

MEDICO-SURGICAL SUGGESTIONS

Vol. IX.

DECEMBER 1940.

No. 12



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A MONTHLY JOURNAL OF PRACTICAL MEDICINE & SURGERY
Editor: Dr. P. R. Venkappaya.

VOL. IX

DECEMBER 1940

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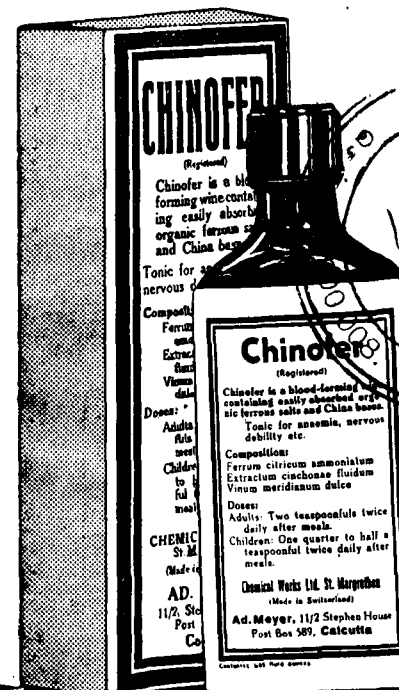
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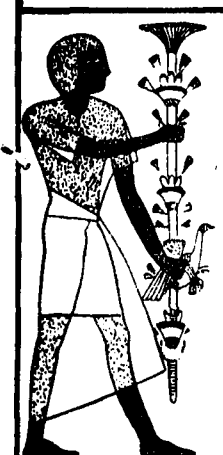
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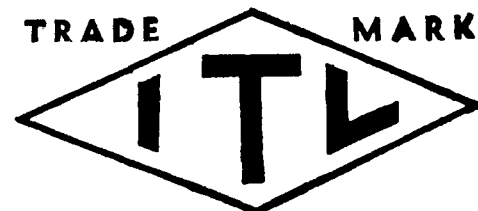
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VOL. IX

DECEMBER 1940

NO. 12

EXPLOITS OF THE SULPHONAMIDE GROUP*

BY PARMANAND AHUJA, M.B., B.S.

Ever since the discovery of this group of drugs the medical literature has literally been flooded with the exploits of this chemo-therapeutic agent. It has enriched the arsenal of therapeutic with a new weapon. It is one of those few therapeutic agents that have found a place in the armoury of the surgeon, physician, specialist and the general practitioner. I am sure every one of you have used this drug in some disease or the other (and the number of diseases in which it is being tried is mounting up day by day) and will come forward to relate your experiences and clinical trials at the end of my talk.

Historical. The idea of discovering specific chemical agents for bacterial infections is as old as bacteriology itself. It was in 1881 that Koch failed to cure anthrax with mercurial salts. In 1904 Ehrlich's success in controlling protozoon infections led to vigorous research in chemotherapy. But it is within the last few years that the sulphonamide group of synthetic compounds has been discovered. In 1908 Gelmo described the preparation of sulphonamide. In 1932 Domagk experimenting with compounds patented for dyeing succeeded in curing streptococcal infections in mice; this was sold as Prontosil.

'Sulphonamide' is a general term and every firm has put up its own trade name like Prontosil (Bayer), Proseptasin (M & B), Bacteramide (G. Loucatas), Streptocide (Evans), Urea-sulphazide (The Urea salt of 4 sulphamidobenzene-azo-1-hydroxy-7-acetyl-amino-naphthalene-3 : 6 disulphonic acid) and Urea sulphazide-T (Acetamido-Amino-Benzene-sulphonate-Urea) (Union Drug Co. Ltd.)

Action. How the drug acts is not clearly understood so far. It is stated that they "act by neutralizing a metabolic function or an enzymatic activity. They affect by invading organisms in the sense of producing a bacteriostatic action which in many cases is not

*(Lecture delivered at the Sind Medical Union.)

Toxic effects. Toxic effects observed due to high doses leading to hyper concentration of the drug in the blood or in the very susceptible persons may be conveniently classified into three categories.

Mild.—Like malaise, depression, headache, vertigo, nausea.

Moderate, like cyanosis, methæmoglobinæmia, sulphæmoglobinæmia, numbness, tingling, abdominal pain, diarrhoea, low grade fever and skin rash.

Severe, toxæmia and Jaundice, leucopenia, agranulocytosis and hæmolytic anæmia.

How to avoid toxic symptoms, and treatment when they develop. Laxatives particularly salines and sulphur containing foods like eggs, onions etc. to be prohibited. Paraffin liquid however is permissible. Analgesia or hypnotic drugs from the aniline and sulphonal groups are better avoided.

If alkalies are given, they enhance the action of the drug and minimise the toxicity. If high doses are given careful watch over the blood picture and other toxic signs and symptoms is indispensable both in the interest of the patient and the professional reputation. But if dosage is controlled within moderate limits, incidence of untoward symptoms could be minimised if not eliminated.

Dr. Deshmukh suggests the use of nicotinic acid 50 m. g. with sulphonamides to avoid unpleasant symptoms.

Preparations like Bacteriamide and Rubiazol with the carboxy group (COOH) and urea-sulphazide with the urea radical produce less toxic effects.

In cases of met-hæmoglobinemia methylene blue by mouth or 1/3 gr. in 10cc. of distilled water intravenously is indicated.

In cases of sulphæmoglobinæmia, colon should be kept free of food residue.

In agranulocytosis, hæmolytic anæmia and other severe toxic manifestations, Sodium Pentanucleotide (Evans) 10cc. twice a day intravenously is advocated.

But as far as possible, appearance of early mild symptoms of toxicity should be a warning to suspend the drug before the grave stage is reached.

In conclusion, I would like to say that we are at present riding the crest of the wave of enthusiasm for the use sulphonamide group and time alone would show whether we are able to retain that position.

DOCTORS PREFER

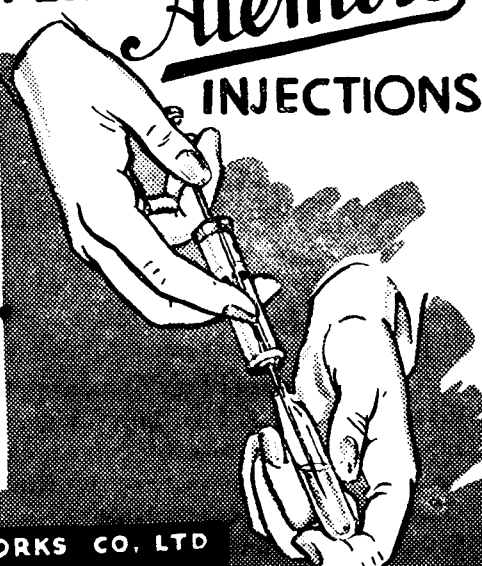
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sufficient to affect sterilization without the co-operation of the defensive mechanisms of the host." But it is all a gamble of words and the whole thing is shrouded in mystery.

I will now give you my own experience of this drug in the following diseases.

Gonorrhea. I have got record of 15 cases out of which three were treated with Uleron (Bayer) and others with ordinary sulphonamide preparations. Alkalies were given simultaneously along with topical treatment in some cases. Discharge was controlled in shorter period with Uleron than with ordinary sulphonamides; the average duration of the treatment in early uncomplicated cases being about 5 days in case of the former and a week or ten days in the case of the latter. Sulphapyridine or Deganan 693 (May & Baker) is reported to be the drug of choice in these cases with or without topical treatment. The general tendency appears to be to dispense with irrigations etc.

Pneumonias. I have record of about 50 cases mostly in children. Out of these ten were treated with Deganan, 693 (May & Baker) and 40 cases with ordinary sulphonamides. Fall of temperature is more rapid with 693 than with the ordinary preparations though incidence of recovery is identical in both the series. The drug is to be stopped after the fall of temperature though general symptomatic treatment is to be continued. If temperature fails to respond in 3 or 4 days to the continuous administration of the drug it will be useless to continue the drug any further.

N.B.—It has been my endeavour to give a trial to ordinary sulphonamides as compared with specific action of its modifications like sulphapyridine in order to test its efficacy with minimum cost to average class of the Indian patient who can ill-afford to go in for the costlier specifics particularly in these abnormal war times when prices have a tendency to soar higher and higher.

Tonsillitis, Pharyngitis and Quinsy. Before pus has actually formed which would require aid of the knife, the administration of the drug in these cases has yielded satisfactory results in my hands. In cases of infective glandular enlargements in the neck in children, the drug is worth a trial in pre-suppurative stage.

Purulent Conjunctivitis. I have a very typical case to report when the whole family consisting of wife, three children and the husband was suffering from a virulent type of infection with profuse discharge, burning, smarting pain and restlessness and photophobia. They had been treated on ordinary lines for a week before they consulted me. I also gave a trial to general remedies for 3 days more before I put them on to sulphonamides and within 48 hours the discharge was controlled and smarting subsided and the children who passed sleepless nights before, were soothed into a restful repose after days of pain and agony. It is reported that topical application

of the sulphonamides in these cases yields favourable results. I have no personal experience of the same. But for the benefit of those who feel interested I may mention 'Eyelex' (5 per cent. Urea Sulphazide) ointment for such eye conditions.

Puerperal Septicaemia. I had a chance to give trial in three cases with success.

Otitis Media. Four cases were treated with good results.

Mastoiditis. Only two cases were treated in the premonitory stage and the administration of the drug parenterally and by mouth succeeded in aborting the attack.

Skin. Moist eczematous eruption in a child of one year following measles and involving practically the whole body was treated with success.

Erysipelas. Two cases were successfully treated.

Streptococcus faecalis and aureus infections of the urinary tract are said to be better influenced by two derivatives of the sulphonamide viz. sulfathizol and sulfa methyl thiazol.

Topical application of prontosil rubrum on *oriental sores* is advocated and dressings of infected wounds and chronic ulcers is recommended. But I personally have not tried in such cases.

Acute Infective Gingivitis. I had an occasion to try in two cases in women. The gums were simply pools of pus and the teeth painful and shaky, and ulcerations present all over the lips, tongue and throat. Previous treatment by well-known dentists had failed to control the disease. Administration of sulphonamides in one case both parenterally and orally and in the other only orally effectively checked and ultimately cured the infection without any loss of teeth etc.

Dosage. Comparatively smaller dosage, than those prescribed by continental writers are required in Indian patients without detracting from the efficacy of the drug. Some favour heroic doses. But with large majority of the general practitioners with whom it is not a practical proposition to control the blood picture and keep a close watch over the patient and who can ill afford to take risks with impunity, the 'safety first' is the method of choice. I am reminded of the wise saying of my professor of medicine, "If your medicine does no good, it must do no harm" which has stood me in good stead all these 22 years of my professional career. Children require 50 per cent., more than adults per unit of body weight.

The duration of treatment may be prolonged by few days but I have had rarely an occasion to face any grave toxic symptoms consequent on administration of sulphonamides to my cases. I have seldom exceeded 6 tablets of $\frac{1}{2}$ gm. each in a day in cases of gonorrhoea and pneumonia and the average dose has been 4 tablets of $\frac{1}{2}$ gm. each in a day.

LEPROSY*

BY DR. J. T. BHATT, M. B. B. S.,

R. M. O. Sheth Vadilal S. General Hospital, Ahmedabad.

Leprosy is a scourge of humanity since time immemorial. The disease is preventable and curable to some extent.

Leprosy is a chronic infective granulomatous disease produced by a sp. bacteria and characterized by lesions of skin, nerves, viscera, eventuating, in local ulceration and great variety of trophic lesions. It is a disease which has a tendency to die out.

Spread.—Hereditary influence plays no part in the spread of the disease. There is abundant evidence from the great leper settlement of Culion in the Philippines where 5,000 lepers live under ordinary family circumstances that inherited leprosy does not occur. Children separated from leper parents at birth have remained healthy to the second generation.

Contagion plays an important part in the spread of the disease. But this should be continuous. Children staying with their leper parents develop leprosy.

Mode of Infection.—The actual mode of infection is not known with certainty. But it is presumed that lepra bacilli escape from lesions of advanced cases of the disease under the skin of persons who come in close contact with infected persons. Abrasions and punctures seem to be the avenue of infection. The bacilli are found in 70 to 90 per cent. of advanced nodular lepers. E. Muir found them in nasal mucous membrane of 37 per cent. earlier skin and mixed type in Calcutta and 3.8 per cent. in nerve leprosy. Ulcers of nodules discharge more bacilli than trophic ulcers of nerve cases. The sites of usual lesions are extensor aspects of extremities, face, parts exposed to pressure such as back and buttocks.

Prevalence.—Found in middle class, crowded localities, slums etc. In India there are about a million lepers. In the world about 4 millions.

Causative organism—Caused by myco bacterium lepra of Harsa an acid fast gram positive rod shaped organism arranged in bunches. It is easily decolorized by dilute acids but less so by alcohol. The bacterium cannot be cultivated except on tissue culture.

Clinical forms.—Leprosy is a systemic disease and is probably in no case confined entirely to any particular tissue. It shows itself clinically in two main forms, which may coexist in one patient:—

- (1). *Nerve* (or maculo-anesthetic) leprosy.
- (2). *Skin* (cutaneous or nodular) leprosy.

* Paper read before the Ahmedabad Medical Society, Ahmedabad.

(1) NERVE LEPROSY.

Two main types:—(a) Macular type.

(b) Acroteric type.

(a) Macular type: Round, oval or irregular areas of skin showing one or more of the following changes:—

1. Loss of pigment.
2. Diminution in cutaneous sensation particularly in the centre.
3. Lack of sweating, and keratosis.
4. Thickening of nerves supplying the macule.
5. Thickening and erythema, particularly at margin.

Of these appearance the two that are diagnostic of leprosy are anesthesia and thickened nerves. There may be only one macule or there may be dozens. They may be very large or very small. They may be found anywhere on the body, may exist for years without spreading, may appear or disappear, may be thick, red and raised at one time and later become flat and pale. Ordinary bacteriological examination of those macules usually gives negative results.

(b) Acroteric type: (Acroteric spreading upwards—from the distal to the proximal portion of the body). This type is due to leprous inflammation of the nerve trunks. The following changes are seen in the distribution of the affected nerves.

1. Impairment of cutaneous sensibility.
2. Loss of pigment.
3. Impairment of sweating.
4. Keratosis.
5. Wasting of muscles with consequent deformity.
6. Trophic ulcers, loss of fingers and toes, etc: frequently there is secondary infection.

The commonest nerves affected are the ulnar (claw hand etc.), the peroneal (drop foot etc.) Other nerves often affected are the superficial peroneal, the radial (drop wrist), the median, the trigeminal (5th cranial) and facial (7th cranial nerves facial paralysis, anesthesia of the cornea, lagophthalmos, conjunctivitis, corneal ulcers, blindness) the great auricular. The affected nerves are usually thickened. In marked cases all the limbs and most of the trunk may be anesthetic. Bacteriological examination of the skin lesions usually gives negative results.

(2) SKIN LEPROSY

The lesions in order of frequency are:—

- (1) Slight diffuse thickening and sometimes erythema of skin.
- (2) Macules differing from the macules of nervous leprosy by not being anesthetic and having an indefinite border.

(3) Nodules.

(4) Ulcers due to breaking down of nodules.

These lesions are most commonly seen on the face and ears and on extensor surfaces and exposed parts of the body. In all well established cases of leprosy, the whole skin of the body is affected, but clinical lesions are visible only in certain sites. Internal organs are found affected. Lungs contain leprosy lesions. Sputum may show lepra bacilli. Lymph glands also contain lepra-bacilli, and they get enlarged.

In skin leprosy, the mucous membranes particularly the nasal mucous membranes, are affected. There is also often iritis.

The course of the disease.—The disease does not usually start with signs of nerve leprosy. In some cases after a period varying from a few weeks to many years, skin leprosy develops, and may increase steadily over a period of years until the skin of the whole body is affected. Unless the patient dies from intercurrent disease, leprosy in time tends to die out, and finally the infection subsides leaving only the scars and deformities. There is at all stages of leprosy a marked tendency to spontaneous arrest which may occur at any stage of the disease, very frequently in the early stages. The whole course of the disease in skin leprosy may be as long as thirty or forty years.

Lepra reaction.—Leprosy is a chronic disease increasing or decreasing extremely slowly but in some cases the condition of lepra reaction occurs with a sudden increase in the signs of the disease. The signs of reaction are as follows:—

1. Swelling up and spread of the old lesions.
2. The appearance of new lesions.
3. General malaise, fever etc.

In nerve cases there is swelling of macules, appearance of new macules, increase in inflammation and thickening of the nerves, increase in acroteric lesions, but constitutional disturbances are slight. In skin cases the infiltrations and nodules of skin and mucous membranes increase, the nodules often swelling up and ulcerating. New nodules appear sometimes in great numbers. There may be profuse discharge from ulcers in the nasal mucous membrane. There is frequently iritis. Constitutional disturbances may be marked, fever up to 104° or 105° and great weakness. Lepra reaction always subsides spontaneously frequently in a few days, but sometimes it takes weeks or months.

Classification of cases.—Cases are classified as N (nerve) or C (cutaneous) and the degree of the disease is indicated by figure 1, 2 and 3. Thus a N-1 case is a slight neural case with one or two macules. C2-N1 means a case with moderate skin infection and slight nerve infection and so on.

Diagnosis.—There are three important diagnostic points in leprosy, namely the occurrence of superficial anesthesia, thickened nerves and acid fast bacilli in the skin. Unless at least one of these three signs is present, do not diagnose leprosy.

Thorough examination is of prime importance. Examine the whole body area in a good light. Look for macules and slightly infiltrated areas of skin. Palpate for thickened nerves. Test for skin sensation. Make bacteriological examination of suspicious areas of skin.

1. Anesthesia usually partial not complete is found in centre of macules of nerve leprosy, or in distribution of affected nerve trunks. Test sensation of light touch by a piece of paper or a feather, and of pain by a pin prick.

2. Thickened nerves (Note: Many normal nerves are palpable. Do not say a nerve is thick merely because you can feel it.). Palpate ulnar, peroneal, superficial branch of peroneal and the skin proximal to macules for thickened cutaneous nerves. Thickened nerves are often tender.

3. Presence of lepra bacilli. Bacilli are rarely found in anesthetic areas or in the macules of nerve leprosy, but there may be a few in the erythematous margins of such macules. In skin leprosy bacilli are always found in the skin lesions and often in the nasal mucous membrane. The methods of examination are as follows:—

In skin lesions (a) The "Snip" method (suitable for examining the ear lobe). With sharp curved scissors snip off a small piece of skin. Hold it in forceps on a slide, scrape off the deeper cellular layer and make a film. Fix by heat. Stain by Ziehl-Neelsen.

(b) The 'Slit' method. Take up a fold of the suspected skin area between the fingers, pressing to prevent bleeding. With sharp scalpel make a slit into the corium. With the point of the scalpel scrape the bottom and sides of the slit. Cells, not blood, are required. Make a smear on a slide, fix by heat, stain by Ziehl Neelsen.

(c) Nasal examination. Inspect nasal septum for lesions. Take an old scalpel and scrape away cells (not blood or mucous) from the septum near the anterior end of inferior turbinate bone. Make smear, fix and stain.

Bacilli if present are usually numerous and may occur in masses.

Differential Diagnosis.—The common diseases which may be mistaken for leprosy are 1. syphilides, 2. tinea. 3. leucoderma, 4. dermal leishmaniasis, 5. psoriasis. Less commonly such diseases as beri beri (polyneuritis), Bernhard's diseases (Neuritis of lateral femoral cutaneous nerve), cervical rib, syringo-myelia, traumatic injury to nerves and other rare nerve diseases may be mistaken for acroteric nerve leprosy, and skin diseases such as morphea, erythema nodosum, lichen planus, occupational dermatitis (Jute) may

simulate macular or skin leprosy. These diseases may be differentiated from leprosy, (a) clinically, in few of them is there any anesthesia and if present it is atypical in distribution, in none of them is there palpable thickening of nerves and (b) bacteriologically—in none of these diseases are acid-fast bacilli found in the skin.

Leprosy and another disease may coexist, for example, leprosy and tinea or leucoderma is very common, leprosy and syphilis is occasionally seen.

Miscellaneous points in diagnosis.—In addition to diagnosing the presence or absence of leprosy, the following points should also be determined. What is the type of case, if neural, is the disease active or inactive? Is the patient infectious? In deciding these points a careful history, a thorough examination, clinical and bacteriological, are necessary.

Prognosis.—In considering the prognosis the following points are to be considered :—

1. The age of the patient.
2. The type and duration of the disease.
3. The general physical condition of the patient.
4. The resistance of the patient as shown by the sedimentation and leprolin tests. Strong reaction points to a good prognosis.

In childhood the prognosis is worse than in adults. In skin leprosy the prognosis is worse than in nerve leprosy. In debilitated patients prognosis is worse than in well nourished active patients. When the sedimentation index is high and the leprolin test gives a negative result, the prognosis is comparatively poor. If the disease is diagnosed early or it has been present in a mild form for a long time with little or no increase, the prognosis is favourable. By a favourable prognosis we mean that the disease will under favourable conditions probably increase little if at all and will become inactive though there may remain some deformity. By an unfavourable prognosis, we mean that disease will remain active for a much longer time and if it does finally subside is likely to cause much more disability and deformity.

Treatment.—Cases suitable for treatment.

- (a) Cases of active nerve leprosy.
- (b) Cases of skin leprosy except very advanced cases.

Cases unsuitable for treatment or not needing treatment.

- (a) Inactive arrested nerve leprosy (either macular or acroteric.)
- (b) Very advanced cases of skin leprosy.

In judging whether the disease is active or not a careful history and a careful clinical and bacteriological examination are necessary.

The following are signs of activity.

- (a) History of recent increase in size of number of lesions.
- (b) Definite inflammation of nerves.
- (c) The presence of lepra bacilli in the skin.

GENERAL TREATMENT.

This is the most important part of the treatment.

- (a) Complicating diseases. A thorough clinical and if necessary laboratory examination. The following diseases are often found complicating leprosy :—Syphilis, hook-worm, chronic malaria, chronic amebiasis. These must be sought for and if found should be treated.
- (b) Diet. An ill balanced diet often aggravates leprosy. The rice diet should be modified to include more proteins, fats, and vitamins.
- (c) Regime. Sufficient exercise and sufficient sleep are essential. Irregular hours, over-work, sedentary occupations are bad. Hygienic living conditions and fresh air are important.

SPECIAL TREATMENT.

(N.B.—There is no specific treatment for leprosy). Many different forms of treatment have been advocated in leprosy, but general opinion of experienced workers is that the best form of treatment is the administration of some preparations of hydnocarpus oil preferably by injection.

The following preparations are in common use.

- (a) Hydnocarpus oil (pure) with 4 per cent. creosote.
- (b) Ethyl esters of hydnocarpus oil with 4 per cent. creosote.
- (c) Sodium hydnocarpate 1 per cent watery solution with 5 per cent carbolic.

The ethyl esters are usually preferred to the oil, but it is more expensive.

The oil is more viscid and more difficult to inject; warming reduces viscosity. Sodium hydnocarpate is little used in India. Supplies must be obtained from a reliable source.

DOSAGE AND REGULATION.

The dose for an adult should be 1 c. c. to begin with and increase to 5 or 6 c. c. once or twice a week. If there is excessive local reaction or pain the dosage should be reduced or a different preparation should be tried. If lepra reaction occurs, injections should be stopped until it has subsided, and then only small doses should be given. The sedimentation index may be used as a guide to dosage.

METHOD OF INJECTION.

The oil and ethyl esters are preferably given by a combination of intradermal and intramuscular injections.

Intramuscular injections should be given deep into the upper part, of the gluteus maximus muscle.

Intradermal injections are given into the dermis of the lesions themselves. A special short intradermal needle is advisable. The needle is inserted at an angle about two millimeters into the skin

and about 1 c. c. is injected raising a little wheal in the skin. Similar injections are given at about half inch intervals over the whole lesion to be injected. The small swellings at the site of injection should subside in two or three days. The lesions should not be injected a second time until three weeks have elapsed. If the lesions are not more than two or three cubic centimeters intradermal injection at one time is given, the remainder (if any) of the dose ordered being given by intramuscular injection. Recently it has been found that snake venom is given in regulated dosage.

Lepra reaction may be caused by excessive doses of injection. When reaction occurs injections of hydnocarpus preparations should be stopped. Mild reactions subside in a few days without any treatment. Severe reactions with fever etc. should be treated as follows. Put the patient to bed, give a purge, and light diet with plenty of fluids, analgesics to relieve pain. Small doses of antimony (P. A. T. '02 gramme) dissolved in 1 c. c. saline-intravenously every other day may be useful in assisting the subsidence of reaction.

EXTERNAL TREATMENT OF LESIONS.

Considerable improvement in the size and appearance of lesions may be obtained by local counter irritation. The most convenient method of applying this is by watery solutions of trichlor-acetic acid diluted 1 in 1, 1 in 2, and 1 in 3. The solution is painted on the lesions with a brush or plug of cotton wool in forceps, the stronger solutions being used for touching nodules and weaker solutions for larger areas of skin. The application should produce a local caustic effect causing loss of the epidermis but not ulceration.

DIATHERMY.

Diathermy is sometimes useful for the treatment of neuritis which may be very severe.

TREATMENT OF IRITIS.

Keep pupil dilated with atropine, warm boric compresses, eye shade, darkened room.

SURGICAL TREATMENT.

- (a) Excision of nodules. Unsightly nodules may be excised.
- (b) Operation on trophic lesions. Necrosis of bones of fingers and toes often with gangrene and secondary infection, may necessitate amputation of digits or part of digits. If neglected may cause loss of hand or foot, arm or leg, or of life. Trophic ulcers frequently have dead bone underlying them, the removal of which will enable the ulcer to heal. The operation should be radical and may involve the loss of digits.
- (c) Operation for neuritis. This is most often done on the ulnar nerve above the elbow. The pain is due to pressure on inflamed nerve bundles inside the nerve sheath. The nerve is exposed and the nerve sheath is freely

incised or preferably—entirely removed over the area of thickening and inflammation.

ASSESSING RESULT OF TREATMENT.

Improvement under treatment is usually slow, often very slow. Assessing results of treatment needs careful observation and records. Careful body charts of the patient before, during and after treatment are necessary. These charts should record the findings of careful clinical and bacteriological examinations. Under, favourable conditions with careful prolonged treatment the following results may be obtained.

- (a) The progress of the disease is arrested and no new lesions appear.
- (b) The lesions already present become less noticeable and may entirely disappear.
- (c) The anesthesia becomes less extensive, but there is often some permanent anesthesia due to destruction of nerve fibres.
- (d) In the less severe forms of skin leprosy after prolonged treatment, the bacilli becomes less numerous and may disappear.

DURATION OF TREATMENT

This depends on the type and severity of the case. In slight macular nerve leprosy (N) the active signs may disappear in a few weeks or months. In skin leprosy treatment has to be continued for years.

In all cases when signs of activity have disappeared the patient should if possible be kept under observation, and examined every few months so that any tendency to relapse may be at once detected.

IS GONORRHEA A BACTERIEMIA?

BY WALTER M. BRUNET, M.D.,

Chief of Clinic, Public Health Institute, Chicago.

A single glance backward on the subject of gonococcus infection will disclose the stern truth that little progress has been recorded in the study of fundamental pathology of the disease within our generation. The reasons for this state of dormancy are due primarily to our inability until recently to successfully inoculate laboratory animals in any routine manner and study the infection in a comparative fashion, and secondly to a lack of skilled investigators, and thirdly inadequate financial support. We have also been guilty of

worshipping for too long a time at the shrine of those pioneers who blazed the trail and this is exhibited by our continually repeating their statements and accomplishments almost without question in our presentations. Very rarely does one physician or a group of physicians declare with clear, loud tones a whole important chapter, or even a paragraph in the record of science. More often the start is made from an isolated and timid voice making an inspired observation and raising a stimulating question. We must turn our thoughts forward and cease fighting the last war on gonorrhea over again and plan a new strategy for the modern conquest.

We are in a ferment at this time, I trust, of some practical adjustment of our conception of gonococcic infections and their treatment and control. We must modernize our concept of the disease and begin the new attack which should bring us nearer to the goal.

All organic functions are modified as soon as bacteria or viruses cross the frontiers of the body and invade the tissues. When disease attacks cells the characteristics of the pathological changes are dependent upon the reaction of the body to the insult. Fever is one of the usual and frequent responses of the body to bacterial invasion. Other adjustments in the body are determined by, and dependent upon, the production of materials—toxins—from the growth of bacteria on the tissues, and by the accompanying disturbance of the activities of the various organs and glands and the lack of certain substances which may interfere with general nutrition and resistance of the individual. The resistance of the body to pathogenic invasions concerns two offensive methods of the economy: on one hand it opposes the entrance of bacteria and attempts to destroy them, and on the other it repairs the lesions caused by the organisms and neutralises the poisonous products of their growth, thus overcoming them. Many human beings have an apparent susceptibility to some infections and others are quite immune. The state of so-called resistance is due to unknown constitutional factors which prevent or oppose the penetration of bacteria or destroy them when they have gained an entrance. This is called natural immunity and it may protect certain individuals from almost any disease. There is also an acquired resistance to disease which may be spontaneous or artificial. The body reacts and adapts itself to bacteria and viruses by the production of substances capable either directly or indirectly of destroying the invaders. This tissue fluids, leucocytes and other cells have the major part to play in this complex and little understood process. Fundamental research has demonstrated that the responses of the organ to invading bacteria are due to unknown chemical substances. It has been shown also that complex polysaccharide substances usually present in the protoplasm of certain bacteria determine the specific reaction of the cells and tissues when

they are united with the protein. The tissues of the body also manufacture carbohydrates and lipoids under special conditions which conceivably serve a defensive function. It is presumably these substances which give to the human organism its power to attack foreign cells and proteins and render them harmless. When an infection begins the blood and lymph are modified by the bacteria and by the toxins set free as a result of this process, and by the waste products of the diseased organism. In disease there are alterations in the entire body and an acceleration of the chemical changes. There are infections which confer a lifetime immunity once they have been contracted and other conditions which like malignancies cause slight response from the body and they are seldom if ever controlled by the organism. So far as our knowledge extends there exists no immunity to the gonococcus either natural or acquired from previous infection or by individual resistance, or racial qualities although there are scores of men and women who have admitted repeated exposures to the disease and yet deny ever contracting it. These escapes may be explained upon the assumption that there were no free gonococci upon the mucosal surface at the time of the exposure, or that both parties were infected with the same strain of the organism and therefore could not be superinfected.

Gonorrhea in the male is an infection of the seminal ducts and any part or all of the system which is subject to attack may be involved. In the female the disease is more serious due to the complexity of the genito-urinary structures. The primary attack is regional in both sexes but the organism, or its toxin, can involve any susceptible tissue in a systemic invasion. Ricord many years ago said that we knew when a gonorrhea begins but God only knows when it would end. From a historical viewpoint this statement is interesting but not now true, for with sulfanilamide, sulfapyradine and allied compounds plus hyperpyrexia the disease on the whole is a short lived one.

In any large urological clinic the number of men and women experiencing potentially serious, and disabling extensions, will average between 40 and 60 per cent. of all admissions. In the male nearly 50 per cent. will have a posterior infection, and of these from 5 to 10 per cent. will suffer an epididymitis, vesiculitis and prostatitis, and 1 per cent. will have joint involvement, an occasional patient will experience lymphangitis, peri-urethral abscess and rarely metastatic ophthalmia will be observed. In the female about 40 per cent. will have a complication and of these the largest number will suffer a pelvic extension, about one-half of 1 per cent. will have arthritis, or tenosynovitis, and an occasional patient will experience an extension to the right upper abdominal quadrant resulting in a perihepatitis. Women are apparently less susceptible than men to arthritis, and metastatic ophthalmia. We have observed one death

from a gonococcic endocarditis in a male patient and this represents the admissions of 35,000 cases of gonorrhea over a ten year period. The rarity of this complication in urological practice is more apparent than real. No informed physician ever minimises the dangers of a gonococcic infection, and especially in the female, for the physiological function of menstruation adds enormously to the possibilities of upward extensions and permanent residuals.

There are striking similarities between the extension of a gonococcal infection to the fallopian tubes and the seminal vesicles, and in writing of the latter condition Belfield coined the phrase "pus tubes in the male" to describe this complication. Many gynecologists have written and taught that the gonococcus reaches the tubes through the fundus of the uterus, the mucosa being a bridge for their passage, and urologists are instructing students that the gonococcus reaches the epididymes through the vas deferens by way of the vesicles and ejaculatory ducts. If the gonococcus is a "surface rider" why is it that specific endometritis is so infrequent, and that vasitis is rarely observed in the most acute epididymal involvement.

Bolnick in his studies has demonstrated that it is impossible to force fluids by high and prolonged pressure through the vas beyond the tail of the epididymis. He explains that were the infection in the epididymis of hematogenous origin the head or body of the organ would be attacked first rather than the globus minor. The globus major is often the first portion of the part involved in a gonococcal infection for it is at this site that the swelling is often first palpable and the pain localised. The testicle or ovary themselves are seldom involved unless the inflammatory process be a very extensive one and in these instances the entire organ may be destroyed.

There are too many physicians who in their zeal to cure gonorrhea fail completely to consider the pathology of the infection or to grasp the fact that the tissues which are vulnerable are those covered with columnar epithelial cells. It has been demonstrated that the fewer the epithelial layers the more violent the invasion. The diplococci rapidly penetrate the superficial cells and cause a local tissue reaction, and the cellular response is usually noticed subjectively in 48 to 72 hours, and in the large majority of patients a full blown infection is well established within five days. The gonococcus elaborates toxins, presumably both of an intra and extra cellular nature, which are considered to be the real cause of the symptoms, but there are elements in the implantation and growth upon the urethral mucosa and the tissue response which are not fully understood. Pelouze states: "It is not the presence of, but the death of, the gonococcus that produces the phenomena of the disease." Is it? This author has demonstrated that upon the death of the gonococcus in vitro a poison highly lethal to its growth is

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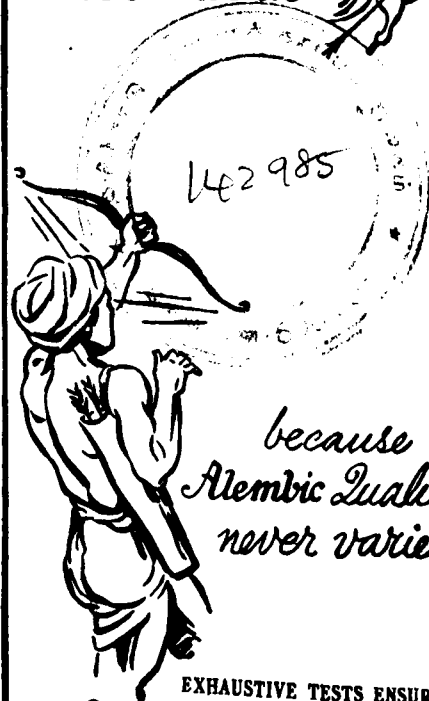
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produced and when a very weak solution is injected into a normal urethra it will speedily produce a purulent discharge which will spontaneously subside in a day or so. This lytic substance will resist high temperatures and it is not destroyed by strong solutions of bichloride of mercury, or phenol. The theory propounded by d'Herelle that every bacteria has its own 'phage which ultimately controls the disease has been disappointing, and not proved clinically insofar as gonococcal infections are concerned. To produce an immune substance for a disease in which no demonstrable immunity is ever developed would be a scientific discovery of the first magnitude.

In the management of gonorrhea prior to 1933 physicians have proceeded to the control and cure of the disease on the grounds that the infection is a superficial one and the manner of extension by continuity of surface. In the decade of great medical tranquility following the first world war, treatment by widely advertised tissue penetrating antiseptic dyes was advocated wholly upon this basis. We know they fell far short of the claims made for them but perhaps the color had a beneficial psychic effect so they were not wholly valueless. When a gonococcal infection is diagnosticable the organisms have already reached the submucous tissues, and this often occurs within 48 hours after exposure. After the disease is well established, at the fifth day, the deeper tissues will show characteristic changes in the wall of the capillaries, round cell infiltration, serum exudation and limited phagocytosis. The actual function of the white blood cells in gonorrhea appears to be that of a "bouncer" as they envelop the invaders in their cytoplasm and in this manner they are discharged from the body. These intra leucocytic gonococci are alive, can multiply and produce a new infection upon suitable soil. Some writers have even expressed though that the phagocytes are a hindrance in that they interfere in some manner with recovery from the disease. However, our knowledge of the function of these cells and their action in this disease is most rudimentary. The superficial epithelial cells are cast off after the infection is established by pressure from the exudate or the action of the toxins or both, and the denuded areas are covered by layers of epithelial cells.

Frequently the deeper structures of the urogenital tract in both sexes are invaded and these extensions together with remote lesions are considered complications. Are these manifestations of the disease true complications or but the expression of the infection in a host whose combative forces are altered in some manner? A patient who once has had an extension of the infection will often experience a congeneric disturbance with a succeeding attack. Distant foci such as iritis, arthritis, perihepatitis and endocarditis are usually discovered in sufficient numbers for a skilled clinician to make a

prompt diagnosis and apply appropriate treatment. How many physicians consider the manner in which the infection reaches these organs? Can these far off lesions be explained upon the basis of continuity or contiguity of tissues? In the posterior extensions in the male is it conceivable that gonococci march through the posterior portion of the urethra to the openings of the prostatic and ejaculatory ducts and invade these organs and continue their journey down the vas to the epididymes and not cause any local disturbance enroute? The infection cannot spread by continuity along the mucosa from the urethra to the epididymes because the dissemination is too rapid. Neither does it travel along the lymphatics for there is no continuous system from the vas to the epididymis. If the sheath of the vas were infected, then a periorchitis would develop for the covering of the vas extends only to the tunica vaginalis which it envelops. In the Female the bacteria are thought to seek the lining of the tubes by passing through the uterine fundus and leaving no damage in their wake. Just a short cross country like! Vaginitis and endometritis caused by the gonococcus are among the incurable in medicine yet in almost any text on urology or gynecology which one may select statements to this effect will be found. The glandular tissue of the uterine fundus is susceptible to attack from gonococcus but why is it that such pathology is not found on pelvic examination or during abdominal operations. There have been 100 cases of gonococcal perihepatitis reported in the past decade and although this complication is more often missed than diagnosed it is potentially serious and many patients are operated upon and a correct diagnosis made at the time of the section. How does the gonococcus reach the right upper abdominal quadrant, by continuity of tissue, the lymphatics or the blood stream? Despite the remarkable advance in the chemotherapeutic attack on the disease the distressing complications and their sequelae will far outnumber, especially in the female, the residual defects of poliomyelitis upon whose victims more financial support is lavished than can be productively spent.

Is gonorrhea a bacteremia? The question is not answered for there is no technic known whereby the gonococcus can be readily recovered from the blood stream and even in well established cases of endocarditis only rarely are the organisms grown in the laboratory. Does the gonococcus of Neisser have a life cycle of which we are wholly ignorant? Studies of the dissociation of the gonococcus have shown that the typical form of the gonococcus may undergo dissociative changes which include pleomorphic forms, gram-positive diplococci, micrococci and beadlike bacilli; these dissociative changes are accompanied by alterations in the fermentation of sugars and by changes in serologic reactions. Dissociative forms may be reverted to the parent type. The changes described may be

comparable to those occurring in the human body during the course of gonococcus infection.

As progressive physicians planning a new strategy for a final assault against an old enemy we are in need of general improvement in diagnostic procedures, treatment methods, more careful case control and contact discovery and adequate appropriations for fundamental research projected on a long term basis. In gonococcal infections we look for the day when our basic pathology will catch up with our therapeutics, our wisdom with our scientific knowledge and our purposes with our powers. Then at last we shall have conquered gonorrhea.

Paper read at the Sixth annual meeting of the American Neisserian Medical Society in New York, on 11th June 1940.

STERILITY IN THE MALE.

ROBERT W. DICKSON, M.D.

Denver.

A sterile union is often the cause of much discord and occasionally of divorce. It is also becoming a serious economic problem because of a falling birth rate produced by both an involuntary and voluntary sterility. Aside from the usual methods employed against conception, vasectomy has recently become very popular. This is often done indiscriminately on all ages. There is a popular opinion, especially among young virile individuals, that continuity can be established easily and at any time. Quite the reverse of this is true.

From time immemorial to the present century, the wife has borne the blame for childless marriages. Only for about the last twenty years has the question of male fertility been studied with any degree of thoroughness. Heretofore, the wife has been subjected to numerous tests, all expensive and occasionally harmful, before a proper examination of her husband has been made. The examining physician is not always to blame, however, because many males are unco-operative in that they refuse to submit to a proper examination.

It is estimated that 10 per cent. of all married couples are sterile. Meaker says only 10 per cent. of these husbands and 5 per cent. of wives are free from all objectively demonstrable evidence of fertility. Dickinson and Carey found that 31.5 per cent. of 1,763 sterile unions were due to poor semen. Mazer and associates found in a study of 389 barren marriages that 40.6 per cent. of the males had various types of

semen abnormalities. Other investigators state that complete or partial infertility accounts for nearly one-half of sterile matings. These figures stress the fact that to ascertain the cause of sterility the examination should begin with the male.

The etiology is often obscure, but presupposing the male is sexually capable and his mate is perfectly normal, an examination of the seminal fluid will likely reveal an aspermia, an azoospermia, an oligospermia, a necrospermia, or the semen will contain blood or pus or other pathological constituents. Aspermia is that condition in which no semen is ejaculated. This is rare and is usually due to a urethral stricture or to an inflammatory destruction of the prostate and seminal vesicles. In azoospermia, the spermatozoon is not demonstrable. This condition is found in about 15 per cent. of cases and is usually caused by a bilateral obstruction of the epididymis or to faulty spermatogenesis. By oligospermia we mean a deficient number of sperm. In necrospermia, the spermatozoa are all found to be dead.

The common cause of obstructive sterility is stated to be bilateral gonorrheal epididymitis. This usually results in an inflammatory closure of the lower end of the epididymes, and less frequently of the ejaculatory ducts or vasa. Partial obstruction may result in oligospermia. In my experience, as well as that of others, the occurrence of male sterility as a direct result of a previous case of gonorrhea is greatly exaggerated. Genital tuberculosis or severe traumatic epididymo-orchitis may also cause an obstructive sterility. It has been my observation that male sources of sterility are usually the result of a general constitutional depression far overshadowing local factors. By this I mean such disorders as anemia, low blood pressure, or a low metabolic rate. The obvious defects in spermatogenesis are caused by cryptorchism, traumatic orchitis, mumps and syphilis. Remote devitalizing factors such as sexual exhaustion, physical fatigue, prolonged emotional stress, and endocrine disturbances likewise cause faulty spermatogenesis. Certain constitutional diseases—namely, prolonged fevers, bacterial toxemias from any foci of infection, especially the teeth, tonsils, or sinuses—will cause a temporary cessation of spermatogenesis.

As in every study, a complete history is necessary. First, of the family, which should include the physical and mental condition of both the parents and grandparents and of both husband and wife. Second, the personal history, including such diseases as parotitis, tuberculosis, syphilis, gonorrhea, rheumatism, mental diseases, and accidents. Third, the sexual history, which should include the habits and sexual life of both husband and wife before and after marriage. The physical examination is also important and should include the type of constitution, the mental and physical health as well as the primary and secondary characteristics. The next most

important step is a competent examination of the seminal fluid to complete the picture.

Before an examination of the seminal fluid is made there should be a pause in intercourse for at least a week. I wish to condemn the practise of examining only the fluid obtained by prostatic and vesicular massage. Too often this test may fail; and too often are we prone to give the male a clear bill of health if a few live spermatozoa are found in either a condom specimen or one taken directly from the vaginal pool. I prefer the collection of the specimen as advocated by Huhner which comprises the examination of the cervical and vaginal secretions following intercourse. This should be done as soon following intercourse as possible, as it has recently been found that the endurance of normal spermatozoa in the vaginal canal is between three and four hours post-coitus. I am not convinced, however, of the advisability of having a bedroom adjacent to the doctor's office as advocated by some. A condom specimen is satisfactory if kept at body temperature. It should be washed and dried thoroughly, because some powders used are detrimental to the life of spermatozoa. The ejaculate may also be collected in a clean wide-mouthed bottle. This also should be kept at room or body temperature.

Normal semen is a mucinous fluid of about a drachm in quantity. Smaller quantities are often pathologic, whereas larger ones are not. An increase in viscosity or a very thick fluid means an increase in mucus produced by an inflammatory reaction in the genital tract and thus is pathologic. The reaction is mildly alkaline. Microscopic examination of a drop of semen placed on a slide should be "alive" with active, motile sperm. If kept moist and at room temperature they should remain alive for many hours. Repeatedly I have kept normal sperm alive for twenty-four hours, whereas if kept at body temperature they will die within ten hours. If, after repeated examination, no sperm are found, then it may be necessary to centrifuge the specimen. If this fails, we can then say we have an azoospermia. Normal semen contains 75,000,000 to 100,000,000 sperm per cubic centimeter. The count is made similar to that of counting red blood cells. Most observers agree that a count of 60,000,000 or less is indicative of infertility. Some necrosperm and fragmentation will be found in any normal specimen. Mazer says that a necrosperm of 20 per cent. means infertility. Numerous double-headed and monstrous forms are also indicative of faulty spermatogenesis.

As to prognosis, various investigators such as Cary, Wolborst, Read, Dickenson, and others, report from 20 to 47 per cent. cures; however, if only a small percentage of cures were effected. I believe it worth the effort and patience expended.

Treatment of the obstructives of sterility, of course, require some types of urological or surgical procedure. For obstruction in

the lower poles of the epididymes, an anastomosis or an epididymovasectomy, as devised by Martin and later altered by Hagner, is done. In some hands this procedure gives very good results, in others it is a complete failure: nevertheless, these procedures are worth trying.

In the treatment of non-obstructive sterility, it is often difficult to determine which is responsible—endocrine failure, depressed constitutional states, or the role played by infection; therefore, careful study and close co-operation of the patient is required. In general the treatment should consist in the eradication of all foci of infection, especially that found in the teeth, tonsils, sinuses, prostate, and vesicles. General hygienic measures, which include mental rest, outdoor exercise, elimination of drugs, alcohol, and excessive smoking, should be instituted. Also, in some cases the curtailment of sexual activity is necessary; in others, complete sexual rest is imperative. A well balanced diet high in calcium, vitamins, and proteins is prescribed.

Stockmen have known for years that feeding plenty of wheat and rye will cause their animals to come in heat sooner than normal, and feed their breeding stock accordingly to increase their fertility. I make it a practice to give wheat germ oil in 5 to 10 c.c. doses daily during the course of treatment.

The endocrines will undoubtedly play a more important role when our knowledge about them increases. Much has been claimed by the manufacturers of testosterone propionate as an aid in treating sterility. It has been of no value in the few cases in which I have used it. At best its efficacy would only be temporary.

Recently there has become available a gonad stimulating hormone of pregnant mares' serum, which I do believe will prove of some value. Its principal indication would be in those cases of testicular hypofunction due to inadequate pituitary stimulus.

Some observers report favourable results with anterior pituitary-like substances, but others, like Herman, have had no results whatever. I believe it is a distinct and in some cases, at least many patients have informed me they felt considerably better, and even if its use is only psychic in nature it is of benefit.

In those cases due to endocrine or metabolic disturbances, such as hypothyroidism, cryptorchism, or generalized obesity, the use of thyroid extract is definitely indicated. Thyroid is one glandular product which when taken by mouth is of value.

In treating sterility, I do not believe we pay enough attention to the prostate and seminal vesicles. Numerous observers as well as myself have noted that regular prostatic massage, as well as stripping the seminal vesicles, even if infection is not present results in an increased tone of those organs which produces a reflex stimulation of spermatogenesis and thus an increase in sperm count.

Conclusion

Because of such a high incidence of male infertility (nearly 50 per cent.), the examination should begin with the male in all questionable cases of sterile unions.

The etiology, which is often obscure, is usually due to a faulty spermatogenesis. Obstructive sterility can be caused by bilateral gonorrheal epididymitis, genital tuberculosis, or severe traumatic epididymo-orchitis. General constitutional disorders far overshadow the local factors.

A competent examination of the seminal fluid is absolutely necessary. Also of importance is the collection of the specimen. A prostatic massage secretion should not be relied upon to give accurate information. Normal semen contains 75,000,000 to 100,000,000 live spermatozoa. A count of 60,000,000 or less is not conducive to fertility. The prognosis as yet is not good. Cures of 20 to 40 per cent. are reported.

Treatment of the obstructive types of sterility require surgical procedures. In the treatment of the non-obstructive types of sterility, special attention should be given to eradication of all foci of infection, hygienic measures instituted, and the combating of all depressed constitutional disorders. Endocrines and glandular products are given when necessary. Regular prostatic massage is recommended to increase its tone and aid in the reflex stimulation of spermatogenesis.

(Rocky Mountain Medical Journal, September, 1940.)

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MEDICO-SURGICAL SUGGESTIONS

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THE WAR AND THE MEDICAL PROFESSION.

The first anniversary of the Second Great War has come and gone and the conflict seems nowhere near its end. It has dislocated the entire human economy and disorganised all professions and trades. The medical profession has also its due share of dislocation. Medical supplies and drugs from abroad are difficult to obtain and slow to reach this country. Drugs of German and Italian manufacture are not available and this fact has given the much needed fillip to the chemical and pharmaceutical industry in India. British firms and some Indian firms too have manufactured good substitutes for articles which formerly were produced only in Germany. These are a few of the silver linings in the dark clouds emanating from the conflagration in Europe and menacingly blowing eastwards.

The medical profession in England was well prepared for the war conditions several months ahead of the actual outbreak of war. The British Medical Association took a hand and collaborated with the Ministry of Health in arranging for beds for the reception and treatment of the war wounded. The patients in the London hospitals were sent to hospitals in the provinces to make room for casualties in the metropolis, which is the main target of enemy attack. The whole of Britain was divided into convenient zones and medical personnel was distributed according to requirements. The voluntary hospitals were taken over by the state and staffed by the state, thus initiating a sort of medical conscription. Medical men from the "evacuation areas" were directed to serve in the "reception areas." The medical practice of several practitioners were disturbed but they willingly put their shoulders to the wheel in the interests of national service. Through the good offices of the British Medical Association, junior practitioners were enabled to attach themselves to senior practitioners. In addition, many medical men took up regular commissions in the army medical service. In a word, one and all in Britain, medical men and non-medical men, have braced themselves and banded themselves together, and are working in a co-ordinated and orderly fashion—thanks to the co-operation of the

state, the medical organisation, and the public—and march forward to withstand and overcome the horrors and sufferings of a modern war with all its air raids, black outs etc. One cannot help giving expression to feelings of sympathy and admiration of the fortitude and courage with which the people in Britain and Londoners particularly, are spending their days and nights in such a nerve-racking atmosphere.

In India the European armageddon has its repercussions. The prices of drugs and other commodities have soared high. Trade and industry has come to a standstill. The burden of taxation has increased and "the money market is dull." The average man's income is just sufficient to meet his normal expenditure, and he would spend on medical relief only when absolutely unavoidable. Medical practitioners who were hitherto considered glowing examples of success are hard put to maintain their former level of income. It is discouraging to the junior practitioner on the threshold of his career to find that it is a very hard job to make a bare living. It is sad to see senior practitioners who had seen their palmy days long ago and who have still to work in their old age. The granting of emergency commissions in the I. M. S. to the products of our medical institutions is a step in the right direction, but several such steps are needed to promote the welfare of the medical profession in India. Among the "independent" medical practitioners there is dissatisfaction, discontent and dismay, and in the absence of any extraneous aid, they are fighting their life's battles in their own way in a spirit of self-help and self-improvement. Many of them just carry on with philosophic resignation, sans patients, sans work, sans income, waiting patiently for better times—probably waiting for the dawn of a new era after the peace and the subsequent reconstruction of the world.

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A TALK ON THE RECOGNITION OF THE NORMAL HEART.

F. A. WILLIUS, M. D., *Section on Cardiology.*

Much emphasis is accorded the recognition of the diseased heart, but few discussions regarding the recognition of the normal heart find their way into medical literature. However, the physician's uncompromising verdict to his patient that the heart is not impaired by disease, a verdict attained by means of careful examination is one of the most satisfying experiences to all concerned. It is toward the willingness of the physician to make this diagnosis under justifiable circumstances that I wish to direct this discussion.

Many instances occur in which the physician appears reluctant to express the opinion that the heart is normal. The explanation of this attitude is found in several sets of circumstances. He may lack confidence in the accuracy of his observations or their interpretation. In spite of the absence of incriminating symptoms and signs his reluctance to tender a positive opinion may be motivated by the fear that serious disease may be present yet undetectable. Again, in the absence of evidence of disease the physician is hesitant to commit himself to the verdict that the heart is normal on the presumption that if the heart is normal now, disease may supervene later and he may thus be censured for not having recognized the abnormality at an earlier time.

Indecision of this character is lamentable because it comprises that form of practice which contributes so generously to that ever-existent horde of cardiac neurotic patients who become so convinced of nonexistent disease that their very existence becomes a horrible eternity of fear and uncertainty. Perfection in diagnosis is as yet not one of the attributes of mortal man and the physician must accept this premise, unhappy as it may be, and willingly accept the hazard of error when made in a conscientious attempt to help rather than to try to avoid such error by always leaving a portal of exit open at the expense of human anguish. Little doubt exists that more errors in diagnosis occur as results of such evasive methods than as the results of the method of conscientious conviction.

Let us presume that the patient has been carefully studied, an accurate history has been elicited, a thorough physical examination has been conducted and pertinent laboratory adjuncts have been correlated. Then let us consider various subjective and objective parcels of evidence that frequently are the basis for the false indictment of the healthy heart.

One of the most prominent symptoms of the diseased heart is dyspnea, yet too many times the physician accepts the complaint of shortness of breath as genuine when in reality it is counterfeit. It

however, it has been found too irritant to comfort, and the practice has been to give the ordinary sulphur ointment treatment in such cases until the pustules dry up. Then the case is treated on the same line as a papular case. The course of treatment is as follows:—

- (1) Paint the whole body below the neck with a 40 per cent. solution of the sodium thiosulphate (hypo).
- (2) Allow it to dry for about 15 minutes.
- (3) Paint with a 5 per cent. solution of hydrochloric acid (over the hypo).
- (4) Allow it to dry for about 15 minutes.
- (5) Repeat the above-mentioned treatment after an interval of two hours.
- (6) Give fresh linen.

The 40 per cent. hypo solution and 5 per cent. hydrochloric acid solution are used in all cases except for children under 7 years of age in whom half the strength of the above solution is used.

Review.

THE ANTISEPTIC. October and November 1940, Special Number on Respiratory Diseases, Parts I and II, 323, Thambu Chetty Street, Madras.

Our distinguished contemporary has brought out these two issues with numerous articles on respiratory diseases. The common disorders of the entire respiratory tract, from the pharynx and larynx down to the lung alveolus, are discussed by the various writers, and the November issue contain some articles on tuberculosis of the lungs. A perusal of the articles in the two issues constitutes a good refresher course for the busy practitioner. This special number keeps to the usual standard that one is accustomed to see in the pages of the *Antiseptic*.

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Medical News & Notes.

THE SOUTH KANARA DISTRICT MEDICAL ASSOCIATION.

The 10th Annual General Meeting of the South Kanara District Medical Association was held on 23-11-1940 in the Canara High School, Mangalore. Captain W. E. Mascarenhas, President, occupied the chair. The annual report with audited statement of accounts was read and adopted. For the first time since the starting of the Association, the General Body elected the following Honorary Members—all belonging to the District, who have made a mark for themselves in their career, highly respected both by the public and the profession.

(1) Dr. U. V. Nayak, M.B., B.S., Ph. D. (Lond.) (2) Lt. Col. V. R. Mirajkar, I.M.S., F.R.C.S., O.B.E. (3) Capt. M. G. Kini, M.Ch., F.R.C.S. (4) Dr. M. B. Prabhu, M.B., B.S., M.M. (5) Dr. U. Rama Rau. (6) Rao Bahadur M. Keshava Pai, M.D., O.B.E. (7) Dr. V. Rama Kamath (8) Dr. P. Venkatagiri, M.D. (9) Dr. G. R. Dinkar Rao, M.D. (10) Rao Sahib Dr. B. Colaco. (11) Dr. L. P. Fernandes, B.A., L.M. & S. (12) Dr. S. R. Pandit.

After tea, the toast was proposed by Dr. M. Umeshe Rao, L.M. & S., T.D.D. (Wales) in very suitable form. Dr. K. R. Kini replied to the toast. Captain W. E. Mascarenhas and Dr. C. P. Kamath were re-elected as President and Secretary respectively for the ensuing year.

After a vote of thanks proposed by the Secretary, the meeting terminated.

THE KRISHNA DISTRICT MEDICAL ASSOCIATION, MASULIPATAM.

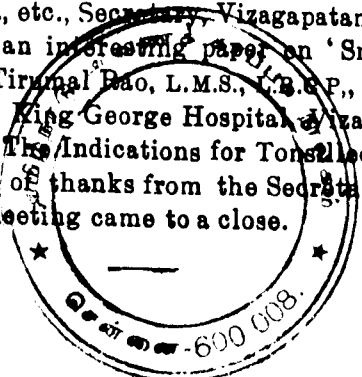
(The Krishna District Branch of the Indian Medical Association).

The minutes of the monthly meeting of the Krishna District Branch of the Indian Medical Association held on the 5th October 1940.

The meeting was presided over by Dr. M. Seshacharyulu, L.M.S., Vice-President, in the unavoidable absence of G. J. Pacheco, Esq. L.M.S., President. 35 doctors, were present.

During the ensuing Medical meeting, Dr. V. Govindan Nair, L.M.S., L.R.C.P., F.R.C.S., etc., Secretary, Vizagapatam District Branch of the I. M. A., read an interesting paper on 'Small Pox and Its Treatment.' Dr. B. Tirumal Rao, L.M.S., M.B., F.R.C.S., F.R.F.P.S., D.L.O., Superintendent, King George Hospital, Vizagapatam, read an interesting paper on "The Indications for Tonsillectomy."

Then, with a vote of thanks from the Secretary to the distinguished visitors, the meeting came to a close.



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