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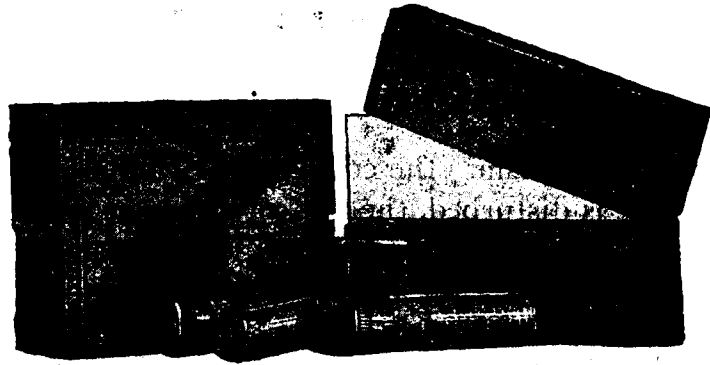
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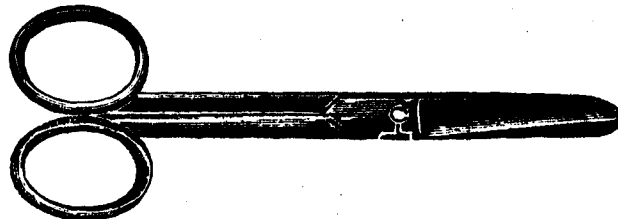
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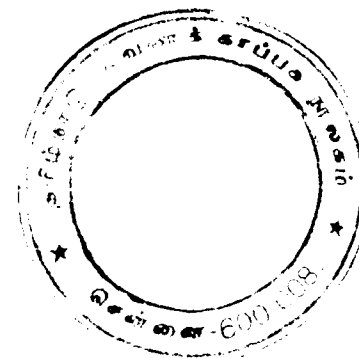
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The Madras Medical College Magazine

EDITOR

Dr. M. R. Guruswami Mudaliar, B.A., M.D. C.M.



Vol. VI. No. 4.

March, 1927.

Nothing is more estimable than a physician who, having studied Nature, knows the properties of the human body, the diseases which assail it, the remedies which benefit it, exercises his art with caution, and pays equal attention to the rich and the poor.—Voltaire.

“Sail - don't drift. Choose the port that you wish to make, then set your course and hold it. There may be contrary winds and storms that will set your sails aback, and drive you far to the leeward ; but Will is a compass and Determination is a helm that will set you right again and bring you at last to the port of your ambition.”

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THE MADRAS
Medical College Magazine

Vol. VI. MARCH No. 4.

A New Year's Greeting to Our
Students

BY LT. COL. A. S. LESLIE, M. D., I. M. S.

Do not chide me, gentle reader—put a little of the blame—put a lot of it on that tall handsome student who slid a copy of the December number of the “Madras Medical College Magazine” into my hand: murmured “something for this month’s issue, please,” and was gone before I could say him “nay!”

I have to inflict myself on you every working day of the week—shame on it that I should also intrude on your leisure!

For, your magazine is to your work, what coffee and liqueurs are to a full meal—something to aid digestion, to promote the feeling of *bien être*, and to banish the cares of the day.

So, I will not discuss a medical topic—does not Dr. Treatemwell, aided by a hundred and one fellow con-

spirators, intrigue you, in repeated editions of ponderous tomes, to learn all that medical science can teach—and do not he and his fellow conspirators go into each subject much more deeply than I can ever hope to do?

And besides: you know already what of medicine I have not forgotten: you know all my pet heresies: how I distrust all stimulant in treatment, but look on other liquids with even more suspicion: how I.....

But no! we will not pursue professional topics. How am I to appease the ingratiating villain who would not wait to hear my "nay"? Show you what wiles can be successfully tried on your future examiners?

Perish the thought! I would have every colleague up in arms against me if I gave away *these* secrets!

Why not let us give a few minutes consideration to the attitude that the medical student ought to adopt towards his teachers and his profession. At once my mind goes back to an old gray college with its weather beaten crown, in the cold keen north.

Over three centuries ago the pious founders built it and over the entrance gateway are engraved the words—

"They say! What say they? Let them say!"

Could the medical student's progress and outlook be more pithily described?

They say: With bowed reverent head he begins his studies and drinks in the knowledge culled from all quarters by his teachers.

What say they? He has reached the stage of criticism. He not only listens: he analyses: he weighs the pros and cons of the doctrines laid before him.

Let them say! He has reached the final stage. He has learned: he has observed for himself: He has reached conclusions of his own. His teacher

and their doctrines are not infallible now, as they appeared to be at an earlier stage; they have brought him knowledge: they have broadened his outlook: they have given him insight: they have taught him to think for himself. They have been good: some of them have been more than good, but further progress, he feels, must now come from himself.

"Let them say!" He still listens—listens with respect—but he now thinks for himself: he satisfies himself as to the genuineness of the premises: he sifts the evidence for and against. He draws his own conclusions: his own individuality comes into play: he is no longer the mere recipient of ideas: he observes: he analyses; he differentiates: his inferences are not mere repetitions of his teachers' words: they are the result of his own individual way of thinking and reasoning: he has taken the step that counts: he is trying to reach the ultimate truth by his own efforts.

And so, gentle reader, let my new year wish for you be that you have reached that stage: let my advice be to you "Think for yourself".

You have absorbed the knowledge of the text-books: you have been helped by the individualities of your teachers: they have given their best to help you along the road to Truth: they have helped you to reach what a short time ago appeared to you to be the veriest Olympian heights.

But there are heights above the heights: and it is by your own efforts that you can scale those heights.

The Romance of the Future lies in your own hands. Never was there greater need for individuality in medicine than at the present day.

Present day medicine teems with half-truths. We know an enormous amount about many of the ills that

flesh is heir to : we do not know the whole truth about a single one of them.

It is the ultimate truth about every disease, that we are in quest of: and we, your teachers, look to you to carry on the struggle towards Truth till the end has been attained. "They say! What say they? Let them say!" May a future generation add to the pithy old motto and say of you and your generation—

"They have said!"

Non-Specific Protein Therapy*

BY S. RAMAKRISHNAN, L. R. C. P. & S., D. T. M. & H.

When one thinks of talking on the use of Vaccines and sera, it is no longer necessary to begin with a defence of such methods of treatment. Those materials have become instruments of the recognised systems of treatment of many diseases, and practitioners have no greater hesitation in using antitoxin in diphtheria than they have in using arsenic or mercury in syphilis—perhaps less.

Again for several years past every student has learnt the principles of immunity and the common therapeutic attempts to produce it. There is no longer therefore any need to trace the gradual development of this science.

Immunity however is a term very wide in its application. Indeed it has become exceedingly complex and involves a considerable amount of expert knowledge of colloidal chemistry for a thorough comprehension of its entire field of application, so much of it is without practical application at the present moment in the plain matter of the treatment of diseases, that any attempt to compress it into proportionate space would result in creating confusion. The fundamental facts upon which rest all treatment by artificial immunisation are these:—

Disease may be caused by infection with microscopic organisms.

Natural recovery from such infections often occurs.

The natural means of recovery are capable of artificial reinforcement.

One great limitation to this means of treatment

* A paper read before the Vellore Medical Association.

should however be constantly borne in mind.

Broadly speaking pathogenic micro-organisms are divisible into two great classes:—Protozoa and Bacteria. Protozoa are incapable or are extremely difficult of cultivation and consequently cannot be obtained in the necessary amount and state of purity which are required before they can be used artificially in the immunising process. Bacteria are readily capable of cultivation so that both their bodies and in some cases their products can be so obtained in the pure state. Protozoa on the other hand can sometimes be abolished by the use of specific drugs such as mercury in syphilis, quinine in malaria or emetine in amoebic dysentery. But drugs are next to useless in the treatment of bacterial infections. Ten or twelve years ago this statement was literally true. Ten years hence it may not be true at all. At present it is practically true. Vaccine and sera are only available against bacterial infections. It has been stated that practical immunisation requires the use of bacteria or their products in what may be called the 'pure' state and in sufficient quantity. Both these substances are of immunising value because they contain proteins which are foreign to the animal body and which will consequently excite as it were some kind of antagonistic response when introduced into the animal tissues. Bacteria and their products are probably the most harmful of such foreign proteins; bacteria have the added danger that they are capable of multiplying; but it is easy to understand that other foreign proteins besides bacteria may under some circumstances gain entrance to the tissues and produce toxic results and that method similar to those used against bacterial infection may be of use both for diagnosis and treatment of such toxic conditions.

Bacterial toxin when injected into an animal induces the appearance in the animal's blood of a substance which will combine with the toxin and render it inert—an anti-toxin. Very numerous experiments on similar lines were undertaken and it was found that if ricin which agglutinates and haemolyses blood corpuscles, was injected into animals in suitable doses, a serum was obtained which when added to ricin destroyed its power. Snake venom stimulates the formation of a substance which combines with it. Blood corpuscles of an animal of one species if injected into one of another species, produce bodies which haemolyse the corpuscles of the species injected and no others. By similar means bodies which coagulate milk and which arrest the movements of spermatazoa may be produced. Thus either animal or vegetable foreign proteins whether bacterial toxin, secretions like milk or venom, formed bodies, like R. B. C. or spermatazoa, will stimulate the production by the tissues of specific anti-bodies.

Three orders of anti-bodies are recognised according to the nature of the foreign protein used for inoculation, and the sera of immunised animals will give reactions whose characters are sufficiently indicated by their different names.

The first class are anti-toxins. The immune serum will neutralise a toxin and render it harmless without producing any visible change in the mixture of the two fluids.

The second order includes precipitins and agglutinins. Their reactions are recognisable in vitro.

In the third order we place lysins which produces reactions of which haemolysis is the best known and most easily seen.

It may now be stated as a physiological law that the body tissues have the power of elaborating anti-bodies to any foreign albuminous substance.

It must be definitely remembered that because anti-bodies are formed, it does not necessarily follow that they can be demonstrated in vitro, but in certain cases antibacterial bodies are definitely demonstrable, both after experimental inoculation and after spontaneous recovery from bacterial diseases, which will agglutinate, kill, dissolve or otherwise disintegrate bacteria.

This is the foundation of the modern view of immunity in infectious disease, viz., that it results from the production of anti-bodies to the invading bacteria so that recovery results from immunity and not immunity from recovery. Immunity can be confirmed by inoculation with the appropriate vaccine apart from infection.

These then are the data which have been available as a result of thirty years research work.

Animal tissues can produce anti-bodies to foreign albuminous substance in general and to bacteria in particular. The anti-bodies so formed were long supposed to be absolutely specific.

In many instances these anti-bodies can be demonstrated in the serum. The animal is then in a state of active immunity.

When anti-bacterial substances are demonstrable in serum, the fact may be used in the diagnosis of bacterial infection.

Bacterial substances may be inoculated into patients to confer active immunity either before exposure to infection for prophylactic purposes, or in the course of infection for curative purposes.

Bacterial suspensions or bacterial products may be injected into animals to produce in them active immunity. Their serum when charged with anti-bodies may be drawn off and injected into patients in the course of infection,

thus neutralising toxins or destroying bacteria and conferring on the patients, passive immunity.

It must be stated that anti-bodies are hypothetical substances. It is not the case that an immune serum contains any special chemical substance capable of analysis, the anti-bodies cannot be crystallised out or presented as the salt of any acid. An immune serum will produce certain demonstrable results on the foreign protein to which it is due and it is convenient to describe it as containing anti-bodies. The substances with which anti-body formation may be induced are collectively spoken of as 'Antigens.'

The Conception of Antigen

Antigens are all substances which when injected into an animal body induce specific anti-body formation. They form a large group in nature and are chemically identical with proteins, indeed we may say that all known proteins may act as Antigens. We may divide antigenic substances into two main classes. One of these comprises all those substances of bacterial, animal or vegetable origin which when injected into the animal body give rise to specific neutralising or antitoxic properties in the blood of the injected animal. These are bacterial exotoxins, the snake venom, powerful vegetable poisons and proteolytic and other enzymes of animals and plants. They are substances which are powerfully active—some of them strongly toxic to the living animal, others true enzymes or ferments. The number of such substances known is limited. The reaction they call forth in the animal body appears to be aimed directly at the specific neutralisation of their respective activities and is so unique and different from that induced by other antigens.

The other class of antigens comprises all proteins

which are inactive, showing in themselves neither toxic nor enzyme-like properties. Introduced into the animal bodies, they call forth a response of a nature entirely unlike that of the anti-toxins, and which so far as we can fathom appears to be directed merely at the assimilation or the removal of the infected substance. (For the cells of the animal cannot utilise the foreign protein as such and thus it is only the foreign proteins injected into an animal that acts antigenically, and no anti-bodies are formed when homologous material is injected). This large group composed of all formed and unformed substances in nature in which a protein structure is involved does not induce the formation of anything like the neutralising anti-toxins spoken of above. The anti-body appearing in animals treated with such substances have been spoken of as cytolytins or cytotoxin and in the case of formed antigens like bacteria—agglutinins or opsonins. We must not forget however that the observation of anti-bodies in the circulating blood is but one of the changes that have taken place in the treated animal. Much has been made of this phase of the problem because serum anti-bodies are readily studied *in vitro*, but their origin of course must be sought for in the body cell in which the original and most profound changes must necessarily have taken place during such treatment, changes the nature of which are to a larger extent still a mystery but on which ultimately depend the important physiological difference between treated and untreated animals.

Thus the trend of modern therapeutic thought has been strongly in the direction of seeking specific substances for the relief and cure of specific maladies. The idea of specificity in other words has dominated such attempts almost exclusively. In spite of this, however there has

gradually grown in the minds of bacteriologists an impression that not all the effects of the injection of bacterial protein were purely specific. Sir A. Wright who has done more than any other immunologist to emphasise the factor of strict specificity in vaccine therapy records his present attitude thus:—"I confess to having shared the conviction that immunisation 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 7 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, we had to take up into our categories the fact that inoculation produces in addition to 'direct' also 'collateral' immunisation". It is because of unexpected benefits derived in this way from injection of solutions of foreign proteins given for other purposes and not specific for the conditions benefitted, that the whole subject has received a very wide amount of attention, and the deliberate use of foreign proteins given by injections for therapeutic purposes is extending and seems likely to afford an increasing field of usefulness in the direct treatment of various kinds of patients and of diverse maladies. It is

probable that every foreign protein when absorbed into the body from the alimentary canal or by subcutaneous or intravenous injection causes one or other of the tissues of the body to produce in retaliation, as it were, certain chemical substances differing in different cases but having this in common, that they would not have been produced if there had not been this injection of foreign protein from without. Even if this is not true of all foreign proteins, it is certainly true of some and a familiar example is the reaction shown more by some individuals than by others after a subcutaneous injection of serum.

The injection of a foreign serum occasionally produces symptoms of sufficient severity to constitute a positive serum disease. It is a matter of indifference whether the serum is normal or immune, the symptoms being due to the foreign proteins introduced and in no sense an anti-bacterial reaction. Three different classes of persons are found as regards reaction to the injection of serum. The first class is unaffected. The second is sensitive enough to give what is called a normal reaction. The third is hypersensitive and give an extraordinary and severe reaction. In general, the larger the dose of serum given, the greater is the risk of serum sickness. The commonest complaint is a rash which is most often urticarial, the lesions being sometimes pale, sometimes surrounded by a red zone. The rash starts in the neighbourhood of the injection and spreads from there, finally becoming generalised and on the whole symmetrical. It is best marked on the extremities especially their extensor aspects. The rashes are highly irritating. The duration is very variable—generally they last three or four days, but may recur. The temperature is often raised, may last for some time, a few days up to even a fortnight.

There is often sweating. The lymph glands draining the site of injection may be enlarged and tender. Slight degrees of oedema occur and may be observed in the face, scrotum and hands. Albuminuria is rare and only slight when present. Joint pains and stiffness occur in a small proportion of cases. The joints are slightly red and swollen but do not suppurate. The joint pains generally accompany the rash and last two or three days; they rarely recur. When it is remembered that individuals vary extremely in their sensitiveness to the serum reaction, that the sera of different horses vary much in their toxicity and that the symptoms vary with the amount of serum injected, it will be realised that serum disease is not a definite clinical entity and is liable to extreme variations in character, degree and duration. This condition of serum disease is generally described as *anaphylactic* and the phenomena described above have long been realised as a non-specific reaction due to the proteins in the serum and not to its anti-toxin contents. Thus the modern conception of protein shock and its application in non-specific protein therapy are stated to have arisen from the 'clinic' and not as the result of any scientific reasoning. It was observed in practice that remarkable results were obtained in diseases having no specific relationship to the remedies employed.

In the practice of non-specific therapy a large number of therapeutic agents have been used.

1. Counter irritants—cautery—blister.
2. Blood and serum—normal and immune.
3. Proteins—egg albumin—milk—gelatin.
4. Enzymes.
5. Vaccines.
6. Bacterial extracts.
7. Colloidal Metals.

Milk.—The intra-muscular injection of milk was introduced by Saxl in 1916. Milk offered the advantage that it would be easily available by the profession. The method is very simple. Ordinary fresh milk is boiled from 5 to 10 minutes and when cooled 5 to 10 c.c. are injected intragluteally or into other muscles—arm, etc. Probably, the most commonly employed non-specific agents are typhoid and colon vaccines, cow's milk, horse serum and various split proteins as Witte's peptone.

The Non-Specific Reaction

The intravenous injection of bacterial suspensions, of colloidal metals, of various serums, the intra-muscular injections of milk are all followed by a reaction that varies from a mere stimulation of leucocytes, to mild febrile reactions, and to extreme shock pictures associated with profound vaso-motor paralysis. The reaction varies with the substances used, its method of application and absorption and its dosage, with the type of infection we are dealing, with the number of previous injections, with the physical condition of the patient, with the duration of the disease from which the patient is suffering, with the temperature of the patient at the time of the injection and other individual factors. Certain of the agents always give a reaction in both normal individuals and in persons ill with disease and give a reaction in a relatively short period of time after the injection. Typhoid vaccine belongs to this category. The colon bacillus more often gives a delayed reaction while some of the protein split products such as the albumoses may produce a reaction in diseased individuals but not in a normal person when the same dose is employed.

The Chill.—Usually the first symptom that attracts the attention of the patient after the injection is a chill or a

rigor. This may set in within half an hour after the typhoid vaccine and from 6 to 8 hours with the colon vaccine. This lasts about 20 minutes to an hour. In cases of arthritis with painful and tender joints, a severe chill is to be avoided by reason of the resulting increased pain and distress. The intra-muscular injection of milk is not generally followed by a rigor. With the subsidence of the chill or during its later stages, the temperature of the patient has as a rule commenced to rise. In typical arthritic cases running a febrile course of from 100° to 101°, the injection of typhoid vaccine will raise the temperature to about 103° or 104°, the maximum increase being recorded in from three to four hours after injection. With intramuscular milk injection, the maximum temperature may not be reached until 6 or 8 hours after the injection. As a rule, the defervescence is more rapid with the intravenous injections, but even in the case of milk injections, the former level is reached within 24 hours.

Pulse.—Coincident with the temperature reaction, the pulse is almost invariably increased to some extent, usually about 15 to 30 beats more, per minute.

Sweating.—Shortly after the subsidence of the chill, the patients sweat profusely; in arthritic patients this is very common.

Nausea and vomiting.—These are occasionally observed after relatively large doses of vaccines. It is usually an indication that the dosage has been too large or that the patient is particularly sensitive. The gastro-intestinal tract will frequently show increased peristalsis. Sometimes there appears to be an increased secretion of milk in lactating women. Headache of the frontal region is a common symptom, but it is not as a rule of long duration. The most notable blood changes affect the leucocytes. As a general rule a leucopenia first appears which is tempo-

rary and followed in 5 to 12 hours by a leucocytosis. This reactive leucocytosis is of a myeloid type that is due to the stimulation of the bone-marrow and is largely made up of neutrophil polymorphonuclears, large mononuclears and transitionals. This leucocytic change is most marked after the first injection and tends to be reduced after subsequent injections.

The nitrogen balance shows considerable variation both experimentally and clinically, following the introduction of proteins and their split products. In patients, the injection of the non-specific agent is frequently associated with an increase in the nitrogen excretion, the urine alone showing an increase in its total nitrogen of 20 to 30 per cent. After about two days the nitrogen excretion reaches the normal. Not infrequently, patients experience a general feeling of improved well being, after one or more injections, and an increase of body weight.

The Mechanism of the Reaction

The effects and results of non-specific protein medication are better known and understood than the mechanism of their production. When the clinical results of non-specific therapy were first made known, we were quite at a loss to account for the results obtained. It represented empiricism pure and simple, and being diametrically at variance with current conceptions of immunity and resistance, it was to be expected that scientifically trained physicians would be decidedly sceptical. But when with increasing evidence of clinical success, the importance of the reaction would no longer be ignored, one has to seek for some explanation to account for the therapeutic benefit even though we had to recast our entire conception of the mechanism of recovery from disease. Weichardt who has conducted a

great deal of investigation in this field, believes that when we make use of N. S. T. we stimulate all the cells of the organism to greater activity in the production of either specific substances, anti-bacterial in character, or merely increase the general resistance to intoxication. This stimulation does not involve any alteration in function. The organism by N.S. reaction acquires no new methods of defence. Probably it does not overcome infection or intoxication through agencies other than those already at its disposal. But the stimulation represents a summation, a cumulative effort of the defensive agencies of all the organs. And as a necessary corollary it is but logical that we can achieve no therapeutic effect when once the organs have by complete exhaustion been rendered incapable of reaction—as in the terminal stages of disease processes. While the stimulation does not involve any new method of defence, differences in reaction exist between normal individuals and individuals ill or sensitised. The cell that has been sensitised responds more promptly with a mobilisation of productive agents, both specific anti-bodies as well as enzymes, and other non-specific factors in resistance. The therapeutic value of the reaction according to *Peterson*, who has written a monograph on the subject, rests upon the detoxicating effects of increased enzyme activity. The symptoms of bacterial disease are due to intoxication by proteins or by protein split products. Detoxication in either natural or induced recovery depends on the digestion by enzymes of these proteins to their lowest degradation products which are non-toxic. The main point of his argument appears to be that bacterial infections give rise to protein intoxication, that these proteins are destroyed by digestion by enzymes and that a production of enzymes is stimulated by the injection of foreign proteins.

The diseases in which benefit by this form of treatment has been noticed fall into four main groups.

1. Infective disease in which the casual organism is known and in which the vaccine is given intravenously both for its specific and possible shock effect *e.g.*, typhoid fever, coliform infections of the urinary tract and the septicaemias.

2. Infective disease in which the causal organism is unknown or not certainly determined *e.g.*, infective or rheumatoid arthritis.

3. Infective disease in which the causal organism is known but in which specific vaccine therapy is of little value *e.g.*, pneumonia, gonorrhoea.

4. Chronic disorders of doubtful or unknown etiology, skin conditions like psoriasis, pemphigus; and bronchial asthma and epilepsy.

Typhoid

Gay states that the milder cases of the disease react better to the treatment than the more severe ones. The usual dose of an initial injection was 25 to 50 millions and the dosage in successive inoculations was slightly increased in order to produce a similar reaction. The injection is followed, in from 15 minutes to one hour, by a chill and is accompanied by a rise in temperature of one to three degrees, reaching its maximum within three hours and then falling. Mild reactions of this kind are desirable, as a general rule the injections are given every two or three days in slightly increasing amount until three or four injections have been given. Most of the beneficial results are attributed to non-specific effects, principally to the hyper-leucocytosis. If the pulse is over 100 and there is evidence of vaso-motor instability, if the patient is profoundly toxic or cachectic, if there has been any bleed-

ing, if the disease has continued for several weeks before treatment is commenced or if there is any evidence of pneumonic complications, it is not advisable to try non-specific therapy.

Asthma

True asthma is to be distinguished from the many other conditions to which the term asthma has often been erroneously applied; for instance, *chronic bronchitis, cardiac asthma, and renal asthma*. N. S. T. is applicable only to pure spasmodic asthma. This is not a disease but a recurrent phenomenon occurring in unfortunate individuals who have particular idiosyncrasies and the idiosyncrasy underlying the spasmodic asthma phenomenon is protein. The attacks are due to upsets of nervous system caused by a great variety of different chemical reactions in the body and brought on, in some as the result of partaking of one or other of a great number of different food substances and in others by the effects of animal products and so on. In one case the idiosyncrasy may be due to some quiet ordinary food substance like an orange or pappoy, in another some out of the way substance like chicory may be the cause, in a third it may be due to an article of clothing like wool or silk fabric and in a fourth to something else. Underlying the great majority of such cases of asthma there seems to be some peculiarity of the chemical reaction occurring in the body as the result of the ingestion of some thing either by inhalation or by mouth or even by contact. It has been found that the injection—generally intravenous—of a solution of peptone sometimes serves to alter the normal chemical processes in such a way that a patient hitherto susceptible to some particular foreign substance liable to cause asthma in him, becomes no longer susceptible and

it is only in this way that solutions of peptone given intravenously are of real therapeutic value in the treatment of asthma. One cannot say that we have as yet very exact knowledge of what dosage of peptone is the best, nor of just how many injections should be given to derive the maximum benefit, nor do we yet know whether peptone is really the best protein to employ. But it has been established on the basis of the results obtained in a fair number of cases of true spasmodic asthma that a five per cent. solution of Witte's peptone in normal saline intravenously, using for the first dose five minims, increasing each successive dose by two minims until a marked general reaction is produced, is the most satisfactory procedure to be followed. About 6 to 12 injections are necessary, the interval between the doses varying from 5 to 7 days. That the treatment does, sometimes by its anti-peptone reaction within the patient, lead to a great amelioration of the tendency to attacks of spasmodic asthma, has now been established beyond doubt though it is difficult to tell before hand whether a particular case is going to receive much benefit by this treatment.

Epilepsy

Upon the theory that there is some kind of relationship between the peculiar chemical processes in the body of an epileptic and the attack he suffers from, attempts have recently been made on a fairly large scale to treat epileptic patients by trying to modify their intrinsic metabolic processes by the administration of a foreign protein in a way allied to that which has been described above for asthma. As in asthma the injections of a 5 per cent. solutions of peptone are given until 20 minims are reached. If no results were obtained after three doses of 20 minims the treatment was given up.

Indications and Contra-Indications

Protein therapy offers a potent method that we have at our command of altering the current of cellular activity. Proper dosage results in transient but well marked stimulation, and if continued for a period of time, the alteration of metabolic processes becomes clinically manifest in a feeling of general well-being. We can intelligently apply protein therapy in infectious diseases, only if we remember constantly and firmly that non-specific therapy is purely a method of stimulation of the cellular forces in the body, and this stimulation will be useless and even disastrous when the cells of the body are profoundly fatigued. As with other new therapeutic procedures, there is still some uncertainty as to the proper dosage. Especially is this true because the dosage depends to some extent on the vigour of the patient, on the type of infection which is causing the disease as well as on the stage of the disease. There is no unanimity of opinion as to whether it is well to give one or two fairly large doses or several smaller ones. Whichever method is decided upon it is safe to be cautious especially when dealing with such toxic substances as vaccines. In general it is to be kept in mind that injections must be given early in the course of any disease, that the injections should be given slowly and that a relatively small dose should be given at the first time. It is unnecessary to point out that the non-specific method of treatment outlined above should under no circumstances be considered as a rival to, or a substitute for, proven specific measures that we have at our command. That a non-specific factor is at times and possibly often associated with the specific reaction may be true, the more reason why both should be studied and both utilised in their proper time and place. From the evi-

dence available so far, the use of specific measures is always in place, whenever a true toxin is to be neutralised by an anti-toxin; on the other hand the non-specific measures find a field of usefulness as adjuvants of drug therapy, the treatment of syphilis both early and late, with salicylates in arthritis, and with luminal in epilepsy. Here, the injections serve at least two purposes. They facilitate the diffusion and distribution of the drug and they increase the general resistance of the patient.

Contra-Indications.—No patient should be subjected to an intravenous injection unless there is strong proof that he will be able to bear the additional strain imposed by the injection. Therapeutic results cannot be expected when the organism is no longer capable of response to stimulation. Alcoholism is an absolute contra-indication. Pregnancy must be similarly regarded. In various cardiac lesions great caution must be observed.

In conclusion, the whole subject of non-specific protein therapy may be said to be still in its infancy. Needless to say, it does require judgment, careful attention, and besides, study on the part of the physician perhaps to a much larger extent than any other therapeutic measure.

Kala-Azar in Madras*

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Summary

1. Introduction.
2. Epidemiology of Kala-Azar.
 - (a) Distribution according to race and caste.
 - (b) Distribution according to age and sex.
 - (c) Seasonal variation of Kala-Azar.
 - (d) Distribution of Kala-Azar in Madras City.
3. Urea Stibamine in the treatment of Kala-Azar.
4. Binomics of Sand-flies and its relation to the incidence of Kala-Azar in Madras.
5. Conclusion.

Epidemiology of Kala-Azar

The observations were drawn from the cases admitted to the General Hospital in 1924 and 1925, the records of the previous years were summarised by Korke and Cunningham and Varda Rajan.

Taking all the 406 Kala-Azar cases admitted during 1924 and 1925 in the General Hospital we find:

Racial Incidence

1. Europeans—2			
2. Chinese—1			
3. Indians—			
	Hindus	105 & 139	... 244
	Mahomedans	6 & 12	... 18
	Indian Chrs.	15 & 16	... 31
	Other castes	26 & 20	... 46
			339
4. Anglo-Indians—64.			
Total—406.			

All races are affected. Although the number of Anglo-Indians is small in percentage of cases when compared with Hindus and Mohamedans the relative incidence of the disease in the community is still high.

* Extracts from the half-yearly reports forwarded to the University of Madras.

Regarding the Chinaman no definite history could be traced to the place from which he got the infection. Considering the size of the spleen and the duration of stay in Madras city it looks as if he got the infection in Madras rather than in China.

(a) Distribution according to Age or Sex

Age.	1924.		1925.		Total.		Total.
	Males	Females.	Males	Females.	Males	Females.	
0 to 5	2	3	2	2	4	5	9
6 to 10	1	6	14	12	32	18	50
11 to 15	30	15	26	11	56	26	82
16 to 20	28	3	23	9	51	12	63
21 to 25	28	5	40	7	68	12	80
26 to 30	20	4	24	10	44	14	58
31 to 35	7	3	12	1	19	4	23
36 to 40	6	2	6	4	12	6	18
Above 40	10	1	6	6	16	7	23
	149	42	153	62	302	104	406
	191		215		406		

(See graph 1)

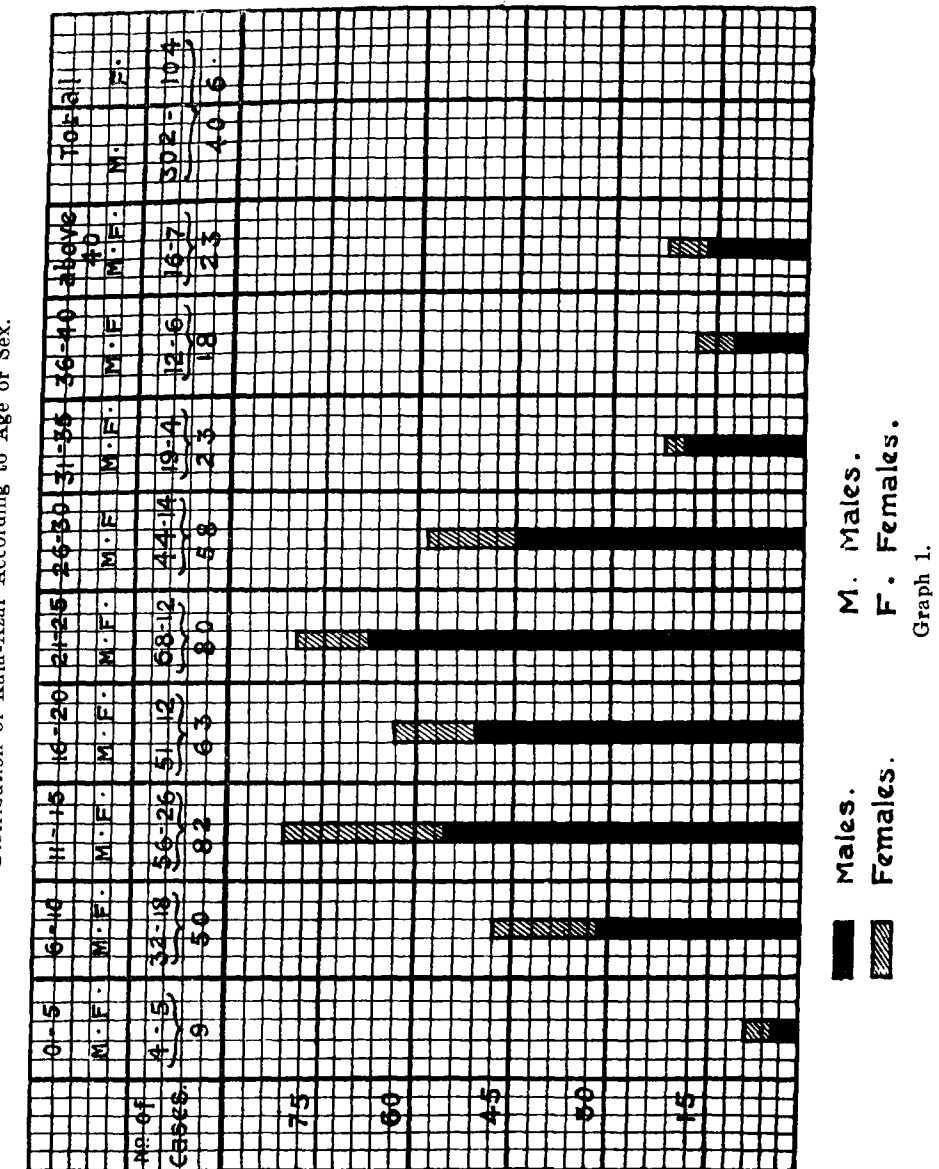
From the above figures it is evident that the minimum age recorded is 3 years and the maximum 62 years, the greatest incidence being between 10 and 30 years.

Sex.—Regarding sex, females are only few when compared with males. This information seems to be correct, only in hospital patients; but private practitioners say that cases occur among females far more than the Hospital statistics show.

	Admission to Hospital		Total
	1924	1925	
January	18	21	39
February	6	18	24
March	17	19	36
April	13	18	31
May	21	30	51
June	11	11	22
July	18	17	35
August	12	21	33
September	23	17	40
October	24	14	38
November	14	18	32
December	12	11	23
	191	215	406

(See graph 2)

Distribution of Kala-Azar According to Age or Sex.



(b) Seasonal Variation of the Disease.

Only 115 out of the 406 cases gave the definite history of onset.

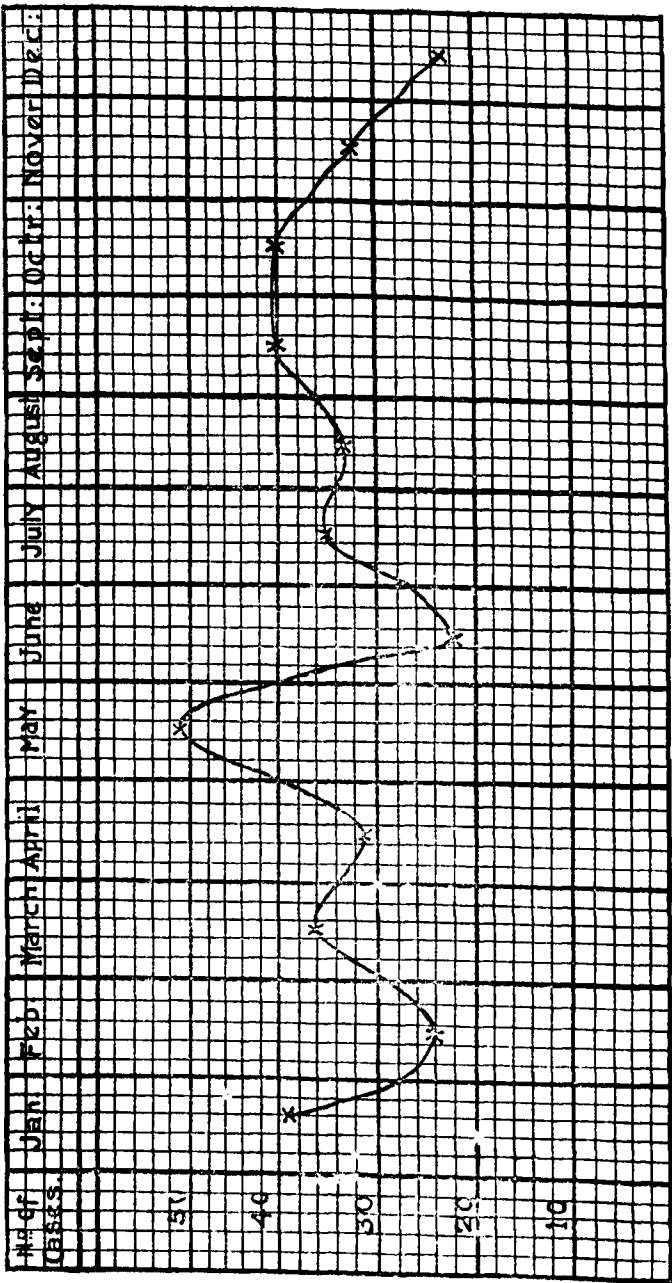
	1924	1925	Total
January	6	13	19
February	4	5	9
March	8	7	15
April	1	2	3
May	3	7	10
June	1	6	7
July	3	5	8
August	3	8	11
September	5	4	9
October	1	8	9
November	1	3	4
December	7	4	11
	43	72	115

(See graph 3)

Comparing the two graphs (2 and 3) obtained from the number of admissions to the hospital and the onset of the disease, one finds that patients come to the hospital very early in the disease and they are mainly of the typhoid type. Here the reason is obvious. But we have to take into consideration the other set of cases that come in with a spleen reaching the level of the umbilicus. In these cases the history is unreliable and does not give any clue as to the seasonal variation of Kala-Azar.

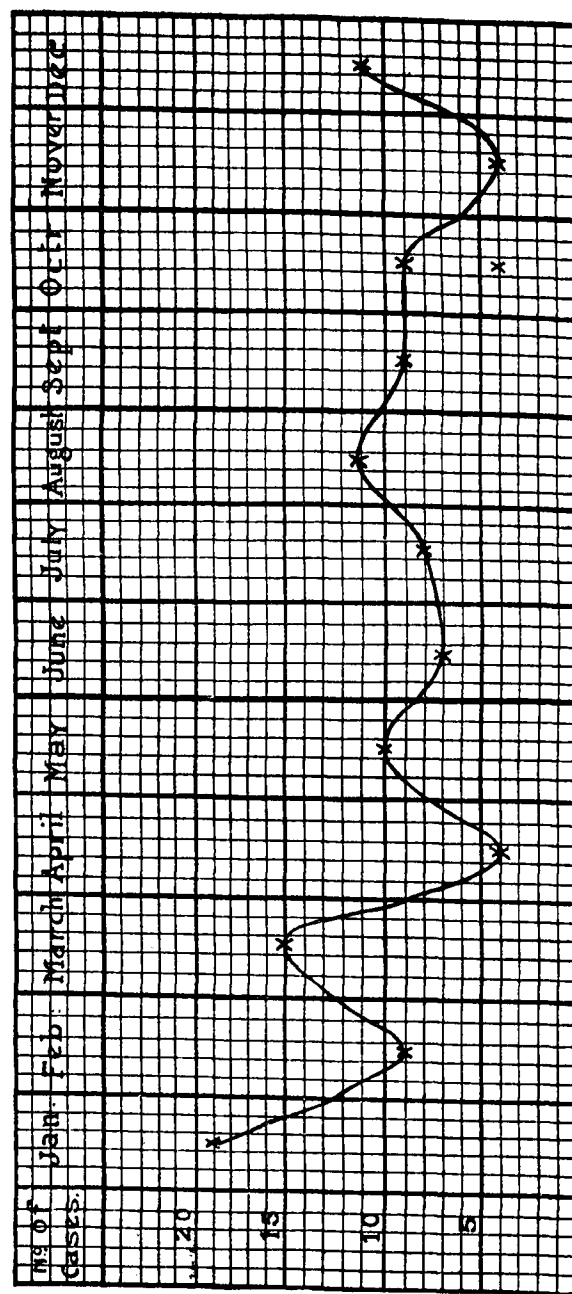
The greatest number of cases show the onset in January, February and March but the curve does not rise in October and November as shown by Napier in Calcutta. In my graph there is a slight rise in December but this I think is due to the close proximity of the month of January in which we have the greatest number of cases. Of course, one has to admit the smaller number of cases with which one is dealing. The two rises in the curve of seasonal variation as shown by Dr. Napier is not seen in the curve of Madras.

Admission of Kala-Azar Cases in the General Hospital.
(1924-1925).



Graph 2.

Seasonal Variation of Kala-Azar.



Graph 3.

Methods Employed in the Diagnosis and Treatment with Results:—

Out of the 191 patients admitted in 1924 the disease was diagnosed by :—

	No. of cases.
1. Finding L. Donovan Bodies in spleen or liver puncture	... 27
2. Finding L. Donovan Bodies in the peripheral blood	... 11
3. By clinical evidence and positive aldehyde reaction	... 87
4. On clinical grounds alone	... 66
Total	... 191

One is not justified in including the last two groups of cases as those of Kala-Azar. Cases which clinically look like Kala-Azar and showing a strongly positive aldehyde reaction were shown to be negative for L. Donovan bodies after spleen puncture and culture in N. N. N. Media.

Treatment Employed and Results:—

	Europeans		Indians		Total
	...	1. Resistant to treatment.	Anglo-Indians	Indians	
1. Total No. of Deaths	...	...	4	20	24
2. Cases that went away without any treatment	...	...	1	22	23
3. Incomplete treatment	...	...	3	54	57
4. Those that went away and came back for treatment	...	...	2	6	8
5. Cases resistant to treatment	...	...	1	1	2
6. Cured	...	...	27	49	76
Total	...	...	38	152	190
Grand Total					191

The word "Cure" means, disappearance of fever, disappearance of spleen underneath the costal margin, improvement in the general condition of the patient and increase in body weight. Only in one case was liver puncture done after treatment and found negative. In this case also no culture of the liver juice was done. Two of the resistant cases were treated with urea-stibamine and all the rest were treated with 2 solution of sodium antimony tartarate.

In 1925 the cases were diagnosed by :—

1. Spleen or liver puncture	...	83
2. Finding the parasite in the peripheral blood	...	15
3. Clinical evidence and positive aldehyde reaction	...	78
4. On clinical evidence alone	...	39
Total		215

The increase in the number of cases when compared with the previous year is evident. The diminution in number in the last two groups is remarkable. Nearly 25 cases more would have come to the third and fourth group of cases showing a doubtful or a positive aldehyde reaction. But these cases were investigated by me and were found to be not suffering from Kala-Azar as shown by the negative spleen-puncture culture.

Treatment and Results

1. Total No. of deaths	...	26
2. Those that went away without any treatment	...	14
3. Incomplete treatment:		
Tartar emetic 40 }		
Urea Stibamine 11 }	...	51
4. Discharged and came back for treatment	...	1
5. First treated with Sodium antimony tartarate and later with Urea Stibamine. None of these were resistant cases.		12
6. Resistent Cases	...	1
7. Cured with :		
Sodium antimony tartarate	...	64
Urea Stibamine	...	45
Amino Stiburea	...	1
Total		215

Of these cured cases, the majority were punctured after treatment and found negative after culture.

(c) Distribution of Kala-Azar in Madras City

Out of the 406 cases admitted to the Hospital during the two years 1924 and 1925 only 288 cases gave a definite idea of the place of residence in the Madras city and they are

	1924	1925	Total
George Town	54	74	128
Vepery including New Town...	32	39	71
Triplicane & Mylapore	14	6	20
Royapuram	7	9	16
Chintadripet	6	9	15
Puraswalkam	4	6	10
Pudupet	3	3	6
Royapettah	5	1	6
Perambur	2	4	6
Kilpauk	4	1	5
Washermanpet	2	1	3
Egmore	1	1	2
			288

Korke in 1912 said that George Town was 'the cradle for Kala-Azar'. That statement is true even to-day and that portion of the city still contributes the greatest number of cases.

They were unevenly distributed, and the majority of them were from :

1. Dravidson Street	7. Coral Merchant Street
2. Bunder Street	8. Presentation Convent Street
3. Broadway	9. Govindappa Naicken Street
4. Sembudoss Street	10. Linga Chetty Street
5. Mint Street	11. China Bazaar Road.
6. Armenian Street	

Triplicane.—One case was that of a student from Victoria Hostel and the other a clerk from the Buckingham Lodge. Regarding the first, the evidence was only clinical. In both these cases it has been very difficult to trace the source of infection. These might have got it from Madras City.

Body-Guard lines and Government house premises were not stated to be infected before. In this series two cases have come from the former and one from the latter.

Kilpauk.—Korke has reported cases from Kilpauk but does not say from which portion of the area. Civil Orphanage Asylum had two cases (two brothers) in 1925. In these two cases the infection could be traced to George Town where they lived four months before the onset of fever. The third case came in 1926. The fourth was an Anglo-Indian girl running a continuous temperature for 15 days. In this case the patient was discharged before a definite diagnosis was made. Clinically it looked like Kala-Azar. We have to wait and see whether that patient comes back again with fever and enlargement of the spleen.

Vepery and New Town.—Every street and by lane of New Town is infected, Naval Hospital Road, the 1st, 2nd and 3rd Street in New Town showing the largest number. Other streets are also infected.

1. Vepery High Road.
2. Sydenham's Road.
3. Maddox Street.
4. Rottler's lane.
5. Choolai High Road.

Chintadripet.—The number of cases are increasing every year in Swami Naicken Street. Iyah Mudali Street and Cooum River Road also have a large number of cases.

Royapuram.—Thana Street, when put in simple terms, can be called the Kala-Azar street of Royapuram. That Kala-Azar is both a house and household infection is clearly shown by a few of the families residing in that part of the city.

Egmore.—The two cases that gave the residence as Egmore contracted the disease in George Town.

Taking the Madras City as a whole, one finds that the

greatest incidence is in George Town, Vepery, New Town and Chintadripet. Nungambakam and Egmore are the only two areas in Madras that are free from indigenous cases.

Imported Cases.—Outside Madras Presidency there were cases from Calcutta and Assam but now from Behar and Orissa. Inside the Presidency cases come from Kayalpatnam, Tinnevely Bridge, Dhanushkodi and Calicut.

Urea Stibamine in the Treatment of Kala-Azar

Case (1) Mr. U. —Indian Christian, male, aged 50 years, spleen palpable 1 2" below costal margin. Liver puncture showed L. D. bodies, R. B. C. 2,700,000. W. B. C. 1800, Hae. 50%, wt. 70 lbs.

Total No. of injections	... 4
Amount of Urea Stibamine	... 0.5 gm
Duration of treatment	... 13 days

After treatment.—Spleen not palpable. Liver puncture culture negative. R. B. C. 3,640,000. W. B. C. 5800. Hae. 55%, wt. 72 lbs.

Case (2) Miss. V.—Indian Christian, female, aged 10 years, spleen palpable 3/4" below costal margin. Spleen puncture showed L. D. bodies, wt. 42 lbs.

Total No. of injections	... 5
Amount of Urea Stibamine	... 0.7 gm
Duration of treatment	... 14 days

After treatment.—Spleen not palpable. Liver puncture culture negative, wt. 49 lbs.

Case (3) Mr. R. —Hindu, male, aged 21 years. Spleen palpable below costal margin. Spleen puncture showed L. D. bodies, wt. 103 lbs.

Total No. of injection	... 7
Amount of Urea Stibamine	... 1.05 gm
Duration of the treatment	... 18 days

After treatment.—Spleen palpable $1\frac{1}{2}$ " below costal margin. Spleen puncture culture negative, wt. 109 lbs.

Case (4) Mr. N.—Mahomedan, male, aged 23 years. Spleen palpable 2" below the umbilicus. L. D. bodies in the peripheral blood, wt. 99 lbs.

Total No. of injections	... 7
Amount of Urea Stibamine	... 0.975 gm
Duration of treatment	... 14 days

After treatment.—Spleen palpable $1\frac{1}{2}$ " below costal margin. Spleen puncture culture negative. R.B.C. 5,072,000 W. B. C. 4800. Hae 80%, wt. 102 lbs.

Case (5) Mr. K.—Hindu, male, aged 19 years. Spleen palpable 4" below the umbilicus. Spleen puncture showed L. D. Bodies. R. B. C. 2,800,000. W. B. C. 2150. Hae. 35%, wt. 94 lbs.

Total No of injections	... 10
Amount of Urea Stibamine	... 1.7 gm
Duration of treatment	... 28 days

After treatment. Spleen palpable 1" above umbilicus. Spleen puncture culture negative, R. B. C. 4,028,000. Hae 80%. W. B. C. 8,000. wt. 107 lbs.

Case (6) Mr. N.—Hindu, male, aged 22 years. Spleen palpable $2\frac{3}{4}$ " below umbilicus. Spleen puncture showed L. D. bodies. R. B. C. 1,950,000. W. B. C. 3,500. Hae 35%, wt. 87 lbs.

Total No. of injections	... 10
Amount Urea Stibamine	... 1.75 gm
Duration of treatment	... 30 days

After treatment.—Spleen palpable 1" below costal margin. Spleen puncture culture negative. R. B. C. 4,300,000. W. B. C. 5,280. Hae. 70%.

Case (7) Miss. K.—Hindu, female, aged 7 years, spleen palpable $2\frac{1}{2}$ " below costal margin. Spleen puncture showed L. D. bodies. wt. 28 lbs.

Total No. of injections	... 12
Amount of Urea Stibamine	... 1.045 gm
Duration of treatment	... 37 days

After treatment.—Spleen not palpable. Liver puncture culture negative, wt. 33 lbs.

Case (8) Miss. S.—Hindu, female aged 8 years. spleen $1\frac{1}{4}$ " below umbilicus. L. D. bodies in peripheral blood. R. B. C. 2,050,000. W. B. C. 3,760. Hae. 35%, wt. 31 lbs.

Total No. of injections	... 13
Amount of Urea Stibamine	... 2.25 gms
Duration of treatment	... 47 days

After treatment.—Spleen not palpable. Liver puncture culture negative. R. B. C. 4,140,000. W. B. C. 5,280. Hae. 75% wt. 37 lbs.

Of all drugs we have used so far in Madras, Urea Stibamine is the best in the treatment of Kala-Azar.

A few of the observations in connection with the treatment by Urea Stibamine are worth mentioning.

Patients are discharged from the Hospital with a spleen still enlarged, after treatment. The criterion of cure in these cases being:—

- (1) Disappearance of fever.
- (2) Increase in weight.
- (3) Increase in the R. B. C., W. B. C. and haemoglobin percentage.
- (4) Reduction in the size of the spleen.
- (5) Negative spleen puncture culture.

As one had the opportunity of studying these cases a month later, it was surprising to find that a spleen which was 3" to 4" below the costal margin had completely disappeared.

The same phenomenon was also observed during the course of treatment in Hospital. When the supply of Urea Stibamine could not be had for a fortnight, patients were

left without any injections, the result being, that the spleen diminished in size even without any treatment.

What does this mean? Patients can be discharged from the hospital as cured even with an enlarged spleen. Of course the negative spleen puncture culture must also be taken in conjunction with disappearance of fever, increase in weight and increase in blood count and haemoglobin percentage.

As one looks back to the former days of Trivalent Antimony (Sodium or Potassium Antimony tartarate) such a phenomenon was not observed at all.

The following additional points require mention:

(1) Reduction in the size of the spleen extending to an inch above the umbilicus, is uniform and rapid.

(2) In spleens reaching as far as the umbilicus or below that, there is no appreciable reduction in the size of the spleen even after 5 or 6 injections (0.9 gm.). After the 6th injection the reduction seems to be rapid and uniform.

(3) From the consistency of the spleen as felt by palpation one can approximately judge whether the reduction will be rapid or delayed.

Firm consistency—in spleen extending to 2" to 3" below.

Firmer—To the level of umbilicus.

Tough—below the level of umbilicus.

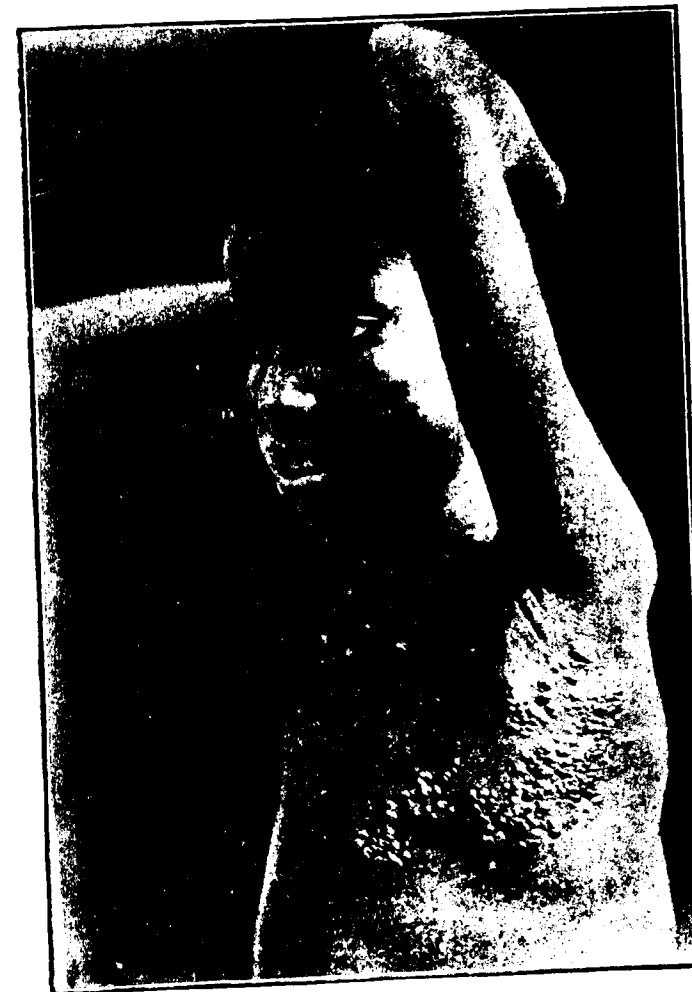
The last one will not show any reduction in size at all even after 4 or 5 injections of Urea Stibamine.

The observations are only relative and in many instances one of personal equation.

Occurrence of Herpes After Antimony Injection in Cases of Kala-Azar. (see photograph).

Case (1) *Distribution.*—along the course of 1st, 2nd and 3rd Dorsal nerve roots.

Herpes Occurring after Antimony Injections



Herpes occurred 4 days after the last injection. Distribution of Herpes 5th, 6th and 7th Dorsal Nerves.

Total number of injections ... 9
 Total number of Antimony ... 0.7 gm.
 Solution used (2% solution of Sodium Antimony-tartrate.)

Case (2) *Distribution*.—along the course of 8th, 9th, and 10th, Dorsal nerve roots.

Total number of injections ... 12
 Total amount of Antimony ... 1.1 gms.
 Solution (2% solution of Sodium Antimony tartarate.)

Case (3) *Distribution*.—along the course of 5th, 6th, and 7th Dorsal nerve roots (left side).

Total number of injections ... 23
 Total number of Antimony ... 1.3 gm.
 Solution used (2% solution of Sodium Antimony tartarate.)

Case (4) *Distribution*.—along the course of 10th, 11th, and 12th, Dorsal nerve roots (left side).

Total number of injections. ... 5
 Total amount of Antimony. ... 0.7 gm.
 Solution used—(2.5% solution of Urea Stibamine dissolved in distilled water).

All these four cases were under Dr. M. R. Gurusawmy Mudaliar and occurred within a period of 5 months.*

In case (2) that showed the distribution along the course of 8th, 9th & 10th, Dorsal nerve roots of the left side the herpes juice from the vesicle showed L. Donovan bodies. No flagellates could be grown in culture since the inoculated media became contaminated.

In case (4) a thorough investigation was made.

1. Herpes juice showed no L. D. bodies.
2. No flagellates from the herpes juice could be grown even after 20 days.
3. The herpes juice was tested for Antimony and no trace of the drug could be detected.

* A brief note of this was published in the Madras Medical College Magazine, April, 1926.

4. Urine showed a negative reaction for Antimony.
Points to be noted here are :—

(1) All the cases showed the distribution of the vesicles along the dorsal nerve roots. Osler says "Herpes is frequently seen in the region of the dorsal nerve roots". So there does not seem to be anything peculiar about the seat of election along the dorsal nerve roots.

(2) All the cases occurred on the 3rd or 4th day after the last dose of Antimony.

The occurrence of herpes after Antimony injections seems to be a rare phenomenon. Both Sodium Antimony Tartarate and Urea Stibamine can produce the same condition showing thereby that Antimony is responsible for the herpes. None of the text-books in discussing the etiology of herpes give Antimony as one of the factors in the production. Only Arsenic has been mentioned by Taylor. Dr. U.N. Brahmachari (in a personal communication) says in his wide experience in the treatment with Antimony salts in Kala-Azar he has seen only a few cases.

Binomics of Sand Flies

Oviposition.—Adult gravid females (*P. Argentipes*) kept in captivity lay their ova in 3 to 5 days and then die. How long they live after oviposition is a very difficult point to decide. On looking into the "chatties" in which the ova are laid two kinds of distribution of eggs could be made out. One, in singles distributed throughout the vessel, the other occurring in clumps in the neighbourhood of which one always can find the dead body of the female. These facts closely prove that the flies might die immediately after oviposition or might live a few hours after the act.

Places selected for oviposition of the eggs are cool and

moist ones. This was decidedly shown in the "chattie" that had been accidentally upset moistening only one half and not the other. All the ova laid were found only in the moist half, whereas there were none in the other half.

Where do sand flies oviposit in nature? Larvae of *P. Argentipes* have been found in the cool and dark corners of stables. Evidently these must be the places where they oviposit.

Larvae. It takes about 5 to 10 days for the development of the larvae from the ova depending on the temperature and the amount of moisture present. When once the larvae come out they want food material to live upon. In nature the organic matter of animal and vegetable origin in the corners of stables form the best food material for the larva. Those in the laboratory are supplied with rabbit's faeces devoid of any fungus growth. The presence of fungus in the "chattie," retards the growth of the larvae and in a majority of cases kills them.

Sufficient food material has to be supplied and the amount of moisture has to be attended to. When a chattie containing larvae is opened none of the larvae will be found. But on turning the lumps of rabbits faeces they can be found either singly or in numbers. If one has accurately observed it is easy to locate the place where the larvae can be found in the "chattie". Excreta of larvae can be seen surrounding the small lump of faeces that covers the larvae.

As the larvae grow in size the amount of moisture necessary is less, but the food material has to be increased. Their activity and movements become diminished, the greyish black appearance of the gut of the larvae disappears and becomes dirty white in colour. This colour of the larvae indicates that they will become pupae within 3 or 4 days. One has to bear in mind that this appearance

alone cannot be taken as characteristic. When the food material inside the "chattie" is attacked by fungus larvae of even 8 to 15 days growth might show the appearance. With a size of 2.5 m.m. lengths and the dirty white appearance one can foresee the development of the pupa.

The whole larval stage takes about 20 to 35 days. Twenty-two days can be taken as an average, but in the cool months of December and January, the duration of this stage may be carried to the extreme limit of even 40 days.

Pupa. This stage lasts from 5 to 12 days. Just as in the larval stage the maximum number of days required for this stage will be in the months of December and January. No moisture is required in this stage and no addition of food material is necessary, since they do not feed.

Adult fly. From the pupae emerge adult male and female sand flies. There does not seem to be any relation at all between the number of males and females bred out. Here is the result of four batches of flies bred out.

	Males	Females
Batch 1	3	7
Batch 2	7	9
Batch 3	7	3
Batch 4	8	9

When kept in muslin cages without any food almost all the females die away by the 3rd day and none are left by the fourth. The males die later than the females. It is obvious, only the females die before the males, for, the males can feed on the residual organic matter. When once the chatties are removed from the cages one will be surprised to find that on the fourth day the flies remaining are females and not males. No doubt a few males survive even to the fourth day.

STAGES IN THE LIFE CYCLE OF THE SAND FLY
(Phlebotomus Argentipes)
 Mag. $\times 20$.

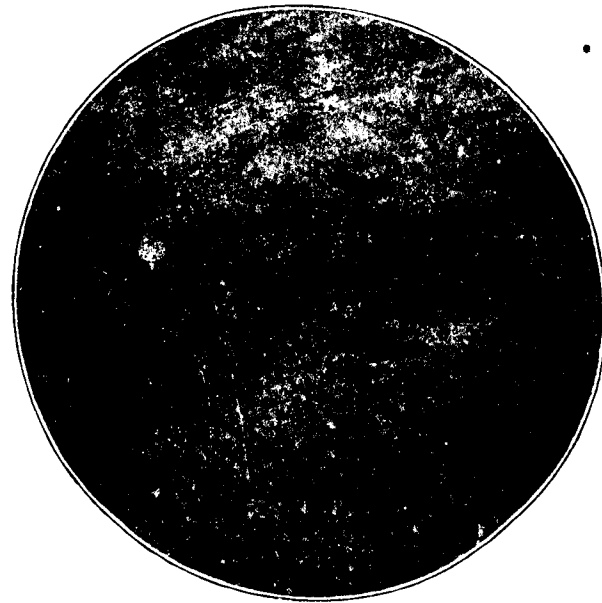


Fig. 1. Ova.

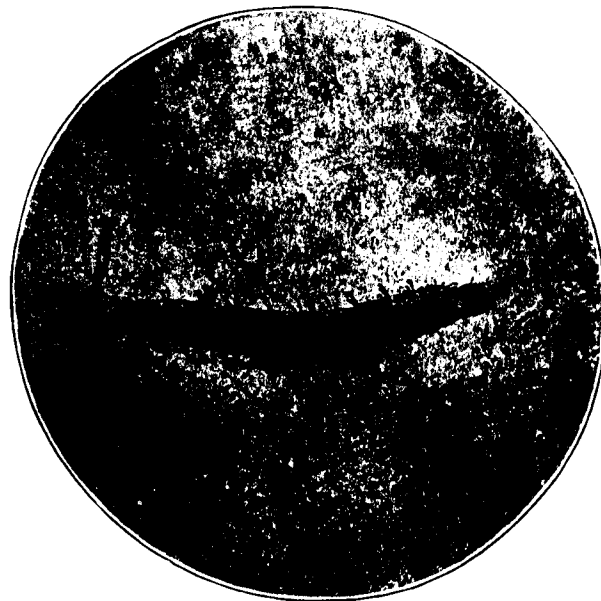


Fig. 2. Larva (13 days old).



Fig. 3. Pupa (from which the adult sand fly has emerged).



Fig. 4. Female.



Fig. 5. Male.

Feed.—Only the females feed on blood, whereas the males feed on vegetables or organic matter. *How long do they take to feed?* By observing through a cage in which a hand is partially introduced one can follow the activities of these insects. It takes some time, probably about $\frac{1}{2}$ an hour for the sand flies to get accustomed to the hand introduced into the cage. The patient can easily say when they are bitten by the sand flies. This makes them change the position of the hand and sometimes this shifting of the hand prevents the fly biting him altogether. I had the opportunity only once to observe the whole process of feeding. It took exactly five minutes. In nature probably this might be completed in still shorter time.

A few of the flies do not get the opportunity of taking a full blood meal and one has been able to see in the course of experiments some partly fed flies.

How many times do sand flies feed? This is a matter for controversy. First of all I never succeeded in feeding them a second time, but later on I succeeded in feeding them a second time, but the third trial never succeeded. The Kala-Azar commission has fed flies a third time but my attempts were quite unsuccessful.

When one goes into the cowshed to catch Sand flies it is very difficult to find at least one unfed fly. This means one of two things—either all females get the opportunity of feeding, or they feed more than once. Under artificial conditions it has been shown that they can feed more than once.

When flies are put in cages the conditions are absolutely different. It does not matter whether one puts 6, 8, 20 or 30 female sand flies together with or without males. The maximum number of flies fed at a time was only *five*.

An interesting observation has to be recorded here in

experimental feeding. The presence of males seems essential even though they do not feed. A dozen females were put in a cage and kept for feeding. One had to wait about three hours. Still none of them would bite. The presence of males however is not absolutely essential in experimental purposes. Under exceptional conditions they feed without the males. But to get greater success in feeding experiments one has to put males also into the cages.

Copulation.—One had ample opportunities of observing copulation taking place in the laboratory-bred sand flies. Copulation takes place both before and after the feed. The majority of them took place before feeding. When one looks into a cage full of laboratory-bred sand flies, at least half a dozen pairs can be seen in the process of copulation. They can be seen attached end to end with the posterior ends together. When disturbed during this process they jump “as they are”, but the movement is not so active as when they are single. The whole process lasts about *seven* minutes. A curious phenomenon was observed once. A female was in the act of feeding when four males surrounded her. All the males were restless but none of these moved more than 2 inches away from the female. One male attempted copulation in the midst of the feed, but one of his rivals came in his way and disturbed him in the process. When feeding was over she flew away, and one was not able to follow as to what happened to the males.

Duration of Life and Habits

When the fly takes only a single blood meal it could be kept alive only for five days. When they are given another opportunity to feed they could be kept alive up to the maximum limit of nine days. Regarding males none of them could be kept alive for more than 5 days.

They could be caught

- (1) From sleeping rooms
- (2) From cow sheds.
- (3) From fowl houses.
- (4) From horses stable.

Regarding sleeping-rooms a few could be caught from the first floor also, the height being about fifteen feet.

Seasonal Incidence of Sand flies and Kala-Azar

Sand flies (*P. Argentipes*) could be caught in large numbers in the months of July, August and September, then at the approach of December and January only very few could be caught and sometimes none at all. During the months of April and May the number of flies are again on the increase. During the 1st fifteen days of May and the whole of June no sand flies could be caught at all this being the hottest season of Madras.

Here in Madras, we find sand flies (*P. Argentipes*) are seen in minimum number during the hottest and coldest seasons of Madras and in larger numbers, during the other months of the year.

Comparing the greatest incidence of sand flies, and the seasonal variation of Kala-Azar in Madras one is not able to find either a positive or a negative correlation between the two.

Before concluding the report I have to thank Lt. Col. G. E. Malcomson, Dr. M. R. Guruswamy Mudaliar and Dr. T. K. Menon for their valuable advice and assistance; Dr. U. N. Brahmachari for the supply of Urea stibamine and Major K. G. Pandalai and Dr. G. V. James for giving me all facilities to work in their laboratories.

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Pitfalls in the Practice of Midwifery*

BY DR. A. LAKSHMANASWAMI MUDALIAR B. A., M. D.

MY object in choosing this subject for to-night's discourse was to place before you some of the common difficulties experienced by the beginner and to warn you to avoid such mistakes as are only too frequently committed by junior practitioners. I propose to speak of the mistakes I have committed and of the pitfalls into which I have seen others fall at the beginning of their career. A very large number of these errors could be avoided with care and I am convinced that it is possible to greatly minimise the risks of mortality and morbidity after child-birth.

Forceps

This is perhaps the most frequently used in obstetrics and while its value cannot be exaggerated, it must be confessed it is a potent factor for mischief in the hands of the inexperienced. Nearly half of the gynaecological troubles following child-birth are due to the mismanagement of labour with forceps and a good proportion of children who die after a forceps delivery owe their death to faults in the technique of the operation.

First make sure that the forceps is indicated. There are only two reasons which justify the use of the instrument. It should be used in the interests of the child or mother, when any definite signs are present, suggestive of danger. Never use the forceps if the head is floating above the brim of the pelvis. In cases of multiparae where the pelvis is of normal size and the head does not get fixed, if the conditions are satisfied, it is far better to

* A lecture delivered at the Giffard School of Obstetrics on 8-10-26.

do version and extract the child than to apply forceps and risk severe lacerations and the chances of post-partum haemorrhage.

Do not mistake a large caput succedaneum for the head come low down in the pelvic cavity. You can easily find it out by the pelvic grip and by carefully feeling for the bones per vaginum.

Great difficulty is sometimes experienced in extracting the head after application of forceps. This is not infrequently due to the fact that the presentation is occipito-posterior and unless forward rotation of the occiput is helped, the delivery will be difficult. If, however, traction is persisted in, two dangers are likely to result—severe laceration of the maternal soft parts with an extensive tear of the perinaeum and undue compression of the foetal head with intra-cranial haemorrhage.

Note that there will be several signs suggestive of an unreduced occipito-posterior presentation. Besides the facts elicited by palpation and vaginal examination, you would note when forceps are applied, that—

1. There is some difficulty in the introduction of the blades.
2. The blades are locked with some difficulty and there is a gap between the handles.
3. As you pull, there is a tendency for the forceps to rotate.
4. Very little of the forceps is outside the vagina.
5. The forceps tend to slip. This may occur also in cases of hydrocephalus, contracted pelvis and in cases where the head is high up. When the forceps slips, it should be removed at once and a careful examination made. If the forceps be allowed to slip, the thin edges of the blades will cause tears in the vagina and later these will become the foci for septic infection.

In pulling with the forceps, remember the direction of the pull. Don't apply too great a force in pulling and always loosen the blades at intervals of 20 to 30 seconds. The correct method of pulling is to stand up and pull with one hand against the buttock of the woman, leaning forward as in a tug-of-war rope pull. The blades should always be removed when the head is almost at the outlet. In introducing or removing the blades of a forceps, remember the law of curves.

The forceps should never be applied when the uterus is in a condition of inertia. The risk of P. P. H. is great. Nor should anaesthesia be pushed to the degree necessary for a major surgical operation. Give just enough chloroform to keep the patient under, only during the time of application of forceps and as the delivery is nearly completed, she should gradually come round.

The choice of a forceps is largely dependent on individual experience, but for a beginner an axis-traction forceps is the best. Milne-Murray's is a good one. If Barnes forceps be applied and it is later noticed that the head is not so low as it was anticipated, it is well to try and deliver by Paget's manœuvre.

The forceps may be applied in (1) the left lateral position, (2) dorsal position, (3) Walcher's position and (4) exaggerated lithotomy position.

Left lateral: This is the position generally adopted; the advantage is the perinaeum can be seen better and saved better.

The dorsal posture is sometimes adopted and it is said that the vaginal manipulations can be made with greater ease.

Walcher's position: This position is useful in moderate degrees of flat pelvis as it increases the diameter of the true conjugate by at least a quarter of an inch. In

Walcher's position the patient lies on her back across a high table with her buttocks projecting over its edge and and her lower extremities hanging free, so as not to touch the ground. It is important to remember that,

(1) the forceps should be applied before the woman is put in Walcher's position,

(2) the operator should squat on the ground and pull directly downwards,

(3) the lower extremities should not be supported, and

(4) after the head has entered the cavity, the patient should be put back in the left lateral or lithotomy position before the head is delivered. This is necessary as the size of the outlet is decreased in Walcher's position.

Exaggerated lithotomy position. This is useful in cases of funnel-shaped pelvis as thereby the size of the outlet is slightly increased.

Breech

I next refer to a few of the pitfalls in the delivery of a breech.

In some cases where the breech is high up and the patient is of more than average proportions, it is difficult to distinguish between the breech and the face by vaginal examination. It is always better to feel for the ear or the groin. In case of doubt, the patient had preferably be put under chloroform and a thorough examination made. Pass the half hand and feel for the bony landmarks and the ear or groin.

In cases of extended breech, once the membranes have ruptured and the os is fairly fully dilated, don't leave it alone. You can always get one foot down and then deliver the child or leave it for natural efforts. It is unnecessary to release both feet; traction on one leg will deliver the child up to the thorax.

The next difficulty is with extended arms and the difficulty is more often due to haste and undue interference.

Do not worry about extended arms till the body is almost fully born and the axillary fold is seen well out of the vagina. Then gently pass a finger along the shoulder of the child up to the elbow and sweep the arm down. Attempts at releasing the extended arm when the child is born up to the umbilicus necessitate the passage of the whole hand inside the vagina and result in increased risk of sepsis and possibilities of fracture of the humerus.

In the nuchal position of the arm rotate the child in the direction in which the fingers are pointing, and when the arm has become only an extended arm, release it in the usual manner suggested.

In delivering the after-coming head note –

(1) that the body should be well delivered.

(2) that flexion should be so promoted that the sub-occipito-bregmatic diameter engages.

(3) that good fundal pressure should be given.

Transverse Presentation

As this has been referred to in detail in another lecture, only one or two points will be noticed here. When the arm is prolapsed outside and the child is dead, the natural tendency is to disarticulate, and then deliver the child by version if the uterus will permit it. I should like to warn you to note that in some cases, a posterior Bandl's ring may form, which is not detected by abdominal palpation. If in such a case, disarticulation be performed, version would be impossible and the other alternative of decapitation would be very difficult.

Decapitation is performed –

1. When the uterus is tonically contracted, child dead and the arm prolapsed in transverse presentation.

2. In locked twins, when the after-coming head of the first child is impacted by that of the second, or by the shoulder and arm of the second child lying transversely.

3. In a thoracophagous monster when the first head is presenting at the outlet and the second is jammed in the pelvis, the first head being decapitated.

4. In a dicephalous monster, when both heads present and labour is obstructed the first head should be decapitated and the monster delivered by version.

The operation may be performed with the aid of a Ramsbotham's hook or a Galabin's saw or a strong pair of scissors. The last is the best. Remember that when any hook is passed into the vagina, the knob should be directed posteriorly, so that if the hook should perchance slip it may not injure the bladder.

Note that the method of delivery after decapitation is to pull on the prolapsed hand, deliver the headless trunk first and then the head by fundal pressure.

(To be continued)

Idiopathic Muscular Atrophy



Fig. 1.
Front and side views of the patient showing marked wasting of the muscles.



Fig. 2.

Idiopathic Muscular Atrophy*

(Erb's Juvenile Type)

BY DR. T. KRISHNA MENON M. B. C. M.

Honorary Physician, Govt. General Hospital, Madras.

LADIES AND GENTLEMEN,

This morning I am going to discuss a case, the details of which are as follows:

Name, etc.—Syed Ahmed, male, aged 25, clerk, Muhamadan.

Complaint.—Wasting, weakness, and difficulty in walking.

Duration.—Two years.

Condition on Admission:—

Circulatory System.—normal.

Respiratory System.—Weak movements, expansion poor and abdominal, diminished breath sounds.

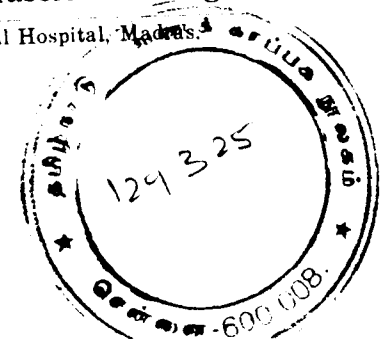
Nervous System.—Average intelligence, no mental disturbance, cranial nerves function normally, superficial reflexes present. Deep reflexes (knee jerk, ankle clonus, and ankle jerk) absent. Romberg's sign, Babinski's sign and A. R. pupil, not present. Vesical and rectal sphincters normal. No tremors.

Muscular System.—Facial muscles not involved. Wasting of:—

(1) Upper extremity—scapular muscles and pectoralis major.

(2) Lower extremity—Gluteal, thigh and leg muscles. Grips are equal and good on both sides; no fibrillary twitching; co-ordination is good; no spasticity or rigidity, no increase in volume; movements are weak and this weakness is limited to the affected muscles. The gait

* A clinical lecture delivered at the Government General Hospital, Madras.



is waddling. He gets up from the squatting position by climbing up his legs (see photographs).

Special Examinations.—

Wassermann.—negative.

Blood pressure.—150—110 m. m.

Electrical Reactions.—Response in muscles is sluggish, requires stronger stimulus and is normal in type. Reaction of degeneration is absent.

Pathological Report:—A small portion of one of the affected muscles, with the nerve leading to it, was excised and examined. Vacuolation of the muscle fibres was seen. There were no changes in the nerves.

History.—Patient has no alcoholic or venereal history. The weakness was noticed two years ago, first in the legs with wasting of thighs and buttocks and has since been progressive. The wasting has become more pronounced during the last six months. The wasting of the shoulder muscles was also noticed during that period. He has had no acute illness. His father's sister was unable to walk and was crawling at 25 years of age.

Points to be noted:

1. Familial history (father's sister).
2. Slow insidious onset, in localised groups and progressive in nature.
3. Absence of acute illness.
4. Electrical reactions.
5. Age of onset.

Diagnosis.—Erb's Juvenile type of Idiopathic Muscular Atrophy.

Muscular wasting occurs in—

1. Disorders due to a lesion of the lower motor neuron, i. e., the peripheral nerves or their cells of origin in the anterior horn of the spinal cord and cranial nerve

Idiopathic Muscular Atrophy

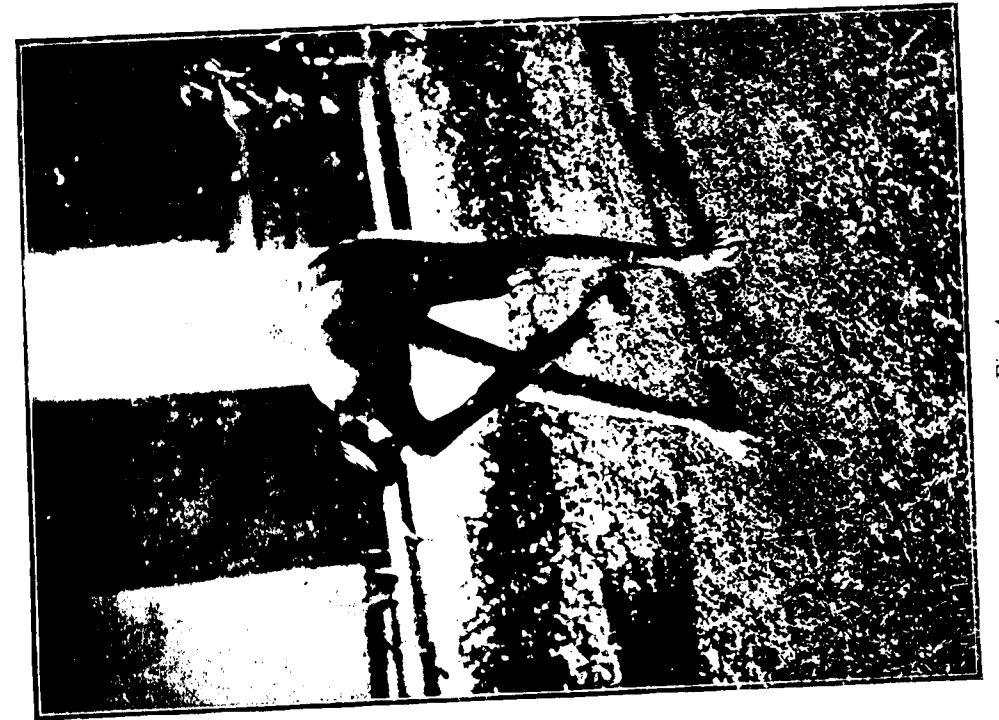


Fig. 3.
Photographs representing the patient climbing up his knees in the act of getting up.

Photos by Capt. Barnard, X-Ray Institute, Madras.

nuclei; in which case it is called atrophic muscular paralysis, amyotrophic paralysis or amyotrophy.

2. Those due to a lesion of the muscle itself or rather where no other lesion is discoverable; in which case one speaks of it as idiopathic muscular atrophy, myopathy or muscular dystrophy.

Aetiology

Under the name 'myopathy' is comprised a group of cases which, though for some time confused with atrophies secondary to spinal cord lesions are now considered to be due, primarily, to changes in the muscle itself, no changes in the nervous or other systems which could explain these muscle changes being present.

The disease begins in infancy though it might become apparent, in some cases, only in later years. It is more common among males than females, especially the pseudo-hypertrophic variety. Heredity is an important factor. A familial tendency is also seen. Sometimes one generation is skipped over, the disease manifesting itself in the offspring of a healthy father or mother of the original stock, transmission thus taking place through males or females. In most instances a neuropathic taint in the ancestry is observed. One makes use of syphilis and alcohol as contributory or accessory causes to explain all such obscure conditions but these factors have not been found to have any effect whatsoever in the aetiology of this disease. Though the changes do not manifest themselves till sometime after birth, it is essentially a congenital defect and well merits the name "Abiotrophy" suggested by Gowers, indicating a defective vital endurance. As usual in such little understood diseases the endocrine system has also been called upon to supply the explanation. Some justification there seems to be in

dragging the endocrines in, as X-ray photos have shown pineal shadows and alterations in the size and contour of the sella turcica and clinoid processes in some of these cases. It is considered to be due to pluriglandular disfunctioning. Another theory is that it is due to changes in the sympathetic system leading thereby to inadequacy of vascular supply and consequent degeneration of the muscle fibres. Whether these vascular changes are of primary sympathetic origin leading to inadequacy of blood supply and consequent muscular degeneration is a doubtful factor. The general consensus of opinion is that the lesion is primarily muscular, the vascular changes being purely secondary.

Early cases show swelling of some of the muscular fibres with increase of sarcolemma nuclei. They become more rounded and hyaline with less well-marked striations. This is ascribed to the swelling of the sarcoplasm. Later on, an increase of interstitial connective tissue is found with a tendency to fat deposit. Small muscle fibres, supposed to be due to splitting up of hypertrophied fibres, are seen in numbers. These are called sarcostyles. Hyaline degeneration, vacuolation of the muscular fibres as well as retrogressive changes leading to connective tissue formation are present, especially near the tendons. In the advanced stages very few muscular fibres are seen. They are of varying sizes and are sparsely distributed in the connective tissue. The muscle spindles (nervous tissue) are found intact even in the last stages. Intramuscular blood vessels show thickening of walls and narrowing of their lumen by connective tissue proliferation around them. Thrombosis has been noted as well as adventitious lymph spaces filled with newly formed cells. Certain obscure changes in the sympathetic system have also been described.

On autopsy, the muscles show a varying appearance. Some are atrophied, some look hypertrophied. The two conditions may exist side by side in the same muscle. Certain muscles might have completely disappeared. The affected muscles vary in colour from pale pink to ruddy red with white bands of connective tissue. These together with fat, may even replace the normal muscle. Calcification of the pineal body has been noted. The bony structures sometimes show rarefaction.

Symptoms

The disease begins insidiously. Weakness, clumsiness and wasting of the muscle are the main subjective symptoms. The wasting may begin in the arms, legs, trunk and occasionally in the face. In one type a pseudo-hypertrophy is noted. The patient assumes unusual and typical attitudes depending on the muscle or groups of muscles involved. A frog like attitude, the angel type with marked projection of the shoulder blades giving a winglike appearance, an abnormally erect position with kyphosis or a hercules type with abnormal size of the lower extremity are all seen. The condition gradually progresses with gradual involvement of other muscles till he is reduced to a semi-helpless state but rarely to one of complete paralysis. The disease runs a slow course, the patient generally dying of some intercurrent disease, as a result of the paralysis of the diaphragm and the attendant complications, or asthenia. Cranial nerves are not involved. There are no sensory changes. Deep reflexes gradually disappear. The motor power is lost or considerably diminished. The electrical irritability of the muscle is diminished and requires stronger stimulus to provoke a response, which however is of a normal character. There is no reaction of de-

generation. The varieties of the myopathies, the appearance of the patient and the associated symptoms like the localised wasting, inability to perform movements, depend entirely on the age of onset, the particular groups of muscles involved, the stage of the disease, and the presence or otherwise of atrophy or pseudo-hypertrophy. The hypertrophic condition is, on examination, found to be mainly one of increase of size with not only an absence of proportional increase of muscle tissue and of the power behind the muscle. It is practically a bloating up of the area with fat and connective tissue replacement of the muscle. A hercules like appearance is nothing but a sham and bluff having no muscle force behind.

Varieties

The main varieties of myopathy are classified according to (1) age of onset (2) change in volume. Whatever might be the method adopted, the main clinical conditions enable any one variety to be transferred from (1) to (2) without any difficulty. These varieties are associated with the names of the particular observers who have described the particular varieties and do not justify, except for purposes of confusion, any distinct claim for separate existence. All these various forms may be seen in members of the same family. The various types are—

(A) With loss of volume

1. Duchenne's infantile type
2. Erb's juvenile type.
3. Landouzy-Dejerine's Fascio-scapulo-humeral type.

Most of these cases begin and predominate in the biceps, triceps, supinator longus, shoulder muscles, teres major, lower two thirds of the pectoralis major. The hands are rarely affected. In the Landouzy-Dejerine type, the

Idiopathic Muscular Atrophy

face is first attacked; both sexes are equally affected; the naso-labial fold is lost; lower lip is projected; a peculiar dull expression is present; and a history of inability to close the eyelids from early childhood may be elicited. Erb's and Duchenne's types are self explanatory, depending purely on the age of onset—either in infancy or adolescence.

(B) With increase of volume

Pseudo-hypertrophic muscular paralysis (Duchenne.)

In this type there is an apparent increased volume of muscle due to interstitial filling up. The main features are that the patient is invariably a boy, between four and fourteen years of age; lordosis is present; he walks with a peculiar waddle and is unable to get up except by climbing up his legs. Knee jerks are absent. The muscles especially of the calves and buttocks, supra- and infra-spinati, are prominent and increased in volume. The lower two-thirds of the pectoralis major, teres major, latissimus dorsi, are apt to be diminished. Weakness of the shoulder girdles with slipping up of the shoulder is found.

(C) Associated forms

I. The Peroneal type, (Charcot-Marrie-Tooth) is a limb between muscular dystrophy and progressive muscular atrophy. It is hereditary, manifests itself in childhood, and shows a gradual onset. Diminished electrical excitability is present. It begins generally in the peroneal muscles but may also begin in the hands. Gradually wasting spreads to all the muscles. Sometimes, sensory loss is present. Fibrillary twitchings are seen. Reaction of degeneration is present. The last three points show the difference of this condition from muscular dystrophy. If without these, it is classified as the distal type of muscular dystrophy (Gowers).

2. Amyotonia congenita.

Here a congenital loss of muscle is present. It has no familial character. There is extreme flaccidity, lowered faradic excitability, and absence of deep reflexes as in the myopathies. Paralysis is not present. The face is usually exempt. The sphincters are unaffected. The limbs are slender, musculature is soft, the hands and feet long and delicate. It is accounted as a disease of muscles in intra-uterine life. Muscles are yellowish and fat. The most obvious change is the small size of the fibres 7-8 μ in length, with occasional giant fibres (140 μ) central vacuolation and a tendency to split longitudinally is present. Extreme thinness of ventral nerve roots, diminution in number of motor cells in ventral horns, paucity of nuclein and fibres in the roots are found.

3. Family Periodic Paralysis.

Familial in type; affects both sexes equally; is transmitted through father or mother and observed generally after six years of age. The attacks are irregular and affect chiefly the proximal muscles of the lower limb. The trunk and neck are affected in severe cases. During the attack muscular response diminishes progressively and equally to all stimuli (volitional, reflex, galvanic, faradic and mechanical). No sensory changes are present. There is no spasticity and attacks pass off leaving no impairment. Before the attack arterial tension is increased. Vomiting shortens, and careful diet eliminates the attack. Urine before and after the attack has been reported to be toxic. There is absence of electrical response. Muscle in section shows association with lymph stasis, œdema, and toxic accumulation around muscular fibres.

Diagnosis

It is very hard to make a mistake once a typical case has been seen. The gradual and insidious onset, the history, diminished electrical response, absence of R. D., absence of sensory changes, localisation of the muscles affected in early cases are characteristic enough to warrant a diagnosis. The characteristic method of climbing up himself is a pathognomonic feature. Differential diagnosis has to be made from—

1. Anterior poliomyelitis—note here the electrical changes and the presence of R. D.

2. Neuritic muscular atrophy—sensory changes are present here.

3. Progressive muscular atrophy—begins in the hands; occurs later in age; fibrillary twitchings are present.

4. Myotonia atrophica—note the prolonged response to stimulus; the atrophy of the sternomastoids.

In an examination of suspected myopathy (A) do not confuse with (1) wasting due to illness, (2) upper motor neuron lesions with later wasting of muscles; (3) disuse atrophy, (4) arthritic atrophy (Charcot) which is found after joint lesions even after a slight contusion. It is limited to the muscles which move the joint, predominates in extensors, do not show any qualitative electrical changes and improve rapidly with proper measures when the joint trouble is removed. The disease is too rapid to be explained by disuse.

(B) Investigate (1) family history, (2) age and sex of patient, (3) method and time of onset, (4) flaccidity of paralysis, (5) absence of knee jerks, (6) sensory changes, (7) distribution (8) presence or absence of hypertrophy, (9) movements, and (10) electrical reactions.

Prognosis

Unfavourable. The disease runs a long course and death results from exhaustion, involvement of respiratory muscles, or intercurrent diseases.

Treatment

Electrical massage and gentle exercise seem to delay the progress of the disease. Endocrine therapy may also be tried. It has been noted that rapid decline begins once the patient takes to his bed, so that every effort must be made to keep up his spirits and continue his exercises.

Abortion

BY DR. M. B. PRABHU, M. B. B. S.

Govt. Hospital for Women and Children, Madras

I am writing a few lines on this subject not because there will be anything new in this paper which cannot be found in the several text-books of midwifery but just to focus the attention of the senior students on some of its aspects.

Abortion is one of the forms of haemorrhages during pregnancy. Of all the complications during pregnancy this is the commonest. Most of the women who had several child-births have had one or more abortions. If you go through the waiting cases in the labour ward of this hospital, the largest proportion have come in for labour pains at full term and the next largest for bleeding before term which in a very large number of cases is due to abortion, threatened or inevitable. It has been estimated that nearly 20 per cent. of pregnancies terminate prematurely.

No distinction has been made here between abortion and miscarriage as both really mean the same, viz., expulsion of the ovum before the child is viable.

Causes.—These are generally classified as maternal and foetal (including its appendages).

Among the maternal causes there may be mentioned constitutional disorders like syphilis, tubercle, lead poisoning, chronic nephritis and the toxæmias of pregnancy, diseases of the blood like anaemia, leukemia and purpura, toxins of fevers like influenza, pneumonia, measles, chicken pox and malarial fever, and local causes like dis-

placements of the uterus, chronic endometritis, pressure of growths and trauma. Nervous causes may facilitate abortion. This may be due to increased irritability in the nerve centres or in the nerves supplying the viscera.

As regards foetal causes, they are imperfect fertility in either one or both the parents resulting in failure of development of the foetus. Diseases of the foetus causing its death result in abortion as the dead foetus acts as a foreign body and sets up by its irritation reflex contractions of the uterus. Diseases of the chorion, amnion and placenta facilitate the separation of the ovum and may also interfere with its nutrition thereby causing its death. Clinically it is not possible to diagnose the disease of the foetus and its appendages; hence it is not possible to forecast whether a pregnancy will terminate prematurely or not.

As regards the maternal causes, clinically syphilis ranks first. The virus of syphilis causes infarcts and early degeneration of the placenta, chorion and amnion, and interferes with the nutrition of the ovum, thereby causing its death in a number of cases. As regards lead-poisoning it is rare in this country. Regarding blood diseases abortion is not so common as one is led to imagine. Cases of severe anaemia in pregnancy have been admitted into the hospital and abortion has not been common except in the most severe types and that too especially when the fatal end was nearing. Though fevers like pneumonia, enteric, influenza, and malaria are possible causes, pregnant women do in large number of cases successfully go to full term.

In small pox complicating pregnancy, abortion is the rule rather than the exception. The virus affects the foetus profoundly. In some cases the eruption is noticeable in the foetus also.

Regarding displacements of the uterus retroflexion

is the most common and even here the growing ovum tends to lift up the uterus from its retroflexed position. Where there is interference with this spontaneous rectification, abortion will result. Considering the nervous causes, one finds that in hysteria and neurasthenia though there is altered irritability of the nervous system, there is no tendency towards abortion. Emotion, and fright may be possible causes but very rarely bring about abortion. Mechanical action as continuous jolting in a bullock-bandy are frequently common in our village parts.

Lastly, it is important to remember that the degree of attachment that naturally exists between the ovum and the mother is not the same in all cases. Just as in fruit trees, in some the stalk of the fruit is held less firmly than in others, so that some fruits fall off more readily in a stormy breeze, so also is the attachment of the human embryo to its resting place.

Clinically we do not know of any method of measuring this degree of closeness and in a certain number of cases of abortion we may find nothing abnormal with the mother or the foetus. I doubt if in this country marriage of girls in their 13th or 14th year has anything to do with abortion in their early married life. The uterus is not sufficiently developed to adapt itself to the changing conditions of pregnancy and added to that the pelvic congestion caused by excessive sexual intercourse may be a factor in its production.

Signs and symptoms.—Haemorrhage in a married woman after a period of amenorrhoea makes one think of abortion. Together with this there is a certain amount of discomfort in the abdomen which in a few cases may amount to actual pain; constitutional disturbance is generally slight. It is important to remember that haemorrhage in the early weeks of pregnancy may also be

due to the relatively uncommon but far more serious condition, an extra-uterine pregnancy. It is also necessary to remember that haemorrhage may not be due to pregnancy at all but to some associated gynaecological condition.

Treatment.—The proper treatment is to treat the cause. But when a pregnant woman comes to you with a history of bleeding, you have to treat the haemorrhage first and then the constitutional condition afterwards. The usual answer to the question, what do you do when a pregnant woman is bleeding is, rest in bed. Injection of $\frac{1}{4}$ gr. morphia and administration of sedatives like bromides. But before doing anything by way of treatment it is very necessary to find out if the abortion is threatened or inevitable; because in the former the line of treatment is to allay the uterine contractions; whereas in the inevitable, you have to assist the uterus to expel its contents. If the case is inevitable and you put the patient on morphia etc., you are only interfering with the natural process. Hence, first decide if it is threatened or inevitable; the points of distinction being:—it is inevitable if there is (1) rupture of the membranes or (2) dilation of the os, more than one finger (3) actual protrusion of the ovum into the cervical canal (4) offensive discharge from the uterus conclusively showing that the foetus has been dead for some time and (5) severe haemorrhage. In severe haemorrhage though it is likely that the ovum has been disorganised by bleeding there are cases on record where the woman had alarming haemorrhage but pregnancy proceeded to full term. Repeated, profuse haemorrhage is a more probable sign of inevitable abortion. Therefore before deciding on any line of treatment you have to make a vaginal examination and find out if the abortion is threatened or inevitable.

In the threatened, you try to control the uterine

action and stop bleeding by rest in bed, injection of morphia, administration of bromides, calcium lactate, etc. If inevitable, what is the line of treatment? Abortion is after all a miniature labour and do not interfere in a case because it is inevitable. Leave the ovum to be expelled and the best result for the mother will be a spontaneous evacuation of the uterine contents. Interference is justifiable in the interest of the mother,

1. When there is severe haemorrhage.
 2. When the pains are sluggish and there is no progress in expulsion.
 3. When it is incomplete.
 4. When there are signs of sepsis.
1. In case of severe hæmorrhage, give a hot vaginal douche, tightly pack the vagina, give a tight abdominal binder, and administer 1 cc. pituitrin, treat for collapse. Very rarely it may be necessary to carry out forcible dilatation and evacuate the contents.
 2. When the pains are sluggish, give hot vaginal douches 2nd hourly or 4th hourly, a quinine draught (15 gr. to 1 oz) or $\frac{1}{2}$ cc. pituitrin repeated if necessary.
 3. When incomplete, manual removal is the best instead of with the aid of instruments. Anaesthesia is advisable. Taking aseptic precautions put your fore finger inside the uterus and evacuate, bringing down the fundus within your reach by pressure from above. In cases of amenorrhoea of 4 months and upwards, if the fundus cannot be reached the ovum forceps has to be used and in such cases the cervix has to be dilated sufficiently to allow the foetal head to come through. It may even be necessary to perforate the head before extracting it.
 4. What is to be done in cases of sepsis? One is to leave the case alone by prescribing ergot and hot douches without interfering with the uterus lest the

septic process should flare up. This is what the American School used to advocate until recently.

The other is to take active measures and get rid of the septic contents. The line of treatment advocated in our hospital is to get rid of the septic contents as soon as possible rather than to leave it to an uncertain future. The uterus during the early months of pregnancy has not such enlarged blood-vessels and sinuses as at full term and evacuation of the septic contents brings down the temperature and there is noticed an immediate improvement in the general condition.

How to evacuate.—Bring the patient to the table and give a hot vaginal douche. Then put in a gloved finger. For the beginner, the bare fore finger is better because he has a better sense of touch without gloves. Then remove the contents manually. Even in cases where the ovum forceps has been used to remove the ovum, digital exploration is essential lest any piece of the ovum has been left behind. After removing the contents with the finger, take the blunt flushing curette. There is no need to curette out the uterine mucosa, but you simply stroke the interior and sweep out the debris. In a few cases where the discharge has been very offensive, I swab the interior of the uterus with an antiseptic like Tr. Iodine. The patient recovers after this line of treatment in a remarkably satisfactory manner.

After treating for the actual condition, find out the possible causes and then rectify them. An abortion is a strain on the health of the mother without the compensation of being blest with a baby, and frequent abortion drains her health and draws upon her nervous system. Hence all lines of treatment, local as well as constitutional, should be explored to prevent the recurrence of abortions in future conceptions.

I have to thank our Superintendent Lt. Col. Hingston, I.M.S., for having given me permission to publish this article.

Infantile Convulsions

BY R. K. CHETTUR

Final Year

The occurrence of convulsions in infancy is dangerously frequent, and whether the convulsions are of a general nature affecting the whole body, as they more often are, or whether the convulsive disorder manifests itself in any of the local forms, it is to be regarded as a condition attended with danger, as the child runs the risk not only of its life, but also in later life from disturbances of its mental equilibrium. The development of the higher functions of the nervous system is not complete at birth, as a result of which the lower centres are not subjected to proper control by the higher ones, and further the nervous mechanism as a whole has not acquired that stability which gradually begins to assert itself only as the child grows. This is substantiated by the fact that as the infant becomes older there is a marked tendency for the convulsions to occur less frequently and finally cease altogether, except in those not unusual cases where there is an inherited neurotic tendency, or where the convulsive disorder occasionally continues and ultimately passes into true epilepsy. Predisposed, therefore, as a child is naturally to convulsions, it is at once obvious that a child whose general resistance has been lowered by some constitutional affection, or whose nervous mechanism has been rendered more susceptible to even peripheral stimuli, is still more liable to convulsive disorders.

Etiology

I. Lesions of the gastro-intestinal canal are considered by some to be the most important factor in the causation of

this condition. Indigestion due to an unsuitable dietary, the presence of intestinal worms as *Ascaris Lumbricoïdes*, and constipation, are regarded as being potent causes, especially the latter. It is to obviate recurrence of the convulsions from intestinal toxæmia due to constipation that the administration, immediately the fit is brought under control, of a dose of castor oil or calomel is so strongly advocated. Convulsions sometimes follow the severe exhaustion resulting from protracted diarrhœa.

2. Rickets is considered by others the most important factor in the production of this disorder, as rachitic children have been found to be particularly liable to infantile convulsions. In *Laryngismus stridulus*, one of the local varieties of convulsion, there is evidence in the majority of cases (75%) of rickets. With most of the systems thrown out of gear in this constitutional affection, and with the increased irritability of the nervous system, the fits are often induced by what might be considered in a healthy child as a trivial source of irritation. For instance, the process of dentition has been known very often to excite convulsions; but dentition *per se* can hardly be considered a cause since it is only effective as one of several factors in weak and ailing children.

3. In infancy the onset of acute diseases, as measles or pneumonia, is often accompanied by convulsions. They apparently take the place of rigors in the adult.

4. Venous congestion of the brain, such as occurs during a paroxysm of whooping cough, may produce convulsions, but how far mere mechanical congestion is responsible for the fits is difficult to estimate with any degree of accuracy. Other conditions of asphyxia as in the paroxysms of laryngismus stridulus or in even a violent fit of crying, have also been known to end in convulsions. There is the authenticated case of a boy in whom the

attacks were brought on whenever he cried from any cause (Still). On being examined the child was found quite healthy but resenting the thorough examination to which it was subjected it burst out into tears, "went bluer and bluer, and then a livid leaden colour, and lost consciousness. Gradually he revived," but the unwise attempt to look into his mouth unfortunately induced another fit of crying, "the child again became unconscious, and was in imminent danger of his life."

5. In some, the convulsions are to be regarded as epileptic, since epilepsy has been known to occur in infancy. It is true that only a small number of children who have had infantile convulsions ultimately become epileptic, but the fact remains that according to Osler, "of 460 cases of epilepsy in children, in 187 the fits began in the first three years of life, and the greatest number 74 was in the first year of life."

6. More rarely, grave lesions of the nervous system in infants are either accompanied by or marked in their onset by convulsions, the commonest of these being the acute encephalitis of children and meningitis, either simple or tuberculous. A few inconspicuous attacks of convulsions, increasing in severity as the child becomes older may be the first sign in some cases, of mental deficiency. While mental defect is acknowledged to be more often the cause rather than the result of convulsions, and statistics prove that at least 22 to 28% of idiots have had convulsions in infancy, thereby substantiating the association of convulsions with mental deficiency, there is, on the other hand, the case of the girl aged 8½ years who remained perfectly normal up to the age of two years and eleven months. Then in one day she had ten fits, and later, often had eight or nine a day. Since then "she has ceased to

talk, and has become imbecile; her tongue lolls out, and she takes little notice of her surroundings."

7. Convulsions are frequently seen in children who are the victims of congenital syphilis.

8. As one of the rarer causative factors might be mentioned uræmia, for uræmic convulsions have been known to occur.

9. Sometimes the attacks start immediately after birth and have lasted for weeks or months. There is, then, a history of difficult labour with instrumental interference, or of a fall resulting in a meningeal hæmorrhage or some serious injury to the cortex. A child aged eleven months fell head foremost to the ground, and as a direct result, it had repeated attacks of severe convulsions, and suspecting a hæmorrhage the skull was trephined and the clot removed. The convulsions stopped and the child recovered completely.

Symptoms

Where the convulsions affect the whole body, the resemblance to a true epileptic fit is very close. The attack may be ushered in by prodromal symptoms as grinding of the teeth, twitchings of the face or a general restlessness. On the other hand the onset has been known to be sudden without any previous warning whatever. This initial stage is followed by (1) tonic contraction with loss of consciousness, (2) clonic convulsion, and (3) the stage of recovery.

Recovery is only likely to be complete when there is one attack at a time with recurrence weeks or months later, for in those cases where the fits are repeated in rapid succession the probabilities are that recovery will never be complete and consciousness be not regained at all between the fits, a condition which when it occurs is comparable to the *status epilepticus* of the adult.

In the tonic stage the eyes are fixed and staring, and may be turned to one or other side. The back is stiff, the limbs being completely extended usually. In the general tonic contraction the chest becomes fixed, and as a result of the temporary cessation of respiration there is lividity of the face. But just as the respiratory distress reaches its acme the tonic stage is followed by the clonic spasms, the lips and eyelids begin twitching, the voluntary muscles of the body contract, very violently at times, and as the respiration is now no longer hindered the face regains its original colour. The convulsions gradually cease, are followed by the stage of recovery in which consciousness is regained slowly, and leave the patient in an exhausted condition.

The definite length of time that a fit lasts varies, for it may pass off in a few minutes as it more often does, or may last from a few minutes to half an hour or even more. Moreover, it is not always that infantile convulsions are so persistent either in their severity or in their completeness, for many cases have been known where there was only a strabismus or a rigidity of the extremities.

The local affections, to which reference has already been made, are laryngismus stridulus, tetany and the so-called "facial irritability". All the three conditions appear with, and disappear after, the characteristic changes of rickets. In laryngismus stridulus there is spasmodic closure of the glottis, usually induced by some trivial stimulus, and as expiration only is possible the face becomes dangerously cyanosed. Although in most cases inspiration occurs at the critical moment, death has occasionally resulted from a spasmodically closed glottis. The spasm usually lasts from 15 seconds to about 2 minutes. Approximately half the number of

cases of laryngismus stridulus are said to be either preceded, or followed, by general convulsions, a fact which sufficiently emphasises the close relation between the two conditions.

In tetany the characteristic contractions, known otherwise as *carpopedal contractions*, nearly always occur in the upper and lower extremities. The fingers of the hand are so contracted and flexed as to form the well-known "accoucher's hand", while the wrist and elbow joints may be flexed. In the lower extremity the toes are huddled together and the feet are extended. Pain may or may not be associated with the condition. Sometimes the hand and feet become oedematous, and since the veins appear swollen the plausible explanation has been put forward that the oedema might be due to obstruction to venous return owing to prolonged muscular spasm, which may last from either five to ten minutes or from one to even three hours. The variety known as latent tetany may be detected by encircling the arm so as to compress the brachial vessels and nerves when in about half a minute the spasm of tetany may be induced. These carpopedal contractions are seen very commonly in association with laryngismus stridulus, the tendency to general convulsions being more marked in the former.

Facial irritability or *Chvostek's sign* is the condition where the motor nerves and muscles of the face, being more excitable, respond very easily to slight external stimuli, a gentle tap on the facial nerve as it emerges in front of the ear being sufficient to produce a contraction of the facial muscles on that side. Although this is generally known, the fact that the same increase in the degree of excitability is present also in the extremities must be borne in mind.

These three forms of local convulsive disorder are to

Infantile Convulsions

be regarded, when present, as real indications of danger, as general convulsions are only too likely to follow at no distant period. The degree of irritability of the nerves present affords us a gauge by which the patient's liability to convulsions may be estimated, for it is obvious that where a mere stroking of the face (*Schultz's sign*) suffices to induce the twitchings the probabilities of general convulsions supervening are greater than when there is little or no response. While therefore, the irritability is effective as a danger signal, its diminution under the influence of bromides is equally efficacious as an indication of the general progress that is being made under treatment.

Diagnosis

Although the recognition of infantile convulsions does not offer great difficulty, affections which simulate, and might probably be confused with, them will have to be excluded. These conditions are strychnine poisoning, tetanus either general or modified, and hydrophobia.

In *strychnine poisoning*, all the muscles of the body contract very violently at the same time, followed in a minute or two by muscular relaxation. Consciousness is retained all through. Moreover, there is the characteristic position of opisthotonos, etc. Hearing and vision are very acute, even the slightest noise being sufficient to provoke a spasm.

In *tetanus*, which when present in newborn infants is due to infection of the navel, the jaw muscles are invariably affected first, the well-known lock-jaw developing. In the intervals between the spasms the muscles remain rigid from contraction. Consciousness is retained throughout. There is likely to be a history of a wound.

In *hydrophobia*, the spasm is at first clonic and as the

muscles chiefly affected are those of respiration and deglutition, there is the characteristic association of the difficulty in swallowing. There is mental excitement in most cases.

Having excluded these conditions it is necessary to discover the cause of the convulsions, as by efficient treatment of the cause alone is there any likelihood of eliminating the possibility of recurrence of the fits.

Prognosis

Immediate:—In considering the immediate prognosis the three factors that must be taken into consideration are the age of the child, the duration of the fit, and its severity. The younger the child the greater naturally is the danger to life, and statistics show that about 90% of deaths from convulsions occur in infants under a year. In cases where there is only slight twitching or a squint the child does not run any serious risk, but where the fits rapidly recur, where the loss of consciousness is more complete than the severity of the fit warrants, or in those rare cases where the condition approximating to the status epilepticus of the adult is present, the outlook is correspondingly graver. Of the local affections, laryngismus stridulus is the most serious as, apart from indicating a marked tendency to convulsions like its allied conditions, death might at any time result from asphyxia due to spasmodic closure of the glottis.

Ultimate:—It is necessary to exercise great caution in giving the ultimate prognosis as it is not possible in most cases to give a definite answer. Where the question of impairment of the child's intellect arises it must be remembered that idiocy has been known to follow severe bouts of convulsions but that more often they are to be interpreted as an indication of the already existing

abnormal mental condition. Of children who have had convulsions in their infancy only a small number ultimately become epileptic so that it is equally difficult to prognosticate at such an early stage as to whether the child might become epileptic or not in later life. In all cases it is invariably the best plan to be wary and always give a guarded prognosis.

Treatment

Of distress and anxiety to the parents, of great risk to the child, and one allowing no time for cogitation on the part of the physician, this is one of those conditions where prompt and active treatment is imperative. Treatment, therefore, must be directed to (1) relief of the attack, and (2) removal of the cause.

Relief of the Attack:—(i) While more effective and certain means of treating the fit are being got ready, the child should be immersed up to its neck in a hot bath, the temperature to be between 100° and 105° F, for about five minutes. It quiets the child considerably.

(ii) The administration of chloral or bromide, or both together. There are two methods by which these drugs may be administered, and they are orally and rectally. Rectal medication is easier and more rapid in its effects during the convulsions, but where the convulsions have ceased and the drugs have still to be continued to prevent recurrence, the former method is to be recommended.

(a) *Rectal Medication*.—To a child under one year of age 8 grains of sodium bromide and 3 grains of chloral, suspended in two ounces of mucilage of acacia or dissolved in an equal quantity of water may be given, and repeated at interval of two to six hours as indicated by the severity of the condition. A soft rubber

catheter is attached to a syringe containing the suspension or solution, or if a syringe is not available a rectal tube may be made to serve the purpose equally well, and introduced for at least 9 inches to enable the contents to be conveyed to the descending colon, where the probabilities of its being retained are greater than if introduced just within the anus. During the operation the child should rest on its left side, with the buttocks elevated on a pillow so that they are on a higher level than the shoulders, a position which ought to be maintained for a few minutes even after the withdrawal of the catheter to aid in the retention of the suspension.

(b) *Oral Administration.*—For repeated administration by mouth, bromides are better as chloral even though effective, is apt to irritate the gastro-intestinal tract. To an infant one year old 5 grains of the sodium salt may be given in $\frac{1}{2}$ ounce of water every four hours till the convulsions are controlled. For an infant one month old one grain of chloral may be given every two or three hours; for one six months old 3-5 grains, and for a child over twelve months 7-10 grains may be given as often as is considered necessary. Where both chloral and bromides have failed urethane may be tried in doses of 1 grain, three times daily, in infants under a year; 2 grains up to the age of four years; and 3-5 grains in the case of children from 6-10 years.

(iii) Morphia also is useful but better avoided, and if the necessity for its use arises at all it must be injected cautiously in doses of $\frac{1}{50}$ grain hypodermically.

(iv) The inhalation of chloroform is to be recommended where the fits tend to recur rapidly, or are unduly prolonged.

(v) In laryngismus stridulus, which is also a condition calling for immediate treatment, the application of

a hot towel over the larynx, and of ammonia to the nostrils may cut short the attack but where the spasm is prolonged or asphyxia appears imminent nothing short of a tracheotomy will probably save the child.

Removal of the Cause.—In order that prophylaxis might be effective the underlying cause must be dealt with adequately. In rickets dietetic and hygienic treatment must be instituted at once, and in addition bromides must be administered regularly to prevent the recurrence of the attacks. Constipation is the exciting cause very often, and a dose of castor oil or calomel immediately the convulsions have been brought under control, followed by strict regularity in diet, has on several occasions been found to be sufficient by way of treatment. Where the causative factor is congenital syphilis cessation of attacks will follow the adoption of anti-syphilitic measures, while for those cases sometimes classed as "idiopathic" the administration of chloral or other sedative is the only line of treatment that remains to be adopted. The advisability of operative treatment may be considered where, after a history of difficult labour or trauma immediately after birth, a meningeal haemorrhage is suspected to be the cause, but when the haemorrhage shows signs of being extensive operation will probably be of no avail.

Case Report

A Case of Human Meischeria (Sarcosporidia).

I have to thank the Professor of Pathology, Medical College, Madras, and the Third Surgeon, General Hospital, Madras, for permission to publish the notes of this very interesting case.

A Hindu male of 55 years, without occupation, was admitted to the Third Surgeon's wards giving a history of an abscess of the chest wall, on the left side, which started as a small pustule and gradually enlarged to about 2 inches in diameter. The patient has had no previous illness, is fairly well nourished, with clean tongue and teeth; the duration of the present one was only 15 days. Before operation, it was observed, that the edges of the ulcer were indurated, the base fungating, adherent to the muscles and presented sieve-like openings all over; Wassermann reaction was negative and the urine presented no abnormality. The ulcer was provisionally diagnosed to be carbuncle chest and excised under gas and later, skin was grafted on. The temperature remained normal all through the 29 days he was an in-patient. The excised tissue was examined and reported on as Sarcocystis.

Tissue.—Macroscopic appearances:—Just over the nipple, there is an irregular excavated ulcerous area roughly $1\frac{1}{2}$ by 1 inch, with undermined edges and pieces of granulating tissue projecting from the sides and the base of the ulcer. The margins are thick, rounded, slightly inverted, dense and sclerotic. On section, the ulcer extends only to a portion of the sub-cutaneous fatty layer, the underlying areolar tissue is thickened, and laminated. The pectoral muscle fibres show pin point white specks and thin lines.

Section of a piece under the microscope:—Numerous tubes of sarcocysts invading the muscle fibres are seen. Each tube is very long and from the tissue received the entire length of the tube could not be discovered and dissected out. Dissection in the fresh state was missed as sarcocystis was not suspected. However, the preserved tissue was dissected out, examined and the following findings were noted. The cysts containing the sporozoites were all not uniform in size or shape. Some were large and mature with septa well developed, while others were immature with ill defined septa. The average dimensions of the mature cysts are—length unknown (in one case a portion measured $3\frac{3}{4}$ c. m.) breadth 195 to 240 microns (average of readings from 12 cysts). In the mature cysts, there are on the average as many as 64 septa, which unite in the central area as a well defined honey-comb like structure. Between the septa, the sporozoites are packed full, whereas in the central honey-comb like area, only a few could be seen. The shape differs according to the inclination of section and the age of the cyst; the younger ones appearing nearly round, the mature ones, oval or sausage shaped. There is a definite hyaline slightly pink staining (with eosin) non-granular, non-laminated, non-striated cyst wall of 3.5 microns thick all round. The cyst is well seen to be in the muscle substance, and the muscle fibrils are sprayed out overlapping the cyst at the ends. Two cyst cavities present peculiar appearances. In the one, there is a ring of large eosinophilic, non-granular, mononuclear cells with abundant protoplasm, all round both on the inner side and on the outside of the cyst wall; a few of these mononuclears are seen within the cyst cavity, amidst a certain quantity of granular debris and destroyed cyst walls. There are a few polymorphonuclears to be seen outside and inside

the cyst cavity. Some of these cells are in the actual process of migration into the cyst cavity. The other cyst cavity is packed full of the eosinophile non-granular mono-nuclears and a few polymorphs. No sporozoites are visible either inside or outside the cyst.

The tubes were then dissected out, the spores were isolated, and examined, unstained and stained; their average dimensions are :—length 9.7 to 12.7 microns, breadth 2.9 to 4.4 microns. Each sporozoite is sickle shaped with one sharp thin extremity and the other somewhat rounded. The nucleus, which is oval with its long axis parallel to the length of the spore, is at a distance of a fourth of the length of the sporozoite from the thin end. At the broader end, a deeply chromatin staining spot (Leishman stain) is visible. Besides, in every spore one or two (in most two) ruby red small chromatin granules are visible between the deep-stained spot at the broad end and the nucleus. The rest of the protoplasm is very lightly staining pink. The size of the nucleus is 3.6 by 2.0 microns. No spore wall to be made out. All the sporozoites are not of the same size; some are plump and some are lean, and some are short, but the majority conform to the same shape and size. The plump ones assume an elongated lance shape.

The interest centering about this case is the rarity of human sarcocystis and the identification of the species discovered. According to Stit (till 1923) only three authentic cases have been reported. In one case, they were found in the biceps, in another in the laryngeal muscles. According to Fantham (1916) six cases are reported.

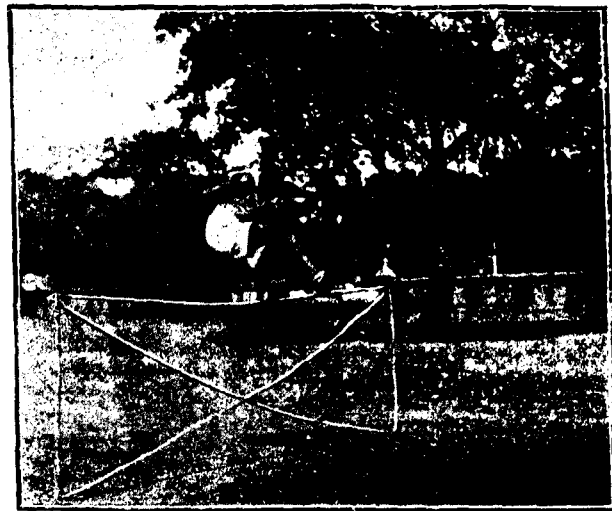
I am unable at present to proceed further with the working out of the details and obtain information of the species.

A. SRINIVASULU NAIDU, B.A., L.M.S.

The Madras Medical College Sports



[Photo by G. Thimma Reddi.]
The arrangements were excellent this year and with the "B" Coy. men on duty, left nothing to be desired.



[Photo by Dr. A. St. C. Bartley, I.M.D.]



[Photo by G. Madhusudanan.]

T. M. John (3rd year) won the Championship in the College Sports, securing the first place in the 10 yards dash, the 120 yards Hurdles, Long Jump, High Jump and 220 yards Dash.



[Photo by Dr. A. St. C. Bartley, I.M.D.]

The Sixth Annual Meeting

THE MADRAS MEDICAL COLLEGE ASSOCIATION

The sixth annual meeting of the Association was held on Wednesday the 9th March, 1927, at 1 P. M. in the Hygiene Hall, with Major K. G. Pandalai, F.R.C.S., I.M.S. in the Chair.

The Chairman, after a short introductory speech, called upon the Secretary to read out his Annual Report, for the year 1926-27. It was passed unanimously.

The next and last item in the agenda was the business of nominating the candidates for the Secretaryship for the year 1927-1928, the following being duly proposed and seconded:

Mr. P. Marthandam Pillai	IV Year
„ A. S. Selvaraj	IV Year
„ S. Hanumantha Rao	IV Year
„ A. J. Eapen *	IV Year

In his concluding speech, the Chairman briefly reviewed the working of the Association, pointing out that the year had been a very successful one. The Magazine was not only superior to college journals but had found a respectable place among the journals of India. There had been complaints received that the Journal was not sufficiently scientific, but would the students remember that the College Journal was meant more to reflect the life of the students than to compete with either the British Medical Journal or the Lancet? Concluding, the Chairman thanked, on behalf of the members, the out-going Secretary, his vote of thanks being repended to heartily by the members.

With the vote of thanks to the Chairman, the meeting came to a close.

The result of the polling on 10th March 1927, (time 11 A. M. to 4 P. M.).—

Mr. A. S. Selvaraj	... 91 votes
„ P. Marthandam Pillai	... 125 „
„ S. Hanumantha Rao	... 185 „
Invalid votes	... 9 „

Mr. S. Hanumantha Rao was duly declared elected.

* Mr. A. Eapen withdrew his candidature after the meeting was over.

Sixth Annual Report

MR. PRESIDENT, LADIES, AND GENTLEMEN,

It was the custom in former years to express great pleasure when submitting Annual Reports, but coming as this does after the unprecedented, unexpected and unhappy ending of the last meeting, it is only natural that what might otherwise have been pleasure should be replaced by an inexpressible feeling of intense relief. As annual reports with their dull figures and even duller statements of facts which everybody knows, inevitably create that ineffable atmosphere of boredom, I may be pardoned for referring you for minute details to the March issue of the College Magazine, for I intend confining myself here to a few general remarks on the working of the Association, on the publication of the Magazine, and end off with a general survey of the finances of both.

The first General Body Meeting was held on the 28th of July 1926, for the election of office-bearers and as you all know the details there is no necessity to go into them now. The one feature however, that must be recorded is the election of the Assistant Secretary, which was conducted on lines similar to that of the Secretary's.

The Graduates' Reception

The Graduates' Reception which was held on the 20th of August 1926, has been admitted by all, except of course, by that not unusual number of dissentients, as having been very much better than in previous years. Several radical changes were made in the arrangements, which, judging by the appreciation expressed by the graduates of the year, other guests, and the majority of the students, I am inclined to think were certainly for the better.

Association Meetings

Apart from the couple of Committee meetings that were held to discuss the rules and bye-laws of the Association, and the details of the Graduates' Reception, there were eight meetings in the year under review, including the inaugural address delivered by Capt. P. N. Basu I.M.S., on the 9th September, 1926. I have appended the list of the lecturers, the subjects and the respective presidents at the end, but there were two meetings that deserve special mention. The Association had the very rare privilege of hearing Sir P. C. Ray speak on "Ancient Hindu Chemistry", on the 1st of December 1926, under its auspices, with Sir R. Venkataratnam Naidu, the Vice-Chancellor of the University, in the Chair. This meeting was thrown open to the public, and even though

Annual Meeting

the lecture was delivered in the Library, the accommodation was so limited that several people went away disappointed without hearing or seeing anything. The other lecture to which I referred was the one delivered by Dr. A. Lakshmanaswami Mudaliar, B.A., M.D., on the "Conquest of Disease," on the 26th of January 1927, for the remarkable feature of the lecture was, that, although there was nothing exceptional in the speaker's command of the language, in that choiceness of diction, and in that rapidity of delivery, a College student, Mr. R. V. Chockalingam, (IIIrd year), presided.

The College Magazine

The fair degree of regularity with which the last three issues of the Magazine have been published is by no means a true index of either the regularity or the promptness of its contributors, for I have experienced very great difficulty in obtaining contributions and it is to this chiefly that delay in the publication of the first issue was due.

A great effort has been made to improve the get-up of the journal, and it can be said without flattering ourselves, that there are very few College Magazines at the present day that compare favourably with ours either from the aesthetic or from the utilitarian points of view. The Magazine has been very favourably reviewed by the Press and have congratulated the Editor on an excellent production. The fourth issue is in the Press, and, I expect, will be out shortly.

Finances

There has been no difficulty at all with the finances this year. By subscriptions we collected a sum of Rs. 491 for the Association, and in this connection I should like to state that while there are 516 students on the rolls of the College, including the BSSc. students etc., 25 of the students have dodged payment. How they managed to achieve this feat is still a mystery, but the fault I am inclined to think, lies with the several Asst. Profs. in charge of the rolls who must have entered the names of the guilty students on the registers without insisting on the production of the required receipts. The major portion of the sum (Rs. 455-12-0) has been literally swallowed up by the graduates' Reception, but the success of the entertainment has more than warranted the increased expenditure. The arrangements for Sir P. C. Ray's lecture, and the printing of the Receipt books have increased the expenditure, and leaves us a not negligible balance of Rs. 3-4-0 (cash on hand). The Association "Rules and bye-laws" have been printed but I have not received the bill for it.

Magazine Fund

On 1st March 1926, there was a sum of Rs. 1730-5-1 in the Bank. By subscriptions we realized, from the students, mofussil subscribers and staff, a sum of Rs. 1106-0-0; by sale of old copies Rs. 25-0-0; by advertisements a sum of Rs. 385-1-7. As might have been noticed the advertisements have now become a paying concern and the money collected is nearly thrice what was obtained in the previous year, Rs. 139-0-0. We have yet to collect a sum of Rs. 170-0-0, also from advertisements from firms in America.

The cost of printing the Magazine has been almost the same as in previous years except for the increased charges for the better quality of paper used at present.

All the bills have been settled except a few minor ones which I have not received, and also, of course, the bill for the 4th issue of the Magazine. There remains at present a sum of Rs. 1802-9-8, of which 294-14-7 is on hand, the remainder being in the Bank.

I cannot adequately express that sense of thankfulness nor repay that debt of gratitude I owe to Major K. G. Pandalai, F.R.C.S. I.M.S., and Dr. M. R. Guruswami Mudaliar, B.A., M.D.C.M., for the interest they have evinced, the counsel they have given and for the help they have always rendered when most needed. To those members of the Committee and other ladies and gentlemen who so kindly and efficiently co-operated with me in the management of the Graduates' Reception, to Dr. Dinker Rao, M.D., who so patiently submitted to the obvious torture of auditing the accounts, I wish to take this opportunity of conveying my sincere thanks.

I should like to thank every one of you for the opportunity you have given me of serving the Association, for I always considered it a privilege to do so, and ever-mindful as I have always been of its traditions I have always endeavoured to execute the responsible duties of my office to the satisfaction of its members, and to the best of my limited ability. Less I have not aimed at, Ladies and Gentlemen, more I have not sought.

MADRAS MEDICAL COLLEGE, }
9th March, 1927.

R. K. CHETTUR,
Hony. Secretary,
1926-1927.

Appendix (A.)
Office Bearers 1926-27.

Patron:

Major General Hutchinson, I.M.S., Surgeon-General
with the Government of Madras.

President:

Lt. Col. C. A. F. Hington, I. M. S.

Vice-Presidents:

{ Major K. G. Pandalai, I. M. S., F. R. C. S. (Eng).
{ Dr. M. R. Guruswami Mudaliar B. A., M. D. C. M.

Asst. Secretary:

Mr. A. S. Selvaraj.

Class Representatives:

Lady Students	Mrs. M. A. Williams.
Military Students	Mr. P. DeMellow.
Final Year	Mr. P. T. Raghavachari
Fourth Year	Mr. Kameswara Rao.
Third Year	„ Govinda Rao.
Second Year	„ E. C. Srinivasan.
First Year	„ G. Verghese.

Magazine Committee.—

President:

Major K. G. Pandalai, F. R. C. S., I. M. S.

Editor:

Dr. M. R. Guruswami Mudaliar, B.A., M.D.

Associate Editor:

Dr. A. Vasudevan M. B. C. M.

Student Editors:

Mr. P. Ratnaswamy.	} Final Year.
„ M. D. Ananthachari.	

Appendix (B.)

Date:	Speaker.	Subject.	President.
9-9-26	Capt. P. Basu I.M.S.	"Rejuvenation its Relation to the ancient Yoga System."	Major K. G. Pandalai, F.R.C.S., I.M.S.
5-11-26	Dr. M.L. Kamath M.D.	"My Reminiscences."	Dr. M.R. Guruswami Mudaliar B.A., M.D.
17-11-26	Mrs. Muthulakshmi Ammal M.B., C.M.	"The Duty of the Profession."	Mrs. O'Brien Beadon, M.B.C.M.
22-11-26	Major. A.J.H. Russell I.M.S.	"Preventive Medicine."	Lt. Col. E.W.C. Bradfield, I.M.S., M.S.
1-12-26	Sir P.C. Ray.	"Ancient Hindu Chemistry."	Sir R. Venkatratnam Naidu.
26 1-27	Dr. A. Lakshmanaswami Mudaliar B.A., M.D.	"Conquest of Disease."	Mr. R.V. Chockalingam, (III year.)
4-2-27	Dr. M. Kesava Pai M. D.	"Artificial Pneumothorax"	Dr. M.R. Guruswami Mudaliar, B.A., M.D.
8-2-27	Dr. James H. Cousins	"The Art of Medicine."	Lt. Col. A S. Leslie, I.M.S.

Appendix (C.)
The Madras Medical College Association Account for the Year 1926-27.

Receipts.	Disbursements.
By Subscription from students at Re 1 each ... 491 0 0	To Graduates' Reception entertainment ... 455 12 0
	Printing receipt books ... 10 0 0
	" Expenditure in connection with the Association lectures ... 22 0 0
	" Balance in hand ... 3 4 0
Total ... 491 0 0	Total. 491 0 0

Secretary:
R. K. Chettur.

Graduates' Reception Expenditure	
	Rs. A. P.
Refreshments	... 346 0 0
Cards, etc.	... 22 0 0
Chairs	... 31 0 0
Peons	... 30 0 0
Flowers	... 11 8 0
Cigars and Cigarettes	... 10 4 0
Miscellaneous	... 5 0 0
Total	... 455 12 0

Appendix (D)
Madras Medical College Magazine.
Accounts for the year ending 28th Feb. 1927.

Receipts.		Disbursements.	
By Balance carried over from the year 1925-26.		To Printing the first 3 issues for the year 1926-27.	
Fixed deposit	... 1,000 0 0	" Remuneration for Clerk	... 918 4 0
Savings bank deposit	... 651 5 5	" Do. Peon	... 120 0 0
As at 1st March 26	... 69 0 0	" Do. Typist	... 6 8 0
		" Stamps	... 3 12 0
" Interest on Savings Bank deposit	1,720 5 5	" Mis. expenditure.	... 90 8 0
" Interest on Fixed Deposit	9 15 8	" Printing the 3rd issue 25-26	... 15 1 0
" Subscriptions from students at Rs. 2/ each	120 0 0	" Do. 4th issue	... 254 12 0
" Mofussil members	506 0 0		164 4 0
" Staff	474 0 0	Balance as on 1-3-27	1,573 1 0
" Advertisement charges and Mis. receipts	126 0 0	Fixed deposit in Bank	... 1,120 0 0
" Suspense	410 1 7	Savings Bank Do.	... 386 5 1
" Balance brought over from the Association	6 0 0	Postage stamps	... 1 6 0
account for 1926-27	3 4 0	Cash in hand	... 294 14 7
	Rs. ... 3,375 10 8		Rs. ... 3,375 10 8

I have audited the accounts with the receipts etc., maintained by the Hony. Secretary and I certify that the accounts as shown above are correct and present a true statement of the affairs of the Fund as on 1-3-27.

(Sd.) Major K. G. Pandalai, F.R.C.S., I.M.S. (Sd.) Dr. M.R. Guruswami Mudaliar, B.A., M.D.
Vice President. Vice President.

(Sd.) G. Dinker Rau, (Sd.) R.K. Chettur.
Hon. Auditor. Hon. Secretary.



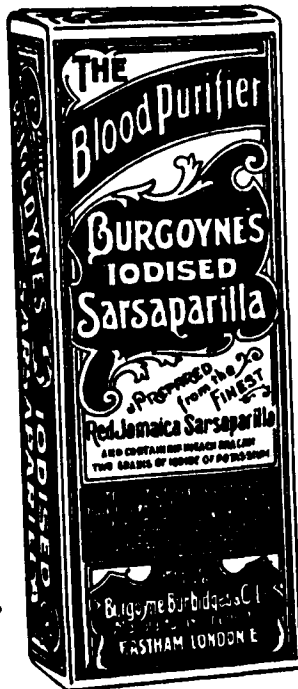
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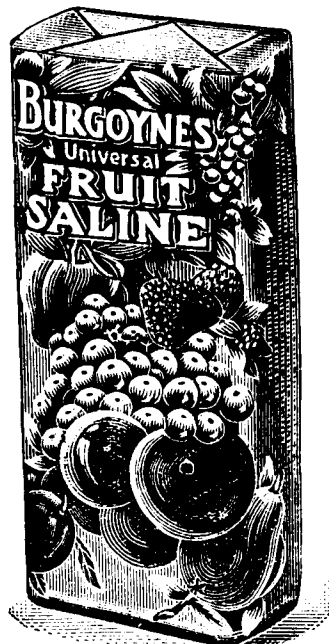
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Editor: Dr. M. R. Guruswamy Mudaliar B.A., M.D.

Secretary: R. K. Chettur.

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