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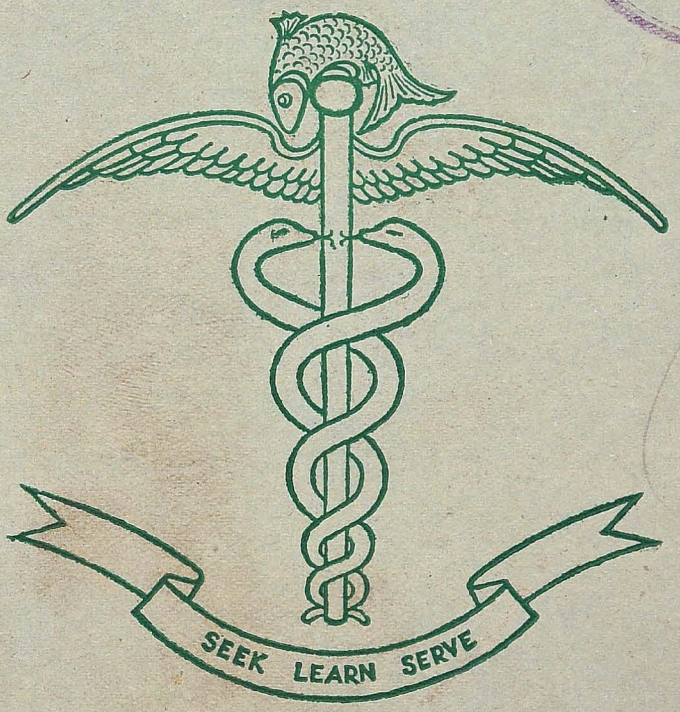

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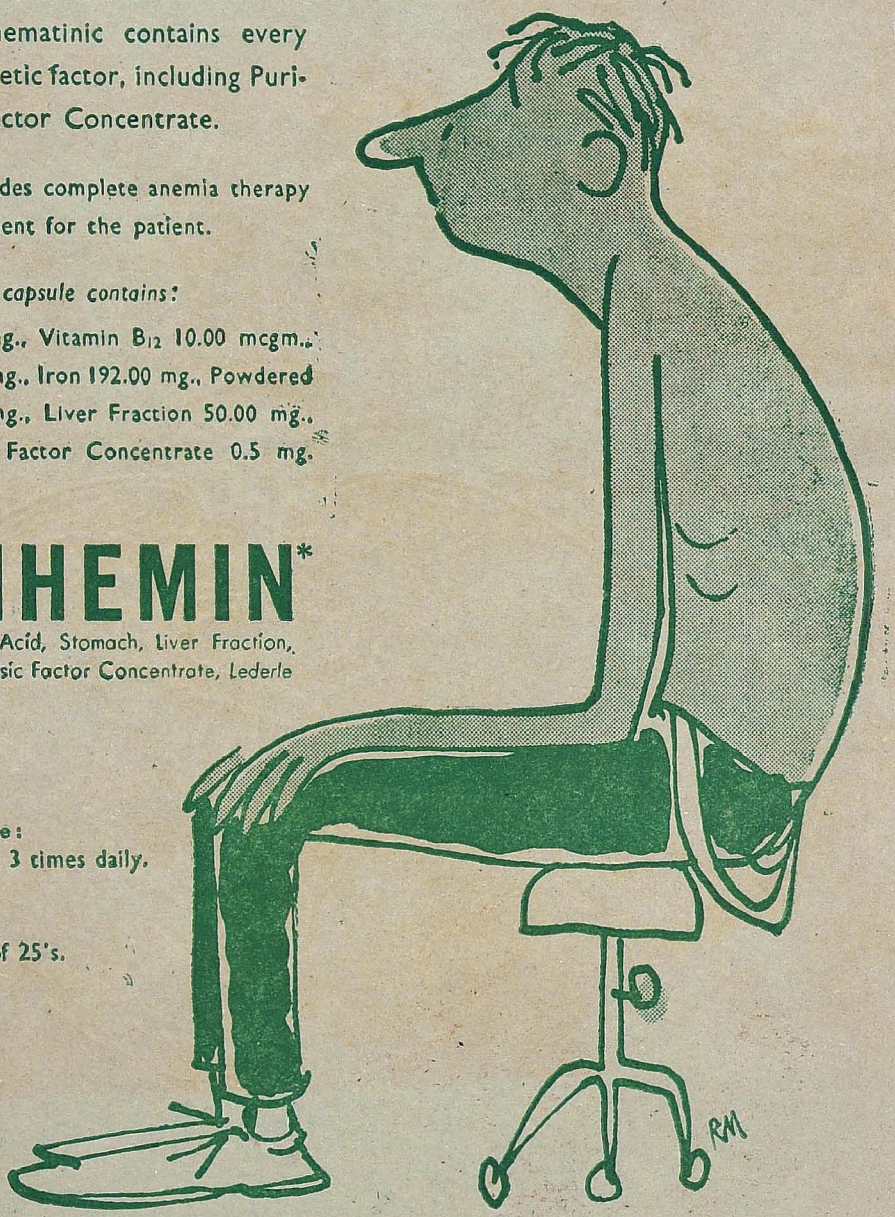

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The Madurai Medical College Magazine

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VOLUME IV ★ NOVEMBER 1957

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CONTENTS

The Editorial

THE Madurai Medical College has now entered the fourth academic year and we deem it a privilege to present this, the fourth volume of our College Magazine.

The New Year itself dawned with very historic events. The Madras University celebrated its centenary and Madurai, with her large number of collegiate institutions — second only to the city of Madras, had her full share in the celebrations, in which our own institution, though young in age, by virtue of her importance in our great national reconstruction programme, contributed her due share. In addition to taking part in the local inter-collegiate sports, exhibitions, entertainments, etc., marking the celebrations in this city, a representative contingent was also deputed to Madras to take part in the celebrations there.

Another notable event was the Rural Health and Medical Care Programme of the College which was inaugurated by Sri. R. Balasubramaniam, M.A., I.A.S., Collector of Madurai, at Uthangudi Village with a population of about 1500. Situated outside the city, about three miles from the college, this rural programme gives the students an opportunity for a closer link with the rural population and understand the important problems of rural housing, sanitation, safe and adequate water supply, good food, and proper environments — requirements so vitally linked with good health. Any scheme of development, particularly in the socio-economic field must have its roots in the health of the village, the very foundation of our national structure. Rightly has the Regional Committee for South East Asia of the World Health Organisation stressed the importance of Rural Health Units as against increasing the number of hospitals and hospital beds. It is not suggested that activities other than preventive have no place in the scheme of things and we are aware of the difficulties in resist-

ing popular demands for curative services which are capable of producing immediate spectacular results. But we, as guardians of the health of the people, owe it to them that we not only cater to their immediate needs, but at the same time plan for lasting results even if it appears at the moment to be at the cost of urgent needs. This is the basic conception which should permeate all our planning and training programmes.

The current academic year started with a further increase in the number of new admissions to our college from 70 last year to 100. The progress in the building and equipment programme has been maintained and the hostel accommodation increased to meet the additional needs.

It had been our desire to bring out two issues of our College Magazine this year onwards by bringing out an additional issue in March. Though much of the materials were ready, our printers were so pre-occupied with the printing of University Text Books and notes etc., that we had most regretfully to abandon this proposal. When November approached, we had to face a new problem — the acute shortage of the quality of paper we had been using so far. These and the late receipt of some of the additional articles promised, as well as other unforeseen circumstances contributed to the delay in the publication of even this issue. We do hope that our readers as well as our advertising patrons would bear with us these shortcomings and enable us to benefit from our past difficulties.

We thank all those members of the staff as well as students who contributed to this issue and our advertisers for their continued patronage. Many of the articles as well as advertisements in this issue were collected by Mr. C. Thangaraj, our previous Secretary to whom our thanks are due.

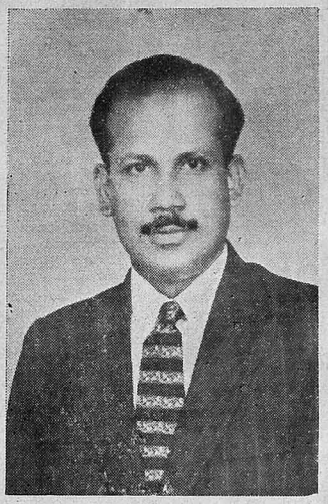
Our thanks are also due to our printers, the De Nobili Press for the excellent work turned out by them.

STAFF EDITOR

Magazine Committee.

— ***JAI HIND !*** —

We Welcome



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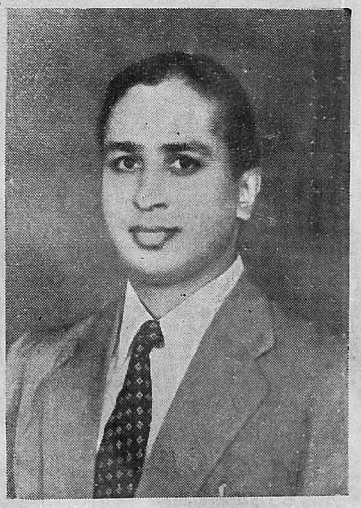

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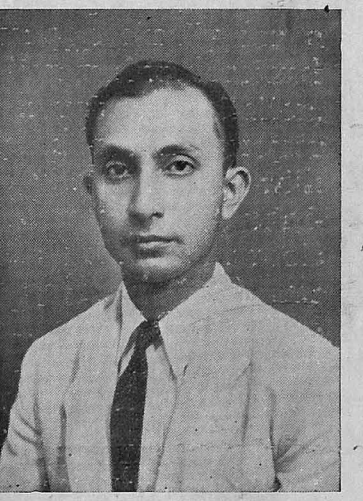

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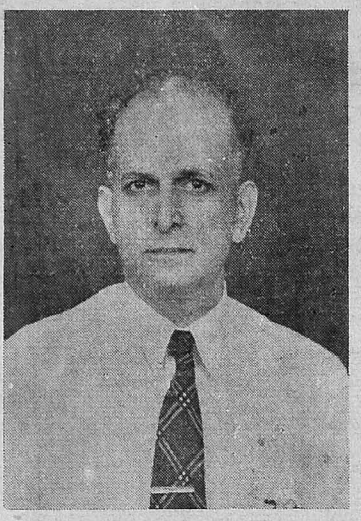

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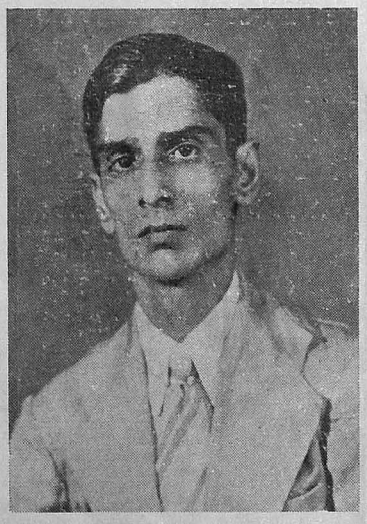

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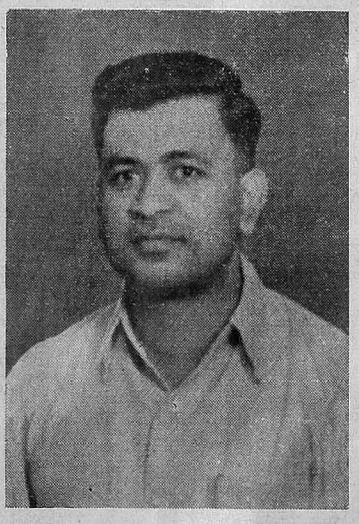

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The Philosophy of a Doctor

by

DR. M. D. ANANTHACHARI M. D.,
Principal and Professor of Medicine,
Madurai Medical College.

WHEN your secretary asked me to address your association, I think I must have left behind me my native prudence and like one belonging to the class that "rushes where angels fear to tread", accepted the invitation. The enormity of the folly of my action I realise now, while facing you, a set of hardened intellectuals, who do not suffer fools nor their folly, readily.

Fools do however philosophise and under cover of their folly as a cloak shoot wisdom as a challenge to their overtly wise brethren.

In evolution when does philosophy appear ? Man as a piece of creation stood apart, from the rest, on account of the development of his brain. With this, he transcended the others in the evolutionary scale. A remarkable change noticeable in him is the development of his psychic function. The brain gave birth to the mind, and the mind registered the features of intelligence, i.e., the faculty of independent thinking on the basis of experience. Intelligence helped man to challenge Nature and question her ways with the purpose of gaining sway over her and order his environment to suit his survival and satisfy his senses. He reached the stage where he could be a demi-god and order life as if he were the Creator himself. Having acquired all knowledge, the mind of man began to think in abstraction, un-relative to a purpose and in pure joy of the exercise of its faculties and thus reached the stage of philosophising.

Philosophy can come into being only after a certain amount of experience of the world and worldly things. You cannot philosophise if you have no observational data at all. One must gain experience and that must be as wide as possible for one to indulge in a philosophy. Next, one must have time for reflection. Immersion in the materialistic pre-occupation of the mundane world cannot provide for the abstract contemplation of philosophy. Reflection calls for calm collection and array of facts of life, gained from all sciences relative to matter, before the drawl of a conclusion based on them, becomes possible. The conclusions drawn by a process of reflective thinking into the causes, purpose, progress and evolution, may become a philosophy. The sciences give facts relative to life and living things. They do no more than collect facts and figures. They are like the numbers in a balance sheet of assets and liabilities. These do not reflect the mind of the business-man to whom they appertain ; the purpose for which he undertook the business is not revealed there; neither his hopes and his fears, nor his prime motive. The exercise by which a contemplative and reflective mind tries to probe into the meaning of facts and truths would amount to a philosophication and

the conclusions drawn for future conduct, would be regarded, a philosophy.

It cannot be gainsaid that philosophy is the by-product of rationalisation of the physical phenomena, a fitting into a common inference. Philosophy has no justification for that appellation if it did not relate to human activity in any sense. The essence of philosophy is its applicability to man and to none else in creation. It gives satisfaction to the inquiring mind; a bias, a purpose and rationale for the activities he contemplates relative to his existence and perchance, raise him above the common herd.

Human psychology has attempted and created various concepts of existence relative to the past, the present and the future. It has attempted to know the origin, purpose and the future of creation. It has rationalised the existence of unity in diversity, death in immortality, a moment in eternity and an atom in cosmos. The outcome of such disquisitions has been the recognition of various types of philosophies such as the physical and metaphysical, the moral and ethical, the theistic, and so forth.

When the greatest function of the brain, independent thinking, has free scope for its intellectual exercise and is fed by the plethora of knowledge (used in its widest sense) gained by the exercise of its appreciative faculties, philosophy is born. For its boundary, it has the entire horizon established by individual and collective experience of the various faculties. There is nothing it cannot embrace in its ambit ; it can question the very existence of God and all His attributes. Its purpose, strangely, is not always evident nor does it set one before itself. What does it achieve ? Perhaps nothing more than the pleasure that comes out of the exercise of a function performed well and completely. " Fancy while thou art, thou art but what thou shall be — nothing — thou shalt not be less ".

It is in this, perhaps the philosopher is derided by those who do not have the gift to take their minds above what their sensorium feels and subscribes to. The outcome of a philosophy may be entirely unproductive of mundane, material values. Of course, no philosophy can fill the empty stomach nor assuage the pangs of hunger. No wonder philosophers are irreverently set apart as impractical dreamers with no connection to reality. A wag has compared philosophy to the exercise one gets in the process of searching for a black cat in a very dark room when it is not there !

When does philosophy pervade a human being ? When he begins to question everything, even himself. Can life be lived out without a philosophy ? Yes ; all the animals around us do that. To have no philosophy is to sink to the subhuman level and deny the existence of that very beautiful thing of creation called the mind — the gift of a part of himself by God to man, his noblest work !

Why should there be need for a philosophy ? Because it gives or tries to give, meaning to all human activities and all human relationships. It guides where there is no direction; it satisfies where there is no fulfilment ; it rationalises where knowledge is incomplete. It has the virtue to raise man above his environment and create around him one that pleases him. Life and death, pleasure and pain, achievement and failure, he can look upon these with equanimity. Yes, make a hell of a heaven and a heaven of hell.

And that is my conception of philosophy. And now what of the doctor ?

A doctor is a specialised specimen of humanity. His special field relates to the human body in the majority and the mind, in a minority. He claims to know Nature's ways. For the exercise of his profession he gets as his tools, what any science can give him. He comes across in his daily practice, humanity in the raw, physically and mentally. Though compelled to go in company with pain and misery, yet like the Happy Warrior, " turns his necessity to glorious gain ". Though these dull his susceptibilities by their constancy, yet sensitise him, for whatever he thinks of and does to others is also applicable to himself. Birth and death are the daily phenomena he deals with, as a speciality. Sympathy and correction is what he purveys. Knowledge and experience are his daily acquisitions. The phenomena of life are his books. His eternal endeavour, is the maintenance of life.

It will be seen that a doctor has all the ingredients necessary for the exercise of an intelligence and the creation of a philosophy. Knowledge of any and every type is exercise to his intelligence. The observations he makes are confounding, inexplicable, and even beyond his comprehension. — Where else can you get a fertile field for philosophy, than in a doctor's practice ? Let us examine a few of his enigmatic problems.

A rose that has blown, for ever dies. The acceptance of birth, implicitly takes in death as well. There never was born, that which did not die. Everything on earth changes. There is nothing everlasting, immutable, and never-ending. The mountains, rivers, oceans, keep changing. Even the universe is changing. The sun and the stars are constantly getting consumed. Life around is undergoing an eternal cycle of change. Time never stands. It marks a change in everything in creation.

A doctor's effort, foolish perhaps, is to resist this change in Man. Like all living thing he too must die : and before that, perhaps become old. The doctors' knowledge tells him that. His daily observation confirms all that — Death is certain, inevitable, may perhaps be essential ! Strangely enough death is friend to life. And yet what is his daily task ? Keep death back, like keeping the waves of the ocean back ? Should he stop this futile nonsense, " seeing that death is a necessary end, come when it will come " ?

But the whole universe is doing just the same ! Life on earth other than human is doing the same. Birth, living, growing, maturing, fructifying and dying, are the greatest common measure of all life. Where is the sense in all this ? From whence hither, willy nilly blowing ? From hence, whither, willy nilly flowing ? Like the wind over the waste and the river across the plains, life seems to have no purpose.

What is the urge for creation ? What is the purpose of its being — what power takes life irrevocably to its destruction and dis-integration ?

To a doctor, Death is a common friend — or enemy ? Many is a situation, where death would be merciful. He sees the cold hand of death daily. But what is it that death has robbed the body of ? Life ? What is it ? A moment before, the body was living. It was an individual. A something with vast potentialities. But a moment later it is a carcass — a decaying organic conglomeration of atoms of various kinds and dissolution begins ere yet the hand that wrought the change has disappeared ! What is that which the protoplasm of the cells has lost ? Life ? and what is that ?

Look at the emergence of life. A seed is inanimate. There is no life in it yet. It can remain unaltered for centuries. The grain found in a Pharoah's tomb, hundreds of years later, when put into moist earth sprouted ! What gave it the life ? What made its chemicals start living and multiplying ?

This continuous change, birth leading to death, has no meaning, none that he, the doctor can yet give. And what philosophy can give him the answer. None. Why be born, if only to die ?

He therefore helps to perpetuate the state in which he finds the body. If it is being born, he helps in its birth without harm. If it is growing, he helps and maintains its growth. He trains it to evolve correctly. When it becomes decadent, degenerate and dying, he helps the exitus lethalis — and renders the descent to the avernus easy. He does not question the Maker, who perhaps is also the Destroyer. He awaits until he knows more. That is his philosophy.

For what purpose is life then ? Will He who made the world in pure joy, in an after-rage, destroy ? Often the question arises, " Why this birth and death, after only a short living " ?

I philosophise thus :

Birth and death are beyond my ken and control. The living alone, I can manage. What am I to do with it ? Who can tell ?

All life other than human is controlled by some outside agency — Nature. They react and act in a pre-destined, predictable way. They are the victims of reflexes from the environment. What is it that they do after they are born and before they die ? They are not responsible for what they do. They are guided by instincts, (and not reason, as in the case of man) to fulfil their allotted purpose in life. They strive to live, mate, procreate and die. Man alone has his mind and intelligence to reason what he should do, while he lives. And how does he live ? The world of to-day is the answer to what he has done to his life. Every other form of life without thinking subserves a function useful to something else than its own self. Man alone, in all creation can be selfish. Need he be ? Why not he copy that which is guided by the same agency that created him also along with the rest?

And so a doctor philosophises !

The man who has not anything to boast of but his illustrious ancestors is like a potato — the only good thing belonging to him is underground.

— *Sir Thomas Overbury*

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Tuberculous Disease of the Abdomen

by

Dr C. K. Padmanabha Menon, M.B., M.S., F.R.C.S. (England),
Professor of Surgery and Superintendent, Government Erskine Hospital, Madurai.

AMONG the extra-pulmonary lesions, tuberculous disease of the abdominal organs forms a very important part. It is said that malaria can simulate many diseases and conditions except pregnancy. This may be said to be true of tuberculous disease of the abdomen because it can mimic not only a number of abdominal conditions such as intestinal ascariasis, carcinoma, lymphosarcoma and amoebiasis to mention only a few of the competitors in the field of diagnosis, but also simulate pregnancy.

It is not possible to go into great details of the various manifestations of this disease in the abdomen. But for a clear idea it is necessary to point out the presenting symptoms in some of these cases and to come to a certain idea of the best way of management of these conditions. It is well-known to general practitioners that the disease starts often in the lymph glands which get infected through ingestion or inhalation. When the tubercle bacilli enter the lymph glands these ordinarily may, after multiplying, get arrested in their deleterious progress and except for the enlargement of the gland, nothing may be noticed. On the other hand, there may be softening of the gland, adhesion of this to the neighbouring blood vessel and haematogenous dissemination of the bacilli may take place to any or all the organs of the body. Thus in this distribution, the gastro-intestinal tract, genito-urinary tract and the lymph field of the abdomen including the peritoneum may be the possible victims.

When tubercules multiply, the changes in the submucous or sub peritoneal layers are responsible for ulcerations and adhesions and collection of peritoneal fluid. When these regress, the result may be restoration to fairly normal functions which is more common than we imagine, and so these changes do not come to our notice. But when the disease progresses for the worse, *ulceration* may lead to stenosis or perforation ; *adhesions* may lead to kinks and bands and volvulus ; *lymph glandular masses* may give rise to pressure and difficulty in the normal function of various abdominal organs. Long continued pathological processes will result in malnutrition, faecal fistula and partial intestinal obstruction which, if unrelieved, may result in prolonged misery and death.

It is possible for us to have a rough classification of tuberculous disease of the abdomen as :—

1. Gastro-intestinal.
2. Genito-urinary.
3. Lymph glandular.
4. Peritoneal.
5. Involvement of other regions.

From a clinical point of view,

(*a*) The disease usually has a very insidious onset and in many instances, it

may be silent until some complications develop.

(*b*) There may be symptoms of chronic dyspepsia with vague abdominal pain which sometimes resemble peptic ulcer, sometimes chronic cholecystitis, sometimes amoebiasis.

(*c*) Patient may complain of chronic intestinal obstruction not being able to pass freely faeces or flatus, and now and then having colicky pain.

(*d*) May come with signs and symptoms of acute intestinal obstruction.

(*e*) May complain of distension of abdomen and on examination be found to be due to free fluid in the peritoneal cavity or it may be localised cystic swelling.

(*f*) There may be multiple lumps in the abdomen.

(*g*) The presenting complaint may be frequency of micturition, pain or pyuria, drawing particular attention to the urinary tract, and

(*h*) occasionally there may be signs and symptoms pointing to the infection and involvement of the genital organs, the epididymis in the male and Fallopian tube in the female.

Before going into details about these conditions, it is necessary to point out that although the patient's complaint may be pointing to the abdomen, the entire body must be examined for signs and symptoms of involvement of other regions of the body particularly lungs, lymph glands that are accessible for palpation and also those of involvement of bones and joints. In many instances tuberculous disease does not affect one organ only, although when we say one organ is affected, we mean that that particular organ is more seriously and extensively involved and is more obvious than the involvement of the others.

I. Gastro-intestinal Tuberculosis :

The commonest site of tuberculous lesion in the gastro-intestinal tract is the terminal ileum and caecum. But any part of the small intestine and even the stomach can be the site of tuberculous lesion ; it becomes less as we trace upwards from the terminal ileum and downwards from the caecum.

Gastric symptoms may be noticed in patients in :

(*i*) the very rare condition known as hyper plastic tuberculosis of the stomach or in some rarer instances, tuberculoma of the stomach. These two conditions are mentioned only to draw attention to the fact that they may be mistaken for either the linitis plastica or for polypoid variety of carcinoma of the stomach. We have to remember that syphilis and more rarely tuberculosis can produce changes in the stomach resembling linitis plastica (infiltrating carcinoma). However, it is better and safer for the patient to entertain the diagnosis of malignancy, under all these three conditions, because they are more common and more dangerous till it is disproved. Gastric symptoms may also be produced by the following other conditions :—

(*ii*) A high jejunal tuberculous ulcer with stenosis : This simulates very closely a chronic stenosing duodenal ulcer. I have in my experience a few cases where the pre-operative diagnosis was chronic stenosing ulcer of the duodenum, whereas at operation, high jejunal ulcer with marked stenosis was the obvious cause of gastric retention. Whenever one suspects a chronic ulcer from the clinical findings, but does not find the ulcer in the duodenum, it may be worth while remembering the possibility of high jejunal ulcer

(tuberculosis) or in many instances the condition of annular pancreas, where a portion of the pancreas surrounds the third part of the duodenum.

(*iii*) A large mass of tuberculous glands may be present in the upper part of the abdomen. This may press on the third part of the duodenum and produce obstruction to the emptying of the stomach. In many instances a good barium picture can reveal a widening of the 'C' curve of the duodenum or it may show obstruction to the third part of the duodenum, the cause of which may be revealed at laparotomy to be a mass of lymph glands. The treatment of these two conditions is obviously a short-circuiting operation, and subsequent treatment for the tuberculous disease. For the hyper plastic tuberculous stomach and for tuberculoma of the stomach a partial gastrectomy of the affected part will obviously be the ideal measure.

II. Intestinal.

A number of cases present themselves with symptoms of chronic obstruction and on investigation they are found to be the effect of lesions in the lower ileum or caecum or both ; these can also be associated with enlargement of lymph glands in the abdomen and in a few instances with a fair amount of fluid in the peritoneal cavity or associated plastic peritonitis. There are also cases where there will be only a mass in the right iliac fossa which may be difficult to distinguish from carcinoma or amoeboma of the caecum or even a chronic intussusception. Under these circumstances the differential diagnosis has to be made by consideration of other factors and if a correct diagnosis cannot be arrived at, it is always safe to do a laparotomy to arrive at the correct diagnosis. In many instances, the patients are not in a suitable stage for a one-stage resection of the part of the bowel involved nor is this desirable if the lesion is extensive. The initial concern should be to overcome the obstruction by suitable short-circuiting operations, to treat the tuberculous disease by anti-tuberculous measures and re-consider further surgical measures at a later stage. Where, on opening the abdomen, multiple strictures are seen at long intervals, excision of several small segments and end to end anastomosis is a method of choice. In two or three instances where I found multiple strictures all along the entire length of the small intestine, where resection would have involved almost removal of the entire gut, I had to content myself with doing nothing but closing the abdomen followed by anti-tuberculous measures. These patients showed considerable improvement. It is possible that some of these ulcers may stenose so tightly that a second operation for overcoming the obstruction may become necessary, at a later date.

The difficulty of diagnosis even after opening the abdomen in some instances between hyper plastic tuberculosis and carcinoma of the caecum is well known. One need not expect to find always tubercles on the surface. In all doubtful cases if the patient can stand a resection, it is better to do a resection. Otherwise, do a short-circuiting operation and remove a gland and get biopsy done to confirm the diagnosis. In most instances of tuberculous disease of ileo-caecal region, I would prefer in the first stage to do a side to side ileo-transverse colostomy. When patients are admitted with acute intestinal obstruction, the diagnosis cannot be obvious and the necessity for a laparotomy will be very pressing. When the abdomen is opened it may turn out to be a very extensive tuberculous disease of the entire

intestinal tract and peritoneum. Recently in one of my cases, the diagnosis of intestinal obstruction was arrived at because few visible coils of small intestines stood out prominently. The diagnosis of tuberculous disease as a cause was very likely, because there was a previous history of treatment for pulmonary tuberculosis. On opening the abdomen there was considerable amount of extensive adhesions, matting up the entire small intestines which had been kinked in various places. This was the cause of the obstruction. With considerable patience and with delicate handling these plastic adhesions were gently but completely separated and the intestines could be released from their kinks. Throughout the entire peritoneal surface, multiple small tubercles could be seen studded all over. With the instillation of streptomycin, the abdomen was closed. The patient made an uneventful recovery. I made mention of this case to emphasise that opening the abdomen is necessary whenever we find visible coils of intestines standing out prominently when there is an attack of colicky pain.

III. Lymph Glandular Tuberculosis :

This condition is more commonly seen in younger age group of people. Very often children are admitted with high fever, severe abdominal pain and some amount of distension. It is not easy to come to a definite diagnosis as worms may be the common cause of such a condition. But almost equally common or even co-existing with this will be tuberculous mesenteric lymphadenitis. If there is no acute intestinal obstruction these patients should be treated on conservative lines and often they recover completely. The presence of enlarged lymphatic glands elsewhere in the body or presence of tuberculous focus in any other part may be a clue to the diagnosis. Sometimes patients come with acute intestinal obstruction in which case operation is unavoidable.

IV. "Ascitic Variety" :

The abdomen gets distended and in some instances the diagnosis of cirrhosis of liver may be made. The fluid in some instances may also be encysted, giving rise to some difficulty in the diagnosis between this and pseudo-pancreatic cysts or cystic conditions of the kidney. If the ascitis is very tense and considerably large collection of fluid is present, there may be no shifting dullness. In one instance of this nature, in an old female patient, diagnosis of malignant ovarian cyst was made. In this patient there was also a calcified mass in the position of the uterus that before the operation, was diagnosed as a calcified fibroid. On opening the abdomen, the condition turned out to be tuberculous peritonitis with enormous quantity of fluid, intestines shrunk and tethered within the fluid, not able to shift its position at all. The calcified mass was a fibroid of the uterus. If the patient's condition had permitted removal of this mass it would have been a very good specimen for the museum. The abdomen was closed after getting rid of the fluid and instilling streptomycin. The patient made a complete recovery and was very grateful for the operation. This case is mentioned to stress the fact that if the diagnosis could be made with certainty, the fluid may be aspirated periodically and streptomycin instillation may be continued with pneumo-peritoneum until symptoms are relieved. But in most instances laparatomy will be required in doubtful cases to first establish the correct diagnosis.

In a few instances as a result of plastic adhesions, the intestines may get adherent to the abdominal wall particularly

at the umbilicus and faecal fistula may develop. The non-tuberculous fistula usually may heal if obstruction below the level of the fistula is removed. The prognosis of tuberculous fistula is always much more serious. After improving the patient's general condition and tuberculous lesion in the abdomen by anti-tuberculous measures, if the fistula does not heal, end to end anastomosis may have to be performed, after resection of the affected part, when the patient is fit enough to stand such an operation.

V. Genito-urinary Tuberculosis :

Apart from the fact that they may confuse the diagnostic issue, there are cases where infection of the genito-urinary tract has gone off, for a considerably long period side by side with intestinal tuberculosis. Careful study of the urinary tract will always be an essential step when patients with intestinal tuberculosis do not respond as well as they should. A detailed discussion of this problem is out of place here.

The *other varieties of tuberculous manifestations* inside the abdomen are comparatively rarer and I only mention here just to complete the list. This will include the tuberculous disease of the spleen and liver and also cold abscesses forming lumps in the abdomen.

Difficulty in diagnosis :

I would like to mention a few cases to point out some of the difficulties. I have already stressed the high jejunal ulcers causing confusion with duodenal ulcer. In one instance, a patient was treated for worms several times but she came back repeatedly saying the relief was incomplete and at laparatomy she was found to have a tuberculous ulcer, resection of which completely relieved her symptoms. Recently, I had a case which was diagnosed to be possibly duodenal ulcer clinically. Radiologically, there was a suggestion of a volvulus of the stomach but further study revealed that this was a pulling up of the stomach due to tuberculous adhesions. Cases of carcinoma of the caecum mistaken as tuberculoma and vice versa have already been stressed. The same is the case with chronic intussusception and amoeboma. The presence of multiple lumps in the abdomen should call attention to various possible causes, the commonest of which is tuberculous lymphadenitis. At the moment, I have a case in the male surgical ward with multiple lumps in the abdomen and partial intestinal obstruction and on examination showing a very large irregular nodular mass in the pelvis, felt per rectum. The diseases of lympho-sarcoma, Hodgkin's disease and multiple secondary nodules in the abdomen are all conditions which cannot be definitely diagnosed, in some instances at least, without a biopsy. In a few instances confusion may be caused also by pseudo-myxoma peritonei which presents itself as irregular nodular swelling in the abdomen diffusely extending throughout.

I have stressed a few of the important clinical features, and tried to enunciate certain common principles in the management of tuberculous disease of the abdomen with special reference to those affecting the gastro-intestinal tract. There is a fair amount of difference of opinion as to the types of anastomosis and as to the one-stage and two-stage procedures with regard to the treatment of ileo-caecal tuberculosis. It is necessary to point out that no fixed rule can be applied in individual cases. The treatment should be adopted after laparotomy then and there to the needs of the particular case and if

and when the two-stage resection is contemplated, it is better to do side-to-side anastomosis in the first stage and at the second stage the intestine can be resected just beyond the anastomosis and the cut ends closed. But these are technical details. I do not wish to go into a lengthy discussion of this problem.

The treatment of tuberculous disease of the abdomen has become comparatively easier and safer since the advent of streptomycin and INH and PAS. These are the drugs which can be used to control and improve conditions of the patient either as a preliminary measure before operative treatment is undertaken or subsequent to the operation and establishment of diagnosis. But I must draw the attention also to the fact that many a time patients have been treated as abdominal tuberculosis; yet, on opening the abdomen, there was not a trace of evidence to confirm the diagnosis. So, wherever a reasonable course of treatment has been given, if the improvement is not satisfactory, it is worthwhile not to persist in these drugs as valuable time may be lost in diagnosing and treating more serious and often fatal conditions. Even after treatment by streptomycin and other drugs mechanical changes that may occur as a result of fibrosis during the process of healing may demand a surgical intervention. When it is found that cases diagnosed as tuberculous disease of the abdomen in any of its forms does not respond as well as should be expected, taking all circumstances into consideration, such patients should be given the benefit of surgery whereby a correct diagnosis can be established, proper line of treatment can be chalked out and dangerous illness which may mimic tuberculous lesions may be excluded.

My thanks are due to Superintendent Erskine Hospital, for permission to use some of the clinical material that has come under my observation as a Surgeon in that hospital and to my colleagues.

That which is everybody's business is nobody's business.
— *Izaak Walton*

* * * *

The wheel that squeeks the loudest
Is the one that gets the grease.
— *Josh Billings*

* * * *

I am not an Athenian nor a Greek, but a citizen of the world
— *Socrates*

Myocardial Damage

by

Dr. K. Ramachandra, M. D.

THE integrity of the myocardium is a prime factor which keeps up the efficiency of the pumping mechanism of the heart throughout life. The properties of the cardiac muscle is maintained only in a heart which is perfectly healthy. Any disturbance of its metabolism or blood supply, may severely interfere with the smoothness of its action, either temporarily or permanently. Apart from physiological disturbances, pathological states may permanently damage the myocardium with serious consequences. The properties of this syncytial structure may be disturbed *in toto* or may be involved in a regular sequence depending on the nature of the disease. The tonicity and distensibility of the myocardium is early affected as a result of slight alteration of the integrity of the myocardium. It is only in advanced states of myocardial diseases that the other properties of the heart such as rhythmicity and conductivity are affected. All properties are sometimes disturbed in certain types of myocardial lesions, when there is reduction in the muscular continuity. The syncytial pattern of the heart with intercommunicating and interlacing structural design is an all-important factor which determines the rest and reserve forces of the heart.

Interference with the individual myocardial fibrils may not grossly disturb the functions of the heart muscle, but the efficiency of the heart as a whole will be affected. Changes in the sarcolemma, sarcoplasm, histological or histochemical, will throw a strain on the proper functioning of the heart, but not seriously interfere with its properties. Exhaustion of cardiac muscle experimentally brought about may disturb the metabolism and thereby interfere uniformly with its properties.

Anatomy of the Heart Muscle :

The muscle fibres branch and anastamose to form a syncytial mass arranged in large sheets or bundles of which four are important. These encircle the ventricular chambers in characteristic patterns. Superficial and deep bulbospiral and sinospiral bundles help in expelling blood from the ventricles.

The muscle fibres are composed of smaller strands known as myofibrils which are segmented and so present a transversely striated appearance in stained sections. The contractile molecules are arranged in the myofibrils in a definite manner that shifting of the molecules occurs during contraction — myosin and actomyosin. The sarcolemma is thin and delicate and surrounds the sarcoplasm. Between the syncytial myocardial fibres there are haemic and lymphatic plexuses, fibrous connective tissue, nerve terminals and wandering cells.

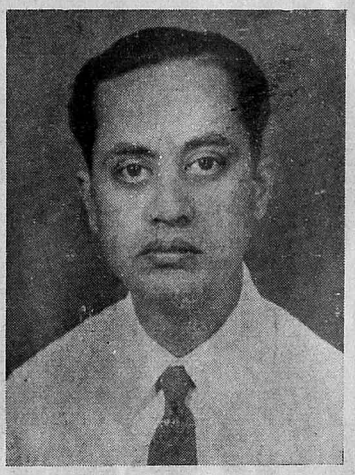
Properties of the Cardiac Muscle :

The contractile power of the heart is due to biochemical and biophysical changes in the heart. The stretched-out protein

molecules in the myocardial fibril are arranged in certain patterns parallel to each other. These molecules have the property of contracting or elongating under different conditions, viz., changes in pH, heat or alteration in water content. The molecules change their size and arrangement during contraction, thus shortening the sarcomeres. The reorientation of these myosin molecules is probably brought about by the breakdown of phosphocreatine into adenylic acid and adenosine triphosphate and lactic acid. The adenosine triphosphate may act as a catalyst to help combination of actin and myosin to the contractile molecule actomyosin.

The phosphocreatine is again resynthesised by the presence of oxygen in the sarcoplasm concentrated there by myoglobin from capillary blood. The myosin thereby gets re-orientated to its former proportion. Heat is elaborated during the breakdown of phosphocreatine which helps expansibility of the heart. This molecular phenomenon is based on biophysical and biochemical changes occurring in the sarcoplasm which determines the properties of heart muscle — rhythmicity, conductivity, contractibility, expansibility, refractoriness and irritability. All these properties may be sequentially affected or all may be depressed uniformly in myocardial disease. The property which is last affected is rhythmicity and conductivity, unless the conducting system is specifically the seat of damage.

Heart muscle is the seat of damage in a variety of diseases, some of which are definitely known, others are obscure. In many of the diseases the changes in the myocardium are not clinically manifest and often pass unnoticed. If an electrocardiogram is taken routinely in the hospital on all patients irrespective of the diseases they are suffering from, it will be realised that the heart is the seat of some form of reversible or irreversible affection. It is an important structure in the body, the functioning of which is modified by internal *milieu* and physical state of other organs and the body as a whole. Circulatory dynamics change from time to time mainly depending on metabolic needs of the body, subject to an altering internal and external environment. The physical status of the body determines the status and integrity of the myocardium. Deficiency states, anaemia etc., interfere with the nutrition of this specialised structure. Infections etc., which the body is subject to, also affect the myocardium altering its anatomical and physiological state. The heart is also subject to a variety of degenerations which are sometimes extremely fatal. Moreover the heart is supplied by blood vessels which are the seat of many diseases, and are mainly responsible for damaging the myocardium permanently. These are the conditions which are responsible for cardiac diseases of old age, and underlying causes of sudden death and myocardial insufficiency. From this short discussion, it is evident that the heart, like any organ is subject to the same dangers and more so because it is an actively functioning structure whose metabolic demands are more comparatively. As its blood supply is from the general systemic circulation, any infection in the body is liable to pass to the heart and damage this structure. A few decades back, the term "myocarditis" was in frequent use to describe obscure causes of myocardial failure. But unfortunately myocarditis has fallen into disuse as a result of intense study of the histopathology and it can be said that myocardial degenerations are more prevalent than myocarditis. This condition is existent but is confined only to certain diseases. Many



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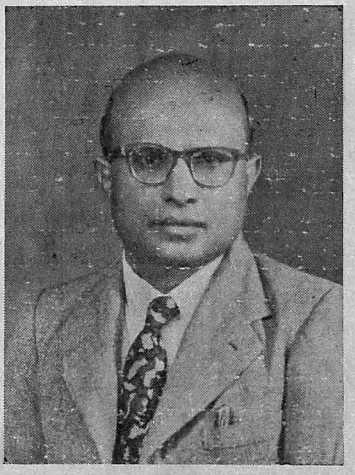

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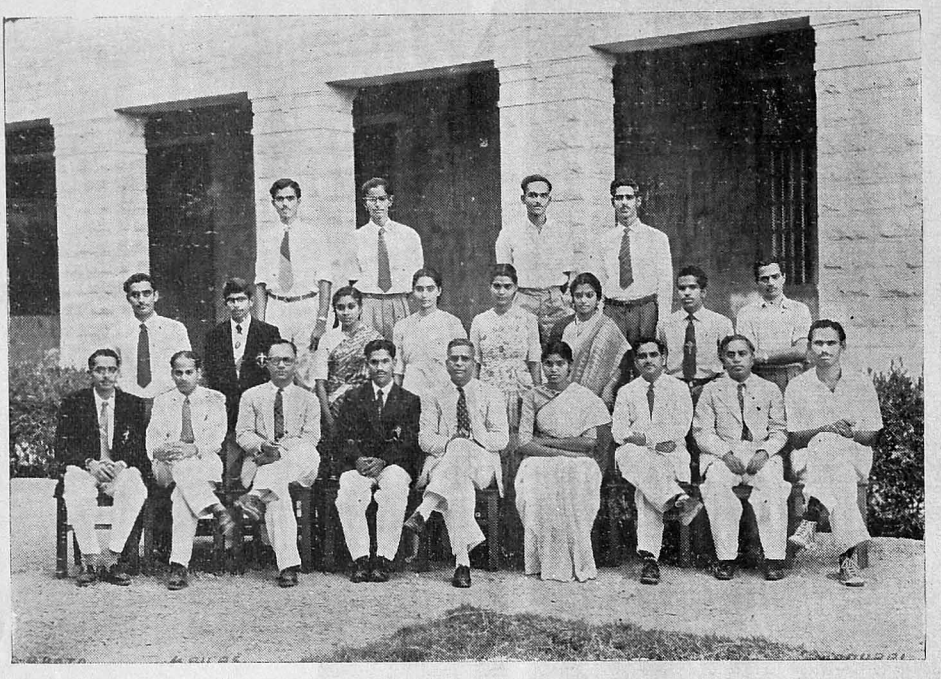
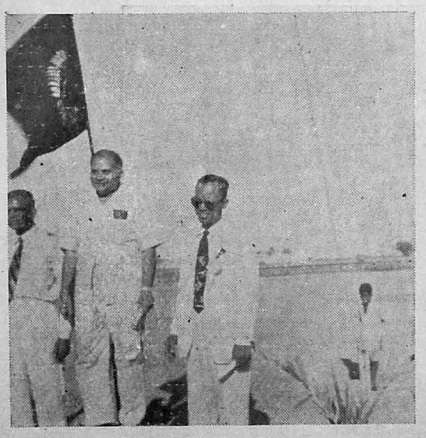


OFFICE BEARERS — SPORTS COMMITTEE 1956—57

The Chief Guest on Sports Day



The Victory Stands

Photos-Courtesy Mr. Abdul Hayeom

of the acute infectious diseases produce not inflammatory but degenerative changes in the myocardium from simple cloudy swelling to necrosis. Inflammatory cells may also be seen in the interstitial tissue of the myocardium side by side with the degenerative changes, but it is uncommon.

The myocardium is also affected mechanically as a sequelae of diseases of the valve apertures or some interference with the circulation as a primary feature. The size of the heart and the thickness of its walls are altered in all the valvular affections. The length and thickness of the muscle fibres are the basic features which determines the size of the myocardium. The largest hearts are often seen in valvular diseases, e.g. — aortic regurgitation. (*Cor bovinum*).

Circulatory disturbances and changes in the calibre of larger vessels and more so the arterioles interfere enormously with the efficiency of the pump. This impediment is overcome by the heart which tends to contract with greater strength and force. As a result of increased load and increased work of the heart, it becomes bigger at the expense of its musculature. Hypertrophy is a fundamental property of all muscles. The greater the work done, the stronger it becomes. Its metabolic needs and anabolism is raised. Work hypertrophy can only proceed to a certain limit after which there is gradual decline of its efficiency if the factors responsible for hypertrophy persist. Many other physiological disturbances in the metabolism and blood supply limit the progressive hypertrophy. This is always followed by decreased myocardial efficiency. Fibrosis of the myocardium results, which helps to weaken the myocardium. Its tonicity is lost and the heart tends to dilate. Summarising, the mechanisms of myocardial affection are as follow :

1. INFECTIONS :
   - (i) Changes in the heart due to toxaemia — mainly degenerative.
   - (ii) Inflammation of the myocardium — rheumatic fever.

2. NUTRITIONAL :
   - (i) Beri-beri.
   - (ii) Anaemia.
   - (iii) Protein deficiency states.

3. ENDOCRINE :
   - (i) Alteration in basal metabolism — thyrotoxicosis, myxoedema.

4. MECHANICAL :
   - (i) Valvular defects.
   - (ii) Congenital diseases.
   - (iii) Systemic and pulmonary hypertension.
   - (iv) Arteriosclerosis of peripheral vessels.

5. INTERFERENCE WITH CORONARY CIRCULATION :
   - (i) Atheroma.
   - (ii) Arteriosclerosis.
   - (iii) Coronary osteal stenosis — infarction.

6. RARE DISEASES :
   - (i) Affection of collagen matrix.
   - (ii) Parasitic diseases of heart.
   - (iii) Tumours of heart.
   - (iv) Degenerations: (*a*) Lipochrome (*b*) Atrophy.

(v) Epidemic dropsy.

(vi) Myopathic affections.

(vii) Myocardial injury ; abscesses, haemorrhages.

It is not possible to discuss in detail, the rare and obscure causes of myocardial damage, which no doubt will be extremely interesting. Here a few details about the commonly seen condition will be mentioned.

MYOCARDIAL DISEASES IN YOUNGER AGE GROUPS

Infections :

Any toxic fever or infectious disease produces myocardial damage pathologically ranging from unobservable histopathological change, to gross alteration in the architecture of the myocardium, and clinically without manifest signs, to gross alterations in all properties of the heart muscle seriously interfering with circulatory dynamics.

The increased rate of the heart is not only due to the raised metabolism of the body, but also due to changes in the myocardial rhythmicity. Sometimes instead of increased rate, there is bradycardia noted in certain varieties of infectious diseases. Here, the conducting system, probably, is the seat of damage which is only a specialised portion of the myocardium. Influenza, dengue fever and the other virus infections like yellow fever produce a bradycardia either during the acme of the fever, or during convalescence. Faget's sign which is characterised by falling pulse rate with rising temperature is very typical of several virus infections. Many of the exanthemata, mumps, whooping cough, diphtheria, pneumonia, typhus, tonsilitis, all produce some sort of changes in the myocardium. Sometimes sudden death is noted in many toxic fevers due to gross changes in the myocardium very often peripheral circulatory failure exists along with cardiac damage as a result of toxaemia. The infectious agent may not be found in the myocardium though sometimes it grows in the valvular and mural endocardium of a diseased heart. Probably the constant state of activity of the heart and high O_2 consumption is responsible for the rare incidence of myocardial infections. The myocardium often shows cloudy swelling, fatty and granular degeneration which may be generalised or localised and patchy. Associated with these changes, in more advanced cases, necrotic foci are seen. The interstitial tissue of the myocardium shows perivascular inflammatory reaction. Congestion and aggregation of leucocytes are noted with arterial changes of the nature of capillaritis. These are the cases associated with myocarditis. So in infectious fevers, the inflammation is mainly an interstitial affection, with alteration in the conduction of impulses in the musculature.

There are certain diseases where the heart shows definite changes histopathologically which give an additional aid in their diagnosis. For instance, in rheumatic fever, the changes are always of a definite pattern in most of the hearts. The myocardium shows cloudy swelling and cellular infiltration is seen in the interstitial portion of the heart muscle in the vicinity of blood vessels. The aggregation of these cells is known as Aschoff's body or nodes,

which is a proliferative phenomenon having a characteristic histological picture. In the centre, there is an area of fibrinoid necrosis or collagenous degeneration, surrounded by Aschoff's cells and lymphocytes, limited peripherally by fibrous tissue. Sometimes changes are seen in the arteries themselves, especially in the collagen diseases like rheumatoid arthritis, periarteritis nodosa, disseminated lupus erythematosis and scleroderma. As a result of the cloudy swelling, there is a tendency for the heart to dilate and appear bigger with gross interference of its functions. Acute congestive failure with conduction defects and other arrhythmias are often noted in this form of Carditis. Tissue destruction here is very little and massive scarring is never a feature of this form of myocarditis.

Beri-beri :

The cardiopathy of beriberi is definitely established and characteristic. The changes in the myocardium are described as hydropic degeneration and oedema of the muscle. Intercellular vascular dilatation has also been noted. This gives rise to acute cardiac enlargement.

Vascular dilatation is also noted in another disease, which is sometimes mistaken for beriberi, clinically — epidemic dropsy. Here the blood vessels especially capillaries show aneurysm-like expansion, and intracellular oedema. The myocardium is secondarily involved. The disease is endemic in places where mustard oil is used.

Hypertrophy and Dilatation :

Hypertrophy and dilatation are often a result of mechanical strain on a particular chamber whose wall becomes thicker to overcome the impediment till a stage is reached when it fails to hypertrophy. This is an inherent property of all muscles to undergo changes as a result of more work and good nutrition. The muscle fibres are seen to grow thicker and proliferate. This is a mechanism by which the reserves of muscle is exteriorised in chronic diseases, unless the muscle has lost such an inherent property due to aging or poor general nutrition or improper blood supply. Under such circumstances, this compensatory phenomenon is not noted. The common diseases where cardiac hypertrophy occurs are valvular affections and circulatory obstructions as hypertension, and co-arctation of aorta. A hypertrophied heart has a poor reserve force and symptoms of decompensation may be manifested. Specific chamber hypertrophy often is seen — auricles in mitral disease, right or left ventricular in *Cor pulmonale,* hypertension, aortic diseases etc.

As a result of muscle hypertrophy, the capillaries in between the fibrils are pushed wider apart and oxygen diffusion into the sarcoplasm becomes increasingly difficult and inadequate, limiting further hypertrophy. This induces fibrotic changes in the myocardium, as a result of which dilatation of the heart may occur. Such changes in the myocardium are the commonest to note.

Fatty changes in the heart are seen in cases of anaemia and myocardial anoxaemia. Fats may be deposited into the subpericardial regions outside the heart or may be deposited as globules inside the protoplasm. The latter condition is very serious as it interferes with the contracti-

bility of the heart by upsetting the biochemical changes already described.

Myxoedema heart may be the seat of a constant type of change indicating the disturbance in protein metabolism as a result of hypothyroidism. The material deposited in the myocardium is mucin, a type of protein complex. It is seen not only outside the myocardial fibrils but also inside the protoplasm. Such a change interferes grossly with the proper functioning of this muscular structure, and circulatory disturbances follow. All the properties of cardiac muscle are uniformly affected. There is marked cardiac enlargement and flabbiness of the muscle. The change in the shape of the heart referred to as the ice-bag heart is very characteristic. Its slow contraction, snail-like due to interference with impulse origination and conduction defects, are noted on fluoroscopic study. Many patients of myxoedema die of progressive cardiac failure or coronary insufficiency as the arterioles share the same type of change.

Thyrotoxicosis :

The heart is dilated as a part of generalised wasting due to depletion of reserves. The sarcoplasm loses much of its glycogen and protein and becomes thinner and less elastic. The same change is noted in general body musculature also. Lymphocytic infiltration is also seen in between the myocardial fibrils. All these changes interfere with the properties of heart muscle.

Heart in Nephritis :

The changes differ with the stage of nephritis. In acute nephritis, cardiac enlargement is noted due to capillaritis and mechanically due to high blood pressure. Myocardial fibrils show fatty and hydropic changes — ischaemia is responsible for the fatty changes. Acute cardiac failure may be precipitated due to sudden dilatation of the heart.

In chronic nephritis, the changes are due to coronary arteriosclerosis and the raised blood pressure. Acute dilatation can occur due to sudden rise of blood pressure. But it is usual to note gradual progressive hypertrophy due to coronary sclerosis. In late stages myocardial fibrosis is the ultimate result.

CHANGES IN OLDER AGE GROUPS

Myocardium shows increased amount of fibrous tissue with replacement of myocardial fibrils in affections of the coronary arteries. Gradual sclerosis of these vessels induces myocardial anoxaemia. The intracellular connective tissue proliferates and often interferes with the myocardial function. In the heart, several foci of myocardial necrosis may be present due to obstructive sclerosis of vessels. The heart usually enlarges in size under these circumstances. Myocardial infarction is also a sequelae of coronary sclerosis when thrombotic episode is superimposed. Here sudden necrosis of the muscle fibres occurs with autolytic changes in the sarcoplasm and nucleii. The products are absorbed into the circulation. Softening of the myocardium is noted and reactionary changes occur. The infarct is usually *red* in the beginning, changing to *white* later. The muscle fibres show various changes from cloudy swelling to necrosis during the period following infarction,

Cellular infiltration occurs and finally scarring follows.

In cases of arteriosclerosis usually the subendocardial layer gradually loses its blood supply and fibrosis is noted. The size of the heart greatly increases and may be mistaken for cardiomegalies of metabolic origin.

Other Changes :

Protein deficiency gives rise to a myocardium whose fibrils are small and narrow with little dynamic activity. The sarcolemma which is the main constituent helping contraction is unable to function efficiently. Cardiac reserve is depleted and atrophic changes are noted — small heart with fatty changes. Later, intracellular oedema may occur due to progressive hypoproteinaemia. So, depending on the changes, the clinical findings vary. Tachycardia is noted in the early stages which is later replaced by bradycardia and other irregularities. Brown atrophy of the heart may resemble the heart of protein deficiency in the early stages. Here the atrophy is due to old age where lipochrome pigment accumulates on either end of the myocardial fibril, partially replacing the sarcolemma. The heart may be the seat of parasitic infection, Trichinella spiralis, taeniasis or tumour formation.

Secondary malignant deposits can occur in the heart muscle. Haemorrhages can also occur, but very rare. These are noted in various bleeding diseases. In bacterial endocarditis small cellular infiltrations are noted in between the cardiac muscle fibres (Bracht — Wächter bodies)

So from this discussion it can be understood that the heart, like any other organ in the body, can be the seat of various pathological changes, due to innumerable disease processes. Patients may succumb to such complications when the changes in the myocardium are gross. Very often these changes retrogress under proper treatment. The irreversible disorders of the heart are often primary and not secondary to diseases in other systems. These are mainly valvular and vascular. Certain myocardial affections may also produce irreversible changes.

Clinical evaluation of myocardial damage is often difficult unless the changes are advanced manifesting as cardiac enlargement, changes in rate and rhythm, changes in intensity of sounds. An electrocardiogram will be of much help to detect early myocardial inefficiency due to disease but interpretation and significance of changes noted is still not well understood.

In people who have severe allergic manifestation to foreign protein like serum, the myocardium has been the seat of specific changes. These features are always constant in many experimental animals. The most important pathological change is intermuscular oedema due to extreme dilatation of capillaries, a state resembling that of angioneurotic oedema seen on the skin. An associated eosinophilic infiltration has been demonstrated at times. In extreme cases, the haemorrhagic phenomenon is a sequelae of the severe capillary damage. Myocardial necrosis and oedema of the fibrils follow later with extreme dilatation of the muscle.

The same features have been noted in many diseases whose aetiology is allergic

due to infective coccal agents. The results are disastrous and not easily diagnosed clinically. Disturbances in the ionic equilibrium in the blood interfere with the proper functioning of the myocardium. K, Na, Ca, and Mg ions are absolutely necessary to keep up the rhythmicity and conductivity of the heart. In certain syndromes, e.g., uraemia, diabetes, loss of fluid by vomiting or diarrhoea, dehydration etc., or use of drugs like digitalis etc., the ionic level is disturbed. Various irregularities of the heart are noted and sometimes cardiac arrest.

Pathological examination may not reveal any myocardial damage, gross or histological. The cardiac arrest is due to interference with the heart's nutrition and contractility. It can function only when these ions are present in proper quantities. Experimentally it can be noted by perfusing the frog's heart with various ions. So very often, the cause of death is not made out in many illnesses. It may be due to changes in the pH and ionic equilibrium. As it is not a routine to use the electrocardiograph for diseases other than that due to the heart, the cause of death in many diseases is often missed.

The Scrotum in Clinical Surgery

by

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THE scrotum is Nature's "all-weather or an air-conditioned house" for the testes. In the animal kingdom, not every species has the scrotum, to accommodate the testes. Some mammals, chiefly aquatics, have the testes in the abdomen all the time. In some, the testis becomes extra-abdominal during the breeding season only. In the great majority, the testes are situated in a scrotum whose purpose seems to be to maintain them at a lower temperature than that of the rest of the body. In warm weather the scrotum relaxes; in cold weather it contracts, bringing its contained testes closer to the warmer trunk. In animals with scrotum, if the testes are warmed by an electric circuit, wrapping the scrotum with wool or by returning the testes to the abdomen, a previously fertile male is rendered sterile. The purpose of the dartos muscle, the hair, and the abundant sweat and sebaceous glands of the scrotum, seem to be to help the scrotum to keep the testes at a lower temperature than the rest of the body.

In the animal kingdom procreation is an important aspect of the life of animals. In the lower strata of animal kingdom, it is seasonal. But during this reproductive season, the very behaviour and attitude of the animals change. Eels leave their ponds and undertake a journey of thousands of miles over land and water to breed. Seals from the whole Pacific flock to the Aleutian Islands. Birds fly from South Africa to the cooler climate of Norway. The reproductive cells, at least the male ones, demand a lower temperature than other systems, for proper function. Probably Nature kept this in mind when later animals were to be evolved in the scale of evolution. Even as an automobile manufacturer improves his later models to rectify the defects of the previous models and to cope with fresh and newer necessities, Nature also has improved her "models", in this case the living beings on her vast market, that is the world. A motor car manufactured about 25 years ago is out of date today not merely by lapse of time but essentially because the requirements of an up-to-date car are different. The modern motor car among many other qualities, is an all-weather car. Even so, Nature, in providing accommodation for the testes in her later "models", has created the scrotum, an all-weather house. As the male reproductive cells demand a lower temperature than the rest of the systems of the body for a proper function, the mammals have been driven to the necessity of extruding the testes from the abdomen to which they belong. Hence the scrotum.

From a clinical point of view, the scrotum affords a site for a variety of diseases.

The structures inside the scrotum are : (a) the testes, (b) epididymis, (globus major, body and globus minor) (c) the spermatic cord (pampiniform plexus of veins, vas deferens and the cremaster), and (d) the tunica vaginalis. "Swellings" of the scrotum will be in connection with these contents ; (e) in addition, the skin of the scrotum itself is the seat of diseases. In South India the most important condition that involves the skin of the scrotum is filariasis, either as a lymph scrotum or filarial oedema, known as filarial scrotum. Among benign swellings, sebaceous cysts (or adenomata) and papillomata are the most important. Scrotal cancer, though infrequent, is of importance since a certain number of them may be industrial in origin.

Inguino-scrotal swellings are inseparable from the discussion of scrotal swellings. These also will be referred to in this discussion.

Clinical examination of a case of scrotal (or inguino-scrotal) swelling involves, inspection, palpation and transillumination. The aid of percussion and auscultation is invoked less frequently here than in other regions. No examination for a scrotal (or inguino-scrotal) swelling is complete unless the abdomen is also examined. In fact it is sometimes helpful to remember scrotum as the tenth segment of the abdomen for purposes of thorough clinical examination. An inguino-scrotal swelling (hernia) may reflect an increased intra-abdominal tension as a result of ascitis, tuberculous in origin in children and malignant in adults. Or a patient may enter a medical ward for dyspepsia or vague pain in the abdomen while he may be really having (enlarged) malignant para-aortic glands due to a testicular growth which the patient may not consider important or perhaps feels shy to speak out. With these general remarks, more detailed discussion can be proceeded with.

Inspection : Size, shape and symmetry of the scrotum and the condition of the skin itself must be noted. In addition, one should inspect whether the swelling is purely scrotal or extends on to the region of the inguinal canal — if so, whether it disappears now and then or becomes more prominent on coughing with an impulse. Now and then, in an inguino-scrotal swelling due to hernia, visible peristalsis of the intestines may be seen.

Palpation : During palpation, the scrotal contents can be examined individually in greater detail. The first step is to determine whether the swelling is purely scrotal or is inguino-scrotal. This is ascertained by performing the test of "getting above the swelling". The spermatic cords are palpated between the thumb and fingers (left hand for right side and right hand for left side) and the structures rolled between the digits. The spermatic cord will be felt as a bundle of soft strands running longitudinally, the contents being the fibres of cremasteric muscles and veins of pampiniform plexus among which the vas deferens can be felt with the consistency and thickness of an ureteric catheter. In a purely scrotal swelling, one can get above the swelling.

The spermatic cord leads down to the testis. During palpation the anatomy of testis, epididymis (the three portions of epididymis) and tunica vaginalis with reference to each other must be recalled.

Before discussing purely scrotal swellings, the inguino-scrotal swellings may be mentioned in passing. The most important inguino-scrotal swelling is an *inguinal hernia,* with its reducibility and impulse. This need not be elaborated further here. Next in importance is the *varicocele,* that fills up on walking or standing for some time and empties gradually if the patient lies down. The feel of a varicocele is aptly described as that of a "bag of worms". At the risk of repetition, it may again be pointed out that abdominal examination should never be omitted, since a varicocele (on the right side especially) may be secondary to a renal neoplasm and an inguinal hernia may be secondary to some abdominal pathology. A very elusive condition is diffuse *lymphangiectasis* of the cord. This gives a soft diffuse swelling of the spermatic cord, spongy to the feel and only very gradually reducing in size on continued pressure of squeezing. Usually there is no impulse on coughing. If the swelling is substantial trans-illumination may be demonstrated. This condition must be borne in mind and recognised since it may be mistaken for a hernia or a varicocele. The condition does not need an operation. The patient needs assurance and reassurance.

Abdomino-scrotal or bilocular hydrocele is a very interesting, though infrequent condition. In this condition the fluid-containing-sac extends from the scrotum along the inguinal canal into the abdominal cavity (not the peritoneal cavity) but the sac is closed at both ends. The swelling is inguino-scrotal and exhibits an impulse on coughing. But it is never reducible (but not strangulated or obstructed hernia, since but for the swelling, the patient has no abdominal symptoms). It is persistently dull on percussion and is brilliantly transluscent as it contains only clear fluid. Aspiration will settle the diagnosis but this procedure is neither desirable nor necessary. In infantile variety of hydrocele the findings are more or less similar but the swelling abdruptly stops at the level of internal ring and there is no "intra-abdominal" extension behind the level of internal ring.

An encysted hydrocele of the cord does not give rise to much difficulty in diagnosis. Other conditions to be remembered are metastasis in the lymphatics of the cord, innocent tumours of the cord like myoma, lipoma and neuro-fibroma, and haematoma of the cord.

If the spermatic cord is normal at the level of the neck of the scrotum, the testis must next be palpated.

Hydrocele : The commonest purely scrotal swelling is a *hydrocele* of the tunica vaginalis. In this condition, neither the testis nor the epididymis can be palpated but there is a diffuse ovoid swelling due to distension of the tunica vaginalis with clear fluid. Transillumination will settle whether the fluid is clear (hydrocele) or blood (hematocele).

Hydroceles may be *primary* (with no apparent cause) or *secondary* as a result of disease in the testis or epididymis. The clinical student should always be alert to spot secondary hydrocele however infrequent they may be. This would be possible if cursory examination is given up and detailed methodical examination is done. A primary hydrocele is a straight

forward problem a non-destructive operation and a permanent cure. But a hydrocele secondary to tuberculosis or syphilitic or malignant disease of the testis is significant since it alters the prognosis. It involves the question of duration of life of the patient after operation if it is secondary to malignancy in the testis. Orchidectomy in tuberculosis is as much a responsible decision as it is an irresponsible omission if orchidectomy is not done in malignancy due to the failure of the surgeon to recognise the condition. The collection of fluid in the sac is not itself important in secondary hydroceles but is important because of the fact that it masks the outline of the testis and hence the disease in it. In cases of doubt one should not hesitate to tap the hydrocele and examine the testis and epididymis for evidence of disease.

A primary hydrocele is the commonest of all scrotal swellings. It is a smooth diffuse swelling and is fluctuant and transilluminates very well. There is frequently a history of trauma. Usually this is symptomless and the patient seeks advice because of its weight or because it attracts too much attention. Sometimes there may be a circular constriction. The testis is situated behind as can be demonstrated by transillumination, if not by palpation. If the fluid is under great pressure the swelling is tense and elastic to the feel rather than fluctuant. Usually the condition is bilateral though they may vary in size.

The fluid of a hydrocele when tapped is clear, golden yellow (straw coloured is the usual description) and contains good quantities of albumin. Hence it is soapy on the fingers, frothy as it is poured into a receiver and soon goes solid nearly like the white of egg. The fluid contains fibrinogen but no thrombin, so that the fluid clots only when blood or tissue fragments are added but not before. In long standing cases the fluid may be brownish and may contain shimmering cholesterol crystals.

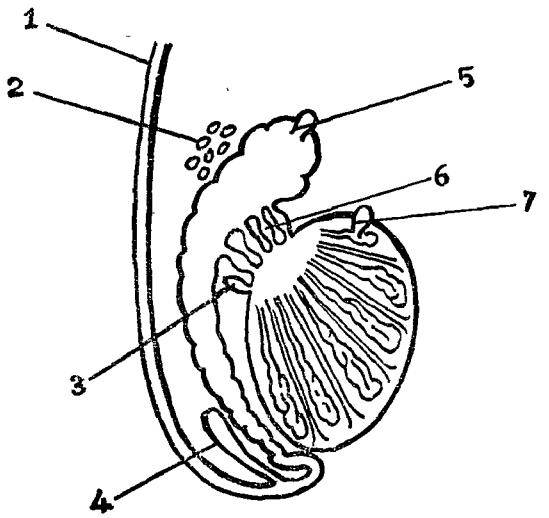
Haematocele :— The tunica vaginalis is distended with blood or blood-stained fluid. The latter variety is more important since they are secondary to lesions of testes, a neoplasm or torsion. Those that contain blood are the result of trauma or due to spontaneous rupture of a vessel in the cord. Diagnosis is made by the fact that though it is a fluid-containing swelling, it is too heavy for its size and that it does not transmit light. Tapping confirms the diagnosis.

Cysts of the epididymis are important in the differential diagnosis if not for anything else, at least for the fact that owing to their infrequency of occurrence, one is not very familiar with these and hence a student is often confused when faced with a case. Cysts of the epididymis do not resemble and are not pathologically related to the other lesions of epididymis which are inflamatory in origin.

The essential points of difference between hydrocele and cysts of epididymis are given below :

	Hydrocele.	*Cyst of epididymis.*
Shape :	Smooth and ovoid.	Group of irregular knobs.
Tension :	Tense.	Usually soft.
Testes :	Not felt.	Can be felt separate from the swelling. An epididymal cyst of moderate size may feel like another testicle.
Transillumination :	With some difficulty in the older hydroceles.	Shines brilliantly (said to shine like a Chinese lantern)
Fluid	Clear, yellow,high specific gravity around 1025.	Opaque and milky — low sp. gr. around 1005.
	Goes solid on boiling.	Slight cloud on boiling.
Microscopy :	No cells, often cholestrin crystals.	Spermatozoa, usually mobile, occasionally dead in more than 90 per cent.

Vestigial structures associated with the testicle
(After Ogilvie.)



1. Ductus deferens
2. Paradidymis
3. Appendix epididymis
4. Vasa efferentia
5. Appendix testis
6. Superior vas aberrans
7. Inferior vas aberrans.

Reference to the diagram will show the structures in connection with which cysts may occur. With the exception of appendix epididymis and organ of Giraldes (both are mesonephric remnants), the tubular structures from which cysts may arise communicate with the seminiferous system. Hence the majority of epididymal cysts are spermatoceles. They contain milky fluid with spermatozoa or their parent cells. These spermatoceles are single or contain groups of two or three cysts and are usually large, quite often larger than testis. They are unilateral and

seen during middle life. Epididymal cysts containing clear fluid probably arise in the organ of Giraldes since they are situated in the head of epididymis. They are bilateral, multiple, each cyst seldom exceeding the size of a pea. They occur in older men and are discovered accidentally.

Testis & Epididymis :— The testis and epididymis must be separately palpated. The diseases involving the epididymis and testes are different in nature, though disease arising in one may eventually spread to the other. The epididymis is the seat of acute, subacute or chronic infections ; the testis, of viral, spirochaetal and malignant lesions.

Swellings involving the body of the testis

1. *The orchitis of mumps* :— Difficulty in diagnosis will arise in this case only if the testis gets involved in the absence of parotitis.

2. *Syphilitic orchitis*:— The salient features are enlargement of testis, painlessness, loss of testicular sensation and a small secondary hydrocele.

(a) *Congenital syphilitic orchitis* :— occurs in infancy and up to about the 3rd year of life. This is a diffuse orchitis resulting in a functionless testis. Gumma does not usually occur.

(b) *Acquired tertiary syphilis* :— Diffuse syphilitic orchitis gives rise to an enlarged painless testis with loss of sensation. Other stigmata of syphilis should be looked for. The gumma is a more localised lesion in the testis which may go on to the typical anterior ulcer with wash-leather slough.

3. *Tumours of the testis* :— As a rule they are malignant. The two varieties are the teratoma and the seminoma (seen between the ages of 20 - 40) the former occurring at an earlier period of life and the latter, rather later in the life. The subject of testicular tumours is interesting and vast and their pathology is full of controversies. Only their salient clinical features may be mentioned here. Testicular tumours present themselves as diffuse enlargements of the testes. They vary in size. Since they may be painless they may go unrecognized in the beginning. For a time the tumour remains confined within the tunica albuginia. The epididymis is stretched over the back of the tumour. There may be a small hydrocele. Undescended and ectopic testes are about 43 times more prone to the development of malignancy than normal testes (Campbell). Metastasis occurs chiefly by the lymphatics to the para-aortic glands, and also by the blood stream to the liver, lungs, and bones. Both seminoma and teratoma produce gonado-trophic substances in the urine. The A. Z. test in the urine may be positive in both. Both tumours are radio sensitive but seminoma more so. The differential diagnosis from tertiary syphilitic enlargement of testis may sometimes be impossible on clinical grounds. The naked eye appearance of cut section of seminoma and teratoma are different and distinct.

Epididymis : The swellings of the epididymis are almost all of them inflammatory. I have come across two cases of leiomyo-sarcomata of epididymis.

The important types of epididymitis are gonococcal, septic (B. Coli and streptococcal) the tuberculous and filarial. Of these, except the tuberculous variety the rest are usually acute or subacute in onset. The epididymis is enlarged as a whole and is tender. The scrotum on the affected side is hot and tender. In the gonococcal variety previous history of urethral discharge might be available or there may be evidence of gleet. Prostatic massage and examination of the smear may reveal gonococci. Septic epididymitis may follow prostatectomy, catheterization, or operations on the urethra. Gonococcal epididymitis usually subsides without suppuration, definitely so with penicillin. Septic epididymitis may end in abscess formation. Filarial infection is often an epididymo-orchitis and funiculitis with sudden onset accompanied by fever and rigon. Acute inguinal adenitis is very often an accompaniment. Blood examination during the acute fever may show microfilaria.

The clinical picture of tuberculous epididymitis is different but quite characteristic. The disease usually commences in the globus minor, then extends to the other parts of the epididymis and may ultimately extend by continuity into the body of the testis. Very often the disease is bilateral when seen. Clinically, the globus minor is felt as a hard craggy nodule (occasionally the globus major too); in addition, the vas deferens is thickened and beaded, a very characteristic finding. If the disease has progressed to the stage of caseation and liquefaction, a posterior sinus adherent to the epididymis may be present. Rectal examination may reveal tuberculous involvement of the prostate and seminal vesicles which are enlarged, irregular and knobby. Evidence of tuberculous infection of the urinary tract may be obtained in some cases on thorough investigation. Other tuberculous lesions of the body such as P. T., lymphadenitis or arthritis must be looked for. Most of the urologists are of the view that tubercle bacilli reach the epididymis from a focus in the prostate in a retrograde fashion along the seminal tract, though embolic (blood borne) infection is also possible.

Finally, there is one condition that involves the testis and epididymis simultaneously. That is torsion. This is an acute condition, mostly occuring in undescended testes — 400 times more common in undescended than in the scrotal testes. Torsion can occur at any age, but more common in children and adolescents. The condition is acute in onset and is accompanied by severe pain, vomiting and shock. If examined immediately after the onset a tense and very tender swelling lacking the outline of the testis will be found at a level higher than the normal level at which the patient had his testis before. This is because during torsion the cord gets tightened and shortened. After some time a haematocele develops and the scrotum is oedematous. Hamilton Bailey's test shows that twisting the swelling in one direction (in the direction of torsion) aggravates the pain, while twisting

in the opposite direction gives a momentary alleviation of pain.

In the above pages, I have discussed the common and important scrotal and inguino-scrotal swellings and their differential diagnosis. Discussion on treatment is beyond the scope of this article.

Happiness is the only good.
The place to be happy is here.
The time to be happy is now.
The way to be happy is to make
others so.
— *Robert G. Ingersoll*

* * * *

Fame is the perfume of heroic deeds.
— *Socrates*

* * * *

We toil for fame,
We live on crusts;
We make a name,
Then we are busts.
— *L. H. Robins*

* * * *

He who has a thousand friends has not a friend to spare,
And he who has one enemy shall meet him everywhere.
— *Emerson*

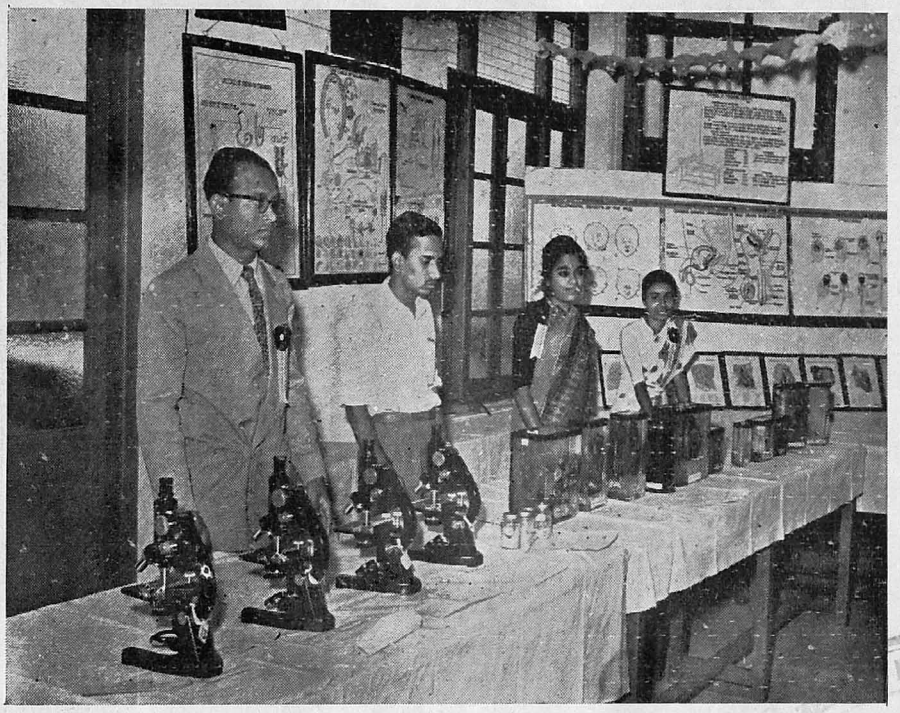
* * * *

Beware the fury of a patient man.
— *Dryden*

* * * *

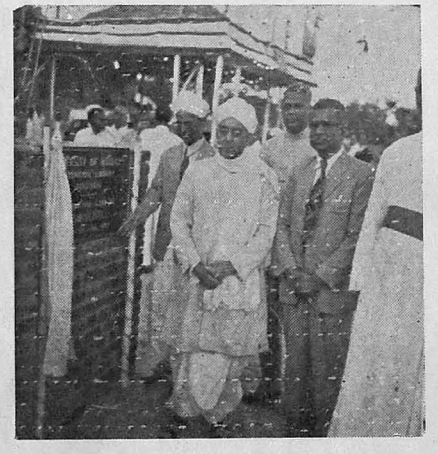
Do all the good you can,
By all the means you can,
In all the ways you can,
In all the places you can,
At all the times you can,
To all the people you can,
As long as you can.
— *John Wesley*

University Centenary Celebrations at Madurai



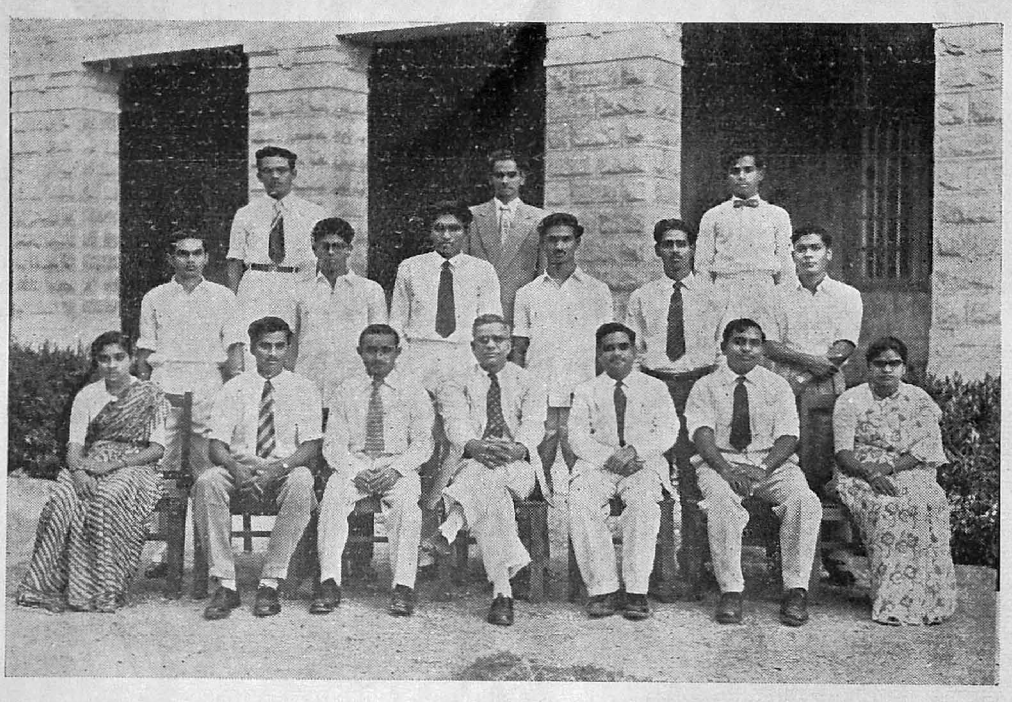
EXHIBITIONS — MEDICAL WING

Victory–Centenary Sports



Foundations for a New Library

Photos–Courtesy—Mr. S. Jamal



MEN'S HOSTEL COMMITTEE 1956 — 57

WOMEN'S HOSTEL COMMITTEE 1956 — 57

The Evolution of Anatomy

by

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THE earliest known portrait of a medical man is a picture found on the walls of the Trois Freres cave in the Pyrenees. This ancient drawing resembles that of an African witch-doctor. Several tools and weapons have been recovered and described as being used by the pre-historic man. It is observed that the Australian natives still use the flint knife in ritual circumcision. It is quite possible that some of the stone implements were used as surgical instruments. Pre-historic skulls have been found which bear the evidence of trephining. During this period, trephining of the skull was done as a magical rite or to cure certain ailments such as head-ache, epilepsy, or insanity.

The preservation of the human body after death is a strange custom started first in Egypt. The dead bodies which were kept in the hot sands of the desert remained dry and became hard. The bodies were in a state of preservation. This suggested to the Egyptians to try other artificial methods of preserving the cadaver. The preservation of dead bodies was practised in Egypt from about 4000 B.C., to 600 A.D. It is approximately estimated that during this period about 700 million bodies were embalmed, and this was a laborious method. Although the bodies were preserved they did not take any interest in the study of the structure and functions of the human body. The Babylonians distinguished between the arterial and venous blood; but they did not know that there are separate vessels carrying arterial and venous blood. In fact they did not understand the circulation of blood. They also believed that certain animals had a knowledge of the future denied to man. This knowledge was reflected in the shape of their liver. So they began to study the anatomy of the liver with the help of models.

Even though the Egyptians practised embalming, they learned very little of Anatomy. Among the Persians, Georg Sticker believed that the fear of contracting diseases from a dead body may be traced to epidemics of plague and anthrax. Even in those early periods, the Chinese and Hindus had some knowledge of Anatomy. The Asian peoples were very much interested in the study of bones. The Hindus supposed that there were 360 bones in the human body. The Hindus further described the heart in its proper place as a round, smooth, boneless organ with a cavity inside. They also described a net-work within the heart. Probably they referred to the trabeculæ carneæ, papillary muscles, and chordæ tendineæ.

The ancient Hindus were also good surgeons with very good anatomical background. Susruta described a number of surgical instruments which were used by him. This work should be considered as

very bold and distinctive. Anatomy was taught on models made from wood, ivory, hollow stems of plants, leaves etc. In this connection it is worth noting that two articulated skeletons made of sandalwood and ivory with good anatomical details have been found in the Tanjore Palace in South India.

Although the Egyptians practised embalming, the Greeks had the spirit of inquiry and thirst for knowledge for the anatomical details of the human body. As far as we are concerned, we must consider Hippocrates as the father of Medicine. Hippocrates was supposed to be a descendant of the fabulous Aesculapius. His contribution to Anatomy was not very much ; but he formulated the methods for use in clinical medicine. Galen was born at Pergamus during the Roman period. During this period of Roman history, the anatomists relied more upon the Barbary ape and not on human specimens. Galen has dissected and describes platysma, buccinator, levator palpebræ superioris, palmaris longus, interossei, plantaris etc., which were originally called by numbers. He was also very much interested in Osteology and advised his students to study this branch in detail. He first described cranial nerves in seven pairs.

During the earlier period, the teaching of anatomy was purely theoretical as people were against dissecting a dead body. Later permission was given to dissect the executed criminal. At the University of Tubingen, it was the custom to dissect once in every three years the body of an executed criminal. All who had taken part in this, shall attend a mass for the individual's soul and later shall accompany the remains to the grave. The dissection was open to scholars and professional men only. No attempt was made to dissect the nerves, vessels and muscles. Under the guidance of Mundinus (1275—1326) the anatomical dissection was considered an essential basis for the medical curriculum. Like Galen and other workers of this period, he also committed many errors. The common among them were as follows : The heart was described as having three ventricles. The liver was said to consist of five lobes. He described the brain to be under the control of a red worm (Choroid plexus) The uterus was described as having seven chambers.

During the 14th and 15th centuries there were many great artists who were interested in the structure of the human body. They engaged themselves in dissection. They were Michael Angels, Raphæl and Albrecht Durer. Greatest among the artists who showed interest in anatomy was Leonardo da Vinci (1452—1519). He was a very gifted versatile artist. He has also made discoveries in anatomy. He was the first to demonstrate that there are air sinuses inside the cranial bones. He also demonstrated the ventricles of the brain by means of wax injections. He must be regarded as a leading pioneer in anatomy.

The foremost anatomist next was Andreas Vesalius (1514—64). He belonged to a medical family in Brussels. His father was an apothecary to Emperor Charles V. He first dissected dogs, mice and other small animals. He went to Paris later to study Medicine. His teachers were Sylvius and Guinther. For six years Vesalius remained in Italy dissecting, studying and teaching at Padua, Bologna and Pisa where countless students had already flocked to his classes. Sylvius was a close follower of Galen. When the structure of the human body did not con-

form to the description given by Galen, he said that the human body must have become changed since Galen's time.

Guinther made no great contribution to anatomy. But he was a very good teacher in anatomy and he recognised and encouraged the work of young Vesalius. Vesalius wanted to learn more and so he undertook at great personal risk "body-snatching expeditions." When he was working in Italy, he found that Galen's teaching was all wrong and could no longer be regarded as a final authority. He was also interested in anthropology. His ship was wrecked on the island of Zante at the entrance to the Gulf of Corinth and there in October 1564 Vesalius died.

Gabriele Fallopio (1523—1562) of Modena studied under Vesalius at Padua. In his twenty-fourth year, he was appointed as Professor of Anatomy at Ferrara and in 1548, he accepted the chair of Anatomy at Pisa. In 1551, he obtained the chair of Vesalius at Pauda. He was a very good dissector and described the structures in great detail. After Fallopio came Contanzo Varolio (1543--1575) who was an eminent anatomist and surgeon. He was very much interested in the study of the brain. Archangelo Piccolomini (1526—1605) taught anatomy at Rome. Other great anatomists during this period were Columbus and Cæsalpino.

Before 1600, Anatomy and Physiology were taught together. The Seventeenth Century was an unsettled time in Europe. Nathaniel Highmore (1613—1684) a Physician at Sherborne demonstrated the difference between the lacteals and mesenteric veins. Thomas Bartholin (1616—1680), Francis Glisson (1597—1677) Johan George Wirsung (1600—1643), Thomas Wharton (1610—1673), Niels Stenson (1638—1686) are all well known to the students of Anatomy. These anatomists will recall to the mind of the reader who knows his anatomy, a duct, a gland or some other anatomical structure named after the discoverer. During this period Hendrik Meibom (1638—1700) is credited with the discovery of the Tarsal glands.

In the latter part of the 17th century contributions were made to the study of the glands by two Swiss students Johann Conrad Peyer (1653—1712) and Johann Conrad Brunner (1653—1727) after observing these glands in the intestine of the cat. Marcello Malphighi (1628—1694) was born near Bologna and studied Philosophy and Medicine there and received the doctorate in 1653. He held professorship at Pisa, Messina, and Bologna. In 1691 he was physician to Pope Innocent XII.

During the 18th century, a worthy successor to Malphighi was Antonio Maria Valsalva (1666—1723). He was very much interested in the correlation between Anatomy and Physiology. G. D. Santorini (1681—1737) was the Professor of Anatomy at Venice during this period. His name is well known to the students in conjunction with the accessory pancreatic duct. Jacques B. Winslow (1669—1760) of Odensee, Denmark was Professor of Anatomy at Jardin Royal in Paris.

During the 19th Century Henle was an eminent anatomist and later Hyrtl (1820—1894) who developed the technique of making "corrosion preparations" — the blood vessels of an organ injected and other tissues dissolved away by acid so that all the vessels were seen clearly. Every student of anatomy is no doubt familiar with the name of Henry Gray (1827—61) Lecturer in Anatomy at St.

George's Hospital whose early death from small-pox was a great loss to the medical science. Embryology received its stimulus from Carl Ernst von Baer (1792—1876) of Konigsberg. Anthropology owed much to Paul Broca (1824—80). Among the eminent anatomists during the 19th century may be mentioned Charles Bell, George A., Piersol, Franklin, Paine Mall, Franz Kiebel, Karl. F. Burdach, J.F.G. Henle, His, Camillo Golgi, Waldeyer and others.

To-day the science of anatomy and its affiliated fields are again expanding. The department of anatmy has included radio-graphic anatomy, physical anthropology, statistical methods, topographical anatomy, surgical anatomy and dynamic anatomy which were previously unknown or used only to a very limited extent.

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I was angry with my friend
I told my wrath, my wrath did end.
I was angry with my foe ;
I told him not, my wrath did grow.

— *William Blake*

* * * *

If a man can write a better book, preach a better sermon, or make a better mouse-trap than his neighbour, though he builds his house in the woods, the world will make a beaten path to his door.

— *Emerson*

* * * *

Hating people is like burning down your own house to get rid of rat.

— *H. E. Fosdick*

Why I Eat Meat

by

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1. *Nature* :—

Those with fangs eat those without fangs ; those with fangs and claws eat those with fangs ; those with hands eat all the above is the natural law. This is well expressed by an English adage :— Frogs (or toads) eat worms, snakes eat frogs, pigs eat snakes, man eats pig and worms eat man. This is the natural cycle.

2. *Evolution* :—

The evolutionists declare after careful study that the lower strata of life exist for the sustenance and evolution of higher life. So, to kill and eat such lower strata of life is not a sin.

3. *Chemistry* :—

The rabbit is the most wonderful chemical factory in the world to convert inedible food into edible food. But for this wonderful live factory, grass will be of no use to man. So with other herbivorous animals and birds. Ordinarily man eats herbivorous animals and birds and not carnivorous animals or birds.

4. *Practical Life* :—

The Chinese, who had suffered more often from famine than any other race declare that everything in this world is for the use of Man. So they say that whatever can be used as food should be used for food. The following anecdote will explain this. A farmer said that he was going to slaughter a fattened pig the next day. The pig on hearing this, was very sad and asked the cow in the byre why such a big beast as she is not killed. The cow replied 'I yield while I live and you after you are dead'. This is a trite saying. The cow is endowed by Nature with four udders capable of producing milk over and above the needs of her offspring, which extra milk man uses for himself. If the cow produced only a small quantity sufficient for her young only, then she will be put to the plough or probably killed for her meat. A cow is the most docile of all domesticated animals. Further, the milk that she yields is from consuming that which man cannot consume. The cow eats grass which man cannot eat as menticned above. Man takes the paddy and throws the straw to the cow. He takes the rice and gives the bran to the cow. He makes use of the cotton fibers and gives the seeds to the cow. He extracts oil from oil seeds for himself and feeds the cow with oil cakes. Having thus fed himself with the best, he throws the refuse to the cow which she eats and produces milk of which he gives a small quantity to her calf and takes the maximum quantity of it for himself. Thus the cow-adoring humanity is utterly selfish in its adoration. Because the cow yields while it lives he cherishes it but he kills the pig when mature, if not kept for breeding, and eats its meat.

5. *Balance of Life and Self Preservation*:-

One of the pioneers to Australia introduced a few pairs of rabbits into the country thinking then that when they multiplied he can shoot a few for his rab-

bit pie. Now ? They have multiplied in such large numbers that the Australians have to kill them off in thousands to preserve their wheat fields as otherwise they will be starved to death. They have erected thousands of miles of wire fence to protect their cultivation. The Government offers rewards to the destroyers of these pests. It is said that in the Uttar Pradesh there are as many monkeys as there are people. What these monkeys destroy is more than what they can eat.

Thus it is evident that man must destroy the excess of such pests for self preservation. Again, in 1871 and before, the wild buffaloes that roamed in Kansas (N. America) in vast numbers, had to be killed as they destroyed more than what they ate and no man-made fence could keep off these beasts from cultivated lands. Further, such indiscriminate destroyers were replaced by cattle and sheep which could be controlled and kept within limits.

Thus, it is evident that the Balance of Life must be maintained.

That man is great, and he alone,
Who serves a greatness not his own,
For neither praise nor pelf :
Content to know and be unknown :
Whole in himself.

— *Owen Meredith*

* * * *

Babies haven't hair ;
Old men's heads are just as bare ; —
Between the cradle and the grave
Lies a haircut and a shave.

— *Samuel Hoffenstein*

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The best sauce for food is hunger

— *Socrates*

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To be ignorant of one's ignorance is the malady of the ignorant.

— *A. B. Alcott*

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It is a principle of human nature to hate those whom you have injured.

— *Tacitus*

Blood Changes During Drug Administration

by

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IN the treatment of many diseases, we are using day in and day out several pharmacologically potent drugs. These may be used mainly in therapeutic doses but occasionally in larger doses and for prolonged periods. It is but natural that a therapist must be aware of the toxic or other potentialities of the drugs he is using and be prepared to deal with them suitably as and when the occasion arises.

Of the many toxicities that may be manifested by the drugs, we are now focussing attention on the haematological changes that may occur concurrent with the therapeutic or toxic use of drugs.

A preliminary definition of some of the common haematological changes that may occur, will be pertinent to this subject.

The blood picture may show manifold changes in poisonings and these may be of great importance for the diagnosis.

Polycythemia is characterized by an increase in the proportion of red blood cells per volume unit of blood. It is considered a compensatory mechanism due to hypoxemia and therefore may result from congestive heart failure, pulmonary edema, changes of the blood pigments, and high altitude. Edematous conditions, especially of the lungs, and dehydration because of severe diarrhœa, may also reduce the amount of circulating plasma and thus increase the relative number of red blood cells.

Anemia may be due to a destruction of the red blood cells, injury of the blood-forming organs, and loss of blood because of extensive hemorrhages.

Hemolytic anemia is characterized by an active destruction of red blood cells at a rate greater than that of their production by the blood-forming organs; an increased fragility of the red blood cells and an increased number of microcytes and reticulocytes. It may be due to hyperactivity of the spleen, but in most instances it is caused by an injury of the cell membrane from toxic substances.

Aplastic anemia is characterized in most cases by a more or less severe inhibition of the activity of the bone marrow as indicated by a decrease in the number of red blood cells, granulocytes, and thrombo-

cytes. The hemoglobin is decreased in proportion to the erythrocytes, so that the color index may be little changed. In aplastic anemia there is no evidence of regeneration, as illustrated by the absence of reticulocytes, normoblasts, and stippled cells. There may be a relative lymphocytosis.

Pernicious anemia is characterized by a marked reduction of the red blood cells and there may be a high degree of poikilocytosis and anisocytosis, polychromatophilia, stippling, and basophilia. Nucleated cells and megaloblasts are seen frequently. In contrast to aplastic anemia, the hemoglobin is not reduced in proportion to the number of red blood cells, so that the color index is consistently above 1.

Pathological erythrocytes are often present in the circulating blood of anemic patients. Most common defects are *anisocytosis* and *poikilocytosis*. The former is characterized by red blood cells of varying diameter and the latter by erythrocytes of varying shape ranging from oval to very irregularly shaped cells. They are seen in severe anemias of other than the aplastic type.

Nucleated red blood cells and reticulocytes are immature erythrocytes which are thrown into the circulation in severe anemias in which there is an active regeneration in the bone marrow. They may, therefore, be found in anemias from blood loss, hemolytic anemias, and pernicious anemias.

Shadow cells are colorless erythrocytes seen in hemolytic anemias and in some poisonings. *Polychromatophilia,* characterized by an increased affinity of the erythrocytes for Wright's stain, may be seen especially in pernicious anemia. *Stippled cells* are basophilic red blood cells. They are indicative of erythrocytic regeneration and may be seen in anemias in which the bone marrow is active or hyperactive as in lead poisoning.

Thrombocytes or blood platelets are intimately connected with the coagulation of the blood, being the chief source of prothrombin in the blood plasma. Their decrease, *thrombocytopenia,* may be due to injury of the bone marrow, specifically the megakariocytes, or to the destruction of thrombocytes in the circulation.

A phenomenon which may be helpful in the diagnosis of certain poisonings is the presence of *Heinz bodies* in the red blood cells. These are round, oval, or serrated very refractile granules, one or more in each cell, and sometimes protruding from the cells. Their nature is not definitely established, but they appear to consist largely of denatured protein and to be indicative of some injury to the erythrocytes, which, if severe, may lead to hemolysis and anemia. The presence of Heinz bodies is often, but not necessarily, associated with methemoglobinemia.

Many poisonings are associated with an increase or decrease in the number of *white blood cells.* An increase of the leukocytes, *leukocytosis,* is indicative of infections or inflammatory processes. It may be seen in poisonings subsequent to primary injuries such as pneumonia, ulcerations, and corrosions, hemorrhages, and liver injuries. *Lymphocytosis* may only refer to the ratio between leukocytes

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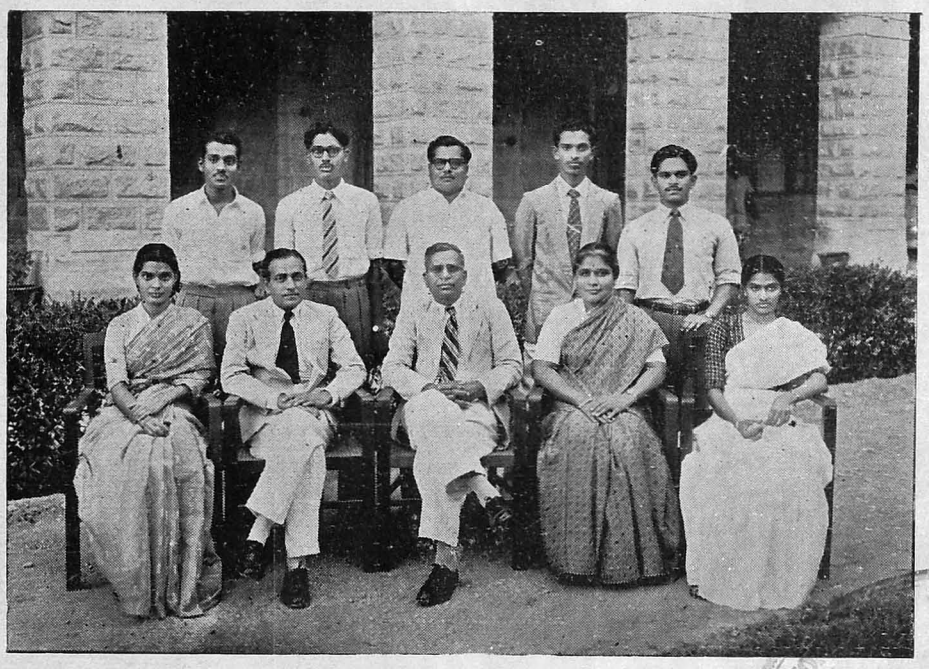

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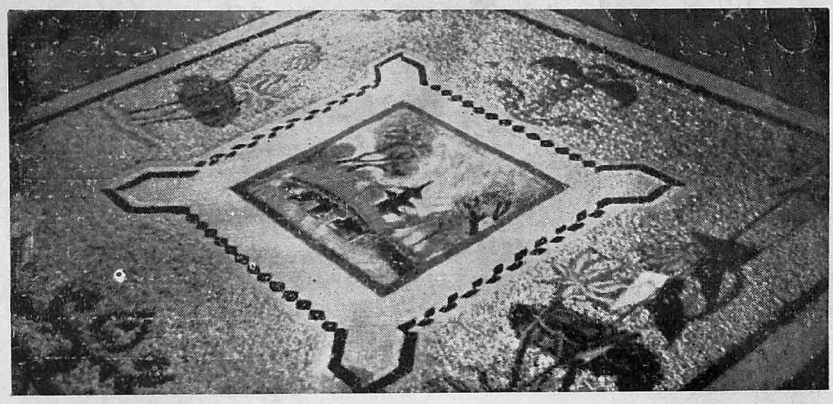

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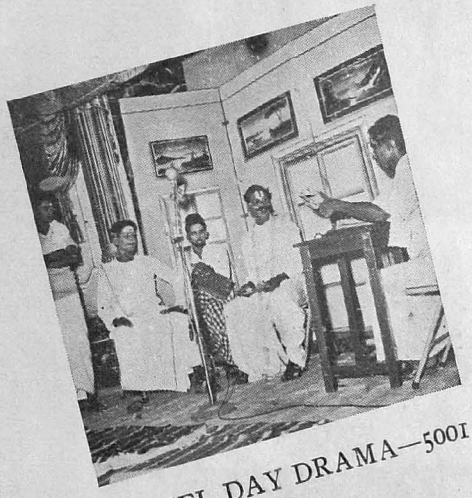

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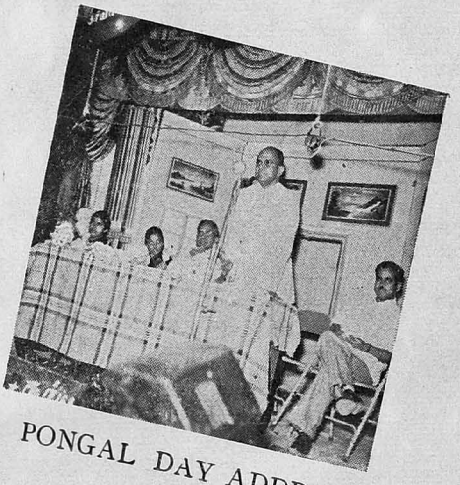

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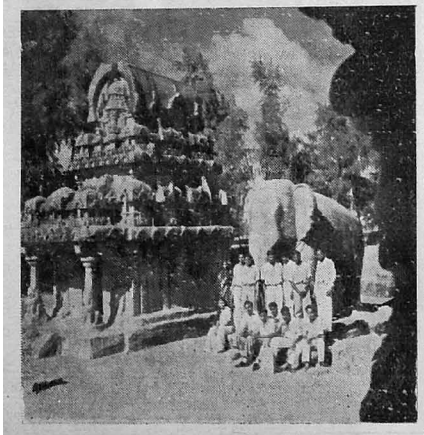

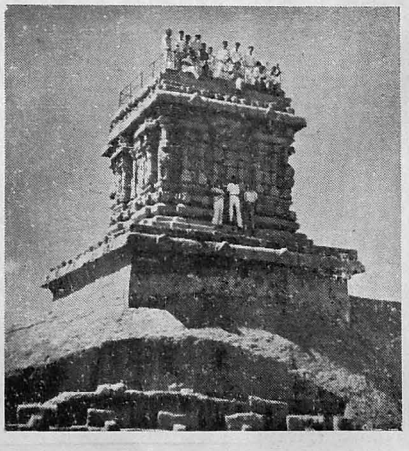

AT MAHABALIPURAM

Photos-Courtesy S. Jamal.

and lymphocytes in absence of an increase of the total white cell count — *relative* lymphocytosis. If the number of white blood cells is increased because of an increased number of lymphocytes it is called *absolute* lymphocytosis. The former may be seen when the hematopoietic activity of the bone marrow is depressed, so that a smaller number of leukocytes is thrown into circulation, and the latter may be indicative of a stimulation of the lymphopoietic tissue.

Eosinophilia, or an increase of the basophils, is often associated with hypersensitivity to plants, drugs, and chemicals.

Leukopenia is characterized by a decrease in the number of leukocytes. If mild, it may be indicative of poor nutrition, if more pronounced it is indicative of a toxic inhibition of the hematopoietic functions of the bone marrow.

Agranulocytosis, a marked reduction of the number of white blood cells, especially of the neutrophils, is often associated with a relative lymphocytosis and sometimes with a high platelet count. The clinical picture of agranulocytosis is characterized by fever, prostration, and septic throat. It is indicative of the absorption of some leukocidal poison and of injury to the bone marrow, and it is often due to hypersensitivity to certain drugs or chemicals.

In an examination of the blood, the first noticeable characteristic is its color. *A bright red color* of the blood may be indicative of changes of the blood pigment as produced by carbon monoxide, of interference with the oxygen consumption as caused by a cyanide, of changes of the circulation, and of the absorption of organic acids.

A dark color of the blood may be indicative of decreased oxygen absorption as in pulmonary edema or of increased oxygen consumption as produced by dinitrophenol.

A dark color of the blood is very often due to *methemoglobinemia.* Methemoglobin is an oxidation product of hemoglobin and differs from oxyhemoglobin in its brown color, its inability to give off oxygen, and its absorption spectrum.

The fragility of the red blood cells may be increased, or their resistance to hypotonic salt solutions decreased, in certain poisonings which may lead to hemolytic anemia and hemolytic jaundice.

Decreased fragility of the red blood cells, characterized by a greater resistance to hypotonic salt solutions, has been explained as a "tanning" effect of some sort on the cell membrane, or by a selective destruction of the less resistant cells.

The sedimentation rate of red blood cells is influenced by a number of factors such as the specific gravity of the blood cells, the viscosity of the plasma, the corpuscle size, the protein composition of the serum, and its pH. An increased sedimentation rate is seen in pregnancy and in the presence of inflammatory processes.

The *coagulation time,* the interval between the withdrawal of the blood and its conversion to a clot, may be indicative of a number of pathologic conditions. *Increased* coagulation time will result in a tendency to prolonged hemorrhages and may be indicative of thrombocytopenia, hypocalcemia, obstructive jaundice, and infectious processes, especially pneumonia.

Haematological changes produced by a number of drugs are described below :-

CENTRAL NERVOUS STIMULANTS

Caffeine — shortens the prothrombin and coagulation times of the blood, similar to theophylline and theobromine.

Amphetamine — shortens clotting time.

CENTRAL NERVOUS DEPRESSANTS

HYPNOTICS :

Barbiturates— blood changes—marked leucocytosis with eosinophilia (73,500 leucocytes with 32% eosinophils), myelocytes and erythroblasts in the peripheral blood, agranulocytosis. Anaemia has also been reported.

Chronic poisoning — haemolytic anaemia, anaemia, (porphyrinuria).

Sedormid — Many cases of thrombocytopoenic purpura have been observed after the use of sedormid. There is some reason to try BAL, when the thrombocytopoenia does not subside spontaneously. It is possible to use this drug regularly; yet one tablet may suddenly cause a serious thrombocytopoenia. Not only skin haemorrhages but also mucosal haemorrhages may occur in such cases. (It has been observed to cause melena, epistaxis and hematuria).

Carbromal — (Adalin) — thrombocytopoenia.

ANTI-CONVULSANTS :

Sulphonal, methylsulphonal — (haematoporphyrinuria)

Chloral hydrate — purpura.

Phenurone (Phenylacetylurea) — leucopoenia, thrombocytopoenia, fatal aplastic anaemia.

Hydantoin derivatives — Nirvanol (Phenylethylhydantoin) — eosinophilia, thrombocytopœnia, leukæmoid reactions.

TRANQUILLISERS :

Chlorpromazine — Agranulocytosis.

Meprobamate (Miltown) — purpura.

Pacatal — agranulocytosis.

ANALGESICS :

Codeine — eosinophilia.

ANTIPYRETICS :

Pyrazolone derivatives.

Antipyrin (Phenazone) — rarely causes blood changes.

Aminopyrin (Pyramidon) — There are over 50 preparations containing aminopyrin including veramon and cibalgin. Agranulocytosis often occurs after the use of the compounds containing aminopyrin.

It is estimated that in England 50 patients annually die from agranulocytosis after the use of aminopyrin.

It also causes lowered prothrombin content of blood. It has been deleted from the British Pharmacopoeia (1953).

Phenyl butazone (Butazolidine) — Agranulocytosis, fatal aplastic anemia, granulocytopenia.

p-AMINOPHENOL DERIVATIVES :

Acetanilide — (Antifebrin) — Cyanosis due to methaemoglobinaemia, (haemo-

globinuria, porphyrinuria). Prolonged use may give rise to secondary anaemia.

Phenacetin — (Acetphenitidine) — is present in Saridon, etc., haemoglobinuria, sulphaemoglobinaemia, methaemoglobinaemia, icterus, haemolytic anaemia, (few cases of haemolytic anaemia were reported after the use of saridon, 2-6 tablets per day, during a period of one to ten years).

SALICYLIC COMPOUNDS :

Aspirin (Acetylsalicylic acid), Salicylic acid, Sod. Salicylate, Methyl salicylate, Salol (Phenyl salicylate).

These cause : (a) Haemorrhages due to capillary damage. One should allow the aspirin tablets to disintegrate in water before swallowing.

(b) Haemorrhages due to lowering of the serum prothrombin — haemorrhages have been observed in kidneys, brain, liver and mucus membranes. Therefore, it seems advisable to give 1 mg. of vitamin K with every gram of salicylate.

(c) Haemorrhages due to thrombocytopoenic purpura — deaths due to *i.v.* and oral salicylate therapy — have been reported.

Even very small amounts of acetylsalicylic acid may provoke the most violent symptoms.

Salicylamide — 144 G. salicylamide over a period of three months is reported to have caused severe thrombocytopoenia in a patient. Acute haemolytic anaemia, thrombocytopoenia.

ANESTHETICS :

Changes in the organism, which may occur with all inhalation anesthetics — polycythaemia or decrease of Hb. content, leucocytosis.

Blood coagulation is accelerated with *Ether*. Later on, blood prothrombin content decreases and blood coagulation slows down when the liver is damaged. This prolongation of prothrombin time is most marked about the fourth post-operative day. Ether also inhibits phagocytosis.

Chloroform — A fall in blood prothrombin content is a sign of liver damage. Increased haemolysis, anaemia.

Cyclopropane — increased coagulability.

Hexobarbital (Evipan) — agranulocytosis.

Thiopental, Kemithal, should not be administered to patients if they are anæmic.

LOCAL ANESTHETICS :

Ethylaminobenzoate (Benzocaine)

In infants — cyanosis and anoxia due to methaemoglobinaemia.

Agranulocytosis from the use of procaine amide (pronestyl) over a period of weeks has been reported, as well as from another procaine derivative.

MUSCLE RELAXANTS :

Mephenesin — Anaemia, leucopenia (and haematuria). (Haemoglobinuria is reported to have occured in a patient following the injection of 1.5 g. of 5% Mephenesin).

GANGLIONIC BLOCKING AGENTS :

T. E. A — Leucopenia.

SYMPATHOLYTICS :

Care be taken with *Gynergen* in Anaemia.

Benodaine — Thrombocytopenic purpura.

PARA SYMPATHOLYTICS :

Diparcol — Leucopenia, Agranulocytosis.

Myanesin — Haemoglobinuria, haemolysis, thrombosis.

ANTIHISTAMINES :

Rare — Agranulocytosis. (In most cases after an administration of 3-8 weeks' duration). Pancytopenia, haemolytic anaemia.

The structure of pyribenzamine is somewhat similar to that of Aminopyrin; they have the benzaminic molecules in common. A non-thrombocytopenic purpura has been noticed.

Benadryl — Agranulocytosis — Haemolytic anaemia due to prolonged uses of benadryl and pyribenzamine have been reported.

Neoantergan — Agranulocytosis, severe haemolytic anaemia following prolonged use.

Antergan — Agranulocytosis.

Neohetramine — Nonthrombocytopenic purpura due to hetramine. While 33 tablets were tolerated well by a patient, a year later one tablet caused purpura haemorrhagica.

Antistine — Agranulocytosis.

C. V. S. DIGITALIS GROUP OF DRUGS :

Coagulation time of blood is reported to be shortened by digitalis but this observation is denied by others. Digitoxin is very rarely capable of producing thrombocytopenic purpura.

Quinidine — Thrombo-cytopenic purpura (In one case after injection of 100 mg. Quinidine) Leucopenia with transient splenomegaly.

Nitrites — Cyanosis. Methaemoglobinaemia- (Methylene blue should be given).

METALS AND METALLOIDS

Arsenic — (Fowler's solution) anaemia, Erythroblastosis.

Arsphenamine — Agranulocytosis, Aplastic anaemia, thrombocytopenia (Arsenogenic purpura), purpura fulminans (Henoch). Acute haemolytic anaemia. A remarkable fact is that agranulocytosis is much more frequently seen in women than in men, in 3% and 3/1000 respectively of cases reported in a series.

Eosinophilia during Arsphenamine treatment constitutes a warning, not to be under estimated, to suspend the injections. Arsphenamines and other metallic antisyphilitic drugs have a coagulation promoting influence. Decrease of prothrombin concentration.

Organic penta-valent Arsenicals — Atoxyl, Stovarsol, Tryparsamine, carbarsone produce agranulocytosis.

Bismuth — Thrombocytopenia, agranulocytosis, anaemia, Basophilic granules in the erythrocytes. Bismuth and other

metallic anti-syphilitic drugs have a coagulation promoting influence on the blood. The use of alkaline Bismuth nitrite by mouth may lead to symptoms of methaemoglobinaemia with cyanosis and asphyxia (a fatal case in a child).

Iron — Parenteral — Haemolysis, transient granulocytopenia, haemosiderosis.

Gold — Leucopenia, Eosinophilia (5 to 45%) — (Symptoms which may serve as warning not to continue the injection). In mild cases the injection may be discontinued and started later with small doses. In some cases it may have to be given up.

Blood changes — Eosinophilia, leucopenia, Agranulocytosis, pancytopenia, thrombocytopenic purpura (At present splenectomy, sometimes recommended), hypochromic anaemia and prolonged clotting time. (Vitamin K deficiency).

Mercury — Mercury is incorporated in various medicinal compounds which may be used in a variety of ways. Symptoms which must be ascribed to Mercury absorption may be provoked by even minute amounts. The absorption may take place in the gastro-intestinal tract, skin, mucous membrane and by injections. Typical symptoms may occur in people hypersensitive to Mercury, by absorption of minimal amounts of mercury as from a toothfilling.

Greatly varying mercury compounds may thus give rise to identical toxic and allergic symptoms.

The organic mercurials are used as diuretics now-a-days. When used sensibly they will only rarely give rise to the symptoms specific of Mercurial poisoning. Some Commercial preparations are *Salyrgan, Neptol, Novasurol, Mercuprin, Mersalyl, Mercuhydrin, Mercuxanthine.*

Blood changes — Anaemia after prolonged use, leucopenia, occasionally agranulocytosis, Marked neutropenia (1000 per c.mm.) (Good response to BAL). Eosinophilia, thrombocytopenic purpura.

During the period of Diuresis, a shortening of the blood coagulation time is observed and the acceleration of clotting is proportional to the volume of urine excreted. Agranulocytosis.

Lead — Although the symptoms of lead poisoning are mainly seen when the drinking water contains lead, these symptoms may also be caused by absorption of lead from ointments or dressings (Goulard's water). It is also possible in children who suck a nipple shield made of lead.

Anaemia with basophilic granules in the R. B. C. aplastic anaemia, (porphyrinuria), Agranulocytosis. (For lead poisoning, sodium citrate 4 grams 4 times a day for one to four weeks B. A. L. EDTA).

Thallium — Eosinophilia, lymphocytosis.

Potassium Chlorate — May lead through haemolysis to anaemia and through haemoglobinuria to anuria. This may occur after prolonged use or too much of a gargle containing it is swallowed.

Potassium thiocyanate — Anaemia thrombocytopenic purpura.

Iodine — prolonged use may lead to anaemia. Thrombocytopenic purpura may also occur (Tincture Iodine and Potassium Iodide).

CHEMOTHERAPEUTIC AGENTS

SULPHONAMIDE :

When sulphonamides are given for local intestinal action (succinyl sulphathiazole, sulphaguanidine, etc.) the possibility of Vitamin K deficiency should not be forgotten. The prothrombin level may fall to such a degree that haemorrhages occur. It is impossible to discuss all sulphonamides separately as they are too many. It is advisable to accept that the side effects may be caused by all the sulphonamides.

Blood Changes — Vary greatly in character. The bone marrow may be more or less damaged. Leucopoeisis may be stimulated which gives rise to a marked leucocytosis. (30,000 to 40,000 c/m.m.). The stimulation may be so strong that it should be recorded as leukaemoid reaction. It has been sometimes thought that the sulphonamide may give rise to leukaemia.

Eosinophilia is often seen and it should be considered as warning sign. As much as 62% of Eosinophils have been noted in sensitised persons. More often there is an inhibition of leucopoesis. It seemed there was a connection between the degree of leucopenia and the extent of the skin rash. A calculation showed that at the height of sulphonamide era, 20% of children admitted to a pediatric clinic had leukopenia. When Penicillin came to be used increasingly, the number of children admitted to that clinic, with Leucopenia dropped 6 to 7%.

Agranulocytosis and pancytopenia have been observed following all sulphonamides, Agranulocytosis, leucopenia and thrombocytopenia may be induced after succinyl sulphathiazole.

The development of agranulocytosis is also possible even after sulphonamide therapy has been suspended for 2 to 3 weeks. Even if leucocyte counts are routine procedure, the very acute development of agranulocytosis may take many by surprise. Leucopenia and agranulocytosis have been noticed after prophylactic administration of sulphonamides.

With the arrival of Penicillin era, the prognosis of agranulocytosis has improved. It may occur that the erythropoiesis are undisturbed and that only thrombocytes suffer — Thrombocytopenic purpura.

Formerly when sulphanilamides were exclusively used, cyanosis was more frequently observed than now-a-days with the new sulphonamides. Originally, it was thought that the formation of methaemoglobin and sulphaemoglobin was the cause of the *cyanosis*. The current opinion is that the verdo-haemoglobin is more often the cause, as sulphaemoglobin and Methaemoglobin are frequently absent in marked *cyanosis*.

Following Irgapyrin marked cyanosis may be observed. Porphyrines are also found under the influence of the sulphonamides and are excreted in the urine.

A mild anaemia with basophilia may be seen after prolonged use of sulphonamides.

Acute haemolytic anaemia is usually a serious complication due to sulphona-

mides. It may occur 2 to 6 days after the beginning of treatment (Irgaphen), after a period of nausea. Marked anaemia, icterus and risk of renal obstruction by hæmaturia. Blood transfusion and the administration of alkalies and copious fluids are the necessary measures. The haemolysis does not always run a stormy course.

In haemolytic anaemia, Henz's body could be shown in the R. B. C. agglutination and cold agglutination, the R.B.C.S. have been observed after the use of sulphonamides. Coagulation of the blood may be delayed slightly. Sedimentation rate of blood may be slightly increased. (The plasma protein may also be increased).

Recently introduced, sulphanylurea derivatives, namely BZ 55 and Tolbutamide, evince the same bone marrow depressant action as any other sulphonamides.

SULPHONE COMPOUND:

(*Sulphetrone, promizole, promin* and *Diazone*) — Are sulphonamides used in the treatment of tuberculosis and leprosy. The following side effects are noticed :

Anaemia due to haemolysis, leucopenia, agranulocytosis, cyanosis due to methaemoglobin formation, (haematuria).

ANTIBIOTICS

PENICILLIN :

Blood changes — Eosinophila, Thrombocytopenic purpura, Leucopenia, shortening of coagulation time. Strangely Penicillin itself has been reported to have caused Agranulocytosis. The prothrombin time is reduced by 50% in nearly all the patients receiving penicillin. In contrast to this, other investigators found a normal coagulation and prothrombin time, in many healthy people who receive penicillin. Probably there is a relationship between the increase in the number of cases of thrombosis and the embolism since the war and the frequent use of penicillin. Patients who were freely treated postoperatively with antibiotics showed a greater percentage of thrombosis than those who had not received antibiotics.

Streptomycin and Dihydro streptomycin — Eosinophilia, leucopenia, Neutropenia, Agranulocytosis, Aplastic anaemia, Leucopenia (1000 — 3000) not related to granulocytopenia repeatedly occurs in patients, with the suspension of streptomycin treatment. Leucopenia in haematogenus form of tuberculosis is more dangerous in this case. Streptomycin has to be stopped. Haemorrhages of the skin and gums occur due to Aplastic anaemia.

In 45% of cases in a report, treated with streptomycin a fall in the R.B.C. count of an average of one million per cubic/m.m. was observed during first week, all counts returning to pretreatment levels between eight and twelve weeks.

Thrombocytopenic purpura, slowing of coagulation time, a fall in the prothrombin content of blood plasma may result when streptomycin is given orally, due to the reduction of intestinal flora. Folic acid deficiency must be taken into consideration.

Aureomycin — Common intestinal flora are killed, giving rise to Vitamin K deficiency and leading to haemorrhages, notwithstanding that initially the blood

coagulation time is shortened by Aureomycin.

Chloramphenicol — At first shortening of coagulation time, later delay of the blood coagulation due to Vitamin K deficiency, marked leucopenia, marked anaemia with signs of insufficient maturation of erythroblast. Cases of granulocytopenia with signs of inhibition of bone marrow have been reported and even complete picture of aplastic anaemia. Agranulocytosis was also observed. (Haemoglobinuria)

Polymyxin — Eosinophilia, Leucocytosis.

ANTI-MALARIALS :

Quinine — Haemolysis, Black water fever, diminished blood prothrombin content. Thrombocytopenic purpura, sometimes due to even very small amount of quinine and the manifestation of haemorrhagic diathesis, Agranulocytosis.

Mepacrine — Hypoplastic anaemia, pancytopenia, Haemolysis with haemoglobinuria may occur even after a dose of 120 to 125 mg. Eosinophilia.

Pamaquin — Cyanosis, Methaemoglobinaemia, acute haemolytic anaemia, haemoglobinuria, haemolytic icterus, leucopenia.

Chloroquin — Methaemoglobinaemia.

Pentaquin — Methaemoglobinaemia. Acute haemolytic anaemia. The toxicity of pentaquin increases when it is combined with Meta Chloride (SN 11437) or sulphadiazine. A marked leucopenia and agranulocytosis may result. The combination of pentaquin and Mepacrine causes greater risk of methaemoglobinaemia and increased haemolysis. Pentaquin and Quinine cause methaemoglobinaemia and loss of active haemoglobin.

AMOEBICIDES :

Vioform — Agranulocytosis.

ANTHELMINTICS & INSECTICIDES :

Male fern — haemolytic icterus.

Phenothiazine — haemolytic anaemia.

Thymol — Eosinophilia and (eosinophilic rhinitis).

D. D. T. — Mild anaemia.

OTHER CHEMOTHERAPEUTIC DRUGS :

P. A. S. — Purpura — Insufficient excretion of P. A. S. gives rise to disturbed haematopoiesis, leucopenia, Eosinophilia, leucocytosis, Basophilia, Monocytosis, Acute haemolytic anaemia, haemoglobinuria, methaemoglobinaemia.

There may be a decreased prothrombin content of blood due to P. A. S. treatment. Haemorrhagic diathesis may occur in which the coagulation time may be prolonged. It is advisable not to give other salicylate preparation during P. A. S. treatment. Agranulocytosis.

THIOSEMICARBASONE :

(Tibione, Contiben) — Increased haemolysis, epistaxis, Eosinophilia, Monocytosis, Leucopenia, Agranulocytosis, Thrombocytopenic purpura, Methaemoglobinaemia. The combination of thiosemicarbozone with pyramidone, quinine or sulphonamides is hardly tolerated.

Chaulmoogra Oil — Haemolytic anaemia.

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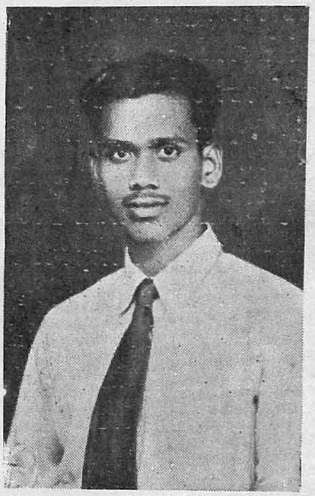
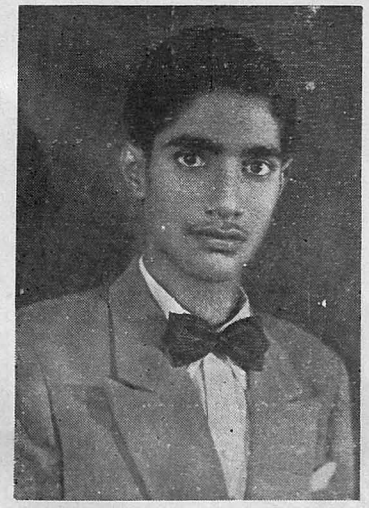
Ladies' Champion



P. KUPPUSAMY
Men's Champion

Sports and Studies



M. S. RAJENDRAN
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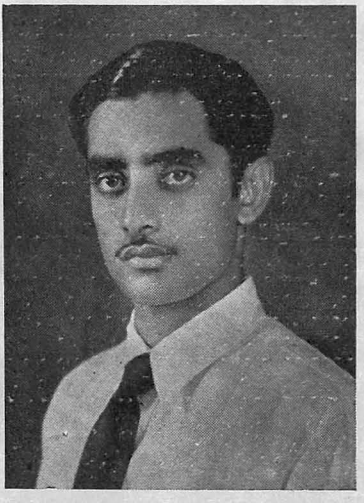

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. *Mandelic Acid* — Icterus (through increased haemolysis).

I. N. H. — Eosinophilia.

Purpura with and without thrombocytopenia, Agranulocytosis, leucocytosis.

HORMONES

Cortisone & ACTH — Polycythemia, increases or decreases blood coagulation. Thrombosis, Haemorrhagic diathesis due to vascular changes and Vitamin C deficiency under the influence of large amounts of ACTH. Acute Monocytic myelogenous leukemia. Hodgkin's disease may be accelerated. Lymphopenia, leucocytosis, thrombocytosis.

Oestrogens — In Male — Thrombocytopenic purpura.

In female — lower erythrocyte count, Haemoglobin level and haematocrit value, purpura, thrombocytopenic purpura.

Anti-thyroid drugs — Thiourea, Thiouracil, Methyl thiouracil, propyl and *Amino thiouracil* — Agranulocytosis. We have to bear in mind the fact that agranulocytosis occurs in 2% of cases. Granulocytopenia occurs more frequently, also after propyl thiouracil. It is advisable to do leucocyte count thrice a week and differential count once a week. However, one should not rely too much on a normal leucocyte count, for often the agranulocytosis occurs suddenly when the previous leucocyte count has been normal. It is perhaps more important, especially in outpatient treatment, to instruct the patient to stop taking the drug if he develops a temperature, sore throat, erythema, parasthesia or other signs of malaise. In this case, an extensive blood examination is necessary. It is stated that agranulocytosis occurs between 5th and 8th week, but it may occur sooner and on the other hand not until the patient has been taking the drug for 2 years. This may occur with propyl thiouracil also. Agranulocytosis has been described even in a patient who had only 50 mgm. of propyl thiouracil in 4 months. A case of non-fatal agranulocytosis occurred with Methimazole or *Tapazole.*

Thrombocytopenic purpura due to propyl thiouracil.

Blood coagulation may be accelerated by decrease of thrombin inactivating properties of blood. Prolongation of prothrombin time may also occur. Haemolytic diathesis with normal platelet count but markedly prolonged prothrombin time and coagulation time following the use of propyl thiouracil. Recovery follows administration of blood serum. Anaemia.

ANTI-COAGULANTS :—

Heparin — Haemorrhagic diathesis — Fortunately the haemorrhages which occur due to heparin, are by no means as frequent and severe as after the use of Dicoumarin preparations. The heparin is inactivated some hours after the injection and the bleeding stops spontaneously. If necessary, protamine sulphate may be injected as an antidote. If necessary, a blood transfusion should be given. Usually heparin haemorrhages occur exclusively when there is wound surface, for instance, a bladder haemorrhage when heparin had to be given after prostate operation.

RBC in the urine—A large haematuria sometimes occurs on subcutaneous or intramuscular injection (which may be very painful).

Dicoumarol (and allied coumarin derivatives) — Haemorrhages after dicoumarol are often more severe than after heparin. What is said of Dicoumarol is also true of Tromexan. Haemorrhages of some severity occur in 2% of patients with Dicoumarol in spite of all precautions.

Bleeding may occur from any site. Haemorrhages may still arise even after the administration has been suspended for a week. There are persons, e.g. elderly people who are particularly sensitive to dicoumarol. Fatal issues may occur in hypertensive and Arteriosclerotic C. V. Disease, despite a therapeutic level usually considered as safe. Widespread and fatal haemorrhages may occur even if prothrombin time is above 20% of normal.

In disease of the liver, prothrombin content falls so steeply after a small dose that a damaged liver constitutes a contraindication to Dicoumarol treatment. The same holds for bad renal function. Hence Dicoumarol should not be given when there are signs of haemorrhagic diathesis. In cardiac insufficiency, the prothrombin content falls after a very small dose.

The haemorrhage may continue after prothrombin content has become normal again. It is also possible that the prothrombin remains low and none the less the bleeding stops. Use of Dicoumarol should be avoided in pregnancy. Foetal deaths due to haemorrhage in Thymus, thorax and pericardial cavities have been reported.

Treatment of cardiac infarction with too much dicoumarol may give rise to a serious condition due to haemopericardium.

It is necessary to give repeated transfusion with freshest possible blood in haemorrhages due to hypoprothombinaemia. The effect of Vitamin K is less certain.

BLOOD & BLOOD DERIVATIVES AND PLASMA SUBSTITUTES :

Haemolysis with grave risk of shock and lower nephron nephrosis. A transfusion should be stopped immediately when the patient complains of pain in the lumbar region or oppression in the chest. If the patient shows symptoms suggestive of incompatibility, the transfusion of compatible blood should be carried without delay plus alkali to counteract the formation of haematin in the renal tubule.

Haemolysis may arise —

(a) From errors in the determination of blood group — A blood transfusion should never be preferred without a cross test.

(b) Because of failure to consider the Rhesus factor.

(c) From conditions concerned with the container.

(d) Sensitisation to sub groups (M.N. K. L. & S antigens).

(e) When the donor's plasma contains powerful agglutinins.

When too many transfusions are given, the body receives too much of iron. This may lead to haemochromatosis of liver and pancreas. Pulmonary embolism may occur due to small fibrin clots.

Serum albumin — Purpura.

Dextran — pseudo agglutination of R. B. C.

Acacia — Agglutination of R. B. C., capillary haemorrhage, delayed blood coagulation, hypoxaemia due to the fact that erythrocytes are covered by a layer of gum acacia.

VITAMINS

Vitamin A — Over-dosage — anaemia, fall of blood prothrombin. Minimal toxic dose of Vitamin A is estimated at 75,000 i.u./day. When this amount is taken for six months at a stretch, the clinical symptoms of A — hypervitaminosis appear.

Folic Acid antagonists — Aminopterin, Pteropterin.

Haemorrhages (skin, nasal, gastrointestinal) leukopenia — Thrombocytopenia, aplasia of bone marrow. Megaloblasts appear in the bone marrow, the leucocytes are markedly segmented and very large myelocytes may be found; symptoms such as are also found in pernicious anaemia occur and are caused by neutralisation of the effect of folic acid.

PABA — Leucopenia, granulocytopenia.

In leukemia, the leucocytes are increased under the influence of PABA, with sulphonamides on the other hand diminish.

Vitamin C — Increase of thrombocyte count.

Vitamin D_2 — Intoxication. — Normochromic anaemia, increased BSR.

SERA & VACCINES

Serum Disease — Purpura, delayed blood coagulation, leucopenia, eosinophilia.

Typhoid Vaccine — A haemorrhagic diathesis was obtained in intestines, spleen, liver and meninges of patients who developed typhoid fever immediately after vaccination against this disease.

UNCLASSIFIED THERAPEUTIC DRUGS

Benzene — Pancytopenia, leukaemia, haemolytic anaemia.

Caronamide — leucopenia, prolonged bleeding time, increased capillary permeability.

Cinchophen — Purpura, agranulocytosis, acute haemolytic syndrome.

Colchicine — Leucopenia and (porphyrinuria) Aplastic anaemia.

Congo red — delayed blood coagulation.

Menthol — A patient is described who developed purpura with a normal thrombocyte count as a consequence of smoking cigarettes containing Menthol. A caution to such cigarette smokers !

Nitrogen Mustard — Bone-marrow damage, causing leucopenia, agranulocytosis, thrombocytopenia, anaemia, lymphopenia, delayed blood coagulation. It is said that severe side effects may be prevented by previous injections of 100 mg. pyridoxine.

Papain — Eosinophilia.

Phenolphthalein — Purpura, haemorrhagic diathesis.

Phenyl hydrazine — haemolytic anaemia, jaundice, aplastic anaemia.

Picric Acid — haemolysis

Pyridine — Methaemoglobinaemia.

Radioactive isotopes — P^{32} — leucopenia, anaemia, thrombocytopenia. These symptoms do not occur until weeks to months following the last administration.

A patient with polyerythraemia developed thrombocytopenic purpura following treatment with P^{32}.

Acute Radium Poisoning — Haemorrhagic diathesis by a fall of thrombocyte count, by capillary damage and because a heparin-like substance is formed — Anaemia.

Urethane — Porphyrinuria, Leucopenia. After Leucocyte count falls to 2000/c.mm. Agranulocytosis, thrombocytopenia, aplastic anaemia, increased hæmolysis.

Sodium Morrhuate — Prolonged bleeding time with a tendency to generalised bleeding.

Suramin — increased haemolysis.

Thorotrast — Aplastic anaemia.

Radium Compounds — (Thorium X) — After prolonged use Aplastic anaemia and leukaemia.

So far we have dealt with the haematological changes met with during the administration of a number of drugs. This is not a comprehensive list. Newer drugs are being used in several disease conditions and their potentiality to produce haematological changes are under study. In course of time, many more will have to be added to the above list.

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If my theory of relactivity is proven successful Germany will claim me as a German and France will declare that I am a citizen of the world. Should my theory prove untrue, France will say that I am a German and Germany will declare that I am Jew.
— *Albert Einstein*

* * * *

Anger is a kind of temporary madness.
— *Gandhiji*

Birth, Re-birth & Birth-day

by

M. D. A.

To be born again ; Wherefor ?
Hast thy song and sojourn been so good,
So useful, so complete, that the world
Wouldst hear once again thy song ; see thee
Tread thy path of old ; try to make good
What thou hadst failed in ? Be it so,
If thy past was good, beneficial, useful ;
Otherwise, it were better that re-birth
Be stopped for ever ; the day of thy nativity
Forever consigned to oblivion,
The bosom of Eternity from which
You did come forth, but a brief moment to spend.

Let each time, at the turn of the year,
That month, that day and hour of thy birth,
In grace given to you, perchance,
Given thee the chance of remaking
The love for thy neighbour,
Which thou didst scorn ; speak
The truth which thy cowardice urged
Thee to hide ; scatter the gains of greed
You hoarded whilst pleading penury
Passed by thine shut eyes,
And charity clamoured for her share !
Return of the day, year after year.

Of the day of thy birth, were worth only,
If thy story year after year on this earth
Has been of common good, to all things living,
Great and small ; be it bird, beast,
Plant nor flower, bee or butterfly,
That God had made in common with man.

Is immortality a gain ? not to be born to die !
The hordes of Heaven with never birth nor death,
With no chance to make better their good,
Transform angelic unto God-like !
Wherefore was Son of God born as Son of Man,
But for the chance that birth gave him
By sacrifice and death to be a Saviour ?
The cross that did raise Him above ground
Raised Him, also to the Heavens above ;
Yea, for a place on the right hand of God.
By birth did Son of God gain the right
To title, God the Son and seat beside God,
the Father.

What of the Avatars ? Ten times was God
Born as man or in the guise of beast ;
Half human, half animal – born each
Time just to save this world from sin of Man.
Birth ! be born again ! 'Twere a happy event,
If each rebirth, each birthday were a chance,
To sacrifice self for the common good –
Man, aspiring to a seat in Heaven, and
Beside God Himself ; what better way
Hath he to gain that which angels seldom get,
To bring God to earth, and take Man to Heaven,
Than by being born, to live like Man,
Suffer like him : prove the worth in him
That divine spark, shine and glorify.

The world is not things, but persons ; no natural beauty of inanimate things can equal in interest the profound fascination of the human character. — *Katherine T. Hinkson*

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Guinea - worm Disease or Dracontiasis

by

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Senior Entomologist, Department of Social and Preventive Medicine, Madurai Medical College, Madurai.

GUINEA-WORM infection, which is also known as Dracontiasis is one of the oldest known tropical diseases. The disease is caused by the invasion of the connective tissues by a long thin worm called *"Dracunculus Medinensis"*. The female worm comes to the surface of the skin and causes a blister, generally on that part of the body which comes in contact with water. This is a common parasite of man in certain parts of India, Arabia, Egypt, and other parts of Africa. This has been imported into West Indies and South America.

In India this disease has been known from early days and is widespread mostly in certain rural areas and in a few towns, where there is scarcity of water, with low rainfall and rocky soil. The incidence of the disease is known to be fairly heavy in Rajasthan, Madhya Pradesh, and Bombay. In South India, the West Coast regions like Malabar, South Kanara and the Nilgiris which have heavy rainfall are practically free. The following areas are endemic for the disease :

(1) Cuddappa, Kurnool, Bellary and South Arcot. (*Heavy infection).*

(2) Plains of East and West Godavari, Guntur, Nellore, South Arcot, Salem and Coimbatore. (*Medium infection).*

(3) Chingleput, Tiruchirapally, Tanjore, Ramnad, Tirunelveli, Anantapur, Chittoor, Vizag and Krishna. (*Light infection).*

Although this disease is not fatal, it causes a good deal of suffering and physical deformity. The affected persons are incapacitated for a long period and are unable to earn their daily wages. As medical aid and advice are not easily available in villages, the people fall easy victims to the disease.

During the period of incubation which is about a year, no symptom generally occurs. But symptoms occur just before the manifestation of the parasite beneath the skin. There is a feeling of intense itching all over the body. Nausea, vomitting and giddiness may follow. These symptoms are attributed to the toxins excreted by the worm. Redness of the skin at the spot and burning sensation will be the external symptoms noticed. At that time the patient immerses the affected part in cold water which has a soothing effect. A few hours after the onset of the foregoing symptoms, development of a blister may be noticed. When the blister opens, a raw red ulcer is exposed. About the middle of the ulcer there is an opening which is connected with the tunnel in the tissues where the female worm is lying.

Though the worms mostly appear on the lower extremities which generally come in contact with water, cases are not uncommon where the worms make their appearance on the shoulder, arms, chest and other parts of the body. Sometimes the worm may fail to appear and die before maturity. In such cases it either becomes calcified, forms an abscess or gets absorbed. Generally a patient gets one worm at a time but occurrence of multiple infection is also noticed.

In rural parts where this disease is common, the people are in the habit of winding the worm on a small stick, when the head emerges from the blister and continuing the process by giving a turn or two to this stick every day. This is an old and tedious method, requiring about 21 days for the complete removal of the worm. Removal of the worm will be much easier if it is done after the worm has discharged all the embryos. The worm can be made to discharge the embryos by douching with cold water, as the contact of cold water induces the worm to discharge the embryos. Extraction of the worm after killing it by injecting certain drugs like acriflavine, perchloride of mercury solution etc., is also done. Removal of the worm by surgical methods by making incisions is also done. Great care must be taken to remove the whole worm unbroken. If the worm breaks during the process of extraction, not only will it be extremely painful but many complications will occur resulting in the swelling up of the tissues in the neighbourhood of the ulcer, fever and severe pain. Secondary bacterial infection may also supervene.

A full-grown guinea-worm measures 12″ to 48″ in length and 1/16″ in diameter. It is round, smooth and milky white in colour. The head end is tapering and rounded, while the tail end is also tapering and curved like a blunt hook. A mature female worm is fully packed with embryos for most of its length. Per worm there will be roughly about three million embryos. The mouth is smaller and triangular and is surrounded by eight papillæ. The secretion from the head gland is an irritant and causes blister on the skin of the host. There is a double ovary at the posterior extremity and the whole worm is occupied by the double uterus packed with embryos. The male worm is practically not met with in man. It measures about 3 c.m. and after fertilizing the female it dies and is absorbed.

When an infected man having the mature worm protruding from the ulcerated surface steps into the water of a step well or any water source in the village bare-footed, the worm discharges a milky fluid which is teeming with the embryos. An embryo will be about 0.6 m.m. long. Its body is flattened and the tail is tapering. When discharged it is coiled up, but very soon becomes straight and moves about by performing jerky movements.

These embryos are swallowed by minute aquatic crustaceans called *Cyclops* or the water fleas. They migrate into the body cavity of the cyclops and undergo development. It loses its tail, becomes cylindrical in shape and undergoes two moults. All these take from 2 to 6 weeks depending upon the temperature of the water. Inside the cyclops no further development takes place. As many as ten

embryos may be swallowed up by a cyclops at a time, but too many embryos may kill the cyclops. In endemic areas as many as 40% of the cyclops may be infected.

When such infected cyclops are swallowed by man along with drinking water, they are killed by the hydrochloric acid present in the gastric juice and the larvae are set free. They pierce through the intestinal wall and reach the retro-peritoneal tissues. It is here that fertilization of the female takes place. They develop further and migrate to the other parts of the body. Full development of the worm takes place in about 8 to 12 months.

When the female becomes gravid, it migrates to the surface of the body, legs or feet, and secretes some irritant material which causes the formation of a blister. Finally, when it comes in contact with water the blister breaks and the embryos are discharged as already described. These embryos survive for about a week and die if they are not swallowed by cyclops within the period.

The average life of a cyclop is about three months. But this is determined by various factors like the temperature of the water and its acidity or alkalinity. When the temperature of the water is raised to 60°C. the cyclops present therein die.

CONTROL

1. *Protection of water sources* : Infection from this disease is got only by drinking water which contains cyclops infected with guinea-worm larvae. So, this could be prevented by protecting the source of drinking water from being polluted by the guinea worm patient. Generally in villages, the sources of water supply will be wells, step wells, small ponds, hollows of rocks etc. The step wells should be converted into pucca protected draw wells or into pump wells, by carrying out suitable alterations, such as construction of parapet walls on all sides etc. Similar arrangements may also be made to the other water sources so as to prevent people from physically coming into contact with the water.

2. *Addition of certain chemicals,* such as caustic potash, perchloron etc., in certain concentrations kills the cyclops. But, such dosages are found to be unsafe for human beings. Lime is the most practicable substance and is found to be very efficient in killing cyclops. So this may be used at the rate of 60 grains of lime to one gallon of water.

3. *Biological* method of control may also be followed by introducing cyclopicidal fish like Barbus puckelli and B. ticto into the wells. The density of the cyclops may be brought down to a considerable extent, as these fish voraciously feed upon cyclops.

4. *Personal prophylaxis* that can easily be followed will be either to boil the water or to filter the water well through fine muslin. As the cyclops are large enough they are retained in the cloth.

5. *Health Education* : The dissemination of the knowledge about this disease and the mode of its prevention and treatment among the rural folk of the endemic areas will go a long way in preventing them from falling a victim to this disease.

The density of cyclops of the suitable species is an important factor. Cyclops survive well in areas where there is moderate or low rain fall. They increase in number with the onset of summer due to the concentration of rich organic matter in the water, which serves as food for them. As the intake of water is larger in summer on account of greater thirst, the chances of infection are also greater at that time. The infection spreads from one village to another by the migration of the infected villager who carries the worm in his body and allows it to discharge the embryos in the uninfected water source of the new village. So, to prevent the spread of the disease, such persons should not be allowed to come in contact with the water source.

If you really will, you surely can —
But you never can unless you will.

* * * *

If you are tempted to reveal
A tale someone to you has told
About another, make it pass
Before you speak,
Thro' three gates of gold :
Three narrow gates of gold — first,
"Is it true ?"
Then, "Is it needful ?" In your mind,
Give truthful answer ; and the next
Is last and narrowest,
"Is it kind ?"
And if to reach your lips at last
It passes through these gateways three
Then you may tell the tale nor fear —
What the result of speech may be.

— *Anonymous*

Calcium and Life

by

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HARDLY any day passes in the routine of a Biochemical Laboratory without there being an estimation of blood calcium. Calcium is one such element which occupies a privileged and enviable position in biochemistry and biophysics and it can be unreservedly stated that calcium is the main actor in the drama of life played by various elements.

If we burn any living thing (God forbid !), it becomes dead — this does not require a scientist to investigate and assert — and the bulk of it vanishes. Water contained in it escapes as steam and the organic matter is converted into carbon-dioxide. The little ash left behind is found to consist of various salts of sodium, potassium, magnesium, calcium, etc., Thus the stuff of which living material is made of, is of the very cheapest. The salts constitute the very important part of the protoplasm. Of these, calcium in the form of its salts is the most essential.

Calcium is the vital structural material in bones and teeth. The rigidity of the bones and teeth is due to calcium. When bones are treated with acid, they lose their stiffness and become pliable, as their calcium-content is removed by the acid.

Calcium is an important ingredient in the cement that binds the cells together in the tissues. This has been demonstrated in the case of the sea urchin. This organism is a great mass of cells clinging together. When placed in a solution antagonistic to calcium, the cells fall apart.

The formation and firmness of the cell membranes also are dependent on calcium. The protozoan amœba is a single cell consisting of a drop of protoplasm having a nucleus and surrounded by a fairly rigid cell wall. When the membrane of an amœba is broken, the outflowing protoplasm soon forms a new membrane. But if the cell is kept in a solution which would remove calcium, no new membrane is formed to replace the one that has been ruptured.

The reaction whereby a broken cell forms a new membrane in the presence of calcium is termed "surface precipitation". Surface precipitation reaction in protoplasm is a form of clotting. The reaction of blood to calcium ions is similar to that of protoplasm. The clotting of blood — a boon and blessing in preventing blood losses — is wonderfully influenced by calcium. The blood-clot is a net work of the threads of fibrin (an insoluble protein) in which the corpuscles and residue of plasma are entangled. Fibrin is produced from fibrinogen (a soluble protein) by the action of thrombase (Thrombin). The formation of thrombase from prothrombin present in the blood is facilitated by calcium ions. There would

not be any clotting of blood if the calcium content of blood is removed by adding a little oxalate (Calcium ions are precipitated down as Calcium oxalate). That is why oxalic acid and oxalates are called metabolic poisons. In this connection, it may be interesting to note that an over-abundance of calcium can *prevent* clotting both in blood and in protoplasm. This perhaps proves the common saying that anything in extremes is always calamitous and catastrophic.

Incidentally, the curdling of milk also is a calcium-influenced process. The clot of milk is essentially the insoluble calcium salt of casein (phosphorus-containing protein), calcium caseinate and as such nutritionally rich though some people consider it unpalatable.

Muscle protoplasm like the protoplasm of the cells in general is extremely sensitive to calcium ions. Thus when a muscle fibre is cut, the cut surface immediately produces a plug very much like the new membrane that forms at the surface of the exuding droplet of protoplasm of an amœba. The more the calcium in the surrounding fluid, the more rapidly the plug forms. The sensitivity of muscle protoplasm to calcium can be shown by the injection of small amounts of calcium directly into the interior of the muscle fibre with a needle of extremely fine bore. The injection of a very dilute solution of calcium makes the muscle contract.

Calcium is essential for the contractibility of involuntary (e.g. heart) muscle, acting antagonistically to sodium and potassium. It has great influence on excitability of nerve fibres and centres.

Plasma calcium must be maintained near the normal (10 mgs. per 100 c.c.). This appears to be controlled by the parathyroid hormone. A low blood calcium produces tetany.

Calcium has also a role in the division of cells. The mechanism of cell division is of particular importance as it has a bearing on cancer. Perfection in the research of the role of calcium in cell division might open vast vistas in combating cancer.

Aging of cells and aging of animals and human beings is due to the accumulation of calcium in the cells in an insoluble form. In fact 'stones' removed during operation from any part of the body are specially tested for calcium salts.

Methods of estimating calcium especially in blood and urine have been refined, perhaps to such an extent that it may not be possible to refine it further. The Flame Photometer is to a biochemical laboratory as calcium is to life. With the use of this versatile instrument, estimation of even traces of calcium is done with utmost accuracy in a jiffy.

Thus, by virtue of its multiplicity of function and indispensability to life, calcium has a unique and unchallengeable role in Biochemistry and Biophysics.

A good marriage would be between a blind wife and a deaf husband.

— *Montaigne*

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Hypertensive Encephalopathy

by

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Introduction :

THE term "Encephalopathy" denotes any degenerative disease of the brain. The following are the varieties of Encephalopathy.

1. Toxic Encephalopathy which includes poisoning with heavy metals such as lead, arsenic, gold, and manganese.

2. Hypertensive Encephalopathy.

3. Saturnine Encephalopathy — In acute lead poisoning, a lead encephalopathy develops, characterised by alteration in blood pressure, raised cerebrospinal fluid pressure, papilloedema, acute mania, convulsions and coma. Pallor of the patient is ashen-gray. In the case of chronic lead poisoning, fits may replace delirium.

4. Wernicke's Encephalopathy — An inflammatory haemorrhagic type of encepalitis with lesions about the cerebral aqueduct, occurring mainly, but not always in chronic alcoholism. It is marked by paralysis of the eye muscles, a reeling gait, and disturbance of consciousness.

5. Nicotinic Acid Encephalopathy — Characterised by clouding of consciousness, cog-wheel rigidity, uncontrollable sucking and grasping movements. Mental symptoms are in the form of confusion, confabulation, dementia, depression, delirium, mania.

6. Demyelinating Encephalopathy — due to degeneration of the white matter of the cerebrum. This group includes Schilder's disease, Pelizaeus-Merzbacher disease, Krabbe's disease, Scholz's disease, Balo's disease, Greenfield's disease.

7. Progressive Subcortical Encephalopathy.

8. Malaria Encephalopathy.

9. Heat-stroke Encephalopathy.

This article deals with Encephalopathy caused by hypertension.

HYPERTENSIVE ENCEPHALOPATHY :

Definition :

This condition is a cerebral symptom-complex correlated with arterial hypertension. It begins with severe headache, followed by vomiting, drowsiness, convulsions and coma. These attacks may be

preceded or followed by focal symptoms and signs. This clinical picture is seen in essential hypertension, malignant hypertension, hypertension due to nephritis, eclampsia, and lead poisoning. There is a sudden rise of an already elevated blood pressure followed by unconsciousness with or without convulsions. Hypertensive retinopathy may be present, papilloedema may occur in consistant cerebral oedema. Blood urea is not raised, and the attacks are not due to uraemia.

Symptoms and Signs :

The clinical picture comprises Prodromata, Convulsions-tonic and clonic, and Coma. In the prodromal stage headache, nausea, vomiting, increased apathy or somnolence, loss of appetite, physical or mental weakness, restlessness, parasthesias preceding convulsions, pallor of the skin, rise of blood pressure, diminished urine output may be seen. Convulsions start all in a sudden. If tonic, the body is thrown into opisthotonus, the limbs rigidly extended, the fists clenched, and respiration ceases. This is followed by clonic spasm involving the arm, face and then the whole body. Minor seizures occur in the form of few twitches in single muscle groups. Froth in the mouth, rise of temperature, dilated pupils, sphincter disturbances are also observed. The convulsions last for a few seconds to ten or more minutes. Babinski's sign, neck rigidity, Kernig's sign are often found.

The patient becomes comatose during the more severe fits. On returning to consciousness, he may feel well, but suffers from amnesia.

In general, paroxysmal dyspnoea and Cheyne-Stokes breathing may occur in these patients. Lumbar Puncture reveals greatly increased cerebro-spinal fluid pressure, ranging from 300—400 mm. of water. Retina may show papilloedema, narrowing of retinal acteries. The blood chemistry is normal throughout until either recovery or death. Additional manifestations of this syndrome are amaurosis, auditory disturbances ; and mental symptoms of short duration such as stupor between the excited phases, visual or auditory hallucinations, melancholia.

The hypertensive cerebral vascular crisis appears in the later stages of the disease. It is of abrupt onset, short duration, disappearing suddenly and takes the form of transient attacks of amnesia, hemiplegia, aphasia, or some other cerebral episode.

Pathogenesis :

The cerebral symptoms of Hypertensive Encephalopathy are manifestations of circulatory disturbances in the brain brought about by hypertension. An increase in the tone of the cerebral arterioles results in increased arteriolar resistance to blood flow through the brain. The blood flow through the brain remains normal, in spite of the rise in arterial pressure. This adjustment of cerebral circulation could be disturbed in two ways : (a) In some cases, the cerebral arterioles do not constrict well to counterbalance rise in blood pressure, resulting in rise of pressure in the brain capillaries, transudation, impaired reabsorption and oedema of brain (as in glomerular nephritis, toxaemia of pregnancy). In this variety, increased intracranial pressure is manifested. (b) The cerebral arteries develop a spasm, resulting in ischemia of the part of the brain sup-

plied by these vessels. Thus fainting, fits, transient palsies, and aphasia are produced without increased intracranial pressure.

Rosenberg examined the brains in 17 cases of malignant hypertension and in 15 controls. In the former, massive haemorrhage was found in 4, small haemorrhages with numerous infarcts of similar size in 5, single large infarcts in 3. Multiple miliary infarcts he found in 12 cases in the basal ganglia, cortex, white matter, brainstem, and cerebellum. These small lesions had been ascribed to vascular spasm by Westphal & Bār and Spielmayer, because no adjacent organic arterial lesion could be found. Rosenberg suggested that cerebral symptoms were of 3 kinds in malignant hypertension : (a) due to increased intracranial pressure — headache, vomiting, drowsiness. (b) due to multiple miliary cerebral lesions — a wide variety of transitory disturbances often without physical signs. (c) due to large cerebral vascular accidents. The obstruction is organic and not spasmodic. In hypertension, local cerebral attacks are most probably due to the sudden organic occlusion of a cerebral vessel. The nature and extent of the symptoms will depend on the site and extent of the area rendered ischemic and thus on the site and size of the vessel occluded. The speed of recovery will depend on the size of the vessel, the location of the vessel, and the efficiency of the collateral circulation.

The current view as to the origin of cerebral oedema is due to ischemia of the brain — the cerebral arterioles are more constricted than those elsewhere, so that the cerebral blood-flow becomes so small that the capillaries are damaged by anoxia and so allow abnormal amounts of fluid and protein to pass out into the cerebral substance. Further light on the mechanism by which arteriolar necrosis is produced, is offered by Byrom who after careful experiments infers that Hypertensive Encephalopathy is the result of ischemia of the brain consequent on the arterial spasm provoked by the grossly raised intra-arterial pressure.

Diagnosis :

Hypertensive Encephalopathy is one of the three cerebral manifestations of Bright's disease, the other two being Uraemic symptoms due to renal insufficiency, and Symptoms arising from cerebral arteriosclerosis. The diagnosis is made clinically in the presence of intense headache, vomiting, sudden impairment of vision, and papilloedema. Convulsions and coma are frequently observed. However, this condition is to be differentially diagnosed from ; (1) Uraemic states of malignant Hypertension ; (2) Low Salt Syndrome ; (3) Cerebral Vascular Accidents ; (4) Epilepsy ; (5) Meningitis ; (6) Brain Tumour or abscess.

Prognosis :

This is a matter of serious concern. In Hypertensive Encephalopathy not accompanied by azotaemia, prognosis is decidedly better than when the cerebral symptoms are due to uraemia. Since salt restriction has become routine in treatment, oedema of the brain in acute glomerular nephritis has become rare and the outlook far better.

Treatment :

A. Medical : The aim of treatment is to prevent and alleviate oedema of the

brain, as well as to enforce rapid relief of the arterial hypertension. The medical line of treatment includes the following procedure :

(1) *Sedation.* Most patients respond satisfactorily to intravenous Sodium Amytol 0.5 G. repeated at intervals in smaller doses to maintain sleep and a decrease of arterial pressure over 12 — 24 hours. In the older type of patients, Phenobarbitone gr. 1½ — 2 daily is indicated in addition to the general line of treatment.

(2) *Venesection.* This is indicated in cases with severe manifestations of oedema of the brain. In adults 400 — 500 c.c. of blood may be let out and repeated if necessary. Convulsions disappear. If the procedure is undertaken during the prodromal stage of the condition, prevention of the onset of the attack and violent headache are ensured.

(3) *Lumbar Puncture .* Is indicated for the severe symptoms, and can be only undertaken with particular caution in view of the danger of a sudden decrease in pressure, permitting the medulla to be forced downwards into the foramen magnum resulting in sudden death. The cerebrospinal fluid removed should be proportionate to its tension and the tapping helps through lowering intracranial pressure.

(4) *Dehydration of the brain.* This is indicated when intracranial pressure is enormously increased. Intra-venous hypertonic solutions are helpful. 50% Dextrose solution 200 c.c. given as a slow intravenous drip is of value ; if necessary, several further injections of 100 c.c. Dextrose solution could be given within the next 24 hours. This is followed by profuse diuresis, abolition of convulsions, and relief of headache. Sucrose has been shown to produce renal lesion such as Osmotic Nephrosis. 25% salt-poor Serum albumin, 60-80 c. c. given in 8-10 minutes intravenously is also helpful. Magnesium Sulphate, 2% solution 250 c.c. given very slowly produces rapid fall in blood pressure.

(5) *Anti-hypertensive drugs.* Veriloid, intra-muscularly or intra-venously ; Apersoline, intra-venously ; Ansolysen, subcutaneously or intra-muscularly; Hexamethonium compounds, subcutaneously ; are of value in the treatment of Hypertensive Encephalopathy particularly in the high blood pressure levels.

(6) Other drugs. A mercurial diuretic even in the presence of a mild degree of albuminuria may be given. For the acute manifestations of cerebral oedema, 1 -- 2 c.c. of Mersalyl may be given intra-venously. Sodium nitroprusside, intra-venous drip (0.15 — 3 c.c. per minute) of a solution of the salt in 5% glucose may also be tried. Overdosage of the drug is indicated by a decrease of arterial pressure to normal or below, emesis, increased confusion and restlessness. Aminophylline 0.1 — 0.2 G, four times daily has been recommended by Reese and Kant as a cerebral vasodilator in cerebral arteriosclerosis and the so-called Hypertensive Encephalopathy. Epileptiform attacks should be treated with soluble Phenobarbitone gr. 3 given intramuscularly. Sodium Amytol given intravenously or Paraldehyde 7 c.c. given intramuscularly or intra-venously have profound control over convulsions. When

convulsions have stopped, the patient is to be kept absolutely quiet in a darkened room. Chloral hydrate should be given by rectum. In cases showing papilloedema, 25% solution of Magnesium Sulphate in the form of retention enema is indicated.

B. SURGICAL : Nephrectomy offers a better prospect for patients with a gross unilateral renal disease.

* *Statistical Evidence* :

Tables I, II and III present a review of Hypertensive Encephalopathy cases admitted to two major Government General Hospitals in Madras City, viz., Government General Hospital and Government Stanley Hospital during the period 1951 to 1954.

Table I presents an analysis of number of cases treated during 1951, 1952, 1953 and 1954 showing total number of admissions and deaths, according to sex. It is seen that more male patients were admitted than female patients (in the ratio of 3 males to 1 female). Death rate also shows a preponderance in the males, percentage of deaths in the male being 47.1 and in the female 36.3. Maximum number of cases occurred in the 6th decade of life, youngest in the series being 15 years and the oldest 72.

Table II deals with the Chief Complaint, duration of illness, past illness, and short detail of Present illness. Similar complaints or closely allied conditions were reported under history of past illness by 17 patients.

Table III tabulates positive clinical findings and results of certain laboratory investigations. Fundus changes were present in 5 cases, cranial nerves were involved in 6 cases, blood pressure was raised above 160 & 90 mm Hg in almost all cases — highest systolic pressure recorded being 286 mm. and highest diastolic 190 mm ; urine showed albumin or casts in 6 cases, blood urea raised above normal in 13 cases, CSF was under tension in 4 cases.

Summary :

(1) Introduction, (2) Hypertensive Encephalopathy — Its definition, Symptoms and Signs, Pathogenesis, Diagnosis, Prognosis, and Treatment, (3) A summary of Case Records, comprising 45 cases of Hypertensive Cerebral Encephalopathy.

Acknowledgment :

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* Based on a Paper read at the Joint Conference, 1956 (Association of Physicians of India) at Bombay.

TABLE I

Year	Admissions		Deaths	
	Male	Female	Male	Femal
1951	9	—	4	—
1952	4	2	1	2
1953	18	1	10	1
1954	3	8	1	1
	34	11	16	4
Total	45		20	

TABLE II

	No.	Sex	Age	Expd	Complaint	Duration	Past Illness	Present Illness
1951	1.	M	65	+	Giddiness, Drowsiness	1 day	...	Fell down because of giddiness
	2.	M	50	.	Inability to walk, Tingling and numbness	1 year & 4 months	...	...
	3.	M	54	.	Unconscious	2 days	...	Headache, dimness of vision, Unconsciousness
	4.	M	40	+	Coma	1 day	...	...
	5.	M	50	.	Fits	within 24 hrs.	...	Fits, skew deviation of eyes & head, Unconsciousness, frothing of mouth
	6.	M	50	.	Fits	within 24 hrs.	...	Onset sudden, conjugate deviation of eyes and head, frothing of mouth
	7.	M	40	.	Inability to use left half of body	4 days	...	Onset sudden
	8.	M	50	+	Fever, Head-ache, pain in lumbar region	3 days	Similar complaint 6 months back	Onset sudden, drowsiness, Convulsions, T 102
	9.	M	50	+	Fits, incontinance of urine	2 hours	...	Onset sudden, Unconsciousness
1952	10.	F	65	+	Coma	1 day	...	...
	11.	M	58	+	Coma	1 day	Fits with unconsciousness 3 years back	Fell unconscious while taking food
	12.	F	65	+	Coma	1 day	...	Sudden onset, flaccid right limbs
	13.	M	51	.	Headache, Giddiness	1 week	...	Epigastric pain, vomiting, giddiness, headache, tinitus
	14.	M	45	.	Fits	10 days	Similar complaint 4 years back	Tingling, numbness left half of body, fits, unconsciousness
	15.	M	40	.	Unconsciousness, convulsions	within 24 hrs.	Hypertensive Failure	Onset sudden, Unconsciousness, Convulsions
1953	16.	M	35	+	Breathlessness	1 week	...	Breathlessness, loss of movements, left limbs

	No	Sex	Age	Expd	Complaint	Duration	Past Illness	Present Illness
1953	17.	M	55	+	Mental derangement	1 day	...	Onset sudden, Giddiness, mental derangement
	18.	M	52	+	Unconsciousness	2 weeks	...	Itching sensation all over the body, next day could not walk, later unconsciousness
	19.	F	19	+	Unconsciousness	1 day	giddiness 4 months ago	Unconsciousness
	20.	M	69	.	Dyspnoea, then Unconsciousness	within 24 hrs.	Similar complaint 2 years back	Dyspnoea followed by unconsciousness, passes urine in bed
	21.	M	60	.	Weakness Rt. arm, difficulty in speech	3 days	Headache & giddiness 10 years back	Felt giddy, went to bed, weakness and heaviness of Rt. arm ½ hour later speech affected
	22.	M	22	+	Breathlessness, Oedema of extremities	2 months	...	Fever 4 days, Oedema of feet, breathlessness
	23.	M	59	+	Unconsciousness	within 24 hrs.	Hypertension	Unconsciousness
	24.	M	35	.	Fever	2 days	...	Felt giddy, does not talk properly, by night becomes semi-conscious, Severe headache
	25.	M	52	+	Unconsciousness	3 days	Similar complaint 6 months back	Fever followed by unconsciousness
	26.	M	54	+	Unconsciousness	2 days	...	Fits, then unconsciousness
	27.	M	56	.	Loss of consciousness	5 hrs.	...	Palpitations, loss of memory, loss of consciousness
	28.	M	55	.	Loss of consciousness	10 days	Hypertensive Hemiplegia 1 year back	Weakness of Lt. limbs, Headache, giddiness 4 days, followed by unconsciousness
	29.	M	72	+	Dribbling of urine and in-continance of faeces		...	...
	30.	M	50	.	Inability to use Lt. limbs	1 day	...	Fell down unconscious later regained consciousness, inability to use Lt. limbs, patient semi-conscious, passes urine and motion in bed
	31.	M	60	.	Weakness Rt. arm, difficulty in speaking	3 days	Frequent attacks of headache, Occasional giddiness 10 years back	Felt giddy, went to bed, later developed weakness and heaviness of Rt. arm, ½ hr. later difficulty in speech

	No.	Sex	Age	Expd	Complaint	Duration	Past Illness	Present Illness
1953	32.	M	45	+	Unconsciousness	within 24 hrs	...	Became unconscious suddenly
	33.	M	48	+	Coma	1 day	Hypertension	Sudden unconsciousness, vomiting, T. 102
	34.	M	40	.	Giddiness	10 days	...	Giddiness followed by unconsciousness
1954	35.	F	30	.	Severe headache, giddiness	4 days	Similar complaint 4 months back	Onset sudden, Headache, giddiness, Semi-consciousness
	36.	M	57	.	Headache	3 months	...	Onset sudden, Aphasia $\frac{1}{4}$ hour Headache, Unconsciousness
	37.	M	42	+	Blurred vision, Dyspnoea	4 years	Similar complaint 1 year back	Onset gradual, blurring of vision, exhaustion, Dyspnoea
	38.	F	60	+	Unconsciousness	within 24 hrs	Similar complaint 6 months back	Onset sudden, Heaviness of head, fell unconscious, convulsions arm.
	39.	F	60	.	Unconsciousness	within 24 hrs	Similar complaint 3 years back	Unconsciousness during sleep
	40.	F	56	.	Headache, Nausea	10 days	Similar complaint 10 years back	Headache, nausea, slight paresis left arm
	41.	M	44	.	Unconsciousness	2 days	...	Unconsciousness during sleep, right limbs affected
	42.	F	15	.	Headache, fits	3 days	...	Severe headache, fits, swelling of face
	43.	F	65	.	Semiconsciousness	4 hrs	...	Giddiness, vomiting, Unconsciousness
	44.	F	43	.	Unconsciousness, inability to use left side of body	...	...	Unconsciousness. Incontinance of urine
	45.	F	38	.	Loss of speech	1 day	...	Sudden inability to speak, felt giddy, next day regained speech

TABLE III

	S. No.	Speech	Cranial Nerves	Motor System	Sensory system	Reflexes	Fundus	B. P.	Urine	Blood Urea	C. S. F. Tension	C. S. F. Pr. G.	C. S. F. Cl: S.
1951	1.	—	—	—	—	—	—	220.110	—	—	—	—	
	2.	—	—	Power poor 4 Spasticity 4	—	Abd. lost Crem lost Deep + + + Plantar ↑ ↑	—	220.120	—	—	—	—20	—760 58
	3.	—	—	—	—	Abd, lost Crem. lost Deep −4	—	285.190	Alb + + Gra. cast	97	—	—	
	4.	—	—	—	—	—	—	220.110	—	—	—	—	
	5.	—	—	—	—	—	—	160.100	—	—	—	—	
	6.	—	—	—	—	—	—	160.100	—	—	—	—	
	7.	—	7th UMN	Power Poor Rt Limbs	—	Deep + + + Rt Plantar ↑ R	—	170.105	—	—	—	—	
	8.	—	—	—	—	—	—	180.120	—	—	—	—	
	9.	—	—	—	—	Deep poor 4 Plantar ↑ ↑	—	170.120	—	—	—	—	
1952	10.	—	—	Flaccid Rt	—	Plantar ↑ R	—	170.100	Alb + +	—	+	—	
	11.	—	7th Rt	Flaccid 4	—	Deep lost 4 Plantar ↑ ↑	—	190.100	—	—	—	—	
	12.	—	—	Flaccid Rt	—	Plantar ↑ R	—	170.100	Alb + +	—	+	—	
	13.	—	—	—	—	—	Papilloedema Hges.	230.150		—	+	—	
	14.	Normal	Normal	Normal	Normal	Normal	—	195.100		—	—	—	
	15.	—	Pupils contracted, not reacting to light.	—	—	Deep + + + legs & Rt. arm Plantar ↑ ↑	—	200.140		—	—	—	

	No.	Speech	Cranial Nerves	Motor system	Sensory system	Reflexes	Fundus	B. P.	Urine	Blood Urea	C. S. F.				
											Tension	Pr	G	Cl	S
1953	16.	—	—	—	—	Deep + + Lt Plantar ↑ L Bladder +	—	230.120	—	—	+	20	—	750	57
	17.	Slurring	—	—	—	Deep—Rt	—	240.120	—	—	—			...	
	18.	—	—	Power lost 4 Spasticity 4	—	Deep + + + Bladder + Rectum +	—	235.130	—	105	—	30	—	600	56
	19.	—	—	Power N. 4 Tone N.	—	—	—	236.148	—	—	—			...	
	20.	—	—	—	—	—	—	240.120	—	75	—	20	—	750	57
	21.	Dysarth.	—	Power Poor Rt. upper Tone + + +	—	Deep + + +	—	220.100	—	—	—			...	
	22.	Normal	Normal	Normal	Normal	Normal	—	220.150	Alb +	320	—	30	—	650	50.8
	23.	—	7th Rt.	Flaccid Rt. limbs	—	Deep + + Rt Plantar/R Abd. lost Rt	—	244.120	—	—	—			...	
	24.	—	7th Rt.	—	—	Lower Abd— Crem. lost	Hypertensive Retinopathy	200.120	—	—	—	20	—	650	53.2
	25.	—	—	Spasticity Rt.	—	Deep N Plantar ↑ ↑	—	162.98	—	—	—	20	—	750	41
	26.	—	—	Flaccid 4	—	Plantars ↑ ↑	—	190.100	—	—	—			...	
	27.	—	—	—	—	Deep + + lower limbs	—	190.145	—	—	—			...	
	28.	—	—	Power poor 4 P	—	Plantars ↑ ↑	—	170.108	—	60	—	80	+	600	40
	33.	—	—	—	—	—	Haemorrhages	210.110	—	153.8	—	40	—	700	132.5
	34.	—	—	—	—	Deep lost lower limbs	—	160.118	—	45	—	20	—	750	53.5

	No.	Speech	Cranial Nerves	Motor system	Sensory system	Reflexes	Fundus	B. P.	Urine	Blood urea	C. S. P.				
											Tension	Pr	G	Cl	S
	31.	—	—	Power poor right arm, Tone + +	—	Deep + + + Rt arm	—	210.100	—	—	—			...	
	32.	—	Pupils dilated	Power poor 4 Tone poor 4	—	Deep + + 4 Plantars ↑ ↑	—	260.110	—	—	—			...	
	29.	—	—	—	—	—	Haemorrhages	210.110	—	153.8	—	40	—	700	132
	30.	—	—	—	—	Deep lost lower limbs	—	160.118	—	45	—	20	—	750	535
1954	35.	—	—	Neck rigidity +	—	—	—	160.110	—	—	—	150	+	700	100
	36.	—	—	—	—	—	—	170.120	—	—	—			...	
	37.	—	—	—	—	—	Disc swollen Exudate + + Art. Sclero + +	198.146	Alb +	120	—			...	
	38.	—	—	—	—	Deep N	—	174.110	—	83	—			...	
	39.	—	—	Neck rigidity + Kernig's + Rigidity Rt	—	Plantar ↑ L Deep + + 4	—	160.90	—	—	—			...	
	40.	—	—	—	—	Deep + + left leg Plantar ↑ L	—	224..124	—	35	—	30	—	700	41
	41.	—	—	Tone poor Rt	—	Superficial lost Rt	—	—	—	60	—	40	—	750	52
	42.	—	—	Normal	—	Normal Plantar ↑ ↑	—	150.110	Alb ÷ ÷ ÷ RBC +	—	—	40	—	700	91
	43.	—	—	—	—	—	—	180.100	—	—	—			...	
	44.	—	—	Power poor Lt Tone poor Lt	—	—	—	200.110	—	30	—			...	
	45.	—	—	—	—	—	—	150.98	—	—	—			...	

Yawning and Its Significance

by

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YAWNING is a phenomenon familiar to everyone and is usually associated with boredom, fatigue or drowsiness. Though an interesting physiological phenomenon occurring now and then in normal healthy individuals and excessively in certain clinical conditions, scientific literature on the subject appears to be surprisingly scanty. Most standard textbooks of Physiology make no mention of it and the few that do, dismiss it in two or three lines by referring to it as a modified respiratory act. Few textbooks of Medicine refer to it under symptomatology. This is largely because the act of yawning has not been subjected to much study to elucidate its physiological or pathological significance. Some lay magazines however hazard explanations and attach significance to yawning that have little scientific basis. A monograph by Russel (11) has been described by Heusner (3) as "the most exhaustive account of yawning" and that it "should be consulted in the original by anyone interested in whimsical physiology".

Mechanism of Yawning:

Yawning is an involuntary reflex act. It consists of a prolonged deep inspiration with the mouth fully open, followed by a short sharp expiration mostly through the mouth. Mayer (5) divides the act into (i) inspiratory phase consisting of an initial part and acme and (ii) expiratory phase. During the inspiratory phase there is a slow tonic contraction of chest muscles, diaphragm, muscles of larynx, tongue and lower jaw. When the acme is reached, these movements are maintained or accentuated. During the expiratory phase there is a gradual relaxation of the previously contracted muscles; lachrymation and swallowing may also occur. Yawning may be associated with automatic tonic movements involving frequently the muscles of the upper limb and sometimes those of the legs, trunk and abdomen. These movements are of a stretching character and are referred to as 'pandiculation'. Since yawning and pandiculation frequently occur together it has been suggested that they are two components of a single "yawn-stretch" act. Kuntz (4) states that yawning and stretching reflexes appear to be intimately related in their phylogenetic origin and that man has only by practice learnt to separate the two reflexes and voluntarily suppress the stretching reflex. Yawning which is a fractionation of the yawn-stretch reflex by voluntary inhibition of the stretching component, appears to be an adaptation acquired to meet the demands of society. However, since yawning unaccompanied by stretching frequently oc-

curs in infants, and as man has a tendency to yawn without stretching before sleep and stretch without yawning on waking, other factors than voluntary inhibition appear to be concerned in the association or dissociation of the reflexes.

Neural Basis of Yawning :

Yawning has been described as a subcortical basal ganglion reflex and the presence of a yawn centre in the corpus striatum or subcortical ganglia has been suggested (10). Though the existence of a yawning centre or its location has not been proved, the occurrence of yawning in animals with only the medulla intact suggests that if a yawn centre exists, it is probably situated in the medulla close to the respiratory and vasomotor centres (3). Impulses from the higher cortical levels can initiate or inhibit the act of yawning.

Clinical Observations :

Excessive or morbid yawning is associated with some clinical conditions. It has been observed to occur frequently after severe haemorrhage (9), in gastrointestinal disorders especially duodenal ulcers (1) but is most often seen in neurological manifestations. Penfield and Jasper (8) report frequency of yawning in diencephalic autonomic seizures and describe the case of a young man referred to them because of attacks of continuous yawning varying in duration from a few minutes to three hours. Excessive yawning occurs as a sequel to epidemic encephalitis (4, 14), intracranial tumours, especially of the posterior fossa (10) and in infants after birth injuries. (12). An interesting clinical observation is the stretching of the paralysed limbs which occurs sometimes when a patient with severe hemiplegia yawns. The tonic extension and abduction of the paralysed flexed fingers and elevation of the paralysed adducted arm raises false hopes in the patient's mind of a return of voluntary power in the affected limb (10). Heusner quotes the interesting observation of Yakovlev (13) that in basal ganglia lesions yawning with pandiculation is frequent in Huntington's Chorea but rare in Parkinsonism.

Experimental Observation :

Experimental observations on yawning are few. Heusner (3) observed that yawning was accompanied by a digital vasoconstriction and transient cardiac acceleration in normal subjects and in patients with Reynaud's disease or Buerger's disease. In patients after therapeutic preganglionic sympathectomy, digital vasoconstriction was absent or greatly diminished but cardiac acceleration persisted. These findings are similar to previous observations of reflex digital vasoconstriction resulting from voluntary deep inspiration.

Significance of Yawning :

Yawning is variously stated to be a means of toning up the body or relaxing mental tension (2), an automatic expression of cerebral fatigue (5), an effort of the nervous system to correct by an extra deep inspiration, the venosity of blood due to inactivity produced by ennui, or grief (6), an indirect vascular reflex which serves the purpose of improving circulation (4) and that it serves to empty the veins and favours increased arterial supply to the body especially the brain (10). It is said to be due to anoxia or arterial hypoten-

sion in haemorrhage and to afferent impulses from the affected region of the brain modifying the rhythmic activity of the medullary respiratory centre, in brain tumours. (7).

Though much is known about the movements associated with yawning and the experimental finding of reflex vascular response is significant, further observations on normal subjects and in clinical conditions are necessary for a better understanding of this phenomenon. To propound theories regarding the physiological or pathological significance of this act in the present state of our knowledge will be more speculative than factual.

Summary :

The literature on yawning is briefly reviewed and the mechanisms, clinical and experimental observations and significance of yawning are discussed.

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You have no more right to consume happiness without producing it than to consume wealth without producing it. — *Bernard Shaw*

* * * *

I never think of the future. It comes soon enough.
—*Albert Einstein*

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MADRAS—1.

Some Uses of ACTH and Cortisone

by

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ACTH and Cortisone raise the circulating level of the adrenal hormones in the blood — Hydrocortisone. ACTH stimulates the adrenals to secrete the hormone while cortisone is absorbed and converted into the closely allied hydrocortisone within the body. Hence, for the action of ACTH the adrenal cortex should be intact, and so it is useless in the treatment of Addison's disease where cortisone will be the drug of choice.

Normally 25-50 mgm. of Hydro-cortisone is produced and diffuses into the tissue and is soon converted into various metabolites. A balance is maintained between production and destruction. But when the cortex is stimulated by ACTH, the level of blood hydro-cortisone can rise to 50-100 mgm. There is clinical variation in hydro-cortisone production. There is increased production during exercise, stress, anxiety and emotional upsets.

The rise in concentration of circulating hormones enhances the ability of the body to resist noxious agents, thus preventing their adverse influence during an infection which produces stress and strain to the body. But it is difficult to explain how this is brought about. On this is based the alarm reaction and the general adaptation syndrome.

ACTH is obtained by an extract from the anterior pituitary of mammals. Its function is to stimulate the intact adrenals to secrete the hydro-cortisone. This is effective only by the parenteral route. Varying responses are obtained by different preparations, as it is an extraction and not a synthetic product. There may be some inactivation at the site of injection due to local destruction. The adrenal of the average individual responds to a maximum of 2 mgm. per hour. So, if 25 mgms. are given rapidly by I.V., method the adrenal responds only to 1 mgm. by which time the 24 mgm. will be destroyed in the body and becomes useless. The life of ACTH in the blood is only about 15 minutes. So the best method of administration without any wastage will be to give the ACTH in I.V. drip, slowly, taking 10-12 hours (25 mgm.) and 2 mgm. in 1 hour. By this process 10 times the stimulation of adrenals will be achieved more than when given I.V. rapidly. Another variability in the effect in a person may be due to hypo or hyperactive adrenals. If the adrenals are hyperplastic as in mild Cushing's syndrome, a big response will be obtained to the ACTH.

The methods of administration are :

(1) By repeated small doses in 24 hours, but this is not well tolerated.

(2) In combination with gelatin or zinc and an intra-muscular depot is created with one or two daily injections.

(3) Slow I. V. transfusion in saline or preferably in distilled water as a slow drip.

The actions of ACTH are varied, as a number of hormones are secreted by the adrenals by the stimulation of ACTH, namely the Sodium hormone or mineralo corticoid, S-hormones or glucocorticoids (mainly on carbohydrate metabolism), the nitrogen retention N-hormones promoting the anabolism of the body. Of the varied functions of the adrenal cortical steriods, secreted by adrenals, after stimulation by ACTH, an important function, namely the cellular response to infections is one. In the normal course of events whenever there is a noxious stimulus to the body by way of an infection, the mesenchymal tissues react to the agent. The reaction of the tissue to infections is by way of : (a) inflammatory reaction ; (b) allergic reaction. Both types of reactions can be reduced to a minimum if there is an increase in the adrenal cortical activity, compound F (Glyco-corticoids i.e. Cortisone and hydro cortisone) and related cortical hormones. It is by abolishing these fundamental reactions of mesenchymal tissue to injury that the late sequelae of fibrosis and degenerations are prevented. This *Mesenchymostatis Effect* whereby sickness in disease is eliminated, forms the basis for the use of adrenal cortical hormones therapy in diseases other than adrenal cortical insufficiency. In cases such as rheumatoid arthritis, where there is slow progressive inevitable inflammation taking place, by suppressing or reversing this process, amelioration of symptoms is achieved by ACTH. There is a remarkable reduction of the inflammatory process that is proceeding. The clinical relief that the patient achieves by way of reduction in pain, swelling, tenderness and effusion as a result of reversal of the process is immediate. By reducing the inflammatory reaction of the body by ACTH, the defence mechanism that will develop for any coexisting inflammation is also impaired and this is a great draw back in the clinical use of this valuable drug.

CORTISONE

What is achieved by stimulating adrenals by ACTH can be obtained by cortisone orally. Cortisone acetate is very well absorbed and is converted into active hydro-cortisone. Hydro-cortisone is available as the free form as well as the acetate.

Hydro-cortisone (free alcohol)

Hydro-cortisone acetate.

Cortisone acetate.

Hydrocortisone and its acetate are prepared and made as local application for the skin or injection into a serous cavity. Cortisone is not locally useful as it cannot be readily converted into active form locally due to lack of some enzyme. It can be given I. V., or intramuscular, as suspension, oral tablets or topical applications. The dosage depends upon the particular compound, the nature of the disease etc. Roughly cortisone is required about 50-200 mgm. a day.

The broad indications for cortisone are :

(a) Substitution therapy.

(b) Inhibit and control over-activity of abnormal adrenals.

(c) In disease states which empirically respond to use.

It is the last which forms the basis for the widespread use of the drug.

SIDE-EFFECTS and TOXICITY

Any drug has its therapeutic actions as well as its toxicity or side effects and ACTH and cortisone are no exception to this general rule.

SIDE-EFFECTS OF ACTH AND CORTISONE

1. *Gain in weight* :

Due to reproduction of symptoms of mild Cushing syndrome. "Mooning of face occurs during prolonged administration, due to accumulation of fat." The gain in weight is due to :

(a) Accumulation of fat due to increased appetite.

(b) Due to retention of fluid.

This can be overcome by reducing the caloric value of diet and by restricting the Sodium intake.

2. *Psychological changes* :

Euphoria, may be due to steriod therapy or due to relief of pain, change of moods, elation and depression. Some may develop psychosis.

3. *Electrolyte disturbance* :

Na. retention and hypo-potassioma as shown by asthenia, and muscular weakness. By restricting the intake of sodium and giving K side by side with cortisone, this can be overcome.

4. *Androgenic effects* :

Hirsutism and acne vulgaris, amenorrhea or oligomenorrhoea may occur but are rare.

5. *Altered tissue reaction and injury* :

Excess of cortical hormone interferes with the formation of granulation tissues and delays the healing of wounds. So theoretically, surgery should not be carried out in these cases but in practice operations are safe in patients receiving 100 mgms. of cortisone a day or less. Hormones may produce suppression of symptoms. Acute peritonitis without pain, fever or rigidity have developed from the rupture of a gangrenous appendix. There is liability of the lighting up of healed tuberculosis foci due to failure of the defence mechanism during prolonged cortisone therapy. But this does not apply to Addisons disease of tuberculous aetiology.

6. *Gastro-intestinal* :

May increase gastric acidity, bleeding and perforation of peptic ulcers.

7. *Carbo-hydrate metabolism* :

Transitory glycosuria and hyperglycaemia may occur. Hence in severe cases of diabetes, Cortisone is to be avoided.

8. *Cardio-vascular system* :

Raises the blood pressure and is very important in cases where there is pre-existing hypertension.

9. *Blood* :

They may develop thrombo-embolic phenomenon.

10. *Neurological* :

Convulsion may occur especially in children during therapy.

NEWER DRUGS :

Prednisone — (Cortisone)

Prednisoline : (Hydro-cortisone) — The anti-inflammatory and anti-rheumatic effects of these drugs are equal to cortisone, but weight for weight they are 5 times more potent, and hence supplied in 5 mgm. and 1 mgm. tablets. The side effects of the drugs are the same except for the Na retention. This makes them less useful in Addison's and more useful when there is oedema or hypertension.

Cortisone therapy should not be abruptly stopped but is to be tapered down. Normal secretion of adrenal cortical hormones are suppressed due to inhibition of the pituitary (the patient's own ACTH). But this reduced dosage is not necessary, if ACTH is employed. Although there are very many diseases for which ACTH and cortisone are being given and tried with very successful results, I am not going to enumerate all those conditions.

BRONCHIAL ASTHMA

ACTH and cortisone have revolutionised the treatment of asthma. Patients who are longstanding asthmatics do go into Status Asthmaticus very often. Due to the prohibitive cost of the drug and undesired side effects, it should be used only when the time-honoured remedies fail to relieve an attack. Many cases of true bronchial asthma, as well as eosinophilic lung having spasms are relieved by the ACTH drip 20-25 units I.V. daily for six days. Again when the patients cannot be hospitalized, it is given as I.M. injections daily for six days. But the drip method is the method of choice when available. The concurrent antibiotics to control the infection should not be given up. Penicillin with Sulpha as a routine in ordinary cases and in cases of resistant infections, Penicillin with Streptomycin is the drug of choice. In certain instances broad-spectum antibiotics have to be administered by mouth. Cortico-tropin gel 120 mgm. in divided doses may be given in the 1st day and reduced to 80, 60, 40, etc., on the successive days. If, however, oral administration is preferred cortisone 200 mgm. should be given by mouth daily in divided doses. One word of caution is that the drugs should be used only when repeated adrenaline aminophiline fail to evoke any response in a given patient.

Arthritis :

Another group is the one of rheumatoid arthritis. The treatment by ACTH and Cortisone is one that brings about a remission and not a cure of the disease. Here again, despite the prohibitive cost of the drug, it has a definite place in the therapy of the disease. The severe pain that develops during the exacerbation is very troublesome and the patient will be in agony. Here we have but to use the hormones. A person with rheumatoid arthritis with psoriasis all over the body with severe bodily pain was treated on this line and felt relief and the psoriasis also disappeared. Here again in hospital practice, ACTH by the drip method is the method of choice.

Another case of arthritis (post-dysenteric) who was not relieved in the usual

line of treatment, was given the hormone therapy and on the third or fourth day after starting the treatment became better and was able to walk without any pain.

Leukaemia :

Acute leukaemia is one of the blood diseases which is rapidly progressive with fatal termination. So the treatment of this condition is essentially a palliative one. Treatment of the condition with cortisone or prednisoline concurrently with folic acid antagonists or when therapy with folic acid antagonists fail to evoke a response, gives rise to clinical remissions with well being of the person, improvement in the general condition and cessation of bleeding etc. By this procedure, the fatal result is postponed and life prolonged a little more. There is no doubt about the concurrent use of transfusions repeatedly, if possible an exanguinous transfusion, along with haematinics other than folic acid to correct the secondary anaemia which goes hand in hand with the leukaemic process. One should not entirely depend upon this hormone therapy for the treatment of acute leukaemia.

NEPHRITIS :

In oedema of renal origin where there is persistent oedema with massive albuminuria, cortisone and ACTH are given to promote diuresis. It should be made clear that after the usual methods are found insufficient or unsuccessful in relieving the oedema, this should be tried. The other routine methods, one by giving a salt-restricted diet with a high protein diet. Then diuretics like aminophiline, diamox and marsalyl are given. Finally, if failure is met with in this line of treatment, we may have to use the hormone ACTH or Cortisone. The diuretic effect is due to the withdrawal of the hormone after therapy for a few days to produce a relative insufficiency of adrenal cortex. But in practice, it will be noted that the diuretic effect occurs much earlier even before the drug is stopped.

IN RHEUMATIC DISEASES :

Its usefulness lies in the acute phase and has very little use when the valvular lesions are very well established. Once a valvulitis has occurred and fibrosis has taken place with a residual damage to the valve cusps, the hormone is useless unless it is used to control a recurrent rheumatic infection.

It has all the value when there is an acute attack developing with joint manifestation and (pan carditis) the heart takes part in the inflammationory process. The cortisone therapy not only controls the infection and brings about dramatic relief in the symptomatology of the patient, but also makes the cardiac changes retreat. The slight dilatation and the systolic murmur due to myocarditis will melt like ice. The E. S. R. will come to normal due to the anti-inflammatory effect of the hormone. The only disadvantage is that it may precipitate a conjestive failure in a border-line case due to its salt-retaining power. But this should not stand in the way of its wide use in acute rheumatic conditions.

RHEUMATIC CHOREA :

The course is quite uninfluenced by the use of the hormone. Hence it has no place in the treatment of rheumatic

chorea, but can be used in other rheumatic manifestations which also occur along with chorea.

Of the other conditions, one is tropical eosinophilia where there are marked symptoms with wheezing. *ACTH can be used with advantage during the acute phase* but however it may be necessary to administer the usual arsenical compounds in this condition.

In cases of meningitis when, during the fight, the organisms take the upper hand, parenteral administration of cortisone or ACTH may be beneficial.

Of course, the use of the drug in diseases of the skin, eye etc., is not dealt with in this article. Thus, it will be seen that the steriod hormone has become one of the modern weapons that has a very wide use against systemic and local diseases.

"In no country in the world perhaps is more affection lavished on the child and yet who that knows the conditions of India can deny the terrible waste of mother's and child's life that goes on daily."

— *President Rajendra Prasad*

* * * *

Experience is a comb given by Nature to old people when they are bald.

— *Thai Proverb*

* * * *

Some people grumble that roses have thorns; perhaps we should be thankful that thorns have roses.

* * * *

If you want a thing well done, go and ask a busy man; the other kind never has the time.

* * * *

A smile is a thing that adds value to your face without anything to be spent to get it.

* * * *

Nothing contributes more to cheerfulness than the habit of looking at the good side of things.

* * * *

Humility is the surest sign of strength.

* * * *

Hope is the companion of power and the mother of success, for whosoever hopes strongly has within him the gift of miracles.

— *Smiles*

Emulsifying Agents

by

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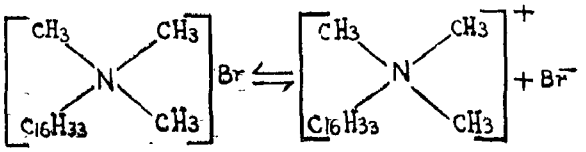
EMULSIFYING agents are substances of widely different chemical properties. Most of them are naturally occurring substances, not well defined chemically and often difficult to classify. The modern method of classification is to group emulgents into non-ionizing and ionizing types.

The ionizing types are divided into "anion active" and "cation active". For example sodium oleate will ionize as follows :—

$$C_{17}H_{33}COONa \rightleftharpoons C_{17}H_{33}COO^- + Na^+$$

The anion, $C_{17}H_{33}COO^-$ is the part of the molecule which possesses the emulgent properties, and this emulgent is therefore spoken of as anion active.

On the other hand, Cetyltrimethyl ammonium bromide is called a cation active emulgent. It ionizes as follows :—



The characteristic group ($C_{16}H_{33}$) is the cation.

Emulgents may be classified as below :

1. Non-ionizing substances : Esters, Ethers etc.
2. Anion-active : Soaps, sulphates, sulphonates.
3. Cation-active ; Quaternary ammonium compounds.
4. Natural Colloids : Gums, mucilages, waxes, proteins, clays.

Non-ionizing substances :

Generally these are esters derived from fatty acids, with 12 to 18 carbon atoms in the chain and an alcohol with suitable properties. The general formula for such emulgents will be RCOOR' where R represents the hydrocarbon and R' represents the alcohol. Both oil/water and water/oil emulgents are possible, depending chiefly on the alcohol chosen. If the polar nature dominates the non polar property of the fatty acid, the result will be an O/W emulgent and vice versa. Monohydric alcohols are of no value in this connection since the simpler types such as ethyl alcohol (C_2H_5OH) are too weakly polar and fatty alcohols such as palmityl alcohol are too strongly non-polar.

Whether the esters formed will be O/W or W/O will depend upon the number of unestrified — OH groups or other polar groups in the molecule. If these are few, the compound will be a W/O emulgent. Glycol mono-stearate or distearate and glycerol mono-stearate are examples of W/O emulgents.

Conversely alcohols with numerous OH groups, or polyethylene glycols with a chain of four or more — CH_2CH_2O groups, combine with fatty acids to form O/W emulgents. Examples of this type are propylense glycol monolaurate, propylene glycol monostearate and sorbitan monolaurate.

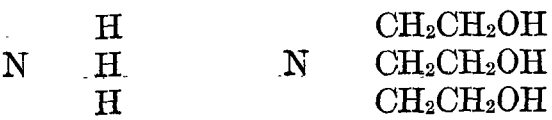
Anionic Emulgents :

This type includes all those emulgents in which the "active" group is included in the anion.

Soaps :

Alkali soaps and other soaps of inorganic elements are simple and typical examples. Soaps of monovalent elements or groups such as sodium or ammonium form emulsion of the O/W type. Soaps of di- and trivalent elements such as calcium and aluminium form emulsion of the W/O type.

In recent years a number of organic amino compounds which form soaps with fatty acids have been tried with success as emulsifiers and have assumed technical importance in different industries. The most important in pharmaceutical practice is triethariolamine. Structurally it resembles ammonia.



Ammonia Triethanolamine

It combines with fatty acids to form soaps as does ammonia.

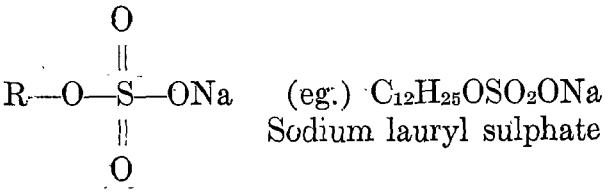
Commercial triethanolamine contains approximately 2 per cent of monoethanolamine $NH_2CH_2CH_2OH$ and 11 per cent diethanolamine — $NH(CH_2CH_2OH)_2$ — these having similar emulsifying properties.

Mono-, di- and tri- isopropanolamine have also been found useful in the preparation of cosmetic emulsions.

Organic Sulphates and Sulphonates :

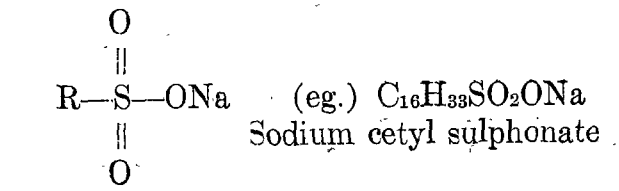
In sulphates, the sulphur is linked to carbon through an atom of oxygen, whereas in the sulphonates there is a direct linkage of sulphur to carbon. Such compounds may be exemplified by the following emulsifying agents :-

Sulphates



General Structure

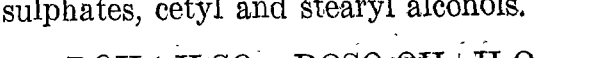
Sulphonates



General Structure

Sulphates :

An example well known to pharmacists is lanette wax S X ; this consists of a mixture of cetyl and stearyl alcohols which have been partly (10 per cent) converted to sulphates. Halden's Emulsifying Base (H. E. B.) also contains sulphates, cetyl and stearyl alcohols.



Lanette wax SX which acquired a deserved popularity with pharmacists for the preparation of water-miscible ointment bases, was first used officially for the preparation of penicillin creams. It has been replaced by emulsifying wax the preparation of which from cetostearyl alcohol and sodium lauryl sulphate is described in the pharmacopoeia.

Sulphonates:

These are more difficult to prepare than the sulphates, the direct link between sulphur and carbon requiring the use of more vigorous reactants.

Igepon-T is one of the many patented products, being the sodium sulphonate of the oleic acid derivative of taurine :

$C_{17}H_{33}CONH\ C_2H_4SO_3Na$. This emulgent is interesting, as it is similar in general structure to the natural product sodium taurocholate, one of the bile salts which plays the role of emulgent in nature. Sodium taurocholate :

$$C_{23}H_{36}(OH)_3CONHC_2H_4SO_3Na.$$

Cationic Emulgents:

The emulsifying properties of certain quaternary ammonium compounds were described by Reychler in 1913 and in recent years have assumed considerable importance because, in addition to their emulsifying properties, a few of them have a bactericidal action and are used in ever increasing quantities in skin disinfectants in medical practice. A very good example of this type is cetyl trimethyl ammonium bromide.

Natural Colloids :

Some of them long used in pharmaceutical practice are — gums and mucilages from vegetable sources, starch, alcohols of bees wax and wool wax, sterols from woolwax, lipids from egg volk, proteins such as gelatine and casein, bile salts etc. In addition, derivatives of certain natural substances (eg.) cellulose and alginic acid are well known.

The best examples are gum and mucilage of Irish Moss. The former is the best known emulsifier in dispensing practice, producing as it does good O/W emulsions by mortar and pestle methods. Mucilage of Irish Moss, which is prepared from the seaweed Chondrus crispus by digesting it in water on a water bath, yields excellent O/W emulsions by mechanical methods.

Phospholipids:

Lecithin, the chief emulcifying factor in egg yolk, is a phospholipid. Previously egg yolk was much used for emulsions containing acids since it is stable in acid solution.

Carbohydrates:

Dextrin is the emulsifying agent in codliver oil and malt extract.

Starch is a poor emulsifying agent, but is sometimes used in the preparation of enemas containing oils.

Cellulose derivatives commonly ethers, have become very popular in recent years as stabilizers, having excellent thickening properties.

Soluble salts of alginic acids are good stabilizers, giving mucilages of good viscosity. Alginic acid is a polymer of dextro mannuronic acid.

Sterols:

Cholesterol from wool wax is one of the commonest of these. It produces O/W emulsions, although the wool wax and wool alcohols derived from wool wax promote W/O emulsion. The bile salts sodium taurocholate and sodium glycocholate are also familiar examples.

Proteins:

A number of proteins of which gelatin and casein are examples are also used on occasions in pharmaceutical emulsions.

Solids as Emulsifiers:

The two following are worthy of special mention, being used in pharmacy :-

Aluminium Hydroxide:

A form of this substance, prepared by a special process and marketed under the name of Unemul, is an emulgent of the O/W type used in the preparations of water-miscible ointment bases and creams.

Bentonile:

This is a colloidal clay found naturally in parts of America. It has a wide use as suspending and emulsifying agent. Purified forms are used in medicine both for internal and external use.

Very often he that his money lends
Loses both his gold and his friends.
— *C. H. Spurgeon.*

* * * *

A liar needs a good memory.
— *Quintilian*

* * * *

Of all the causes which conspire to blind
Man's erring judgement, and misguide the mind,
What the weak head with strongest bias rules,
Is pride, the never-failing vice of fools.
— *Pope*

* * * *

Better never trouble Trouble
Until Trouble troubles you ;
For you only make your trouble
Double-trouble when you do.
— *David Keppel.*

* * * *

Personality is to a man what perfume is to a flower.
— *Charles M. Schwab*

Metamorphosis of a Medico

by

MR. F. J. RAYEN, IV YEAR, M.B.B.S.

Enters the freshman with a soul so bright
Only a few hours and his face looks like a mite
For no senior condones cranial weight
Which makes puny pedants think mighty great.

Tip-top to the dissection theatre he goes
Pin-drop silence he meekly shows,
Releases the tie-knot for fear of strangulation
Preserving that noose for the day of graduation.

Holds with unsoiled hands a frog
Dissected and it twitches as if it tasted grog
Cuts the muscle and the nerve sciatic
Leaving the amphibian a poor paralytic.

To the examination he goes undaunted
Annoying the examiners with things unwanted;
Then says "No medical course is complete
Without a short but airy respite."

That respite he gains by being subjectional
To a short course as an additional;
Rejuvenating with refresher courses
Which have in them inherent forces.

The second M. B subjects hurt him to the core
They really make him very sore,
So from his unconscious stores
Thoughts fly of the one he adores.

Medicine and surgery are his pet subjects
'Cause the practical victims are human objects,
Which gives him that great feeling
That he is already doctoring.

Wards are places to show off
Cheering the patients with a laugh;
Carrying a rubber contraption called a stethescope
Washing his hands in lotion instead of soap.

Coming out of the course as a student interne,
That name it dotli sound so funny
Wishfully dreaming one day over a cup of tea
How to get M S. or M. D.

90% of the friction of daily life is caused by the tone of voice.

* * * *

Your friend is one who knows all about you and still likes you.

* * * *

Do not feel discouraged — it may be the last key in the bunch that opens the door.

* * * *

A man may fail many times but he is not a failure until he begins to blame somebody else.

* * * *

There are only two ways of being happy, — either augment your means or diminish your wants.

* * * *

Tomorrow

1. He was going to be all that he wanted to be — tomorrow;
   None would be kinder or braver than he — tomorrow.
   A friend who is troubled and weary he knew
   On him he would call and see what he could do — tomorrow.

2. Each morning he stacked up the letters he would write — tomorrow
   And had'nt one minute to stop in his way —
   More time I would have to give to others, he would say — tomorrow;
   And thought of the folks he would fill with delight — tomorrow.

3. The greatest of workers would have been — tomorrow
   The world would have hailed him, had he ever been — tomorrow.
   But in fact he passed on, and he faded from view
   And all that he left here when living was through
   Was a mountain of things he intended to do — tomorrow

* * * *

When we have not what we like, we must like what we have.

* * * *

The fly sat upon the axle-tree of a chariot-wheel, and said, 'What a dust do I raise!
— ***Francis Bacon***

Case Notes

A CASE OF OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY

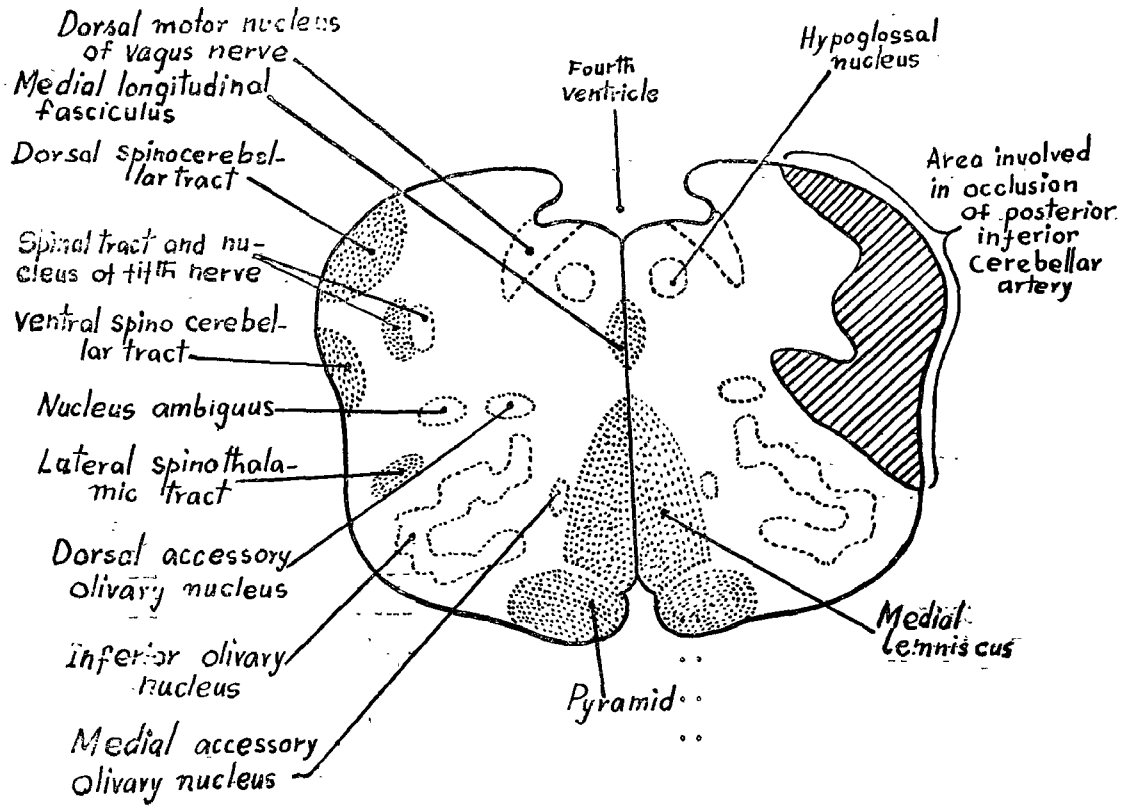
(Lateral Bulbar Syndrome)

by

MISS S. KRISHNAVENI, II M.B.B.S. class.

Introduction :

THE study of a case of occlusion of the Posterior Inferior Cerebellar artery is an exercise in applied Neuro-anatomy. This artery is the longest and largest branch of the vertebral artery, arising at right angles to the parent stem. It passes between the rootlets of the hypoglossal and vagus nerves and courses obliquely over the inferior cerebellar peduncle to reach the inferior surface of the cerebellum, over which it ramifies. The artery is noted for its variations in distribution and in a large majority of cases, the penetrating branches supply the upper and outer half of the medulla. In a few cases these branches reach as far as the sixth and seventh nerve nuclei in the pons. The accompanying diagram shows the anatomical structures supplied by this vessel and if this area is rendered ischaemic, the symptomatology may readily be followed.



The aetiology of occlusion is thrombosis of the vessel and the course is usually benign. Extension of the thrombus to the vertebral artery results in death.

Case:

Soundaram Chettiar, a male aged 49 years was admitted on 1—2—1957 with a history that a fortnight ago he experienced a sudden violent vertigo and nasal regurgitation of fluids, followed by inability to use the left half of the body. He stated that he fell down after the attack of vertigo and an attempt to stand was followed by a fall to the left. He was given fluids to drink and he noticed nasal regurgitation and dysphagia. Almost simultaneously he felt inability to use the left half of the body and developed hoarseness of voice.

General Examination :

A well nourished individual. Pulse, temperature, respiration and B. P., were normal. He had no syphilitic stigmata and was a non-diabetic.

C. N. S. — Higher intellectual functions : Normal.

Cranial Nerves :

1st and 2nd normal.

3, 4, 6 : Ptosis, enophthalmos and miosis on the left (Horner's Syndrome). Horizontal nystagmus on movement to the left.

5th : Sensory :
subjective — nil.
objective — Loss of pain and temperature sensations over the distribution of ophthalmic and maxillary divisions, on the left.
Motor : Normal.

6, 7, 8 : Normal.

9th and 10th : Uvula deviated to right. Palatal and pharyngeal paralysis left. Hoarseness of voice — laryngeal paralysis.

11th and 12th : Normal.

Motor :

The abnormal signs were noted on the left half of the body below neck. Impaired voluntary movement of the left limbs and inability to turn to the left. Loss of tone of muscles. No wasting, intentional tremor and dysdiadokokinesis present.

REFLEXES :

Cutaneous :

Corneal and Conjunctival :
Abdominal and cremasteric : } Lost on the left side.

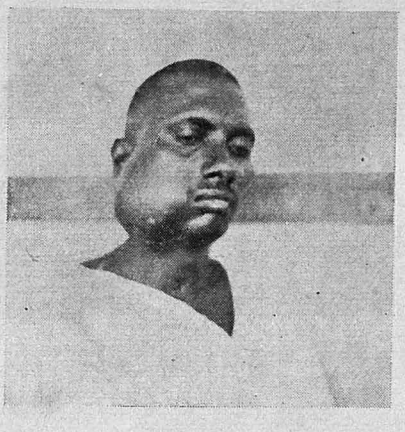
Tendon :

	Right	*Left*
Biceps ..	++	+
Triceps ..	++	+
Supinator ..	++	+
Knee ..	++	±
Ankle ..	++	±

(++ = normal.)

Babinski : Present on the left.

Clinical Quiz



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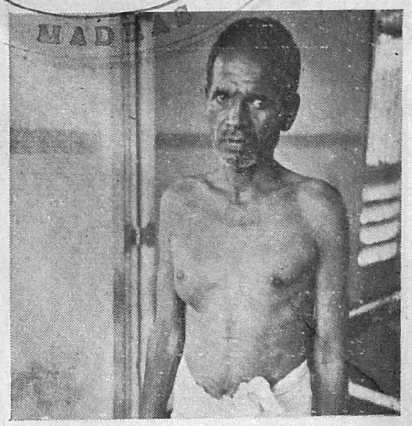

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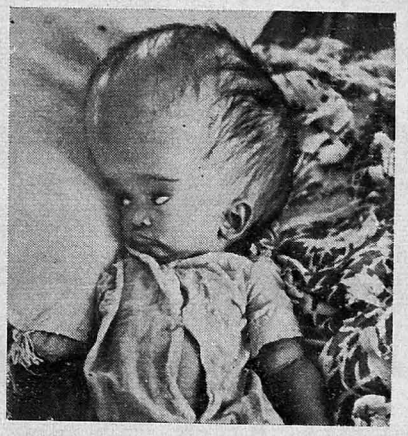

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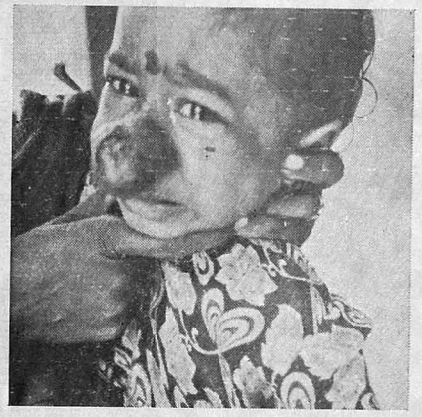

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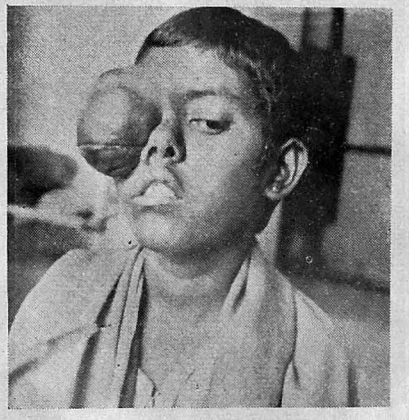

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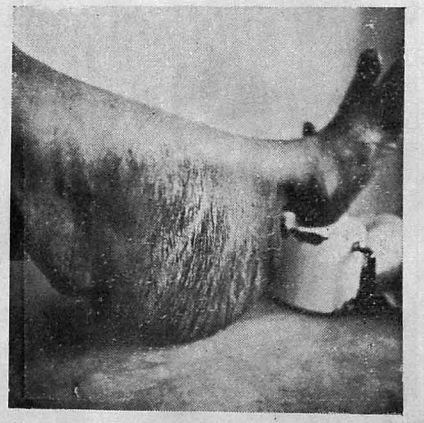

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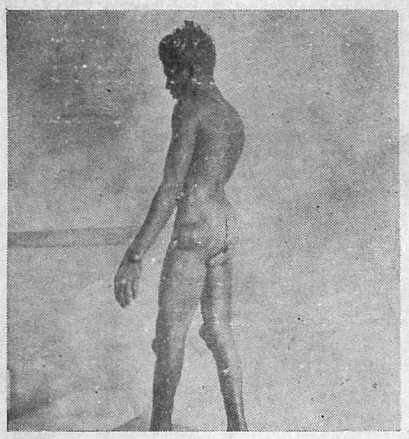
Clinical Quiz (contd.)



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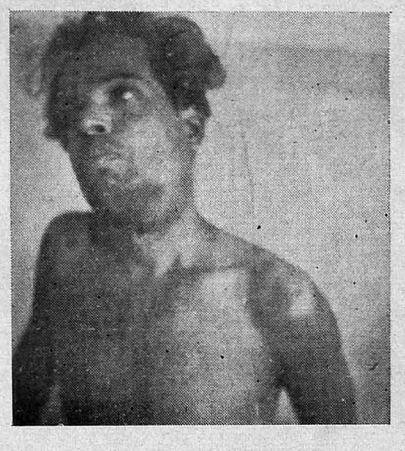

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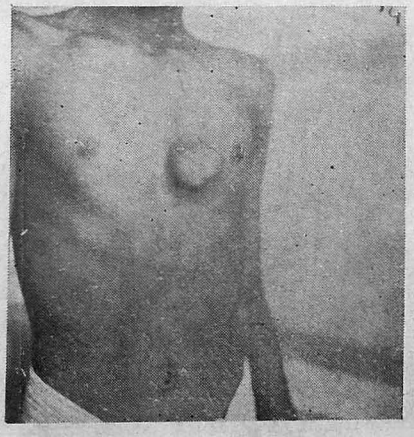

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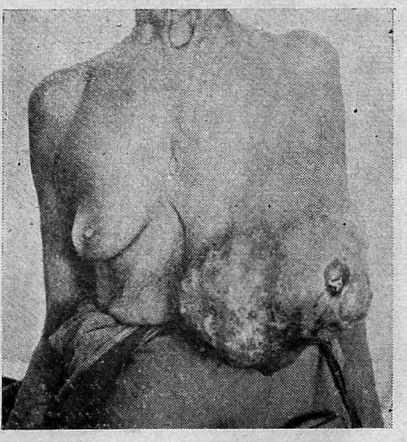

11

SENSORY :

Subjective	:	Nil	
Objective	:		
		Right	*Left*
Pain and Temperature		Lost below the angle of the mandible.	Lost over the maxillary and ophthalmic divisions of trigeminal.
Touch	:	Present.	Present.
Posterior column	:	Present.	Present.
Stereognosis	:	Present.	Present.

AUTONOMIC :

Horner's syndrome	:	left.
Sphincters	:	Normal.

OTHER SYSTEMS : Nil abnormal

Investigations :

Lumbar puncture	:	Clear fluid under normal pressure.
Biochemical	:	Nil abnormal.
Blood Kahn	:	Negative.

Treatment and Progress :

(i) Nasal feeding with milk and orange juice.

(ii) I. V. Nicotinic acid 200 mg. in a pint of 5% glucose saline.

The patient showed a sudden change on the third day of admission. At 10-30 A.M. he became unconscious with conjugate deviation of the eyes to the right, pallor of the face and stertorous breathing. He died an hour later.

Discussion :

The sudden onset of violent vertigo followed by fall to the left are the manifestations of ischaemia to the region of the Deiter's nucleus. Loss of function of 9th and 10th nerves account for the ipsilateral palatal, pharyngeal and laryngeal paralyses resulting in nasal regurgitation, dysphagia and hoarseness of voice. Cerebellar ataxia, hypotonia of muscles and dysdiadokokinesis on the left, result from lesion of the inferior surface of the cerebellum. Lesion of the sympathetic fibres on their way down to the cilio-spinal centre results in Horner's syndrome on the left. The presence of Babinski's sign on the left is due to oedema spreading on to the pyramidal tract. Involvement of the descending root of the trigeminal explains the loss of pain and temperature over the ophthalmic and maxillary divisions on the left. Loss of pain and temperature sensations on the right are due to involvement of the crossed spinothalamic tract.

The acute onset and characteristic symptomatology discussed, places the lesion in the left posterior inferior cerebellar artery. The aetiology is probably thrombosis of an atherosclerotic vessel. The

sudden change in the condition of the patient on the 3rd day of admission and subsequent death can well be due to the extension of the thrombus to the parent stem.

Summary :

A case of occlusion of the Posterior Inferior Cerebellar Artery has been recorded. A study of correlative neuro-anatomy explains the symptomatology of this syndrome.

Acknowledgments :

My thanks are due to the Superintendent, Government Erskine Hospital, Madurai for permitting me to publish this case.

My thanks are also due to Dr. M. D. Ananthachari M. D., Physician and Professor of Medicine, Madurai Medical College, Madurai for his guidance and also to Dr. R. S. Rajagopalan M.D., D.T.M., Medical Registrar, Madurai Medical College for his kind help.

Gratitude is the memory of the heart.
— *J. B. Massieu*

What is history but a fable agreed upon ?
— *Napoleon Bonaparte*

There is so much good in the worst of us,
And so much bad in the best of us,
That it hardly becomes any of us
To talk about the rest of us.
— *Anon.*

There are four sorts of men :
He who knows not and knows not he knows not : he is a fool — shun him ;
He who knows not and knows he knows not : he is simple — teach him.
He who knows and knows not he knows he is asleep — wake him ;
He who knows and knows he knows : he is wise — follow him.
— *Arabian Proverb*

Ignorance of the law excuses no man : not that all men know the law, but because 'tis an excuse every man will plead, and no man can tell how to confute him.
— *John Selden*

We cannot hope to build a better world without improving the individual. Towards this end, each of us must work accepting his share of responsibility in the general life of humanity.
— *Marie Curie*

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The Drama of Human Life

by

Mr. C. Dhanasekara Pandian, II M.B.B.S., class

HUMAN life starts, not at the time of parturition but at the moment of conception i.e. the union of the male and female germ cells in the mother's genital tract. Both the parents contribute equally to the physical and mental features of the offspring in the form of chromosomes. It is at this time, that familial diseases like haemophilia, colour blindness and diabetes, can also be inherited.

The single cell zygote or fertilised ovum derives all its nourishment from the hospitable mother and grows at a terrific speed and becomes a full-term foetus at the end of nine months.

The two main processes, namely, growth and development, are maintained during the creation of the miniature human being, the fetus, as well as after its birth. Growth means cellular proliferation. By development is meant the maturity of the cell i.e., the cell attaining its specific functional capacity.

Growth of brain cells and voluntary muscles cease in the intra-uterine life itself. These cells cannot be replaced by new cells, if they happen to die in later life. Most of the organs, including liver, stop growing at birth. Only those tissues like the dermis and blood which are short-lived and are incessantly undergoing wear and tear, retain the power of regeneration. The intra-uterine life is mainly engaged in growth while the post-natal life is chiefly occupied by development.

It is wonderful how this small, feeble and dependent newborn infant becomes a clever and cunning man or a charming and smart lady. When the infant first comes into this cooler atmosphere, sensory impulses from the skin stimulate its respiratory centre and the act of respiration begins by the entry of air into the lungs. The heat regulating centre is not working properly until the age of two, whence it gets slowly stabilised till puberty, in parallel to the development of the thyroid gland. The thyroid looks after the development of both the brain and the body.

During the first two years of life the digestive system undergoes a lot of growth and development. The stomach increases its capacity 10 to 20 times in the first two years and more later. The liver is relatively large at birth and its weight increases about 10 times while reaching adulthood. At the sixth or seventh month of age milk teeth arise. By the end of 18 to 25 years the permanent set of teeth fully set in.

At birth the communication between the left and right auricle is blocked and so also that between the aorta and the pulmonary artery. The lungs receive all

the blood from the right ventricle. The blood pressure steadily rises and the pulse falls down with age. The heart and kidneys are relatively large at birth and a major portion of their growth occurs at the first two years of post-natal life.

The skeleton is mainly made up of cartilage at the time of birth. During early years of life secondary centres of ossification appear in the epiphyses of long bones. The metaphysis or the epiphyseal cartilage becomes the site of growth and it is converted into bone only after the linear growth of the bone is completed. As the child begins to walk and do other muscular exercises, the skeletal muscles and the bony spines and tubercles develop. The osteoclasts and osteoblasts work constantly to maintain the thickness and shape of the bone.

The maximum amount of growth of the cranium occurs in the first year during which time its capacity is doubled. By the end of 18 months the fontanellæ close completely. The cranium reaches its full size by the age of six years. When compared to the growth of the cranium, the bones of the face lag behind. Their active growth takes place during the teen age.

In the first two years of post-natal life, the brain attains over half of its full size. At the end of 9 years, growth is practically complete. But the nervous system is very immature at birth. Its development occurs slowly. Hearing and touch sensation of lips and tongue are present at birth. Only after 3 months the baby is able to visualise objects. It cries when its stomach is empty.

The infant then slowly learns to turn the head, focus the eyes and co-ordinate them in following a moving object. The child cries or smiles to express its emotional feelings. The movements of eyes and hands are co-ordinated. It distinguishes strangers from friends. In the second year the child begins to walk and gains control over the rectum and bladder. It is curious to know about all things around.

Between the ages of 6 to 11 i.e. the early school life, the character and behaviour of the boy or girl, are determined. Understanding and intelligence develop rapidly. Skills, motor and verbal, advance quickly with practice.

During the critical period of puberty and adolescence, maximum changes take place both in the body and mind. Growth and development in this period, namely between 11 and 21 largely depend upon the hormones secreted by the anterior pituitary, adrenals and sex glands.

At this stage the legs lengthen, the trunk grows in girth and the vital capacity increases. Blood pressure is stepped up. While the cranium has finished growing, the face bones suddenly grow fast. The pores of the skin of the face are enlarged and the sebaceous secretion is increased. As a result of this, there may appear pimples in the face for which lads and lasses need not worry at all, as they will automatically disappear in the appropriate time. The gonads, stimulated by the pituitary, act on the internal and external accessory sex organs which become mature.

Puberty is attained about two years sooner in girls. The girl begins to mature

at the age of 11. She then shoots up 3 inches or so in a year changing from a plump little girl to slender young maiden almost over-night. The changes are first seen in the breast by enlargement, accompanied by widening of pelvis. The hips are thrown outward and the thighs converge to the knees. Hair appears around the external genitals and later in the axillæ. At 13, in average the girl has her first menstrual period, dignified, as menarche indicates the release of the first ovum from the ovary. By the age of 15, the adolescent girl, now called woman, reaches her maximum stature.

Puberty sets in for the boy on the average at 13. He gains rapidly in height and strength. His shoulders widen and the muscular contours become prominent and his voice turns hoarse. As the accessory sex organs mature, spermatogenesis begins. Appearance of the pubic hair precedes that of the armpit and face. By 17, the growth of stature is almost complete.

The psychological changes occurring at puberty are profound. Oestradiol and testosterone work a revolution in the attitudes of young girls and boys. The girl becomes self conscious and modest. She becomes sensitive to criticisms of others, especially of the opposite sex. She tends to develop more interests of a passive nature.

Adolescence is followed by adulthood, middle age, and old age. The bones undergo progressive changes from adulthood. They lose their minerals and become fibrous and brittle with the advancing age. Folds and wrinkles begin to appear in the skin which gradually loses its shiny appearance and elasticity due to gradual withdrawl of the subcutaneous fat. There is gradual loss of elasticity in the lungs. Organs of digestion do not suffer much from the effects of aging except the tendency to develop cancer, perhicious anæmia and formation of gall stones. The cartilages of the respiratory tract tend to ossify. Maintenance of blood sugar level, heat regulation and acid-base balance suffer as the individual is getting old. Wounds take longer time to heal. There is a steady decrease in the number of taste buds and the sense of smell also declines. In the brain and spinal cord white matter suffers more than the grey and the frontal cortex more than the rest.

The reproductive system finishes its activities in the fifth decade of life. Menopause is sharp-cut in the female and occurs between 45 to 50. It is usually fairly late and insidious in the male. There is no age limit for spermatogenesis.

The heart and blood vessels first feel the effects of ageing. "A man is as old as his arteries". Arteries lose their elasticity and their lumen gets narrowed by subendothelial deposition of cholesterol and other lipids. Blood pressure rises to increase the strain of the ageing heart. Thrombosis and extravasation frequently happen.

There is a slow decline in all learning activities. The factor of speed is lost but experience comes into play in all activities. We must engage ourselves in those interests and activities that will keep us young at 90.

Death slowly steps into our body activities. It brings to a close the perpetual

expenditure of energy that is characteristic of life. Until our prime, disease and accidents are the great enemies of long life. Then we have to meet with gradual wearing out of the bodily mechanism.

Below the age of forty, death due to accidents is many times more than to diseases in the more advanced countries. There, most of the dreadful killing diseases like typhoid, cholera, plague, smallpox, diphtheria and pneumonia are won over by man. When a man escapes all the traps of death by infectious diseases and accidents and enters his sixth decade of life, he is usually engulfed by coronary catastrophe, apoplexy or cancer. Cancer, which is the greatest and still challenging enemy of mankind, scores over the rest. Let us await with fond hope the day of declaration of victory over this — the greatest enemy of the human race.

We have the past with its memories, the present with its duties, and the future with its anticipations — one for wisdom, one for action, and one for hope.

* * * *

A hasty man drinks tea with chopsticks.
— *Japanese proverb*

* * * *

Enthusiasm is one of the most powerful engines of success.

* * * *

Education is not information, but the ability to use information.

* * * *

We are given two ears, but only one mouth ; because we should talk only half of what we hear.

* * * *

Kind hearts are the gardens,
Kind thoughts are the roots,
Kind deeds are the fruits.

* * * *

He who cannot forgive others, breaks the bridge over which he must pass himself, for every man has need to be forgiven.

* * * *

An optimist sees an opportunity in every difficulty ; a pessimist sees a difficulty in every opportunity.

On Pleasure

by

Miss K. Kamala, III year M.B.B.S.

COMING forward and seating himself on a big stone, an old deaf man began to talk to his semi-deaf friend who was at brown study.

Old man : I have left to the last, the little room for winter evenings.

Friend : Sure, sure. The strong-backed and neat-bound volume published last winter was nothing but nonsense. Because.....

Old man : I rather prefer to go walking than to be bumped about in your old bullock cart.

Friend : Without doubt buying and selling are perfectly straight-forward matters. I say it on the basis of .....

Old man : Stop it. How foolish you are to think so low of our profession.

Friend : You mean that brown-capped gentleman. I thought of the new gardener.

* * * * *

I couldn't help laughing when I heard this conversation. My friend who had been indulging all this time in plucking a cluster of flowers from the thorny bush, turned round and asked me what pleasure I had in laughing at. Without waiting for my answer she again concentrated her attention on the flowers.

"*Pleasure*" — what sort of mixed cloudy feelings this single word evoked. The sun was sinking below the horizon. Puffed by the sweet breeze and eternal beauty of the evening atmosphere, I plunged into the sea of thoughts to emerge with a good resolution.

"*What is pleasure?*". It is after all a sense of delight or enjoyment. In olden days pleasure was something which people indulged in, in an intellectual effort. The great poets immortalised the out-bursts of their pleasure as poems and sonnets, the essayists as literature, the artists as lovely portraits and beautiful scenes and so on. The people actually merged with the art and found pleasure in it.

The new scientific inventions changed all that. Now-a-days, personal initiation is buried in depths. Ready-made pleasures are available at cheap rate, since cinemas, radios and televisions have made their appearance with bustling and pride. Not even the participation of the pleasure-seeker is needed here ; all the intellectual effort demanded of him, is his sitting and seeing the cinema or hearing the radio. Nobody minds whether he understands or not, what his senses perceive.

In our field, what do we do as students? Our greatest pleasure is to buy a heap of new books in the beginning of the term. " Pathology ", " Pharmacology ", Bacteriology", "Ha.....Ha". How high-sounding they are ! The very feel of the smooth paper delights us. The smell of the new books swells us. How patiently and impartially we turn the pages of each book — the beautiful diagrams ! the X-Ray plates ! ! the clinical photographs ! ! ! — We become practically mad with joy. With the overcoat on our shoulders and steth. in our hands we fancy ourselves as great physicians and surgeons though in reality we can hardly do a single minor operation. Carried by these illicit imaginary pleasures, we have little time to focus our attention to the real pleasure of learning and becoming wise. Now let us join with the good English writer and say " Real pleasure lies in true wisdom. True wisdom is to know what is best worth knowing, and to do what is best worth doing."

Knowledge not gratis comes. Nor without fees,
On sick men would attend Hyppocrates.
When any doctor first attends a call,
The patient would bestow the world and all;
But once relieved and safely out of bed,
The doctor's aid from memory has fled.

— *Medical School of Salerno, Code of Health.* (11th. Cent.)

* * * *

Doing an injury puts you below your enemy;
Revenging one makes you but even with him;
Forgiving it sets you above him.

* * * *

Strong foundations are necessary for a towering structure.

Reports of the Madurai Medical College Students' Associations

REPORT OF THE FINE ARTS SECTION
1956—'57

The election of the office-bearers of the Section for the year 1956—57 was held in September, 1956 and the following were elected as the office bearers :

Secretary :
Mr. J. D. Babu Bryant.

Joint Secretary :
Mr. C. Kesavan Kutty.

Lady Representatives :
Miss T. Padmavathy.
Miss B. Kamala.

Class Representatives :
Mr. Sam E. Rajiah.
Mr. T. Rajagopalan.

Dr. T. S. Chelvakumaran, B.A., M.B.B.S., M.S. (Mich.) Professor of Anatomy, Madurai Medical College, has accepted the office of the Vice-President. Dr. V. C. Anguli, B.Sc., M.D.. Lecturer in Pathology and Dr. K. Arumugam, M.B.B.S., Tutor in Pathology, Madurai Medical College, are the staff advisers.

The section had no funds at the beginning as no regular subscription was collected from the students this year. On 4—3—1957, a benefit performance was given by Mr. N. Sundaram, B.A., Shakespearean mono-actor from Madras, at our College premises. Students, staff members of the College and other distinguished gentlemen of the city made moderate contributions as voluntary donation and attended the entertainment in large numbers. We are grateful to Mr. N. Sundaram for his excellent performance.

An orchestra was arranged as one of the main items for the Hostels Day. It was considered as a fine one. The section took part in giving the necessary guidance and help to the variety entertainments staged by the students of the college on Hostel and Pongal Day celebrations.

Greater activity is expected with the beginning of the next academic year in July when we expect a good number of new students to the institution.

C. Kesavan Kutty,
Joint Secretary.

HOSTELS ANNUAL REPORTS 1956—'57

MEN'S HOSTEL

I feel very privileged to place before you the annual report of the Madurai Medical College Hostel (Men's Section) for the year 1956—'57.

The Hostel strength is 115. The freshmen numbering thirty were given a rousing welcome on their arrival at the hostel.

The Hostel elections were held on the 11th of August, 1956. Every post in the executive committee was keenly contested. The elections were held in the best of spirits and the following were elected as office-bearers for the year 1956-57. :

Mr. N. Ramachandra
General Secretary

Mr. V. Navaneethakrishnan
Non-Vegetarian Mess Secretary

Mr. K. Kumaraswami
Vegetarian Mess Secretary

Mr. K. C. Nambiar
Sports Secretary

Mr. A. Thiagarajan
Reading Room Secretary

Mr. S. Rajendran
Member-in-Charge of Radio

NON-VEGETARIAN MESS MEMBERS

1. Mr. Muthuvirumandy
2. Mr. Mohammad Kasim
3. Mr. M. P. S. Menon
4. Mr. D. Raja

VEGETARIAN MESS MEMBERS

1. Mr. N. Soundararajan
2. Mr. Veerabadran

Dr. M. D. Ananthachari, M.D., our Principal inaugurated the Hostel Student's Association on 31st of August, 1956. In his address he stressed the value of democracy and told us to practise it in as best a way as we could. In his college days, he said, he had missed experiencing hostel life ; further, he wanted us to achieve distinction in all our activities.

Dr. K. P. Anandan, M.B.B.S., M.SC., who was the Warden, Men's Hostel, till 3rd September, 1956 had been transferred to Madras. A combined College and Hostel dinner was given in his honour. The members of the Hostel expressed their gratitude and affection for him for the interest he had taken in running the activities of the hostel. We wish him all the best in the future.

MESS

Our Mess Secretaries have been competing very hard to attract members to their respective messes. The average daily rate in the Non-Vegetarian Mess was Rs. 1—9—0 and the average daily rate in the Vegetarian Mess Rs. 1—8—6.

READING ROOM

The following dailies and magazines were provided in the reading room :

Daily : The Hindu, The Mail and the Tamilnad.

Weekly : The Time, Illustrated Weekly, Sports and Pastime, Mathrubhoomi, Kalki, and Kumudam.

Monthly : Reader's Digest, Life, Kalaimagal, Kalai-Kadir and Manjari.

RECREATION HALL

Our Recreation Hall besides being equipped with amenities for indoor games — such as Table-Tennis, Chess, Carroms, is also equipped with a seven valve Philips Radio and a record changer. By the generous donations from the members of the Hostel, we have purchased nearly fifty records containing songs in Tamil, Hindi and English.

We are grateful to our Principal for the help and guidance he has given us in running the activities of our hostel.

We are also grateful to our Warden who despite being the Assistant Principal has given us all the encouragement necessary in running the activities of our hostel.

To conclude, I can say that life in the Madurai Medical College Hostel is as exhilarating as ever.

N. Ramachandra
General Secretary

WOMEN'S HOSTEL

Building and accommodation :

The building of the Women's Hostel consists of 26 rooms in the ground floor for the accommodation of 52 students. The present strength of the hostel is 26, the number having increased by 16 during the academic year.

Hostel Executive Committee :

There is an Executive Committee common to both the hostels with three representatives from the ladies' Section. The following students were unanimously elected as the office-bearers for the Women's Hostel Executive Committee on 10—8—1956.

Miss J. Saroja
Hostel Secretary

Mrs. V. Sangeetham
Non-Veg. Mess Representative

Miss K. Karthiyayini
Veg. Mess Representative

Miss S. Krishnaveni
Reading Room Secretary

Mrs. K. Vijayalakshmi
Clinical Representative

Miss T. Padmavathy
Pre-Clinical Representative

Mess :

There is no separate mess for the women's hostel. The food for the women students is brought to the dining room of the women's hostel from the men's hostel mess and served.

Sports :

The women students have got facilities for playing throw-ball, badminton and tennikoit and our students have won laurels in the inter-collegiate matches held at Madurai in badminton.

In the common recreation room, the students have got facilities to play carroms.

There is a magazine section in the common hall where the students are provided with the following newspapers and journals :

The Hindu.
Sport and Pastime.
The Illustrated Weekly of India
Reader's Digest.
Journal of the Indian Medical Association.

Out of the amenities fund collected for the past one year from the students, one Philips Radio has been purchased for the hostel at a cost of Rs. 600/-. The students use the radio only at fixed timings, so that it does not interfere with their study hours.

Extra-curricular activities :

Apart from their activities in sports, the students take keen interest in exhibiting their talents in music and dramatic activities in all the college and hostel functions.

J. Saroja
Hostel Secretary

MADURAI MEDICAL COLLEGE STUDENTS' ATHLETIC ASSOCIATION REPORT FOR '56—'57

The Students' Athletic Association of the Madurai Medical College was formed as a separate body as soon as the freshers came in August, 1956. A constitution was framed for the association and the committee members were elected.

President	—	Dr. M. D. Ananthachari M. D.
Vice-President	—	Dr. M. N. Guruswami B.A., M.B.B.S., M.S., (Michigan)
Treasurer	—	Dr. T. Narayanaswamy M.B.B.S.
Staff-Secretary	—	Mr. V. S. Austin B.A., D.P.E.
Staff-Representatives	—	Dr. K. Sivarama Mudaliar M.B.B.S.
		Mr. A. Alagiriswamy B.A.
Student Secretary	—	Mr. K. P. Mathew
Lady Representative	—	Miss Charlotte Indra Bai.

The following Captains were elected for the following games :

MEN

ATHLETICS	...	K. C. Madhavan
CRICKET	...	F. J. Rayen
FOOT-BALL	...	K. C. Nambiar
HOCKEY	...	Sembon David Clement
VOLLEY-BALL	...	G. Vijayarengam
BADMINTON	...	R. Thangavelu
BASKET-BALL	...	S. Selvaraj
TENNIKOIT	...	S. Desingaraja
TENNIS	...	M. Bhaskaran

WOMEN

ATHLETICS	...	Miss K. Karthiayini
BADMINTON	...	Miss T. Padmavathi
TENNIKOIT	...	Miss G. Rukmani
THROW-BALL	...	Mrs. Vijayalakshmi

Early August 1956 saw the beginning of our sporting activities, just after the P.W.D. had spread out our sports fields. Facilities were provided for all games for both men and women. The government sanctioned Rs. 7000/- for the purchase of materials and as such our college is one of the best equipped institutions in sports in Madurai to-day.

Our teams entered the fields in nearly all the games conducted by the Madras University in the inter-collegiate tournaments. I can, with all pride, state that we did well for a college which has just been weaned.

Our cricket team did extremely well this year. On the whole we played eight matches, won five, drew two, and lost one. We entered the finals of the Madurai zone in the inter-collegiate tournament.

Our Hockey and Volley-ball teams also fared well. Though we had strong teams in foot-ball and basket-ball, we were unfortunate to have lost in the very first round but not before we gave our opponents a close fight.

Badminton is a very popular game in our college and we have some very good players amongst us. In the Sivaswamy Memorial All-India Tournaments, held in Madurai, we came up to the quarter-finals.

Our Tennis-club was innagurated in the last week of August '56 by Dr. T. Narayanaswamy our Assistant Principal. A good number of tennis enthusiasts had to be disappointed due to lack of accommodation in our two courts. We have some promising players in our club and

M. Bhaskaran our tennis captain was the runner-up in the Madurai zone.

Our lady students were also not behind the boys in the field of sports. They were runners up in Badminton in the Madurai Division. These fair folks brought a name to our college for, four of them were selected to represent our Division in the University sports held at Madras. This is the first batch of students to have represented our college in the University sports. Miss G. Rukmani kept our flag aloft by coming second in the Shot Put.

Our Intra-mural tournaments are looked forward to, always with anxiety and fervour by our students and their sprightly spirit can be seen in all its glory. Four houses were the basis on which the competitions were held. Many were the games included and this year mixed events were also introduced adding to the colour.

Blue House won the games shield.

For the second year in succession, our Annual Aquatic Championship was conducted in the Municipal Swimming Pool. In addition to the Back-stroke, Free-style and Relay, this year, Breast stroke was also included.

The aquatic shield went to Green House.

The Annual Sports meet was held on the 23rd February, '57. Dr. M.D. Ananthachari our principal took the salute at the March Past and declared the meet open. Miss N. Shakunthala, Principal, Fatima College, presided over the function. Later in the evening she distributed the prizes and addressed the gathering.

Many students took part and there was keen competition in every event. Till the last event of the day, the House championship was in the balance, whilst finally the Blue annexed the shield by a narrow margin of 6 points over their nearest rivals, the Red House.

Kuppuswamy of Blue House and Vatsala Kumari of Green House won the individual championship for men and women respectively.

The highlight of the day was the tug-of-war between the staff and the students in which the sheer weight of the staff prevailed. The day ended with the distribution of the prizes. The annual report was read by Mr. V. S. Austin and Mr. K. P. Mathew, Sports Secretary proposed the vote of thanks.

This report will not be complete if I do not thank the members of the association. Our Principal and the Vice-President did a lot and gave us valuable guidance. I thank all the other staff members, and finally my colleagues and friends, the students of this infant institution.

K. P. Mathew,
(Sports Secretary)

Rules

The Madurai Medical College Athletic Association is established to promote all sports and games for students of the Madurai Medical College, Madurai.

2. The Association shall be managed by a General Committee which shall be constituted as follows :—

President. (Principal will be the ex-officio President of the Association).

Vice-President

Honorary Treasurer.

Secretary from among the Staff (Physical Director).

Secretary from the Students.

Two Staff Members.

(Nine) Captains of Football, Hockey, Cricket, Tennis, Volleyball, Basketball, Badminton, Tennikoit & Track & Field Sports.

One Lady Representative for Women's Section.

Seven members shall form a quorum.

3. *Members* :

(a) Official Members :- All Professors, Lecturers, and Assistant Professors, Demonstrators and Assistant Lecturers on the staff of the College during the session are expected to become members of the Athletic Association, Professors and Lecturers paying a subscription of Rs. 15/- and Assistant Professors Rs. 10/- Demonstrators and Assistant Lecturers Rs. 5/.

(b) Ordinary Members :- All students of the College who should pay an annual subscription of Rs. 8/- each which is collected along with the other college fees.

4. The colours of the Association to be black with the College crest worked in white on the front of the Jersey. The College blazer shall be black flannel, wth the College Crest in white on the breast pocket. The county cap to be black with the College crest worked in white on the front.

5. The Association shall consist of the following sections : (1) Cricket (2) Football (3) Hockey (4) Tennis (5) Volley-ball (6) Basket-ball (7) Badminton (8) Tennikoit (9) Track & Field Sports. Other sections may be added from time to time by the General Committee.

6. The following shall be the members of the Executive Committee of the Association :

(1) President (2) Vice-President (3) Honorary Treasurer (4) Secretary (Physical Director) (5) Secretary (Men student) and (6) Representative (Women Student).

7. The Vice-President, Honorary Treasurer and Secretary (Staff) shall be appointed by the President. The Executive Committee of the Medical College Association shall appoint two Staff members to serve on the General Committee.

8. The Medical College Association shall elect the Secretary (Student). The Captains of the various sections (Viz., Cricket, Football, Hockey, Tennis, Volley-ball, Badminton, Tennikoit and Track and Field Sports) will be elected from among the panel of players selected by the Executive Committee to represent the College.

9. The General Committee shall, at the commencement of the College year, appoint a staff member to audit the accounts. It shall estimate the expenses for the current year and apportion grants as required and submit the balance sheet for the information of the Principal.

10. If the grant of any section is exceeded, the General Committee shall consider the special circumstances and shall have the power to vote an increased grant.

11. The General Committee shall appoint two members to serve on the Inter-collegiate Athletic Association.

12. Entrance for any tournament or competition shall be made by the Secretary (Physical Director) with the consent of the General Committee.

13. The General Committee shall have the power to fill up any temporary vacancy which may occur excepting those appointed by the President.

14. THE TENNIS CLUB :

Membership is open to all members of the Madurai Medical College Athletic Association on payment of a special subscription and subject to other rules and regulations framed by the General Committee as and when required.

Results of the Intra-mural Tournaments, Aquatics and Athletics

CRICKET

Winners — Red House

(1) C. S. Raveendran (Capt.)
(2) F. J. Rayen
(3) C. Rajan
(4) K. M. Rajendran
(5) Babu Brayant
(6) K. C. Nambiar
(7) Selvaraj
(8) Abdul Hamid Khan
(9) Chandrasekaran
(10) S. Jamal
(11) Chandramohan

FOOT-BALL

Winners — Green House

(1) Kandaswamy (Capt.)
(2) K. P. Mathew
(3) K. C. Madhavan
(4) Sudersanam
(5) Pandian
(6) Kalimuthu
(7) Venkatasubramaniam
(8) Ramachandra Kurup
(9) Jeyaraman
(10) Veerabhadran
(11) Ramaswamy

HOCKEY

Winners — Yellow House

(1) M. P. S. Menon (Capt.)
(2) Sembon David
(3) Mahendran
(4) Abdul Hayoom
(5) Ian Sundararaj
(6) Chandragraham
(7) Kichley
(8) V. P. Bhaskaran
(9) Venkataraman
(10) Vedanayagam
(11) Rajamanickam

BASKET-BALL

Winners — Green House

(1) S. Rajendran (Capt.)
(2) Pandian
(3) Sudersanam
(4) Thangavelu
(5) Shanmugavelu
(6) K. C. Madhavan
(7) K. Ramaswamy

VOLLEY-BALL

Winners — Yellow House

(1) Chandragraham (Capt.)
(2) Sembon David
(3) Pandi
(4) Venkitaraman
(5) Raghavan
(6) Mahendran

TENNIS

Singles :

Winner — M. Bhaskaran
Runner — K. P. Mathew

Doubles :

(1) M. Bhaskaran & Rengarajan
(2) F. J. Rayen & Raveendran

TABLE TENNIS

Singles :

(1) F. J. Rayen
(2) K. M. Rajendran

Doubles :

(1) F. J. Rayen & K. M. Rajendran
(2) C. Rajan & A. Hamid Khan

Mixed Doubles :

(1) F. J. Rayen & Lalitha
(2) Balan & Hemavathy

BADMINTON

Singles :

(1) Thangavelu
(2) Chandrabose

Doubles :
(1) K. P. Mathew & Thangavelu
(2) Devarajan & Pandian

Fives :
(1) Devarajan
(2) K. P. Mathew
(3) Thangavelu
(4) Pandian
(5) Sudersanam

Mixed Doubles :
(1) Sundaram & Padmavathy
(2) Chandrabose & Hemavathy

TENNIKOIT

Singles :
(1) Desingarajan
(2) Rajamanikam

Doubles :
(1) Chandragraham & Rajamanickam
(2) I. Soundararaj & A. Sunderaraj

Mixed Doubles :
(1) Desingarajan & Victoria Indrani
(2) Chandragraham & G. Rukmani

WOMEN

THROW-BALL

Winners — Yellow House
(1) Vijayalakshmi (Capt.)
(2) G. Rukmani
(3) Valli
(4) Bhagyalakshmi
(5) Gladys
(6) Sivakami

BADMINTON

Singles :
(1) Padmavathy
(2) G. Rukmani

Doubles :
(1) Padmavathy & Hemavathy
(2) Vatsala & V. Rukmani

TENNIKOIT

Singles :
(1) G. Rukmani
(2) Bhagyalakshmi

Doubles :
(1) G. Rukmani & Vijayalakshmi
(2) Bhagyalakshmi & Valli

CARROMS

Singles :
(1) Padmavathy
(2) Madona Antony

Doubles :
(1) Padmavathy & Madona Antony
(2) V. Rukmani & Shantha

TABLE TENNIS
(1) Shantha
(2) Gladys

RESULTS OF THE AQUATICS

Free Style
(1) K. P. Mathew
(2) Victor Jesudason

Back Stroke
(1) Radhakrishnan
(2) Ramaswamy

Breast Stroke
(1) Ramachandra Kurup
(2) Kuppuswamy

Free Style Relay
(1) K. P. Mathew
(2) Victor Jesudason
(3) Ramachandra Kurup
(4) Ramaswamy

RESULTS OF THE ATHLETICS

Events for men

(1) 100 *Metres Dash*
(1) Chandramohan
(2) Abdul Hamid Khan

(2) 200 *Metres Dash*
(1) Chandramohan
(2) Sembon David

(3) 400 *Metres Dash*
(1) P. Kuppuswamy
(2) Chandramohan

(4) 800 *Metres Race*
(1) Kuppuswamy
(2) P. R. Srinivasan

(5) 1500 *Metres Race*
(1) P. Kuppuswamy
(2) P. R. Srinivasan

(6) 110 *Metres Hurdles*
- (1) I. Soundararaj
- (2) F. J. Rayen

(7) *Javelin Throw*
- (1) K. P. Mathew
- (2) P. Kuppuswamy

(8) *High Jump*
- (1) C. S. Raveendran
- (2) Sembon David

(9) *Long Jump*
- (1) I. Soundararaj
- (2) Abdul Hamid Khan

(10) *Hop-Step & Jump*
- (1) Sam E. Rajiah
- (2) K. Pandi.

(11) *Pole-vault*
- (1) I. Soundararaj
- (2) Kalimuthu

(12) *Shot-Put*
- (1) K. S. Rajaguru
- (2) Muthuveermandy

(13) *Discus Throw*
- (1) Rengarajan
- (2) Kandaswamy

(14) *Hammer-Throw*
- (1) Kandaswamy
- (2) Rajaguru

Events for women

(1) 100 *Metres Dash*
- (1) Madona Antony
- (2) Ummu

(2) 80 *Metres Hurdles*
- (1) Karthiayini
- (2) Rajalakshmi

(3) *Long Jump*
- (1) Vatsala Kumari
- (2) Karthiayini

(4) *Slow Cycle Race*
- (1) Usha Rani
- (2) Victoria Indirani

(5) *Shot Put*
- (1) G. Rukmani
- (2) Bhagyalakshmi

(6) *Discus Throw*
- (1) Vatsala Kumari
- (2) Padmavathi

(7) *Javelin Throw*
- (1) Vatsala Kumari
- (2) Bhagyalakshmi

(8) 75 *Metres Skipping*
- (1) Ummu
- (2) Rajalakshmi

(9) *Obstacle Race*
- (1) Ummu
- (2) G. Rukmani

Key for Clinical Quiz

1. Adamantinoma
2. Hydrocephalus
3. Retrobulbar Sarcoma
4. Osteochondroma
5. Cavernous Haemangioma
6. Plexiform Neurofibroma
7. Lipomatosis
8. Hydatid Jaw
9. Meningocele
10. Fibrosarcoma
11. Papillary Cystadenoma

Photos — Courtesy : Mr. S. Jamal

We Welcome :

1. Dr. V. N. Ambalavanan, M.B.B.S., T.D.D., — *Lecturer in Tuberculosis.*
2. Dr. A. S. Annamalai, M.B.B.S., — *Hony. Lecturer in Orthopaedics.*
3. Dr. (Kum) P. Bangaru, M.B.B.S., — *Tutor in Midwifery.*
4. Dr. K. Bhaskar Rao, M.D., — *Lecturer in Midwifery.*
5. Dr. S. Gandhamadhanan, B.SC., M.B.B.S., — *Tutor in Forensic Medicine.*
6. Dr. C. B. Gopalakrishnan, M.B.B.S., — *Lecturer in Forensic Medicine.*
7. Dr. P. Govinda Rau, M.B.B.S., F.R.C.S., (Eng.) — *Hony. Lecturer in Principles and Practice of Surgery.*
8. Dr. T. M. Ibrahim, M.B.B.S., — *Lecturer in Anaesthesia.*
9. Dr. A. K. Joseph, M.D., — *Lecturer in Midwifery.*
10. Dr. T. Kadirvel Raj, M.B.B.S., — *Tutor in Biochemistry.*
11. Dr. K. A. Krishnamurthi, B.SC., M.D., M.R.C.P. (Edin.) — *Asst. Professor of Medicine.*
12. Sri S. Krishnamurthi, B.SC., — *Demonstrator in Biochemistry.*
13. Dr. D. Lakshmanan, M.B., M.S., — *Assistant Professor of Surgery.*
14. Dr. Madhav Gadiyar, M.B.B.S., — *Tutor in Anaesthesia.*
15. Dr. P. Manickam, M.B.B.S., B.S.SC., — *Asst. Professor of Social and Preventive Medicine.*
16. Dr. T. D. Mohannathan, M.B.B.S., — *Tutor in Pathology.*
17. Dr. Md. Ubeidullah, M.B., M.S., — *Lecturer in E.N.T. Diseases.*
18. Dr. R. G. Murugesan, M.B.B.S., — *Tutor in Pathology.*
19. Dr. N. M. Muthiah, M.B.B.S., — *Demonstrator in Physiology.*
20. Sri R. Muthusubramaniam, B.SC., — *Demonstrator in Biochemistry.*
21. Dr. R. S. Rajagopalan, M.D., D.T.M., — *Assistant Professor of Medicine.*

22. Dr. S. Ramasami, M.B.B.S., — *Demonstrator in Pathology.*

23. Dr. D. V. Ramamurti, M.B.B.S., Ph.D., (Mac. Gill) — *Lecturer in Bacteriology.*

24. Dr. M. C. Rama Iyengar, B.A., M.B.B.S., D.V., — *Lecturer in Venereology.*

25. Dr. C. Raman, M.B.B.S., — *Tutor in Anaesthesia.*

26. Dr. K. Ramachandran, B.Sc., B.D.S., — *Lecturer in Dental Surgery.*

27. Dr. V. S. Ramachandran, M.B.B.S., — *Tutor in Mental Diseases.*

28. Dr. (Smt.) Sarojini Santhosham, M.B.B.S., — *Tutor in Anaesthesia.*

29. Dr. H. D. Singh, M.B.B.S., M.Sc., — *Lecturer in Physiology.*

30. Dr. V. Sivarajan, M.B.B.S., — *Tutor in Bacteriology.*

31. Dr. V. Srinivasan, M.B.B.S., M.R.C.P., (Edin.) — *Hony. Lecturer in Therapeutics.*

32. Sri L. R. Subbaraman, B.Sc., — *Demonstrator in Biochemistry.*

33. Dr. (Smt.) Suraiya Begum, M.B.B.S., — *Tutor in Biochemistry.*

34. Dr. (Smt.) Thankammal, M.B.B.S., D.G.O., — *Tutor in Midwifery.*

35. Sri K. Valmikinathan, M.Sc., — *Non-Medical Assistant in Biochemistry.*

36. Dr. S. P. Vedachalam, M.D., — *Professor of Pathology.*

37. Dr. Victor Gnanadhikham, M.D. — *Additional Professor of Medicine.*

For the want of a nail the shoe was lost
For the want of a shoe the horse was lost,
For the want of a horse the rider was lost,
For the want of a rider the battle was lost,
For the want of a battle the kingdom was lost —
And all for the want of a horse shoe-nail.

— *Franklin*

* * * *

Not only our future economic soundness but the very soundness of our democratic institutions depends on the determination of our Government to give employment to idle men.

— *Franklin D. Roosevelt*

We bid farewell to :

1. Dr. V. C. Anguli, B.SC., M.D., — *Lecturer in Pathology.*
2. Dr. K. Arumugam, M.B.B.S., — *Tutor in Pathology.*
3. Dr. K. Bhaskar Rao, M.D., — *Lecturer in Midwifery.*
4. Dr. K. Jagannathan, M.B.B.S., D.T.M., — *Tutor in Medicine.*
5. Dr. P. Natarajan, M.B.B.S., — *Tutor in Pathology.*
6. Dr. N. Padmanabhan, M.B.B.S., M.SC., — *Lecturer in Physiology.*
7. Dr. N. G. Pandalai, M.D., F.R.C.P., D.T.M., — *Professor of Bacteriology.*
8. Dr. K. Ramachandra, M.D., — *Lecturer in Medicine*
9. Dr. (Smt.) Ramani Sivaraman, M.B.B.S., D.G.O.,—*Tutor in Midwifery.*
10. Dr. S. Ramasami, M.B.B.S., — *Demonstrator in Pathology.*
11. Dr. K. Sivarama Mudaliar, B.SC., M.B.B.S., — *Tutor in Bacteriology.*
12. Dr. S. T. Sundararaj, M.B.B.S., — *Tutor in Physiology.*
13. Dr. M. I. Varkki, M.B.B.S., B.S.SC., D.G.O., D.P.H. (Lond.) — *Asst. Professor of Social and Preventive Medicine.*

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