out after each dilatation. It is best done upon a bladder partly filled with antiseptic solution. The best technique for massage is to have the patient on the table in the knee-elbow position and the surgeon with his gloved finger in the rectum carries out gentle pressure upon the lobes palpable, using downward stroking motions directed towards the urethra. Massage is also carried out first once a week and then once in two weeks and finally once a month.

Sulfadiazine or sulfathiazole is given in the small doses mentioned above and may be repeated for a second course 6 to 10 days after the lapse of a week or two.

Spices and alcohol should be omitted from the diet and constipation avoided.

Prostatic abscess is most frequently due to the *Gonococcus* but may also occur as a complication of systemic infection or may be secondary to superficial pyogenic infection such as carbuncles and boils.

Symptoms are, pain in the perineum, chills, fever and frequent and painful urination, which may progress to complete retention. Chronic abscess of the prostate may be present in which the only symptom is difficulty of urination.

Diagnosis is made out by rectal palpation when a tender soft prostate is felt. In chronic abscess tuberculosis should be kept in mind.

Prognosis—An untreated prostatic abscess may rupture into the urethra or rectum or it may burrow into the perineum, bladder or even the peritoneum with dire results. If proper drainage is carried out convalescence is usually satisfactory.

Treatment—Evacuation of a prostatic abscess through the posterior urethra is the method of choice. A sound is introduced into the urethra and a mid line incision made in the perineum. The urethra is exposed and incised upon the sound. The sound is removed the finger inserted into the prostatic urethra which breaks through the urethral wall into the abscess cavity, thus evacuating it. A drainage tube is inserted into the urethra for drainage of urine and pus and the skin is closed with interrupted sutures.

Evacuation of a prostatic abscess may also be done outside the urethra. An inverted V incision is made in the perineum, the intervening tissues carefully dissected apart and the prostate is exposed to view. It is then incised, pus evacuated and a drainage tube left in.

Tuberculosis of the prostate gland—Tuberculosis of the prostate is almost always a secondary involvement in a progressive tuberculosis which is extending throughout the genital tract. Between 50 to 70 per cent of all cases of tuberculosis of the male genital tract show involvement of the prostate.

Symptoms—With well walled off tubercles in the prostate there may be no untoward symptoms whatsoever. If the tubercles have coalesced and finally ruptured into the urethra there will be frequency, dysuria, hæmaturia and pyuria.

Rectal examination reveals a nodular elastic gland usually affected on one side only and differing from a carcinomatous condition in that it lacks the board like consistency usually associated with the latter. The secretion may show tubercle bacilli. Guinea pig inoculation of the secretion will clinch the diagnosis.

Treatment—In tuberculosis of the prostate, operation is contraindicated and radical removal of the genital tract is also inadvisable in patients with extensive involvement of the prostate.

Good results have been obtained with the usual hygienic, dietary and therapeutic measures advised for tuberculosis. Injections of gradually increasing doses of Koch's old tuberculin (beginning with 1 cc. 1 in 10 million dilution of Koch's old tuberculin) have been found to be of benefit.

Prostatic calculi—True prostatic calculi are fairly common, being like tiny seeds in the cleavage plane between the hypertrophied prostate and the compressed capsular tissue in benign enlargements of the prostate. These are rarely of clinical importance. Larger stones may form in connection with chronic inflammation.

Diagnosis may sometimes be suspected from rectal palpation, particularly when so marked as to allow crepitation on palpation. The diagnosis is usually incidental to X-ray for other conditions when radio opaque bodies are made out just behind and sometimes slightly above the upper part of the pubic symphysis.

Treatment is seldom necessary for prostatic calculi alone, as they seldom cause obstruction or symptoms. However they may act as foreign bodies in perpetuating infection when removal may be necessary.

Benign hypertrophy of the Prostate—Benign hypertrophy of the prostate gland occurs in from one third to one fifth of all men over 50 years of age. Many theories have been advanced as to why the prostate tends to hypertrophy with age. The chief are (1) that the hypertrophy is due to true tumour formation (2) that it is a fibroepithelial growth akin to myoma of the uterus (3) that the condition is a hyperplasia having its basis in chronic infection and (4) that the hyperplasia is endocrinopathic and due to an improper balance between male and female hormones.

Benign hypertrophy of the prostate most frequently occurs in the middle and lateral lobes, the anterior lobe being affected rarely and the posterior lobe practically never.

Symptoms—Enlargement of the prostate is usually an insidious disease which develops slowly and is marked by gradually increasing frequency and nocturia (nocturnal frequency). This nocturia is characteristic. The patient rests for four or five hours during the first part of the night, is disturbed at 2 or 3 a.m. and may pass water several times after this. The stream is slow in starting and lacks force. Men take this as evidence of advancing age and often pay little attention to it, occasionally hæmaturia may occur and acute retention may suddenly supervene in a patient when there have been no previous symptoms. Most patients with enlargements of the prostate have decreased libido and impotence. Occasionally however attacks of priapism occur due to the congestion of the Verumontanum.

Diagnosis—The patient should be given a careful general and special examination, including rectal palpation, an estimation of the amount of residual urine, determination of the renal function and examination with a McCarthy's Panendoscope to ascertain the exact nature of the enlargement. The size of the prostate as felt per rectum has nothing to do with its obstructiveness and cystoscopy (or endoscopy) or cysto-urethrography is essential to determine the extent of the intravesical and intraurethral intrusion.

Treatment—Many patients with benign hypertrophy of the prostate have no obstructive symptoms and do not require surgery. In benign hypertrophy without residual urine, palliative treatment is usually advisable, this consists of periodic prostatic massage urethral dilations, urethrovaginal irrigations and when there is urinary infection, suitable chemotherapy.

Hormonal therapy is still in the experimental stage. Some investigators believe that in prostatic hypertrophy there is an improper balance between the male and the female (or the oestrogenic) hormones. In the normal male under the prostatic age, the two sex hormones are supposedly in balance with each other, and with the hormones of the Pituitary and the adrenals. According to these people, benign hypertrophy is due to a deficiency of the male hormone so that the hyperplastic activity of the female (or the oestrogenic) hormones is not inhibited. Hence administration of the male hormone (or testosterone propionate) is carried out.

Contrary to the above view is the opinion that benign hypertrophy is due to a deficiency of the female hormone, normally present in males. Treatment of 28 patients with large doses estrone (or female hormone) resulted in marked improvement. It is considered by some that the benefit obtained by treatment with male hormone is only due to the stimulation of the detrusor muscle and continued treatment may not only do no good but may be actually harmful.

X-ray therapy of benign hypertrophy is an accepted procedure. There is definite alleviation of associated congestion and oedema in selected cases. Residual urine becomes diminished and patient is relieved for a considerable time. However, it is no substitute for prostatectomy or resection.

Surgical treatment is usually required in cases with residual urine.

Before carrying out surgical treatment preliminary examination and preparation are of the greatest importance.

A blood urea estimation gives a rough indication whether preliminary drainage is necessary or not. When the blood urea level is below 20 mgm per 100 c. c. prostatectomy can be carried out at once. When it is above 20, drainage by either an indwelling urethral catheter (the method most commonly adopted at the New York Hospital) or the usual suprapubic cystotomy is carried out and maintained, until the blood urea settles down. In cases where the blood urea is higher than 50 or 100 mgm per 100 c. c. lengthy drainage is necessary before the patient is fit for operation. At present with blood transfusions when necessary, and sulfanilamide and penicillin, it is possible to operate on patients with blood urea levels up to 50 mgm per 100 c. c.

In addition to blood urea estimation, a preliminary plain X-ray is always taken to determine the size, conformation and location of the kidneys. Routine cysto-urethrograms are also carried out and they give an idea of the size of the prostate. An electrocardiogram is essential and blood pressure estimations are most helpful. In the presence of infection chemotherapy as mentioned above is necessary,

The operative methods available are :—

1. Trans-urethral resection.
2. The punch method as practised at the Mayo Clinic.
3. The Suprapubic prostatectomy.
4. The Perineal and
5. The Retropubic.

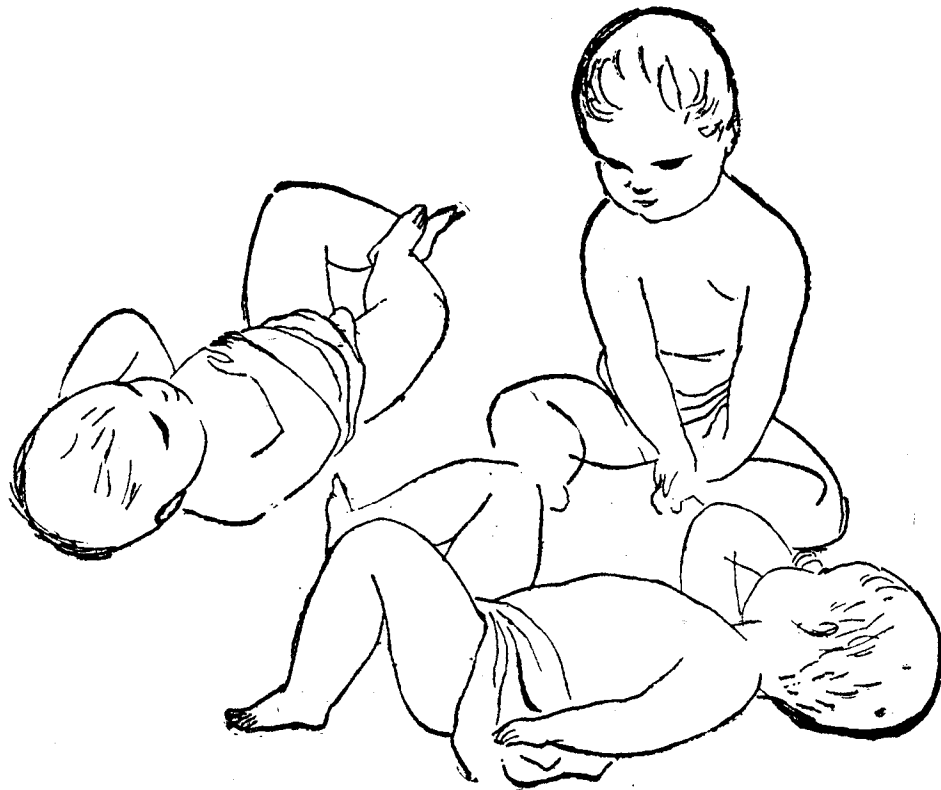
Trans-urethral resection requires costly equipment and a long period of training is necessary before good results are possible, even in the best hands operation has to be repeated twice and sometimes thrice to get rid of all the obstruction, and severe strictures requiring long and continued treatment, occur fairly often after trans-urethral methods. On the other hand resection can be undertaken in people who are not fit subjects for any other type of operation, and in median bars and such like obstructions, trans-urethral methods provide an easy way of relieving obstruction.

The Punch method has its adherents in only those of the Mayo Clinic and their disciples.

The Suprapubic operation is the usual method adopted.

The Perineal is considered best for early carcinoma for total prostatectomy.

The Retropubic method as revived by Terence Millen of London has been advocated by him as the ideal approach for the prostate. The



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method has been modified at the New York Hospital and the results upto date have been very gratifying.

Contracture of the bladder neck and median bar formation—These are common and were once referred to as "Prostatism sans prostate" because in spite of prostatism, (prostatism is the clinical situation resulting from urinary obstruction, which is located at the bladder outlet) the gland is not enlarged, in fact, is usually small. Fibrosis of the bladder neck and prostate most often due to chronic inflammation, is the cause. These occur in an earlier age group than benign hypertrophy—generally about forty. Contracture means a general constriction and immobility of the outlet while bar formation is a more localized process producing a ridge across the floor of the outlet which looks not like a dam endoscopically. The diagnosis depends on the history, the general examination and is clinched on endoscopic examination. The principal method of treatment is trans-urethral resection. The Retropubic approach can also be used, a longitudinal incision made over the capsule of the prostate, the vesical neck is exposed and a V shaped section of the posterior lip of the vesical orifice carried out, with relief of the obstruction.

Carcinoma of the prostate gland—According to Young a fifth of the male patients who seek relief of obstruction of the vesical neck have carcinoma of the prostate. In autopsies on men over 50 years of age carried out at the Johns Hospital, 14 per cent showed carcinoma of the prostate and of these 68 per cent had not shown any symptoms. In only 5 per cent of cases is diagnosis made sufficiently early to carry out successful treatment. However, suffering may be relieved and life prolonged for varying periods by methods at present at our disposal.

As with carcinoma elsewhere, aetiology of prostatic cancer is unknown but a very important relationship has been shown between androgenic hormones and carcinoma of the prostate.

Prostatic carcinoma is insidious and slow growing and may remain confined to the prostate for long periods but there is a high frequency of skeletal metastases in carcinoma of the prostate and the pelvis and the lumbar vertebrae are especially affected.

Symptoms—Disturbances of urination, as in benign hypertrophy, are usually the first symptoms, but advanced carcinoma may be present without urinary symptoms, as cancer of the prostate most often arises in the posterior lobe of the prostate. Pain referred to the sacroiliac regions, penis, and scrotum is a relatively early symptom in 65 per cent of the cases and may be due to bone metastases.

Diagnosis—Responsibility for the early recognition of prostatic cancer falls primarily on the general practitioner. A routine check up of men over 45 years of age should include a rectal examination and this is of special importance when patients give a history of urinary disturbances.

Rectal palpation reveals a board-like hardness of the prostate. In some cases a single hard nodule may be felt. Occasionally the nodule has sharp edges—a point specially characteristic of cancer.

X-ray examination of the pelvis and lumbar vertebrae reveals spotty areas of increased density in the presence of metastases.

There is one important procedure useful in diagnosis as well as in suggesting the line of treatment in cancer of the prostate, i.e., estimation of the serum acid phosphatase. In cancer of the prostate with skeletal metastases, serum acid phosphate is greatly increased. When these high levels are present indicating metastases, radical prostatectomy is inadvisable, but employment of hormonal therapy is indicated.

Prognosis—Although very few cases are seen at a sufficiently early stage to warrant radical prostatectomy, with present methods of treatment suffering can be alleviated and death postponed for a number of years.

Treatment—Total prostatectomy by the perineal or by the new retro-pubic method or partial prostatectomy by supra-pubic or trans-urethral methods may be carried out according to the stage of the growth.

Radon seeds can be implanted in the prostate after a conservative perineal prostatectomy. Surface application of Radium, interstitial application of radon and deep x-ray therapy help in some cases.

Hormonal therapy—Dr. Huggins of Chicago was the first to show that carcinoma of the prostate is composed of epithelial cells of a mature type which like adult prostatic epithelium is responsive to depression of the level of androgenic hormones in the organism. Eliminating the gonadal androgens by castration or supervoltage irradiation of the testicles or neutralizing their activity by oestrogen therapy, decreased the activity of the prostate neoplasm.

In 21 patients who were castrated, 15 showed great clinical improvement as evidenced by marked diminution of pain, increase in body weight shrinkage of the primary growth and regression of bony metastases.

Administration of the female sex hormone is routine treatment in cancer prostate with skeletal metastases. This inactivates the gonadal androgens and sometimes even after castration, it is given with the object of dealing with androgenic hormones still present.

Castration or oestrogenic therapy is not curative of cancer and in early cases the only correct treatment is total prostatectomy.

In conclusion, it may be said that it has not been possible to cover all the diseases, nor mention all the points in connection with them. An attempt has been made to give an idea of the present tendencies with regard to diseases of the prostate and their treatment.

Extracts.

ENTERIC FEVER.

By J. C. PATEL, EL AL.

The authors review 100 cases of fevers out of which 51 were enteric cases, 37 of these being culture-positive. The remaining 14 were serologically positive. The 36 cases were clinically typical, one was atypical, the patient being ambulatory throughout the course. Treatment with purgative and quinine during the first week seemed to produce adverse effect on the subsequent course of the disease. 11 cases were treated with purgatives and four died, six were treated with quinine and four died: while in 20 cases treated in other ways only, two died. This adverse effect of purgative and quinine has been noted by others also. Complications developed in 24 cases. In 18 cases there were one or more of the major complications like hæmorrhage, perforation, pneumonia and hyperpyrexia. Hyperpyrexia was seen in six cases, intestinal hæmorrhage in seven. The culture positive cases were treated by two different methods:—

1. Massive doses of penicillin and sulfathiazole.
2. Bacteriophage.

The Diet: Every patient was given a basic diet consisting of 120—150 ozs. of fluids per day including milk, tea, fruit, juice and water. He gets about 40 ozs. of milk or butter milk, 12—16 ozs. of fruit juice, 16 ozs. of tea, egg preparations and as many bannanas as he can take. The same diet was given successfully in 2 cases that developed hæmorrhage. If diarrhoea or distension developed, the only change in diet made is that milk is changed to butter milk and fruit juice stopped, but the solid food is continued unchanged. The diet generally provided 1000—1500 calories. Patients were encouraged to drink large quantities of fluids. As soon as the patient feels better and starts taking interest in his surroundings, he is encouraged and coaxed to take the full diet in spite of fever. The patient is able to sit up in bed within a day or two of his temperature becoming normal and is able to walk about the eighth day of convalescence. The fatality rate among the 37 culture-positive cases has been 27 per cent. In 9 cases bacteriophage treatment was tried in only two cases there was the expected immediate improvement. 18 cases were treated with penicillin. In 11 cases penicillin and sulfathiazole were given as follows:—

An initial dose of 2 gms. of sulfathiazole was given, followed by 1.5 gms. 4 hourly day and night for five days. Penicillin 2 lac units was given intramuscularly every two hours until a total dosage of 10-12 million units is administered. Penicillin alone was used in 2 cases. In two the temperature touched normal and remained normal at the end of treatment. In two other cases the combined treatment brought the morning temperature down to normal, but it was 6—8 days before the evening temperature became normal. In one case penicillin alone had a similar effect.

In one case the first course of combined treatment had no effect and blood culture remained positive at the end of it. However, the second course of 5 days brought down the temperature to normal. One patient was severely ill and semi-conscious and was administered the combined course. There was marked improvement in general condition, rowdyism and toxicity, but the temperature ran its course for five weeks before touching normal. One female patient was given penicillin alone. There was marked improvement in the general condition and the temperature remained normal for a day. But later she died of perforation. Two cases who were given combined treatment died of hyperpyrexia. Another died of pneumonia and pulmonary edema during the combined treatment.

(Abstracted from the Indian Physician).

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THE MODERN TREATMENT OF CEREBROSPINAL FEVER.

By L. J. M. LAURENT, M.D., M.R.C.P.

Under the term cerebrospinal fever are included all manifestations of disease due to the meningococcus.

Varieties :—1. Acute septicæmic form. A purpuric rash is always present. Signs of meningitis are absent. The cerebrospinal fluid is clear and sterile.

2. The common meningitic form. Well marked signs of meningitis are present. Rash may or may not be present. The cerebrospinal fluid is turbid and contains meningococci. The patient is conscious or delirious but not comatose. His B.P. is normal.

3. The meningitic form with encephalitis. After 24 hours, or so of the illness the patient becomes deeply comatose, unresponsive to stimuli and has stertorous breathing.

4. Post-basic meningitis. This is seen in infants. It is a mild form and head retraction is present.

5. Acute septicæmic with adronal syndrome. It is usually seen in children. Large purpuric spots are present, the B.P. is very low and so the pulse is almost imperceptible. There is frequent vomiting with cyanosis and air hunger. Death usually comes within 24 hours of the onset.

6. Fulminant septicæmic form. This form may be fatal in 1.8 days from circulatory failure.

7. Chronic septicæmic form. The patient has red nodules and bouts of pyrexia for weeks.

8. Catarrhal rhino-pharyngitis. A condition little more than a carrier state.

Diagnosis : In every case the clinical diagnosis must be confirmed by examination of the cerebrospinal fluid and by blood culture.

Lumbar puncture : Lumbar puncture is necessary for diagnosis. It may be done under local anæsthesia, but in few cases general anæsthetic may be required. Lumbar puncture is repeated when a patient is unconscious or fails to improve after 48 hours' treatment; if there are convulsions, and if there is some doubt about the diagnosis.

Specific treatment of an uncomplicated case :—Sulfonamides: Sulfanilamide and sulfapyridine though effective, have been given up due to toxic effects. Sulfathiazole, sulfadiazine or sulfamezathine can be chosen almost indiscriminately.

1. The initial or loading dose. This should be large with the object of obtaining rapidly a high concentration of the drug in the blood and cerebrospinal fluid. It is best given intravenously even in conscious patients. About 2 gm. at a time is enough.

2. The next dose is given by mouth within the next two hours and thereby regularly every four hours by day and night until there is manifest improvement. In practice this means that Kernig's sign has become negative, the neck is no longer stiff and the patient can chin his

knees easily. This usually occurs in 3—5 days' treatment. Thereafter the drug is given at six-hourly intervals, for another two days and finally discontinued. The course of treatment should never be less than five days and rarely longer than eight days.

3. With every four-hourly dose of the drug alkaline mixture containing 20 grs. each of soda bicarb and soda citras should be given as an adult dose. When the urine becomes alkaline only half the dose is given. With six hourly doses and with sulfanilamide this alkaline mixture is not required.

4. The adult patients should be made to drink at least five pint of fluids of all kinds daily while taking four-hourly doses of the drug. The children should be given correspondingly smaller quantities.

Dosage:—At the age of three months the initial dose is 0.5 gm. then 0.5 gm. every four hours for 2 days and subsequently 0.25 gm. every six hours. Dosage from 3 months to 3 years 0.75 gm. 0.5 gm. and 0.5 gm. respectively. Dosage from 3-10 years 1.0 gm. 0.75 gm. and 0.5 gm. respectively, dosage from 10-15 years 1.5 gm. 1.0 gm. and 0.75 gm. respectively. Dosage above 16 years 2.0 gm. and 1.0 gm. and 1.5 gm. respectively. If parenteral injections are required, 'soluthiazole' should be used as it is a neutral solution which can be given intravenously or intramuscularly 5 c.c. are equivalent to 1 gm. of sulfathiazole.

Penicillin:—Intramuscular injections every three hours are given. But intrathecal injections are also needed. A single dose intrathecally should not exceed 10,000 units of even the purest brand. In infants these injections may cause convulsion, collapse and apnea. Penicillin should be reserved for 1. cases of kidney disease and urinary obstruction; 2 cases of congestive heart failure which need restriction of fluids.

It is better to combine intramuscular penicillin with full doses of sulfanilamide orally.

Treatment of Special Cases.

1. Patients in Coma. Keep flat with the mouth and air way clear. Feed and give all fluids by nasal feeds four hourly. Sulphonamide treatment is carried out by intramuscular injections. Catheterise the bladder and tie in a catheter. Do a lumbar puncture. Give intravenous injections of 50 per cent glucose in doses of 40 c.c. 2-3 times a day.

2. Delirious and restless patients: Paraldehyde and somnifaine are given to adults. To children syrup of chloral. Morphine and opium are best avoided.

3. Patients with adrenal syndrome: 'Eucortone' in doses of first 10 c.c. and then 5 c.c. every six hours should be tried until the blood pressure rises. Oxygen tent is used for children. Intravenous drip of 2½ per cent glucose in normal saline may be used.

(Abstracted from the Medical Press).

SYPHILIS.

By F. W. REYNOLDS & J. E. MOORE.

Bismuth Preparations: Three compounds were studied by Eagle:

1. Sodium and potassium bismuthotartrate. Water soluble.
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These preparations are from ½ - 2/3rd as active therapeutically as oxophenarsine hydrochloride.

Bismuth gingivostomatitis rarely occurs when there is good oral hygiene.

Arsenical Preparations: Dichlorophenarsine hydrochloride is safe and effective arsenical.

Tryparsamide is not to be used as it may damage vision and is of doubtful value.

Toxicity of Arsenical Compounds: Death after neoarsphenamine 1 to every 35,861 injections, after oxophenarsine hydrochloride 1 to every 159, 410 injections. Death results from hæmorrhagic encephalitis and exfoliative dermatitis.

Arsenical Encephalopathy: Out of 75 cases described in literature 57 were fatal. Usually the symptoms occurred after the second or third injections. The initial symptom is intense headache, accompanied by fever, shivering and vomiting. The next day there is epileptiform convulsion, with clonic spasm, followed by unconsciousness. Often death supervenes within 48 hours of the onset of the symptoms. Treatment is unsatisfactory. Bal is worth trying.

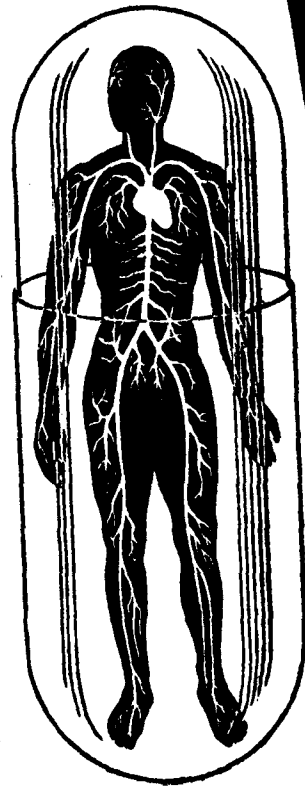
Agranulocytosis: It was seen to occur towards the end of the scheduled course of treatment.

Semi Intensive Arsenobismuth Therapy of Early Syphilis: This consists of administration of forty injections of oxophenarsine hydrochloride and sixteen of bismuth subsalicylate in a period of twenty six weeks. The results of this therapy were assessed by Leifer. An apparently favourable outcome was observed in 9.83 per cent of the sero-negative primary cases and 94.5 per cent of the sero-positive primary cases and 89.3 per cent of the secondary cases. The relapse rate was 4.6 per cent cerebrospinal fluid was abnormal only in 18 cases out of the total.

Death rate was 1 in 38,000 cases treated. But toxic effects necessitated the termination of therapy in 4.8 per cent of the cases.

Penicillin in Early Syphilis. Sodium penicillin in aqueous solution is used by the intramuscular route every 2-3 hours for a minimum of 7½-8 days.

The minimum dose for seronegative primary syphilis should not be less than 3.6 million units; for seropositive primary and early secondary syphilis, not less than 5.4 million units.



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Relapse: For a first relapse. Above penicillin course is supplemented with six intravenous injections of 60 mgm. each of mapharsen plus six intramuscular injections of bismuth subsalicylate 0.2 gm. each twice a week.

For a second relapse, penicillin is omitted entirely and a 26 week course of 40 injections of mapharsen and 16 of bismuth subsalicylates is carried out.

Penicillin In Beeswax and Oil; A single injection of 6 lacs will produce a therapeutically active blood level for from 20-28 hours. This injection is given daily for 8-15 days in early syphilis. Bismuth and Arsenic as supplements to penicillin should be reserved for relapse cases.

Results: Sternberg and Leifer have analysed the Army records of 1400 soldiers with early syphilis treated with 2,400,000 units of penicillin in aqueous solution given in divided doses over a period of 7.5 days. Satisfactory progress was observed in 94.3 per cent of 600 cases of seronegative primary syphilis, in 89. per cent of 564 cases of seropositive primary syphilis and 88.9 per cent of 286 cases of secondary syphilis. The cerebrospinal fluid was normal in 714 out of 719 cases.

Penicillin In Late Syphilis: Benign late syphilis. Visceral (hepatic) gummas heal promptly under therapy with penicillin. This form of therapy is at least as effective in the treatment of visceral gummas as other forms of antisyphilitic treatment, with the additional advantage of being completely nontoxic.

Cardiovascular Syphilis: Russek treated 15 patients with cardiovascular syphilis, including 4 with aortic aneurysm with penicillin in relatively large doses (40,000 units every two hours). Significant reactions necessitating discontinuance of the use of the drug were not encountered.

Jarisch-Herxheimer reactions were rare. Four of the 15 patients in the present series showed distinct improvement in coronary reserve after treatment.

Penicillin in Neurosyphilis: Gammon, Stokes et al report in 161 patients with various manifestations of syphilis of the central nervous system the greatest effect on symptoms and signs occurred with respect to the psychoses, the incoordination, the tremors and the speech defects of dementia paralytica and the lightening pains of tabes dorsalis. Abnormalities of the spinal fluid responded dramatically. Within two years the spinal fluid in 62% of their patients were normal or 'near normal'. In all types of neurosyphilis the outstanding therapeutic response was a gain in weight, usually accompanied with a sense of increased well being. In dementia paralytica, mental symptoms improved in 72% of 47 patients, the most notable results being observed in those with severe symptoms of short duration. In juvenile paresis, improved mental status was noted less frequently. There were 40 patients of dementia paralytica with tabetic pains and of these 38 (82%) were 'improved'. In the relief of tabetic pain the results of penicillin therapy compare favourably with results of other forms of therapy. In other symptoms of tabes no striking change were noted. In meningovascular and asymptomatic neurosyphilis,

the results were measured by the response of the spinal fluid. Fourteen patients had convulsions, a symptom which failed to improve notably after penicillin therapy.

Neural Herxheimer reactions were noted. Transient increase in mental abnormalities developed in 6 paretic patients, 4 with tabes dorsalis had exacerbations of their pains and in two with spinal syphilis, there developed transverse myelitis.

Koteen, Doty, Webster and Mc Dermott call penicillin therapy on neurosyphilis equal to fever therapy. They treated with penicillin alone 111 patients with various forms of neurosyphilis. In 6 out of 11 with dementia paralytica there were satisfactory remissions that have been sustained approximately for two years. There was subjective improvement in 38 out of 41 patients with tabes dorsalis. In meningovascular neurosyphilis the results after penicillin were comparable to those usually observed after arsenotherapy. The treatment had a striking effect on abnormalities of the cerebrospinal fluid.

Heyman treated 141 patients with syphilis of the central nervous system with penicillin. With 40 lac units of penicillin, 'arrest' of the neurosyphilitic process was obtained in approximately 85% of the cases. Results in early cases of neurosyphilis are better. Heyman recommends penicillin for the treatment of early neurosyphilis and late asymptomatic neurosyphilis.

Penicillin in Asymptomatic Neurosyphilis: Penicillin exerts a profoundly favourable effect on spinal fluid abnormalities in asymptomatic neurosyphilis. This effect is manifest, in order of promptitude and extent on all counts, protein content, colloidal test and a Wassermann reaction. Reappearance of increased cell count, increased protein or both is an indication for retreatment.

Penicillin In Dementia Paralytica. Penicillin with malaria gives better results than penicillin alone 41 patients with dementia were treated with penicillin alone, 17 with penicillin and malarial therapy. Penicillin alone, in doses ranging from 20 lacs to 100 lacs produced at least some degree of clinical improvement in 46% of cases. The response was better among patients whose symptoms were of recent onset. No patient whose symptoms had been present for a year or more prior to the institution of penicillin treatment showed any clinical improvement. Penicillin with malarial therapy caused improvement in 58% of cases.

Penicillin in Abnormalities of the C. S. Fluid. Reynolds describes the results of 149 patients with late neurosyphilis treated with penicillin or penicillin with malaria. Improvement in the spinal fluid generally was apparent. As a rule the cell count and protein content promptly became normal and remained so. Colloidal gold test, Wassermann reaction improved more gradually, the improvement usually being well sustained.

Neural Jarisch Herxheimer Reaction: Tucker and Robinson reported reactions in 26% out of 208 patients. The majority of these reactions

were febrile, but transitory mental confusion or agitation also occurred. In 2 cases paranoid trends and in 2 convulsion developed.

Penicillin in Prenatal Syphilis: penicillin is the ideal drug for prenatal syphilis. It proved efficacious even when administered late in the course of pregnancy.

Penicillin in the Treatment of Syphilis in Children: Yampolasky treated 32 infants with penicillin and satisfactory results were obtained in 23 of them. An immediate clinical response was observed in all patients and healing of cutaneous and mucosal lesions often occurred by the end of the course of treatment. Osseous lesions also showed prompt improvement, but complete resolution usually did not occur for 2-3 months. Penicillin failed in interstitial keratitis, Clutton's arthritis and Juvenile paresis. In late congenital asymptomatic neurosyphilis, the results were satisfactory. The results in children depend on the nature and duration of the disease. Penicillin appeared of little or no value in the treatment of the late manifestation of the disease. The total dosage of penicillin sodium be not less than 75,000 units per pound of body weight and the course of therapy not less than fifteen days. Platou et al treated 252 infants with penicillin in a single course administered over a period of 7½-15 days. Satisfactory results were present in 78% unsatisfactory in 9.1% and uncertain in 17%. Dosage of more than 40,000 units per kilogram of body weight yielded better results. Most babies became seronegative between the 4th 12th months after therapy. They recommend at least 1 lac units per kilogram of body weight divided in 120 intramuscular injections given every three hours over a period of 12-15 days.

(Abstracted from the Archives of Internal Medicine).

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TREATMENT OF MALARIA

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Malignant Tertian Malaria: 0.3 Gm. twice daily for ten days. In severe infections the dose may be increased up to 0.5 to 0.6 Gm. twice daily for the first three days.

Benign Tertian and Quartan Malaria: 0.3 Gm. daily until the fever subsides, followed by 0.1 Gm. every three days, for six months. This after-treatment favours the eradication of the persistent forms of the parasite and thus reduces the possibility of relapse.

CLINICAL CURE

All types of Malaria: For the control of the acute attack, 0.3 Gm. daily until the fever subsides.

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Correspondence.

To

The Editor,

Medico Surgical Suggestions,

Madras.

Dear Sir,

Certain changes in the dosage of 'Paludrine' for the treatment of malaria have been announced in the medical press this month and I would be glad of the hospitality of your correspondence columns in order that I may amplify this announcement for the information of the Medical Profession in India.

After the original clinical trials of 'Paludrine' by professor N. Hamilton Fairley in Australia, by far the most extensive clinical trials were carried out in India and Pakistan under the auspices of the Malaria Institute of India in the years 1946-47. The results of these trials together with Professor Hamilton Fairley's published work led to the dosage recommendations contained in our present literature (leaflet Pal. 1 June 1947 and brochure Pal. 2 dated July 1947). Concurrent with clinical trials in India, however, investigations were carried out in many other countries, and, in the reports from Africa particularly, it was apparent that certain strains of the falciparum parasite in Africa required a heavier dosage of 'Paludrine' than was recommended in our literature. A few workers in India also were of the same opinion and felt that in certain cases of severe infection with some Indian strains of both the P. falciparum and P. vivax parasites, a heavier dosage was advisable.

Amongst the trials set up in Africa were trials on the prophylactic aspect of 'Paludrine' for protection against malaria in endemic and hyperendemic areas. Some of the individuals treated in these trials developed malaria in spite of taking one tablet (0.1 Gm.) twice-weekly at spaced intervals. Apart from these occasional "break-throughs", some doctors in Africa thought that a daily dose of one tablet (0.1 Gm.) was easier for the individual to remember than a twice-weekly dosage at evenly spaced intervals. They thought that taking one 'Paludrine' tablet (0.1 Gm.) daily would be easier, and would obviate the occasional "break-throughs" that had occurred in their trials.

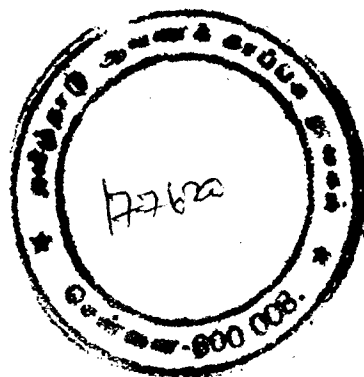
In deference to these views, we have therefore amended our recommendations for the treatment of malaria and these recommendations are as follows:—

(1) For Radical Cure, 0.3 Gm. twice daily for 10 days. In the case of severe infections the dose may be increased up to 0.5 or 0.6

Gm. twice daily for the first three days. (2) For a Clinical Cure, that is to say, control of the acute attack of malaria, a single daily dose of 0.8 Gm. should be given until the fever subsides. This is the dose best suited for malaria arising in rural areas, (which has been called by some Indian writers "Village Malaria") where medical attention is not always easily obtainable. In the case of protection against malaria, however, although we have carefully considered the reports from Africa we do not think that we are justified in recommending the Medical Profession in India to adopt the daily dosage of one tablet (0.1 Gm.) for prophylactic purposes. There are many well controlled Indian trials, published, and unpublished, which show conclusively that on a dose of one tablet (0.1 Gm.) twice-weekly at evenly spaced intervals, or three tablets (0.8 Gm.) once-weekly the incidence of malaria can be reduced to almost negligible proportions, provided the administration of the drug is regularly maintained during the transmission season. We therefore do not purpose to make any alteration in our recommendations for prophylaxis and protection against malaria.

Full details of the new recommendations have been given in our advertisement in your journal and I am writing this letter in the hope that these remarks will be of interest to the Medical Profession when considering the new dosage. The matter has not been decided by my Company alone, and the Medical Profession in India may rest assured that the dosage we are now advising in our literature and advertisements has the support of the highest authorities on malaria, both in India and Pakistan and the U. K.

Yours faithfully,
(J. M. MUNGAVIN, M.B., B.Ch. (Cantab),
Medical Service Department, I.C.I., India, Ltd.,
Calcutta-1.



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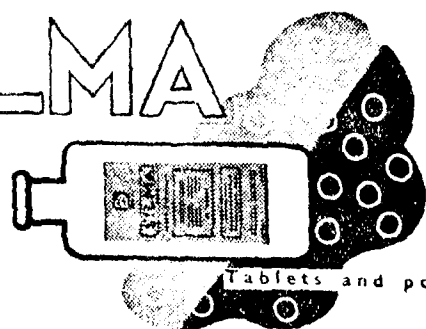
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MEDICO-SURGICAL SUGGESTIONS

(Established 1932.)

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MADRAS

STOMATITIS IN CHILDREN

G. COELHO*

Stomatitis is a generalised or localised inflammation of the mucous-membrane of the mouth including the lips, gums, inner side of the cheeks and the tongue,

This inflammation is due to various causes, some of which are known. It is not possible, however, to determine the cause in many cases.

The known causes are :—

- A. Infections : (1) virus (2) fungus (3) anærobic organisms (4) associated with an upper respiratory tract infection (5) associated with measles (6) diphtheria (7) pneumococcus, streptococcus (8) multiple organisms.
- B. Vitamin deficiencies—vitamin B complex, vitamin C, singly or combined.
- C. Allergic.
- D. Blood disorders—agranulocytosis.
- E. Traumatic—injury with toys—drinking hot liquids.
- F. Constitutional—glossitis marginans.

CLINICAL PICTURE

Stomatitis without Ulcers.—The tip of the tongue is red, the gums are swollen, there is excessive salivation. There is a bad smell from the mouth. When the child is old enough, he may complain of a bad taste in the mouth. The children may dislike taking food.

Stomatitis with few "herpetic" ulcers.—In addition to the above signs and symptoms which are present in rather severe degree, vesicles are present. They are found on the tongue, lips and inner side of cheeks. They may be single or in groups. The vesicles may rupture and leave small, superficial ulcers with an yellowish exudate. There may be fever. The child is irritable. That acute gingivostomatitis is caused by the herpetic virus is now proved. Youmans was one of the first, in 1932, to demonstrate it. In 1938 Dodd, Johnson and Buddnigh reported twelve cases in which they had conclusively proved herpetic virus was the cause by inoculating into the cornea of rabbits. In 1942 Black published a report on 45 cases with identical conclusions after the same method of experimental investigation.

Stomatitis with severe gingivitis with extensive ulceration.—This is a serious condition. The gums are swollen, bluish purple in colour, bleed

*From the Department of Pediatrics, Sir J. J. Hospital, Bombay.

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easily. The edges of the gums are ulcerated. Ulcers are also present on the edges and dorsal surface of the tongue. They are larger than in the previous condition. They are also present on the inner side of the cheeks spreading towards the angle of the mouth. A cauliflower-like collection of debris may be seen at the angle of the mouth. Opening of the mouth is difficult and is associated with pain and bleeding from the angle of the mouth. Eating of any food and swallowing is very painful and the child refuses food. There is excessive salivation and thick saliva drips from the half-open mouth. The mouth smells very badly; the observer often feels sick with the smell. The submental and submaxillary glands are enlarged and quite tender. The child is fretful and looks miserable. There is usually fever ranging from 99° F to 103° F. Smears from the ulcers usually show spirillæ, bacillus fusiformis and in some cases pneumococci and streptococci besides pus cells. The role of these organisms is still a matter of controversy. A healthy child usually harbours in his mouth one or other type of the aerobic or anaerobic organisms. The oral spirochætes, fusiform bacillus, spirillæ or vibrios, singly or in combination, are not capable of initiating infection or a pathological lesion in man. But material obtained from the lesions of Vincent's angina is able to produce pathologic lesions in experimental animals. This material, however, contains streptococci or staphylococci. It is, therefore, possible that though the aerobic organisms normally live a commensal existence in the human mouth, yet when the tissue has been changed by the action of the streptococci or staphylococci, they multiply there in large numbers and thus present the characteristic clinical picture. Their action is thus subordinate to the aerobic bacteria.

The ulcers may be covered with a thick membrane which peels off easily. This membrane may spread over the tongue, inner side of the cheek, the pillars of the tonsillar fossa, the tonsils, and the pharynx, though not in a continuous sheet.

Thrush is caused by the fungus *Oidium albicans*, which was discovered by Burg in 1841. A dry mouth is an essential prerequisite for its development. This condition is met with physiologically in the newborn and pathologically in the dehydrated and in the seriously ill child. It is generally seen in debilitated infants, particularly in the first 2 months of life. Those suffering from acute gastroenteritis, chronic diarrhoea, chronic fever may end up with this condition. It also spreads as an epidemic in badly kept nurseries. The dryness of the mouth is followed by desquamation of the surface of the tongue which looks red and shiny. Then thrush appears as white patches, like bits of milk, alone first on the tongue, then on the inner side of the cheeks, the palate and lips. The patches vary from a pinhead to a half-anna piece. On removing the patches, the mucous membrane is red and bleeds easily though there is no ulceration. These may fuse and form an extensive patch of varying thickness and irregular margins covering the entire palate, inner side of the cheek and a portion of the tongue. The condition extends rapidly

and in 24 hours the uvula, the pillars of the fauces and the pharynx may be affected. There is no foetor oris, no extensive salivation. The glands are not affected. Fever will depend on the original cause. But there is some dyspnoea and the infant may refuse to suck the breast or the bottle. Thrush infection is carried to the mouth through dirty nipples, soothers, infected hands or toys. Newly-born babies may be infected while passing out of the vagina or by contact with the bed-linen, clothes and person of a mother who has monilial vaginitis. Anderson, Sage and Spaulding cultured the material from the mouth of newborn infants and found that oral thrush developed in each instance in which a *Monila Albicans* was found in the mouth of the infants. They also report that oral thrush appeared more frequently in infants whose mothers had vaginal moniliasis.

Diphtheritic Stomatitis.—The incidence of diphtheria is increasing in Bombay. In this condition there is a thick membrane which may be a patch on the tip of the tongue or over its side, or may extend over its anterior two-thirds, or may be on the inner side of the lips or cheeks spreading to the gums. The salivation is not much. The submental and submaxillary glands are always enlarged. Though there is foetor oris it is nothing compared to that in ulcerative stomatitis. Some children look acutely ill; some look surprising not ill at all. There is generally a history of some previous illness, most often a gastroenteritis. When a reliable history is available, the duration of the membrane is a matter of five to six days. Fever is not a prominent feature.

Noma, the gangrenous type of stomatitis, is rare. It starts as a small dull indurated nipple or a group of vesicles on the inner side of the cheek. Very soon a local ulcer is produced which is covered by a dark grey slough with surrounding swelling and oedema. In the next stage the slough becomes black and gangrenous. The cheek is indurated, and if no surgical measures are adopted, is soon perforated. The child is very ill, prostrated, delirious and runs a high temperature. The breath is very offensive. The slough extends rapidly and may involve the gums, and tongue. The teeth may loosen and drop out.

The exact cause of noma is not known. Avitaminosis has been considered, but though the out-patient department of our hospital abounds in children suffering from avitaminosis, noma is very rare. A specific virus or agent acting on tissues that have been debilitated by previous illness has been suggested as the cause. Ekstein who observed 40 cases in the course of two years at the Children's hospital of the Model Hospital Ankara, found that out of 22 cases 19 suffered from malaria. Noma is a local condition. Except at the end, general intoxication is rare. Ekstein reported that "good appetite and absence of pain are paraxodic to the severity of the disease." In his cases the leucocytic count did not exceed 10,000 per c.mm. Investigating the cause Hutinel and Noblecourt pointed out the symbiosis of the fusiform bacillus with the Vincent's spirillum. Aschoff, Kumer and Muhleus agreed that this symbiosis, the "Plaut-Vincent symbiosis" probably involves the noma virus.

Ekstein investigated his cases and found that the necrotic area of noma contained all the micro-organisms that are found in normal or pathological conditions of the mouth. This was not very helpful. But on examining the fluid obtained by pricking the apparently healthy tissue just beyond the necrotic area he found fusiform bacilli, spirilla and a few leucocytes without any other organism. He was, therefore, of opinion that the Plaut-Vincent symbiosis plays an important part in the etiology of noma, the action of these organisms being facilitated by any condition that lowers the vitality especially of the reticuloendothelial system.

Allergic Stomatitis.—Though skin manifestations of allergy are so common, yet stomatitis is far less common. The oral manifestations are swelling of the lips, gums and tongue. The tongue may be so swollen that it fills the whole mouth and movement is painful, speech difficult, if not impossible. The list of substances to which children may be sensitive is very long but the common ones are milk, egg albumin, fish, orange juice, liver extract, cereals like barley, vegetables like carrots, brinjals, fruits like bananas and sweet limes, drugs like castor oil, milk of magnesia, dilantin sodium. Injections of serum may also cause this swelling. The swelling comes on suddenly, and disappears in about 24 hours under treatment. Unless the offending substance is scrupulously avoided there is a chance for recurrence. It is surprising what small amounts are enough to cause the disturbance. When stomatitis is a part of general allergic condition, there may be fever.

Vitamin Deficiencies.—Of these the commonest is the riboflavin deficiency. The tongue is just furred and then small areas are denuded of this coat. These areas, generally oval in shape, look atrophic, and the boundaries whitish and raised. In these denuded areas the papillae appear as tiny raised pinheads giving the tongue a granular appearance. The edges of the tongue may be fissured. The lips look red just inside the vermilion border. At the angle of the mouth the mucous membrane looks sodden, later it is red as if it has been stretched and torn, the crack extending as a tapering line both inside and outside but more on the outer surface. A good clinical picture is seen in infants acutely ill with diarrhoea and vomiting—red tongue, red lips, and a red streak at angle of mouth.

In nicotinic deficiency the tongue is first sore at the tip, then becomes swollen and red. There is excessive salivation. Later there is desquamation of the epithelium of the lingual papillae, the papillae become engorged and then atrophied. A raw reddish band appears along the side of the tongue, which is much indented. The surface of the tongue then looks swollen, glossy smooth and red. There is a similar reddish appearance of the gums and inner side of cheeks. There is pain and the child declines to eat any spicy or pungent food. There may be ulcers and these may contain Vincent's organisms.

Deficiency of vitamin C has been recognised for a long time by sponginess of the gums. In the milder cases the margins along the sides

of the teeth bleed only on firm pressure; there are no visible changes in the gums. But when the deficiency is marked the gums are swollen, invade the teeth, are purplish in colour and bleed easily and profusely. The invasion of the teeth by the spongy gums is best seen in infants who are just cutting incisor teeth. There is no excessive salivation, the tongue is not affected and the submaxillary and submental glands are normal.

Glossitis Marginans.—This condition starts at about the age of one year. At first a slightly elevated circular white patch appears on the dorsum of the tongue. This enlarges rapidly. In a day or two the centre of this patch assumes a different appearance and here desquamation starts. The desquamation spreads as the original patch enlarges and always nearly overtakes it leaving a raised waxy whitish border between it and the healthy tongue. The patch may spread over a large area on the dorsal area but it never spreads to the inferior surfaces. There may be a patch on either side and on the tip. Two or more patches may coalesce to form a large one with very irregular borders. The desquamated area is red and the fusiform papillae in that area are swollen and red. Healing begins from the pole distant from the raised white border. The white border itself gets subdued. The progress is so rapid that the picture of the tongue changes quickly, when the patch heals another may appear. The condition is chronic and may last up to puberty. Very rarely is there any discomfort to the patient. The salivation is not increased nor are the sub-maxillary or submental glands enlarged.

Various explanations have been put forth since Rayer first described it in 1831. Parrot considered it to be a manifestation of syphilis, but it has been proved it is not so. Others considered it as "eczema" of the tongue, but strangely these children do not exhibit any other manifestation of allergy. There is a semblance to riboflavin and nicotinic acid deficiency but continuous administration of riboflavin and nicotinamide has not prevented its recurrence. The Germans considered it a part of exudative mechanism.

In this, oval or irregular areas on the edge and upper surface of the tongue are denuded of the surface epithelium. A small area of the tongue may be permanently so, but at varied intervals of time this area increases. Then there is pain and salivation. The rest of the tongue is furred. At this time there is loss of appetite for food, the breath is offensive, the stools are whitish, and there may be a pruriginous eruption on the body.

Stomatitis with Blood Disorders.—In agranulocytosis, leukaemia, thrombocytopenic purpura, aplastic anaemia, infectious mononucleosis there may be stomatitis. Sponginess of gums is a feature. The mucous membrane is ulcerated and necrotic because of the absence of polymorphonuclear cells. In acute lymphatic anaemia the condition will probably have been diagnosed long before the stomatitis is seen, but in acute agranulocytosis fever and sponginess of the gums may be the only clinical finding till a routine blood examination reveals the real condition. The

presence of Plaut-Vincent's organisms in the mouth lesions may lead one away from the correct diagnosis.

DIAGNOSIS

The various conditions are diagnosed by :

1. Clinical appearance.
2. The presence of evidence in other parts of the body as in avitaminosis, allergy, infections and blood disorders.
3. Examination of a smear from the lesions and culture of material from them.

PROGNOSIS

The general condition of the patient has an important bearing on the prognosis. Other than in noma the prognosis is favourable though acute ulcerative stomatitis caused by the fuso-spirochaetal symbiosis is a serious condition, lasting for ten to fifteen days. Diphtheritic stomatitis, when it is an extension from faucial diphtheria, has a grave prognosis. Stomatitis caused by deficiency of nicotinic acid and riboflavin, particularly the former, takes a long time to improve. In scurvy the improvement is more prompt. Allergic stomatitis may recur. The geographical tongue persists for years with periods of exacerbations.

PROPHYLAXIS

1. The mouth should be cleaned after administration of food ; this cleaning must be done gently.
2. The use of soothers must be discontinued. As infants in the course of their development introduce their hands, toes, and toys into their mouths, these must be kept clean. Nipples of the mother's breasts and of the feeding bottle should be kept clean, the former by the use of weak boric lotion, the latter by boiling.
3. During acute infections the mouth must be kept moist by maintaining the fluid intake. The salivary secretion must be stimulated by giving barely sugar or lemon drops to suck.
4. Mothers who suffer from monilial vaginitis must be treated antepartum.
5. Adequate supply of vitamins should be included in the diet.
6. Care should be taken that children are not administered food when it is very hot. Similar vigilance should be exercised while using a steam inhaler as there is a danger of the child burning his mouth.

TREATMENT

Local Treatment :

If possible, the child should gargle with 1 in 5,000 solution of potassium permanganate, or any other mild antiseptic like Listerine or Glycothymolene, three or four times a day.

When the mouth infection is bad, the mouth should be cleaned with a solution of hydrogen peroxide in water, diluted in the proportion of 1 to 2 to 1 to 5. The cleansing is easier if a spray is used. This cleansing must be done thoroughly twice a day. After this the following local applications may be used, twice a day.

1. In mild conditions one per cent solution of gentian violet in water.
2. In cases of ulcerative stomatitis :—
 - (a) five per cent solution of gentian violet in water.
 - (b) when Vincent's organisms have been found either of the two :
 - (i) ten per cent solution of neosalvarsan in glycerine ;
 - or
 - (ii)

Liq. Arsenicalis	dr. 1
Vin. of Ipecac	dr. 1
Oleum Mentha Pip	ms. 2
Glycerine	oz. 1
 - (c) in severe and resistant cases seven per cent solution of chromium trioxide. During its application to the ulcers the rest of the mouth should be protected by cotton ;
 - (d) when only pneumococci or streptococci have been found local application of a suspension of sulfadiazine 60 grains to one ounce

3. In cases of thrush, 1 in 4,000 solution of mercury cyanide or five per cent solution of gentian violet in water, twice a day.

Recently we have applied penicillin solution, 5,000 units to 1 c. c. twice a day in 4 cases of ulcerative stomatitis and five of severe thrush. The penicillin solution is applied after preliminary cleansing with hydrogen peroxide. The cases of ulcerative stomatitis showed marked improvement in 3 days and were quite clean in 8 days. The thrush disappeared in one day. The results, therefore, were much better than with the other lines of treatment mentioned above.

4. For noma local applications of formalin, neosalvarsan, have been tried, but they are not effective. Ekstein injected 10 c. c. of anti-gas gangrene serum locally in the healthy tissue adjacent to the necrotic area, daily, till sloughing of the necrosed tissues took place. In the cases that recovered signs of demarcation were visible after the first local injection.

General Treatment :

1. As supportive measures, adequate nutrition and sufficient fluids must be administered. When there is difficulty in opening the mouth food should be offered as milk, thick soups, egg flip, custard, fruit juices and water. This may even be given nasally. In infants and in older children who refuse to drink, fluids will have to be given by lavage or subcutaneously or intravenously.

2. In infection with Vincent's organisms, when there is a tendency for the infection to spread to the tonsils and pharynx, neosalvarsan should be injected intravenously.

3. In vitamin deficiencies, nicotinamides, riboflavin and ascorbic acid should be administered. Though the clinical picture may suggest a predominant deficiency of one vitamin yet as these deficiencies are generally multiple, it is advisable to administer all of them. I suggest

nicotinamide 50 mg., riboflavin 10 mg. and ascorbic acid 100 mg. daily by injection and thrice a day by mouth.

4. In diphtheritic stomatitis, inject anti-toxin, the dosage being determined by the extent of the membrane and general condition of the patient.

5. In allergic stomatitis inject 0.2 c.c. to 0.5 c.c. adrenalin chloride solution 1 in 1,000 subcutaneously, once in 6 hours, and calcium gluconate 10 per cent solution 2 c.c. to 5 c.c. intramuscularly or intravenously daily. Give Syrup Benadryl (P. D. & Co.) thrice a day orally.

6. For noma inject anti-gangrene serum 10 c.c. to 20 c.c. intramuscularly and intravenously, daily, till sloughing of the necrosed tissue takes place. Ekstein injected about 500 c.c. in each case.

7. In exacerbated states of "Geographical tongue" fractional doses of calomel and a saline purge are useful.

8. Should there be fever, it is advisable to put them on sulfadiazine 1 grain per pound weight per day.

SUMMARY

1. Stomatitis is caused by various causes, only some of which are known.
2. A general clinical picture is described first, followed by salient points in known causes.
3. The prophylaxis and treatment of these conditions is described.
4. Attention is drawn to the value of a dilute solution of penicillin in ulcerative stomatitis and thrush.

(Indian Journal of Medical Science) May 1948.

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CEREBRO-SPINAL FEVER IN BOMBAY.

By S. G. VENGSARKAR.

In Bombay City the disease was present in sporadic form from 1912 but, the incidence was high in the years 1921 to 1925, 1935-39 and it has been again high since 1942.

The disease is mainly spread through carriers and rarely by contact with an actually ill patient. It was found that about 70 per cent of the contacts of cerebrospinal fever cases become carriers (Flügge 1906). They are usually (80 per cent) temporary carriers harbouring organisms in the nasopharynx for about three weeks only. A few (20 per cent) of them however may become chronic carriers.

The use of Sulphonamides 3 to 4 grams daily for 4 days, clear the nasopharynx of both group I and II Meningococci.

Aetiological Factors.

(i) Causative organisms. It was found that in a vast majority (87 per cent) of cases of cerebrospinal fever in Bombay, Meningococcus Group I (Types I & II) was responsible.

(ii) Season. The disease has been found to prevail during late winter and spring, particularly during the months of January to May as confirmed by its incidence in the City of Bombay. It is less prevalent in Summer with dry and less humid weather conditions.

Age: most of the cases were found in adolescents and young adults of the age group between 15-30 yrs, unlike in other countries.

Predisposing Factors.

The most important factors that predispose to cerebrospinal fever are overcrowding, insufficient ventilation, nasopharyngeal catarrh and the recent introduction of susceptible persons (like new recruits) into a community and the presence of highly virulent organisms. Spacing of beds in barracks or dormitories should be at least six feet apart and if this is impossible, sleeping arrangements should be so made as to make the occupants of alternate beds have their heads towards the feet of their neighbours. Attendants on patients such as nurses, doctors and menial staff, particularly those having a cold or chronic nose trouble should invariably wear face-masks while on duty, to prevent themselves from becoming carriers.

Clinical Course of the disease under treatment.

325 cases of cerebrospinal fever have been reviewed for this purpose in the present paper. The cases were over a period of twenty-six months from 1st of September, 1944, to the end of August, 1946. The total number of cases of cerebrospinal fever admitted during this period was 400, whom 122 expired giving a mortality rate of 30.5 per cent. However excluding the 77 moribund cases that died within 40 hours of admission (and gave no chance for response to thorough and effective treatment) the mortality was 45 out of 325 cases i.e., 13.8 per cent.

The cases under consideration were admitted mostly in the acute condition with classical signs and symptoms of the disease.

The onset was more or less sudden in 266 cases (81.8 per cent). It was insidious in 59 cases (18.2 per cent). 189 patients (57.8 per cent) were brought in an unconscious or semi-conscious condition and 111 of them (34.1 per cent) had delirium. 224 cases (68.9 per cent) gave history of headache or backache. 197 cases (60.6 per cent) had vomiting. 45 patients (31.8 per cent) had on admission a pulse rate slower in proportion to the rise in temperature. Out of these 13 maintained a slow pulse rate till they expired. In 22 cases, within 24 hours of treatment the pulse rate corresponded to the respective temperature curves, which had settled down to normal by that time. The remaining ten cases maintained the relatively slow pulse rate for some length of time. 141 patients (43.8 per cent) had temperature above 101 F. and 4 cases (1.2 per cent) had normal or subnormal temperature on admission. All these four apyrexial cases were treated with sulfadiazine and all of them made a complete recovery. Three had however, a history of fever for 2 to 6 days at home but no history of medical treatment before their admission into hospital. The fourth case had subnormal temperature and was in an unconscious and collapsed condition on admission. The pulse was very small. This case also responded well to sulfadiazine, and became conscious within 24 hours taking ten days for complete, recovery.

The clinical state simulated Waterhouse Friderichsen Syndrome. He responded well to circulatory stimulants and there was no necessity of administering sodium chloride or adrenal cortical extract. All except 23 had a positive Kernig's sign (92.9 per cent). In only 82 (35.2 per cent) was the Brudzinski's sign positive. Cervical rigidity was present in all except 4 (98.7 per cent); 58 cases (14.7 per cent) exhibited an extensor plantar reflex.

To sum up a vast majority of cases had a sudden onset. The most common and constant symptom were (i) headache or backache (ii) vomiting or nausea, (iii) unconsciousness or semi-consciousness with or without delirium and (iv) pyrexia. The commonest signs were (i) Kernig's sign and (ii) cervical rigidity.

Cerebrospinal Fluid. In almost all cases the cerebrospinal fluid was under tension. The cerebrospinal fluid of 279 cases (85.9 per cent) was either turbid or purulent and that of 46 (or 14.1 per cent) hazy. The period during which symptoms of the disease were present before obtaining hazy spinal fluids, was from 1 to 2 days showing a period of septicemia before the meninges are involved.

The cell counts of cerebrospinal fluid varied from 850 to 9600 cells per c. mm. (polymorphonuclear cells (85 per cent—95 per cent). Sugar was absent in 304 (93.5 per cent) and diminished in 31 (6.5 per cent), proteins were increased in all the cases. Micro-organisms were detected in 306 cases (94.1 per cent) either by smear or by culture. They could not be detected in 29 cases (5.9 per cent) only, in which all the clinical and other cerebrospinal fluid criteria were however, present. The ratio of the extra to the intra, cellular organisms was 4:1. The mortality rate

in cases with extracellular organisms was 11.1 per cent. It is interesting to note that in our series of cases none exhibited any rash or spots on the body. Only two cases were admitted in the septicemic stage of meningitis. On clinical grounds, they were diagnosed as cases of cerebrospinal fever, but lumbar puncture after 4 days however, the cerebrospinal fluid showed the characteristic changes.

Complications.

The dreaded complications described in old books are now very rare. The following were noted in the cases under review:

(a) Affections of the cranial nerves:

(i) **Squint:** Four cases developed a squint during the course of the disease. Two had a squint along with a history of fever of 3 days' duration. The squint in both the cases improved under treatment within 4 days. The squint in the other two cases developed after over 15 days' duration of the disease. Of these one was admitted with squint and loss of speech and died within 72 hours of admission. The other case was admitted into the hospital with seven days' history of symptoms of cerebrospinal fever and was treated with penicillin and later with sulfadiazine. On the 9th day of his hospital stay the patient got relapse of the disease, on the 18th day he developed squint, and expired on the following day.

(ii) **Diplopia:** One patient developed diplopia on the 6th day of admission into the hospital and it disappeared in 2 days.

(iii) **Ptoxis and Facial Paralysis.** One patient developed this complication in the hospital on the 12th day of the disease and improved after a week's treatment. Another patient had also facial paralysis of lower motor type. This was developed on the 5th day of disease (and 3rd day of hospital stay). The palsy disappeared after 15 days.

(b) Hemiplegia.

One patient had hemiplegia. The onset of the disease was with fever and hemiplegia. He developed a bed sore and expired after 10 days' stay in hospital.

(c) Skin Manifestations:

One case developed a rash. It appeared after two weeks of sulfadiazine treatment in hospital. The rash disappeared ten days after the sulfadiazine was discontinued under treatment with nicotinamide, liver extract and non-specific protien therapy.

Prognosis.

For a good prognosis adequate and bold treatment is also essential. Timid and irregular treatment gives rise to higher mortality or chronicity and relapses. There must be an early response to adequate therapy, say within 24 to 48 hours. If there is no such response seen clinically or in changes in the cerebrospinal fluid pictures within a week, the prognosis is unfavourable, though over 50 per cent of such patient do respond to the alternative-specific therapy or a combination of both.

From almost 100 per cent mortality of preserum and serum therapy days the mortality varied roughly between 44 per cent and 34 per cent with prontosil and sulphapyridine, and has come down to about 30 per cent with sulfadiazine and penicillin.

Treatment.

(a) Older methods. (ii) Serum Therapy.

A study of series of cases mentioned in the Annual Reports of The City Fever hospitals, Bombay, since 1926 to 1932 of seven years during which anti-serum was the only treatment reveals that the mortality rate in this disease has been 100 per cent.

(ii) Combined Serum and Sulphonamide Therapy.

4 tables 4 hourly, i. e., 12 gms. in 24 hours orally and 2, 5 gms. of sodium sulfadiazine intravenously in 24 hours in an average case.

In our series of 325 cases critically studied, 29 were given sulfadiazine and anti-serum combined. With the usual dosage of sulfadiazine 50 cc. of antimeningococcal serum was given intramuscularly for three successive days. Out of these 29 cases 24 recovered and 5 expired giving a mortality rate of 17.3 per cent against the total mortality rate of 88 per cent.

Sulfadiazine Therapy.

Of the 325 cases reviewed, 207 were treated with sulfadiazine. After establishing the diagnosis of meningitis, a solution of 2.5 gms. of sodium sulphadiazine diluted to 50 c.c. with distilled water or glucose solution, was given intravenously. At the same time 2 gms. (4 tablets) of the same drug were given orally and continued, every 4 hours. The intravenous dose was repeated after 24 hours in moderately acute cases. Usually the temperature reaches normal level in such cases within 24 hours. In less acute cases sodium sulfadiazine could be given intravenously at the onset in 1.25 gms. doses, duly diluted, at the intervals of 12 and 24 hours according to the response to the therapy. In toxic and hyper-toxic cases, associated with delirium and unconsciousness, it was found necessary to give 2.5 gms. of sodium sulfadiazine intravenously every 12 hours for 2 days till consciousness was regained, or in the alternative, 5 gms. as an initial dose, followed by 2.5 gms. after 24 hours and repeated as required thereafter.

The average total intravenous dosage of sodium sulfadiazine was 10 gms. in 3 days. Orally, sulfadiazine tablets were administered as follows. First 2 days, 2 gms., 4 hourly (i. e., 12 gms., in 24 hours). Then 6 gms., per day for the next 3 days. And later the drug was administered in gradually diminishing doses, so that the average total oral dose was 65 gms. in 12 days by which time the clinical condition and the cerebrospinal fluid became normal.

In some cases a total oral dose of about 100 gms. of sulfadiazine was required as adequate response was not forth-coming in due time with the average dosage. The minimum total oral dosage administered in this series was 20 gms. and the maximum was 104 gms.

Precautions :

The usual precautions with chemotherapy were taken as follows: (i) Plenty of fluids by mouth and whenever necessary by subcutaneous infusions of normal saline with 5 per cent glucose. (ii) Frequent checking of W. B. C. count to detect any contra-indication for continuation of chemotherapy. (iii) Careful observation of urinary excretion (Character and quality). (iv) Administration of nicotinamide as a routine along with sulfadiazine.

Penicillin Therapy.

In our series 71 cases were treated with penicillin. An intrathecal injection of 10,000 units of penicillin diluted in 10 c.c. of normal saline was administered and the drug was also given intramuscularly at three hourly intervals in dosage varying from 40,000 to 80,000 units according to severity of the case on admission. The diagnosis was immediately established and the treatment further continued. In a majority of the cases penicillin was given intrathecally once a day for four days, but in some more acute cases it was given twice a day. We are of the opinion however, that it should be given intrathecally twice a day as a routine in moderately severe cases as this has been found to shorten the course of the disease. It has also been observed that the total dose of penicillin by the intramuscular route for beneficial effect is less when the drug is administered intrathecally twice a day than otherwise. From 2 to 15 intrathecal injections of 10,000 units each, duly diluted were required in this series of cases. The range of total dosage given intrathecally varied from 20,000 to 1,15,000 units, the average being 40,000 units.

At the editorial suggestion of a medical journal in one case of this series only intrathecal injections of penicillin were given for the first two days; but as the general condition of the patient did not improve, intramuscular injections were also started and the patient recovered.

In very acute cases, 40,000 units were given intramuscularly every 8 hours, during the first 24 hours while in the less acute cases the dose was 30,000 units intramuscularly every 8 hours. According to the improvement noticed in the clinical and cerebrospinal fluid pictures the dosage was later reduced to 30,000 and 20,000 units 3 hourly till towards the state of recovery the dosage was brought down to 10,000 units and the interval increased to every 4 hours. The total intramuscular dose of penicillin required in this series varied from 2,00,000 to 17,70,000 units, the average dose being 7,60,000 units.

Duration of Penicillin Treatment: On an average days were required for recovery with combined intrathecal and intramuscular administration of penicillin. It was required to be continued upto 10 days in more acute cases. The intrathecal injections were continued till there was recovery clinically as well as by cerebrospinal fluid picture. Intramuscular injections were however continued still further though in gradually diminishing doses till not only was there a complete clinical improvement but until the cerebrospinal fluid became completely normal and remained so

as regards its appearance, cell count, the sugar and protein contents and disappearance of organisms, on three consecutive examinations at the interval of 3 to 4 days, after which the patient was declared, as a rule as cured and fit for discharge from hospital.

	Sulfadiazine Series.	Penicillin Series.
(i) Percentage of Mortality.	4.3 per cent (3 died out of 70).	10 per cent. (7 died out of 70).
(ii) Clinical Response.	It took 2 to 9 days for clinical recovery. On an average 3 days only.	From 2 to 7 days for clinical recovery. On an average 4 days.
(iii) Effect on Temperature	Temperature became normal in 2 days in 77.1 per cent of cases.	Temperature was normal within 2 days in 60 per cent of cases.
(iv) C. S. Fluid Picture.	C. S. Fluid completely normal in 5 days.	C. S. Fluid completely normal in 7 days.

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Extracts.

THE TREATMENT OF CORONARY DISEASE.

By: JOHN MARTIN ASKEY

Prevention: The measures indicated for the prevention of atherosclerosis in general are used.

Treatment: Clinical signs of coronary artery disease are shown by the symptoms of myocardial ischemia usually angina pectoris. The pain may occur spontaneously in the course of ordinary daily activity or as part of a diagnostic measure, it may be induced in latent cases by the exercise tolerance test or the anoxemia test of Levy.

In the majority of patients in whom angina is the first cardiac symptom, old complete occlusions have occurred in at least two of the three main coronary arteries. These are the patients without valvular heart disease or arterial hypertension. Angina pectoris occurring in patients with aortic stenosis or arterial hypertension is associated with less extensive coronary artery disease. In a heart free from valvular defect or hypertensive load, the demand for coronary blood is relatively small and the coronary circulation has to be markedly impaired before ischemia develops.

The fundamental objects in the treatment of coronary artery disease are first to promote the development of the maximum collateral circulation and to maintain its vasodilation and second to reduce the load upon the heart muscle and third to avoid any sudden disproportion between demand and supply of the blood inducing cardiac catastrophes.

Effects of Drugs.

The only drugs of value for angina cases is nitroglycerine.

Specific Treatment of Various Types of Coronary Diseases.

Angina Pectoris: In a person with angina pectoris, another occlusion of coronary arteries may occur at any time. The rationale of treatment is that longer an occlusion is delayed, the more time anastomoses have to form. Pain of angina pectoris is the index of cardiac ischemia and therefore, sufficient restriction of activity to avoid pain is of paramount importance. Along with this the use of vasodilators is essentially the medical treatment. The only drug of value is nitroglycerine which can give tangible benefit by its use not for relief of pain but as a prophylactic of pain. Any activity or event which is regularly productive of anginal symptoms should be preceded by nitroglycerine. The avoidance of emotional tension is important. The emotional tension may increase the heart load by 30-40% due to tachycardia and increased blood pressure. It is essentially the intensity of activity that is harmful. Nitroglycerine dose is 1/200 gr. which is chewed and held in the mouth. In some case 1-500 gr. is given at hourly intervals.

Treatment of myocardial infarction Classification of Wood is helpful in this respect :—

1. Severe case, a patient with prolonged pain, leucocytosis, shock, tachycardia, fever and signs of cardiac insufficiency such as dyspnea, cyanosis, pulmonary edema, and basal rales.
2. Moderately severe case. There is pain, electro cardiographic signs of infarction, fever, and leucocytosis but no signs of cardiac insufficiency.
3. Mild case. There is pain, electrocardiographic changes, but no leucocytosis, and no signs of cardiac insufficiency.
4. Very mild case. Brief pain lasting for 5-20 minutes. This probably represents occlusion without infarction.

Rest. A careful course avoiding the hazards of overactivity and the hazards of complete rest should be adopted. The dangers of over activity are the induction of sudden death, rupture, ventricular aneurysm, and the production of systemic emboli. Bed pan also favours sudden death and so a commode at the side of the bed is better in less severe cases. It is not necessary to worry about the bowels for several days. Straining at stools is to be avoided. A liquid diet is preferable for the first few days.

Rupture of the heart occurs in about 35% of patients, usually before the 16th day. Absolute rest does not prevent rupture.

Therefore for several weeks sudden physical strain should be avoided. Emotional strain should be guarded against.

Hazards of complete rest are thrombosis and embolism, edema of the lungs, hypostatic pneumonia, pulmonary infarction and secondary myocardial infarction. It is not necessary to keep the patient recumbent and it is preferable for him to sit up in bed.

A patient with severe attack should have 4-6 weeks bed rest. No walking is allowed until the sedimentation rate returns to the level before infarction, and the electrocardiogram has become stabilised. Bed rest for the patient with a moderately severe attack is three weeks. Rest for the patient with mild attack is from 7-10 days for very mild attack. Activity must be restricted during that time.

Symptomatic Treatment.

Pain: At the beginning of the acute attack morphine sulfate is given intravenously. Then it may be repeated subcutaneously. Usually not more than 1 gr. is necessary in 24 hours.

Shock: It is not yet decided whether to treat the shock of myocardial infarction with intravenous plasma or other fluids. It is better to give oxygen and wait. Atropine and papaverine are of doubtful value and should not be used.

Quinidine should be used for the prevention of ventricular arrhythmias or for auricular flutter or fibrillation. These are serious cardiac emergencies and must be treated immediately with digitalis and quinidine. Digitoxin 1.2 mg. is given orally quinidine is given 6 gr. every two hours until the arrhythmia is terminated. The development of paroxysmal

ventricular tachycardia is serious and quinidine also should be given as much as digitalis to avoid to initiate and perpetuate the ventricular tachycardia.

Digitalis alone in myocardial infarction is reserved for cases with definite left and right congestive heart failure with regular rhythm. If auricular fibrillation occurs digitalis must be combined with quinidine as digitalis alone in such cases produces an increased number of systemic emboli.

A peculiar complication of myocardial infarction or severe angina pectoris is pain in right or left shoulder which may occur soon after or several months after the attack. This is followed frequently with a painful hand with a smooth glossy skin and often fascia may develop. These conditions are treated symptomatically.

(Abstracted from the American Practitioner.)

HEADACHE

By S. BUTLER AND W. A. THOMAS.

To understand the mechanism of headache, it must be remembered that the following structures in the head are pain sensitive :—

1. The tissues covering the cranium, especially the cranial arteries ;
2. Intracranial structures such as the dura mater, the dural arteries, the venous sinuses and the cerebral arteries at the base of the brain ;
3. Certain nerves such as the sensory branches of the 5th, 9th and 10th cranial nerves and the I, II, III cranial nerves. Other structures which are sensitive are the cranium itself, the parenchyma of the brain, the pia, arachnoid and most of the dura, the lining of the ventricles and the choroid plexuses.

Stimulation of pain-sensitive structures on or above the superior surface of the tentorium cerebelli produces pain in front of a line across the ears and is carried by sensory branches of the trigeminal nerve ; structures below the tentorium cerebelli produces pain which is felt behind the ear and the pain is carried by branches of the hypoglossal, vagus and upper cervical nerves

Headaches are produced by: 1. Distention and dilatation of arteries inside the skull (mostly the branches of the external and internal carotid arteries)

2. Traction on the vessel within the skull.
3. Edema and spasm of muscles of the scalp and neck.
4. Direct irritation of pain carrying nerves or structures by inflammation or pressure.

Severity of Headaches.

The following headaches are usually very severe :—

1. Tic douloureux.
2. Vascular headaches ; migraine and histamine cephalalgia.

8. Inflammations within the cranium, meningitis, encephalitis, brain abscess.

The following are usually moderately severe.

4. Drainage headache.
5. Neuralgias of the scalp, including toothache, earache and sinusitis.
6. Traumatic headache.
- These headaches are usually mild ;
7. Tumors of the brain.
8. Eye strain.
9. Toxic headache including hypertension.
10. Tension.
11. Psychic 'headache.'

Vascular Headaches.

Vascular headaches are those primarily resulting from a dilatation of blood vessels. They include migraine, histamine caphalagia, and tension headaches. They are usually made worse by vasodilators such as nicotinic acid and relieved by vasoconstrictors like ergotamine tartrate and ascorbic acid. Epinephrine and ephedrine contract the extra cranial but not the intracranial vessels.

Migraine: There are three stages:

1. Preheadache or aura consisting of disturbances of the special senses mediated by cranial nerves and showing no pain. These are scintillating scotomas, hemianopsia or diplopia; hearing with tinnitus, or deafness, or vertigo; paroxysmal tachycardia, taste and smell, and various cutaneous paresthesias or peripheral vasomotor disturbances. Preheadache is cortical in origin, due to vasoconstriction of the internal carotid artery and its branches.

2. Headache itself. It begins as a throbbing unilateral localized headache, later becoming more generalized; it is steady in character. The headache is usually accompanied by nausea and vomiting. This is the stage of vasodilatation of the external carotid artery.

3. Steady headache. The headache is steady and grinding. It is usually more generalised and often bilateral. The scalp over the affected area is tense and tender. It is due to stretching of the scalp with edema of the walls of its vessels and pressure on pain carrying nerves.

Vasodilators are of value in stage one. Ergotamine tartrate in 70-90 per cent of headaches in early stage of headache itself. There is no treatment for stage three.

Histamine Headache.

It is a cyclic, paroxysmal headache, occurring several times in twenty-four hours, with unilateral pain, lacrimation, nasal engorgement and flushing of the involved side. Patients are awakened as often as 5-6 times during the night by severe pain which is relieved after arising; they return to sleep only to be awakened by subsequent attacks.

Treatment is with increasing doses of histamine, one or more daily. Better results are noted with the long continued drip of histamine as used in the treatment of migraine in which 2-75 mg. of histamine acid phosphate in 500 c. c. of normal saline is administered intravenously on six successive days at a rate as fast as can be tolerated. The rate is governed by appearance of headache, characteristic histamine flush or fall of blood pressure, all of which can be relieved by injection into the rubber tubing of 1 gm. of ascorbic acid or by slowing the injection. This headache is due to vasodilatation of the cerebral branches of the internal carotid artery.

Tension Headache.

These headaches are usually bilateral throbbing or pulsating, occipital described by the patient as a pressure in the back of the neck. They show no aura, no visual disturbances and usually no nausea or vomiting. They are benefitted by the intravenous use of histamine.

Psychic Headaches.

These are not really headaches. They are due to fear.

Intracranial or traction Headache

This is seen after spinal puncture after removal of spinal fluid.

Tumors of the brain: The headache is low grade, deep, aching and steady. It is usually increased by coughing or straining.

Extracranial Headaches

The commonest cause for headache is irritation or inflammation of the sensory, cranial or upper cervical nerves outside the skull. Neuralgias and myositis belong to this class. Irritation in areas of sensory distribution of the 5th, 9th and 10th cranial and upper cervical nerves produces headache, such as stiff neck, earache, dying dental nerves, or tick douloureux, inflammation in the nose or accessory sinuses and eye troubles.

Toxic Headaches.

These occur in uremia, metallic poisoning and eclampsia.

(Abstracted from the J. A. M. A.)



Correspondence.

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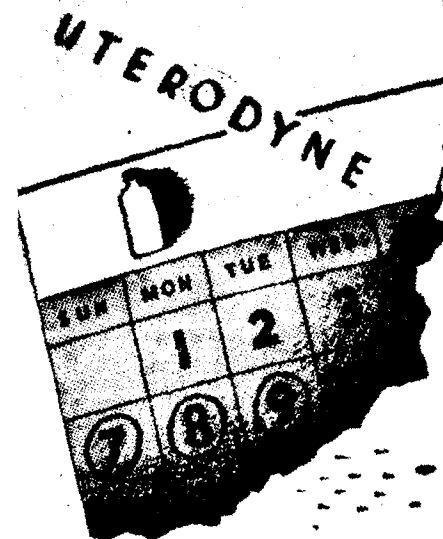
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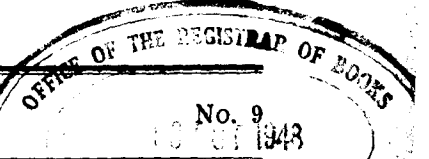
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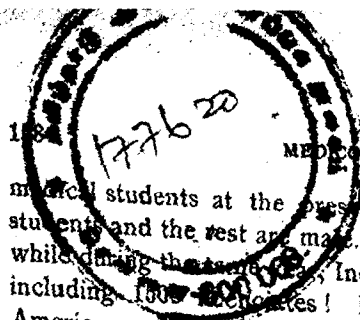
POST-GRADUATE MEDICAL EDUCATION

BY SUBODH MITRA, M.B. (Cal.), M.D. (Berlin), F.R.C.S. (Edin.),
F.R.C.O.G., F.N.I.,

*Professor of obstetrics and Gynaecology, R. G. Kar Medical College ;
Surgeon, Sir Kedar Nath Maternity Hospital ; Senior visiting
Surgeon, Chittaranjan Seva Sadan ; Director,
Chittaranjan Cancer Hospital, Calcutta.*

During my recent tour in U. K. and U. S. A. I availed myself of the opportunity of studying the system of Post-graduate Medical Education. The first thing that struck me specially in U.S.A. was that the authorities lay more emphasis on Post-graduate Medical Course than on the qualifying course for Graduation. Students up for qualifying examination are not to go through a severe detailed and meticulous examination and provided they have worked regularly and diligently in the under-graduate years, practically all the students pass their examinations. They learn detailed practical work during the intermediary period between the passing of the examination and the reception of the diploma, which is the license to practice medicine. Detailed theoretical parts of each speciality are generally taken in the Post-graduate medical studies. Knowing fully well my limitations, because the subject is so vast, I shall first try to give you a few details of American system of medical education.

In America, pre-medical course requires 2 to 3 years (mostly 3 years), after which students are qualified for the regular medical course. This medical course is finished in 4 years. Medical schools admit students in the first year, which is popularly called the Freshman class, all throughout the year, though mostly in the month of *June* and *March* and least in the month of August. In the United States 4.5 students per 1,00,000 of the population enter the medical schools every year. Will it ever be realised in India? In 1946, out of 5,745 students admitted, 60 per cent were male veterans on their return from war 11 per cent physically disabled persons (also war casualties) 7 per cent occupationally deferred and 12 per cent women. That shows how America is trying to utilise most of the war casualties by providing medical education to them in preference to others who are physically fit and young and naturally more serviceable in other departments. There are altogether 26,199



medical students at the present movement, of which 2108 are female students and the rest are male. In 1946, 8889 students were graduated, while during the same year, India produced 2200 qualified medical men including 1000 females! Even during war, the armed forces of America used to get their school and college courses by correspondence through the "Armed Force Institute" which operates in collaboration with colleges and universities. Graduate Training for Physicians is termed either as "Graduate Medical Education", or "Post-graduate Medical Institution" or "Continuation Study".

Graduate Medical education in the broad sense covers all types of instructions which follow the award of M. D. degree. In a more restricted sense, the term applies to well organised, systematic, scientific and clinical instruction planned for the benefit of the student under the supervision of medical school, hospital or other educational institution. It may or may not lead to higher academic degrees. Graduate education includes apprenticeships in the form of residencies (which we in India call House Surgeons or House Physicians) as well as fellowship which are more often university than hospital appointments. The term "post-graduate" applies to short periods of study undertaken by the physician after licensure and after he has established himself in practice. Currently two general forms of continuation study are offered: the first offered in metropolitan cities with facilities for systematic instructions and clinical and laboratory investigations. The second form offered to practising physician is by lectures, demonstrations and clinics in close proximity to the physicians' home so that there might not be any serious interruption of their practice. The purpose of this continuation study is to give instructions in subjects which were incompletely or unsatisfactorily taught during their undergraduate period and subjects of practical value such as recent advances in the prevention, diagnosis and treatment. I shall give you a short sketch of such training specially in obstetrics. Each of these intramural courses is designed to operate on a full one week or two week's daily schedule with special classes for a limited number of physicians who are given ample opportunities to observe obstetrics as it is practised under desirable hospital conditions. The course consists of lectures in obstetrics and pediatrics, conferences, seminars, bedside instruction, antenatal and post-natal clinics, manual demonstrations, laboratory exercises and home delivery services. In one hospital there are 26 instructors for a class of 10 physicians. Fees vary from nothing to 10 dollars a week for one or two weeks instruction. There are at least 12 such institutions where a practising physician can spend a short period of residence to learn the modern methods used in obstetric practice. You will be interested to know how these post-graduate courses are appreciated by the physicians. In 1945-1946, the attendance reported was 45,955. This is how these American doctors utilise their post-graduate courses. The education of physician cannot be completed with undergraduate curriculum. His development is a life long process associated with

unremitting toil and assiduous devotion to the most exacting of profession. In medicine the dissemination of new knowledge is as important as its acquisition.

The situation, obviously, is more complicated than in the undergraduate education. On the one hand are those who desire to fit themselves for the practice of specialities, and on the other hand, large group of physicians, already in practice, who want to keep abreast of the latest advances in medicine.

An important difference in the economic status of newly passed graduates and the practising physicians necessitates for each a widely different type of curriculum. The aspiring specialist who has just completed his internship frequently has no capital except his education. Often he is encumbered with financial obligations. For his further education he cannot pay in cash: he must pay in services. This he can easily do, for his time is not yet money. Hence apprenticeship in which student receives instruction and maintenance in return for his services as a resident or assistant are being developed. While hospitals are primarily engaged in the care of the sick, they are also deeply concerned with the training of medical, nursing and technical personnel necessary for the continued maintenance of high standard of hospital service. Approximately 300 hospitals closely associated with medical school participate in the instruction of undergraduate medical students, 752 are approved by the Council on Medical Education and Hospitals for intern training, 852 hold Council approval for residencies in specialists, 1,229 maintain accredited schools of nursing education, 191 offer acceptable affiliated course for student nurses, and 11 conduct approved schools for medical record librarians, 161 for X-Ray technicians, 294 for medical technologists, 24 for physical therapists and 18 for occupational therapists. There is an earnest desire on the part of the medical Council and hospital authorities to arrange for these facilities and the students are equally earnest in utilising these opportunities. Since 1941, the number of approved residencies has increased from 5256 to 10422. They get a stipend ranging from 15 dollars to 200 dollars according to seniority.

The physician who has built up a practice, however, has an economic value which does not permit him to spend long years in service in order to obtain the instruction which he needs but does permit him to pay reasonably for what he wants. His programme must be systematically and intensively arranged so as to permit his speedy return to his own country.

Besides usual post-graduate courses there are any number of medical Congress and Conference sponsored by different medical, surgical, gynaecological and radiological societies. These Conferences are mostly convened during summer and are well attended by professors, senior and juniors specialists as well as by general practitioners. In order to help the busy practitioners even Travelling Post-Graduate courses are arranged. Different medical societies are up and active in preventing

general practitioners from getting stalemates. I shall end the chapter of American medical system by giving a brief sketch of Mayo's Clinic. During my lecture tour throughout the States, I was lucky enough to be invited by the Mayo Foundation to give a talk on Radical Vaginal Operation for Cancer of Cervix-Uteri.

It was a delightful pleasure to have such a learned circle as my audience. This Mayo Clinic is the creation of one man Dr. Mayo. It appears like a myth how this Mayo with his two doctor sons could build such a magnificent clinic, which is decidedly one of the most amazing acquisitions of America. Mayo Clinic proper is a polyclinic, where patients come not only from different states of America but also from different countries of the world for proper investigation and diagnosis of their ailments. Thousands of patients are attending his clinic daily almost all by previous appointments. Their number varies 3-5000 per week. The procedure is something like this: as soon as a patient is admitted in the Clinic, a brisk general examination is made with a view to find out which actually is the system mainly affected. Next step as a general routine measure, radiography of the chest, flocculation test, and examinations of blood, urine and sputum are made. If any system is found to be chiefly affected, experts are either called in or the patient is sent in that department. By the time the patient reaches the specialist, all the previous records are already there sent through the vacuum conduit system. If this expert wants some special examination *e.g.*, radiological examination, urological examination, biochemical, ophthalmic and so on and so forth, the Secretary takes notes and arranges for these examinations as per schedule. If the opinion of other experts are needed, their service is at once requisitioned. When all the examinations are finished, these experts sit together, discuss and make the final diagnosis and the patient is advised accordingly. Some patients go back home with their ailments diagnosed and the line of treatment chalked out. The surgical cases are admitted to 5 hospitals incorporated with the Mayo Clinic. Daily surgical work of the clinic is simply staggering. On June 28, 1947, I found about 200 operations in the list consisting of 7 prostates, 6 thyroids, 6 hysterectomies, 3 thoraco-lumbar sympathectomy for essential hypertension, pneumonectomy, ligation of patent ductus, etc., etc. Besides this usual surgical routine, seminars for urologic diagnosis proctoscopic diagnosis, Dermatology, Radium and X'ray Therapy, etc., are held almost every afternoon. There are about 800 Staff doctors and 500 Post-graduate fellows working at the Clinic. With the exception of a few physicians all the doctors of the town (which is a small town having 30,000 inhabitants) are subsidised by the Clinic and are full-time workers. Their subsidy varies from 2,000 to 20,000 dollars per annum. It is the characteristic of Mayo Clinic surgeons to do both General Surgery as well as Special Surgery. For example, I found Dr. Counsellor doing Abdominal Hysterectomy, Inguinal Hernia, Cancer Penis, Kidney Tumour and Proctentia operation in seriatum. The Research section of Mayo Clinic

is the cream of the whole thing. Fully equipped laboratories for experimental surgery, histopathology, biochemistry, biophysics, etc., are well utilised by the Post-graduate fellows under instructions of experts. Volumes of their research works are published in the bulletins of Mayo Clinics, and are regularly sent to doctors all over the world.

The British Post-graduate Medical Federation is a school of the University of London, and its first aim is to provide for advanced education and research. For the graduate who has already started his specialist training and comes to London to continue his education in his speciality, University departments at the Post-graduate Medical School at Hammersmith Hospital and Special Institutes in association with special hospitals in London, provide organised post-graduate training. Advanced instruction is given in general medicine, general surgery, obstetrics and gynaecology and pathology. Special Institutes which are now functioning satisfactorily are those of Laryngology and Otology, Child health, Neurology, Ophthalmology and Psychiatry. Institutes for Orthopaedic Surgery, Urology, Cardiology, Dermatology and Dental Surgery are at various stages of development and are able to provide training for limited numbers. In addition to these provisions for advanced education and training in specialities, the central office of the Federation arranges *refresher courses* for general practitioners. These are held at non-teaching hospitals in London and the Home counties.

Post-graduate medical education is long overdue in our country. Knowing fully well our limitations, I submit herewith a workable and less ambitious scheme, which, if developed later on will be of great practical value.

The whole Post-graduate Medical Education may be divided into 3 sections, namely,

- I. Refresher Course.
- II. Special Training-College Course.
- III. Special Research and Higher Research Course.

I. *Refresher Course*.—Recent advances mostly from clinical and practical side in medicine, surgery and obstetrics may be taken up in this course. This will be a 3 months' course held twice a year and each course having 30 lectures on specific subjects. This course will be of great practical value to the general medical practitioners as well as to newly passed students. Lectures are to be given mostly in the hospitals by lecturers specially appointed by the University.

II. *Special Training-College Course*.—This will be very important, because it will train-up young students to be future specialists in respective subjects, and will introduce a spirit of systematic research work in medicine. It will be a course of 3 years divided into 6 sessions for specialised study and work. At the end of 3 years, there will be a University Examination for a Post-graduate degree of the nature of D.Phil (Med.). The preparation of a thesis good enough for publication in any first class scientific journal will be the first requisite for this

examination. There will be two theoretical papers as well as clinical and practical part of the examination. A pattern of the distribution of work for six sessions may be as follows: (taking midwifery as an example).

1st Session: Ward work and Clinical Laboratory work.

2nd Session: Junior Hospital Assistant work and General Pathology.

3rd Session: General Pathology, special Pathology and Post Mortem examination.

4th Session: Resident Obstetricians' work; to take up a subject for thesis.

5th Session: Resident Obstetricians' work; Assistant to Surgeons; to continue thesis work.

6th Sessions: Resident Obstetricians' work; Practical surgical work under the direction of the Professor: to complete the Thesis, which is to be certified by the Professor.

Besides these, there will be 200 hours' theoretical lectures on higher and applied Anatomy and Physiology, Embryology, Pathology general and special as well as on obstetrics and gynaecology.

Similar courses may be charted for medicine and surgery for the present and later on for other medical subjects.

III. *Special Research and Higher Researches.*—There will be two sections. In one section students passing D.Phil. Examination will work further for one year to prepare a more comprehensive thesis, and appear at M.D., M.S., or M.O. Examinations of the Calcutta University. In the second section, the research workers will take up different medical problems for study and research. These workers will be awarded Research Scholarships to continue their work.

In order to implement the Post-graduate medical education, the Calcutta University will have to requisition the services of all the first class medical institutions and medical colleges, which will of course retain their individuality so far as the undergraduate work is concerned. Thus there will be a federation of different institutions and one Director, who will be appointed by the University, will group the different institutions into different categories according as specific subjects of medicine, surgery and midwifery are taught. These different groups will be under the direction of three Deputy Directors who will be in touch with respective professors of grouped or federated institutions and arrange for the admission of students in different groups. These Directors and 3 Deputy Directors, Surgeon General and Vice-Chancellor will form the Post-graduate Council of Medicine. This Council will appoint University Professors and Lecturers of different subjects, subject to the sanction of the Syndicate and the Senate of the Calcutta University.

In conclusion, I would like to point out that 45 years ago Sir William Osler spoke on the educational value of medical societies. A well-conducted medical Society should represent a clearing house in which every local physician should obtain his intellectual rating and in which every physician could determine his assets and liabilities. "We doctors

do not 'take stock' often enough" said Osler, "and are apt to carry on our selves stale out-of-date goods. The Society helps to keep a man 'up-to-the times', and enable him to refurnish his mental shops with the latest wares. It keeps his mind open and receptive and counteracts that tendency to premature senility which is apt to overtake the man who lives in a routine". Should not the Calcutta Medical Club appreciate the sentiment wisely expressed by Osler? Should not the Indian Medical Association like the American Medical Association try to cultivate and advance medical knowledge through the federation of its numerous branches all over India? I am sure, it will.

(Calcutta Medical Journal, May 1948.)

ENIGMATIC TALES TOLD WITH CLUES

By Lt. Col. P. V. KARAMCHANDANI, M.B.B.S., F.R.C.P. (Edin.), Late I.M.S.,

District Medical Officer, Bellary.

What is the etiology of nephritis (Bright's Disease), what are the clearly distinguishable types, what is the relationship of nephritis with hypertension, what are the easily available tests of renal function within the range of a general practitioner and what is the rationale of therapy? On etiology depends every thing else. This etiology has been an enigma so far. Workers from time to time have put forth various theories following various tracks which have not led to fruitful consummation. Some recent clues have come to hand at last. It is probable that these clues may provide a final answer. I intend discussing these clues and other connected problems in this article.

Etiology:

Whilst I was Medical Specialist, Jubbelpore area in 1945 I was called for consultation in the British Military Hospital, Jubbelpore. The patient was an English child 4 years old suffering from anasarca, hyperpiesia, albuminuria, cylindruria. History showed that he had had an attack of streptococcal infection (tonsillitis) some time back from which he had recovered. After this, acute nephritis developed. This set me thinking hard. Then I remembered to have seen cases of acute nephritis in England, following in the wake of scarlet fever. A question at once shot within me. If acute nephritis had been the result of direct invasion by the streptococcus or its toxins, why had it not developed at the height of infection? This was clue number one.

Soon after I was posted as District Medical Officer, Trichinopoly, where a big maternity section was attached to the Government Head quarters Hospital in the same compound and I saw cases of acute nephritis in pregnant women which presented following features:—

- i. development of disease during later months of pregnancy.
- ii. cure on emptying the uterus.
- iii. history of a similar occurrence during previous pregnancy. I queried myself what was the cause of this pregnancy kidney, a condition

which resembled acute nephritis, yet was directly related to pregnancy? This was clue number two.

Then we had a visit by Dr. C. S. Ramachandar Rao who gave us a profitable demonstration in Rh blood groups. I had private discussions with him. It dawned on me whether same antigenic incompatibility existed between the mother and the child in Brights' disease in pregnant women, just as it had existed with the Rh groups, the foetus producing an antibody to its mother's kidney, which the mother absorbs via the placental route. This was clue number three.

Soon after I happened to read Penrose (1946) and Kalmus (1946) and Platt (1947) and the picture cleared up from the haze just as spot light puts into relief the panorama of a fading vision. This is what the picture was and it fitted so well in the frame of my conception that I quote the above authors verbatim. "These peculiarities might be explained if nephritis were the result of an abnormality connected with the development of immunity—some process analogous to allergy or to anaphylaxisin human disease the streptococcal infection in some way causes the renal tissue to become antigenic and when immunity develops, an acute antigen-antibody reaction occurs in kidney." A question may arise, why should there be localization of disease in kidney and not elsewhere? The answer is offered by the above authors in the following terms "because kidneys contain the most vulnerable part of the vascular system viz., the glomerular capillaries."

How beautiful the picture—too beautiful for words! whether this picture is a reality or a hypothesis will be proved by time. About 80 per cent of this type recover completely, few die in the acute stage or within few months and the rest called chronic nephritis type I, run a latent course of several years duration, remaining apparently well enough, although revealing hyperpiesia, albuminuria, and slight renal dysfunction. Ultimately either the tube bursts (vascular crisis—cerebral haemorrhage) or filter chokes (kidneys fail—uraemia).

But whereas this is the picture in acute nephritis what is the picture in chronic nephritis? The latter disease is so indelibly marked on my memory because it occurred in a very near and dear relation of mine, on whom I waited day and night in suppliant attendance, that my minute observation can be relied upon to substantiate what I am going to say hereafter. In this very near and dear relation of mine started the disease insidiously at the age of 42 years. I was the first to discover commencing oedema over his face which even he had not noticed. Urine showed heavy albuminuria and hyperpiesia. There had been no infective illness or any sort. In spite of treatment which varied from one system of medicine to another, he ran a rapidly downhill course and expired exactly two years later. I was mortified. Question after question shot within me. What was the etiology? The answer came again and again, the patient had had hyperpiesia at the very start and that he was dead within two years. I observed several cases later on and they proved

true to the type i.e., those with hypertension at the start, ran a rapidly down hill course and were dead within two years. This was clue number one.

In 1939 Wilson in experiments on rats produced hypertension of a malignant type by constructing the renal artery on one side. The opposite kidney as a result of high blood pressure showed secondary changes viz., necrosis of arterioles which in long standing hypertension was almost a perfect reproduction of certain forms of chronic Brights' disease in man. This was clue number two.

In 1942 Ellis in his Croonian lecture clarified the picture and I would like to quote him verbatim. He says "The demonstration by Wilson and Byron that hypertension can produce in the kidney progressive histological changes formerly termed chronic interstitial nephritis.....the production of designed experiment of such renal lesions as I think, definitely established their hypertensive origin and this is crucial to an understanding of chronic Brights' disease. I suggest that chronic interstitial nephritis as we have known it in the past, is merely the result of hypertension." The ultimate cause of Brights' disease may still be obscure but the fact that hypertension can produce renal changes found in chronic Brights' disease now stands undisputed.

Naturally the question now arises is whether there are three stages of nephritis which text books call acute, subacute (with oedema) and chronic? My answer is definitely no, and the sooner we discard this wrong conception the better it is, as it reveals an ignorance of onset, course and prognosis. These cases of chronic Brights' disease (with gross persistent oedema) are different from the acute Brights' disease at the very start, as they occur at any age, are not ushered in by any illness, creep in insidiously and usually the patient discovers oedema of the ankles or face while the doctor discovers heavy albuminuria. Those with hyperpiesia at the start run a rapidly down-hill course and die within two years. Others however who run a more favourable course with varying degrees of albuminuria and oedema for many years, have a normal blood pressure and are called Nephrosis. But the latter is not a separate disease, for, these ultimately develop hyperpiesia, then renal failure and die.

Thus then we have two main types of nephritis which Ellis (1942) has called type I and type II.

Type I has abrupt onset with headache, vomiting, haematuria, transient oedema, is related to infection usually following a streptococcal one, occurs in young people with 80 per cent complete and permanent recovery rate—the remaining run a latent course often lasting many years and remain apparently well but terminate ultimately in hypertension and uraemia. A few (5 per cent) die within a few months of onset.

Type II commences insidiously, is unrelated to infection, occurs at any age and is marked clinically by a prolonged stage of hypertension and persistent oedema. How simple and clear! Well this is the picture

which the general practitioner may profitably bear in mind. There are a few exceptions to the above e.g., acute focal nephritis where haematuria occurs at the height of infective illness etc., but the whole object of this article is not to write a text book but to focus thought on the subject of renal disease, to simplify diagnosis, clarify prognosis and emphasise treatment.

Tests of renal efficiency.

Although to-day we have reached stages where we can test separately the functions of the glomeruli and the tubules of kidney, yet I propose describing tests of practical value to a general practitioner for whom this article is primarily written.

1. Dilution and concentration tests.

First day (dilution test). After passing urine, patient drinks two and half pints of water within half an hour. He is then made to pass urine at half hourly intervals for next 4 hours. Each specimen is separately examined for (a) volume (b) Sp. gravity. Normally the volume should be $2\frac{1}{2}$ pints and Specific gravity minimum of 1002 or less.

Second day (concentration test). He takes only 16 ounces of fluid in 24 hours, four ounces six hourly. Fruit is not given and patient takes his ordinary food.

Urine is passed as and when he likes. Each specimen is kept separately and examined for (a) volume (b) specific gravity. Normally the volume should not exceed 25 ounces and specific gravity maximum of 1027 or more.

In renal inefficiency volume of urine on the first day is too little, while on the second day too much because excretion tends to continue uniformly irrespective of body requirements. As for the specific gravity the more and more it is narrowed, the more and more disease is supposed to have progressed. The dilution test may show minimum of 1005 while concentration maximum of 1015, and still the patient complains of no symptoms while blood shows no evidence of uraemia.

Concentration test is more reliable than the dilution test, but both set up a rigid standard of what constitutes the normal renal function.

2. Fixation of specific gravity.

Patient takes no fluid of any sort for 16 hours from 4 p.m. to 8 a.m. next day. The evening urine is discarded. The morning urine is tested for specific gravity. If renal function is satisfactory specific gravity should rise to 1025 or more. In elderly person 1020 is good enough.

The above tests distinguish benign hypertension with albuminuria. result of cardiac decompensation (where renal concentration power is preserved) as from malignant hypertension or nephritis with renal failure (where renal concentration is poor).

3. Urea concentration test.

Patient is made to empty his bladder and given 15 gms. of urea dissolved in 100 c.c. of water flavoured with tinct. orange. Urine is passed one, two and three hours after, and urea percentage tested in each

by hypobromide method. There should be at least 2 per cent urea in one of the three specimens. If it is 2.4 or more it is satisfactory. Diet has a great bearing on this test because if the patient is taking low nitrogen diet the tests have less value.

Treatment.

The motto to be remembered is "in type I, attend to the kidneys, in type II attend to the general condition."

Type I—Functions of kidney are excretion of water, waste (products nitrogenous), acid (products), salt (to maintain optimum concentration 0.6 per cent in tissue cells) and toxins.

Kidney is defective, it cannot throw out salt, therefore salt is retained in tissues. Since body cells must have 0.6 per cent Sod; Chloride, water is retained to give this optimum concentration result is oedema.

Kidneys cannot excrete water, it is retained, blood becomes so to say watery and water oozes out into the tissues increasing oedema.

Kidney is diseased, it cannot throw out waste, therefore nitrogenous waste products are retained. Result is rise in blood urea. Now then, there is plenty of urea (the best diuretic) in blood, there is plenty of water in the system (oedema) and there is high blood pressure, why is there no polyuria? Because kidneys are inflamed and renal epithelium paralysed by retained toxins.

What is the rationale of treatment?

Rest to the kidneys and relieve them of their work. Kidneys cannot excrete water, waste (nitrogenous products) and salt. Therefore restrict water, food and salt intake. Patient should not be allowed to be thirsty but maximum quantity of fluids allowed should not exceed 8 pints in 24 hours for adult and half that for a child of 12 years.

As for food, milk contains 4 per cent protein i.e., 0.56 per cent nitrogen. It will do no harm to deprive the patient of nitrogen for a time. I have said, in type I, concentrate attention on the diseased organ. Therefore diet at the outset should be fruit juices, glucose water, barley water with little milk added and modified imperial drink (i.e., potassium acid tartarate, half a lemon and sugar added to 20 oz. of boiled water strained and cooled) in moderate quantities. Toffee made of butter and sugar may be allowed. As the patient improves milk may be allowed

Withhold table Salt. Instead give a mixture of formates, citrates and phosphates e.g., ruthmol.

Kidneys cannot excrete acids therefore combat acidemia, by giving saline diuretics e.g. Pot: citrate, Sodi bicarb etc. We try to achieve three objects. (i) we raise concentration osmotic pressure of blood, reverse the current and draw water from tissues into the blood. (ii) Keep urine mildly alkaline. (iii) Help possible diuresis.

Kidneys cannot excrete toxins, therefore we must divert toxins to other channels e.g., keeping bowels open by a daily laxative, promoting function of skin by mild diaphoretics and protecting skin from chill.

They say drugs are useless, Perhaps this emphasises the other extreme end. Equilibrium lies in the centre.

Complications like convulsions, may need special treatment like lumbar puncture, I.V. injection of 50 cc. 50 per cent sucrose, and rectal administration of 8 oz. 25 per cent mag. sulph. When convulsions are associated with failure of right heart venesection help considerably.

After the patient is cured, we tell him so and let him lead a normal life. If however he enters a latent stage (after many weeks in bed there is albuminuria and hypertension), it is better to tell him that although albumen is left by nephritis his kidneys are functioning well, and the disease is no more active. It is no use submitting the patient to the stress of anxiety neurosis and make him a nervous invalid during the limited span of his life. The relatives also should not be told too much lest they pass the anxiety on to him, although one day we will have to admit that the disease has become active again. "Plate of herbs and contentment is better than oxtali and strife." This saying was never more true before than it is today.

Type II—Nephritis. No drug treatment is known. Therapy consists mostly in combating oedema. There is no defect in excreting nitrogenous waste products by kidneys, which is in contrast to what happens in type I; but there is great retention of fluid in tissues and excessive loss of protein in urine. Therefore rationale lies in removing excessive fluid and preventing its reaccumulation. This is accomplished by diet depending upon renal function tests i.e., blood urea and urea concentration, which should be 20-40 mgm per cent and 2 per cent respectively.

There is excessive loss of protein in urine, which depletes plasma protein content, which in turn lowers osmotic pressure, thereby allowing water to ooze out into the tissues. If we raise the plasma protein content, we raise the osmotic pressure in the circulatory system, we reverse the process and empty the oedematous fluid from tissues into the blood stream which is thrown out by the kidneys. How can this be achieved?

(1) By high protein and low salt diet.

Give meat, chicken, cooked eggs, fish, cheese, and beans in large quantity. Oedema disappears in 6-8 weeks, then proteins in diet are gradually decreased, fat and CHO increased till 60-70 grms of proteins are being taken daily. Salt may be replaced by ruthmol, because thereby we avoid increasing the miseries of an incurable disease. I have used mercurial diuretics in selected cases with success (i.e. where oedema is persistent, renal function is adequate and there is no anaemia) and also used intravenous plasma.

Some authors challenge the above and attribute good results to diuretic action of urea formed from high protein diet. This may be true. With that object in view urea which is a strong diuretic, gms. 15 in oz. ii of water B.D. for 7-10 days, then stopped and continued off and on for one year is recommended. Its failure augers ill for the patient.

Whatever be the mechanism, so long the end is achieved, means do not matter, provided these be judicious and controlled.

As for the recent high fluid (7 pints per day) and low salt diet therapy. I should say that this regime in cases with cardiac and renal oedema, helps excretion of sodium and consequently of water. There is initial gain in weight during the first few days followed by intense diuresis and weight reduction with complete disappearance of the oedema. A word of warning is necessary here. This method should not be tried in cases of extreme oedema because initial retention of fluid might carry away the patient with laryngeal and pulmonary oedema.

Finally in terminal renal failure—so called chronic nephritis and uraemia—even the most recent peritoneal dialysis is a palliative measure. We have to admit defeat. We have no remedy except the palliative and the psychological.

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THE STATUS OF PENICILLIN IN THE TREATMENT OF SYPHILIS.

REPORT OF THE COUNCIL OF PHARMACY AND CHEMISTRY, U. S. PUBLIC HEALTH SERVICE, *J. Amer. Med. Ass.* 136 : 873-879 (Mar. 27) 1948.

Though 5,00,000 patients have been treated since 1948, available data on penicillin therapy is still provisional and no formal routine schedules are yet laid down. Nevertheless, penicillin is the principal agent against syphilis in the U. S. A.

Penicillin G is the most effective type of penicillin for the treatment of syphilis. Intramuscular injections are the preferred method of administration; with intravenous injections after reaching the maximum the concentration falls too sharply. Intrathecal injections are unnecessary because satisfactory results are attained with intramuscular injections. Oral treatment is not advised for syphilis. Amongst other methods of prolonging effective blood levels of penicillin, intramuscular penicillin in oil and bees-wax is the only method which seems practical in the treatment of syphilis.

Penicillin is relatively non-toxic to man, except when administered intrathecally. Some reactions appear to be due to impurities in the drug, but usually they are allergic in nature.

The Jarisch-Herxheimer reaction is common and occurs in about 50 per cent of cases, appearing within 12 hours and lasting 24 hours. It is not considered dangerous in infantile congenital syphilis, but may have been a contributing factor in some deaths. About 25 per cent of cases of late and latent syphilis have experienced mild reactions. It is not uncommon in neuro syphilis. Penicillin is now known to be without danger to pregnant women.

Isolated instances of possible penicillin resistance in the experimental (rabbit) and clinical (late benign gummatous) syphilis have occurred. There is little clinical evidence of anything analogous to arsenic and bismuth resistance, or sulphonamide resistance in gonorrhoea. The effect on syphilis of penicillin used against other diseases is of practical importance. All patients treated with penicillin for gonorrhoea should be followed by monthly serological tests for syphilis for at least 4 months. A sharp febrile reaction within the first 24 hours after the use of penicillin in gonorrhoea suggests concurrent early syphilis (Herxheimer reaction). Patients previously treated for pneumonia, surgical infections etc., should be retreated if they have not received the minimum amount of penicillin suggested in the following sections.

Penicillin in early syphilis—In this condition the value of penicillin routine is evident. The results are expressed in terms of cumulative failure, the term failure including:—Clinical relapse—the re-appearance of any clinical evidence. Serological relapse—includes rise in titre or development of spinal fluid abnormalities. Seroresistance—if after 12 months, the titre is 16 Kahn units or higher.

When penicillin alone is used in early syphilis the optimum dose is 4.8 million units, duration of treatment 7½–8 days, individual doses 50,000 units at two or three hour intervals. The failure rate after one year of observation is about 10 per cent. The critical period for relapse is 4–9 months after treatment. Results are slightly better in sero-negative than in sero-positive cases, but there is no justification for lower dosage in the former. Retreatment is necessary following relapse, but the failure rate is higher. Where penicillin in oil and wax is used, hospitalisation is unnecessary. A single intramuscular injection of 6,00,000 units provides a sustained measurable blood level for 20–26 hours. 10 daily injections provide a failure rate (about 15 per cent after 1 year) which is comparable with that following the use of 4.8 to 9.6 mega units of penicillin in aqueous solution. Local discomfort and allergic reaction are slightly more frequent by this method. Sometimes penicillin is administered concurrently with other treatment methods. The value of combining penicillin with an arsenic or bismuth compound or during artificial fever is not established; results are not improved and the treatment is a more dangerous one. The method is not advised as the first course of treatment in early syphilis, though there may be practical advantages in using the combination in patients who have not responded to a course of penicillin alone.

Re-treatment of Penicillin failures—Retreatment of patients with clinical relapse including re-infections, serological relapse, or significant sero-resistance is the same as for the original course. A further course of penicillin with simultaneous or subsequent treatment with arsenic and bismuth should be considered.

LATENT SYPHILIS.

Penicillin is no more effective than arsenic and bismuth on the serological tests for syphilis. It is possible that penicillin may prevent the appearance of clinical manifestations of the disease for serological reversal is not essential for success; moreover sero-resistance is general in syphilis of more than five years duration. In latent syphilis an extensive observation period up to 25 years will be necessary to ascertain whether penicillin ensures the same cure rate as the six months of arsenic and bismuth therapy.

In benign late syphilis a total dosage of 2 to 4.8 million units or more is as effective in healing lesions of benign late gummatous syphilis as arsenic and bismuth, though the ultimate clinical outcome is still undetermined.

Cardiomascular Syphilis

A large series of observations on the expectancy of life of penicillin-treated patients and patients treated with other drugs is required. Extremely small initial doses of penicillin do not avoid the Herxheimer reaction and since therapeutic shock in such patients may be a serious matter, it may be advisable to withhold penicillin or give preparatory treatment with heavy metals. A total dosage of 5 million or more units

of penicillin is suggested in individual doses of 25,000 to 40,000 units over 15 or more days.

Ocular Syphilis

The penicillin treatment of syphilitic iritis is rapidly effective in a dosage similar to that of early syphilis. Its value in interstitial keratitis is uncertain even with a total dosage of 10 mega units, and it may be necessary to supplement penicillin therapy with artificially induced fever and arsenic and bismuth therapy. In the treatment of primary optic atrophy induced malaria, being of established value, should be combined with penicillin. Several years observations are required to assess the value of penicillin alone.

Pre-natal Syphilis

Penicillin is nearly 100 per cent effective; arsenic and bismuth therapy is unnecessary and undesirable. It is equally effective at any stage of pregnancy and should be used as soon as the diagnosis has been established. A total dose of 4.8 to 6 mega units administered at 2-3 hour intervals over 8-15 days is required. Adequate follow-up of the mother and infant is essential.

Congenital Syphilis

The recommended total dosage in infantile congenital syphilis is 1,00,000 to 400,000 units per kilogram of body weight administered in aqueous solution two or three hourly day and night for eight to fifteen days. Late congenital syphilis responds to treatment in the same manner as similar manifestations of the acquired disease. Its effect in arresting the progression of late or latent congenital syphilis infection requires observation over many years.

NEURO-SYPHILIS

The spinal fluid abnormalities in asymptomatic neuro-syphilis respond well to penicillin treatment especially in the earlier phases of the disease and the effect on blood serological tests is similar to that of arsenic and bismuth.

The effect on the spinal fluid findings in a symptomatic neuro-syphilis is equally beneficial. The effect on clinical manifestations is more difficult to assess but is better than that obtained with metals. Comparison with fever therapy is not yet available.

Acute syphilitic meningitis requires a total dosage of 5-10 million units for the immediate improvement and disappearance of symptoms: the spinal fluid and blood reactions revert to normal.

Penicillin improves the expectation of life in dementia paralytica beyond the average four-year period. About one-half of the cases show definite improvement in mental conditions though there are signs of increase in many others. Reactions are more frequent after penicillin therapy than after fever therapy. The value of penicillin therapy combined with malarial therapy in paresis and tabo-paresis is not yet determined.

The effects of penicillin on the course of tabes dorsalis and tabo paresis are very variable. It is more effective than arsenic and bismuth and is less toxic; it may be more effective in combination with malaria. Penicillin has little effect on diffuse meningovascular neuro-syphilis, syphilitic epilepsy and Erb's syphilitic spinal spastic paraplegia. It may have a favourable effect on syphilitic nerve deafness and a great improvement of hearing or return to the normal state is attained.

The dosage of penicillin in neuro-syphilis depends on the amenability of the condition to treatment. 4-10 million units in aqueous solution given in 7½-21 days is suitable for many readily treated cases of neuro-syphilis, but in later and more resistant conditions, a total of 10-20 million units given over a period of 12-20 days combined with induced malarial therapy (unless this is contraindicated) is advisable.

POST-TREATMENT OBSERVATION

Post-treatment observation for an indefinite period of time is essential for syphilitic patients treated with penicillin. Many years must pass before the ultimate outcome of penicillin therapy for syphilis can be definitely stated. The importance of the continuity of his own post-treatment follow-up must be impressed on the patient.

Early syphilis:—During the first year, monthly physical inspections and blood tests are essential. The spinal fluid should be examined at six months. If normal, and there are no other signs of syphilis, the spinal test should be repeated two years after treatment. If still negative, no further spinal test is necessary. If there are no signs of relapse, blood tests should be made three-monthly during the second year and six-monthly during the third and subsequent years. Annual physical examinations for an indefinite period to detect possible ocular neurological and cardiological symptoms are essential.

Infantile Congenital Syphilis:—Pregnant women with early syphilis or early latent syphilis of less than four years' duration, should be followed-up as for patients with early acquired syphilis. Infants born of mothers with primary or secondary syphilis during pregnancy should have blood tests at birth, thereafter every two weeks, during the first two months, then at three and four months. Infants born of mothers with latent syphilis should have blood tests at birth, thereafter every six weeks and then at three and four months. X-rays of the long bones should be taken at the first and sixth week. If the infant is sero-negative or otherwise normal at four months it is considered to be non-syphilitic.

For any type of late syphilis excepting neuro-syphilis, six monthly blood tests and annual physical surveys for the duration of life should be made.

Neuro-syphilis:—The spinal fluid should be retested every six months for the first two or three years, thereafter at longer intervals. The cell count and of protein content are the most important tests. If these become normal and remain so, regardless of the spinal fluid findings, clinical relapse or progression is unlikely. If the cell count

and or protein count remain high, a further outbreak is likely and treatment, preferably penicillin combined with fever, should be given. Physical examinations should be repeated every six months for two to three years and thereafter at yearly intervals.

M. H. N.

PREVENTION AND TREATMENT OF PRICKLY HEAT.

It is very much easier to prevent than to cure this condition. The aims of prevention are the avoidance of the condition that favour and precipitate the condition :—

- (a) Avoiding as much as possible the environmental conditions, namely, high temperature and humidity, that produce Prickly heat; much can be done by the use of air conditioning and electric fan.
- (b) Avoiding unnecessary exertion unless the immediate removal and change of clothing if possible.
- (c) Avoiding hot baths unless ample time can be allowed for cooling off before redressing.
- (d) Avoiding the taking of long draughts, especially of warm tea or coffee, when fully dressed.
- (e) Reducing clothing to a minimum and avoiding constrictions around the waist, neck, wrists, etc. Children should be allowed to be naked or at most wear a pair of pants, and infants should be spared from wearing even a diaper.
- (f) Wearing fine cotton or linen underpants and vest next to the skin and changing these as often as possible, at least twice during the day.
- (g) Bathing and/or taking a shower-bath with tepid water at least twice a day.
- (h) Avoiding the too free use of ordinary soap in the bath but washing over with special antiseptic soap and leaving the leather to dry with the precaution that sensitivity to such soaps must be carefully tested by each individual.
- (i) Drying the body thoroughly, cooling off under an electric fan and, finally before putting on the clothes, dusting the whole body (except sensitive spots), and especially those areas most liable to prickly heat, with an antiseptic dusting powder.
- (j) At night, in the absence of air conditioning or an electric fan, using a hard mattress, with a sheet of grass matting between it and the under sheet, a firm pillow, and a 'Dutch wife' that is a hard bolster or an open rattanwork cylinder over which the legs and arms can be thrown to keep these apart and from lying against the body.

- (k) Keeping a supply of hand towels for mopping the face, arms and other exposed parts of the body repeatedly; these must be washed and dried frequently.

It may be considered that much of this is counsel of perfection, but it is by following these practices that many experienced sojourners avoid prickly heat altogether. The less fortunately placed must adopt as many of these practices as possible.

In addition to the above, judicious sun-bathing is to be recommended, as the tanned skin is not so liable to prickly heat. This is particularly true of infants and children.

Some observers consider that a reduction of the fats and carbohydrate in the diet with an increase of the protein is of value in reducing liability to prickly heat, but the only dietary modification from which the writer has direct benefit is the addition of extra salt, (2 gramme tablet to each pint of water). This is an important point which should be emphasized. The taking of a balanced diet with all vitamins will of course help to maintain good health and indirectly reduced both prickly heat and more particularly, its complication.

It is usually worth prescribing an antiseptic hair lotion, as the hair is undoubtedly a prolific source of fungi and other micro-organism that play a part in the syndrome. A lotion consisting of castor oil and spirit with 2 per cent curesol (a colourless resorcinol preparation) is very effective. The use of some inoffensive mildly antiseptic oil for thorough inunction of the body has been very successful in some instances. It is a rational producer and it has been suggested that in entire freedom prickly heat of many native populations is due to the practise of inunction with mustard or coconut oil; these oils will be too pungent for the average sojourner, but olive, almond, or other vegetable oils can be substituted. The aims of treatment are much the same as those of prevention. It will however usually be necessary to adopt more aggressive tactics.

All, or as many as possible of, the above precautions should be observed rigidly. After the bath the affected parts must be lathered with antiseptic soap which should be allowed to dry on the skin. The tolerance of each person to each soap must be ascertained. Some sensitive individuals will be "burnt" if "neco" soap, for example, is left on for more than 2 minutes, so that after this interval it must be sponged off; fifteen minutes is usually the time recommended, but in the case of the less sensitive it is unnecessary to wash off the soap at all. 'Asepso' 'Afridol' and 'Ineko' soap are to be recommended. The two former being the better.

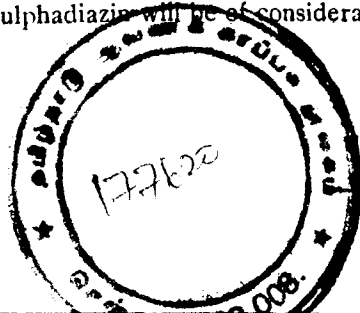
As an alternative to the antiseptic soap and to be applied at other times is a lotion of 1 in 2000 perchloride of mercury in 95 per cent. alcohol to be wiped over the affected part after it has been dried, and itself allowed to dry. (This will not suit all skin). Then dusting powder, consisting of camphor—5 parts, boric acid—200 parts, zinc oxide—300 parts made upto 1000 parts with fine talcum powder, should be applied.

As an alternative to the mercury spirit lotion and powder, a white lotion made up zinc oxide 20 per cent, menthol 1 per cent, camphor 2 per cent, in 80 per cent, alcohol should be dabbed on the affected area with a piece of cotton wool. It is usually inadvisable to apply this more than twice during the day. At other times an aqueous calamine lotion phenol will be soothing.

As in the case of most skin diseases, there is greater danger from over treatment than from under treatment, and whenever any treatment appears to be irritating the skin it must be discontinued and simple aqueous calamine lotion applied.

The sulphonamides have no effect on uncomplicated prickly heat, but for the complication in which pyogenic organisms play a part, sulphathiazole and sulphadiazine will be of considerable value.

(Ind. Med. Rev. XIX 41, '46).



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Vitamin A Deficiency:- Xerophthalmia, Keratomalacia, Phlyctens, Night blindness, Hyperkeratosis, Retarded growth; Preventive against Respiratory, Urinary and Skin diseases.

Vitamin D Deficiency:- Rickets, Osteomalacia, Spasmophilia, Carious Teeth.

As a Reconstructive Tonic:-

Tuberculosis and Pre-tuberculosis, Scrofula, Marasmus, Debility, Malnutrition, Underdevelopment, Anaemia, Cachexia, Anorexia, Convalescence, Pregnancy and Lactation, Wasting diseases, Deficient Dietary with its sequelae.

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