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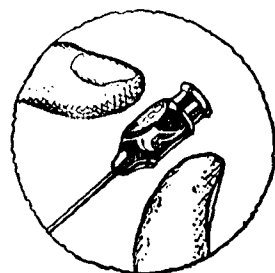
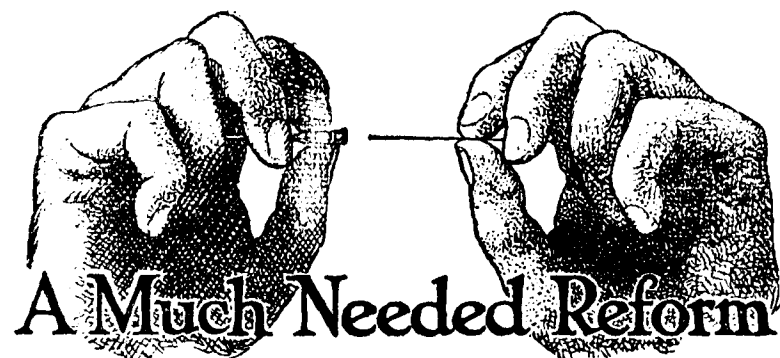
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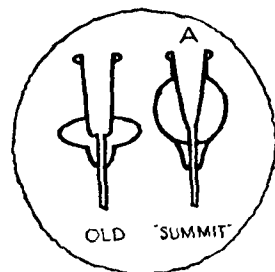


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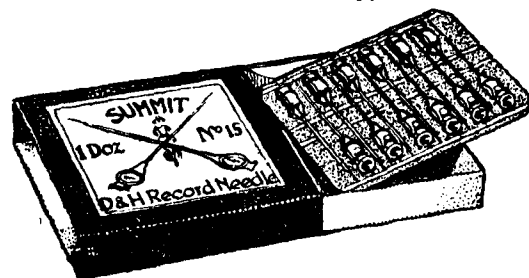
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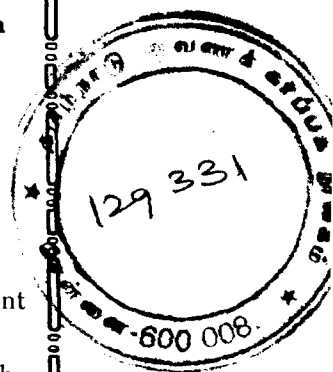
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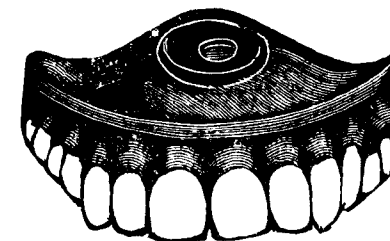
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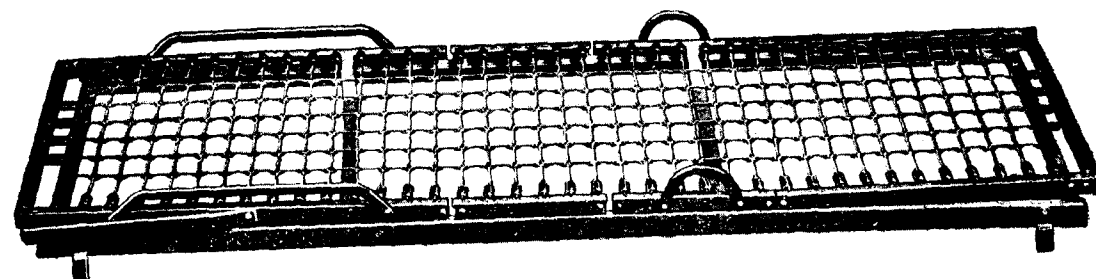


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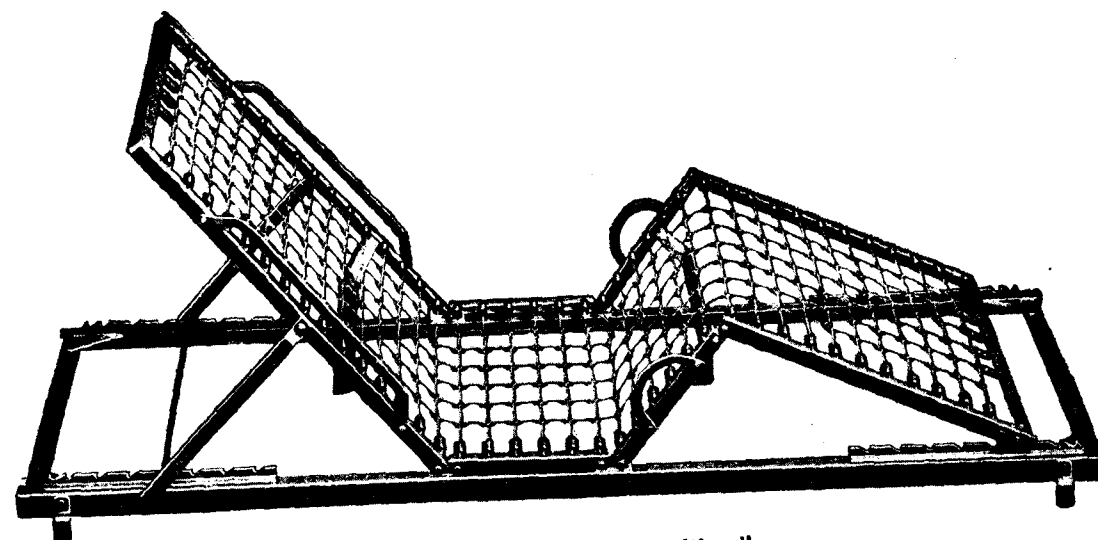
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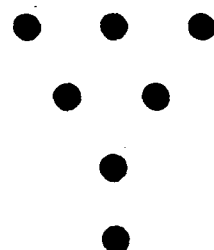
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Vol. X	July 1930	No. 1
CONTENTS.		PAGE.
1 NON-SPECIFIC PROTIEN THERAPY		
By Dr. K. V. Krishnan, M.B., M.R.C.P.E., D.B. (London)	...	1
2 SPRUE, ITS ETIOLOGY AND SYMPTOMS		
By Dr. K. Narayana Murthy, M.B., B.S.	...	12
3 THE LIVER AND NITROGEN METABOLISM		
By Dr. K. C. Paul, M.B., B.S.	...	21
4 MENTAL HYGIENE		
By Dr. S. Venkatasubha Rao, M.D., D.P.M.	...	32
5 CLUB FOOT		
By Dr. M. G. Kini, M. ch., F.R.C.S.	...	37
6 A FEW HINTS ON STAINING METHODS AS APPLIED TO BACTERIA		
By Dr. Seshadrinathan, M.B., B.S., D.T.M.	...	42
7 FLOCCULATION TESTS FOR SYPHILIS		
By Dr. K. V. Krishnan, M.B., M.R.C.P.E., D.B. (London)	...	45
8 SOME OBSERVATIONS ON ASCITES		
By Dr. N. S. Narasimhan, F.R.C.S.	...	50
9 EDITORIAL		52
10 OFFICE-BEARERS OF THE MADRAS MEDICAL COL- LEGE ASSOCIATION		54
11 DODGING THE DOCTOR		
By "the Inaugral Strutter"	...	55
12 IN LIGHTER VEIN		59
13 RANDOM NOTES		
By I. J. E. David, Student Editor	...	66
14 CASE REPORTS		
By Ratnaswamy, B.A., M.D., and Dr. C. S. Sundaram, M.B., B.S.		72
15 ASSOCIATION NOTES		72
16 "ROLL OF HONOUR"		74
17 UNIVERSITY OF MADRAS—Medical Examination Results...		75

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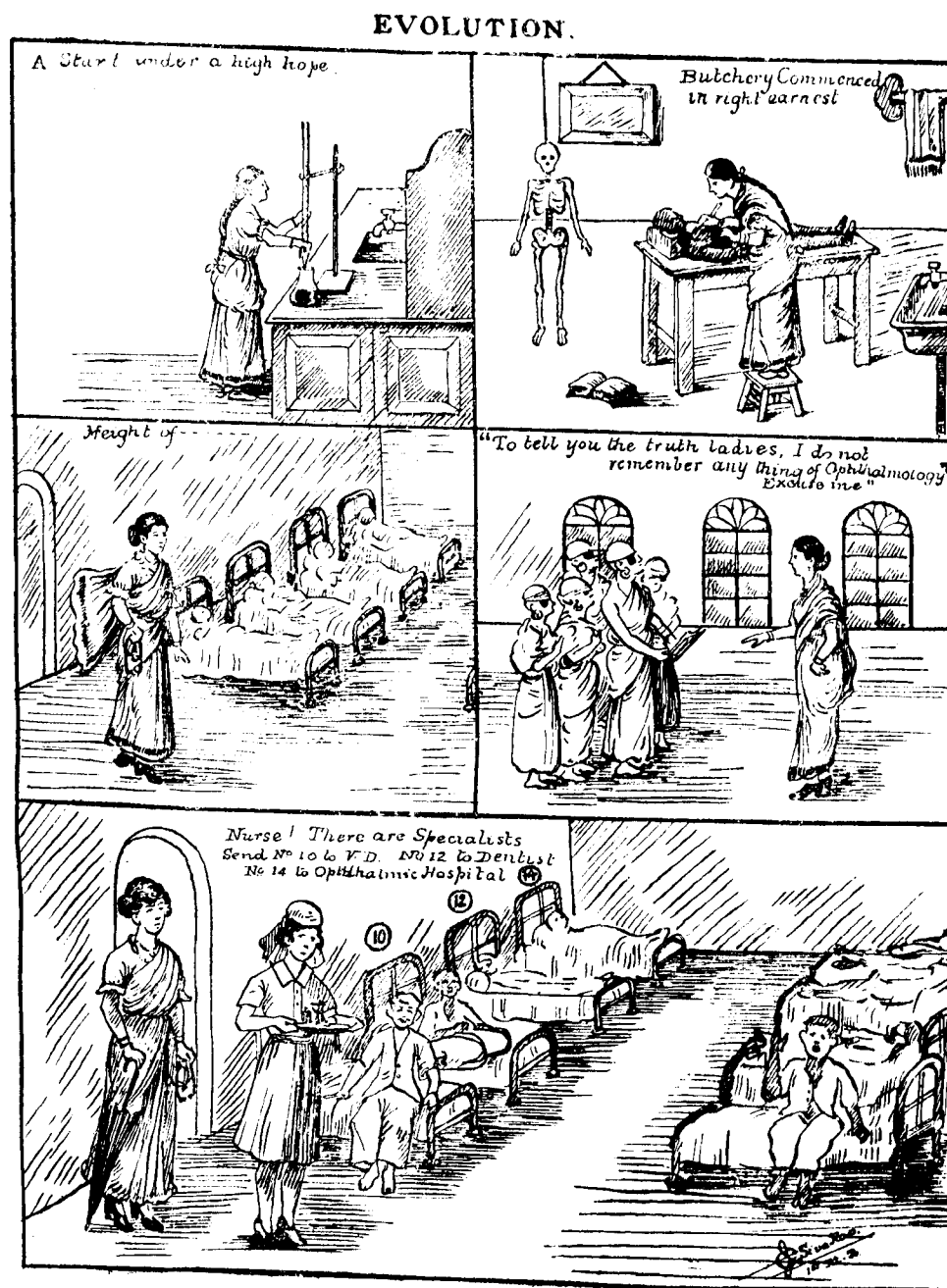
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VOL. X

JULY 1930

No. 1

Non-Specific Protein Therapy*

BY DR. K. V. KRISHNAN, M.B., M.R.C.P.E., D.B. (LONDON).

Within the last few years non-specific protein therapy has been acclaimed a very valuable remedial measure. The amazingly beneficial results at times obtained by the use of this remedy in several pathological conditions have led some clinicians to over-emphasize its value and importance. As a consequence there appears to be a growing tendency on the part of medical practitioners to recommend this method freely to all patients who have failed to react favourably to other remedies. Recent researches on the nature of the reactions following the administration of different protein substances lay a good deal of stress on the necessity for a judicious use of these substances in select cases alone and also point out the disastrous nature of the results likely to follow their improper use. In the light of these evidences it appeared worth while writing this article on the mode of action of non-specific protein substances more from the point of view of the immunologist rather than the clinician and then to put forward suggestions regarding the proper method of application of this therapy. It is hoped that those who are desirous of using this method of treatment will find the information contained in this article both helpful and profitable.

Historical.—The first scientific observation that beneficial effects can be produced in human beings suffering from infectious diseases by treating them with injections of proteins of a non-specific nature was made about forty years ago by Bockenham. He, in 1890, reported that diphtheria serum was effective in typhoid and streptococcal infections. Pfeiffer and others confirmed this and observed that non-specific sera were useful therapeutic agents in specific infections. In 1893 Rumf

* Being part of a paper read before the immunological department of Johns Hopkins School of Hygiene and Public Health, Baltimore.

reported a series of typhoid patients treated with pyococcus vaccine and Romer working along similar lines noted beneficial results by the use of various bacterial vaccines on tuberculous animals. Koch and Matthes observed that tuberculous guinea pigs reacted not only to tuberculin but also to injections of deuterio-albumose. In 1894 Gilbert introduced the method of auto-sero-therapy for treatment of pleurisy with effusion. He injected small amounts of pleural exudate under the skin of patients suffering from pleurisy with beneficial effects. Zimmerman next showed that injections of peptone solution instead of the pleural exudate would yield the same beneficial results. Similar instances of successes in treatment, with purely non-specific proteins such as coley's fluid, the leucocyte extract of Hiss and Zinsser, auto serum, plasma, whole blood, etc., can be found in several clinical reports of the past. But these observations having been made in an era of specificity, that had witnessed the brilliant triumphs of diphtheria and tetanus anti-toxins could not be made to fit in with the prevailing mode of thought. Naturally therefore the claims of this new therapy were put forward half heartedly and its merits remained long unrecognised.

Coming to more modern times the advocacy of non-specific protein therapy may be said to have started as a sequel to Wright's vaccine therapy. About 1916 Wright wrote "I confess to having shared the conviction that immunization is always strictly specific. Twenty years ago, when it was alleged before the Indian Plague Commission, that anti-plague inoculation had cured eczema, gonorrhoea, and other miscellaneous infections, I thought the matter undeserving of examination. I took the same view when it was reported in connection with anti-typhoid inoculation that it rendered the patients much less susceptible to malaria. Again, seven years ago, when applying anti-pneumococcus inoculation as a preventive against pneumonia in the Transvaal mines, I nourished exactly the same prejudices. But here the statistical results which were obtained in the Premier Mine demonstrated that the pneumococcus inoculation had, in addition to bringing down the mortality from pneumonia by 85 per cent, reduced also the mortality from other diseases by 50 per cent., From that on we had to take up into our categories the fact that inoculation produces in addition to 'direct' also 'collateral' immunization." Reports such as this, materially helped to break down the prevailing conception of rigid specificity in vaccine therapy and the scepticism regarding the value of non-specific protein substances in the treatment of disease. From hetero-bacterio therapy, it

was but a logical step to attempt the intravenous injection of bacterial components and bacterial split products, and then of protein split products of non-bacterial origin and finally to the realisation that any substance which may be capable of inducing a reaction on the part of the patient, would result generally in the same therapeutic change. Thus a large number of substances have been found useful in non-specific protein therapy and it will not be out of place to give a list of them here.

Substances used in 'Non-Specific Protein Therapy'—

1. *Blood and blood sera.*

Normal sera.—human, horse, sheep, beef, goat, and chicken sera.

Immune sera.—Human convalescent sera (Measles Poliomyelitis etc). Pneumococcus, Streptococcus and Dysentery sera, Diphtheria and Tetanus anti-toxins.

Whole Blood.—Human.

Related agents.—Blister fluid, pleural and ascitic fluids, C.S. fluid.

2. *Proteins.*—Egg albumen, serum albumin, milk, casein, gelatin, agar, and other plant proteins.

3. *Protein split products.*—Proteoses Deuteroalbumose and Wittes peptone.

4. *Enzymes.*—Trypsin, amylase and leucocytic extracts.

5. *Tissue extracts.*—Tumour autolysates and vascular extracts.

6. *Vaccines.*—Practically all vaccines. T.A.B. and Coli vaccines in particular.

7. *Bacterial extracts.*—Tuberculin, Coleys fluid, and Phylacogens.

8. *Colloidal metals.*—Gold, Silver, Manganese, Sulphur, Iron, Platinum, Mercury.

9. *Miscellaneous.*—Hyper and Hypotonic salt solution, Distilled water, Turpentine, and Glucose solution.

It will be noticed that in the above list a number of non-protein substances are also included. The therapeutic effects of these, though variously explained previously are now recognised to belong to the same category. All the substances included in the list act as non-specific stimuli and help to produce the anamnestic reaction on which probably their beneficial effects mostly depend. The anamnestic reaction, as is known, is one in which an increase in the concentration of an antibody is brought about by the non-specific stimulus of a substance which can possibly have no chemical relation with the antigen that reacts in the test tube with the antibody that has increased.

Diseases in which Non-Specific Protein Therapy is applicable.

They may be classified as follows :—

1. Arthritis—all kinds, acute, rheumatic, chronic, gonorrhœal and syphilitic.
2. Typhoid and paratyphoid fevers.
3. Gonorrhœa and its complications.
4. Acute infectious diseases.—Anthrax, Diphtheria, Bacillary dysentery, Erysipelas, Measles, Pneumonia, Septicaemia, Scarlet fever, Tetanus and Poliomyelitis.
5. Miscellaneous diseases—Asthma, Hay-fever, and Haemorrhagic diathesis.
6. Nervous diseases—Tabes, G.P.I. Epilepsy, Subacute combined degeneration, certain forms of Neuritis and Neuralgias.
7. Skin diseases—Psoriasis, Eczema, Acne and Furunculosis.
8. Eye diseases—Trachoma.

Reaction. The reaction that follows injections of various protein substances into man, may be considered under two heads, *i.e.* clinical and immunological.

The reaction as observed by the clinician may vary from a mild febrile attack to extreme shock associated with profound vasomotor paralysis—the degree being dependent upon :

- the nature of the substance used
- the dosage
- the method of application
- the rate of absorption
- the type of infection of the patient
- the tissue involved in the diseases process
- the physical condition and temperature of patient at the time of injection and so on.

For purposes of description, however, a typical reaction may be described as follows. It usually starts with a slight chill rise of temperature, rapid pulse, and occasionally nausea, vomiting, headache, etc. There may be a slight fall in blood pressure after an initial rise. These may be followed by increased glandular activity, sweating and diarrhoea. Local pain, swelling redness and heat at the site of inoculation may be complained of. There may be a lighting up of the focus of infection irrespective of its nature with aggravation of symptoms. All these may gradually subside leaving the patient in a clinically improved state.

The changes noticed in the blood and blood forming organs, on the other hand, are very variable and uncertain. The results obtained by different workers, both in man and experimental animals, are not concordant and do not warrant any definite conclusions. However, excluding for the time being observations such as increase in the permeability of capillaries, increase in the rate of flow of lymph, and in the number of blood platelets whose significance and value it is difficult to assess, one may consider the more important changes that are recognised to have some bearing on the value of non-specific protein substances as therapeutic agents. These may be summarised under the following three headings:

Erythrocytes. The changes in Erythrocytes are usually transient and variable. There appears to be 3 different views on the subject.

1. No change in Erythrocytes at all.
2. There may be variation in their numbers. Either an increase or a decrease. (Muller, Butah and Agrafonoff).
3. There is not only a numerical but also a qualitative change in the Erythrocytes. Normoblasts Megaloblasts and Polychromatophilia may occur. (Lange, Weichardt, Schittenhelm, Griessh and Ammer).

The more recent work of Spekforovoskapa (1925) shows that in 93 per cent of cases the erythrocytes markedly increase in number from the 2nd day to the 5th day and then go back to normal. Probably there is a stimulation of the erythrocyte producing tissues, following correctly dosed injections of non-specific protein substances.

Leucocytes. Leucocytic changes have also been noticed from the very commencement of this therapy. In the great majority of cases the leucocytic response consists of two distinct stages—a primary stage of leucopenia and a secondary stage of leucocytosis.

Spekofrovoskapa (1925) found that in 80 per cent of the cases there was an initial leucopenia lasting for about 3 days. From the 3rd to the 7th day there occurred a moderate increase in leucocytes which again went back to normal. Weichardt and others found that the leucopenia lasts only a few hours and that the rise in leucocytes commences after 8 to 10 hours and in 1 to 2 days there is hyperleucocytosis which may last from 3 to 5 days. The degree and duration of leucocytosis seems to be dependent on various factors one of which is undoubtedly the dose of the protein.

Regarding the nature of the leucocytosis produced there appears to be some difference of opinion. While some find an increase in the

granular series of leucocytes, others find an increase in the non-granular series. Ling (1925) found that after injections of proteins like milk, peptone and typhoid vaccine there was an increase in neutrophils and after injections of distilled water and crotalin there was an increase in the mononuclears. It is possible that there is some sort of relationship between the chemical nature of the substances introduced and the type of leucocytic response obtained. A clearer knowledge of this subject will undoubtedly be of enormous benefit and importance both to the clinician and the immunologist. When this is available it would be possible to choose such substances for injection as would yield a leucocytic response helpful to the normal cellular mechanism of defence. For there appears to be some evidence to suggest that the leucocytosis produced is to a certain extent responsible for the therapeutic results obtained.

Antibodies. It has long been noticed that if injections of non-specific protein substances are made into persons who have been previously immunised, these will usually respond with an increase in the titre of antibody already present in their serum. Although this, by no means is a constant affair, yet a good deal of stress has been laid on this being one of the important factors responsible for the beneficial effects noticed in patients after injections of non-specific protein substances. It would therefore be useful to record here some of the more important experimental evidences available on the subject.

Using *peptenos and other protein materials*. Obermayer and Pick (1904), Fleckseder (1916), Weichardt and Schrader (1919), Matsuda (1924), Ling (1925), and Buzello (1926), all noted an increase in the titre of antibody.

Next using *milk*. Uddgren and others reported that in distinct Wassermann and Widal reactions become distinct and Gonorrhœal patients giving a negative complement fixation reaction gave a positive reaction.

Using one *Vaccine* as a primary specific stimulus and another as a secondary non-specific stimulus. Dreyer and Walker (1909), Conradi and Beiling (1916), Tsukahara (1921), Rosher (1924), all noted an increase in titre of antibody for the primary antigen.

Using various *colloids* such as agargslatin, silver sols and iron sols, Steaben (1925) found that they had no power to increase antibody titre when this had become steady but that when they were given at the same time as the bacterial antigen, they appeared to increase the antibody response.

Non-Specific Protein Therapy

Using various *metallic salts* of manganese, cobalt, beryllium, etc., Walbum, Madsen Schmidt and their colleagues (1923-25) noted that these caused increased production of anti-toxins, agglutinins, etc. But McIntosh, Horgan and others failed to confirm them.

Although the summary of the results given above may at first sight carry to the reader the impression that the results are more or less uniform, it has to be pointed out that they, as recorded in the original papers, are rather irregular, confusing and at times contradictory. Topley, after weighing the evidences contained in all the available literature on the subject, beautifully summarises the results as follows.

1. Non-specific stimulus cannot cause the appearance *de novo* of any of the known serum antibodies.
2. They may cause an increase in any of the normal antibodies present.
3. They may cause an increased production of immune antibodies when administered during the early stages of immunisation.
4. They may cause a secondary rise in titre when administered after the effect of the preliminary specific immunisation has begun to decline.
5. All the above responses are exceedingly irregular usually trivial and never comparable to those following the re-injection of the specific antigen.

In view of the above conclusions it is hard to decide what the true value and significance of the increased antibody output can be. And although beneficial results have often been recorded in the absence of any increase in the demonstrable antibody, it seems probable that the increased antibody output plays some important part in certain types of diseases at least, if not in all.

Theories regarding the mechanism of cure. It has been shown that several changes occur after injections of non-specific protein substances. To which of these the beneficial effects obtained are attributable is rather difficult to see at present. However the theories that have been put forward from time to time by various people are herewith given.

1. **Weichardt's theory.** He assumes that the non-specific agents instead of attacking the pathological cause, as do the specific agents, aim at altering the "reactivity of the soil". All the cells of the organism are said to be stimulated to greater activity with consequent production of substances that increase the general resistance and speed

up the "mechanism of detoxication". The process is assumed to be a physiological rather than pharmacological one.

2. *Dollken's theory.* This assumes that non-specific stimulation is not 'omnicellular' as suggested by Weichardt but that certain organs alone such as the liver, spleen, kidney and bone marrow as stimulated to greater activity with the result that the immune processes work more efficiently.

3. *Larson's theory.*—In this the injections of foreign proteins are said to liberate the so-called sessile antibodies (previously fixed in the tissues) and get them into the circulation so as to be readily available at the focus of disease.

4. *Whelmann's theory.*—This supposes that in addition to the increased production of antibody, complement, etc., there is "a great parallelism between portein injections and the sympathetic nervous system. In the mechanism of cure the sympathetic nervous system plays a very important part which is difficult of explanation."

5. Injections of proteins act by enhancing the activity of the endocrine glands with their recognised ability to influence general metabolism.

6. *Starkenstein's theory.*—That the proteins alter the permeability of the capillaries and cell membranes, thereby establishing a free exchange between the blood plasma and cells and allowing the washing out of the tissue fluids.

From the different theories quoted above it will be seen that our ideas regarding the mechanism of cure after protein injections are still very vague. It appears that the beneficial effects are attributable not to one but to a combination of factors. It is not uncommon to obtain striking clinical results quite independent of any of the changes recorded above. One may therefore safely suggest that the therapeutic results are attributable to the stimulation of the tissues concerned in the efficient working of the immunity mechanism whatever its true nature may be.

Practical application.—When one goes through the mass of literature on the results of practical application of non-specific protein therapy, one is struck by the great variability and inconsistency of the recorded findings. It is quite as common to find reports of amazing successes as it is to find reports of disastrous results. This is not at all surprising when one remembers that not only is our knowledge of the mode of action of this therapy vague and imperfect but also that there are no fixed standards regarding the choice of protein, dosage, intervals

between injections and so on. Furthermore at present it is not possible to lay down any general hard and fast rule for the guidance of practitioners. Factors such as age of the patient, nature, duration and type of his illness and so on have all to be carefully weighed before deciding that particular substance or dose would benefit the patient most. However the following tentative suggestions may be considered with profit.

Choice of protein.—Although it is recognised that all non-specific protein substances produce reactions fundamentally alike, it will be too presumptuous to say that the choice of the protein is not a matter of very great importance. While it is a fact that in certain cases quite simple proteins produce as good results as highly specialised protein substances, it is not to be supposed that this would work in all cases. For instance for intravenous injections protein split products seem to be more satisfactory than vaccines; where mild reactions are desired sera appear to be more useful; and where more severe general reactions are indicated intramuscular injections of boiled market milk will be found excellent. Again in certain septic infections the colloidal metals seem preferable. The correct choice of the protein therefore appears to be of no mean importance, and experience alone is the best guide to it.

Dosage.—This undoubtedly is a very important matter on which the success or failure of the therapy depends to a large extent. What exactly is the correct dose and how to determine it for each patient, are things dependent upon a number of factors. Theoretically it is only possible to suggest that good results may follow small gradually increasing doses at suitable intervals, particularly during the positive phase that follows a reaction—the idea being to make the negative phase as short as possible and the positive phase relatively intense and protracted. Apart from this general rule the dose for each case should be judged on its own merits.

In young children, in acute illness, and when the route of administration is intravenous the dose should be relatively smaller. Again when the focus of disease is situated in sensitive tissues like those of the lung or brain one has to be particularly careful about the dose. In chronic diseases of adults, involving tissues such as those of joints, bones and skin, an excessive dosage may not prove dangerous.

Other guides to treatment.—The best guide to correct treatment is the patient himself. The right dose will always cause the greatest clinical benefit even after the very first injection. Weichsel, who is a great protagonist of this therapy, is of opinion that the reaction that follows an injection when carefully studied and properly interpreted

would form an excellent guide to treatment. For example there is the *focal reaction*. This, he thinks, is desirable but not a strong one. The best results are usually obtained when the focal reaction is slight.

Next there is the *temperature*.—This, he says, should rise but slightly and last for a short time only. A high rise in temperature lasting for a long time usually does harm.

Lastly there is the *leucocytic curve*.—The focal reaction and temperature follow very closely this curve and therefore it may be considered the best indication of the ability of the person to react to the foreign stimulus. It will tell in most cases definitely, if the stimulus will act favourably or not. Very strong stimuli produce long continued leucopenia followed by hyperleucocytosis (above double the usual number) both of which are generally undesirable. Well dosed stimuli on the other hand produce an initial mild short lived leucopenia, which is quickly followed by a definite but not great increase of leucocytes. The degree of leucopenia and leucocytosis are not in every case of the same significance. For instance in a chronic disease like arthritis, it is the chill, the severity of the reaction and the hyperleucocytosis that appear to be beneficial. Whereas in a disease like asthma or typhoid fever such a reaction would be disastrous.

Lastly it should be pointed out that even when one takes all necessary precautions as to dosage, choice of material, etc., one has to remember that the application of this therapy is limited, there are certain contra-indications. A few are definitely known and a great many may yet be revealed. The more serious contra-indications according to Peterson are extreme physical depression, history of serum sickness or hypersensitiveness, severe nervous diseases, alcoholism, diabetes and pregnancy.

Conclusions.—From the preceding pages it will be evident that our knowledge regarding the mode of action and uses of non-specific protein therapy is still vague and imperfect. Under the circumstances it is but fair to state that it should not be applied either as a routine measure or in unselected cases. Great caution is desirable at least until more information is at hand regarding the correct method of its application. Furthermore it is necessary to realise that at least this therapy is but a poor method of stimulation for short periods of the defensive mechanism of the body and as much would be useless if overdone or applied when the body is in a state of intense depression. Perhaps there is no better way of concluding this paper than by quoting Peterson who writes

“Needless to say, non-specific therapy does require judgment, careful attention and bedside study on the part of the physician, perhaps in greater measure than any other therapeutic procedure. It should never be a routine; to be useful it must be an individualized therapy, with dosage and preparation and time of application varied to the disease, its intensity, its duration and the resistance of the patient. So used, non-specific therapy should prove to be one of our most useful measures both in acute infectious diseases and chronic inflammatory lesions.”

Sprue, its Etiology and Symptoms

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Definition.—Sprue is a Tropical disease which manifests itself by morbid changes of a chronic inflammatory and degenerative type in the alimentary canal, due to an unknown cause, with symptoms of raw tongue and mouth and by the passage of large, pale, frothy motions and its progress being often marked by remissions and exacerbations, occurring at considerable intervals. This is only a tentative definition.

History.—The disease had been described 150 years ago by Hillary. It has been variously described by different writers as psilosis, tropical aphthæ, white chronic tropical diarrhoea, Ceylon sore mouth, etc. With more consistency Dutch physicians have adhered to the local term current in Java "Indische Spruw" and this word anglicised into Sprue, has gradually been adopted throughout the East to designate what is now a recognised and familiar disease.

Climate.—Sprue is of peculiar interest in that it has a characteristic type of regional distribution as opposed to climatic disease. That is to say, it is almost invariably seen in persons who have lived within a certain well-defined area beyond which it is practically unknown. It is endemic and widely prevalent in the countries where it is present. In regions of similar latitude, and atmospheric conditions it does not occur. The Endemic home of Sprue appears to be Asia especially Malaya, Sumatra, Java, Burmah, and also extends into India, Ceylon, Australia, Fiji Islands and Japan. In tropical states of America and in England it is very seldom seen.

Etiology—I made a close and careful study of 33 cases of Sprue admitted and treated at the School of Tropical Medicine, Calcutta. I have found that this disease was more common among Europeans or Anglo-Indians rather than in Indians by which I mean specially the Bengalis. It starts insidiously in the Europeans who live in the tropics especially among those of the well to do classes. It seems to be a disease of adults, who are above 25 years and it is very rare under

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10. It occurs in families wherein a number of persons suffer from it. Close contact seems to have a bearing on the etiology of the disease. Therefore it is very essential to take the family history and also the previous history of the patient. I have come across three cases which gave the family history of the father suffering from the disease while some of the children of those parents subsequently suffered from the disease. So the factor of heredity seems to play some part in the etiology of the disease.

Regarding prolonged contact theory I came across one case where there was history of the husband suffering from Sprue and subsequently his wife contracting the disease. There was no history of the incidence of the disease in either the husband or wife's family. The disease in the husband fairly advanced rapidly and he died of it. Later the wife contracted it. By the time she came under our care she was neurasthenic and she had typical symptoms of sprue. She was afraid that the same fate as that of her husband would overtake her and she was anxious to know that her disease was not sprue. Mere utterance of the word sprue was enough to startle her. On the day of admission she overheard it mentioned that her case was one of sprue, she got very upset and passed more number of motions that day. This state continued until the physician himself gave the assurance that the disease she was suffering from was not the same from which her husband had suffered and died. This assurance made her slightly better even on that day. After that she continued to get better and better day after day from the treatment. This case in question suggests two possibilities, namely (1) that the infection might take place by prolonged contact, (2) through food, utensils, etc. The incidence of more than one case in the same family had been reported from Bombay and Ceylon. This observation favours the infective theory. It is very important to keep the patient uninformed of the nature of his or her disease. Sprue produces a marked depressant effect on the patient. It is of interest to note, that although a patient suffering from Tuberculosis and a patient suffering from Sprue are equally emaciated and appear alike, the former feels always hopeful even on the day of his death while the latter feels most despondent.

While speaking on heredity I might say that the endocrine factor is also to be thought of in the etiology of the disease. The right function of the endocrines happens to be an acquisition from the parents. If all the endocrines become exhausted the body emaciates and gets the

appearance of an old man. In sprue it is likely that premature senility is brought about by the premature exhaustion of the endocrine system. When the function of the endocrines is defective or the defensive mechanism fails or is exhausted, a toxic or infective condition which we see in cases of sprue may be found after all as a super-imposition. I bring before you the endocrine insufficiency as an etiological factor in sprue because as in senility, (1) there is a loose skin often forming wrinkles, (2) there is no subcutaneous fat, (3) all the internal organs of the body as the heart, liver, including the musculature of the stomach and the intestines are atrophied. Such a uniform atrophy of all organs can be noticed in sprue. It cannot be said that a particular gland is at fault, but I believe the whole system of the endocrines may be at fault.

Let me mention briefly the other theories of causation of sprue. I shall first mention those theories that were in vogue for some time and now obsolete but yet text-books copy them from book to book. I shall reserve to the last those theories that are now upheld and require some discussion.

(1) *Helmenthic theory*.—*Stercoralis strongyloides* was considered to be the cause, but this theory is given up since the worm is found in normal individuals without causing any pathogenic symptoms.

(2) *Protozoan theory*.—Various organisms as amoebae, spirochaetes, flagellates, etc., have been found in cases of sprue, but none have been demonstrated to be the cause.

(3) *Food with deficiency of vitamins*.—This theory was ably put forward by Nicholls and Cantli and subsequently by Elders. Elders thinks that deficiency of Fat, Sol. A, Vit. B and unbalanced tropical diet brings about the disease. But his theory did not receive support.

(4) *Calcium deficiency theory of Scott*.—Fourth theory was propounded in 1923. He found calcium in blood was deficient in sprue, especially the toxic or the organic form. He reported cases of rapid recovery after administration of calcium and parathyroid. W. C. Vines believed that sprue is due to acid dyspepsia induced by excessive protein fat and diet and citrates in fruits, resulting in depletion of free calcium in the blood. This free calcium in blood is found to be reduced by about $\frac{1}{3}$ of the normal. I have failed to confirm his results in my 33 cases.

(5) *Syndrome theory*.—In chronic dysentery especially in the bacillary type and also in pernicious anaemia, sprue like symptoms are found

to develop. But sprue is a definite clinical entity as it has, typical symptoms and a typical course. The etiological factor may be different, i.e., it is possible to get sprue associated with a fungus or of bacterial infection or a sprue invaded by a protozoan. But personally I think that some of the cases of bacillary dysentery that are to become chronic before they settle finally as sprue, have to pass through three phases of the disease.

(6) *Bacterial theory*.—(i) *The first phase* in sprue is Post Flexner bacillus infection view. It is to be remembered that Mild Flexner infection does not cause, severe dysentery. It frequently starts as a diarrhoea resembling the "Hill Diarrhoea" or low country morning diarrhoea. The bacillus of Shiga is as a rule a very toxic organism producing very powerful toxins, which give rise to the severest types of dysentery with fever. These toxins are threefold in nature. They are, an exo-toxin, an endo-toxin, and pressor bases. Col. Acton, Chopra and Boyd (1923) have shown that these pressor bases are formed from the amino acids of the protein molecules in the gut, and these are very toxic to man. Therefore Shiga is found to be not the causating factor in Sprue since we did not find these toxic symptoms developing in Sprue. Quite contrary to the infection of Shiga we find B. flexner is less toxic and gives rise to much less severe forms of dysentery. It has been found to produce an endo-toxin but no pressor bases. It subsequent to B. Flexner group of organism, infection with secondary organism, like *asciatica*, ferments many sugars with the production of irritating acids. (Archibald products).

Therefore, I am inclined to call early stages of sprue as post Flexner bacillus infection because (1) the serum taken from those patients give a positive agglutination reaction with B. Flexner to a titre 1:160, (2) a number of chronic diarrhoea cases with infection by B. Flexner passing on gradually to one of the stages in Sprue. It may be pointed out here that there is a great difficulty of isolating the B. Flexner in all those cases where there is secondary infection. This difficulty is either due to (1) the original infection of B. Flexner completely disappearing or lie latent in the mucus membrane leaving behind the ulcers which get secondarily infected with a haemolytic streptococci, (2) B. Flexner lies dormant for some time when the secondary organisms find time to get in.

(ii) *The next phase* of the disease is the occurrence of streptococci on the tongue and also in the stools. With the occurrence of these we also find a change in the symptoms of the disease. Rogers

and Nicholls in 1914 as a result of successful treatment of sprue cases with autogenous streptococci isolated from the tongue suggested that sprue was due to the infection of the oral cavity particularly the tongue with the streptococcus viridans. This streptococcus gradually passes down the alimentary canal, giving rise to some more symptoms. Absence of HCl in the stomach favours the growth of the streptococci. But in my cases there was really no deficiency of HCl in the stomach contents. X-Ray photographs taken after a barium meal showed a hurried action of the contents of the stomach into the ileum owing to the condition of the atony of the pylorus. Regurgitation of the alkaline contents of the duodenum took place into the stomach and so the chloride content will be higher. Therefore it is important to estimate both the free HCl and the chloride content of the stomach in cases of sprue. The organism isolated from the tongue was found to be non-haemolytic but gave positive complement deviation with the patient's sera. Nicholls believes that there are two factors operating in a case of sprue, one the infection with the bacteria and the other is the dietary factor. The streptococci isolated from the tongue are very delicate and are very difficult to be cultured. Vaccines were prepared from them and were used in the treatment of sprue by Sir Leonard Rogers at the S. T. M. From his case sheets, I understand that a great majority of his patients, improved remarkably. His method of isolating the streptococci and the preparation of auto vaccines for treatment is only feasible in cases of sprue with the affection of the tongue. But in cases with no symptoms of the tongue streptococci can be isolated from the stool by using Contradi Draglaskees medium. These streptococci are found to be either haemolytic or non-haemolytic. From these streptococci vaccines are prepared and injected intra-dermally. The patients with this method of treatment at S. T. M. improved marvellously. Now we have given up Sir Roger's treatment with the streptococci grown from the tongue in treatment of cases of sprue; because we come across (1) cases of sprue without tongue symptoms; (2) of the difficulty of isolating the streptococci from the tongue; (3) it is far easier to isolate strepto from the stools.

(iii) *The third phase* in the disease is the phase of the infection of *Monilia psilosis*. Kohlbrugge in 1901 found in Java this fungus in cases of sprue which he called *Monilia albicans*. His observation was speedily confirmed by other workers. Castellani in 1912 described several other species of the same fungus. In 1914 Manson-Bahr in Ceylon isolated the fungus from the tongue scrappings and from the

contents of the intestines. Ashford in 1915 working at Port Pico isolated a fungus which he named as *Monilia psilosis*. He produced on his experimental rabbits, gaseous diarrhoea gradual emaciation and death by the injection of cultures of the fungus. He proved that such cultures are intensely toxic to white mice, guinea pig and rats, when they are injected intra peritoneally. Michael in 1918 using emulsions of *Monilia psilosis* as an antigen obtained complement deviation in the sera of not less than 400 cases of sprue. Anderson in 1917 thoroughly made a study of the normal and abnormal fungi in the intestinal tract. He says that "wild yeasts are present in normal individuals but the fungi obtained from the phases of the sprue entirely differ from the normal forms." He named these as *Para-Saccharomyces Ashfordi*. Dold and Fischer in 1918 claim to have experimentally produced a sprue-like condition by feeding white mice and monkeys with cultures of those fungi.

On the other hand Manson-Bahr in 1923 recorded failure of producing sprue in dogs fed on monilia. Further he also recorded failure of isolating monilia from cases of sprue seen in London. Hence he is inclined to think that M. P. is only an accidental or a secondary invader further he recorded cases of non-tropical sprue recently. Mackie and Chitre were invariably led to the same result. They were unable to produce sprue like symptoms in animals by inoculating with monilia. Lt. Col. Knowles at Shillong isolated monilia psilosis of Ashford from three cases of sprue. These strains were found to be non-pathogenic to white rats and guinea-pigs on feeding as well as by intra-peritoneal injection. Two of those cases were treated by autogenous vaccine prepared by autolysing cultures of this fungus and both recovered. Therefore I am inclined to believe that the primary etiological factor in sprue is Post flexner infection. Mackie and Chitre in the I. J. M. R. Memoirs, 1928 August studied 252 strains of yeast like fungi isolated from stools both from typical sprue and other cases. They divided the strain into two main groups A and K. Class A are all maltose fermenters and usually ferment, glucose, lactose and galactose and are of the type of monilia psilosis (Ashford). Class K are non-fermenters of maltose and this type is monilia Krussi of Castellani. The yeast like fungi commonly regarded as pathogenic are of the A type. None of the strains are found to liquify gelatinic and none are found to be haemolytic. The distribution of the different types of yeasts is found by them to be similar in

sprue and other diseases and no type is particularly frequent in either a diseased or healthy condition. Consequently they adduced no evidence that yeast has a causative relation to sprue. In 1929 April, in the I. J. M. R. experiments of complement fixation with M. P. were done in sprue, which all gave negative results.

The symptoms and signs of sprue:—The symptoms and signs of sprue are described very well in all text books of Tropical Medicine and so I intend to be as brief as possible. My description of the symptoms are exactly those that we see in India, but not a catalogue of symptoms that are likely to be found in sprue prevalent in other islands.

The onset of the disease is slow and the period of incubation is not known. The prodromal symptoms are a peculiar characteristic feeling of lassitude and malaise, a sense of failing strength and undefined illness. There are invariably present preliminary to an actual attack. This description of the symptoms you can get only from an intelligent patient. In most cases you are not likely to elicit symptoms of this prodromal stage of the disease.

After some weeks or months, it starts first with morning diarrhoea. The patient passes two or three stools a day often without any griping. The colour of the stool is yellowish and it may contain little mucus. The patient in this condition may not take any note of his general health, as the evacuation of stools is not associated with griping and as he in fact experiences a sense of relief after the motions: he is free from the motions for the rest of the day.

As time passes on the patient loses the appetite, motions may increase in number and he may feel acid eructations with distension of the abdomen and discomfort. The tongue becomes sore. The patient may not at this stage notice that he is anæmic and has lost weight. The symptoms of sore tongue, indigestion and diarrhoea now worry the patient and so he comes to consult a doctor at this stage of the disease. The temperature may be sub normal. On culture of stools one can easily isolate B. Flexner or B. Morgan from stools at this stage of the disease.

The patient now confines himself to liquid or semi solid food. The symptoms of anæmia, flatulence and weakness develop. The patient loses weight rapidly. On examining the tongue it looks raw, with cracks and fissures on its dorsum. Small indolent and exquisitely painful sores are found all over the mouth and pharynx.

Crombies molar ulcer is not seen in all cases I have examined. Lime juice, spirits, wine and even aerated waters are intolerably irritating; he is unable to smoke and in many instances cannot even take tea or coffee without suffering. The development of these mouth lesions is accompanied by a persistent and continued accumulation of mucus in the fauces. The secretion is so viscid and adhesive that it is a matter of difficulty for the patient to clear the throat. These symptoms are found only in a pre-sprue stage of the disease. At this stage of the disease streptococci can be isolated from the stool. Flexner group of organisms may not be isolated. From this the disease passes into a typical condition of sprue or a slight intermission in improvement may occur. Deglutition and mastication become painful. On swallowing a burning pain is felt over the sternum. The patient is emaciated, depressed or irritable mentally. The skin hangs loose. Abdomen markedly distended, discomfort and pain is felt before each evacuation of stools. The stools are copious, offensive, frothy, clay-white in colour and they stop after midday of their own accord. Blood pressure is low. Oedema may be present over the ankles.

On examination of stool it shows mucus, epithelial debris, many yeast like fungi, bacteria and fatty acid crystals. Fat constitutes 50 to 60 per cent of the stools and this is due to a defective absorption from the intestines owing to the ulceration in the gut. The colour of the stool is probably due partly to the presence of excess of fat and partly due to the leuco bases. Blood shows marked anæmia. R. B. C. and W. B. C. are considerably reduced. This passes on to an Addisonian type of anæmia and at this stage of the disease it is difficult to differentiate between sprue and pernicious anæmia. Urine is scanty and it shows increase of indican and urobilin. At this phase of the disease we can isolate monilia psilosis. After several remissions and intermissions it passes on to a cachetic stage finally ending in death either due to syncope and exhaustion or more frequently by an inter-current disease.

This however, even without treatment is happily not the invariable termination of sprue. A considerable number of our cases pass into a latent condition, and though always liable to certain remissions, the patient enjoys fairly good health and live for many years without any symptoms of toxæmia. In a still large proportion of cases that are left untreated the disorder is protracted indefinitely, its urgency at times abating and at times increasing. But when Toxæmia is once established

and if treatment is not prompt and effective there is little hope of averting a fatal issue.

It will thus be seen that the usual cases of sprue under treatment run through three different phases. Each phase has its distinct group of symptoms which are determined by the bacteriological findings occurring in each.

Pathology of Sprue.—Marked changes take place in the small intestines. The Muscular layer atrophies and looks anaemic. In mucus membrane, we find shrinkage of villi and round celled infiltration. The vessels of submucosa are dilated and there may be certain amount of fibrosis taking place in the gut wall. Later the villi and glands disappear, or cystic dilatation of the follicles and even small abscesses in places may be seen. Those cystic dilatations can be seen with a sigmoidoscope.

Finally, areas of the mucosa, chiefly in the lower end of the ileum and in the colon become eroded, and definite patches of ulceration may be seen. This change may be general throughout the bowel but more often patchy in distribution.

Pancreas.—Slight fatty or granular changes and inflammatory infiltration of the connective tissue are noticed. In the far Eastern Science Congress, 1927, Sokhey and Malandakar reported, that there was no deficiency of Pancreatic function in sprue.

Liver and heart get atrophied. Iron content in liver not increased.

In kidneys and adrenals degenerative changes are seen. In bone, marrow there may be a certain amount of aplasia.

(To be continued.)

The Liver and Nitrogen Metabolism

WITH SPECIAL REFERENCE TO THE STUDY OF LIVER EFFICIENCY

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It has long been recognized that the liver plays an important part in the nitrogenous metabolism of the body. But the exact role of the organ remains yet undefined. It is no doubt an intricate one, and probably many factors of which we have no definite knowledge enter into it. The complexity of the problem is such that an adequate discussion of it would entail a complete survey of the physiology of nitrogen metabolism. As such a discussion is beyond the scope of the present article, only a bare outline of the subject will be attempted here.

The nitrogen metabolism of the body may be considered under two headings: I. Protein metabolism; and II. Purin metabolism. Both of them may be either *exogenous* or *endogenous*. The nitrogenous bodies taking part in the exogenous metabolism come from the ingested food; while those concerned in the endogenous are derived from the wear and tear of tissue protoplasm.

I. *Protein Metabolism.* (The following account is based mainly on the writings of Van Slyke.²⁶)

The proteins of the food are broken down in the alimentary canal into their ultimate building stones, the amino acids, by the proteolytic enzymes of the digestive juices. These amino acids are absorbed into the portal blood and conveyed to the liver. A large proportion of them is arrested here and deaminized, or, possibly, synthesized into reserve protein. A small part passes out into the general circulation, and is carried to the various organs and tissues of the body. These organs and tissues absorb as much of the amino acids as they can, and utilize those which they need for the repair of their protoplasmic elements.

The breaking-down and the building-up of the proteins incorporated in living protoplasm are incessant processes which are expressive of

vital activity. The products of the integration of these proteins are also amino acids. Under ordinary conditions of protein intake, the amino acids required for the building-up process come from the absorbed amino acids; probably, the endogenous amino acids are also used for tissue repair. During starvation, as exogenous amino acids are not available, there is an increase in the breaking-down of the proteins of the inferior tissues, and the amino acids thus derived are made use of for the repair of the more vital structures.

The amino acids not required for tissue repair, be they of endogenous or of exogenous origin, are not stored in the body, but deaminized and got rid of.

Deamination consists in the conversion of the amino group of the amino acid into urea. In bringing about the conversion, the amino acid is first split up into two portions, a (nitrogenous) amino radicle (and a non-nitrogenous) fatty acid radicle. The amino radicle yields ammonia. This ammonia combines with the carbonic acid of the blood and tissues to form ammonium carbonate. Dehydration of the ammonium carbonate (withdrawal of two molecules of water from the ammonium carbonate) results in the formation of urea. The fatty acid group, however, is not thrown out of the body; it is utilized for the energy needs of the organism, probably after conversion into carbohydrate.

The seat of these deamination changes has been the subject of considerable discussion. Van Slyke²⁶ is the chief exponent of the view that the formation of urea is almost exclusively a function of the liver. He does not deny that the break down of amino acids into ammonia and fatty acids may take place in tissues other than the liver. But he maintains that the ammonia formed in other tissues is conveyed to the liver for conversion into urea. Folin and Denis¹¹ had previously come to the conclusion that the power of deamination was not confined to the liver, but was characteristic of all tissues. Taylor and Lewis²⁷ also came to the same conclusion. A similar view is held by Carlson⁶ as well.

Van Slyke has shown that whereas the liver saturates itself with amino acids when protein digestion is taking place, within two or three hours after absorption has ceased, the organ desaturates itself. He explains this desaturation as the result of deamination by the liver, which begins immediately after absorption has commenced. Folin and Berglund⁸ criticize this view. They hold that the liver has no special

function of deamination; the organ simply acts as a buffer or safety-valve between the portal stream and the general circulation, and prevents the flooding of the general blood stream with the enormous amount of amino acids present in the portal blood during protein digestion; the amino acids disappear from the liver, because they are gradually washed out of the organ for absorption by the various tissues.

Macleod¹⁶ says, "It must not be imagined that the conversion of the amino acids into urea is exclusively a function of the liver. On the contrary, it is well known that this process may occur in animals from which the liver has been entirely removed." But the recent experimental work of Mann and his associates on dogs seems to establish the validity of Van Slyke's contention that the liver is the sole seat of deamination and urea formation.

Bollman, Mann and Magath³ found that when the liver was totally removed from a dog, whose kidneys remained active urea, rapidly disappeared from its blood and tissues, and in a few hours the urea content of its urine also diminished. After about 12 hours, its blood, tissues, and urine contained only traces of urea. In an animal with a high urea content of the blood, brought about by the removal of its kidneys, total hepatectomy stopped further increases in the blood urea. They noted also that the urea found in the blood and tissues and excreted in the urine after the removal of the liver accounted for all the urea present in the animal at the time of the operation.

Mann¹⁷ says, "Removal of the liver is followed by a progressive increase in the amino acid content of the blood. If the kidneys are removed at the same time as the liver, the amino acids increase more rapidly in the blood.....There was never any evidence of the formation of urea following the injection of amino acids into the hepatectomized animal."

But the amount of liver tissue necessary for the conversion of liver amino acids into urea seems to be very small. Mann and Bollman¹⁸ have been unable to demonstrate any decrease in the formation of urea even in those animals with the greatest reduction in the amount of hepatic tissue. "More over, in such animals, they have not found 'any significant quantitative changes in the amino acid nitrogen of the blood';...the 'amount of amino acid nitrogen excreted in the urine of these animals has been but slightly greater than that of the normal animal.'" They have noticed, however, that the "injection of amounts of amino acids that are without visible effect in a normal animal

produces cardiac irregularity, increased respiration and often collapse of animals if the amount of hepatic tissue has been greatly reduced."

With regard to the question of storage of "reserve protein", Pfluger's teaching was that the liver should be regarded as a storehouse for protein. He held that the liver cells not only synthesized protein from the exogenous amino acids, but also held it in reserve to meet "urgent demands from the active tissues" (Cathcart?). Van Slyke says that though protein storage by the liver is "surely not proved", neither does the evidence at hand disprove it. The current teaching is, however, that there is no storage of protein anywhere in the body as there is of carbohydrates and fats.

The liver does not seem to be concerned in the formation of ammonia. The organ, of course, converts any ammonia which reaches it into urea.

As deaminization and urea formation are probably exclusively located in the liver, it could be argued that in hepatic disease, deaminization and urea formation would not proceed satisfactorily, and, as a consequence, there would be a definite alteration in the distribution of nitrogen amongst the various nitrogenous bodies in the blood and urine. Theoretically, the change would consist in an increase in the amino acid and ammonia nitrogen of the blood and urine at the expense of the urea nitrogen, which would be decreased. The amino acid nitrogen of the blood would increase as a direct result of the deficient deaminization occurring in the liver; and when the amino acid nitrogen increases in the blood a greater excretion of amino acids into the urine would follow. The ammonia nitrogen would be increased in the blood and urine because of the deficient formation of urea.

Before satisfactory methods of blood analysis were evolved, attention was focussed entirely on the nitrogen partition in the urine. But the innumerable studies of urinary nitrogen partition did not reveal much that was of clinical utility in hepatic disorders. In 1913, Rowntree, Hurwitz and Bloomfield²¹ reviewed much of the current literature, especially the work of Falk and Saxl, and came to the conclusion that in severe hepatic disease there was an increase in the amino acid nitrogen and the ammonia nitrogen at the expense of the urea nitrogen.

With the development of clinical methods for studying the nitrogen partition of the blood, a new impetus was given to the study of nitrogen metabolism in hepatic disease. Rowntree, Marshall and Chesney²² were among the first to study the nitrogen partition in the blood in a

large series of hepatic cases. They did not observe any nitrogen retention in the blood except in the cases with renal disease. In some cases of hepatic disease, they noted a lowering in the urea concentration of the blood; they concluded that a low percentage of urea nitrogen in comparison with the total non-protein nitrogen was suggestive of hepatic insufficiency.

The trend of thought of the present day is to place no great reliance on these nitrogen partition studies in evaluating hepatic function. Pezzali²³ says that no reliable conclusions could be drawn as regards liver disease from the urea and the ammonia in the blood and urine. Like statements have been made by several other writers also.

The unsatisfactory nature of the nitrogen partition studies in hepatic disease is due to several reasons:

1. The amount of functioning liver tissue necessary for the adequate performance of the deaminization function is but small. A disturbance in the nitrogen partition in the blood and urine is not therefore to be expected until a great reduction in the functioning liver tissue has taken place. Such a reduction in hepatic tissue is seldom met with in clinical practice.

2. The amount of urea excreted in the urine is dependent on the diet of the individual and the state of his kidneys. On a high nitrogenous diet the urinary output of urea is considerably raised; but on a dietary of low nitrogen content the amount of urea excreted in the urine falls to a low level. If the kidneys are not functioning adequately, the urinary output of urea will be markedly reduced and a retention of the substance will occur in the blood.

3. An increase in the ammonia nitrogen of the urine need not be an indication of hepatic failure, but may only show that there is an increased amount of acid in the body to be neutralized. Ammonia is one of the bases, in combination with which acids are eliminated from the body.

4. A marked increase in the amino acid content of the blood is not likely unless there is impermeability of the renal epithelium. Folin and Berglund have shown that the amino acids are not threshold bodies, and are excreted by the kidneys in much the same way as galactose. Moreover, amino acids are absorbed in large amounts by the muscles.

It has been known for a long time that certain amino acids when administered by mouth give rise to an increase in the urinary output of

urea. Since their deaminization is effected in the liver, it was held that in disease of the liver part of the amino acid administered would be excreted in the urine unchanged. Glaessner¹³ attempted to make practical use of this inference. He found that in health an individual could ingest 25 grammes of either alanine, aspartic acid, or glycine, or 20 grammes of leucine without giving rise to amino-aciduria. In hepatic disease the administration of such an amount of these amino acids resulted in an increase in the amino acid output in the urine. The following table shows the results he obtained:—

Pathological condition.	Amino acid given.	Dose.	Result.
Fatty liver	... Aspartic acid.	20 gms.	Marked rise in output of amino acid in the urine.
Syphilitic liver	... Alanine	20 gms.	Marked rise in output of amino acid in the urine.
Cirrhotic liver	... Glycine	25 gms.	Rise in output of amino acid equal to about 70 per cent. of the glycine taken.
Cirrhotic liver	... Glycine	25 gms.	Rise equal to about 51 per cent. of the glycine taken.
Cirrhotic liver	... Aspartic acid.	25 gms.	Rise in output equal to about 40 per cent. of the aspartic acid taken.
Cirrhotic liver	... Aspartic acid.	25 gms.	About 87 per cent. of the aspartic acid taken excreted.
Cirrhotic liver	... Aspartic acid.	25 gms.	About 90 per cent. of the aspartic acid taken excreted.
Cirrhotic liver	... Alanine	20 gms.	About 51 per cent. of the alanine taken excreted.
Phosphorus poisoning	... Glycine	20 gms.	Very marked rise in the output of amino acid in the urine.

(Cathcart⁷)

Folin and Berglund, who do not hold that the liver is specially concerned in deaminization, say that this test is not one of hepatic function, as the function it is supposed to test is a non-existent one. They do not, however, say that it is valueless in the study of hepatic disorders. According to them, it may be used to measure the ability of the liver to prevent the amino acids from overwhelming the general circulation, that is, its storage capacity. As this ability of the organ is dependent upon the integrity of its functioning epithelium, it must be maintained that any test which will measure it should be regarded as a test for hepatic function.

Amino-aciduria.—Attention may be drawn here to the occasional appearance of the amino acids, leucine and tyrosine, in pathological urines. They were discovered in the urine by Frerichs,¹² who found them in cases of acute yellow atrophy of the liver. Their presence in the urine was for long regarded as pathognomonic of this condition. But now it is recognized that they may occur in the urine in any disease in which great destruction of tissue takes place in the body. It is true, however, that in no other malady do they occur in the urine so frequently or in such large amounts as in acute liver atrophy. Of course, in several cases of this disease they were absent from the urine.

Mann and Bollman¹⁸ did not find leucine or tyrosine in the urine of dehepatized animals or of those whose liver tissue had been greatly reduced. They say, "It would seem that these substances are products of necrosis of the liver and that their presence is not due to the absence of hepatic tissue."

Leucine and tyrosine have occasionally been found in the urine in some cases of cirrhosis of the liver; here, it would appear, their presence is due to an acute necrotic process, which has been grafted on to an already damaged liver and giving rise to a condition of "secondary yellow atrophy" (Bradford⁵). These amino acids have been found also in the urine from a case of primary carcinoma of the liver. In the diffuse toxic necrosis of the liver, caused by phosphorus, leucine and tyrosine make their appearance in the urine, but only in small amounts. In some cases tyrosine has been absent, and in a few both.

Frerichs and Murchison²⁰ found leucine and tyrosine in the urine in some cases of typhoid fever, typhus fever, and small-pox. They ascribed the amino-aciduria to an acute degeneration of the liver produced by these infectious processes. These bodies have been noted in the urine in a few cases of erysipelas, yellow fever, and cholera. Von Noorden²³ found them in the urine of some cases of pernicious anaemia. They are sometimes excreted in the urine by patients suffering from leukaemia. Abderhalden¹ has repeatedly demonstrated the presence of leucine and tyrosine in the urines of cystinurics. Dixon Mann¹⁹ found them in the urine in two cases of hepatic congestion secondary to heart disease. Rietschel and Langstein²² noted leucine in the urine of a pneumonic child. Smith²⁶ has reported a case of abundant leucinuria in a young woman who was in *excellent health*, but for occasional attacks of hypogastric pain and a frequent desire to pass water.

Resenbloom and Gardner²³ found tyrosine in the urine of a healthy pregnant woman.

Leucine appears in the urine as spheroidal masses or discs, tinged yellow and radially striated. These bodies are refractile and look as though they possess concentric laminae. They may be easily detected with the aid of a microscope. But as Beale² wrote in 1861, "Urate of soda and many other substances crystallize in spherical globules, like leucine. Crystals of this form, however, which are soluble in alcohol and again crystallize in spherules, can hardly be anything but leucine. This substance cannot, therefore, be recognized by the form of the crystals alone." Harley¹¹ was of opinion that oily droplets might be a source of confusion. More recently, Dixon Mann has said, "In the course of a series of investigations on urines, which for various purposes were evaporated to a syrupy consistence, some specimens being derived from healthy persons, I have frequently seen under the microscope large, spineless crystals of ammonium urate, and also other bodies which, as regards size, contour, translucency, and colour, closely resembled leucine crystals." Leucine is soluble in alcohol and ether, and this fact may be made use of in its detection.

Tyrosine occurs in the urine partly in solution and in part as fine, colourless needles arranged in sheaves and rosettes. Harley¹⁴ wrote, "The tyrosine is recognized by appearing in the form of spiculated balls not unlike a rolled-up hedge-hog, with the bristles sticking out in all directions." The clusters of tyrosine needles can be easily made out under the microscope. But not infrequently, crystals of uric acid, sodium phosphate, calcium phosphate, calcium and magnesium salts of the fatty acids, bile pigments, and other substances appear in the urine as sheaves and rosettes and are liable to be mistaken for tyrosine. But the identity of these bodies could be easily established by chemical means.

It is said that when leucine and tyrosine make their appearance in the urine, the amount of urea is generally much below the normal (Bradford).

II. *Purin Metabolism.*—The general aspects of purin metabolism do not concern us here. In the summary which follows, the subject is dealt with only in its relation to the liver.

The role of the human liver in purin metabolism, as far as present evidence goes, seems to be limited to the formation of uric acid, which is 2-6-8-trioxypurin. In other mammals the organ seems to be

endowed with the power of destroying uric acid. Mann and his co-workers³ have shown that in the dog removal of the liver leads to the accumulation of uric acid in the blood and tissues.

Uric acid is formed chiefly from the nucleo proteins, which may be of endogenous or exogenous origin. These nucleo-proteins are compounds of protein molecules and nucleic acid. Nucleic acid is a complex substance which contains phosphoric acid, purin bases, pyrimidin bases, and carbohydrate radicals. When nucleic acid is broken down in the body the purin bases are set free. They are adenine, guanine, xanthine, and hypoxanthine. Adenine and guanine are amino purins. They are deaminized and converted into hypoxanthine and xanthine. Xanthine and hypoxanthine are oxidized into uric acid. These changes are effected in the body through the agency of a variety of enzymes. The enzyme which brings about the final conversion of xanthine and hypoxanthine into uric acid is known as xanthine-oxidase. This enzyme, so far as man is concerned, is found only in the liver. Therefore it follows that the liver is the site of origin of uric acid in man.

It has been mentioned that in the dog and other mammals the liver is concerned with the destruction of uric acid as well. This uricolysis is brought about through the agency of an enzyme, uricase, which is not present in any of the tissues of man. Jones¹⁵ says that there is no destruction of uric acid in the human body. Folin, Berglund and Derick⁹ have brought forward evidence to show that certain amount of destruction of uric acid does occur in the human body, and that the seat of this destruction is the blood. But, as Wells²⁰ puts it, "It is highly probable, in view of all available evidence, that in man most of the purin absorbed from the food, and practically all the purin from cell metabolism, is converted into uric acid and excreted as such, and there is as yet no actual demonstration that there is any destruction of uric acid by human tissues."

Mann and Bollman, as a result of their experimental studies on hepatectomized dogs, suggest the determination of the "amount of uric acid eliminated in the urine during a standard period of time following the ingestion of a standard meal with high content of purin" as a test for hepatic function. They found that in the dog this proved to be the best test for determining the degree of liver involvement. They conclude, "Whether or not such a test will be of value in man remains to be proved".

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Mental Hygiene

BY DR. S. VENKATASUBBA RAO, M.D., D.P.M.

"Prevention is better than cure"—this saying is as true in mental as in physical health, nay, it is even of greater importance, in view of the unsatisfactory result by treatment of the end products of mental disease.

Our attempts have so far been to improve the methods of physical hygiene and sanitation, to fight against the ignorance of the masses about the causation of diseases—that they are not products of sin and that pacification of the mysterious powers, by offerings, prayers and sacrifices—would not be a rational remedy, to reduce the prevalence of preventable diseases and to increase the longevity of life. With this object, the resources of the different branches of science are taken advantage of to devise new methods of prevention.

The position regarding mental diseases is even worse. Knowledge regarding the factors for the breakdown of mental health is wanting even amongst the educated, not to mention about the superstitious ideas held by the masses. Many people even now believe that "insanity" is either a disgrace or a mysterious affliction that cannot be prevented or cured, that insanity is a single disease, of only the most serious kind, that it comes suddenly without warning and people are helpless to prevent it, that emotional shock, loss of dear ones, disappointment in love, loss of money, etc., cause insanity, that "insane asylums" are dreadful places and that to go to one means never to come out and that all kinds of insanity are inherited.

They are ignorant of the facts that "insanity" includes a group of the more severe types of mental disease, that patients need early medical treatment just as in tuberculosis or heart disease, etc., and that no one need be ashamed of this sickness, that there are many different kinds of mental diseases—some mild, some serious—that mental disease develops gradually and displays warning signs in advance, that the danger signals exhibited as symptoms of an approaching breakdown can usually be recognised and if these are given prompt medical attention, the threatened mental disease can often be prevented, that mental shocks may precipitate a mental disorder already in incipient stage but not start it, that our "asylums" are now Hospitals from which a good many patients

are discharged as recovered or improved and that mental disease may also arise from inability to adjust to difficult environment.

They are also unaware that there are milder degrees of mental defect, apart from the extreme low grade cases who are helpless and that the milder defectives, if taken in hand early, are trainable to do manual work according to their capacity and inclination and to lead a useful life and that if neglected, they are susceptible to be the victims of bad influences and to become delinquents, criminals, chronic alcoholics, prostitutes, dependents and beggars.

To the public, between normal and abnormal mind, there is no middle way. There is no hygiene of mind for everybody.

The mental hygiene movement was started in western countries, not only to improve the treatment given to the mentally sick by the attendants and physicians in charge, in better type of institutions, but also to investigate the causes, lines of preventive measures for the reduction of mental disorder, defect and delinquency, to give special training and supervision for the feeble-minded, and to disseminate reliable information regarding mental hygiene matters amongst the public. For this purpose, full advantage is taken of the basic sciences as biology, biochemistry, anatomy, physiology, pathology bacteriology, psychology, psychopathology, etc. Mental hygiene is an art, like medicine, drawing its material from the fundamental sciences. It is the study, understanding and hygiene of the emotional, social and intellectual life of an individual. It teaches us to recognise mental inadequacies of various types, whether, it be mere defect, due to intoxications, constitutional diseases or incapacity of the organism for the adjustment with proper reactions and so on. By its aid, a maladjusted person who cannot get on with other people, and the one with problems of behaviour are recognised as sick individuals, requiring specialised treatment. It serves to conserve the mental health of a community by preventing mental breakdown, and aiding for the highest development of talent and happiness by its steady influence from the cradle to the grave and by keeping away injurious influences, both bodily and mentally. From eugenic point of view, it helps to prevent the propagation of the species by mentally defective and disordered individuals, so that unfortunate mental hereditary tendencies may be prevented in the progeny. It makes an individual function to the best advantage in all phases of life, and make the best of his resources, internal and external, to make his life healthy, contented, efficient and useful to himself and society. The

quality of life lived is improved in all aspects. It improves the social conscience as well, so that he learns to be sympathetic and kind to his fellow beings, to respect the feelings of others, to be considerate in his dealings, so that the human relationship may be of the most cordial type.

For the success of work in mental hygiene, co-operation of varied types of people is required. To mention a few, psychiatrists making a thorough scientific study of the subjects concerned in mental hygiene and generally acting as advisers for the others in the field, psychologists, for the study of mental development, abilities, reaction patterns, etc., psychiatric social workers, trained in psychology, psychiatry and social sciences for an intelligent investigation of the social history, family setting, and other environmental factors as economic, religious, etc., and also for tactful handling of troubles the patient has to deal with and adjusting him in the community, parents and teachers, to put into practice, methods of training the young, in proper habits and behaviour, probation officers in charge of the delinquent, to help in the social adjustment of their wards and so on.

Apart from taking advantage of other branches of science, full use is made of the teachings of modern psychology in developing mental hygienic methods. It tells us that habits good and bad and reaction patterns are mostly formed in the early years of life, and how to understand human behaviour, why a man acts as he does, feels as he feels and reasons as he reasons. It shows that the important governing factors in our mental life and behaviour, are our emotions—our feelings of fear, anger, sadness, worry, discontent, pleasure, love, jealousy, hate, etc., and our intellect is after all a slave of our emotions. Mental life is also intricately associated with the physiological system of digestion, circulation, glands of internal secretion, etc., so that it is impossible to divide the activities of man into those of the mind and those of the body. In other words, a human being functions as a unit finely integrated in its mechanisms. Behaviour and activity function as one aspect of the biological process of individual adaptation to circumstances and surroundings. Faulty adaptation or poor mental health may express itself in headache, fatigue, irritability, sleeplessness, discontents, palpitation, discomfort in stomach and so on. These unhygienic reactions are mental when they occur in response to some external disagreeable situation, or in a setting of personal failure to deal with one's emotional problems. These represent unconscious bodily protests against facing life. The progressive physician goes over such a patient carefully to exclude actual

disease process but the investigation does not end there. He enquires, is this patient worried over business, struggling with debts, entangled in domestic unhappiness, or vocationally ill-adjusted, is there conflict between instincts of ethics and so on. In such cases their bodies are made the scapegoat of their unhealthy state of mind. Intelligent method of approach is to find the root cause and treat such. Facts of life problems and personal difficulties are discussed with the above object in view.

To return to the subject of mental hygiene, in countries where mental hygiene movement has been in existence for some years, marked improvement in the care of the mentally sick, has taken place. A modern mental hospital is really a luxurious institution, with its reception wards fitted with special departments of dentistry, gynaecology, E. N. T., urology, X-ray and Hydro and physiotherapy equipments, psychological, biochemical, pathological and bacteriological laboratories, separate wards for medical, surgical cases, operation theatre, wards for disturbed patients, colonies for convalescent patients, workshops and occupational therapy departments of various types, recreational hall, music room, facilities for outdoor games and so on. It has become a place of research in addition to affording thorough diagnostic, therapeutic, teaching and nursing facilities. Psychiatric wards in General Hospitals or special buildings in the vicinity known as Psychopathic Hospitals, afford ample scope for diagnosis and treatment of early mental cases as well as for teaching and training personnel. Being situated in University centres, these hospitals have on their staff research scholars working on different problems of psychiatry and psychology.

The results of treatment of the mentally sick being still unsatisfactory when advanced, more encouragement is given for admission of cases in the incipient stages. Here the co-operation of the practitioners is much appreciated.

As bad behaviour patterns and reactions can be traced back to early life, preventive institutions of various types, to deal with the very young, are coming into existence. Habit and child guidance clinics, either stationary or travelling are established, to advise parents about training the children in good habits of feeding, sleeping, attention to toilet, control of the emotions, personality disorders and so on. Psychiatric clinics in connection with schools to handle cases of misbehaviour and educational difficulties and similar clinics in juvenile courts, special residential training schools for mental defectives to teach good habits, and

hand work suited to their capacity and inclination, form other important branches of mental hygiene, to make children become happy and useful citizens in after life. Exhibitions of maladjustment of children to their environment are recognised as something gone wrong in the mental health and attempts to nip in the bud, these incipient personality traits are made, to prevent later delinquency, criminalism, dependency and mental disorders.

Clinics are also introduced into Courts and prisons, to investigate individual criminals, so that the causes for their anti-social conduct may be ascertained, proper classification made and suitable rehabilitating methods adopted.

Teaching of psychiatry forms an important item in the curriculum of medical studies, especially in America. Psychology, Personality, study and Psychopathology form preliminary courses before clinical and theoretical psychiatric courses are started. Doctors are thus well equipped to detect early mental trouble and advise suitably.

Propaganda in mental hygiene, by means of lectures to parents, teachers, social workers and so on is systematically undertaken, so that, after some years the number of the maladjusted, the unhappy, the dependent, the inefficient, the delinquent and the mentally sick may be reduced.

The culminating success for the mental hygiene movement came with the holding of the First International Mental Hygiene Congress in Washington during last May. Over fifty nations and three thousand delegates took part to make it a great success. About fifty papers on mental hygiene subjects were discussed by experts of various countries and an International Committee of Mental Hygiene was established to make the movement world wide active.

It is hoped that through the efforts of the medical profession in our presidency, full benefit of the mental hygiene methods would be made available to increase the happiness and efficiency of our fellow citizens.

Club Foot

BY DR. M. G. KINI, M. Ch., F.R.C.S.

Club foot a term which was synonymous with talipes has been put on a more rational basis according to the pathological anatomy found in the deformity. Talipes is a generic term from the words talus (ankle) pes (foot) and is used as a prefix to other associated deformities in the foot.

The common deformities that are found in the foot are the following :

1. Talipes equinus (Rare in congenital condition).
2. Talipes equino varus, most common in congenital condition.
3. Talipes equino valgus, rare in congenital conditions.
4. Talipes calcaneus with or without varus or valgus but always associated with flat foot unlike acquired deformity.

The causes.—1. Congenital

2. Acquired.

1. There are various surmises regarding the actual causation of congenital talipes. Heredity seems to play a very important part as this condition has been known to occur in several generations.

2. Congenital talipes is often associated with other deformities like *spina bifida* hare-lip, congenital high scapula and congenital dislocation of hip. And sometimes follows the lines of Mendelian principle.

3. Berg has adduced a fine theory and he believes that the causation of congenital talipes is due to the persistence of the foetal position in the embryo of three months old.

4. Diminution of amniotic fluid has been attributed as one of the causes of this condition.

In acquired condition, acute anterior polyomyelitis contributes the majority of club feet.

The common deformity in congenital condition.—The most common deformity is talipes equino varus. Next comes Talipes equino valgus. Last Calcaneus 80 per cent of the deformities are of equino varus type.

Sex.—Male children are affected more than females in the proportion of 3 to 1.

Both feet are more frequently affected but sometimes only one is affected.

Pathological Anatomy.

1. *Deformity*.—The foot is plantar flexed at ankle joint adducted and inverted at the mid tarsal joint, looking like a club with a prominent convexity on the upper and outer aspect of the foot.

Changes in bone of the foot.—Astragalus. The neck is longer and retains the embryonic type and according to Keith this is one of the most important causes in the causation of club feet. It is also adducted and inverted.

Body.—The body of astragalus is dislocated forwards with the result it is broader on the superior surface and will be an obstacle while correcting the deformity in restoring the complete dorsi-flexion.

The medial surface is smaller and ill-developed and lateral surface is wider.

Oscalcis.—The anterior end is inverted.

The medial surface may be pointing upwards.

The whole of the oscalcis looks as if it is dislocated inwards.

Sustanteculum tali is nearer the medial malleolus.

Peroneal tubercle is further away from external malleolus.

Cuboid.—It becomes wedge shaped, wider laterly and narrower medially.

Scaphoid.—This is displaced medially and tubercle is very much nearer the medial malleolus and may be very prominent.

Other small bones participate in this deformity but are not of greater importance as the deformity is essentially at the sub-astragaloid and the mid tarsal joint. Sometimes the meta-tarsal bones are splayed out and the first meta-tarsal may be markedly adducted, so as to cause meta-tarsus varus resembling the pre-hensile foot.

Muscles.—The anterior group of muscles are stretched and elongated with the exception of tibialis anticus which is contracted and displaced medially.

The medial group chiefly the tibialis posticus is contracted and at its attachment to the navicular a dense structure often likened fibro-cartilage develops, which prevents the correction of the deformity.

Posteriorly the tendo achilles is contracted and may be displaced medially, a factor which should not be ignored while reducing the deformity as the tendo achilles which is displaced medially acts like a strong inverter of the foot.

Laterly the peronei are stretched.

Ligaments.—Changes in the ligaments correspond to the changes in the muscle and the contractions are the same as in the case of muscles. The calcaneo navicular and the anterior part of the medial lateral undergoes marked contraction and the calcaneol navicular appears like fibro-cartilage.

Plantar fascia also becomes contracted especially the medial limb. The posterior capsule of the ankle joint shares the same change.

Sole of the foot.—It is usually directed medially in mild cases but in very bad cases it is directed upwards and backwards and a distinct crease may be found at the level of the mid tarsal joint.

On the dorsum of the convexity in cases that have walked for a long time a callosity develops with a distinct bursa underneath.

It is often found that associated with this is a rotation of tibia at junction of lower and middle third, a factor often ignored but has been responsible for the recurrence of the deformity after its supposed cure.

I am not going into the signs and symptoms of the condition because a club foot does not require elaborate methods for diagnosis. I have tried to explain the pathological anatomy found in this condition with a view to impress that the cure of this deformity depends upon the proper understanding of the contractures and treating the same in a systematic way.

Prognosis.—Club-foot is a curable deformity but there are several types. A foot without heel and any with marked meta-tarsus varus does not give the same aesthetic result as in the case of a club foot with a prominent heel without the meta-tarsus varus, though functionally it may appear sound.

Treatment.—It is the first duty of every Surgeon who treat this deformity to impress upon the relatives the prolonged nature of the treatment and to get a successful result their co-operation is absolutely essential.

For treatment the club feet are roughly and arbitrarily divided into three groups.

1. From the time of birth
2. From the time of walking up to 5 years.
3. From this period to adult life.

It is difficult to put all club feet in these water-tight compartments but for the sake of treating in a systematic way it is necessary to have a definite plan.

In the first category we have a foot which can be easily moulded and for moulding the following points have to be noted in correcting this deformity.

1. Correction of inversion of the whole foot.
2. Correction of adduction of foot.
3. Correction of equinus condition of foot.

At the same time one has to deal with contracted soft structures which easily yield to moulding.

The treatment, therefore, really begins from birth, and the moulding of the foot is done by the Jones's grip and manoeuvre.

After correcting this it has to be maintained and this is done by adhesive plaster (ZO) preferably.

I have corrected one case so far which came to me 7 days after birth and it took me to correct the deformity nearly 1½ months. Splints have been designed to correct this deformity but it is a matter of personal choice.

The use of plaster of paris is not advocated in children of tender age, that is less than 3 months old but can be used after that period to maintain the correction which has been attained after the manoeuvres and manipulation.

Several plasters may have to be applied before the final correction is attained. And the mother must be warned that she must co-operate to prevent the recurrence by frequently coming to the clinic and secondly wearing a splint at night to prevent the deformity. This want of co-operation has resulted in many recurrences.

In the second category the deformity is more marked and the structures have contracted more with the result that simple manipulations will be of no avail. So instrumental aid like wedges, wrenches have to be sought for, to correct the deformity under anaesthetic and after correcting the foot must be put in plaster of paris. The correction may have to be done in several stages before a cure results. If manipulative methods fail, operative treatment has to be resorted to and various operations have been devised on soft structures, like that of Ober, Elmslie and Trethowen for correcting the contractures on the medial side. If bones have to be dealt with, operations like that devised by Hope, Elmslie Albee and Jones may be done.

In the third category there is only one treatment and that is operative. Though preliminary faciotomie and tendon lengthening may

have to be done and the operations of choice in this condition is either the one devised by Dunn or by Whitman.

My object in writing this paper is to impress upon all medical students the need for educating the public regarding the curable nature of this condition, and that children born with this condition may not be cripples for life.

Summary.—1. Club foot is a common deformity which is curable.

2. The deformity is in the mid tarsal joint and the sub-astragaloid and also in the ankle joint, with the resulting contractures.

3. In the treatment of this condition the quicker the treatment is undertaken after birth the better the result.

4. Tenotomy of Tendo achilles is an operation seldom done and tendon lengthening has to be done after great consideration as once the bow string action of Teno achilles is gone both the knee and ankle will be useless for weight bearing purpose.

5. In adult condition operative treatment gives the best result.

A Few Hints on Staining Methods as Applied to Bacteria

BY DR. SESHADRINATHAN, M.B., B.S., D.T.M.

The usual method of study of the morphology of bacteria is by making an emulsion of the organism (if obtained in culture) in saline and noting the various characters under the microscope.

First what is known as a hanging drop is made, which should be thin. A drop of the emulsion is transferred to the centre of a coverslip with a sterile platinum wire loop. The loop should be as small as possible about 2 m. m. diameter. The drop should be of good volume and should not spread out. Since the object of the hanging drop is to give us the information whether the bacterium is motile or not, the emulsion should be fresh and the loop sterilized and cooled, as the bacteria will lose their motility when a hot loop is dipped into the emulsion. A hollow slide is then taken and the edge of the hollow is lined with vaseline. This is then applied to the coverslip with the centre of the hollow facing the drop on the coverslip. The coverslip will stick to the slide and the drop will be suspended into the sealed chamber thus formed. The bacterial emulsion can then be examined under the microscope through the coverslip.

In examining the hanging drop students usually focus the edge of the drop. This may lead to error in judging motility. Owing to the adhesion of the bacteria to the margin of the drop where even motile bacteria will be seen to be stationary. Hence it is necessary to bring the centre of the drop into view to note true motility.

At the time of the examinations it is usual for students to lose their 'nerve' which at other times they exhibit to a remarkable degree. The result is that even the best students commit inexcusable blunders. For instance students have been known to describe 'motile cocci.' Though the remote possibility of motile cocci has been described in a few text books (probably they are tiny bacilli resembling cocci), for practical purposes it may be accepted that all cocci are non motile.

Some highly imaginative students have been found to describe bacteria moving with lashing flagellae. Flagellae of bacteria are seen only by special methods of staining and not in hanging drop preparations.

A Few Hints on Staining Methods as Applied to Bacteria 43

Economy of time is important to be observed at examinations. Much time can be saved in practical bacteriology examinations by preparing one or two hanging drop slides first, then half a dozen smears for staining. The hanging drops can be examined at leisure while the smears are allowed to dry in air. There will thus be no necessity to boil the emulsion in hurried attempts to dry the smears over the flame. To fix the smears the slides should be passed over the flame quickly two or three times. Some of the bad results in staining are due to the bacteria being boiled up in a wet smear or to charring of a dry smear while fixing.

Proper staining of bacteria is not a simple process but it is also not very difficult of achievement.

The simplest stains commonly used are dyes derived from coal tar products, the so-called aniline dyes. They are divided into basic aniline dyes and acid aniline dyes. The basic dyes are the stains of choice for bacteria since they have greater affinity for the bacterial body which is chiefly composed of nuclear material. The staining power of these are augmented by such substances as carbolic acid, aniline oil, and some metallic salts.

To study the morphology of bacteria at first a simple stain such as a watery solution of methylene blue is used, the smear being covered with the stain for about 5 minutes, then washed with water, dried and examined under the oil immersion lens. This will give a rough idea of the morphology of the bacteria contained in the emulsion.

The most important differentiating stain is what is known as the Gram's stain. By this method of staining bacteria can be grouped into Gram positive and Gram negative organisms. The steps in this method of staining are as follows:—

The staining solution consists of gentian violet in aniline oil, or methyl violet. The aniline oil has some augmenting function like carbolic acid.

The smear having been dried in air and fixed over a flame, is covered with the violet solution for $\frac{1}{2}$ to 1 minute. It is then washed with water and covered with iodine or flooded with iodine solution, which is allowed to remain or better changed several times for full 2 minutes. The essential feature of this method of staining is the treatment with iodine. The iodine fixes the stain on to the gram positive bacteria so that the subsequent treatment with a decolourising agent like alcohol is unable to remove the stain. On the other hand with gram negative organisms the

violet colour is easily removed so that another but lighter stain has to be used to enable us to see the bacteria whose colour has thus been removed. This stain is called a counter stain. It may be a weak solution of carbol fuchsin or neutral red etc. In treating the stained film with alcohol it is neither advisable to be frugal or to use excess of alcohol. Pour the alcohol drop by drop until no more violet stain appears in the out flowing alcohol. There are certain difficulties met with here. The gram method of staining is a puzzle to the students. Most students strongly believe that success in this method of staining is a chance. Considering the percentage of successful staining one will be tempted to concur with the students. The pity is that the examiners insist on a good gram stained slide being shown. The temptation to get inspiration from neighbours is therefore yielded to by some candidates with disastrous results. If the bacterium given is known, through such inspiration to be a gram positive one imperfect decolourisation with alcohol is resorted to or no alcohol is used. The stain deposit etc., around the bacterium will betray the student.

It may be adduced that some old cultures of gram positive bacteria from the laboratory do not retain the violet stain, usually such old cultures are not given at the examinations and the supply of alcohol is not also spared, so that the student may give liberal wash to the smear with it, the only essential being that the iodine has been left on the smear for full 2 mts. before decolourising.

After decolourising, the counter stain consisting of a dilute solution of neutral red or carbol fuchsin is used. This stain is left over the smear for 2 to 5 minutes. Gram negative organisms and tissue cells will be stained pink with this stain.

The slide is then washed in water, dried and examined under the microscope.

There are modifications of gram method of staining but the students can attempt to learn them when they become specialists in the subject.

Having known that the bacterium is gram positive or gram negative the next attempt should be to apply special method of staining to identify the same suggested by observations on the morphology and motility.

These special methods of staining will be dealt with in a subsequent number.

Flocculation Tests for Syphilis

By DR. K. V. KRISHNAN, M.B., M.R.C.P.E., D.B. (LONDON)

(Member, Kala-Azar Commission).

Introduction.—The great simplicity and fair reliability of the different flocculation tests for syphilis are making them increasingly popular all over the world. In the United States the Kahn test has practically replaced the Wassermann reaction in most of the important laboratories. In Great Britain, the staunch adherence to the time honoured Wassermann reaction is being slowly broken down and it is now not uncommon to see some of the leading laboratories in London, Glasgow and Edinburgh adopting the flocculation test with very promising results. In continental laboratories also, one or other of the many flocculation tests is being used in the routine diagnosis of syphilis. There is no doubt that with certain improvements in technique and in the method of standardisation of the antigens used, the results obtained with these tests will be as reliable and as specific as those of the Wassermann reaction itself. For, after all, when one considers the relation between the flocculation tests and the Wassermann reaction, it appears the interaction between the serum and antigen is essentially the same in the two cases. In the flocculation tests the ratio between the reagents is such that the end result of their inter-action becomes readily visible, whereas in the Wassermann reaction the ratio is quite different and the end result is not visible, a special indicator in the nature of a haemolytic system being used to detect the reaction. Dean, who is a recognised authority on the nature of these reactions writes "the process which causes precipitation and complement fixation is in all probability the aggregation of molecules present in the anti-sera by the action of the antigen. When sufficient antigen is present aggregation takes place energetically and flocculi are rapidly separated. These are the conditions favourable to the precipitation reaction. If a relatively smaller amount of antigen is present the process of aggregation is slower and less complete. These are the conditions where the individual particles which form the precipitate are extremely small, but the surface afforded by all the particles of such a precipitate must be larger than that offered by a precipitate composed of larger flocculi. It is indeed possible that

there is a direct relationship between the surface area of the particles of the precipitate and the amount of complement absorbed..... It can, in fact, be shown experimentally that either complement fixation or precipitation can be produced with the same antigen and the same antibody by merely varying the proportions of the mixtures." From this work of Dean, which has been further confirmed by Sachs, it is but just to presume that in the very near future these simple flocculation tests will become the standard methods for the routine diagnosis of syphilis.

The flocculation tests.—Although a very large number of flocculation tests have been described for the diagnosis of syphilis, the two most popular and reliable are The Sachs Georgi and The Kahn tests. And these alone need be described here.

The Sachs Georgi test.—(Mackie's modification):—

This, of course, is the simplest and the cheapest of the flocculation tests.

The antigen.—Is a cholesterolised alcoholic extract of sheep heart muscle and is made as follows:—

20 grammes of sheep's heart muscle (wet) freed from fat and fibrous tissue are taken, ground with sterile sand in a mortar to a fine consistency and transferred to a 250 cc. flask. Then 100 cc. of 96% alcohol are added and the mixture allowed to stand for 4 days during which time the flask is shaken once or twice a day. Finally it is filtered and sufficient cholesterol added to saturate it. It is now ready for use.

The articles of *apparatus* required to perform the test are the ordinary graduated pipettes (1 to 10 cc.), glass tubes (size 7.5 cm. length by 1 cm. diameter) and incubator. All these are usually available in every laboratory.

The *test* itself is done by adding one part of a 1 in 6 dilution of the antigen to two parts of varying dilutions (1 in 2, 1 in 4, 1 in 8, 1 in 16, 1 in 32 and 1 in 64) of freshly inactivated serum and incubating the mixture from 4 to 12 hours at 37°C., before reading off the results. The quantities of the reagents used are large (.4 cc. and .2 cc.) and do not allow of any great error from measurement as in the Kahn test.

The *results* obtained with Mackie's modification of Sachs Georgi test in the past though fairly satisfactory, did not completely agree with those of the Wassermann reaction. This lack of agreement was chiefly due to the absence of an efficient method of standardising the antigen. This defect, however, has recently been remedied by the writer (1929)

Flocculation Tests for Syphilis

who has described a method of standardisation by which each new preparation of antigen can be brought up to uniform optimum sensitiveness.

The second complaint against the Sachs Georgi test is that it is not as rapid as the Kahn, the results being obtained only at the end of 4 to 12 hours incubation. This delay, if it may be called so, is inevitable, due to the very dilute form in which both serum and antigen are used in the test. It is well-known that in all precipitation reactions the time taken to obtain results is closely related to the ratio and the concentration of the reagents used. The nearer these two factors approach the optimum conditions the quicker the result. Also mechanical agitation hastens flocculation. Taking into consideration the fact that syphilis is not an acute illness in which a very urgent laboratory diagnosis is needed a delay of 4 to 12 hours in obtaining results is not to be considered a point against the test.

The third and probably the gravest charge against the Sachs Georgi test is that, as a result of incubation for about 12 hours, bacterial growth is likely to occur in the serum-antigen mixtures. This would, if present, undoubtedly, interfere with the reading of results. As perfect aseptic precautions are not taken in the performance of the test this objection is certainly of some importance. The writer's experience with a large number of these tests shows that with ordinary cleanliness and care this objection becomes indeed very negligible.

The Kahn test.—The greatest advantage of this test is the rapidity with which results can be obtained.

The *antigen* is made by a more elaborate method and involves greater time, labour and cost. It is a cholesterolised alcoholic extract of aether-extracted dry, powdered heart muscle and is made as follows:—

Twenty-five grammes of dry powdered beef heart muscle are extracted in a 250 cc. flask at 10 minutes intervals with 100, 75, 75 and 75 cc. of ether successively. After the last extraction the re-dried powder is extracted for 3 days at room temperature with 95% alcohol in the proportion of 5 cc. per gramme of powder. To the alcoholic extract is then added cholesterol in the proportion of 6 mgrms. per cubic centimeter. It is then standardised by the use of two reagents for correction (1) 0.6 % alcoholic solution of cholesterol for diluting antigens more sensitive than standard and (2) a "sensitising reagent", (made from the ether extractives obtained in the preparation of his antigen) for antigens less sensitive than standard. This introduces fresh errors in the antigen.

The *apparatus* required to perform the test is not quite as simple as in the Sachs Georgi, special accurately graduated pipettes for

measuring small quantities such as '05, '025 and '0125, shaking machines, etc., being required. And these help is enhancing the relative cost of the test.

The test itself is very simple and is performed by adding varying amounts ('05 cc., '025 cc., '0125 cc.) of the antigen saline suspension (1+1'1 saline) to a definite quantity (0'15 cc.) of fresh inactivated serum and shaking the mixture in an electric shaker for 3 minutes. The mechanical agitation, together with the optimum concentration of the two reagents used, help to bring about the maximum flocculation in the minimum period of time. To each of the tubes after the shaking 1 cc. of saline is added and the results read off.

A study of the results of the Kahn test shows that in most hands, there is very close agreement with those of the Wassermann reaction. The lack of complete agreement sometimes complained of is mainly brought about either by errors in the measuring of the very minute quantities of the antigen saline suspension or by the use of antigens imperfectly standardised. The latter, probably, is more often the case, for in the experience of many, the Kahn antigen is rather difficult of accurate standardisation even when one follows his directions fully. Some of the antigens obtained from Kahn himself are at times found to be below the optimum sensitiveness.

The Standard antigen.—It has been recently shown by the writer (1930) that an antigen called the "standard antigen" can be prepared for use in all flocculation tests and the Wassermann reaction. The only difference is that different dilutions of the antigen are used in the different tests. The results so far obtained by the use of this "standard antigen" (in a dilution of 1 in 6) in several hundreds of Sachs Georgi tests carried out in the Department of Bacteriology of Edinburgh University are in complete agreement with those of the Wassermann reaction. This antigen is also being used in the Wassermann reaction, (in a dilution of 1 in 12) and in the Kahn test (in a dilution of 1 in 1) with very satisfactory results. (For details regarding the Standard antigen reference may be made to the original articles).

Conclusion.—From the observations made above it will be evident that the chief drawback in all flocculation tests has so far been the absence of a reliable method for the efficient standardisation of the antigens used. Now that a simple reliable method is available for preparation and standardisation of antigen it is hoped that these simple tests will soon replace the complicated Wassermann reaction. There

seems to be no doubt that all flocculation tests are equally good. Each one has its own special advantages and there is very little to choose between them. It is purely a matter of personal choice what flocculation test is used. Provided the antigens have been properly standardised all must give absolutely concordant results.

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Some Observations on Ascites

BY DR. N. S. NARASIMHAN, F. R. C. S.

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The normal peritoneal cavity absorbs fluid with great readiness and saline solutions, glucose and blood are often injected by this route. The healthy peritoneum can absorb fluid equalling 3 to 8% of the body weight in an hour. Water and crystalloids are absorbed by the sub-peritoneal capillary blood vessels and the more diffusible colloids may pass into the blood stream in this way. Coarser colloids and particulate substances such as blood and bacteria are absorbed through the diaphragmatic lymphatics into the thoracic duct. The basement membrane, which elsewhere forms a continuous sheet under the surface of the peritoneum, breaks into a fenestrated membrane on the surface of the diaphragm and particles are able so pass through the fenestrations to reach the lymphatics. They are forced into the lymphatic system by the contractions of the diaphragm and the abdominal muscles. So in chronic peritonitis, ascites occurs, as the peritonium is thickened and the diaphragmatic lymphatics are blocked.

Similarly sepsis in the abdominal cavity produces a basal effusion in the pleural cavity but no cases have been ever recorded of infection in the abdominal cavity from the pleura. Reverse flow of lymphatics does not take place in this region.

During abdominal operations, especially for Duodenal ulcer, the liver has been found to be deranged to a varying degree congested, enlarged or cirrhotic in varying degrees. The same pathogenic factors which produce the cirrhosis may contribute or aggravate factors tending to the production of the duodenal ulcer.

The ascites of cirrhosis is usually attributed to obstruction to the portal blood flow. In uncomplicated cases the peritoneum is smooth and a little thickened and shows no signs of inflammation. The fluid has the characteristics of a transudate—low Sp. Gravity, little albumin no fibrin, the cells being desquamated epithelium.

Ascites occurs in about 2/3 of the cases of sudden thrombosis of the portal vein, disappearing gradually as the collateral circulation develops and ascites can sometimes be produced in animals by ligation of the

portal vein. Portal hypertension acts mainly as a predisposing cause and in most instances an additional factor is present. The reasons for this conclusion are that advanced cirrhosis and portal congestion are often found post mortem or during operations without ascites, and ascites in cirrhosis is usually a terminal event; duration of life after it occurs is 6 months. The preliminary symptoms may have lasted nearly for 6 years, and liver plays a large part in the normal metabolism of water as is shown by the fact that generalised dropsy may occur in acute yellow atrophy and in toxic jaundice. Evidence of a disturbed water balance is shown by the diminished output of urine, by the absence of the normal variations in the quantity and concentration of the urine at different times of the day and by failure to eliminate ingested water with the same promptitude as in health.

Patients who return for repeated tapplings over periods of 5, 10 or 15 years are not suffering from cirrhosis but from chronic peritonitis.

Portal obstruction alone is not the cause of ascites in cirrhosis and if the patient does not respond to medical treatment he is not likely to benefit by an operation. The Talma-Morrison operation for ascites is falling into disuse; and has not been of any benefit in these cases in my experience.

Editorial

We offer a hearty welcome to Major-General C. A. Sprawson, C.I.E., M.D., F.R.C.P., who has taken over the duties of Surgeon-General with the Government of Madras. His keen interest in the future of the Medical College and of Medical Education in general will we hope be of great value to us during these controversial times.

The representatives of the Indian Universities have all met in conference and have decided to constitute an All-India Medical Council so as to create a uniform standard of medical education all over India. The Council to be constituted will be presided over by the Director-General, Medical Services, and will have one representative of Medical teaching from each University, besides one elected by the Medical Graduates from each University and one nominated by each Government. Besides these, there will be three nominated by the Government of India. The Director-General of the Medical Services will preside over the Council for the first three years after which the President will be elected from amongst the members.

The recommendations of the Medical Education Committee, report of which has been published, are of great interest. Though we are not inclined to agree with the general trend of economizing at the expense of officers who are already in service where professorial appointments are concerned, we are in general agreement with the main features of the Committee's report. Increasing the course of the Medical Schools so as to cover a period of five years is certainly a step in the right direction but one is inclined to wonder if it is wise to keep the standard of general education as it is at present. If the minimum qualification

for admission to Medical study is kept down to the School Final standard, difficulties of language and scientific training would have to be met. It is expected that about 100 students will be trained in the Medical Schools of Royapuram and Tanjore.

The General Hospital Out-patient department is now nearing completion and we hope to be able to carry on diagnosis of treatment of out-patients on more modern and scientific lines. The foundations of a new surgical block have been laid and this together with the new Out-patient department will form the new block of the hospital. With the shifting of departments in the Medical College the Anatomy department is to take the place held by Physiology so far and Physiology will be taught where Hygiene has been before.

We take the opportunity to welcome back from England Dr. Guruswamy Mudaliar, the President of our Magazine Committee and till lately the Editor of this Magazine.

We extend our hearty congratulations to the Graduates of the year and to the candidates who have secured the B. S. Sc., M. D. and M. S. degrees. To the old students of the College who have secured distinction abroad in foreign Universities we offer our hearty congratulations.

The amalgamation of all the separate tiffin rooms into one large common room is an happy event and a sign of the times for which we congratulate the students and their enterprising Secretary. The opening of the new buffet we hope will prove of great help to the students.

Office Bearers of the Madras Medical College Association

The Executive Committee :

President.—Lieut.-Col. C. A. F. Hingston, I.M.S.

Vice-Presidents.—Major Pandalai, I.M.S., Dr. Lakshmanaswami Mudaliar, M.D.

Secretary.—Mr. G. M. Thambidurai (Student, Final year).

Assistant Secretary.—Mr. Purushothama Pai (Student Fourth Year).

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Fourth Year—Mr. Fred. Martinus.

Third Year—Mr. George Fletcher.

Second Year—Mr. K. Rama Rau.

First Year—Mr. Syed Ali.

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B.S.Sc.—Dr. Shanmugasundaram,

M.B.B.S.

Representative of the Military Students.—Mr. A. Saldhana.

" " *Women Students.*—Miss A. Kolandavelu.

The Magazine Committee.

President.—Dr. Guruswami Mudaliar, M.D.

General Editor.—Dr. Bhaskara Menon, M.D., M.R.C.P. (Lond.)

The Two Vice-Presidents of the Executive Committee.

Associate Editors.—(1) Dr. Mangesha Rao, F.R.C.S.E.

(2) Not yet elected.

Student Editors.—Isaac J. E. David (Final Year).

Mr. Pubala Rayan (Fourth Year)

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The Executive Committee of the Athletic Club.

President.—Major Pandalai, I.M.S.

Vice-President and Treasurer.—Dr. Mangesha Rao, F.R.C.S.E.

Secretary.—Mr. Ravi Varma (Student Fourth Year).

Office Bearers of the Madras Medical College Association 55

Major A. M. V. Hesterlow, I.M.S. *Secretaay of the Intercollegiate Athletic Association.*—Dr. S. Ramakrishnan.

The Captains of the various teams :—

Cricket : Fred. Martinus (Student Fourth Year).

Hockey : A. Saldhana (Student Final Year).

Football : Balakrishnan (Student Fourth Year).

Tennis : J. Eapen (Student Final Year).

Dramatic Club.

Joint Secretaries.—Miss B. Mahadevan (Final Year).

• Mr. N. Srinivasan (Fourth Year).

Dodging the Doctor.

BY "THE INAUGURAL STRUTTER."

"Cough," he said, and I coughed; not exactly coughed, not exactly, but made noise calculated to attract attention.

"Breathe deeply," he said, and I tried my level to ape an express labouring up a stiffish bit.

"Open your mouth," and I gaped my jaws like a wee birdie hoping for the early worm.

The Doctor prodded an aseptic but foul-smelling forefinger into my mouth, and it took all the saint in me to refrain from clapping the incisors together.

"Well," he began -- I was mentally preparing myself for a long sea voyage, a sanatorium, Malt extract and even an asylum for incurable consumptives," and eagerly awaited the expert's choice.

"Well," he said "You are not particularly bad now." His voice had the ring of appreciation in it.

"Then, I guess we shall part company in the best of spirits!", I said grinning.

"Not so fast, not so fast," he said quickly, "although I don't find you are phthisical, I still think that you should go through a course of touching of the throat." This matter, touching the throat, I did not grab clearly, and I contracted the brows into a question mark. "You see there are aggregations of Lymphoid tissue all over the shop, and the throat has the favourite's share of it. Now, and again these aggregated lymphoid masses, and of these, pre-eminently the throat, go off on a spree, so to say, and of those gone off on said spree none so far gone as the throat. Hence you see, or you don't, according to your hereditary tendencies, there is a lot of trouble brewing in the throat, ready to assume a threatening look, and this said look of threat may be fulfilled, unless the malady is tackled and mind you properly tackled, by which I mean efficiently tackled.

"Right O!" I said, "tackle it."

Yes I had said the word; I who had fought shy of being tickled between the eight and ninth (I think it is the eighth and ninth) ribs when I was barely eight or nine, (I think I was barely eight or nine then) I had given permission to an utter stranger of a man to tackle it. I did not even halt to question whether he had a diploma to tickle, not to say tackle throats: nor even to ponder if he would kill or cure; and if it came to "dispatch premature" whether the jury would regard it as "A wilful

Dodging the Doctor

57

Murder" or "A death due to sudden Heart-failure" well, well, all this I did not stop to consider, nay, the whole lot of possibilities did not even occur to me. Which is surprising, as the whole lot of possibilities did occur to me later; but of this more later.

Well, well, to make a long matter short or to make a sour matter sweet, which amounts to about the same thing the doctor (?) advised (or do you spell it with a 'c'?) me to do various things of which the following deserve mention:—

"Take this mixture three times a day.....er... ..yes after meals." It sounded as though it did not matter a rap whether the bottled bilge was taken before or after food.

Another item on the prog. was "Rub this linement over your chest"; another was "Use this Paint as directed." And a few others were "Warm this Paste and dabb freely on the neck, chest, and throat; Use this gargle as frequently as you can; take this sleeping draught when you are not asleep, etc., etc.

With reference to the aforementioned gargle, I only want to point out that a 'can' is one thing and a 'will' is another. "Obviously!" I can hear some one mutter; quite so.

What with the drinking of 'The Mix.'; and the rubbing with the 'Linement' and the painting with the 'Paint'; and the dabbing with the 'Paste'; and the gurgling with the gargle, I say what with all this, I am surprised that the old Adam's Apple didn't go on a blooming strike till better relations could be established; or, what would have been more to the point, how it did not get cut right across from left to right or from right to left for the matter of that.

* * * *

I wish to pass over this painful quarter in my life and hurry over to record that in the long run I was cured. It is to this day a moot point between the Doctor and myself whether he cured me or his medicines did (which is quite a different thing don't you know?); but cured I was, and I can very well imagine the anxiety of the much relieved Doctor to reap the harvest of all his labours. If I could not imagine all this the doctor's bills which came like fleas at a dead rat subsequently, amply served to illustrate the greed that lurks under the guise of professional zeal in some quarters.

Those were not pleasant days to linger over. The Doctor was a peculiar man; with him the question of monetary recompense assumed

At the Oral Exam :

Examiner : Can you deliver the goods ?

Student : I shall try Sir.

Examiner : Classify Nephritis.

Student : Nephritis ; Nephritidis ; Nephrosis ; Nephropathies ; Unilateral Nephritis ; Nephrectomy ; Nephrotomy ; Nephews and Nephralgia.

Examiner : I sentence you for another six months.

" Well," said Ram " you don't notice the knock in my car so much now, do you ?"

" How did you manage it ?" asked Dhar.

" Oh, I just loosened one of the mudguards."

" Annie " (apologetically) : " I took the recipe for this " vadai " out of the cookery book."

" Anna " (tactfully) : " You did quite right, darling. It never should have got in there."

" Now," thundered the Professor on a morning of unusual density on the part of his students, " You are all blockheads, but there must be one among you who excels in something, even if only in crass ignorance. Let the biggest dunce stand up."

To the prof's surprise a student stood up.

" Oh, said the Master, I am glad to see that one of you has the honesty to admit his ignorance."

" 'Tisn't that, Sir," said the student ; " but I 'adn't the 'cart to see you standin' there by yourself !"

Geo Kuri took his " Levis " to a garage for repair.

" How much did you pay for this contraption ?" asked the mechanic.

" As a matter of fact," replied G. K., " My Mother-in-law gave it to me for nothing for my wedding."

The mechanic shook his head. " You've been swindled," he said.

" Topics in Brief."

The Romans made their roads straight so that the Britons could not hide behind corners.

Thomas A. Becket lived a dissipated life. Three nights killed him.

The penalty for bigamy is seven years and two Mothers-in-laws.

A psychologist claims that woman wears better than man. She may wear better but not nearly so much.

In many Eastern countries, we read, it is the correct thing to remove the shoes before entering a house. Many late husbands have unknowingly been copying the customs of the East for a long time.

In some of the larger cities schools for teaching women to shop are being established. Now it is up to somebody to endow our leading universities with a professorship to let birds know what their wings are for.

" Who invented high heels ?" asks the writer. One theory is that it was the idea of a short pretty girl who was continually being kissed on the forehead. So little girls... ..

Some lipsticks have been declared poisonous, but women always did love men who defied death.

It's our guess that even if long skirts do succeed in coming in they won't stay long.

According to a doctor singing warms the blood. We have heard some that has made ours positively boil.

Falling in love is recommended in threatened nervous break down. A cynical correspondent says that a far less dangerous remedy is to fall in front of an Omnibus.

There were fifty-eight slight earthquakes in one day recently in Hawaii. No wonder, those Hula maidens can dance !

A married man reports that while the cooing stops soon after the honeymoon the billing goes on for ever. So, marriage comprises of ' billing and cooing ' !

Dr. from Kilpauk reports that the Kilpauk football team while playing against a Town team displayed unique stunts and won a glorious victory in spite of having kicked the ball into their own goal now and then.

At a meeting of our Association a Pre-registration student proposed his own name for the sports Secretaryship and appealed to the chairman to get somebody to second him.

At the above meeting to get the quorum Thambi had to pull out studious students from the library and the dissecting theatre, and also had to search the highways and byways and the bus stands where tired students were waiting to go home. He was even about to phone up to the different hostels but the timely advent of a dozen grandly but uncomfortably dressed sweating " Pre-registrations " saved the situation.

colossal proportions ; and the question of expenses over stationery was obviously a minor matter.

Those, you know where pre-war days, and paper was cheap, at least it was not so dear as it is now-a-days, (" why waste so many then ? " I can hear some one hiss !) and the Doctor bought a ream " slightly shop-soiled, excellent value " at a ocean-bed price and he spread no time or patience to send me bits of the same brand regularly ; which I should say was very kind of him seeing that I more often than not treated them like cake at a stranger's party.

I, an enthusiastic member of the Society for the suppression of Bills, had little trouble in dodging these tokens of Forget Me Nots from the Doctor. Old war horse that I was I hung up a neat natty board at my door with the crisp but constant information that the owner of the board was " Out ".

Whilst thus engaged in peering into the Relief Map of the Doctor's servant, an inadvertant sneeze betrayed the Hidden Peril and I had to come out and deny myself thrice before the cock would crow and leave the spot. Well, this Johnny proved to be the trusty valet and all that. The Pest had been enquiring in the neighbourhood and some of my neighbours not knowing anything about the strained state of affairs between me and the Pest, (then why didn't they keep quite ?) gave out my cognito with the result that I had to tip the Pest a couple of my room-mate's cigars before I could get the landscape clear ; and quite like the Pest that he was he returned daily for his two smokes per diem ; even repeating his unwelcome visits twice in the day ' when he felt chatty ' as he chose to put it ; till at last I had to promise to pay up on pay day. My friend's cigar box was getting dangerously emptier every day, the Doctor was as insistent upon payment as ever, and I said to myself " look here laddie the game is up and it is time to quit ! "

I mortgaged the rooms and their contents, (my friend was out of town you see) scrapped some money and made a dash for the metrop.

Well, well, here we are at the citadel so to speak, and the old tram is going merrily along as of yore with the " Amjikai " buses, and everything bright and chirpy. Days roll on as they have a nasty habit of rolling on, and the mortgage money along with them. What next ? Just then whom should I meet on stepping out of the Tram ?—The bloke the Doc ! I made a dash into a barber's saloon which stood invitingly open and had a leisurely shave. But lo, I had an empty purse ! How to pay ? The barber, instead of a pound of flesh, took revenge upon me by shaving off my mush ! what a disaster ! The glory of a man is his mush and I lost it !

In Lighter Vein.

(BY THE STUDENT EDITOR.—I. D.)

" Laugh and the world Laughs with you."

" Smile a while, and while you smile
Another smiles and there are miles and miles of smiles."

Doctor (to patient).—" Have you tried gargling with salt water ?"
Sailor.—" I should think so ! I've been shipwrecked six times."

Doctor.—" This is a very sad case. I am afraid your wife's mind is completely gone."

Husband.—I am not a bit surprised. She has been giving me a piece of it every day for the last fifteen years."

Thamby is in the habit of opening his Bible at random and taking the first thing his eyes alighted upon as something that would be helpful to him.

The other day the first verse he read was, " Judas went out and hanged himself," and not thinking this quite suitable he tried again at another place, and this time he read " Go thou and do likewise." He tried a third time and to his amazement his eyes fell upon the verse " What thou doest, do quickly."

P.—I say, there is a nice film at The Wellington to-morrow.

D.—What's that ?

P.—" Lies Miserable." They ought to call it " Miserable lies."

D.—Who is acting in it ?

P.—Victor Hugo

D.—What ! Did not " The Man who Laughs " write

" Victor Hugo " ? " Victor Hugo " is the name of a book.

He.—" Darling, in the moonlight your teeth are like pearls."

She.—" Oh, indeed ! And when were you in the moonlight with
4 earl ? "

Flowers Gathered from Many Gardens.

"Whatsoever things are lovely, whatsoever things are honest, whatsoever things are just, whatsoever things are true, whatsoever things are of good report, whatsoever things are pure and whatsoever things are mighty, think on these things."

—PAUL THE APOSTLE.

"Strength of numbers is the delight of the timid. The valient of spirit glory in fighting alone. And the valour of the spirit cannot be achieved without sacrifice, determination, faith and above all humility."

—M.K.G.

"Laugh and the world laughs with you ;
Weep, and you weep alone ;
For the sad old earth
Must borrow its mirth,
It has trouble enough of its own.
Sing, and the hills will answer ;
Sigh, it is lost on the air ;
The echoes bound
To a joyful sound,
But shrink from voicing care."

—ELLA WHEELER WILCOX.

The problem of prevention of plague and extermination of rats was a very distressing problem in Hamelin Town, in Brunswick, five hundred years ago. We would have heard about that old story in childhood.

RATS!

They fought the dogs, and killed the cats,
And bit the babies in the cradles,
And ate the 'iaddus' out of the vats,
And licked 'gulab-jan' from the boys' own saucers,
Split open the kegs of salted fish,
Made nests inside double breast coats,
And even spoiled the women's chats,
By drowning their speaking
With shrieking and squeaking
In fifty different sharps and flats.

Victory.

To suffer woes which hope thinks infinite,
To FORGIVE WRONGS DARKER THAN DEATH OR
NIGHT,

To DEFY POWER WHICH SEEMS OMNIPOTENT,

To love and bear, to hope till hope creates
From its own wreck the thing it contemplates,

Neither to turn nor falter nor resent ;

This like thy glory, Titan, is to be—

Good great and joyous, beautiful and free,—

This is alone life, joy, empire and victory.

—FROM SHELLEY'S "PROMETHEUS VINCTUS."

How Does Love Speak ?

How does Love speak ?

In the faint flush upon the tell-tale cheek,

And in the pallor that succeeds it ; by

The quivering lid of an averted eye—

The smile that proves the parent of a sigh :

Thus doth Love speak.

How does Love speak ?

By the uneven heart throbs, and the freak

Of bounding pulses that stand still and ache,

While new emotions, like strange barges, make

Along vein-channels their disturbing course,

Still as the dawn, and with the dawn's swift force :

Thus doth love speak.

How does Love speak ?

In the ... of that which we seek

The sudden suence and reserve when near ;

The eye that glistens with an unshed tear ;

The joy that seems the counterpart of fear,

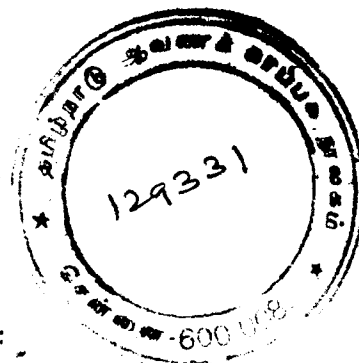
As the alarmed heart leads in the breast,

And knows, and names, and greets its godlike guest :

Thus doth Love speak.

How does Love speak ?

In the proud spirit suddenly grown meek,



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The haughty heart grown humble—in the tender
 And unnamed light that floods the world with splendour;
 In the resemblance which the fond eyes trace
 In all fair things to one beloved face;
 In the "hyperaesthetic" hands that thrill and tremble;
 In looks and lips that can get hyperaemic;
 Thus doth Love speak.

How does Love speak?
 In wild words that uttered seem so weak,
 They shrink ashamed to silence; in the fire,
 Glance strikes with glance, swift, flashing, high and higher,
 Like lightnings that precede the mighty storm,
 In the deep, soulful stillness; in the warm,
 Impassioned tide that sweeps thro' throbbing veins,
 In the embrace where madness melts in bliss,
 And in the convulsive tremor of a kiss:

Thus doth Love speak.

—ELLA WHEELER WILCOX.

A Puzzle Verse.

"There's a River that flows for ever
 Into the SILENT Sea:
 No one ever could tell me whether
 It's known to any but me!
 . . . Could you?"

—J. J. TAYLOR.

Memories.

There where the silent river flows,
 There where the golden kingcup grows,
 There where the silver sunlight glows,
 There where the sighing zephyr blows,
 There in a nook that nobody knows,
 Where in Maytime a hawthorn shows,
 And a rosebush in June bears a single rose,
 There I hear at each day's close
 Thy voice a-calling me.

In Lighter Vein

And it sounds so sweet in the twilight blue,
 My eyes grow moist as I dream of you—
 I remember the time when that call was true,
 And I loved thee; and I knew that you knew
 My heart was calling thee.

—MARY E. NIVISION
 —(Culled from "Madras Mail.")

"We look before and after
 And pine for what is not!
 Our sincerest laughter with some pain is fraught,
 Our sweetest songs are those that tell of saddest thought."
 —SHELLY'S "SKYLARK"
 The best flower with a brimming cup may stand
 And share its dew drop with another near"

—E. B. BROWNING.

Random Notes

By L. J. E. DAVID, STUDENT EDITOR,
Final Year.

The inaugural meeting of our College Association was held on Monday, the 21st of July when Dr. Kini, M. CH., F.R.C.S.E., gave the Inaugural Address and Dr. Gurusamy presided. The address inspired us to seek and acquire a real longing for research. We appeal to the Doctors to devote a little more of their busy time to encourage, guide and initiate us in our clinical work.

It is a great pity that our inaugural meeting was attended by only a moderate number of students. We ought to put in more enthusiasm into such gatherings where alone we meet together. It is *our* Association, and we ought to strengthen it and uphold it by all means possible.

We are very happy to have Dr. Gurusamy back in our midst in our wandering in "Florence", "Hobart", and "Pottinger". We don't mind being teased by his incessant volley of questions!

We also take the opportunity of expressing our joy and pride in welcoming into our midst Dr. Koshy and Dr. Kini, who have come back to us with fresh honours.

Our thanks are due to Dr. McGrath and our indefatigable Secretary for giving us a common dining room. Much improvements are needed before it could be much of a dining room, but all the same, the separating walls which for years all of us wanted to get rid off have at last come down with a great crash. No walls divide us in our college, but this pulling down of the offending structure has been very symbolic of the various sectional walls which are falling down in rapid unison all round us, since recent years, throughout the length and breadth of our country. "And, behold, the veil of the temple was rent in twain from the top to the bottom and the earth did quake and the rocks rent!"

It is a remarkable feature that this year a lady student was elected unanimously as the "Rep." of the final year, and a military student was

Random Notes

67

elected as the "Rep." of the fourth year. Last year a military student was our Asst. Secretary. We wonder, whether there is any necessity for having separate representatives in our Committee from the ladies and the Military students. Originally there was a practical wisdom underlying this, but this is not the ideal. We are all members of The Medical College Association in the capacity of being students of the college. The present method smells of "Reservation of seats" and "Communal electorates"!!

We raise our voice with one accord once more appealing for a hostel. This shows our faith! Faith never dies!! Like refugees we wander from place to place all over the face of our city. During April, May and June we suffer most. The doors of all the hostels are then closed against us. 'The foxes have holes, the birds of the air have their nests,' but we, busy hardworking students, do not have a lap to rest our head! Joking apart, we appeal to the Powers that be to feel for us deeply, right deep in the recesses of their hearts. We have lost many of our comrades due to Enteric and other maladies in recent years.

Our Indian Restaurant started this year is a great boon to us who do not often have time to go out to have our inner man satisfied. Our thanks are due to our energetic Secretary Mr. Thambidurai.

Case Reports

BY P. RATNASAMY, B.A., M.D.

Looking over my notes of cases when I was a House Surgeon at the Government Maternity Hospital, I found that the two following cases were not only rare but also lend themselves to discussion:—

CASE I. Mrs. A. aged 32. ^{L. C.} IV Para. _{R.C.}

EXTRA UTERINE FULL TIME "LIGAMENTOUS" PREGNANCY.

History of previous pregnancy: The third pregnancy was an extra-uterine on the left side which was operated on and removed; the pregnancy was most probably in the left Fallopian tube.

History of the present pregnancy: When the patient was seen at what was apparently the 6th month the shape of the uterus was irregular and there was also apparently a cystic tumour which could be felt per vagina. When seen some time later the shape of the uterus was still irregular and the cystic swelling was noticed to be receding from the vaginal dome. X-Ray taken showed a foetus of about six months with the 'breech' at the lower pole.

Examination: Notes on 21st February 1928. No tumor or cyst felt per vagina. Shape of uterus still irregular; advised to come back a month later if there was any trouble.

The patient was admitted on 10th March 1928. Examination showed a thin, anæmic woman; shape of uterus irregular; height about 8 months; position of foetus R.O.A.?; F.H. 144 per min; heard feebly in the right lower quadrant. There was a slight brownish discharge.

Patient was again examined on 4th June 1928. The foetal heart was not inaudible; the yellowish discharge persisted.

On 13th June 1928. Palpation of the abdomen did not reveal any satisfactory details. F.H. was inaudible; uterine souffle was heard on the left side. The general condition was not satisfactory. She had a low temperature and a quick pulse. There was still a dirty, brownish discharge; os was patulous admitting one finger and the cervix was pointing upwards and forwards. A rounded cystic swelling was now

Case Reports

felt at the posterior fornix. The foetus was supposed to be dead. As labour did not set in, a sound was passed into the uterus on 23rd June 1928 which went in for 3" and was deflected to the right side of the main swelling. The foetus was diagnosed as extra-uterine. The general condition of the patient was none the better. She was operated on 25th June 1928. On opening the abdomen it was found that the pregnancy was in the stump of the left tube. The gestation sac had ruptured into the left broad ligament. The sac was incised and a full term macerated (1st degree) foetus was extracted. The dead placenta was removed as also the major portion of the gestation sac. The sac had contracted adhesions all round especially to the transverse colon. The uterus was pushed to the right side and to the right of the gestation sac. The uterus was enlarged and soft. Right tube and ovary healthy and normal. Supra-vaginal hysterectomy was done. The patient died of shock two hours after the operation.

The interesting points in this case are:

1. The repetition of pregnancy in the stump of the left tube.
2. The viability of the foetus in the ligamentous pregnancy as evidenced by foetal heart.
3. The absence of urgent signs during the primary rupture.
4. The danger of extensive manipulation during the removal of the sac wall. It is not out of place here to say that "marsupialisation" of the gestation sac is advised.
5. The advisability of investigating thoroughly a case of uncomplicated pregnancy which gives a history of "brown discharges."

CASE II. INTRA-UTERINE PREGNANCY—RUPTURE OF THE CESARIAN SCAR—SECONDARY ABDOMINAL PREGNANCY

K. Hindu aged 22. VI para. admitted on 17th April 1928 with a history of pregnancy and bleeding per vagina since the previous day.

History of previous deliveries: The first four were natural, the fifth was a cesarian.

Examination on admission showed height of uterus about 8 months level; F. H. was not heard and uterus was not acting. There was slight tenderness over the lower abdomen and bleeding per vagina.

On 28th May 1928 she was again examined. Os was completely closed; F. H. not audible no uterine souffle was heard; there was a lump in the pouch of Douglas. The foetus was believed to be dead and the

patient was allowed to proceed to a normal labour. There was still bleeding per vagina.

On 14th June 1928. Temp. 104° P. 120. Slight bleeding per vagina.

On 16th June 1928. Temp. 102° - 104° there was offensive discharge per vagina.

She was operated on 19th June 1928. On opening the abdomen the foetus was found free in the abdominal cavity in an advanced degree of decomposition. The placenta was sloughing and adherent to omental tissue. This portion of the abdominal cavity was completely shut off from the rest of the cavity. There was no trace of the body of the uterus, only a small stump of the cervix was seen. The foetus was removed and also such portions of the tissue that easily came away on handling were removed. The vagina was opened into through the Douglas' pouch and a drain inserted. The abdominal wound was closed with through and through sutures and a drain was put in through this wound also.

The patient made a satisfactory recovery after a prolonged convalescence.

In conclusion, I have to thank Lt.-Col. Hingston, Superintendent, for permitting me to report these cases.

A CASE OF MYXO-SARCOMA OF THE BLADDER.

FROM S₁ WARD.

BY DR. C. S. SUNDARAM, M.B., B.S.

House Surgeon, General Hospital, S. I.

History.—Patient P—aged 60, was admitted on 3rd July 1930 with a history of constant dribbling of urine of two months duration. Patient said he had difficulty in passing urine and motion for three days two months back. Since then he had been having incontinence of urine.

Condition on admission.—On admission the bladder was found to be very distended and the patient was catheterized. Catheterization was painful. Foul smelling urine mixed with pus and blood was drawn. After catheterization a hard rounded tumour was felt in the suprapubic region. It was movable. The patient's general condition was good except that he was anæmic.

Rectal Examination.—Prostate was found to be enlarged, about the size of a big lime, middle lobe being very prominent. Prostate was very hard and slightly tender. It was not fixed.

INVESTIGATIONS.

URINE: No Sugar. No albumin. Few R.B.C. Pus cells and phosphates.

Urea Concentration Test.

	Quantity	Percentage of urea.
Before urea	25 c.c.	·4725.
One hour after	67 c.c.	·5925.
Two hours after.	88 cc.	·754.

Blood urea 39 mgms %.

Urine urea ·6174%.

Blood Count R.B.C. 2,580,000.

W.B.C. 6,250.

Haemoglobin 45%.

Wassermann Reaction:—Negative.

TREATMENT.

Operation on 9th July 1930 was done by S₁:—Suprapubic cystotomy. The mucous membrane of the bladder had a granular appearance. The whole of the bladder wall was thickened $\frac{1}{4}$ ". Looked like Scirrhus carcinoma. A section from bladder was taken. A Pesser's tube was left in the bladder through the suprapubic wound and continuous drainage established.

Pathological Report.—Myxo-sarcoma.

21st July 1930—Radium Insertion.

Six needles, three milligrams each. They were suspended inside the bladder through the suprapubic wound.

29th July 1930.—Needles were removed.

Total dose $6 \times 3 \times 24 \times 8 = 3456$ mgm. hours.

Curiously it was noticed that the patient had no irritability of the bladder. The suprapubic wound was still open and draining. A Pesser's tube was put in and the patient was discharged and advised to come and report every fortnight.

The interesting feature of this case is the rarity of a myxo-sarcoma arising from the bladder. A myxoma of the bladder itself is very rare. But a Sarcoma of the bladder though equally rare may arise from the bladder irrespective of age, both in young and old people. Then it takes the form of a pedunculated tumour growing into the bladder. But a myxo sarcoma of the bladder wall producing a diffuse thickening is about the rarest of tumours met with in the bladder.

I am indebted to Lt.-Col. E. W. C. Bradfield, I.M.S., First Surgeon, for kindly giving permission to publish this case.

Association Notes

(BY THE SECRETARY MR. G. M. THAMBIDURAI)

The association had the opportunity of welcoming the freshers. It is a pity that we are not in a mood to "Rag" the freshers before they could identify themselves as members of our fold. We can choose a day as our college Rag day and have a lot of fun for ourselves and get the Freshmen to introduce themselves to us. I make an earnest appeal to those public spirited members of our Association to come forward with practical suggestions, constructive criticisms and hearty co-operation to further the activities of the Association. The Secretary can only act as a key to open out new avenues of activities and as such expects to be used in the proper way. Our thanks are due to Captain McGrath for his unfailing interest in the welfare of the students. Only one who has been moving very closely with him can understand the great trouble he took to push forward matters favourable to students. He has actually expressed concretely the brotherhood and "no party" prejudice that exist in the hearts of the students of our College by knocking down the great partitions of the dining hall. We hear that he takes every opportunity to go to the Jail department to see how our marble topped tables and chairs are being constructed.

We are now having an Indian caterer and students find it a great convenience. We are grateful to our President, the Principal of our College, Lt.-Col. Hingston. He has been kind enough to inspect the rooms and sanction the necessary things to make the students comfortable. We hope to get few more things from him.

Elections. — Lt.-Col. Hingston, Principal of our College, continues to be the President of our College Association. Vice-Presidents are Dr. Lakshmanaswami Mudaliar and Lt.-Col. K. G. Pandalai. It is always a matter of joy to see these friends of students mingling with us. It is no surprise that they are re-elected to the places of honour. The Vice-President Lt.-Col. K. G. Pandalai in spite of his protests was compelled to accept the place.

Dr. Guruswami Mudaliar was elected as the President of the Magazine Committee in the place of Dr. Mannadi Nair, who will be

Association Notes

73

going to England within a short time. Dr. Mangesha Rao is one of the Associate Editors. Another is to be elected. Mr. J. E. Isaac David and Mr. G. P. Royan are student Editors. The representatives of all the classes are also elected. The Pre-registration students elected Mr. A. E. Shunker. The 1st year students elected Mr. Syed Ali. The 2nd year students elected Mr. K. Rama Rau. The third year students elected Mr. George Fletcher. The Fourth year students elected Mr. F. Martinus. The final year students unanimously elected Miss A. Y. Manickam as their class representative. It is perhaps the first time that the class elected a lady student as their representative. I may venture to say that this year might have many other events which might break the previous records of our Association. Miss A. Kolandavelu is to represent the lady students of our College. Mr. A. Saldhana represents the Military students. Dr. Shanmugasundaram represents the Health Officer's class. Mr. Ravi Varma 4th year student was elected as the Secretary for Sports. Mr. Purushotham Pai 4th year was elected as the Assistant Secretary to the Association.

Mr. N. Srinivasan and Miss Mahadevan continue as the Secretaries for the Dramatic club.

Obituary.—The association expressed its heartfelt sorrow on hearing the sad news of the demise of Mrs. Cheriyan by passing a resolution of sympathy. It was not long after this that the sad news reached us that Lt.-Col. Pandalai lost his mother. The Association passed a resolution of condolence.



"Roll of Honour."

The Madras Medical College Council has awarded the following Medals and Prizes for the session 1929-30:—

MEDALS.

- The Johnstone Gold Medal (Best outgoing student)
K. Narasimha Rao.
- The Blacklock Gold Medal (Clinical Medicine)
L. T. Lushington.
- The Chipperfield Gold Medal (Clinical Surgery)
K. Narasimha Rao.
- The Bharati Lakshmi Gold Medal (Midwifery)
Miss Mercy David
- The Raja Sir Ramasamy Mudaliar Gold Medal (Midwifery)
K. Narasimha Rao.
- The Savalai Sethuram Gold Medal (Diseases of Children)
K. Narasimha Rao.
- The Lady Grant Duff Gold Medal (outgoing Hindu woman student) Mrs. S. Janaki.
- The Bannerman Gold Medal (Bacteriology) P. C. Koshy.

PRIZES.

- The Maitland Memorial Prize (Clinical Medicine)
L. T. Lushington.
- The Branfoot Memorial Prize (Midwifery) V. Rangachari.
- The Sibthorpe First Prize (Surgery) C. T. Rangaswami.
- The Sibthorpe Second Prize (Surgery) Miss B. Mahadevan.
- The Sibthorpe Second Prize (Surgery) A. Seshagiri Rao.
- The Price Memorial Prize (1st in Medicine)
K. K. Chintan Nambiar.
- The Allan Ewan Grant Prize (2nd in Medicine)
Miss B. Mahadevan.
- The George Smith Prize (Pathology) P. Hari Vittal Rao.
- The Muhi-ud-din Sharif Memorial Prize M. A. Razak.

University of Madras. MEDICAL EXAMINATIONS, DECEMBER 1929.

PRE-REGISTRATION EXAMINATION.

SECOND CLASS.

Rank.	Reg. No.	Name of Candidate.	Rank.	Reg. No.	Name of Candidate.
1	110	Sarojini, R.	22	73	Narasimhan, M. G.
2	122	Srinivasan, S.	23	134	Thomas, K. E.
3	137	Valisvaran, S.	26	139	Van Ross, Paul.
4	72	Nagaswami S.	27	74	Narasimhan, R.
5	130	Susainathan, G.	7		Augustine Altarikattan.
6	103	Rangachari, P.	12		Beetles, B. N.
7	80	Narayana Prabhu, A.	15		Bindhusekhar Basu, T.
8	125	Subramanyam, M.	20		Cheriyar, Mary P.
9	116	Sitarama Rao	24		Desaradharma Reddi, P.
10	9	Balakrishnan, M.	28		Easwara Chandra Vidyasagar Kandula.
11	54	Krishnan Nair, P.	31		George, K. J.
12	102	Rangabrahmananda Rao, I.	48		Kasim Pillai, A.
13	97	Raman Nayar, M. K.	53		Krishnan Nair, K.
14	65	Madhavi Amma, P. T.	89		Pereira, H. J. A.
15	133	Thomas George.	90		Prakasam, R. N.
16	51	Koman Premela.	108		Saraswathi M. K.
17	78	Narayanan, G.	115		Sitaramayya, P.
18	151	Visvanathan, G.	127		Sundaram Pillai, V. O.
19	145	Venkatalakshamma, K.	138		Vangeysel, H. G.
20	126	Sundara Babu, B. V.	141		Varkkey, C. E.
	57	Lakshmikanthan, M.	144		Venkatacharya, P.
21	11	Basi Reddi, K.	154		Wagenmaker, Wilhelmina
	56	Kunju Krishnan, V. K.			

FIRST M.B. & B.S. EXAMINATION.

PARTS I & II.

SECOND CLASS.

234	Abraham, K. A.	
		NOT CLASSIFIED.
237	Chacko, K. C.	238 Damodaran, T.

PART I ONLY.

NOT CLASSIFIED.

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
156	Abraham, P. Z.	194	Mathai, P. M.
157	Achyuthan Pillai, C. A.	197	Narasimbachari, R. K.
158	Asirvatham, M.	200	Pancharathnam Kanaga mbai P.
159	Balakrishna Pillai, P.	201	Pandidurai, C. S.
160	Burby, G. E. J.	202	Parthasarathi, S.
161	Chacko, Lizamma	205	Pereira, J.
164	Dandayudhan, D. A.	207	Raghavachari, K.
168	George, P. V.	209	Rajaram, S. R.
169	Gopala Pillai, M.	210	Ramakrishna Upadhyaya, K.
173	Jagannathan, N.	211	Ramanuja Ayyengar, N.
176	Jegaroyan, M.	212	Rama Rao, K.
177	Muhammad Jehangir.	213	Rama Wariar, E. W.
179	Joseph, P. T.	214	Ramankutti Wariar, T. V.
181	Kasivisvanatha, P.	215	Rodricks, J.
182	Keshava Aiyer, K.	218	Seshappa Sheno, L.
183	Kesavana Nair, R.	219	Srinivasan, T. V.
186	Krishnaswami, A.	220	Subhadra Devi, I. J.
188	Kuruvilla, V.	221	Subramanya Sarma, A. V.
192	Mamen, Mary.		

FIRST M.B. & B.S. EXAMINATION—(Continued)

PART I ONLY—(Continued).

NOT CLASSIFIED—(Continued).

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
222	Syed Sibgatullah.	224	Vaidyanatha Iyer, A.
223	Titus, K. K.	226	Vasudeva Shenoi, V.

PART II ONLY.

SECOND CLASS.

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
255	Abdul Jameel.	339	Muthuswami, L. K.
257	Alexander Chandi.	343	Narayana Menon, K. N.
271	Bhaskara Menon, T.	344	Narayana Menon, M.
272	Bhushanam David.	345	Narayanan Nayar, K.
276	Dakshyani, M.	349	Pais, O. P.
278	David, C.	352	Ponnamma, K.
279	David, C. S.	352A	Pubalarayan, G.
281	Dhanapathi, Y.	353	Purushotham Pai, A.
284	Francis, M. M.	355	Rajagopal, K.
292	Govindan, V.	367	Rangayya Gupta, P.
297	Janakiammal, A. R.	370	Rukmoni, C. N.
299	Joseph, C.	372	Samuel, Mariam, P.
302	Kalyani Ammal, E. V.	375	Shanthasundari, D.
306	Kesavaraj, R. S.	376	Shiva Rao, B.
311	Koshi, P. C.	379	Sivasankara Pillai, K.
313	Krishna Menon, M. K.	382	Srinivasan, N.
314	Krishnamurthi, C. S.	383	Srinivasan, K.
315	Krishnamurthi, Rao, B.	384	Subraya Prabhu, M.
317	Krishna Rao, B.	385	Subramanya Ayyar, S. V.
320	Kumara Pillai, S.	386	Subramanyan, N. C.
325	Lashminarayanan, P. R.	387	Subramanyan, T. S.
328	Madhusudhanan S.	393	Trivikrama Pai, R.
331	Mariam, C. V.	396	Varghese, A. D.
333	Masilamoni, H. A. Premavathi	398	Vaz, O. G. C.
332	Mascarrhenas, F. P.	399	Velayuthan, P. K.
338	Muthukaruppa Pillai, S.	404	Venugopalachari, M.

NOT CLASSIFIED.

Reg. No.	Name of Candidate	Reg. No.	Name of Candidate
241	Gopalakrishna, U.†	319	Kumara Menon, M.
247	Kuruvilla, P. J.†	327	Madhavan Nayar, O.
254	Sripada Rao, M.†	334	Mathew, M. M.
260	Aravamudan, N.	346	Natesan, M. R.
263	Balakrishna Menon, P.	348	Padmanabha Pillai, S.
265	Balakrishnan, K.	350	Pereira, Daisy.
267	Balakrishna Pillai, P. R.	351	Philip, M. J.
269	Balasubramanya Pillai, S.	356	Rajagopalachari, K. S.
274	Chaturbhujdas, M.	362	Ramakrishna, C. M.
280	Devadason, N. P.	369	Razak, M. A.
288	Gonsalves, E. G. J.	371	Sambasivan, G.
289	Gopala Menon, A.	373	Sanjiva Rao, B.
291	Gnanavolivu, Jane M.	388	Subramanyan, V.
298	Jesudian, K. T.	394	Trivikraman, U.
305	Kesava Marar, P. V.	397	Vasudeva Rao, G.
308	Kolondavelu, Anne.		

* Yet to pass Part I

† Yet to pass in Pharmacology.

SECOND M.B. EXAMINATION.

Rank.	Reg. No.	Name of Candidate.	Rank.	Reg. No.	Name of Candidate.
1	447	Sampath Ayyangar, N. K.	435	Oommen, Sosa P.	
2	438	Raghava Rao, D.	439	Raghavendracharya, P.	
	411	Cartner, Dorothy, E. L.	441	Raman Pillai, K.	
	413	Chokkalingam, P. M.	445	Robert, G. W.	
	414	Cotelingam, J D.	450	Savarirayan, J. C.	
	416	David Mercy	453	Siva Ayyar, S. V.	
	421	Hart Floerence, M.	459	Sundaram, V. K.	
	423	Jacob, M. K.	458	Sundaram, T. P.	
	424	Janaki, S.	455	Sriramulu, G.	
	425	Johnson, A. S.	460	Sundaram Ayyar, R.	
	426	Kalamegham, K.	462	Vaidyanathan P.	
	428	Kumara Pillai, V.			

The following candidates appeared for the Second M. B. Examination, but qualified for the Third L. M. & S. Examination.

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
409	Balasubrahmanyam, V. *	431	Muhammad Safi-ul-lah, †
410	Bower, Enid, M. †	434	Tharaken, P. §
412	Chacko, T. N. *	440	Rajagopalan, T. R. §
417	DeLemos, J. B. C. †	461	Sundararajan, M. S. §
427	Krishnaswami, V. †		

* Obtained exemption for L. M. & S. in Ophthalmology

† Obtained exemption for M. B. in Hygiene.

‡ Obtained exemption for M. B. in Ophthalmology.

§ Obtained exemption for M. B. in Pathology.

FINAL M.B. & B.S. DEGREE EXAMINATION.

PART I.

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
471	Chacko, Anna W.	494.	Srinivasan, E. V.
488	Rangachari, V.		

PART II.

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
506	Bhimesvara Rao, K.	563	Puvayya Accamma, C.
510	Curairaj, G.	567	Rajaratnam, J. N. Appeared for Obstetrics and Gynaecology, and passed (He has yet to pass in Medicine and Surgery).
514	Gnanavolivu, J. K.	575	Rangachari, T. E.
515	Ganesvaran, S.	579	Sambasiva Rao, M.
518	Govindankutti Menon, I.	581	Sankaran, N. S.
520	Govinda Rao, P.	601	Verghese, Anna K.
526	Jacob, K. C.		
530	Joseph, P. M.		
531	Joseph, T. J.		
539	Krishnamurthi Rao, S.		
553	Narayanaswami, B.		

The following candidates appeared for Part II of the Final M. B. & B. S. Degree Examination but qualified for the L. M. & S. Degree:—

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
500	Alankaram, S. †	551	Muhammad Abdul Hadi
508	Chockalingam, R. V. *†	552	Narayana Nayar, V. R. *
517	Govinda Menon, K. †	557	Natesan, P. * †
525	Itty, Sarah P. *	562	Philip Sosa, C. §
542	Lakshmanan R. ††	580	Sankaran, C. †
550	Mathew, M. I.	599	Venkataramam, M. P.

* Obtained exemption for M. B. in Medicine.

† Obtained exemption for M. B. in Surgery.

‡ Obtained exemption for M. B. in Obstetrics and Gynaecology.

§ Qualified for the L. M. & S. Degree by combination of results.

SECOND L. M. & S. EXAMINATION.

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
264	Balakrishna Marar, P. V. *	326	Madhavan, K. R. *

* Obtained exemption for M. B. in Pharmacology.

THIRD L. M. & S. EXAMINATION.

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
412A	Chathu Achan, V. N. §	443	Ramaswami, K. S. †
415	Devasikhamani Mudaliar S. *	451	Satur, Josephine, F. †
418	Dharmalingam, C. †	452	Sankara Rao, A. †
419	Ganapathi, K. V. †	454	Srinivasulu Reddi, N. *
420	Gangadharan, N.	456	Subramanya Iyer, V. *
422	Jacob, C.	457	Sndarsanam, G. C. †
429	Lushington, L. T. †	463	Varughese, P. K.
430	Madhava Menon, P.	465	Venkatasubrahmanyam, V. †
436	Paramanandam, Edward.		
437	Peter, J. G.		

* Obtained exemption for M. B. in all subjects.
 † Obtained exemption for M. B. in Hygiene.
 † Obtained exemption for M. B. in Pathology.
 § Obtained exemption for M. B. in Ophthalmology.

FINAL L. M. & S. DEGREE EXAMINATION.

PART I.

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
467	Achyutha Menon, P. *	478	Martius, Shelga, A. *
468	Achyuthan Nair, M. *	482	Padmanabhan Nambiar, K. T. *
472	Chathu Achan, V. N. *		
474	Dharmarajan, C. K. *	484	Rajagopalan, S. *
477	Kuppuswami, M. *	495	Subramanyam, M. K.

* Appeared only for Forensic Medicine and obtained exemption for M. B.

FINAL L. M. & S. DEGREE EXAMINATION.

PART II.

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
507	Chandra Rao, K. †	578	Ratnavelu, M.
519	Govindarajulu, V.	591	Sundarayya Chetty, V. §
537	Kondalarayudu, K. †	598	Venkataramanayya, G. †
558	Nemraja Alva, K.	602	Verghese, Rachel, M.
566	Rajamannar Nayudu, K. †	603	Virayya, P. †

* Obtained exemption for M. B. in Medicine.
 † Obtained exemption for M. B. in Surgery.
 † Obtained exemption for M. B. in Obstetrics and Gynaecology.
 § Qualified for the L. M. & S. Degree by combination of results.

B. S. Sc. DEGREE EXAMINATION.

PART II.

608 Nityanandam, P. G.

MEDICAL EXAMINATIONS, JUNE 1930.

The following is the list of successful candidates at the recent Medical Examinations held in June 1930.

Medical College, Madras.

PRE-REGISTRATION EXAMINATION,

SECOND CLASS.

Rank.	Reg. No.	Name of Candidate.	Rank	Reg. No.	Name of Candidate.
1	88	Vaidyalingam, K. V.	37	Kitto, S. R. E.	
2	50	Muhammad Ismail. M.	39	Krishna Prasad, R.	
3	75	Santanam, N.	41	Lalitha, N.	
4	87	Syed Ali Ahmed,	43	Luke, V.	
5	14	Chandrasekhara G. V. Ayyar	47	Manikya Rajayya, A. P.	
6	36	Karunakaran Nayar, K.	51	Muktha Bai, B.	
...	2	Abraham, K. J.	52	Muniswami Chetty, M.	
...	3	Achyutan, T.	54	Narasimha Prabhu, G.	
...	4	Achyuta Panikkar, M.	56	Narayanan Kuttii, A.	
...	7	Avndiappan, T. S.	57	Nayanar, Kiruda Bai	
...	9	Beetles, E. A.	58	Padmavati, K. K.	
...	10	Buchinarasimham, M.	59	Paranjothy, Prakashid.	
...	11	Chacko, P. T.	60	Paul, M. A.	
...	12	Chakrapani Menon, M.	61	Paulose, P. P.	
...	15	Colledge, Merlyn	63	Radhakrishna Reddi, G.	
...	16	Damodara Mallayya, B.	64	Raghavachari, G.	
...	17	Damodara Taravanar, T.	72	Rudrani Amma, G.	
...	19	Dwaraka Bai, G. S.	73	Rukmani, C. S.	
...	22	Garson, R. A.	79	Sivarama Ayyar, N.	
...	25	Goda Varma Raja, P. K	80	Sivarama Mannadiyar, E.	
...	29	Isvaran, N	82	Srikumara Kurup, T. N.	
...	30	Jagadisan, A. N	83	Srinivasan, O. V.	
...	32	Janardhanan, T.	85	Subramanya Chetti, M.	
...	34	Joseph, C.	89	Vaidyanathan, K. G.	
...	35	Kantappa Hegade, K.	92	Viraraghavan, K. D.	
			93	Visvanathan, V. N.	

FIRST M.B. & B.S. EXAMINATION.

(Old Regulation.)

SECOND CLASS

Register number and name of candidate,

13, Chandra Rao, K.

27 Govindarajulu, V.

FIRST M.B. & B.S. EXAMINATION.

(New Regulation.)

PARTS I AND II.

SECOND CLASS.

Register number and name of candidate.

174 Kondalarayadu, K.

NOT CLASSIFIED.

170 Govindankutti Menon, P. *

176 Parukutti Amma, K. *

* Yet to pass Pharmacology.

PART I.

SECOND CLASS.

Register number and candidate

153 Sripada Rao, M.

FIRST M.B. & B.S. EXAMINATION—(Continued).

PART I—(Continued).

NOT CLASSIFIED.

Register number and name of candidate.	
102 Cheriyan Mary, T.	130 Manjappa, H.
103 Chinnamm, V.	131 Mathew, Achy.
105 Daniel, P. H.	134 Narasinga Rao, B.
108 Ganapatisundaram, S.	139 Pereira, F.
109 Gopala Menon, K.	140 Porinchu, A. A.
110 Gopala Pillai, P. P.	141 Rajam, K.
111 Govindan S.	145 Sankara Ayyar, G.
112 Govindan Kutti Nayar, A.	146 Sankaran Nayar, K. N.
114 Janardhanan Poti, A.	148 Shankara Adyanthaya, N.
117 Koshi, C.	151 Siva Rao, C. J.
119 Krishnamuti, V.	154 Thomas, M. A.
120 Krishnamurti Rao, P. S.	155 Thomas, P. G.
122 Krishnan Unni, T.	156 Vaidyanathan, S. R.
123 Kunniraman, K.	161 Vedachalam, V. R.
125 Kurien, P. V.	162 Venkatesvara Mallaya
126 Kuruvilla, P. J. *	

PART II.

SECOND CLASS.

Register number and name of candidate.	
185 Aravamudan, N.	221 Kolappa Pillai, N.
188 Balakrishna Menon, P.	226 Kumara Menon, M.
191 Balakrishna Pillai, P. R.	228 Madhavan Nayar, C.
193 Balasubrahmanya Pillai, S.	233 Mohammad Muntaz Ali.
196 Chacko, K. C.	238 Narayana Ayyar, K. V.
197 Chaturbhujdas, M.	239 Natesan, M. R.
198 Christodas Williams, J.	240 Padmanabha Pillai, S.
199 Damodaran, T.	241 Pereira, Daisy.
200 Devadason, N. P.	242 Philip, M. J.
206 Gnanavoliam, Jane M.	250 Kazak, M. A.
207 Gonsalves, E. G. J.	251 Sambasivan, G.
208 Gopala Menon, A.	252 Sanjiva Rao, B.
213 Jesudian, K. T.	253 Srikanthvaran, U. V.
214 Joseph Selvam.	262 Vasudeva Rao, G.
220 Kolandavelu, Anne.	

NOT CLASSIFIED.

Register number and name of candidate.

191 Balamani, M. *	222 Koshi, K. C. *
205 George Fletcher, S. *	223 Krishna Ayyangar, V. *
209 Gopalan Nayar, P. T. *	243 Raghava Ayyangar, S. A. *
210 Govindarajan, S. *	256 Sivasankaran, V. *
215 Jubai, M. *	

* Yet to pass Pharmacology.

SECOND M. B. & B. S. EXAMINATION.

SECOND CLASS.

Rank	Reg. No.	Name of Candidate.	Rank	Reg. No.	Name of Candidate.
1	315	Srinivasan, P. R.	5	272	Chintan Nambiyar, K. K.
2	282	D'Souza, H. C.		335	Vedavanam, D. C.
3	308	Ramalingam, E.			Ranj.
4	281	D'Silva, C. B.			

SECOND M.B. & B.S. EXAMINATION—(Continued).

SECOND CLASS—(Continued).

Rank.	Reg. No.	Name of Candidate.	Rank.	Reg. No.	Name of Candidate.
7	305	Raju Shetti, Y.	16	319	Subbaraya Prabhu, M.
8	309	Rangaswami, C. T.	18	321	Sundaram, T. K.
9	332	Velayuthan Nayar, P.	19	293	Kuriyan, G.
10	274	Balakrishna Shenoi, K.	20	289	Jacob, J.
11	313	Seshagiri Rao, A.	21	298	Manikkam, Alice Y.
12	277	Chandrasekharan, N.	23	326	Thomas, A.
	287	Harivital Rao, P.		284	George A. T.
	297	Mahadevan, Bharathi.		283	Eapen, J.
15	286	Gopalaram, A. K.	24	276	Bhaskar Rao, R.
16	318	Subbalakshmi, S.		320	Subramanyan, N.

FINAL M. B. & B. S. DEGREE EXAMINATION.

PART I

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
312	Sampath Ayyangar, N. K.	352	Raman Pillai, K.
347	Cotelingam, J. D.	353	Robert, G. W.
349	Hart, Florence M.	356	Sundaram, V. K.

The following candidates are eligible for the M.B. and B.S. Degree:—

SECOND CLASS.

Rank.	Reg. No.	Name of Candidate.	Rank.	Reg. No.	Name of Candidate.
1	505	Vasudevan Nambissan, T.	13	397	Janaki, S.
2	381	Duraiwami Ayyar, R.	...	358	Abraham, K. A.
	363	Anantanarayana Ayyar, P.		361	Aiyaswami, T. V.
4	430	Narasimha Rao, K.	...	362	Alankaram, S.
	499	Sundaram Ayyar, R.		364	Arul, E. J. A.
5	445	Parthasarathi, G.	...	365	Arumugam Pillai, K.
6	453	Raghavendracharya, P.	...	374	Chokkalingam, R. V.
7	421	Mahadevan, M. N.	...	387	Govinda Menon, K.
8	426	Medappa, T. B.	...	393	Ithy, Sarah P.
9	444	Padmanabha Panikkar, T. G.	...	398	Johnson, A. S.
10			...	401	Kameswara Rao, D.
11	351	Oommen Sosa, P.	...	415	Lakshmanan, R.
12	371	Cartner, Dorothy E. L.	...	449	Philip Sosa C.
			...	473	Sankaran, C.
			...	481	Savarirayan, J. C.

The following candidates appeared for the Final M.B. & B.S. Degree Examination but qualified for the L.M. & S. Degree:—

Reg. No.	Name of Candidate.	Reg. No.	Name of Candidate.
372	Chacko, Anna W.	459	Ramachandra Kamath, V.
404	Kesava Menon, C.	468	Rangachari, V.
411	Kumara Pillai, V.	472	Sambasivan, K.
416	Lakshminarayana, T.	474	Sankaranarayanan Unnithan, S.
425	Mathew, M. I.	484	Siva Ayyar, S. V.
435	Narayanaswami Ayyar, A.	489	Srinivasan, E. C.
439	Natesan, P.	510	Venkatarangam, M. P.
442	Padma, A. S.		

SECOND L. M. & S. EXAMINATION.

* † 217 Kannankutti Meron, E. 257 Soolapani Wariyar, N.

* Obtained M.B. marks in Anatomy.

† Yet to pass Pharmacology.

THIRD L. M. & S. EXAMINATION.

Register number and name of candidate.

Reg. No.	Name of candidate.	Reg. No.	Name of candidate.
268	Achyuta Menon, P.	§ 323	Syed Abdul Ghani.
269	Alagasigari Nayudu, P.	324	Thambidurai, G. M.
§ 273	Balakrishnan Nayar, M. K. R.	§ 327	Thomas, T. C.
288	Isaac J. E. David.	328	Thomas, T. S.
296	Madhusudanan, G.	330	Vaidyanathan, J.
303	Pakiam, J.	§ 333	Venkatasubba Rao, K.
§ 307	Rama Rao, S.	§ 336	Zachariah, V. O.

§ Obtains M.B. marks.

FINAL L.M. & S. DEGREE EXAMINATION.

PART I.

Reg. No.	Name of candidate.	Reg. No.	Name of candidate.
§ 337	Ganapati, K. V.	348	Dharmalingam, C.
338	Gangadharan, N.	350	Jacob, C.
§ 340	Paramanandam, Edward.	354	Satur, Josephine F.
341	Ramaswami, K. S.	355	Sundarsanam, G. C.
344	Balasubrahmanyam, V.	357	Varughese, P. K.

§ Obtains M.B. marks.

WHOLE EXAMINATION.

The following candidates qualify for the Final L.M. & S. Degree :—

Register number and name of candidate.

§ 345 Bower, Enid M.

§ Obtains M.B. marks.

PART II.

The following candidates qualify for the final L.M. & S. Degree :—

Register number and name of candidate.

* 359	Achyuta Menon, P.	† • 380	Dharmarajan, C. K.
† § 360	Achyuta Nayar, M.	• 383	Ganapati, C. K.
§ 366	Azariah, E. P.	• 385	George, M. C.
† 367	Balakrishna Menon, K.	390	Hanumanta Rao, S.
• 368	Balaraman, D.	• 396	Jagannatharaju, M.
§ 369	Booth, A. H.	† 403	Kanniyappan, K. G.
§ 376	Damodaran Nambiyar, N.	† • 407	Krishnaswami, N.
† • 378	Daivasikhamani Mudaliyar, S.	† † 412	Kuppuswami, M.
• † 379	De Lemos, J. B. C.		

§ Obtains M.B. marks.

† Obtains M. B. marks in Medicine.

• Obtains M.B. marks in Surgery.

‡ Obtains M.B. marks in Obstetrics and Gynaecology.

WHOLE EXAMINATION—(Continued).

PART II—(Continued).

Register number and name of candidate.

§ 417	Lushington, L. T.	• 469	Ranganathan, A.
420	Madhavan Nayar, K.	† 470	Rangathan, T. K.
• 423	Martandam Pillai, P.	• 475	Sankarama Ayyar, R.
• † 436	Narayanawami, G. R.	• 479	Satyanarayanamurti, T.
440	Nilakantan Nambiyar, M. P.	• 483	Shinappa Bandari, K.
† • 443	Padmanabhan Nambiyar, K. T.	• 490	Srinivasalu Reddi, N.
• 450	Radhakrishnan, C.	§ 491	Subbaraman, N.
• 452	Raghavan, R.	§ 492	Subrahmanya Ayyar, K.
† † 455	Raghunathan, R.	† † 493	Subrahmanya Ayyar, V.
463	Ramayya, B. J.	† † 494	Subrahmanyam, M. K.

§ Obtains M. B. marks.

† Obtains M. B. marks in Medicine.

• Obtains M. B. marks in Surgery.

‡ Obtains M. B. marks in Obstetrics and Gynaecology.

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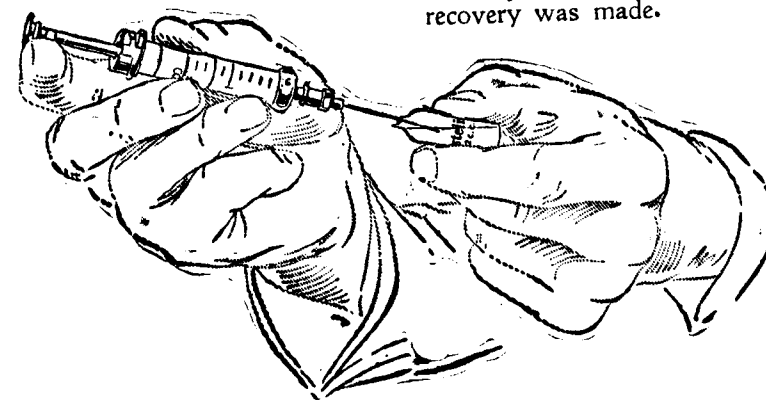
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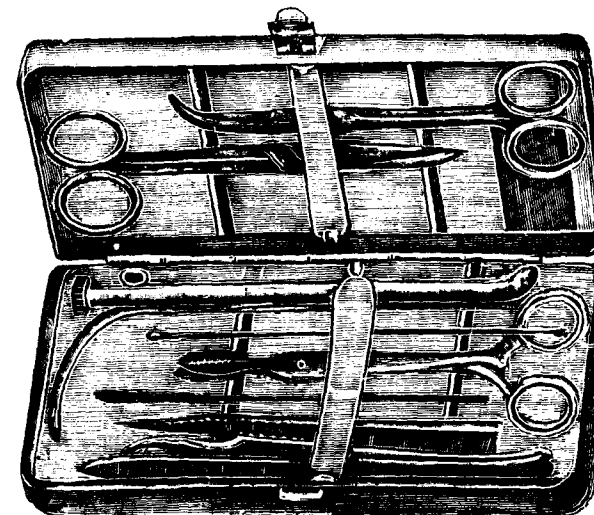
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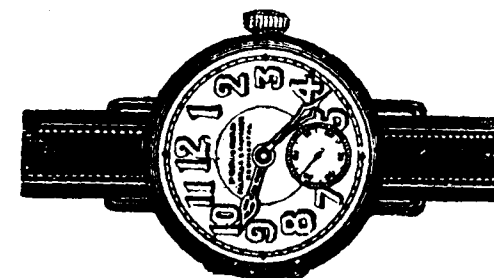
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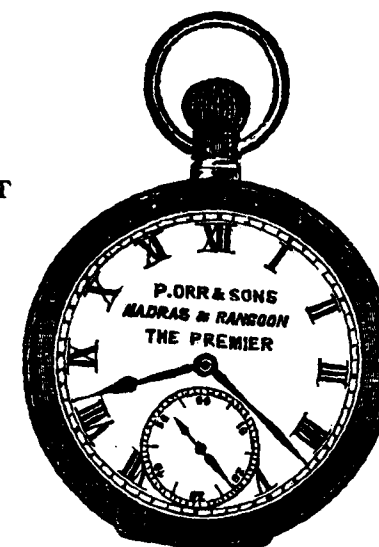
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