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SOUVENIR 1957

THE MADURA MEDICAL ASSOCIATION

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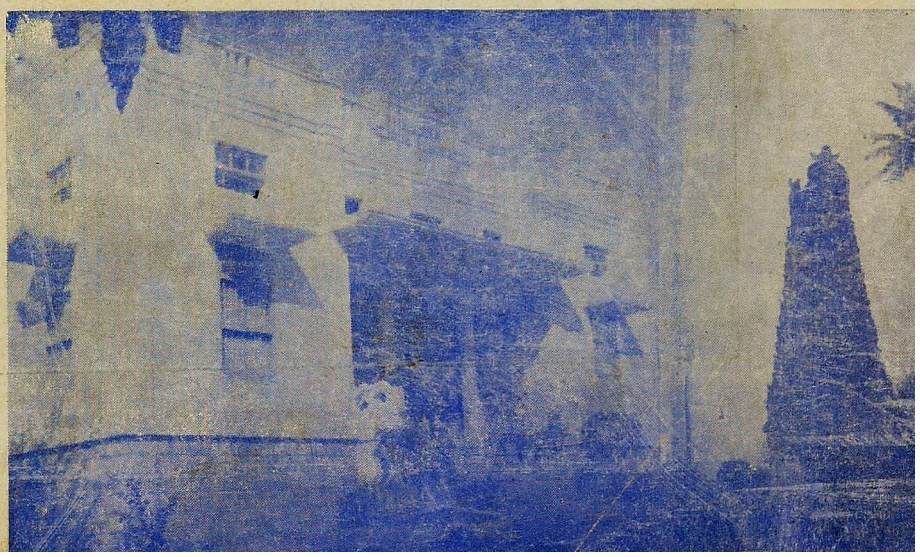

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The Madura

Medical Association Souvenir

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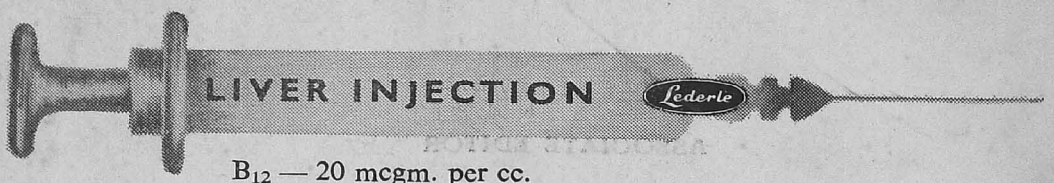

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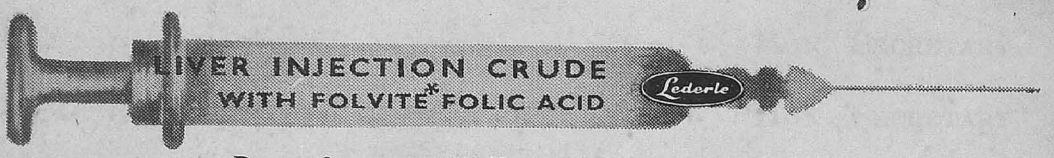

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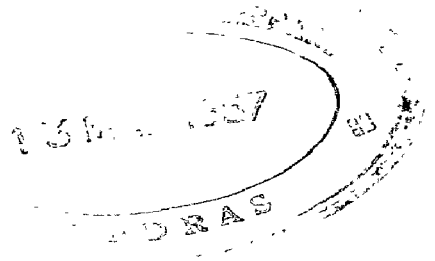

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THE SOUVENIR

THE year that has just rolled on, has been made most eventful by the enthusiastic secretary Dr. K. A. Ramalingam who has evinced a keen interest throughout the year.

I am thankful to the Governing Body of the Association for giving me the opportunity of publishing this year's Souvenir. This publication has become a regular feature at the time of our Annual Meeting and needless to say, a popular and useful enterprise. I have been greatly encouraged by the readiness with which the appeal for articles was responded to by the members of the Association.

I must specially thank Dr. A. Ananthanarayana Iyer B.A., M.B.B.S., M.Sc., F.A.Sc., & Dr.M. Thangavelu, M.D. for their special contribution of articles to this souvenir; similarly I thank the various other authors and advertisers for co-operating and making this souvenir a great success.

Lastly I thank Messrs. De Nobili Press for their unfailing courtesy and ready co-operation and the promptness with which they undertook and published this souvenir, in a most attractive manner bestowing their care and skill.

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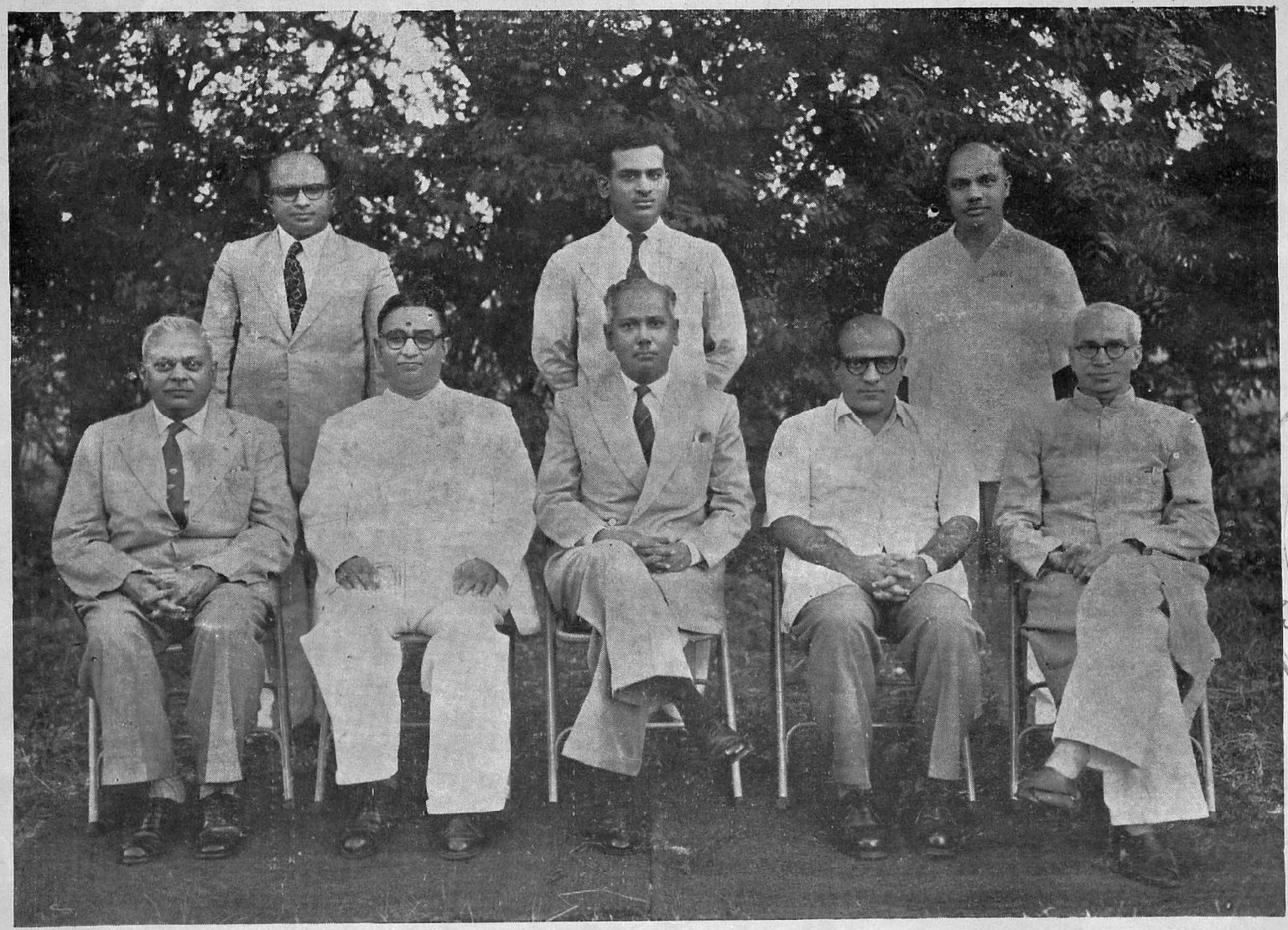

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The Surgery of Peptic Ulcer

P. VADAMALAYAN, M.B.B.S.,
Honorary Surgeon, Erskine Hospital, Maduari

HAVING had the opportunity of working for nearly a quarter of a century in a large Surgical centre like the Erskine Hospital where a considerable portion of major surgery done centres round surgery of the stomach, I am tempted to write about the Surgery of the Peptic Ulcer.

I have a record of 643 cases of Gastroenterostomies and 325 cases of Gastrectomy done by me during the period 1939 to 1956. This will be very roughly one-fifth of the cases done in the Hospital during this period.

HISTORY

I think Madura has attracted a large number of cases of Peptic Ulcer for surgery because surgeons like the late Col. Harley was one of the first to do Gastroenterostomy as far back as 1920. Dr. H. Sommervielle, the Missionary Surgeon farther south in Travancore was a pioneer who operated on a large number of Peptic Ulcer cases. The first gastrojejunostomy was done in the Madras State by Col. Niblock about the year 1915. He had done six cases before the year 1920 for Duodenal obstruction. Col. Symmons and later Col. Bradfield had visited the Mayo Clinic and introduced Gastroenterostomy in the Government General Hospital, Madras on a large scale after 1920.

A first gastrectomy was performed for carcinoma of the stomach about seventy years ago by Pean, a French surgeon. Gastrectomy was done only for carcinoma first and in the teens and twenties of this century gastroenterostomy was the operation for Peptic Ulcer, though Finsterer of Vienna had started doing Gastrectomy before 1915. Gastrectomy for Peptic Ulcer as a recognised operation is only 20 years old. It is interesting to know that the surgical section of the Royal Society of England as late as November 1944 had decided that, with certain exceptions, Gastroenterostomy was still the surgical operation of choice for Duodenal ulcer. Gastrectomy, rather gastro-duodenectomy began to be practised in England on a large scale from 1930 for Duodenal Ulcers. A few years later, Gastrectomy was started in the Madras Government General Hospital for Duodenal Ulcer. Till 1937 only gastroenterostomy was done in our Hospital for Peptic Ulcer. Gastrectomy was practised in increasing numbers for Duodenal Ulcer in our hospital from 1938. I remember in the early days of my surgical career Gastroenterostomy being done for Cancer Stomach and Gastric Ulcer with impunity for cases which at present we will not think of anything but a proper Gastrectomy.

INCIDENCE AND ETIOLOGY

Five to seven per cent. of hospital admission is said to be due to Peptic Ulcer all the world over, and this seems to be the case in this part of the country also. Ten per cent. of the population of America is said to have Peptic Ulcer at some period

of their lives. The incidence would seem to be somewhat less in England but England also would seem to be rapidly emulating that figure. Though we are not so unfortunate, at least 5 per cent. of the South Indian population seems to have Peptic Ulcer at some period of their lives. Roughly, we have 5 per cent of the admission to our hospital for Peptic Ulcer. The prevalence of Peptic Ulcer in South India as compared with North India is striking. According to McCarrison it is 58 times more common in South India than in North India. I was surprised in my last tour of the United States and United Kingdom to find in any big surgical centre, the daily lists of operation for Peptic Ulcer figure so highly even as we have in this part of the world. But the paucity of the Gall-Bladder surgery here as compared with those countries and the fairly high incidence of gall-bladder disease in North India set me thinking about the etiology of Peptic Ulcer. The similarity in incidence in gall-bladder disease in western countries and North India may be due to the diet. But the Duodenal Ulcer which seems to be so common here in the poor Madras peasants who do not smoke and who do not worry as in the case of the western countries must have something common in etiology. Certainly the etiology of the Peptic Ulceration is still wrapped in mystery.

"All the best people have Peptic Ulcers. It is not the prerogative of the well-to-do. There is no doubt that ulceration favours a certain age group and economically it is of vast importance that that age group should be the wage earning — producing one. These sufferers are, on the whole, the ablest, the most hard-working and the most conscientious members of the community. Their lives are made miserable and their working efficiency is diminished by pain and indigestion and they come to us for relief."

"Peptic Ulceration is disabling but not necessarily fatal and it is therefore our duty to advise that treatment most safe, simple and satisfactory. Medical treatment which has, or rather which admits of no mortality is therefore the first choice. It is still the first choice. But since the end of the War there have been a notable swing of opinion towards surgery, a tendency to resort to it more often and earlier. The reason for this is two-fold — that medical treatment though simple and safe has not proved particularly satisfactory and that surgical treatment though less simple than formerly is very much safer and more satisfactory than it used to be."

I shall here like to give an account of the cases done by me in the Hospital and outside :

GASTROENTEROSTOMY

Cases done in Govt. Erskine Hospital

From 1939 to 1947.

	Cases done.	*Mortality.*
MEN	402	12
WOMEN	72	1
Total	474	13

Percentage of Mortality: 2.8 per cent.

From 1948 to 1956

	Cases done.	*Mortality.*
MEN	129	3
WOMEN	28	-
Total	157	3

Percentage of Mortality: 1.9 per cent.

CASES DONE IN THE NURSING HOME

MEN	11
WOMEN	1
Total	12

Mortality : Nil.

Grand Total of Gastroenterostomy done : 643 cases.

DETAILS OF GASTRECTOMY DONE WITH RESULTS.

Cases done in the Government Erskine Hospital

From 1939 to 1947

	Duodenal ulcer.	*Gastric ulcer*	*Anastomotic ulcer.*	*Cancer stomach.*	*Total*
MEN	56	56	6	2	120
WOMEN	7	20	–	1	28
Total	63	76	6	3	148

Total Cases : 148

Mortality : 11

Percentage of Mortality : 7.4 per cent.

From 1948 to 1956

	Duodenal ulcer.	*Gastric ulcer*	*Anastomotic ulcer.*	*Cancer stomach.*	*Total*
MEN	78	39	9	10	136
WOMEN	12	16	2	5	35
Total	90	55	11	15	171

Total Cases : 171

Mortality : 5

Percentage of Mortality : 2.9 per cent.

CASES DONE IN THE NURSING HOME

Duodenal Ulcer.	*Gastric Ulcer.*	*Cancer Stomach.*	*Total*
2	2	2	6

Grand Total of Gastrectomies : 325 cases.

In the first nine years, the Gastro-enterostomies were thrice the number in the second half. The mortality in the first half was 2.8 per cent. which is fairly high and is due to nine cases of cancer stomach included in the list and also due to the fact that blood transfusion and antibiotics were not in use in the hospital during this period. In the second half, the mortality is less than 2 per cent. About the gastrectomy in the first half, the mortality of 7.4 per cent. is a high percentage and it is accounted for by the very few transfusions given at that time and absence of exhibition of antibiotics. In the second half, the mortality was 2.9 per cent. which is also somewhat high. This will account for by a number of cancer stomach cases. There was one case of death in cancer stomach which was for a total gastrectomy for partial leather bottle stomach at cardiac end. If we exclude this, in the selected cases of gastric and duodenal ulcers, the mortality will be less than 2 per cent., which result will favourably compare with the results published.

It will be interesting to note that in this series in the last 75 cases of Gastrectomies done by me in the Erskine Hospital, there were no deaths — though there was one death due to vicious vomiting in a case of gastroenterostomy.

THE INVESTIGATIONS DONE BEFORE UNDERTAKING THE OPERATIONS

I. Careful clinical history — Periodicity and location of pain — vomiting — visible gastric peristalsis — Hematemesis and Malena — occult blood in stools.

II. Gastric analysis :— Fallacies — low acids in stomal ulcers by the neutralisation of bile by a Ryles Tube passed beyond the stoma — low acid also may be due to Gastritis.

III. Barium Meal X-Ray with screening.

IV. Gastroscopy :— Not practised in Erskine Hospital but extensively used in big Gastroentrological clinics.

VARIETY OF OPERATIVE PROCEDURE

I shall mention only the three varieties of operative procedures adopted in the treatment of Peptic Ulcers :

1. Various short-circuiting operations.
2. Vagotomy alone or combined with other operations.
3. Partial Gastrectomy.

Operations as Vaso-ligation as advocated by Dr. Sommervielle and neutralisation of acid chyme by bile as advocated by Aylett by anastomosing resected proximal loop of jejunum to stomach in different places require only to be mentioned as they are not extensively practised. I have done a few Vaso-ligation combined with Gastro-enterostomy in the beginning of my career but I cannot assess their results. I presume they all had the same fate as Gastroenterostomy failures. As regards the Aylett operation, I have no experience but I hear a colleague of mine who did about eight cases in the Erskine Hospital has given it up as he thinks that if the gastroenterostomy gave rise to one stomal ulcer, Aylett operation gave rise to two. Personally, I think it is nothing more than a gastroenterostomy and so it stands condemned for the same reason as gastroenterostomy. I had a personal conversation with that high ranking gastric surgeon, Mr. Norman C. Turner of St.

James Hospital, London who would not have a good word to say about the operation.

First and foremost comes the time-honoured Gastroenterostomy operation. As I told you before, Gastroenterostomy adopted as an operative procedure for Peptic Ulcer is nearly 40 years old. Twenty years ago, it was the accepted treatment for Peptic Ulceration. Many thousands of patients obtained permanent relief from it, but many series of figures in other countries have shown that on this scale this will have 25 per cent. failure rate ; obviously too high to balance its slightly lower mortality rate and procedures which succeeded it. Ideally it is an operation suited for Duodenal obstruction, result of healed duodenal ulcer. In this country, where one is accustomed to see mammoth ovarian tumours and elephantoid tumours often weighing more than the patient, one will see long-neglected cases of Duodenal obstruction where the whole of abdomen is filled by the dilated hypertrophied stomach having a 48 hour or 72 hour barium retention. These patients are dehydrated and hypo-proteinaemic and they do very well with a short-circuiting operation as a Posterior Gastro-Jejunostomy with a little blood or plasma transfusion just to get over the operative shock. It has been the experience of many of us that some of the patients showed so much improvement that we went on doing more and more Gastroenterostomies. It was common knowledge that young surgeons anxious for gastric surgery imagine a patient has got pyloric obstruction by giving a bowl of water and demonstrating V. G. P. and submit him to gastroenterostomy. In some cases, we used to rely on a low acid in the stomach after a F. T. M. and hope the Gastroenterostomy will be ideal for the patient. But in young patients with dilated stomach and chronic gastritis, the acidity certainly increases a few months or years after Posterior Gastro-Jejunostomy and one would be surprised to find that the patient joins the group of gastroenterostomy failures. In a General Hospital, it is a common knowledge that one meets daily with one to two cases of gastro-enterostomy failures. Medical treatment is so very unsatisfactory here because of economical conditions and want of hospital beds so that when I see a young patient with a duodenal ulcer without any definite indication for surgery as perforation or repeated haemorrhage, I put off the operative treatment and think of surgery, only when he returns three or four times with recurrence of symptoms for admission — Intractability. The number of Gastroenterostomy failures of my own cases have been considerable. I cannot give you exact figures for want of follow up, so that, in recent years, I have been practising more and more partial gastrectomy for Duodenal Ulcers.

VAGOTOMY

High acid in the stomach was thought to be the cause of the ulcer and so by cutting down the gastric secretion by Vagotomy, the ulcer is expected to heal. When it came first after Lester Dragstedt's animal experiments, it was extensively practised because of simplicity and low mortality. Vagotomy tide, has, on most surgical shores, receded, because of its "inconsistent, variable and in most cases unpredictable" results and also because of its numerous and not infrequent complications as (1) gastric distension (2) vomiting (3) loss of weight (4) Dysphagia (5) Paralytic ileus (6) Diarrhoea (7) recurrence or persistence of duodenal ulcer in 10 per cent. of the cases (8) gastric ulcer may develop on a duodenal ulcer after Vagotomy and (9) Diaphragmatic

Hernia. I have done it in ten cases including three cases for Stomal Ulcer with total failure in anastomotic ulcer cases. Two of the anastomotic ulcer cases after Vagotomy I had to do partial gastrectomy.

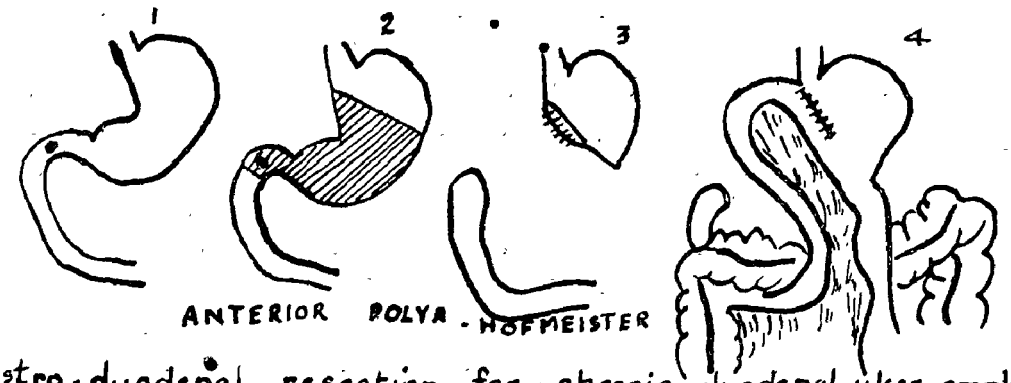
PARTIAL GASTRECTOMY

Gastrectomy is at present the treatment of choice for the treatment of peptic ulceration whether gastric or duodenal. "It is based on sounder reasoning and can show far better results than any alternative procedures, and will therefore remain until something that can be shown to be better comes to replace it. Gastrectomy, properly planned and executed, lowers the acid secretion to a safe level without abolishing it and restricts that secretion to the time that the meal is in the stomach. It thus retains gastric secretion but abolishes the risk of reulceration."

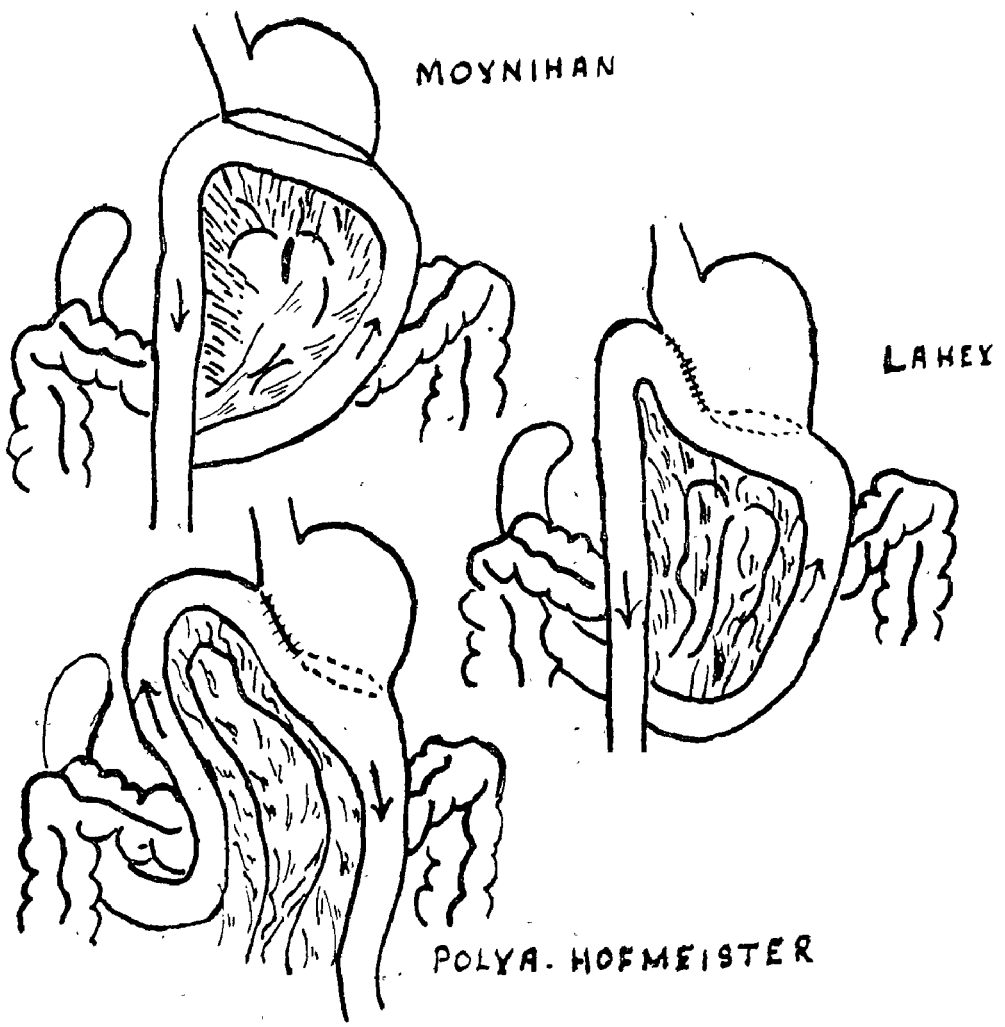
I shall now describe the technique of Gastrectomy which I am practising. There are two main types of Gastrectomy — Billroth I and Polya type of Gastrectomy. I have done only two Billroth I type of operation though I have seen it to be increasingly practised in Western countries. (Diagram page). There are three main types of antecolic Polya operations — one practised by Moynihan, second by Lahey and third known as Polya-Hofmeister. I am practising the Polya-Hofmeister antecolic anastomosis in almost all cases. In some cases where the meso-colon is long and lax, I do a Finisterer Polya type of retro-colic anastomosis.

The patients are prepared for operation with careful attention to replacement of water and electrolytes and of protein by frequent administration of fluid meal with amino-acids if these are required and to clearance of distended stomach by a few days of gastric lavage. A high right paramedian incision is employed, the abdomen opened and upper abdominal organs are palpated for verification of the diagnosis. The great omentum is then divided. The gastro-colic ligament is then picked up well to the left of middle line and then divided between ligatures from there to the duodenum. The right gastro-epiploic artery is divided where it emerges below the pylorus. The pylorus and duodenum are then gently elevated forward from the pancreas by gauze dissection as far as the line of the gastro-duodenal artery. Attention is now turned to the lesser omentum. An opening is made in this above the pylorus and a strip of gauze is passed round the stomach and clamped. This is used for traction on the stomach when the duodenum is dissected from the head of the pancreas at least an inch and a half from the pylorus or beyond the duodenal ulcer after ligature of right gastric vessels. This part of the operation is easy in case of gastric ulcer or carcinoma of stomach and exceedingly difficult if there is a large penetrating posterior wall ulcer. If it can be effected, the duodenum is divided beyond the level of the ulcer and the duodenal stump is closed. The duodenum is crushed by a small Payers clamp beyond the ulcer and sectioned. The stump is closed by continuous catgut suture and is invaginated by a secondary sero-muscular suture of catgut. I know most surgeons are using interrupted silk for the sero-muscular stitch but in all my cases I used only catgut for the two layers. A third layer of continuous stitch which brings the peritoneum over the duodenum and pancreas completes the closure. Particular attention is paid to the closure to avoid a burst stump. Sulphanilamide powder is dusted over this area. If penetration of posterior wall ulcer makes difficult the

THE MANAGEMENT OF ABDOMINAL OPERATIONS.



Gastro-duodenal resection for chronic duodenal ulcer employing the ante. colic Polya-Hofmeister technique.



Three popular types of Ante. Colic Polya operations

separation of duodenum from pancreas, the duodenum may be circumcised around the ulcer to leave the ulcer clear and divided beyond the right edge of the ulcer or the ulcer may conveniently be left and the duodenum divided between the left edge of the ulcer and the pylorus. If the ulcer is left, the anterior edge of the duodenum may be sutured to the right edge of the ulcer and the ulcer extruded as it were from the duodenal stump, the sero-muscular coat of the anterior wall being doubled over the ulcer. In any event, the entire mucosa of the antrum must be removed ; if it remains, a Jejunal ulcer is almost inevitable. In case of extreme difficulty, Bencroft manouvre has been suggested which consists in sectioning the pyloric end of the stomach and removing the mucosa of the antrum as far as the pyloric ring and enclosing the muscular walls of the stump of the antrum over it. I have never adopted this procedure. Tanner suggests a beginner in case of difficulty may leave the antrum and remove it later as a second stage. In the rare ulcer of the second part of the duodenum, no attempt is made to remove the ulcer. It has been advised that if the closure of duodenal stump offers particular difficulty, a tube may be inserted in it and the stump closed around it by purse string suture, the tube being brought to form a temporary fistula. I have never adopted this technique.

Attention is now turned to the curvatures. The division of the gastrocolic ligament which was previously effected to a point just to the left of the middle line is carried round the greater curvature with ligation of the main gastroepiploic vessels, and is carried up the gastrosplenic ligament to the level of the upper pole of the spleen. The stomach is now pulled over to the left, if there is any gastric ulcer penetrating the gastrohepatic omentum, the stomach is torn from the ulcer leaving the base behind. With further traction of the stomach, the left gastric artery is put on the stretch and ligated high and divided. This releases the stomach considerably so that the stomach can be clamped high. Roughly, three-fourths of the stomach is to be removed for duodenal ulcers and two-thirds for the gastric ulcer. A loop of jejunum about eight inches from its commencement is chosen to be anastomosed to the stomach in the iso-peristaltic fashion (the proximal loop to the lesser curvature). This is clamped and the small bowel sutured to the stomach distal to the clamp by a continuous OO chromic catgut by an atraumatic needle. Stomach is sectioned three-fourths to one inch distal to this and a Hofmeister valve is provided in the stomach by giving a spur and the anastomosis is completed as usual. I have been always using catgut for both the layers and had no occasion for leak in the anastomosis. I have seen most surgeons in the West using interrupted silk for the peritoneal coat. Abdomen is closed with or without drainage. I saw a number of surgeons leaving a Cigarette drain over the stump. This might perhaps help a burst stump or a leak. It did help in a recent case done by a colleague. I have had only one case of burst stump in one of my early cases and no case of haemorrhage which ended in fatality or which required any special treatment.

ANAESTHESIA

Most of the cases done in the Erskine Hospital in the first half are by light percaine spinal anaesthesia — giving 11 to 13 c. c. of light percaine supplemented by local 1 per cent. Novocaine for closing abdomen in some cases. Of late, heavy percaine 1.8 c.c. combined with half a

grain of Ephedrine in 1 c.c. is used for the spinal injection in the second space. This seems to give perfect relaxation and anaesthesia lasting for 3 to 4 hours — only very rarely does one require to supplement with local Novocaine. A few cases of pentothal sodium in saline drip and muscle relaxants have been used, but because of want of more anaesthetists, we have been successfully carrying on with the spinal anaesthesia. This may sound strange to those who are accustomed to the present day luxuries in general anaesthesia — as intratracheal gas and oxygen and muscle relaxants.

To sum up, partial Gastrectomy is the operation of choice for Gastric and Duodenal Ulcers. In gastric ulcers, partial gastrectomy is to be advocated early, because of the excellent after-results. The indications for operation in duodenal ulcers are less clear-cut. The absolute indications will include intractability repeated bleeding in young patients (or a single massive haemorrhage in persons after 50) perforation and stenosis. Gastro-enterostomy has largely been discredited as a method of treatment for Peptic Ulceration because of the incidence of jejunal ulceration. Even if it is performed in a patient who has pyloric stenosis and a low acidity, hyperacidity often develops after the stomach is drained. It is probably the treatment of choice, for example, if pyloric stenosis is present in a patient over 50 who has no active ulcer, no pain, no haemorrhage and a very low acid ; in very aged and feeble patients with pyloric stenosis or in young patients with pyloric stenosis who are debilitated or who have concomitant disease, cardiac, pulmonary or renal.

I thank Dr. C. K. Padmanabha Menon, M.S., F.R.C.S., Superintendent, Erskine Hospital, Madurai for allowing me to have access to the hospital records.

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THOUGH treatment of Urinary Calculi is usually considered a surgical problem, medical management becomes of importance when surgery is inadvisable or in prevention of recurrence.

Proper medical treatment will help in spontaneous expulsion of small stones (Stones less than 0.5 cm. in diameter) in many cases, and also prevent them getting larger. Recurrence of stones after operations for removal is nearly 40% when the necessary measures are not employed, but can be brought down to 4.7% as claimed by Higgins, if the patient is placed on correct treatment. Phosphaturia and oxaluria, two conditions which annoying by themselves also predispose to Calculi are amenable to medical treatment.

The main causes of calculi are infection, vitamin deficiency disturbances of metabolism and stasis.

Infection plays a leading role, in the formation of many stones. Stones form frequently and often quite rapidly in kidneys infected with Proteus bacilli and some strains of Staphylococci. These organisms possess the power of splitting urea in the urine into ammonia and carbon dioxide with the resultant formation of Am. carbonate. This combines with the magnesium salts and phosphates to form Ammonium Magnesium Phosphate which is insoluble, and by rendering the reaction of the urine alkaline results in the precipitation of Calcium Phosphate. Many of the large stag horn calculi found in kidneys and encrustations found in the bladder are formed in this way. One should remember however that infection is not a prerequisite for the formation of stones.

Vitamin deficiency

Vitamin A has a specific effect upon epithelial structures and an adequate amount either in food or in concentrated form helps to keep the mucous membrane of the urinary tract in healthy condition. Calcium Phosphate stones are readily formed in animals deprived of vitamin A.

Vitamin B has been advised in the prevention of oxalate stones as intestinal putrefaction which produces oxalic acid is said to be diminished by administration of vitamin B. Complex.

Metabolic Disorders

Metabolic disturbances often cause an excessive secretion of crystals in the urine which become a factor in the formation of calculi. Cystinuria, a condition in which cystine is exerted in the urine is due to an inborn error of metabolism and probably is caused by failure of the renal tubules to reabsorb cystine and some other amino-acids. Renal calculi occur in about 2.5 % of cases of cystinuria.

Uric acid and urate crystals are often excreted in the urine of sufferers from gout. Persons who eat large quantities of protein food and lead sedentary lives are believed to be subject to uric acid stones.

Altered gastro intestinal function can produce phosphaturia. Calcium Phosphate stones occur in patients long bedridden. Calcium and phosphate-ions are also excreted in excess in hyperparathyroidism and the formation of stones occurs in a large proportions of these cases.

Stasis and Recumbency

Stasis, as the result of obstruction anywhere in the urinary tract, is often associated with calculi. It also renders the field suitable for bacterial growth and may be instrumental in making the urine alkaline.

Immobilization as in the treatment of Pott's disease and other similar condition acts in two ways to influence the production of urinary calculi, (1) by impairing renal drainage and (2) by producing changes in calcium metabolism.

The types of stones are mainly three.

(1) Organic, such as uric acid, cystine and xanthine stones, which are mainly due to metabolic disorders and constitute not more than 10% of all urinary calculi. These stones are found in acid urine.

(2) Calcium oxalate stones. Prien of Boston considers that most calcium oxalate stones have a nucleus of Calcium phosphate, which latter is soluble in acid urine. Ordinarily ca oxalate calculi may be found in alkaline as well as in acid urine.

(3) Calcium phosphate and Magnesium ammonium phosphate stones which are often found together and the rare calcium carbonate stones. These three are generally associated with infection and the urine reaction is always alkaline.

Calcium containing calculi constitute nearly 90% of all urinary calculi and if Prien's observation that even Calcium oxalate stones have a nucleus of calcium phosphate is correct then the main problem of the treatment of all calcium stones is the treatment of the Calcium Phosphate factor. Before undertaking medical management of urinary calculi it is necessary to find out the nature of the calculus. If a stone has been passed and is available a proper chemical examination should be done to find its composition. In other cases enough information can be gained about the nature of the calculus from the reaction of the urine and from microscopical examination of the crystals in the urinary deposit. A plain skiagram of the urinary tract will also help us to determine the type of stone in a fair number of cases. The metabolic stones is the cysteine and the urate calculi show a homogeneous density and are fairly translucent, the cart wheel pattern is characteristic of the oxalate calculus and the laminated appearance is diagnostic of the phosphate stones.

Treatment

It must be remembered, that surgical treatment is always necessary for any calculus which is not likely to be expelled spontaneously, and obstructions in the urinary tract require appropriate surgical measures in association with the treatment of the calculus. Also when a parathyroid adenoma is likely to be the cause of frequent recurrences of stone, a painstaking search for and removal of a parathyroid adenoma is the only way to permanent cure. Prevention of Stones in immobilized patients requires that these people are moved from side to side frequently in addition to the various medical measures described below.

Medical Treatment.

Details of medical treatment are :

(1) ADMINISTRATION OF VITAMINS

Vitamin A in a dosage of 25,000 units daily is considered necessary in all cases of urinary calculi. Vitamin B. complex has to be given in cases of oxalate calculi to prevent intestinal putrefaction and excess formation of oxalic acid. It is necessary to keep in mind that vit. D in excess can itself produce hypercalciuria and be a cause of calculi and so this vit. D, in excess must be avoided.

A High fluid intake

Urine is a highly saturated solution of various substances and supersaturation and precipitation of a particular crystalloid cystine in the case of cystinuria, Ca Phosphate or oxalate in phosphaturia or oxaluria, is the first stage in the formation of urinary calculi. It is therefore very essential to prevent supersaturation by a high fluid intake sufficient to produce a urine output of at least 70 to 80 oz. in 24 hrs. To produce this volume of urine it is necessary that the patient not only drink freely during the day but that he should have an additional two glasses of water on retiring and a further two glasses of water at 2 A.M. Only in this way can supersaturation be avoided. This regime has been found sufficient not only to prevent formation of stones in cystinuria but also to initiate dissolution of stones which have already formed. As mentioned above, this regime is also necessary in the prevention and treatment of other types of calculi.

Diet

The Diet of a patient with urinary calculi can be so modified that the reaction of the urine he produces is acid or alkaline. An acid urine is able to keep a larger amount of Ca salts in solution and so prevents precipitation, and similarly in an alkaline urine cystine and uric acid crystalloids are held in solution.

The type of diet to produce an acid urine is called an acid ash diet i.e., the total acid ash in the diet exceeds the total basic or alkaline ash. Explaining this briefly, it may be mentioned that foods are called acid ash foods, neutral ash foods and alkaline ash foods according to the ultimate residue they leave after metabolism in the body. When a food leaves a residue or ash in which the acid forming elements as chlorine, Sulphur and Phosphorous predominate it is an acid ash food ; when the ash or residue of the food is one in which the basic elements as Sodium, Potassium, Magnesium and Calcium predominate, then it is an alkaline ash food. When the two elements acid and alkaline are equal, then it is a neutral ash food. The common acid ash foods are meat, fish, chicken, egg, rice and other cereals. Milk, vegetables and fruits are alkaline ash foods. Citrus fruits as oranges and limes, although they contain a weak acid are metabolised into carbonates in the body and hence are alkaline ash foods.

It is therefore necessary that a person with a phosphate stone be prescribed an acid ash diet, a diet which contains strictly limited quantities of milk, vegetables and fruits and a fairly high proportion of animal foods (excluding milk) and rice and other cereals. For the sufferer with cystine or uric acid calculus milk, vegetables and fruits can be prescribed freely to make and keep the urine alkaline. In South India, it is very difficult in most cases to prescribe and enjoin an acid ash diet and in such circumstances acidifying drugs should be administered to make the urine acid.

Other modifications in the diet of use, in the prevention of stones are the low

calcium diet and the low oxalate diet. The low calcium diet, useful in all cases of calcium containing calculi is one in which milk and milk products as cheese etc. are strictly limited, as milk is the main sources of calcium in diet. (Butter however can be used freely). The low oxalate diet, useful in oxaluria and in oxalate stones is a diet from which beetroot, chocolate, cocoa, rhubarb and spinach are excluded. Too much coffee and sweets should also be avoided as coffee contains fair amount of oxalates and sweets encourage fermentation and formation of oxalic acid.

Medication

Medication in the treatment of renal calculi has two important objects. One is the eradication of infection and the other is the prevention of supersaturation of the urine with stone forming substances.

(a) Eradication of Infection necessitates a bacteriological examination with the object of finding out the infecting organism, when infection is associated with stone formation.

Penicillin, Streptomycin, Sulphon amides as Urolucosil, Gantrisin, and sulphadimidine are the various drugs used to eliminate infection, the choice of the drug depending on the organism causing the infection. Urolucosil and sulphadimidine, especially the former can be given for long periods in small doses without toxic side effects.

(b) *Prevention of Supersaturation*

(1) Acidification and alkalinisation of urine, as necessary is carried out to prevent precipitation of crystals. In phosphate stones and phosphaturia, sodium acid phosphate 20 grains three times daily or ammonium chloride 10 grains TDS can be used to acidify the urine. Acid Hcl dilute 10 m Q.D.S. is also helpful. For cystine and uric acid stones adequate alkalinisation can be carried out with Sodium citrate 1 dram 4 times a day.

(2) Administration of Amphogel.

Amphogel in doses of 1 oz. 4 times a day orally has been recommended for the prevention of phosphate stones. Its action is to combine with phosphate ions in the gastrointestinal tract to form an insoluble aluminium phosphate, which is entirely excreted in the Stool, thus reducing the phosphate output in the urine. Aluminium Hydroxide with Magnesium trisilicate is considered to be more useful than Al. Hydroxide alone in cases of duodenal or gastric ulcer. Salicylamide was used in same dosage when it was found to be equally useful if not better. This therapy was found very effective in 17 patients who had a previous history of recurrent calcium containing calculi. They were observed for 12 - 18 months on this therapy and during all that period they did not produce any new stones. An unexpected and spectacular result of this therapy was the complete inhibition of encrustation on inlying catheters in the bladder, in cases where previously frequent obstruction of catheters by Ca and Magnesium phosphate deposition had taken place. Previous to this, therapy catheters had to be changed at one to two-week intervals because of obstruction by phosphate deposition.

This salicylate therapy offers an ideal treatment for the prevention of Ca Phosphate stones. As we have mentioned earlier that calcium oxalate calculi often have a nucleus of Calcium Phosphate, this treatment will be useful for almost all calcium containing calculi. In addition it was found that the effect of this therapy

is not less even in the presence of urea splitting infections and obstructions in the urinary tract.

(3) Oestrin administration and the use of Hyaluronidase. Oestrin administration augments the urinary citrate and this increase in citrate excretion helps to keep calcium salts in soln and prevent formation of renal calculi. Hyaluronidase parenterally increases protective colloids in the urine which is considered to be of help in keeping crystalloids in solution. Further work has shown that oestrin administration is not of great help and hyaluronidase can sometimes produce sensitization and greatly increase the formation and growth of calculi.

(4) Salicylate Therapy.

Very recently Prien has recommended acetyl salicylic acid or salicylamide to increase solubility of calcium phosphate in urine. These two drugs are excreted in the urine as complex glucuronides and solubility of Ca Phosphate in urine is increased by these complex glucuronides. Aspirin was used first in doses of 2 gm. (30 grains) daily in divided dosage but because of occasional ringing in the ears in some people, and its unsuitability.

Summary

Medical management of urinary calculi is of great importance. These calculi can be divided into calcium containing calculi which are nearly 90% of all the stones and the cysteine and uric acid calculi. The latter, cystine and uric acid stones can be treated by a high fluid intake and adequate alkalinisation of the urine. The calcium containing calculi of which the most important is calcium phosphate, require a proper regimen which involves the administration of vitamins, a high fluid intake, a proper low calcium, acid ash diet and the eradication of infection.

The latest help in the eradication of this common type of stone is the use of acetyl salicylic acid or salicylamide which may prove to be a highly effective method. A proper appreciation of the various factors involved in this problem will help greatly to ameliorate the sufferings of patients with these conditions who are at present given no advice or the wrong type of advice with sometimes very bad results.

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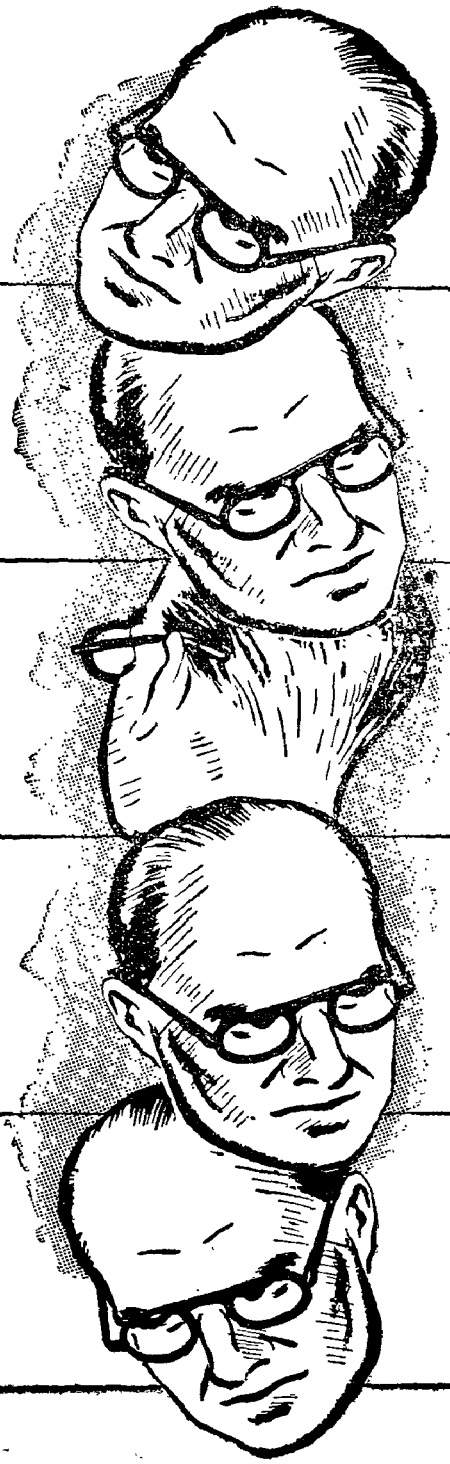

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Tablets, Elixir and Injection

"PHILIPS HOUSE", CALCUTTA-20,

Complications of High Blood Pressure

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MUCH has been said about the aetiology and pathogenesis of high blood pressure in recent years. Volumes have been written and no periodical or journal is exempt from an article on Hypertension, contributed by the most learned members of the profession interested in some aspect of this subject. At last, at present a status quo has been reached about the pathogenesis of Essential Hypertension and many of the workers in the field of vascular diseases have agreed that it is basically a psychogenic disease. It has been grouped under the psychosmatic conditions, a group of disorders so highly prevalent at the present day where the whole tempo of life is moving at an accelerated pace. Stress and strain is the keynote of modern civilisation that only the genetically strong and the hardy stand the vicissitudes of life without succumbing to its untoward effects. A life of continuous struggle punctuated with unhappy and depressing episodes contributes to this psychosmatic disorder and with each new generations of human beings the incidence of Hypertension is increasing and the age incidence of the disease is receding. Moreover, the type of hypertension and the progress of the disease have taken a new turn. The so-called malignant hypertension is becoming a fairly common condition at the present day. As a result of the economic hardships and the altered pattern of life, genetic transmission of this disease and its diathesis will be the ultimate outcome. Vascular disturbances due to vulnerability of the vasculature to intrinsic and extrinsic factors is only a variation of hypertensive vascular disease and many of them have a hereditary basis. Hypertension runs in families akin to other vascular disorders and its course is modified by the environment to a great extent. The complications of this disease is also in a large measure controlled by environmental factors. Psychogenic influences of a continuous and repetitive type disturbs the neurogenic apparatus and has a damaging effect on the vasculature. As a result of vascular spasm, various organs and tissues suffer progressive damage and the individual succumbs to the adverse results of such complications.

The neuorgenic apparatus mediated through the autonomic Nervous system mainly the Hypothalamus is controlled by psychogenic factors, becomes extremely vulnerable and labile in course of time. Minor episodes in life and disturbances in health tilt the balance of this important control. The threshold for stimuli gets lowered and subminimal impulses from a conscious level upset the status of the vasculature. The individual may not only be a subject of Hypertension but manifests other symptoms of autonomic disturbances. The so-called symptoms of a functional disorder or anxiety state is closely and inextricably interwoven with hypertension. These symptoms may precede High Blood Pressure by several years

and may be the forerunner of this disastrous and destructive syndrome. Several of them sometimes do not develop Hypertension but may manifest other vascular syndromes such as Migraine, Raynauds phenomenon, node bleeds etc. So it is called essential that in the history of Hypertensive individuals, these symptoms must be elicited to exclude within certain limits symptomatic Hypertension. The cardinal symptoms of an anxiety state being pain and palpitation in Praecordium, headache, giddiness, swetting, cold extremities, hot feeling over the body, heavy feeling at the pit of the stomach, polyuria, Backache, pain in the neck, sleeplessness fatigue and a host of other complaints.

People with high blood pressure can live for a fairly long period but unfortunately the life is devoid of calmness and serenity. It is full of problems, very tense and hurried, and it continues to be of this pattern, till the last. These individuals avoid the Doctor till a stage is reached when they can no longer endure the distressing results of continued Hypertension. Many of them may not be aware that the manifestations noted are the results of hypertension till the time of clinical examination.

It is a very common experience to come across hypertensives, who in spite of a very high blood pressure have negligible symptoms as compared to others whose blood pressure is moderately raised but have the severest of complications. What controls and modifies the course of the disease is still a matter of speculation and doubt. Another interesting feature of Hypertension is its varying speed and course. Benign in character in some people, and at times taking on a malignant turn. This behaviour is probably regulated by the inherent quality of the vasculature which can hypertrophy to enormous limits if the level of blood pressure is not high. On the other hand if the Blood pressure reaches higher limits due to intense neurovegetative stimuli, the vascular walls becomes thinned out. A vascularity of the arteriolar walls has a deletrious effect and it is this feature which is responsible for the onset of complications. The more intense the spasm, the greater the tendency for arteriolar ischaemia and in the bigger arteries the same phenomenon is noted due to spasm of Vaso-Vasorum. Other factors secondarily may be responsible for intensifying the ultimate sequel of hypertension such as endocrine factors. So arteriolar necroses in others or both changes may be seen together. Another interesting feature is that in certain vascular areas arteriolar hypertrophy occurs and in other areas arteriolar necrosis. This indicates that the whole vasculature of the body may not share uniformly the pathological changes of hypertensive disease. It is unevenly distributed and this may also be responsible for the varied complications. What guides this distribution is also a matter of doubt probably to be explained on a developmental and genetic basis. Those who have studied anxiety disorders have formulated and observed that the pattern of response is peculiar to the individual at times of stress. Each individual has his own sensitive organ which is the seat of disturbance. This can be compared to the shock organ of Allergic diseases which is easily sensitised to Allergen. So also in psychoneurosis the heart, brain, kidney may be the seat of functional disturbance. There is not much difference between allergy and anxiety state, both are allied and belong to the Psychosmatic disease group. It is the sensitive and highly strung individual who is the subject of allergy. From this

point of analytical study it can be concluded that this sensitive organ faces the maximum insult in response to Hypertension, and easily shares the untoward effects of this Disease. Moreover, the same phenomenon is noted in families, those who have Hypertension develop only a particular type of complication. It is more or less a family disease. No doubt other postulates like organ inferiority, easy susceptibility, occupation of individual may be invoked to explain this oft-noted feature, yet it is more appropriate to explain it on this basis.

The complications of Hypertension are many and varied. Sometimes multiple complications are seen in the same individuals, one having a bearing on the other or independent of the other. Also complications may follow in regular sequence, ultimately crippling the individual. In the absence of complications, the disease is latent unless detected in a routine examination. The onset of the complications may be very insidious or may be sudden and dramatic requiring immediate treatment. It becomes an emergency in hypertensive vascular disorder and may be confined to cardio-vascular, Cerebro-vascular Reno-vascular etc., areas. Sometimes the first attack itself may end fatally within a short time or may be delayed. In studying these complications, statistical details are necessary to list them in order of frequency and in relation to age groups. With a certain degree of accuracy it can be said that Cardio-vascular complications occur in a comparatively younger age group than cerebro-vascular complications. Reno-vascular sequelae is noted in all age groups and in association with the other varieties of complications. In cases of malignant hypertension, Reno-vascular complications are more prevalent and seen in very young individuals. The following is a list of the common complications met with in daily practice.

DRAMATIC ONSET :

1. Coronary thrombosis; Angina Pectoris.
2. Cardiac Asthma.
3. Hypertensive Enchephalopathy.
4. Cerebral thrombosis & Haemorrhage.
5. Mesenteric Artery Thrombosis-Rare.
6. Epistaxis. Haematuria.

INSIDIOUS ONSET :

1. Right-sided failure.
2. Renal failure. Uraemia.
3. Retinopathy.
4. Diabetes Mellitus.
5. Gangrene.

Dramatic onset of complications :

Such complications usually come on without previous warning. They are sudden in onset as to alarm the patient and his friends and it is usually noted in people of robust constitution who claim to have led very healthy lives. Many of them succumb to these complications within a very short time. CORONARY THROMBOSIS is a entity often seen as a complication of essential hypertension and is responsible for a great deal of invalidism and high mortality . It is a very dreaded syndrome. These attacks come on with such suddenness that the symptoms noted are sometimes impossible to combat. The condition is one of the many problems in Medicine, where the physician has to wait with patience for the ultimate result. Though a standard method of therapy has been formulated for the treatment of coronary thrombosis, it may not be of much value in a patient who has massive myocardial infarction. It may be

sceptical to say that drugs are not useful as inherent factors such as extent of damage, degree of shock and the efficiency of collateral channels are factors controlling recovery. Though several authors have given the statistical details pertaining to the efficacy of latest drugs in the management of Myocardial infarction, it is still a matter of doubt whether any drugs are at all useful in the treatment of this condition. No doubt the treatment of shock may be a rational line of approach as prolonging the life of the individual may help the bodily restorative mechanisms to heal the myocardial infarction. Therapeutic nihilism may at times be more useful in the management of these patients than the use of several parenteral and oral drugs. Coronary thrombosis as a complication of hypertension is fairly common and its incidence is on an increase. The Age period of this entity is between 30 and 50 years as compared to coronary occlusion result of coronary Atheroma, which is seen in a older age group. It is very common at the present day to hear and see young individuals between 30—40 years approaching the physician with Ischaemic manifestations referable to the heart. Very often the inexperienced consultant brushes aside the symptoms as of negligible importance and labels the patient as a case of Neuro-circulatory Asthenia, Psychoneurosis, Hysteria etc. but unfortunately sometimes the physician has failed to go into the history carefully. Electro-cardiographic and Radiography very often are of little assistance in the clinical evaluation of the case. On many occasions the scientific consultant is correct in making such a diagnosis. Who can suspect Myocardial Ischaemia in young individuals with vague Praecordial pain ? But at the present day it should be the aim of the Doctor to pay a little more attention to this complaint. Coronary insufficiency as a result of Hypertension is due to Coronary sclerosis associated with a hypertrophied heart. The bulk of the cardiac musculature and its surface area has increased and the dull praecordial pain experienced by such individuals is due more to relative insufficiency of blood. The pain here is intensified on exertion and has all the features of cardiac pain. This syndrome can never be missed especially by one who is accustomed to a regular routine in clinical examination.

ANGINA PECTORIS is also associated with Coronary sclerosis. Sometimes it may be unassociated with changes in the vasculature. Moreover heart may not show any change in size or shape on radiological examination. Here the pain is due to the spasm of the Coronaries due to a raised pressure. The pain is exertional and subsides on taking rest. Coronary spasm is precipitated by Neuro-vegetative stimulii : very often seen in people with a moderately raised blood pressure. These spasmodic episodes become more frequent with advancing age and duration of pain and intensity of pain becomes worse. In such individuals impending coronary occlusion must always be expected as these are the threatening symptoms of the approaching crises. Rest from physical and mental work combined with slow acting Vaso-dilators will be ideal in managing such symptoms. It helps the patient and the Doctor to tide over a major crises, the outcome and the result of which can never be prognosticated. Where such warning signals are present, it may be easy to help a cautious and co-operative patient.

But unfortunately no such prodromal features are present in many individuals. The attack comes like a bolt from the blue. In the previous group there is a chance of collateral circulation developing but in

this group there is no time for collaterals to open out. Moreover during the acute stage the coronary arterioles go into a state of spasm especially those in the neighbourhood of the Infarct facilitating the easy spread of necrotic process into the healthy Myocardium. Usually these sudden attacks are due to intense coronary spasm involving large segments of arterioles or multiple segments. Prolonged spasm induces arteriolonecrosis with subintimal bleeding which precipitates thrombus formation. In the younger age group sudden death is the usual result. Where the Coronary arterial tree shows arteriosclerotic changes, the prognosis during the first episode of coronary occlusion may not be fatal as a certain degree of collateral anastomosis may have taken place. In these individuals there is always prodromal warning.

The clinical features of CORONARY THROMBOSIS are so characteristic that it is rarely mistaken for other conditions. It is only an atypical cases that problematical discussions ensue. The intense, excruciating, Retrosternal pain which may radiate to the left shoulder or arm, to neck or back or right shoulder is the presenting feature. This is associated with dysponea and restlessness which makes the individual toss about in bed. The intense shock and circulatory failure with marked swetting, high pulse rate, fall of Blood pressure, vomitting, hiccough is very diagnostic. Sometimes there may be a Bradycardia in Posterior myocardial infarction.

In atypical cases Paroxysmal Dyspnoea, Dyspepsia and feeling of uneasiness in the chest or abdomen may be the presenting features without any of the other more dramatic symptoms. Blood pressure and pulse always give a clue to the diagnosis especially when a physician is conscious of this entity, Coronary thrombosis when confronted with individuals in the later age group.

The management of the patient is a matter of strict and rigorous bed rest at least for 3 weeks. During the acute stage Morphine susphate ¼ grain may be a useful drug to relieve the pain and combat the shock given repeatedly at intervals of 4 - 6 hours till the pain subsides. It is always helpful to give Morphine without any reservation, when diagnosis is certain and when patient is conscious. Oxygen is an essential requisite even in the absence of dyspnea.

In case where shock is intense with a low Blood pressure, and Anuria is impending, 5% Glucose Saline not more than 1 pint with Nor Adrenaline is a useful restrorative. Other stimulants like Adrenal cortical harmone can also be used to raise the blood pressure.

Use of vaso-dilators to help collateral circulation and prevent spread of infarction is a debated point when Blood pressure is low. But it can be administered with impunity when Blood pressure is normal or a little lower. Papaverne, Ammophylline or Khelline are the drugs of choice but its value cannot be assessed. Anticoagulants at times may be harmful especially when the factor which precipitates thrombosis is subintimal bleeding. It should be given only when the pain continues for a long period of 3 - 4 days without the slightest amelioration. These drugs may prevent extension of thrombosis and quick recurrence of such thrombotic episodes. Mural Thrombus formation and venous thrombosis can also be minimised. These drugs can only be used in an institution and not in private practice without Laboratory aid. Much can

be said about these drugs and their usefulness. The results are only comparative and in a syndrome like Coronary occlusion the value of drugs can never be assessed.

CARDIAC ASTHMA is another emergency in Cardiovascular Medicine in hypertensive individuals requiring immediate treatment. It is a complication seen in an older age group —50 years and above and not so common in young hypertensives unless the individual is a subject of malignant hypertension. These attacks usually come on at night after a heavy day's work, physical or mental, or after a heavy meal. The attacks may repeat every night and each subsequent attack lasts longer than the previous one. (Paroxysmal nocturnal Dyspnoea) Sometimes the first attack may prove fatal especially in an individual with a markedly enlarged heart with Coronary sclerosis and a very high blood pressure. In hypertensives the precipitating factors at times is Coronary thrombosis itself. These episodes are ascribed to acute failure of the left ventricle with intense congestion of the pulmonary circulation giving rise to Orthopnoea, cough and wheezing. If neglected, the condition may prove fatal as Pulmonary hypertension may be followed by Acute pulmonary oedema, a pathological state very difficult to reverse.

The nocturnal periodicity may not at all be maintained and the attacks sometimes last for several days. It is said that an imbalance in the pressure in the two ventricles precipitates these attacks during periods of rest when right ventricle has a better venous return. A diseased and weakened left ventricle cannot increase its working capacity and its arterial return increases. So these attacks are an eventual outcome of stagnation of blood in pulmonary circulation, as a result of a failing left ventricle. So Orthopnoea due to diminished vital capacity is a classical feature of Cardiac Asthma. Intense cough and wheezing occurs due to reflex spasm of the Bronchioles as a result of pulmonary hypertension and congestion. (Vago —vagal reflex—Vascular Bronchial reflex) The blood pressure during these attacks rises further and this also may be a precipitating factor especially when some emotional episode stimulates the rise of a pressure. These attacks have to be differentiated from Bronchial Asthma and when occurring in a subject who is already an Bronchial Asthmatic may give rise to several difficulties. Of course the presence of a hypertrophied heart with high blood pressure will be an aid to the diagnosis but it is not a sole criteria specific to Cardiac Asthma. An individual with Bronchial Asthma can have High Blood Pressure both being Psychosmatic disorders. Moreover during the Asthmatic Attack, clinical examination of the heart will be a problem as sounds are masked by respiratory noises. A cautious approach is always necessary. A gallop rhythm, more Rales than Rhonchi in the lungs especially when Basal in distribution is always helpful in the diagnosis. Other findings like engorged neck veins and tender liver will be associated features as pure left sided—failure never remains as such for long. There will always be associated mild right-sided failure.

The management of these patient is an equally difficult problem. Oxygen administration under high pressure is a valuable procedure which helps in better oxygen saturation and also opposes the pulmonary arterial pressure which is raised. This manouevre helps to prevent Pulmonary Oedema.

Morphine sulphate ¼ grain is one of the useful drugs in cardio-vascular emergencies and its place in therapeutic armamentarum is unique. It is a drug along with its allied alkaloidal preparations e. g. Omnophon which can never be replaced by more potent products. Pethidine hydroch 100 mgms. has also a useful role in emergencies, and at times the drug of choice when vomitting occurs as a side effect of Morphine. In cardiac asthma, it is almost a specific to reduce the dyspnoea and rest the patient. It helps to conserve cardiac energy by reducing its work by depressing the respiratory and vaso-motor centres. The drug should be regularly given as long as dyspnoea persists.

Other drugs of equal importance to combat the failure are digoxin and Ammiphyllme which can be mixed up in the same syrings (Digoxin .5 mgm. s+Ammophyllme ½ gms.) diluted with 50% glucose 20 c. c. and given intravenous very slowly. This helps to control failure and reduce blood pressure and the mild diuresis which is produced favours decongestion of lungs. Other useful vaso-dilators like Khe-llin can be given side by side. If B.P. is very high Gangliopllegic drugs must be given, intramuscular to reduce the B. P. slightly but not to the normotensive level as such a manometric device may induce other drastic complications. Vegolysm or Ansolysin retard are additional accessory measures in the management of these patients.

The value of Venesection in plethoric individuals is inestimable, and should be practised as a routine. Why venesection is gradually falling out of repute is not well understood. In comparison to bloodless venesection, which is practised by making the patient hang his legs down and tying torniquets to three limbs and releasing one after every ½ hour, it is far superior. This method is to reduce venous return to the heart, thereby reducing the burden. Venesection should be done as a routine in Cardiac Asthma of hypertensive origin. It not only controls the attack but also reduces the blood pressure for a longer period thus helping as a Prophylaxis for this complication. Periodical Bloodletting is even now a very useful method of prolonging the life and reducing the incidence of complications in persistant hypertension. Its value should never be underestimated, and this practice should be revived.

HYPERTENSIVE ENCHEPHALOPATHY is another emergency in Hypertensive individuals involving the Central Nervous System especially the brain. It is analagous to Angina Pectoris as the underlying cause is Vascular spasm. Cerebral vascular spasm is often precipitated by some psychogenic factor which phenomenally raises the blood pressure. It is seen in old people with Cerebral arteriosclerosis and in young people who have malignant hypertension. Various focal areas in the brain may be the seat of Ischaemia. Usually generalised Cerebral vascular spasm is a rarity and these enchephalopathic episodes are due to affection of isolated vessels. Such attacks usually come on suddenly and disappear within a short time. These transient focal cerebral symptoms may vary in different individuals and in the same patient at different times, as these attacks may repeat themselves. The common manifestations are hemiplegia, aphasia, visual disturbance, fits, coma, headache, severe vertigo depending on the vessel affected. Sometimes mental disturbances occur—maniacal attacks. Cerebral oedema may follow such attacks if the duration is prolonged. Enchephalomalacia can also result. Simi-

lar to Coronary thrombosis, occlusion may occur in the cerebral vessels if spasm lasts for a long duration especially in an artery which shows sclerotic changes. Arteriolonecrosis is the precipitating factor for the thrombosis.

CEREBRAL HAEMORRHAGE is the final terminal event after several enchephalopathic and thrombotic episodes. The vessel gives way due to softening of surrounding cerebral tissues and marked damage of the arterial wall, helps the rupture. Patients are usually brought in a deeply comotose state with stertorous breathing. There is unilateral paralysis of arm and leg. These individuals belong to an older age group and the mode of onset usually helps in the diagnosis. The cerebro spinal fluid may or may not contain blood. As the progress is watched it will be found that the pulse rate gradually falls, the blood pressure rises higher, finally circulatory failure sets in.

Hypertensive enchephalopathy must be diagnosed quickly, treated early before permanent necrotic changes occur in brain (softening). The blood pressure must be lowered by various methods and intracranial pressure reduced.

Ammophyllme 3½ gr. diluted with 50% glucose 25 c. c. or a drip is very helpful to lower the pressure gradually. Vegolysin or Ansolysin by the intramuscular route is also a necessity in refractory cases. Lumbar puncture reduces the intracranial pressure and is a procedure which also helps in the diagnosis of the case. Conc. Mag. sulphate — XII oz. as retention enema is also useful.

As blood pressure falls spasm passes off and patient gradually recovers from the state of coma or recovers function of the ischaemic areas. Where convulsions are the sole manifestation, sodium phenoparbitone or paraldehyde should be used intramuscular for controlling the fits. Morphia should not be given. Venesection may be performed when blood pressure remains high in spite of all these methods. This is a very effective method of reducing the pressure and thereby the cerebral vascular spasm. The quantity of blood removed must be about 20 ounces or more. Cautions reduction in Blood pressure is necessary as thrombosis may be precipitated. Pressure should not be brought down to normal limits but kept a little above normal. One of the dangers of hypotensive therapy by drugs, venesection etc. is precipitation of coronary or cerebral thrombosis.

Mesentric artery thrombosis has been described as a rare complication in essential hypertension. Sudden pain in the abdomen with rigidity of the abdominal muscles and shock are characteristic of an acute abdominal catastrophe. Associated with these clinical features patient passes frank blood in stools. This is followed by abdominal distension and vomiting. Haemorrhagic infarction of the bowel with ultimate gangrene is the underlying pathology. Treatment is to do an immediate laporotomy and resect the involved gut. It is a serious complication with a high mortality.

EPISTAXIS is sometimes a distressing complication which usually alarms the patient as the nose continues to run blood in spite of all measures to stop it. This is a feature which occurs repeatedly in some patients and subsides spontaneously with or without treatment. These people have usually relief from other symptoms after a bout of Epistaxis. More or less it is a mechanism by which blood pressure tends to get lowered by a

spontaneous venesection. These individuals usually bleed from the arteriolar flexus situated in the nasal septum or sometimes a large vessel gives way explaining the profuseness of blood which is lost. Various routine measures adopted in the control of bleeding makes no change in the leakage. Even packing the nares with gauze soaked in adrenaline is ineffective, the gauze packing usually gets soaked with blood. Epistaxis need not be controlled speedily especially in severe hypertension unless the bleeding is profuse and long continued. The patient should be quitered by Morphine sulphate ¼ gr. intramusculary. Provided there is no other cause for the bleeding, it should be watched carefully till it stops. Usually after epistaxis the blood pressure falls and no untoward symptoms occur, except the psychogenic disturbance associated with sight of blood.

The safety valve mechanisms can be seen in many other organs especially kidneys. Haematuria may be very distressing. It is a painless passing of blood and stops spontaneously. But haematuria must always be investigated to exclude other local organic causes as neoplasms in kidney and bladder even though occurring in a hypertensive patient. Then alone it should be ascribed to hypertension. This must be borne in mind as haematuria is not a common manifestation in hypertension.

RIGHT-SIDED FAILURE is the eventual result of continued myocardial ischaemia, result of coronary arteriosclerosis which is a *Sine Qua Non* of Hypertension. Moreover left ventricular dialation may not be associated with any symptoms for a long period of time and its eventual sequelae is right ventricular failure ; or the patient may have experienced paroxysmal attacks of dyspnoea preceding the more permanent complication of right ventricular failure. At this stage the myocardium usually has no reserve whatsoever and permanent relief is almost nil. With treatment the condition improves but again the attacks are repeated. So it would be correct to say that right ventricular failure is associated with poor chances of long life and healthy existence. A little exertion usually brings on the failure in spite of maintenance dose of digitalis and periodical mercurial diuretics. Some of them do not respond to duretics and it is difficult to keep them odema-free in spite of strict salt restriction and administration of ion exchange resins. These individuals as a result of poor intake of food and improper management develop hypoproteinemia and this to some extent maintains the oedematous state. Some of them have a persistantly enlarged hard liver probably as a result of long, continued, mild failure which induces fibrotic changes — cardiac cirrhosis. This also adds to the Hypo-proteinaemic state.

These patients are brought into the hospital with breathlessness on exertion, severe cough and oedema of legs sometimes with generalised oedema. Anasarca with all the typical feature of congestive heart failure. A careful history taking always reveals a past or present complaint of chest pain with or without exertion. Many of them have episodes of coronary thrombosis which has precipitated myocardial failure and a large percentage have chest pain due to coronary insufficiency. Blood pressure is high but not to the level before onset of congestive heart failure. It is very interesting to note that blood pressure gradually rises with progressive improvement and as compensation is restored. This increases the strain on the myocardium and completes the vicious

circle. Use of hypotensive drugs along with other therapetic measures adopted in the treatment of congestive failure is always beneficial in prolonging the period of compensation. There is a disadvantage with the use of hypotensive drugs which at times may be unfavourable in the long run especially when complicated by failure. When the blood pressure is brought down to fairly low limits, the resistance and tone of the arteriolar walls is decreased and the vascular paralysis which results, decreases the efficiency of the circulation. Oedema persists due more to local causes, and dyspnoea may become worse due to pulmonary vascular paralysis. Sometimes it has been observed that thrombotic episodes are precipitated. As a result of such adverse factors management of hypertensive congestive heart failure is a major problem in Cardiovascular practice.

Patients with congestive failure should be at strict bed rest and sedated well. This is a useful plan to follow as many of them complain of lack of sleep and restlessness. Sedatives are very useful in all cases of congestive heart failure and more so in hypertensive failure. Phenobarbitone is the most useful given in optimum dosage as 2 - 3 grains per day.

Diet of these patients should not be restricted to the low calorie low protein diet for long as it leads on to hypoproteinaemia. Kempners diet is useful in a hypertensive patient with no complications for short periods but not for life. A optimum protein requirement per day is essential and may be given as food, or in the form of protein hydrolysate but the diet should be low in cholesterol and fats. Eggs and fats should be cut out. Carbohydrates can be given liberally.

The question of low salt or no salt in the diet is an important problem which may not suit the taste and temparament of the patient. This is a valuable and age-old dietetic precaution followed in the management of congestive heart failure. Food becomes tasteless and insipid and may also make the patient feel asthenic, more so when mercurials are being used. Under such conditions a low sodium syndrome or low chloride syndrome may result. In order to avoid these disturbances in electrolyte balance patient is given a low sodium diet. Many of the common food stuffs have sodium such as milk, bread, south Indian dishes and it is impossible to do without them.

Salt substitutes contain mainly K salts and are no substitutes for Na ion in taste. Hence many are aversed to it and moreover it may, induce K intoxication if used too freely. Weakness, paraesthenias and even paralysis may occur. Ion exchange resins have not solved the problem of salt substitution for several reasons. The dosage of the drugs are high and cost is enormous, and side reactions frequent. Ammonia intoxication or K intoxication may occur even though two Kation resins are combined. About 14 Gms. of this resin must be dissolved in water and taken with or just before food thrice a day. Sometimes these resins withdraw K and Ca causing hypocalcaema, hypopotassaemia etc. As these resins have only a transient beneficial effect, it is not used extensively in medical practice except in persistant oedemas to withdraw Na ion.

The other useful drugs are digitalis in the form of Digoxin, Cedelanid etc., given initially in large doses to digitalise the patient and then continued for prolonged periods in maintenance dose of .25 mgm. twice a day. Mercurial diuretics by the

parenteral route are the most helpful to overcome failure. Later it can be given once in 10 days or less frequently for some time to maintain the patient oedema-free. Oral mercurials e.g. Merchloran are also helpful given as tablets every 4 - 5 days; Other diuretics like ammophylline can be given with mercurials. It not only potentiates the action of mercurials but in addition has a vaso-dilator action, reduces the blood pressure and dilates the coronary arteries. The drug should be given intermittently at weekly intervals as its action wanes due to tolerance being acquired.

Diamox is also a mild diuretic which may be beneficial to cases of hypertensive failure. It can be used intermittently for 3 days, once in a fortnight or more frequently depending on the patient's condition. In spite of multiplicity of drugs and dietetic precautions and advice, the ultimate result of congestive failure is not good.

These patients in the later stages may reveal different types of cardiac irregularities, depending on the extent and severity of myocardial fibrosis — extrasystoles and auricular fibrillation are often noted. Sometimes different grades of heart block, gallop rhythm and Pulsus alternans. The latter group are more serious and presage serious results. It occurs in patients who have damage of the conducting system.

RENAL FAILURE may occur quite early in young hypertensives due to vascular damage in the kidneys. Arteriolonecrosis is the main vascular lesion in the more accelerated types of hypertension which results in glomerular infarcts. Here acute renal failure is the sequelae which is uncontrollable, haematuria, oliguria and lowered urea excretion is noted. Many of these patients have other complications of hypertension which progress along with renal involvement such as Enchephalopathic crises, Retinopathy and sometimes acute Cardiac failure. It is very difficult to manage these multiple complications and the hypertension itself is irreversible and uncontrollable with hypotensive drugs.

In benign hypertension, renal failure is a late result. These patients usually develop cerebral, cardiac or coronary complications and if they happen to survive chronic renal failure occurs. Very often a coronary attack precipitates renal failure. Many of them have albuminuria for long periods without any change in the specific gravity of urine or alteration of blood urea content. Kidney, like the liver can still maintain adequate function in spite of optimal damage and with dietetic regulation, life can be prolonged. Renal failure is detected usually on routine examination when a hypertensive is brought for other complications. By itself, it does not give rise to disturbing symptoms unless the Renal function is very much disturbed. In such patients, vomiting may be an early symptom; lethargy, excessive sleep, a twitching of muscles, headache, hiccough, sometimes diarrohea. In advanced condition convulsions occur later. Coma and other serious symptoms result.

In patients who have albuminuria, use of hypotensive drugs may precipitate renal failure. These individuals have kidneys, whose vasculature is sclerosed and a certain height of renal pressure is necessary for filtration process. When the pressure is reduced, kidney fails to function and blood urea rises. When the Blood pressure is allowed to rise, the waste

products are easily excreted as its function improves. So, it is advisable to have the kidney failure assessed before the use of these drugs in order to prevent renal failure, which may pass unrecognised. These patients have low fixed specific gravity, albuminia, and oliguria, sometimes polynuria. Many of them have nocturnal polynuria.

It is a problem to manage this condition. Renal function can never improve unless it is only hypertensive Nephropathy where spasm of the renal arterioles is the main pathologic feature. In such crises urine is reduced in quantity with transietalbuminuria. Later a fall in blood pressure relieves the condition. These patients do well with antispasmodics like Aminophylline given intravenously and other dilators like Apresoline. Even without vaso-dilators, sedation may improve this state.

In nephrosclerosis, management is a matter of minimum drug therapy and essentially Dietotherapy. Food should be low in proteins and salt should be restricted. Milk can be given to supply the minimum amount of proteins along with Butter milk. The energy can be supplied by a diet consisting of carbohydrates and fats.

Bulls regime:	Glucose	— 100 oms
	Pea nut oil	— 40 oms
	Acacia	—
	Aqna	— 1000 c. c.

Ammophylline intravenous can be given freely to keep dialation of Renal vessels and induce better duresis. Life can be prolonged. Apresoline in doses of 25 mgms. four times a day raising it 100 mgm.s four times a day has been recommended but in many of the patients treated in the wards, no beneficial results have been noted.

These patients can be given rectal washes and Conc. Mag. sulphate as retention enemata, but they are mere placebos. In countries where blood is available in large quantities, exsanguination transfusion or cross transfusion periodically may help the patient to dispense with the waste products. Other mechanical measures is the artificial kidney technique which is a device to remove excretory products, by means of a dialysate which bathes the blood which is circulated outside the body.

These measures are purely temporary and may prolong life for a few months.

RETINOPATHY sometimes is acute and dramatic in onset in malignant hypertension. Headache, vomiting and papilloedema suggest either enclephalopathy or uraemic syndrome. Patients usually enter the ophthalmic department for sudden failure of vision. Examination reveals retinal haemorrhages and exudates with marked papilloedema. This is usually aggravated by albuminuric retinopathy as uraemia exists very often with retinopathy. Grade IV changes occur so suddenly due to Retinal arteriolonecrosis and papilloedema due to sudden rise of intracranial tension.

In benign hypertension Retinal arteriosclerosis sets in progressively in the four stages described by Keith, Wagner and Barker. It is difficult to see a patient with Grade IV changes as they are usually carried away by other complications before such a stage is reached.

The usual symptoms for which such individuals come to the Department are

gradual failing vision, restriction of field of vision, blind spots in field of vision. These individuals cannot improve much with treatment as permanent changes not only in retinal arterioles but also in retina have taken place due to haemorrhagic exudates and Ischaemic phenomena. Along with retinal changes, cerebro-vascular sclerosis progresses and patients may gradually lose their intellectual capacity to remember, to decide and act. Mental power gradually wanes and dementia may ultimately result. Some of these individuals lose the power of internal thought, speech etc., and lead a helpless existence. Many of them have paralytic strokes in rapid succession and become bedridden. In a few cases Parkinsonian symptoms gradually come on and later motor disability becomes extreme. Talking, eating, walking are performed with great difficulty. Sometimes Bulbar paralysis and emotional disturbances are combined. (Pseudo-bulbar syndrome) Before these patients completely lose sight, other complications are prone to occur which prove to be more serious. Generalised cerebral arteriosclerosis is a well recognised entity occurring in individuals between 50 - 60 years. It may be mistaken for other degenerative disorders of the C. N. S., viz. G. P. I., and senile psychosis and infections of the brain. The high blood pressure which these individuals have makes the clinical entity quite distinct and the progressive nature of the malady punctuated with acute vascular episodes characterises its vascular origin.

Treatment is difficult and reversal of changes is remote. Powerful vaso-dilators and hypotensive drugs can only reduce the B. P. but little improvement is noted in the tissues which have been scarred.

Testo-viron 25 Mgms. for prolonged periods combined with vitamin *E* may inhibit rapid progressive vascular changes. These entities are only of academic interest and not amenable to therapy.

GANGRENE of lower extremities is not a common sequelae of hypertension. It does occur but its incidencies low. Very often diabetic subjects who have hypertension develop gangrene quite frequently. These two diseases often co-exist and it is the Diabetes Mellitus which is the prime disease for such a condition. Hypertension may accelerate the spread of the gangrenous process. Intermittant claudication is an important symptom in such people who have arteriosclerosis of muscular and digital vessels. Some of them have vascular disturbances in the extremities like tingling, numbness (parasthesias) — slight ankle oedema passed off as peripheral neuritis, more so when hypertension is combined with Diabetes mellitus. Vitamin B, is given for long periods with little improvement. Vaso-dilators like Priscol combined with hypotensive drugs produce immediate relief. No doubt vascular sclerosis may be associated with interstitial neuritis., and the vaso-nervorum undergo a similar change. The treatment of both is identical.

DIABETES MELLITUS can occur as a result of sclerosis of pancreatic vessels in hypertensive disease. Such an entity is often seen in middle aged Diabetics who have a persistantly raised B. P. without any history of diabetes in the family. The islets of langerhamm become less vascular and the output of insulin is diminished. Moreover insulin is not thrown into the vascular sinusoids due to thickening of walls of capillaries. Minute areas of necrosis may occur in the pancreatic tissue reducing the Harmonal level.

Very often this is a common observation to come across patients who have glycosuria of a transient nature without much alteration in the diet or exercise. In times of stress and emotional states. glycosuria is a feature in hypertensives and even in healthy non-hypertensives ; Whether the glycosuria is due to disturbance of hypothalamo-hypophysial pathways cannot be conjectured. It should also be due to changes in the calibre of pancreatic vessels as a result of spasm. In course of time permanent atrophic changes occurs in the pancreas. It is in such individuals, who in addition are obese that rest, sedation and dietetic regulation in the early stages help to prevent changes in these vessels.

It can be understood from this short treatise on the complications of hypertension that any tissue and organ in the body can suffer temporarily or permanently as a result of this psychosmatic disease. The vasculature of the body is the main link between the peripheral tissues and the psychogenic apparatus mediated through neuro-humeral mechanisms and it is this musculo-elastic structure which is a barometer of psychogenic influences. It need not result in hypertension but can reveal itself in various types of vascular syndromes. Many organs in the body suffer simultaneously or separately from changes in the calibre of vessels and alteration in the quantity of blood supplied to these tissues. In addition continuous neuro-humeral impulses alter the texture and thickness of these vessels. In addition disturbance in metabolism of fat and carbohydrates occur resulting in Lipoid degeneration of vessels. It is only as a result of these changes and organ disturbances that hypertensive disease is revealed to the patient and the doctor. Otherwise the disease will be hidden and not discovered unless examined routinely for some other trouble. Moreover during the time of examination, raised blood pressure may not be evident and the hypertensive diathesis is often missed. These are problems to the patient and the Doctor.

Even allergic reactions are related to body vasculature since changes in the capillaries are the main alterations which initiates the symptoms. A person of this diathesis is subject to psychogenic influences and several hypertensives are allergic personalities. The discussion of hypertension is an elaboration of blood vessel mechanics and blood vessel changes quite different from the changes noted in old age and in other obliterative diseases of the vascular tree.

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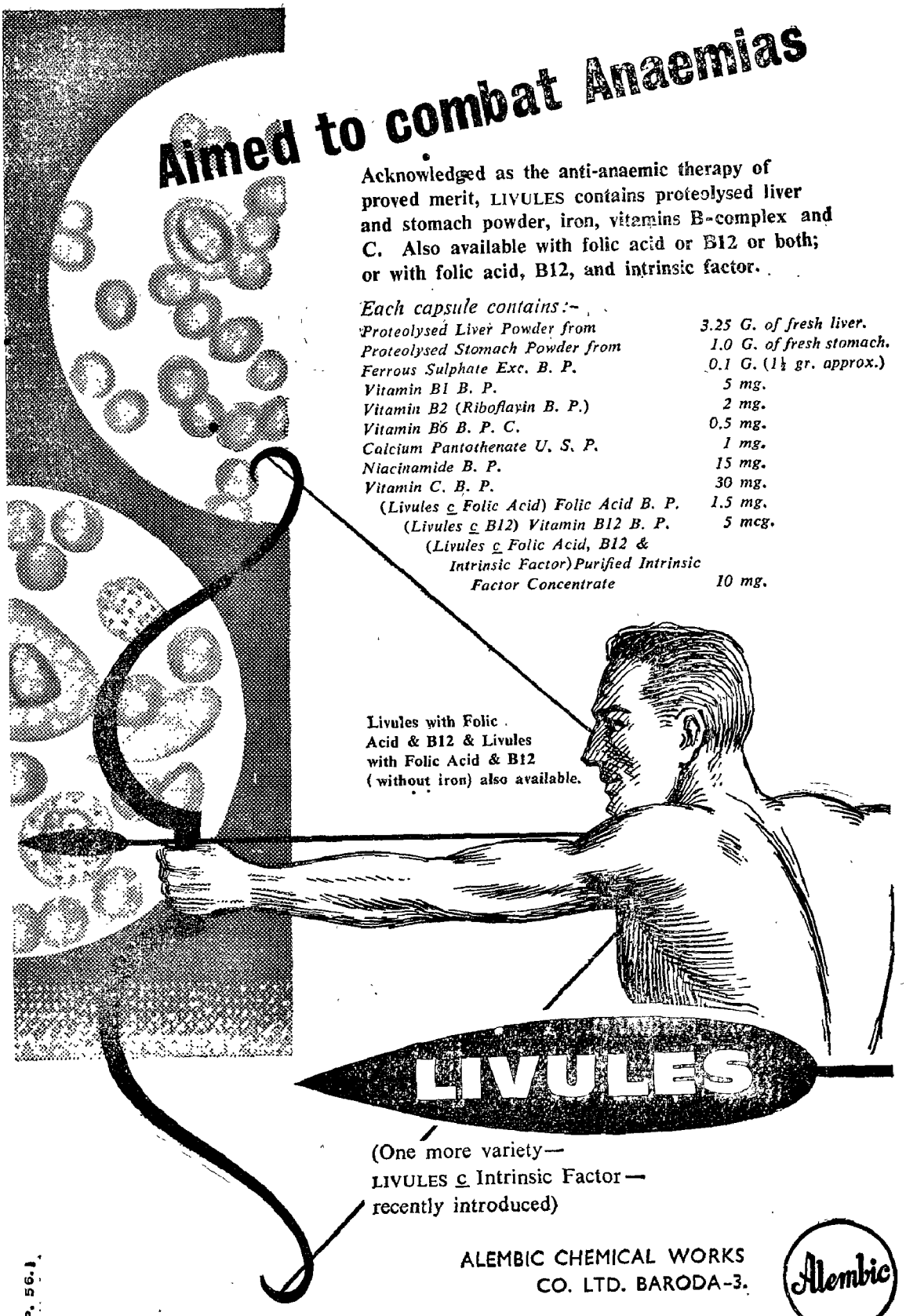

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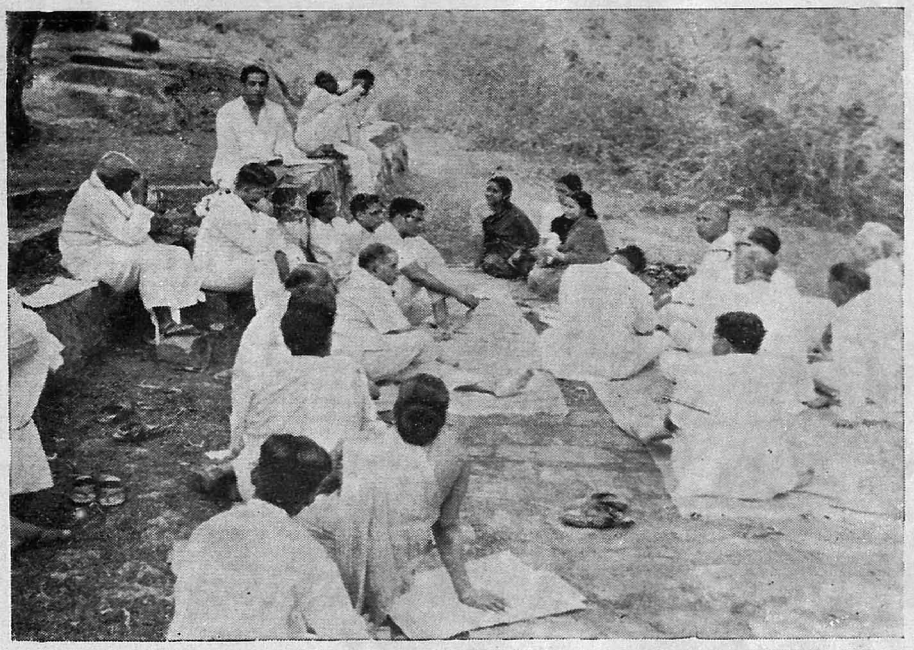
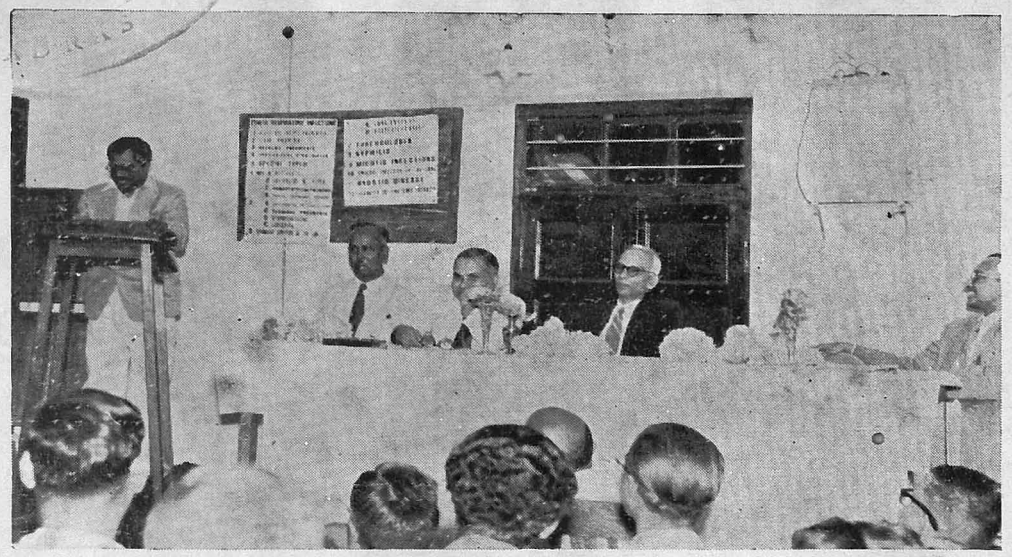


About Coma at Cumbakarai

It is evident they had good lunch at Cumbakarai.

Photo : Dr. S. Bhuvaneshwar



'Symposium'

Cocktail

Photos — J. C. Bose

Tuberculosis of the Large and Small Intestines

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TUBERCULOSIS is a general disease with focal manifestations. This should never be forgotten in the treatment of tuberculous patients, whatever may be the manifestation of tuberculosis in a patient. This point needs to be much emphasised especially in the surgical manifestations of the disease, since in the enthusiasm of treating the local manifestations, the general aspect of treatment is likely to be forgotten or slighted, with disastrous results. It is almost an everyday feature to find, in the surgical Outpatient room, tuberculous patients with involvement of more than one system ; again, after a surgical excision of a tuberculous focus (e.g. Caecum, kidney etc.,) milliary tuberculosis may set in, in the post operative period or some other focus, which was hitherto dormant, may flare up. The early manifestations of tuberculous infection of any system is more often general than local ; likewise, the early indications of a favourable response to treatment of a tuberculous focus is general. I have taken pains to stress this point, to avoid repetition later and to stress the great importance of the general treatment apart from local and specific treatment in every case of tuberculous infection, whatever may be the system involved.

PHASES OF DEVELOPMENT OF TUBERCULOSIS

The various phases of manifestations of tuberculosis in the human body have a similarity to those of syphilis. The cycle of events may be described under four phases, dating from the time of the penetration of the tubercle bacillus into the human body to the development of the most advanced lesions of visceral caseo-cavernous tuberculosis.

(a) *Period of Incubation* : This is a latent period between the entry of the Tubercle Bacillus into the body and the development of a positive cutaneous reaction, which is the first manifestation of an allergy to the tubercle toxin.

(b) *Period of Invasion* : In this stage, the tubercle bacillus reaches the lymphadenoid system. So far as clinical manifestations are concerned, this is a silent phase, although, occasionally general toxic symptoms like intermittent fever, lassitude and anaemia may be observed. Lymph node enlargements may be seen ; resistance in lymph nodes is maintained. Calcification in Lymph nodes may be seen. The cutaneous reaction persists.

(c) *Period of visceral spread* : In this stage, Lymphadenoid resistance is overcome, the lymphatic system, by its cen-

tripetal flow, empties into the blood-stream all organisms which reach the great lymphatic trunks. Implantation of tubercle bacilli are bloodborne to the lungs. It is the stage of spread of the disease to various viscera. Implantations of Tubercle Bacilli occurs into various organs through blood stream, chiefly the lungs, but the bones, joints and other organs may also be involved. The nature of the lesions produced in the tissues by the tubercle bacilli depends upon (i) the virulence and number of organisms and (ii) the resistance of the host.

The lesions may be sparse or milliary, active or relatively quiescent. Should the original infection during the first period of incubation be massive, the second period of invasion follows a rapid course and metastatic foci of infection may become fulminant and rapidly fatal. Should the second period of invasion be resisted by the lymphadenoid system, and the leakage of Tubercle Bacilli to the blood stream remain sparse and infrequent, the lymphadenoid system tends by sclerosis to overcome the original implantations in the lymph nodes, and metastatic foci of infection are subsequently rare in their appearance and slow in their development.

(d) *Period of advanced Caseo-Cavernous Pulmonary tuberculosis* :

The fourth period in the cycle of Tuberculous infection is that of Pthisis, characterised by extensive Caseo-Cavernous or ulcero-cavernous lesions in the lung. Caseo-Cavernous pulmonary tuberculosis is the last phase of Tuberculosis. The surgical manifestations of tuberculosis are the result of implantation of tubercle bacilli in the various systems or organs during the stage of visceral spread and the subsequent development of lesions in these organs.

TUBERCULOSIS OF THE ALIMENTARY TRACT

There are two main forms of Intestinal tuberculosis.

I. *Hypertrophic variety* which occurs at the Ileo-caecal Region is a form of "Primary Tuberculosis" since, as a rule there are no demonstrable lesions in the lung. Tuberculous infection of the ileo-caecal region differs in many respects from the small lesion of the small intestine.

II. *Tuberculosis of Small Intestine* is the other variety ; the lesions are multiple and commonly occur with pulmonary lesions ; Ileo-caecal tuberculosis occurs as a primary lesion in the alimentary tract. Ileal (or jejunal) tuberculosis is ulcerative in type ; Ileo-caecal tuberculosis (or caecal Tuberculosis) is usually of the hypertrophic type and it is due to less virulent strain of tubercle bacilli in a patient whose resistance is quite high. Exceptions to the generalization do occur in both the varieties. It is important to recognise these two varieties of Tuberculosis of the alimentary tract not only because of their differential clinical features but also from the point of view of treatment and prognosis. I shall deal with the two lesions one after the other.

I. *Tuberculosis of the Small Intestine* is quite common. As mentioned already, small intestinal tuberculosis usually occurs along with other tubercular lesions. Thus it may occur in association with pulmonary tuberculosis or is accompanied by tuberculous lesions in the caecum or mesenteric lymph glands. Adolescents and young adults are the usual victims. When in association with pulmonary tuberculosis, swallowing of infected sputum is held responsible for the

disease. The distal part of the ileum is worst affected since this is the most absorptive portion of the gut and, in addition, most richly supplied with lymphoid tissue. The lesion commences first in the Peyer's patches and spreads to form ulcers of typical tuberculous characteristics. The ulcers spread in the direction of the lymph drainage and lie at right angles to the direction of the long axis of the gut. These ulcers rarely perforate ; the most important point to realise is that, in healing by fibrosis they lead to intestinal obstruction of increasing severity. Such strictures of gut are often multiple and are widely distributed in the small gut creating problems for treatment.

Since the contents of Small intestine are fluid, severe symptoms are delayed for a long time ; eventually chronic obstruction ensues which may become acute. Acute obstruction is precipitated by impaction of a foreign body such as a fruit stone, a gall-stone or insipissated faeces.

Frequently, the ulcer infects the peritoneum in its acute phases and leads to the formation of peritoneal adhesions which may produce symptoms by traction upon the gut or by causing obstruction.

CLINICAL FEATURES :

Symptoms are due to :

(a) Those of the accompanying tuberculous lesion, like pulmonary Tuberculosis. These need not be discussed here.

(b) Those due to the tuberculous ulcers of the small gut. The causation of the symptoms are due to the *ulcerative processes* (and spasm of bowel) in the early stages and *due to the strictures* (bowel obstruction) in the later stages : — Diarrhoea, colicky pain, distension. Distension of abdomen (or a feeling of distension) relieved by vomiting is an important feature. History suggestive of complete obstruction relieved by "critical diarrhoea" (like critical diuresis) may come forth. The general health of the patient is much below par, patients being thin, wasted and anaemic, with evidence of hypoproteinemia the consequences of chronic inanition.

Diagnosis : is arrived at from the history of the case, symptoms and general and local examination of the patient. Lumps in the abdomen may be felt. Visible multidirectional (i.e. intestinal) peristalsis may be seen in the abdomen. Coexistant tuberculous lesions like lymphadenitis, pulmonary tuberculosis should be looked for.

Radiology : Plain X-ray of abdomen may show calcified lymph nodes. Barium mean examination (screening and serial pictures) may reveal strictures of small gut with multiple dilated coils of small intestine.

Treatment: General and Specific treatment are adopted. Local treatment is surgical. Surgical treatment is undertaken after suitable preparation of the patient. At Laparotomy for treatment of small intestinal tuberculosis, decision regarding the procedure adopted is not straightforward and easy. This is due to the fact that the strictures are multiple and placed at wide distances along the small gut, each being quite far away from the other. Excision of the entire portion of the gut to include all the strictures may not be feasible or even advisable for physiological regions in view of the anaemia and hypoproteinemia that may ensue if a large portion of small gut is excised. Apart from this physiological bar, these patients are not fit subjects for heroic or adventurous

surgery. It is not possible to generalise the procedure to be adopted. If the strictures are fortunately confined to a small portion of gut, excision of the segment to include all the strictures may be the ideal procedure. Upto about 1/3 of the length of the gut may be excised without grossly upsetting subsequent nutrition of the patient. The general condition of the patient should also be borne in mind, in deciding the step. If excision is decided against for one reason or other, the alternative lies in short-circuiting operation either single or multiple. There is one more procedure. If the strictures are few but far between, multiple local resections of affected segment and restoration of continuity by anastamosis is a sound procedure. These procedures must be guided by the general principles of intestinal resection and anastamosis. Not infrequently the surgeon may have to close the abdomen without performing any surgical procedure.

Out of the seven cases treated by the author, in two, resections including all the strictures were performed. In three cases short-circuiting operations were performed (Ileo-Ileostomy in two and ileo-transverse colostomy proximal to the proximal-most stricture in two cases. In one case, multiple (three) local resections and anastamosis were performed. All survived the operation.

HYPERTROPHIC ILEO-CAECAL TUBERCULOSIS :

As mentioned already, this is a 'primary' form of Tuberculosis and is usually non-ulcerative. The caecum shows thickening of its wall with extreme narrowing of its lumen and increase of subserous fat. The surface of caecum and terminal ileum may show tubercles of various sizes. The terminal ileum shows thickening of its wall and dilatation of the lumen. In some cases, the lumen of terminal ileum will be found to be larger than the lumen of large gut. Now and then small undermined ulcers may be seen in the mucous membrane of the caecum and terminal ileum. Occasionally, ulceration in the Ileo-caecal region seen in the small intestine in association with pulmonary tuberculosis.

Histopathology : Changes are seen in the sub-mucosal layer in the form of follicles consisting of epitheloid cells and a few giant-cells surrounded by a zone of lymphocytic and loose fibrous bands. Only a small percentage of cases show caseation. Occasionally, the histopathology is that of non-specific granuloma. The regional lymph nodes, however, show the usual tuberculous histopathology with caseation.

Clinical features : The most important symptom is *pain.* This pain is intermittent, colicky in nature and situated in the right iliac fossa or in lower abdomen. Distension and borborygmi may be coexistant symptoms. Loss of weight, anorexia are the other symptoms. Alternate constipation and diarrhoea may be noticed in some cases. A mass may be felt in the majority of cases, usually in the right iliac fossa, sometimes higher up due to foreshortening of the ascending colon and the consequent displacement upwards of the caecum. Cough, and haemoptysis, symptoms of pulmonary disease may be present in a small proportion of cases. The disease is at least as common in women as in men, if not more so, and in them, the onset of symptoms quite often follows a recent confinement. The disease is quite commonly seen in the Erskine Hospital, Madurai. During the last seven months, I have come across nine cases in Madurai, of which two had meningeal symptoms in addition to the ileo-caecal

lump, at the time of my first seeing them, and succumbed to the meningitis. The remaining seven were surgically treated. To this may be added four other cases treated surgically at Madras. Two of these four cases came for acute intestinal obstruction. Both these cases were treated by Ileo-transverse colostomy and survived the emergency operation. In addition to these, one case of perforated tuberculous ulcer of small intestine was treated by an emergency operation. *Physical examination* shows that the patients are usually poorly built with anaemia, and wasting. A palpable mass is left in right iliac fossa. Intestinal peristalsis with ladder pattern may be seen if there is sufficient degree of obstruction. I have come across one case with a faecal fistula in right iliac fossa, as a result of caecal tuberculosis. Enlarged lymph nodes may be palpable in some cases.

Investigations : Apart from routine Blood and Urine examination, B. S. R. will be helpful.

X-ray investigation : This is the most important diagnostic procedure. Contrast medium examination of alimentary tract must be done as routine in these cases. Its value is most important when the symptoms and signs are suggestive of the disease in the absence of a definite lump. Barium enema examination must be done first before a barium meal examination. A pilot plain X-ray picture may show calcification in the mesenteric lymph nodes. The changes that may be seen in Barium examination are :-

(a) Filling defect of caecum (Barium meal and Barium enema).

(b) Filling defect of ascending colon when that is involved.

(c) Non-filling of caecum and ascending colon with normal filling of terminal ileum and distal colon, on barium meal examination indicating a spastic condition of the non-filled area resulting from its ulceration — Stierline sign.

(d) Dilatation and delay in emptying of small gut.

(e) The terminal ileum ascends up from pelvis and joins the caecum at a very wide obtuse angle due to the ascent of caecum.

Other tuberculous lesions may be revealed on thorough clinical and radiological examination. This includes lungs, lymph nodes, genito-urinary system, bones and joints.

Treatment : Radical (excisional) surgery gives the best results wherever conditions permit such a procedure. For the Ileo-caecal tuberculosis, a right-sided hemicolectomy is ideal, provided ;

(a) the patient's general condition is satisfactory ;

(b) the mass is fairly mobile ; and,

(c) provided there is no gross obstruction.

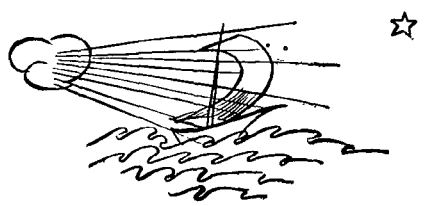
If these conditions cannot be satisfied, an ileo-transverse colostomy serves the interests of the patient best. The aim of surgery in the case of tuberculosis of alimentary tract is two-fold, i.e. removal of a tubercular focus and relief of the accompanying intestinal obstruction. Excisional surgery and restoration of continuity of alimentary tract serves both purposes excellently well. Patients improve in their general health very well and put on weight very rapidly following excisional surgery. If there had been another tuberculous focus in the body, following removal of one focus, the other focus also

improves rapidly. Ileo-transverse colostomy relieves obstruction and avoids the danger of a possible acute intestinal obstruction. This is the only procedure permissible when a patient with Ileo-caecal tuberculosis undergoes a laporotomy for a precipitated acute obstruction. For the treatment of tuberculoma, subsequently reliance must be placed on the specific anti-tuberculous drugs, and probably a second operation.

The Role of anti-tuberculous drugs : With the advent of streptomycin, INH and P. A. S., it may be thought that the necessity for operation may not arise since these drugs will bring about healing of the lesion. While these drugs are very valuable as an adjunct in the treatment of alimentary tuberculosis, they cannot replace them. Even if the drugs could bring about healing of tuberculous lesions, surgery is still the only remedy for relief of the other aspect of the problem, namely obstruction. It must be remembered, that tuberculous lesion in the alimentary tract heals by fibrosis ; and the mere healing of a lesion may lead to obstruction necessitating surgery.

In every case of intestinal tuberculosis (both types), general treatment with sanatorium regime, and administration of specific anti-tuberculous drugs must be followed. It is best to operate these cases, under what may be called a "Streptomycin Umbrella" — i.e. administration pre- and post-operatively.

Tuberculosis of the stomach and left half of colon are rare ; hence not discussed in detail. As an assistant I have come across two cases of tuberculous strictures of colon, one in the descending colon and one in the transverse colon.



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Nephritis

DR. H. A. SATYA JOSEPH, M.B.B.S., M.R.C.P. (EDIN.), D.T.M. & H. (EDIN.).

A FEW preliminary considerations about the kidney, its blood supply and functions before going on to the subject proper :-

The functional unit of renal tissue is the nephron of which there are millions in each kidney. The nephron is composed of the glomerulus and the tubule. The glomeruli are situated in the cortex and the tubules mostly in the medulla.

The Kidney possesses a large reserve of power and has the adaptability or power of varying the volume and composition of the urine to accord with the needs of the moment. Whether this adaptability is achieved by varying number of its functional units in action, or by varying the amount of work done by each unit is not definitely known.

Blood supply : The blood supply is first to the glomeruli by means of the afferent arterioles and through the glomeruli to the tubules by means of the efferent arterioles. Hence when there is an obstruction at the glumerular capillary level the tubules suffer from ischaemia.

Functions : The functions of the kidney may briefly be divided into :- (1) Glomerular function. (2) Tubular function, and (3) Intrinsic function.

Glomerular function is one of selective filtration and clearance. Glomerular Failure will therefore result in retention of metabolites, which are mainly acid radicals and nitrogenous substances, giving rise to acidosis and urea retention.

Tubular function is mainly reabsorption of water and base. Tubular failure will therefore result in dehydration, electrolyte loss and acidosis without any urea retention.

But it should be remembered that glomerular failure of some duration will cause secondary impairement of tubular function, and tubular failure will likewise cause glomerular dysfunction.

Intrinsic function consists of :-

(1) Formation of NH_4 which combines with the acid radicals to be excreted, thus sparing Na^+ and K^+ cations. Failure of this function will cause loss of Na^+ and K^+ cations resulting in acidosis.

(2) Ability to excrete acid phosphate radicals (NaH_2PO_4) when necessary so as to preserve base. Failure of this function will also result in acidosis.

Nephritis :

The term "nephritis" embraces a number of disease states, which may be acute or chronic and which are characterised by proteinuria, cylindruria and often by haematuria. In addition to these signs of local renal dysfunction, oedema, hypertension and nitrogen retention are frequently present.

Classification :

A simple classification of nephritis will be as follows :-

Glomerulonephritis
Acute focal nephritis.
Embolic nephritis.
Acute interstitial nephritis.

GLOMERULONEPHRITIS :

The term glomerulonephritis is used to describe a bilateral non-suppurative affection of the kidneys. It is uncertain whether it includes separate renal diseases or several stages of the same disease. Clinically, cases can usually be placed in one of three clearly defined categories — "Acute", "Subacute" and "Chronic" nephritis. There is great difficulty in correlating the morbid histology with the clinical findings. In the case of acute nephritis which is rapidly fatal, there is general agreement on the pathological features. In cases of acute nephritis which fail to recover completely, and in the clinical syndromes of subacute and chronic nephritis which are progressive over many years, the morbid histology depends upon the duration of the disease and the rate at which it has progressed. The fact that arterial hypertension may accompany some stage of any of these syndromes complicates the pathology still further by the changes due to arteriosclerosis. Consequently there have been many classifications of nephritis based on clinical and pathological findings. But the most recent classification is that of Ellis. From the evidence obtained from clinical and pathological observation of many cases of nephritis over a period of twenty years, Ellis was led to believe that acute and subacute nephritis are two entirely distinct diseases rather than stages of one disease process. In order to avoid further confusion of the nomenclature of nephritis, Ellis referred to them as Nephritis Type I and Type II, acknowledging the fact that both may proceed to the stage of chronic nephritis.

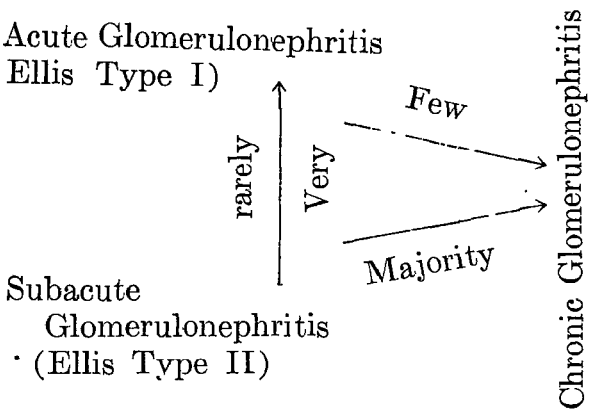
Therefore glomerulonephritis is classified into the following three distinct entities :-

Acute Glomerulonephritis or Ellis Type I.

Subacute Glomerulonephritis or Ellis Type II.

Chronic Glomerulonephritis.

And the interrelationship between them can be represented as follows :—



Acute Glomerulonephritis or Ellis Type I nephritis :

This condition is characterised by a diffuse inflamimation of the glomeruli of both kidneys.

Aetiology :

It is a disease of children and adolescents. There is an antecedent history of infection with haemolytic streptococcus (Group A of Lancefield), such as acute tonsillitis, sore throat or scarlet fever in about 85% of the cases. The history of infection is more easily obtained in children than in adults. A latent period is usual, nephritis developing 1 — 3 weeks after the subsidence of the infection.

Sometimes acute nephritis occurs after other infections like Pneumonia.

Acute nephritis can also occur as acute exacerbations in the course of chronic nephiritis.

The antecedent infection may be slight and may even pass unnoticed. There is no relationship between the severity of the infection and the probability of development of acute nephritis. It is not all cases of streptococcal sore throat that develop nephritis. The most virulent infection often clears up completely or it may cause only a mild nephritis. On the other hand, an infection so mild as to have almost escape notice may give rise to the most fulminant type of nephritis.

And there is also no relationship between the severity of the infection and the ultimate prognosis.

Pathology :

The earliest lesion is a diffuse inflammation of the glomerular capillaries with swelling and proliferation of the endothelium and accumulation of the products of inflammation in the tuft and the glomerular space. In progressive cases there is proliferation of the epithelium of Bowman's capsule and the formation of epithelial crescents which are believed to result from the organisation of haemorrhages into the capsule. Progressive fibrosis occurs and the glomerular capillaries become obstructed resulting in secondary degeneration of the tubules. Ultimately many nephrons may be replaced by fibrous tissue.

Pathogenesis :

The time lag between the streptococcal infection and the onset of nephritis, and the absence of bacteria in the renal lesions and the urine suggest that the disease is due to hypersensitivity.

This hypersensitivity is a diffuse process affecting all the capillaries and arterioles of the body ; but for some unknown and unaccountable reason the Kidney vessels suffer most. First there is spasm of the small arterioles giving rise to capillary ischaemia which damages the capillary wall increasing its permanently When the arteriolar spasm passes off an increased quantity of blood passes through the capillaries still more increasing capillary permeability and even rupturing the capillary wall in some cases. In the kidney, spasm of the glomerular afferent arteriole causes glomerular ischaemia and secondary disturbance of tubular nutrition. There is increased permeability of, and some rupture of the capillaries of the glomerulus resulting in the escape of blood and proteins into the Bowman's Capsule. And because of the disturbance of tubular nutrition there is shedding of tubular epithelium.

Clinical features :

The onset is usually sudden, the most constant features being puffiness of the face, oliguria and blood stained or smoky urine. In addition there may be the general signs and symptoms of an acute infection *viz* : malaise, fever, headache, anorexia and vomiting. Breathlessness due to pulmonary oedema may be present and epistaxis may occur.

Oedema round the ankles may be found if the patient is ambulant. In very severe cases there may be extensive pitting oedema and effusion into the serous sacs.

Urine :

There is oliguria, the daily output being between 300 and 700 cc. due to reduction of the glomerular filtration rate and increased tubular reabsorption. Com-

plete anuria may occur in severe cases. The urine is concentrated and appears red or smoky owing to the presence of blood. The specific gravity is high. There is variable proteinuria. Usually this is moderate, seldom exceeding 4 Gms. per litre. But the amount of protein present is out of proportion to the amount of haematuria and in severe cases the proteinuria is so great that the urine boils solid. Microscopic examination of the deposit reveals plenty of red blood cells, some leucocytes, and blood, epithelial and granular casts.

Heart :

Dilatation of the left ventricle may occur and a soft apical systolic murmur may be heard. The aortic second sound is accentuated.

Blood pressure :

Both the systolic and diastolic pressures are moderately raised.

Blood urea :

Rise in the blood urea level is common.

Fundus :

There is spasm of the retinal arterioles. In severe cases, haemorrhages, exudates and papilloedema may be seen.

Causation of oedema, in acute nephritis :

It used to be thought that the generalised oedema is due to generalised capillary damage. But now, the causation of oedema is believed to be due to the following four factors in the order of importance :-

(1) A decrease in urine formation due to decrease in glomerular filtration.

(2) Increase in the reabsorption of Sodium salts and water by the tubules.

(3) Congestive heart failure with elevation of hydrostatic pressure.

(4) Possibly an increased generalised Capillary permeability.

Course and prognosis :

(1) About 85% of the cases make a complete recovery. The oedema disappears, the blood pressure falls, the daily output of urine increases with disappearance of proteins, casts and cellular elements from the urine.

(2) In a few severe cases, which fortunately are rare, death may occur in a few days from cardiac failure, pulmonary oedema or uraemia.

(3) In a small number of cases, the symptoms and signs persist for weeks or even months and the patient dies from renal failure due to progressive destruction of the kidneys.

(4) In about 10% of cases, the hypertension and haematuria subside and the patient apparently regains normal health. Proteinuria however persists, and after many years chronic nephritis with hypertension and renal failure develops. Such a condition is called "Latent Nephritis."

(5) Very rarely acute nephritis progresses to subacute or Type II nephritis.

Complications of Acute nephritis :

The two main complications are hypertensive encephalopathy and anuria.

Treatment of Acute nephritis :

Prompt treatment lessens the occurrence of chronic nephritis.

Rest :

Rest in bed is essential for as long as it is necessary in order to obtain cure.

It may have to be for months in the less favourable cases. Rest is advisable as long as there is blood in the urine. But in "Latent Nephritis," i.e., in those cases in which proteinuria persists, in spite of apparent clinical cure, one has to use one's discretion in deciding as to when the patient can be let out of bed.

Diet :

Diet Should be a high calorie, low protein diet, consisting of not more than 20-40 Gms. of proteins per day to minimise the endogenous protein metabolism so that excretory work for the kidneys is at a minimum. The calorific value of the diet should be about 2500 calories. At first the diet should consist only of fruit juice and milk. After the acute phase passes off and haematuria subsides, solid food may be given and proteins may be increased up to 50 Gms. per day, provided the blood urea level is not raised.

Fluids :

Should be restricted as long as there is oliguria. The amount of fluid allowed per day is one litre plus the previous day's urinary output. (The one litre of fluid is to replace the 400 c.c. lost as invisible perspiration and the 600 c.c. lost in humidifying the expired air by a human being at rest during twenty four hours). When diuresis begins, fluid administration should be increased to match the urine volume.

Salt :

Salt should be severely restricted during the oliguric phase.

Antihistamines :

Antihistamines are of no use at this stage when nephritis has already set in. They might be useful if it is possible to give them at the stage of hypersensitivity and vasospasm.

ACTH and cortisone :

The general opinion is against the universal employment of these drugs in the treatment of acute nephritis. They may, however, be used in crises in select cases.

Antibiotics :

Penicillin should be administered in all cases. It should be given in doses of 500,000 units daily fill the urine is free from blood. Penicillin gets rid of residual infection and prevents the production of more sensitising antigen.

Removal of frankly infected tonsils or other septic foci should be delayed until convalescence is well advanced, as tonsillectomy, if carried out earlier may be followed by an exacerbation. However, in a few cases of acute nephritis with persistence or repeated recurrence of symptoms and signs, these features may disappear only after the removal of a focus of chronic infection. Any operative procedure should be carried out under adequate Pencillin cover.

Complications and their treatment :

Hypertensive encephalopathy is characterised by intense headache, restlessness, transient blindness or paresis, vomiting and epileptiform convulsions. These nervous symptoms are attributed to the hypertension, spasm of the cerebral vessels and cerebral oedema.

Treatment :

Watch for and anticipate the condition ; continuous record of the blood pressure and frequent fundus examinations for the detection of vasospasm should be made if we are to anticipate the occurrence of the condition. The following therapeutic map be carried out to treat the condition :-

(1) *Venesection* : Up to 500 c.c. of blood may be removed if the blood pressure is high.

(2) *Lumbar puncture* with the slow withdrawal of 30 — 40 c.c. of fluid.

(3) *Parenteral hexamethonium* in 25 — 50 mgm. doses.

(4) *Intravenus therapy with hypertonic solutions* : The effects of this therapy are reduction of blood pressure and production of diuresis. Diuresis occurs only in the absence of acidosis. May have therefore to adminster alkalis in addition, in order to induce diuresis.

The following hypertonic solutions are used :—

(a) 50 % Sucrose solution, to relieve the cerebral oedema. The dose is 1 c.c. per pound of body weight, the usual adult dose being 200 c.c.

(b) 50 % Glucose solution, the adult dose being 200 c.c. But Glucose sometimes produces a secondary rise of blood pressure.

(c) 40 c.c. of 30 % Sodium chloride solution.

Treatment of Anuria :

The aim of treatment is to regulate the fluid and electrolyte balance, to minimise protein metabolism, and to give a diet adequate in calories consisting only of carbohydrate and fat. This is achieved by giving the following mixture through a stomach tube over the 24 hours :—

Glucose	400 Gms.	= 1600 Calories
Peanutoil	100 Gms.	= 900 Calories
		Total 2500 Calories.

Acaciae enough to emulsify
Water to 1 litre.

A plastic tube is better than a rubber tube as it is less liable to be ejected if the patient vomits.

If there is vomiting, the vomit is collected, filtered and added to the drip.

Adequate sedation minimises vomiting. Sedation is induced by intramuscular paraldehyde before the intragastric tube is passed and it is continued either by 3 gr. of Sodium luminol intramuscularly or 1 c.c. of paraldehyde per hour added to the drip.

When the patient begins to pass urine, measure the amount of urine and allow that amount of extra water with the drip.

This method of treatment is known as the *Bulls regime*. This treatment should be continued until the urinary output is 500 — 1000 c.c. per day.

Under the Bull's regime, the daily increase of blood urea is only 17 mgms %. Otherwise the daily increase will be about 50 mgms. %.

The disadvantage of the emulsion used in the Bulls' regime is that it, being so rich in glucose, often produces troublesome vomiting in spite of adequate sedation. Hence the following modified emulsion (after *Brun*) is often used :—

Glucose	100 Gms.	= 400 Calories
Starch	200 Gms.	= 800 Calories
Peanut oil	100 Gms.	= 900 Calories
		Total 2100 Calories

Acacia enough to emulsify
Water to 1 litre.

If intragastric drip is not possible, 1000 c.c. of 50 % Glucose should be given into the Inferior Vena Cava by means of

a polythene catheler introduced through the leg vein. By giving it into the Vena Cava the danger of venous thrombosis is minimised.

In very severe cases, Ex-sanguino transfusions may be undertaken.

Rising potassium level in the blood will give rise to potassium intoxication which can be prevented by the following:-

(1) By avoiding exogenous potassium *Eg.* fruit juices.

(2) By the administration of intravenous glucose, and insulin at the rate of 1 unit for every 2 Gms. of glucose.

(3) By the administration of special sodium charged cation exchange resins either orally or in the form of enemata. Oral dose for a child is 5 Gms. daily and that for an adult 15 Gms. thrice daily.

Three methods to serve as emergency substitutes for functioning renal tissue are being studied. They are :—

(1) *Artificial Kidney* :

Heparinised blood runs from an artery through cellophane tubing where it is dialysed against a bath of approximately the electrolytic composition of normal extracellular fluid. Glucose is added to the bath to balance the osmotic pressure of the blood proteins and calcium salts are replaced by separate injections since they are not soluble in the usual dialysing fluid. The blood is reinjected after dialysis after the elimination of clots and bubbles. There are several types of artificial kidney and they have proved to be effective dialysers, eliminating moderate amounts of urea and other waste products from the body, with temporary symptomatic improvement. The disadvantages of the artificial kidney are the technical complexity requiring a trained team and the necessity for heparinisation with risk of bleeding.

(2) *Peritoneal dialysis* :

The peritoneum is perfused between two incisions with a solution similar to that used in the bath of the artificial kidney. It is temporarily effective in removing waste products and in restoring normal electrolyte balance. After continuous dialysis for a day or two, effectiveness decreases, peritonitis becomes more likely and colic or meterorism may occur.

(3) *Intestinal dialysis* :

The irrigation is done either through an isolated loop of the upper intestine or through a Miller-Abbott tube passed into the jejenum.

None of these methods has as yet been proved to affect the ultimate outcome of acute renal insufficiency. At the present time it would seem best to treat patients conservatively unless some pressing indication exists. But if it is decided to use any one of these three methods, the decision should be one of choice and not one made as a last resort after the failure of conservative and other methods of treatment.

SUBACUTE NEPHRITIS OR ELLIS TYPE II NEPHRITIS

Aetiology is unknown. It is more a disease of adults than of children and adolescents. There is a history of preceding infection in only less than 5% of the cases. Very rarely there is a history of its being preceded by Type I nephritis.

Pathology : Macroscopically, the kidneys are large and pale. After many years the kidneys become small, granular and fibrotic. Microscopically, the glomeruli

show slowly progressive hyalinisation and large amounts of doubly refractile lipoid are found in the epithelial cells of the renal tubules.

Clinical features : The onset is insidious. There are no constituitonal symptoms like fever, headache etc. The predominant features are generalised oedema which may be intermittent at first and gross proteinuria. The oedema involves first the subcutaneous tissue and latter the serous sacs and lungs. The general health is progressively impaired with increased liability to infection of the oedematous tissues or serous cavities. There may be normochromic or hypochromic anaemia.

Urine : The volume is normal or reduced. The specific gravity is normal. There is gross proteinuria and as much as 30 Gms. per litre may be passed. In the deposit, granular and hyaline casts are usually found ; red blood cells may or may not be found.

Heart : Usually no changes are found.

Blood pressure is not raised.

Fundus does not show any changes.

Blood urea level is not raised.

Blood cholesterol is markedly raised to 300-500 mgms.%.

Serum proteins : The total proteins are greatly reduced, but the chief reduction affects the serum albumin so that the albumin globulin ratio is reversed. There are qualitative changes in the various globulin fractions. As a result of the great reduction of serum albumin, there is a fall in the osmotic pressure of the serum and this is held to be responsible for the massive oedema which is the prominent clinical feature.

Course and prognosis : Apparent recovery occurs only in about 5% of cases of subacute nephritis. In the remainder, the oedema persists for months or years with occasional spontaneous but temporary remissions. Many die from exhaustion or from intercurrent infections to which their oedematons tissues are particularly susceptible. In those patients who neither recover nor die from intercurrent infection, a progressive destruction of kidney tissue takes place so that they ultimately develop chronic nephritis and renal insufficiency with hypertension and uraemia like the small number of persisting cases of Type I nephritis.

Some authorities believe that the proteinuria in Type II nephritis is not due to the leaking of ordinary plasma proteins through a damaged glomerular filter, but to the fact that the plasma proteins themselves have become altered in such a way as to render their excretion in the urine possible and necessary. In support of this view it is pointed out that the normal glomerulus which is impermeable to normal plasma proteins, promptly excretes proteins which are foreign to the plasma such as egg albumin, free haemoglobin and its derivatives and Bence-Jones protein even though their molecular weight is about the same as that of serum albumin. Such workers do not regard Type II nephritis as a kidney disease at all but rather as a humoral disorder characterised by a disturbance of protein metabolism and term the condition Nephrosis or Nephrotic syndrome. There is some evidence to show that the plasma and urinary proteins found in this condition differ from urinary proteins found in other kidney diseases. It is difficult, however, to believe that nephrosis is a clinical entity having nothing in common with other types of nephritis, since

many patients, if they do not recover or die from intercurrent infection , ultimately develop hypertension and die in uraemia as in chronic nephritis, and at autopsy the Kidneys are practically indistinguishable from those found in chronic nephritis.

Nephrotic Syndrome is the term usually applied to the association of hypoproteinaemia, oedema, proteinuria and hypercholesterolaemia. Examples of nephrotic syndrome are :—

(1) the lipoid nephrosis of children.

(2) the nephrotic syndrome of diabetes mellitus, otherwise known as the Kimmelstiel-Wilson syndrome.

(3) Amyloidosis.

(4) Chemical nephrosis due to poisoning with heavy metals like mercury, gold and lead.

(5) Compression or occlusion of the renal veins by enlarged para-aortic lymph nodes, by thrombus or as a result of the extension of a renal carcinoma along the renal vein.

All the conditions mentioned above as examples of the nephrotic syndrome should be considered in the differential diagnosis of subacute nephritis.

Treatment of subacute nephritis is mainly symptomatic with particular reference to relief of oedema and maintenance of the general health and of the patients' resistance to infection.

General health must be maintained by ensuring an adequate intake of Vitamins and iron, by removal of septic foci and by efficient treatment of intercurrent infections.

When there is no oedema there is no need for bed rest and the patient may be allowed to lead a nearly normal life with avoidance of excessive and exposure to wet and cold.

Diet : To make good the loss of protein in the urine, the diet should have a high protein content of 100 - 120 Gms. per day.

Fluids : There is no need to restrict fluids when there is no oedema.

Salt : There is no need to restrict salt either.

When there is oedema rest in bed is necessary.

Diet should contain at least 100 - 120 Gms. of protein per day.

Fluids should be restricted according to the degree of oedema and the urinary output. About 1 — 1½ litres a day may be allowed.

Salt should be severely restricted. It is the Sodium ion that matters and therefore care should be taken that no Sodium is given or taken otherwise.

For those who cannot tolerate a salt free diet, salt substitutes (which should be sodium substitutes) like Neoseloram or Citrofinal-N may be allowed.

For the relief of severe oedema, the following measures may be tried :—

(1) *Diuretics* : They are of limited value and any duiretic may be employed in turn Mercurials are probably the best. In certain cases urea in doses of 15 Gms. per day may prove an efficient diuretic

(2) *Administration of Blood and blood substitutes* : The effects are seldom more than temporary.

(a) Transfusion of whole blood.

(b) Intravenous administration of salt-free plasma albumin in doses of 50 Gms. per day for at least 6 days.

(c) Intravenous administration of Protein hydrolysates *Eg.* Casilan or Amigen.

(d) Intravenous administration of 6 % Dextran in doses of 380 c.c. (1 bottle) daily for 6 days.

(3) *Cortisone or ACTH* : The current dosage is 200 - 300 mgms. of cortisone per day for 5 - 15 days or 20 units of ACTH twice a day for the same period. The drugs are stopped when diuresis begins. Usually diuresis occurs only after cessation of therapy.

For the relieve of massive oedema, mechanical measures like Southey's tubes or paracentesis may have to be adapted.

CHRONIC GLOMERULONEPHRITIS

Chronic nephritis is not so much a disease entity as the end result of various types of kidney disease. Chronic nephritis is the end result of either Type I nephritis which has failed to subside, or Type II nephritis. In many cases however, a history of acute or subacute nephritis is not obtained. Chronic nephritis may also be produced by the toxaemias of pregnancy, by destructive lesions of the kidney due to pyelonephritis, polycystic disease, tumours, tuberculosis and by urinary obstruction due to stone, prostatism, stricture etc, and by malignant hypertension.

Pathology : Macroscopically, the kidneys is small and granular with the capsule adherent ; the peripelvic fat is increased and there is great reduction of parenchyma. The normal distinction between cortex and medulla is obscured.

Microscopically, there is filrous replacement of nephrons (both glomeruli and tubules) destroyed by inchaemia from arteriosclerosis or by the inflammation of a preceding acute nephritis. In the remaining nephrons, the glomeruli often show evidence of previous damage and the tubules are frequently dilated.

Pathogenesis : Continuing progressive fibrosis results in the diminution of the number of functioning nephrons. When the number of functioning nephrons is insufficient for the function of filtration and clearance, renal failure begins to occur. But this renal failure is combated by :— (1) a rise in blood pressure which increases the blood flow through the remaining functioning nephrons so that there is greater glomerular filtration through them, and (2) decreased tubular reabsorption so that there is polyuria, and because of the polyuria blood nitrogen level is not raised, provided sufficient water is drunk to keep up the polyuria. This constitutes the *compensated stage of chronic nephritis.*

As the disease progresses still further, the raised blood pressure is not enough or able to maintain glomerular filtration because of the progressive diminution in the number of functioning nephrons. In spite of polyuria blood nitrogen level begins to rise and uraemia occurs. This constitutes the *decompensated stage of chronic nephritis.*

Clinical features of chronic nephritis in the stage of compensation : A history of acute or subacute nephritis is obtained in many cases. Usually the patient first

seeks medical advice because of polyuria, thirst, loss of energy and weakness. There is no oedema. Anaemia is the main cause of loss of energy and weakness.

Urine : There is polyuria and there is approximation of the quantities of day and night urine. (Normally the night urine collected from 10 PM to 6 AM forms not more than a quarter to a third of the total daily output). The specific gravity is low, round about 1010, and there is approximation of the day and night, specific gravities of the urine due to impairement of the concentrating power of the kidneys. There is usually only a trace of protein in the urine because almost all the damaged glomeruli have been completely fibrosed and the fibrosed glameruli do not function at all so that there is no escape of protein through them. There may or may not be red blood cells in the urine. Hyaline and granular casts are found in small numbers.

Blood pressure is raised, particularly the diastolic pressure.

Heart shows leftsided enlargment.

Fundus shows arteriosclerotic changes; there may be haemorrhages, exudates or both.

Blood urea is not increased.

Blood cholesterol is normal.

Serum proteins are almost normal because the proteinuria is only slight.

Renal function tests are impaired.

Clinical features of decompensated chronic nephritis : In addition to the polyuria, thirst, loss of energy and weakness, the patient may complain of nausea, vomiting and diarrhoea due to uraemia which develops as a result of renal failure.

The blood pressure may be markedly raised, and the heart may begin to show signs of failure as a result of which there may be slight oedema.

The Fundus may show papilloedema.

Blood urea is very much raised, sometimes up to more than 300 mgms. %.

Blood creatinine is raised to 3.5 mgms. or often more. There is marked acidosis as shown by the reduced CO_2 combining power of the blood.

Renal function tests show a marked degree of impairement.

Course and prognosis of chronic nephritis : The disease progresses steadily over months or years to a fatal termination. The course may be punctuated by exacerbations of acute nephritis which hastens the progress of the disease to its inevitable outcome. When nitrogen retention and acidosis are severe, the outlook is grave and most patients die within a few months or a year. Papilloedema is a bad prognostic sign and most patients who show it die within 2 years. The cause of death generally is uraemia ; in other cases the patient dies from a cerebrovascular accident or cardiac failure.

Treatment of compensated chronic nephritis :

Every effort should be made to maintain the patient in compensation for as long as possible. Routine regular exami-

nations for the early detection of dehydration and electrolyte loss, concomitant urinary obstruction and infection are essential, and when found, should be promptly and properly treated because these conditions will hasten decompensation if not corrected in time.

Diet : The diet should have a high calorific value, but should not contain more than 75 Gms. of protein per day. But as the disease progresses towards decompensation, the amount of protein should be progressively reduced to 20 - 40 Gms. per day.

Fluids should not be restricted, as compensation by polyuria must be maintained as long as possible.

Salt : There should be a full intake of salt ; otherwise there will be salt depletion due to polyuria and impaired tubular reabsorption and this will hasten decompensation.

Potassium in any form should be forbidden in order to prevent hyperkalaemia. Fruit juices are particularly to be avoided.

Acidosis should be counteracted either by he oral administration of alkalies (Sodium salts only), or by the intravenous administration of 300-500 c.c. of 1/6th molar Sodium Lactate, or 30 - 50 c.c. 8% Sodium bicarbonate administered along with 500 c.c. of normal saline to avoid the possibility of sloughing.

Calcium salts should be administered along with the alkalis to prevent tetany which may be induced by the alkalosis.

Treatment of decompensated chronic nephritis : There is nothing that can be done for the patient after decompensation has set in. The best thing will be to admit defeat and merely try to maintain the patient in as reasonable a state of health as possible till death puts an end to all his or her troubles.

And finally, a few words about the other three types of nephritis :—

Acute focal nephritis is diagnosed when haematuria without any of the other signs of acute glomerulonephritis occurs at the height of an infection especially streptococcal tonsillitis.

The prognosis has been said to be uniformly good, but a reassessment of cases after a long interval of time has shown that the prognosis is similar to that of acute glomerulonephritis. It is doubtful therefore, whether it is justifiable to accept acute focal nephritis as an entity.

Embolic nephritis occurs during the course of subacute bacterial endocarditis. The only manifestation of the renal lesion is haematuria.

Acute iterstitial nephritis : This constitutes an anatomic rather than a clinical entity, characterised by diffuse infiltration of the renal interstitial tissue with red blood cells, polymorphs and other leucocytes. Oedema of the tissues may be present. Thre are no characteristic glomerular or tubular lesions. This disorder of the kidneys is associated with severe sepsis, diphtheria or scarlet fever and constitutes a postmortem finding without known clinical significance.

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Some Factors and Problems of Human Growth

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IT might be stated at the very outset that it is not proposed in this article to go into a lengthy discussion on the precise definition of biological growth. The difficulties confronting such an attempt and a historical review of suggested definitions have been alluded to by many authors (Child, 1941). It would suffice for our purpose to mention that biological growth is not mere increase in size as in the case of an iceberg, nor even an increase in size with a definite pattern and shape as in a growing crystal. Biological growth is the regulated increase or decrease in size of the whole and the constituent parts of a developing or maturing organism towards a predetermined end result of approximately constant adult form. This process involves segmentation and differentiation of cells and a coordinated relative increase or decrease of the size of its parts. Simple cellular inclusions of nutritive or metabolic material or water or other substances is not to be considered as biological growth.

LIFE, GROWTH AND REPRODUCTION

Whenever and wherever life might have first originated on this planet, individual bits of living matter became capable of being aware of the environment and of utilising substances found in the environment towards their continued sustenance. Also living things were inherently incapable of sustaining themselves beyond a limited range of physical and chemical changes in the environment. The elaboration of sense organs for better sensory perception towards a fuller awareness was favoured by increase in size and complexity of design. But increase in mass was, beyond a stage, incampatible with metabolism necessary for sustenance. Hence division or reproduction became a necessity. This, however, endowed such a living organism with the advantage of dispersing its own fragments into a variety of environments, so that if the environment at any one place altered deleteriously leading to its destruction, elsewhere under a more congen-

ial setting a dispersed part it would be able to thrive. Thus growth and reproduction became inherent in living matter even from the very beginning and no living entity escapes these eventualities irrespective of the duration of growth and the mode of reproduction.

HUMAN GROWTH

From a single cell, the zygote or fertilised ovum, the embryo develops. The foetus inside the uterus and after birth the young one grows to the maturity of adulthood. The rates of growth at different stages of this growth process are varied; and the rates of growth of different tissues or organs are also diverse. For any particular character its measurement could be plotted against time to obtain a growth curve. This shows a slow beginning, a subsequent rise to a peak and a flattening out in a stabilised position. In a similar manner the rate of growth could be plotted against time; or the velocity of the growth change or the logarithm of the velocity could be so represented.

Likewise the relative growth proportion between a part and the whole organism or between two separate parts could be studied by a linear correlation. Also a more comprehensive idea of the overall picture of growth could be deduced by a representation based on "Cartesian" coordinates. It is not proposed to go into details of these considerations here.

FACTORS WHICH INFLUENCE GROWTH

Apart from race, sex and genetic makeup, environment, diet, exercise, work, hormones, socio-economic status etc. are important factors that have a marked influence on growth. Really speaking there are no acceptable standards of normal growth which can be applid straightaway to any individual. There is a considerable amount of variation even between normal people depending on constitutional type and individual uniqueness which is a basic biological fact. A growth curve which may be regarded as of some value is a grid or a wide pathway on the graph paper about which the individual will be somewhere located in time sequence. Such grids will have to be constructed from measurements of a large number of healthy people of the particular racial and social stratum if they are to be of any value. We do not have such standard grids yet worked out for the growing children or young adults in India. Hence any medical man would be illadvised to pronounce judgement of retarded growth as regards children or adolescents or adults on comparison with data for western countries. The application of such hypothetical western standards to Indians in medical examination for recruitment to services or insurance has often lead to grave errors. It is necessary to give this warning both to the examining medical personnel and the authorities concerned

who demand the prescribing of such false normal standards of physical make up totally inapplicable to our people.

THE GENERAL GROWTH PATTERN

It will be obvious to a student of growth that all biological growth whether in plant or animal, typically exemplifies distinct periods of 'growing up,' 'filling up' and comparative quiescence occurring in some kind of cyclical manner. In human growth there are rapid spurts of growth during infancy and adolescence with a less marked transient spurt at about the 6th year. This seems to indicate that metabolic background of the "growing up"; stage is slightly different from that of "filling up"; and the organism appears to be incapable of performing both the tasks at one and the same time and hence alternates these two metabolic phases.

GROWTH PATTERNS OF DIFFERENT ORGAN SYSTEMS

Apart from the general growth pattern there are three groups of organ systems whose growth presents characteristic periods of acceleration at different ages. These are (i) the nervous system and sense organs, maturing earliest, (ii) the lymphatic system, slightly later and (iii) the reproductive and locomotor system, during adolescence.

A child of two has acquired the total number of nerve cells possessed by the individual, and at the age of six the cells have attained their maximum size and at the age of twelve the head has possibly attained its final size. The nervous system being the most specialised is the earliest to reach maturity. The equipment necessary for a full appreciation and awareness of the environment is the first necessity for an individual.

The lymphatic system as represented by the thymus, tonsil and other lymphatic aggregations reaches its maximum size much prior to puberty and seems to undergo involution subsequently. This period is the time of establishment of active immunity for protecting the individual against microbic enemies in the environment.

Then during adolescence takes place the spurt of growth of the locomotor system and the reproductive system, the one necessary for the struggle for individual existance and the other for the perpetuation of the species.

The abovementioned triple phasic stages of growth become understandable in the biological economy of human life as phases of 1. elaboration of awareness of the environment 2. biological defence and 3. the advent of adulthood for the fruition of individual life and the perpetuation of the species.

WHY DO GIRLS ATTAIN PUBERTY EARLIER?

This question has been frequently posed and left unanswered. It is

generally admitted that physical and sexual maturation occurs in the adolescent female at an age about two years earlier than the age when it is established in the male. Any suggestions that could be offered to an understanding of this biological problem would be worthwhile being considered for a scientific scrutiny.

There is no evidence forthcoming that the time taken for sex maturation and commencement of reproduction is different in the male and female in any nonmammalian vertebrate, say fish or amphibian or reptile or bird. If this then be a mammalian feature and especially well noticeable in the human, could it be that the stay of the youngone inside the mother for a comparatively long period of nine months has been responsible for this difference. To explain this further, if it be assumed that genetically the time necessary for male or female gonadal maturation be hypothetically of equal duration, the female child during the period of gestation gets a full impact of the hormonal complex of the adult pregnant female, while the male child inside the mother relceives, in spite of the genetic initiation of its basic gonadal development, a feminine hormonal bias as it dwells inside the hormonal complex of a female individual, its own mother. This gives the male child a handicap in its sex maturation. It has to circumvent this handicap during a varying period of postnatal life before it can start on its own slow sexual unfolding. Recent work carried out at the Institute of Anatomy has shown (Patangia, 1956) that the epithelium of the uterus masculinus and certain other adjacent regions of the urogenital system of male foetuses and neonati show metaplasia similar to the vaginal epithelium of female foetus. To cite a parallel, the mastitis of new born is a pointer to maternal hormones influencing the child. We presume that the mother during pregnancy exerts through her hormonal complex a feminine bias in the differentiation of the reproductive system leading to a positive start being conferred on the female child and a negative handicap being imposed on the male child. This Prenatal bias has an abiding influence during the post natal period and is probably the cause of the earlier age of puberty in girls. The suggestion made here towards the understanding of this problem is being presented for the first time.

Yet another passing argument suggests itself relevant to this problem. For some reasons not yet fully undrstood the male sex cells find a congenial environment only in the cooler temperature of the testes situated in the scrotal pouch as contrasted with the female sex cells of the ovary located within the abdomen. This slight difference in environmental temperature might perhaps have a metabolic effect in

accelerating or retarding the rate of growth of the maturing gonad. This also happens to be an original suggestion. It might be stated as regards this problem that there is no evidence to suggest that the hypophysis in the female or the male indicates a primary advance or lag in time in the initiation of the augmented production of gonadotropic hormones incidental to the commencement of adolescence.

ARE THERE SEASONAL CYCLES OF GROWTH ?

Another interesting question is whether cycles of natural units of time have any influence on human growth. These cycles are (1) the diurnal cycle of day and night depending on the earth's revolution with its variations of light and temperature and humidity and the consequently impressed habits on man regarding feeding, rest and sleep, (2) the monthly cycle related to the course of the moon, (3) the yearly cycle with its pageant of seasons with variations in heat, light and rain, and (4) a grouped many-year cycles like the sun- spot cycles of 11 - 12 years which strangely enough coincide with the period of Jupiter's circumambulation round the sun, and (5) even larger cycles of terrestrial or astronomical phenomena about whose influence we have little precise knowledge.

There is every reason to assume on general grounds that in this vast interdependent universe no material object could be sequestered from the influences of the environment, near and far. The difficulty is to prove precisely that correlated changes do exist. How far is human growth affected by such influences ?

There is a diurnal variation in the rate of growth of many organisms. Plant tendrils and shoots and roots have been observed to grow more during certain times of the 24 hour day and less at other times. Similarly children are also said to show a comparable diurnal variation in growth rate, growing more during night and early morning. The monthly cycle of the moon affects all biological life including human growth and has left its impression woman's menstrual cycle. This does not need elaboration here. The various seasons of the year affect growth due to the change in environmental factors; and this variation in growth velocity appears as a superimposed pattern over the basic general growth curve for any individual. There are also certain indications of a more profound change dependent upon the yearly seasons apart from predictable influences due to change in environment as food or temperature or light. Even when environmental factors are to a large extent eliminated there is an increase in the rate of growth of children in late spring and summer and autumn. Whether the terminal part of this phase is a phy-

siological vestige of an ancestral hibernating habit of the stock from which man was derived is a question that can be raised with out venturing on a positive answer.

Then the further question arises whether beyond the annual seasonal cycle any of nature's time units of many-year epochs do have any influence on human growth. The concentric rings of the cross sections of stems of trees are said to indicate definitely the sun-spot cycles of about 11 years. Whether the sun-spot cycles on the sun are correlated to the movements of the planet Jupitar, as the tides of our seas are linked with the movements of the sun and moon, is a question which I can raise without attempting any answer. Do the lives of animals, which subsist on plant life directly or indirectly, also show variations in growth corresponding to sun-spot cycles of eleven years is a query whose answer is yet to be determined. It will be extremely unscientific to accept hypothetical possibilities on insufficient evidence; it will be more unscientific to deny the possibility altogether. One can only keep an open mind, seeking more knowledge.

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Cancer Diagnosis

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"DELAY is Cancers's best ally". — This statement expresses in unequivocal terms the importance of early diagnosis which is very necessary for a high survival rate in cancer cases. It was assumed that cancer in the last stages required no skilled diagnosis because no treatment was possible. But we know at present that even some of the advanced cases of cancer with extensive metastases can be made to undergo retrogression or the patients condition made less miserable if the diagnosis regarding the nature of the cancer is established and appropriate method of treatment either in the form of endocrine or isotope therapy is instituted. Therefore, it is important that cancer should be diagnosed irrespective of its stage of its growth. The knowledge gained from correct diagnosis will enable us to properly assess the biological behaviour of the neoplasm and thus ensure the benefits of modern advances to the cancer patient.

In some of the leading countries great strides have been made to educate the people. They are taught to come to the treatment centre for early diagnosis. The knowledge of cancer is freely imparted even in the schools. This procedure may be criticised by some as causing unnecessary panic in the minds of the public. Apart from causing panic, this has enlightened the public and has paid high dividends in the form of higher survival rates among the population. It is high time that we take up similar measures in our country and organise proper administrative machinery for enlightening the people, for instructing the profession and in addition to help the profession in affording relief to those afflicted with cancer. Free diagnostic centres and free treatment centres should be established in most of the major towns and every attempt should be made to attract cancer patients to these centres and such patients should be given every assurance that this disease is being carefully investigated and treated in these centres. Want of free diagnostic facilities necessitates the physician to adopt methods of trial and error and in many cases valuable time is lost in arriving at a proper diagnosis and instituting the proper therapy at the early stage.

The object of this paper is to place before the profession certain relevant points regarding the diagnosis of cancer. The profession is well aware of the clinical features of the disease and hence it is considered redundant to dwell upon these aspects of the problem. So it is proposed to lay stress on the laboratory diagnosis of cancer. Even the best of clinicians amongst you may recall instances where it was difficult to differentiate a malignant lesion from an inflammatory one. It is no shame to confess that even on an autopsy table this problem confronts an experienced pathologist — often times one mistakes a milliary carcinomatosis for a milliary tuberculosis and vice versa. As one becomes more and more experienced, he becomes non-committal and employs

methods which give reasonably accurate scientific diagnosis. A laboratory is an important adjuvant to the scientific physician, he should never feel shy to make use of a laboratory. It is considered by some that laboratories are luxuries but the time and money saved and also the immense good done to patient by a correct diagnosis will justify the role of a laboratory service.

What are the possible ways in which you can make use of the laboratory in cancer diagnosis ? When one is faced with a case of cancer he studies the tumour in relation to its anatomical disposition The study is incomplete without knowing its histological nature. A biopsy is necessary for this and one has to obtain the material for microscopic study. This material is obtained by (I) Closed biopsy (II) Open biopsy.

CLOSED BIOPSY. (*Aspiration biopsy*)

A sample of the lesion is obtained by aspiration with a syringe and needle. The site to be punctured is anaesthetised preferably with novocaine infiltration and a samll incision is made on the skin through which is inserted a large-bore needle (18-gauge 5-12 cms. long) attached to a large syringe. This incision in the skin is necessary otherwise the skin along the course of the needle puncture is taken into the bore of the needle and thus prevents a successful aspiration biopsy. Having traversed a sufficient depth of the tumour the needle is given a jerky tilt so that the column of tumour cells gets detached from the rest of the tumour and lies in the bore of the needle. The next manipulation is important. The needle is withdrawn and during the withdrawal suction is applied on the piston of the syringe (fig. i & ii) and this ensures the retention of the tumour material in the needle or carries it into the barrel of the syringe. With a little practice more tissue can be obtained by changing the direction of the needle twice or thrice and repeating the procedure through the same incision in the skin. The material thus obtained is squirted into a tube containing a fixative (10% formaldehyde) and sent to the laboratory for processing and report. The site of the puncture is carefully chosen avoiding important anatomical structures and at the same time assuring the entry of the needle into the tumour proper. If the aspirated material is mainly fluid an immediate smear is made, fixed in formaldehyde and stained with haematoxylin and eosin. Another simple method of examination of the fluid is by centrifuging the aspirated fluid transfer a drop of the centrifuged deposit on to a slide, cover with a coverslip and then gently introduce a drop of 0.5% Toluidine blue under the coverslip. On examining the preparation under the microscope the cell details are very well made out. This method is simpler than Papanicolau's and give reliable information. Where a set up for Papanicolau's cytological technique is available the material can be studied using that staining method. The deposit in the centrifuge tube should be treated with 20 volumes of a fixative and material sent to the laboratory for paraffin blocking, sectioning and preparation of sections similar to routine histological study.

OPEN BIOPSY

The site of the tumour is prepared for an open operation under aseptic conditions. Depending upon the site and age of the patient a general or local anaesthesia is used. In the young it is preferable to give a general anaesthesia. A local or regional block anaesthesia will be ideal in a patient who co-operates. It is

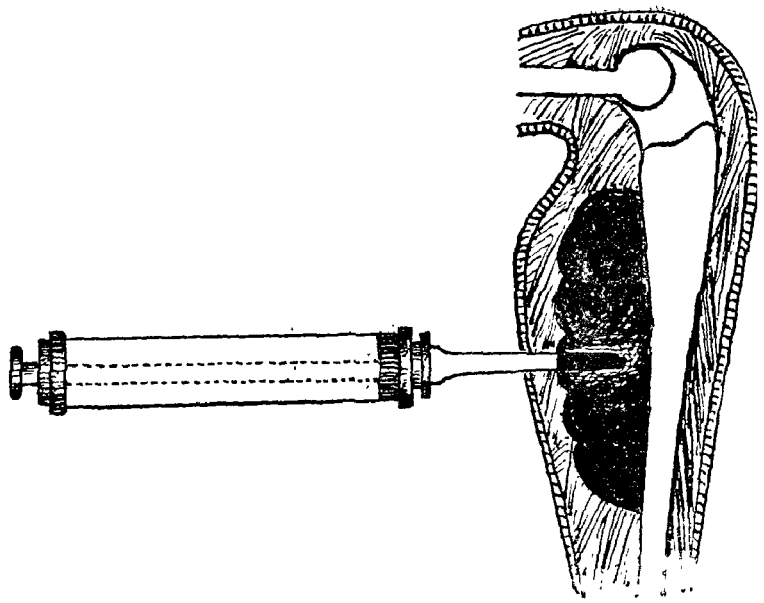


FIGURE 1

Aspiration needle in situ

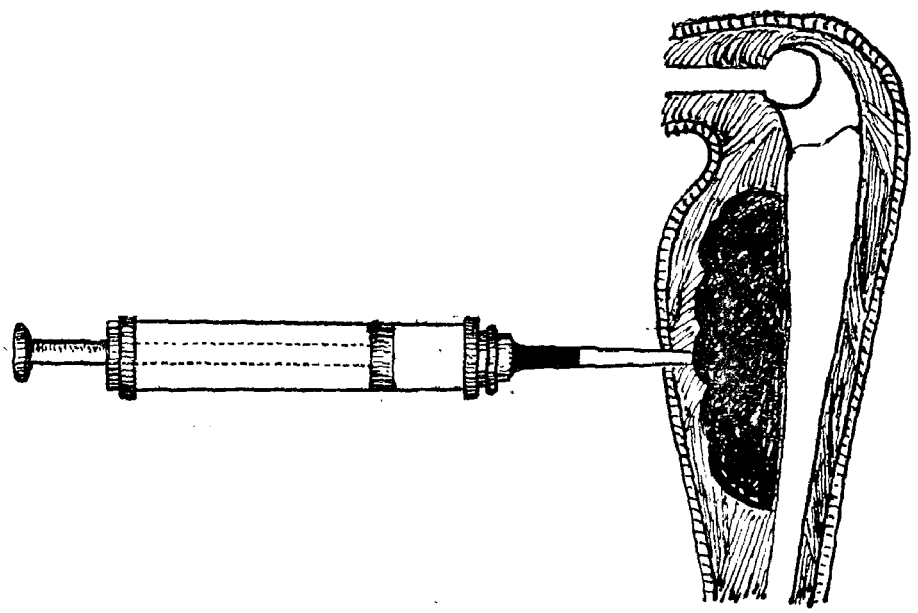


FIGURE 2

Aspiration needle as it is being withdrawn. Note aspirated biopsy material in the needle nearer to the syringe.

better to do a block because local anaesthesia distorts the histology of the tumour. Where the tumour is small the entire mass may be excised for biopsy. This is fixed in 10% formaldehyde (25 cc of commercial formalin + 75 cc. of normal saline and neutralised with lithium carbonate). The material is dropped into a wide-mouthed bottle and 25 times its volume of fixative is added, sealed water-tight, properly labelled and despatched to laboratory.

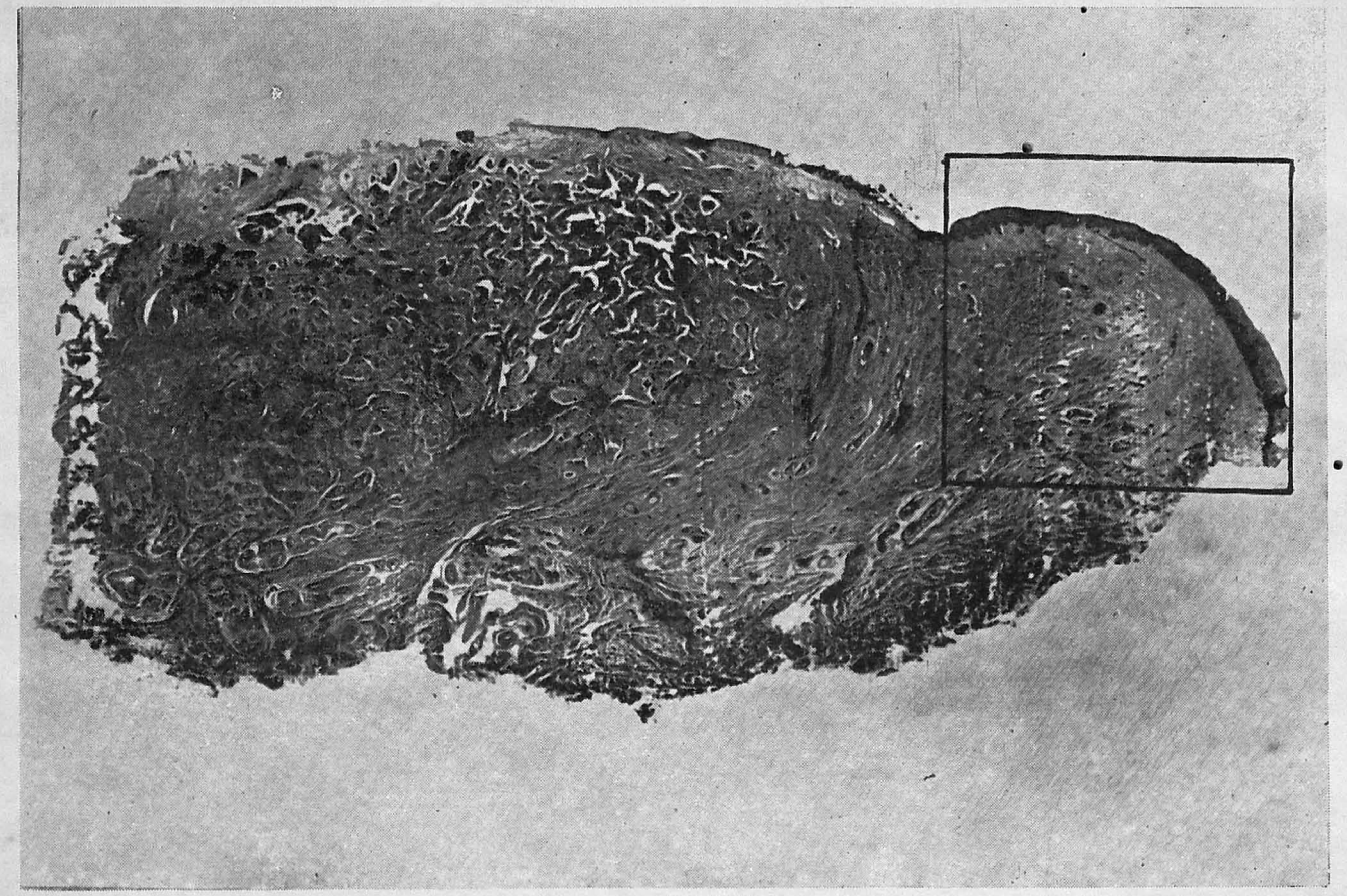
Contra indications for open biopsy :—

(a) Infected lesions — The infection should be controlled by antibiotics and then a biopsy attempted.

(b) Vascularity of tumour — Where the tumour is highly vascular, it is safe to attempt a closed biopsy unless one is prepared to undertake a major surgical operation like amputation or a radical excision etc. A case of chondrosarcoma in the upper end of the tibia was opened into for doing a biopsy and in this case an amputation through the middle of the high had to be undertaken as an emergency to control haemorrhage which could not be controlled otherwise.

It is possible to get a laboratory report on such material within 10 minutes provided a frozen section unit is attached to an operation theatre. A patient may be prepared for a radical operation and if the report on the biopsy under local anaesthesia warrants a radical procedure, the surgeon can proceed with the major operation with a break of 10 minutes. The question of dissemination of the tumour by biopsy is appealing to only the uninitiated in oncology for, we know that a malignant tumour will have metastases when the patient comes to us and the biopsy interference undertaken in the interest of the patient does in no way aggravate the existing condition. Another point worth stressing is that no deep X-ray therapy should precede a biopsy for such treatments seriously interfere with the correct interpretation of the histology of the tumour.

There are no hard and fast rules regarding the nature of lesion and the type of biopsy (open or closed) to be done. The guiding principle is to obtain maximum information from any procedure which could guarantee minimum trauma and inconvenience to the patient. Again the amount of tissue to be obtained is based upon whether enough material could be obtained by a closed biopsy. Even in an open biopsy the nature of the lesion may not be identified if the tissue obtained is composed of necrotic material or the tissue is obtained from the superficial part of the tumour when perhaps a misleading information such as 'chronic inflammatory changes only" may be given due to study of the capsular tissue of the tumour which shows connective tissue with round cell infiltration. A single lymphnode obtained by an open biopsy may not show the lesion and it is ideal to remove a group of lymph nodes for histopathological study. It is also worthwhile remembering that in a case of generalised lymphadenitis, the group of lymph nodes to be removed for study is either lower cervical or axillary because the upper cervical draining the mouth and the inguinal draining the bare-footed lower limb often show inflammatory reaction of a non-specific nature. In open ulcerative type of lesions the tendency is to take the material from the surface which contains only necrotic tissue. Hence, cut out a wedge-shaped piece of tissue from the periphery and this will contain both



Carcinoma cervix — First biopsy done from the area enclosed in the square was negative. The growth was not in the portio vaginalis. It was deeper, infiltrating into the rest of the cervix.

the normal and the neoplastic area which is suitable for study.

The infiltration anaesthesia should be injected a little away from the site of the tissue because this may distort the tumour cells. A cold knife should be used and the temptation to use a cautery should be resisted because tissue thus obtained shows charred material and reveal no histological detail.

While sending the material for histological report it is essential that complete detail of the case should be sent. Very often you will find that your diagnosis is correct while the laboratory diagnosis does not help you. In a case, a piece of tissue for biopsy was sent with a clinical history very typical of cancer. The biopsy material received did not show any evidence of carcinoma. So a suggestion that the case may be one of endocervical carcinoma was made in the report which was otherwise not helpful. The gynaecologist made a careful examination and found a growth in the endocervical region and did a hysterectomy. (Fig. 3) Shows the section from the cervix in this case illustrating the reason for a negative result in the tissue obtained from the portio vaginalis of the cervix. A correct, sincere and sympathetic understanding of the problem between the physician and the pathologist may enable the solution of a number of difficult cases and the patient will be immensely benefitted. The spirit of team work should prevail and whenever an inclusive report is received from the laboratory the physician should be ready for a discussion with the pathologist. Every case of cancer should be reviewed in the presence of a radiologist, a pathologist, a surgeon and a physician. Inadequate and incorrect reports emnate from laboratories as a result of meagre data furnished along with the material sent to the laboratory. A team of workers as suggested with full understanding of the case can do better work. The phycician will be benefitted by the consel of the radiologist and pathologist in doing a biopsy and also in prescribing the treatment.

So far reference was made to biopsy in a general way. There are specialised precedures in diagnosis — exfoliative cytology applied to the study of secretions in the body (vaginal secretion, urine, faeces, sputum, gastric juice, synovial and cerebrospinal fluid), endoscopic examination and biopsy or wash (bronchial washes), tissue culture and inoculation into the arterior chamber or cortex of the laboratory animals and tracer studies with isotopes. Each procedure has its limitations, but an understanding physician can obtain very accurate and reliable results.

Exfoliative cytology when correctly interpreted gives very satisfactory results. Tiny pieces of tissue obtained through a bronchoscope or gastroscope or cystoscope requires a careful study with the full knowledge of the case. Material thus obtained should be immediately fixed in preservative, otherwise such small pieces undergo drying or decomposition with even a slight delay. Washings obtained from endoscopic examinations can yield useful information in the hands of the experienced laboratory worker. Such fluids should be despatched immediately or adequate amount of fixative should be added immediately to the cells lest the cells should get lysed. Tissue culture diagnosis though of academic interest helps in knowing the histogenesis of the tumour and deciding the therapy to be followed. The principle that rapidly dividing tumour cells concentrate substances like

P_{32}, has been used in diagnosis, but this requires an elaborate nuclear-physics laboratory. Some of the tumours of thyroid show functional hyperactivity and exhibit increased I_{131} concentration in such areas. This principle has been made use of in diagnosis and also in treatment. Chemical and biological assay of enzymes and hormones help us in detecting neoplasms of endocrine and generative organs. Whether an evacuated invasive type of vesicular mole had been completely removed or whether it has assumed any malignant propensities can be assayed by such biological and quantitative tests as Aschheim—Zondek, Xenopus toad and Friedmann's tests. These tests can also be made use of in the study of testicular neoplasms. Enzyme estimation in cases of prostatic and parathyroid tumours help us in diagnosis and also in correctly gauging the activity of these neoplasms.

The vista of laboratory diagnosis is as wide as the horizon and this attempt to cover a few essentials is by no means complete. The field is vast and full of benefit to the scientific physician. One need not grow every human neoplasm in the rabbit's anterior chamber or inside its cranial cavity and then the express its benign or malignant nature (though this may be justified in debetable tumours), but it is essential that the correct conception of diagnosis and treatment should be based on unassailable laboratory data.

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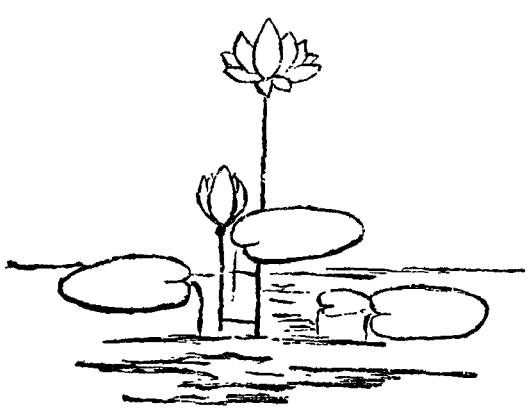

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"Diagnosis and Management of Hepatic Failure"

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Madurai Medical College, Madurai.

IN this article I am making an attempt to bring out the present day treatment and line of approach of hepatic failure complicating, acute or chronic liver disease. I shall be strictly confining only to the cases that are come across in day-to-day practice. Certain fundamental concepts have to be discussed before we actually start the subject.

A diseased liver is different from one having failure. A liver may be diseased yet the hepatic functions may not be altered significantly to call a liver is failing in its functions. A diseased liver may ultimately fail if left untreated or unrecognised. This failure may be acute or chronic. For example, in the common disease, the cirrhosis liver (portal cirrhosis) there exists a various evolutionary phases. Simple steotosis, steato cirrhosis, steato-necrosis and cirrhosis. In these stages only minor impairement of liver functions occur in the early phases. However in a diseased liver hepatic failure will set in sooner or later. So the aim of the treatment of liver disease is to protect the normal portion (uninjured portion) during the various phases of the disease process, so that the liver can function normally after the recovery. If in our attempt we fail, the hepatic failure succeeds, and if we succeed the hepatic failure fails to appear and ultimately the uninjured cells escape out of the danger zone and start regenerating. Liver, as we all know, has got an enormous functional capacity for regeneration. It is said even if 70% of the liver cells are removed or damaged, the 30% of liver cells left, can regenerate to the full extent and can take up the cent per cent function of an uninjured liver. This is what happens in Infective Hepatitis. Most of the liver cells are damaged by the Infective Hepatitis virus of S. H. (Serum Hepatitis) virus, ultimately the liver recovers completely. Of course, various other factors that are detremental to the regeneration like adequate blood flow to liver after injury and free secretion and excretion of bile without any obstruction are necessary about which we are not concerned with in the present article.

They are yet another group of liver diseases which are present say i.e., amoebic abscess liver, hydatic cyst liver, Gumma liver, though are disease entities they do not impair the hepatic function to a significant extent so as to lead on to an acute or chronic form of hepatic failure. It is this one should distinguish clearly between hepatic diseases and hepatic failure which may or may not occur during the course of the disease.

The hepatic failure can occur in an acute, subacute, or chronic form, in the diseases that are very often met with in day-to-day practice. Among infants and children the two things are Erythro-blastosis foetalis or Icterus gravis neonatorum and the infantile biliary cirrhosis. Of the two we are more often confronted with failure in biliary cirrhosis. Portal cirrhosis also occurs in children and can be complicated by an acute hepatic failure due to a haemorrhage of an oesophageal varices or portal thrombosis.

In the young adults metalic poisoning or toxic jaundice due to drugs are ascribed as liver poisons and produce zonal, focal or cerebral necrosis. We do not come across the toxicity of most of the drugs in clinical practice. Very often probably the degree of damage is in a subclinical level or other manifestations draw our notice and this overlooked. Copper sulphate poisoning is one such condition which occurs fairly commonly. Immediately after admission into hospital they do not show any evidence of liver damage. In cases which react badly to the copper sulphate intake they show severe abdominal pain in the second day and some times pass blood in vomiting and motion for first one or two days. In the second or third day though they may be apparently normal they develop jaundice and show bile in urine ; progressive and rapid deepening of jaundice occurs and they go into hepatic coma from which they cannot be restored back to normal.

Some persons who develop toxic jaundice secondary to some other infections, say pneumonia etc., though the liver is involved in the disease thep do not go into or show signs and symptoms of hepatic failure. Another important acute disease in the adult is viral hepatitis. Most of the liver cells are damaged by the diseased process, yet the liver functions are not completely and permanently deranged. During the recovery phase the liver function more or less returns to normal. So 99.8% of people who develop an attack of Infective Hepatitis recover to normal and it is in the unfortunate 0.2% that hepatic failure occurs. The hepatic failure may escape detection if it occurs early in the diseases but however the onset of failure after the disease is fully developed, can be detected with ease.

It was customary in this condition to give a high protein diet with amino acids and a high carbohydrate and a low fat diet. However the concept regarding the role of proteins in Infective Hepatitis has changed. In fact a high protein diet involving the breakdown into amonia compounds is very dangerous to a patient in whom a cholaemia can be precipitated. It is found that dietary protein, choline and methionine are quite unnecessary in the treatment and is unwise to continue them and hence they should no longer be used. There is no rationale in restricting the fat except for the unpalatability of it, in an anorexic patient who has a strong distaste for fat. When the stools are acholic dietary fat is restricted.

The third group is the cirrhosis liver occurring in the adult population. As mentioned before extensive damage to the liver in these conditions is quite compatible with normal function of liver cells, the disease is then called a well compensated one. Hepatic insufficiency or Hepatic failure occurs when the liver cell function is impaired. Here again methionine and choline as dietary supplements do not hasten the recovery and are not utilised in patients developing hepatic

failure. There is no valid indication for their use in liver diseases. An acute form of the failure can occur when there is a profuse gastro-intestinal haemorrhage due to portal hypertension which depresses the liver function, or by a portal thrombosis or a pyaemia which precipitates the failure. Ammonia salts, ammonia exchange resins, methionine, urea, diamox etc., may also precipitate the failure.

Gastro-intestinal bleeding increases the intestinal nitrogen content and thus subscribes to the failure. The result of the haemorrhage, hypotension and anaemia further impairs the liver functions and precipitate cholemia. Special reference to be made to ammonia salts and diamox. Since these are used as diuretics by themselves or to enhance mersalyl to relieve the oedema and ascites may be detrimental in the production of liver failure. Paracentesis abdominalis may also bring about an attack. The mechanism by which it brings about a sudden attack of failure is not known. The surgical shock due to a sudden release of pressure may be a cause ; a severe protein drain may have a marked strain on the liver which is already functioning at a very great strain.

In olden age group all the diseases described above for adults can give rise to hepatic failure and also carcinoma of liver as well as secondaries in the liver. Terminal phases of obstructive jaundice also can give rise to failure. There are yet groups of conditions, the Reticulosis, Leukaemia, like disorders where there is primary involvement of liver tissue and in them hepatic failure does not set in commonly.

So let us consider the hepatic failure that sets in common diseases as hepatitis and cirrhosis of the liver, of the varieties of the failure we are more concerned is with the acute form of failure which leads the patient to a state called cholemia.

PATHOGENESIS OF ACUTE HEPATIC FAILURE :

The essential defect lies in the fact that the absorptions of intestinal content directly straight into Systemic blood without detoxication by the liver. The liver is a useful intermediary barrier preventing gastro-intestinal poisons entering the systemic circulation and then the brain. The failing liver in hepatitis and cirrhosis is unable to fulfil the functions. Various factors that help the absorption of toxins :—

(1) The failing liver in hepatitis and cirrhosis is unable to carry out the functions adequately.

(2) The bypass is accentuated by the collateral communication between the portal and systemic circulation due to portal hypertension.

(3) Again if the portal vein is thrombosed the whole portal blood is deviated through the collateral channel.

This theory of portal-systemic encephalopathy or auto-intoxication explains the clinical features of hepatic failure.

Poisons which enter the systemic circulation includes :

(a) Bacteria causing fever ;

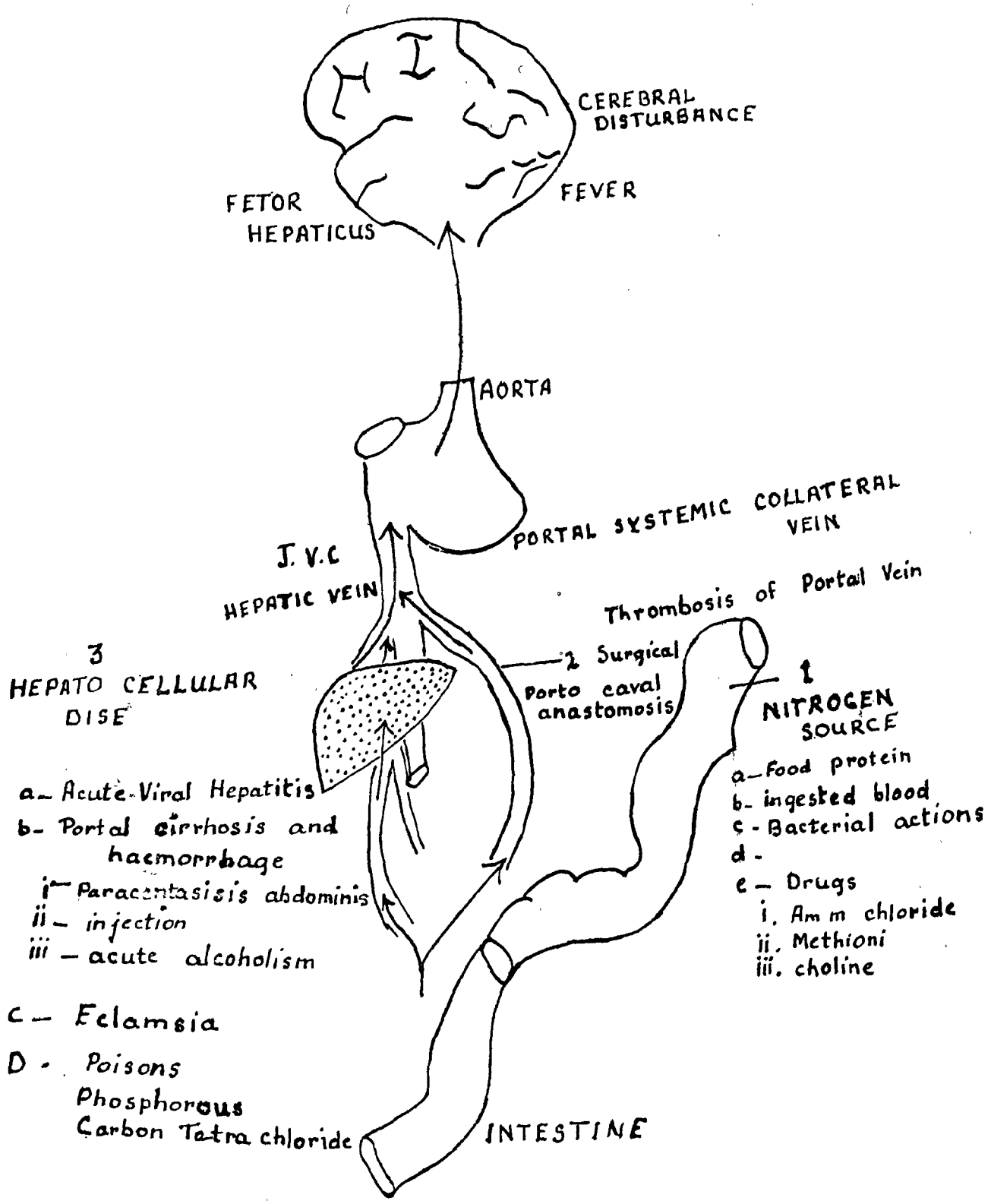
(b) Various intestinal toxins causing Fetor hepatitis ;

(c) Certain nitrogenous substances causing cerebral disturbances.

AMMONIA AND HEPATIC COMA :

Ammonia is highly toxic when given to animals. The Ammonium compound is

MECHANISM OF NEUROLOGICAL CHANGES LEADING TO HEPATIC COMA.



produced by bacteria of intestinal contents. These are quickly converted to urea by the liver. In hepatic failure the ammonia accumulates. The ammonia in the brain cells combines with glutanic acid. In hepatic failure the ammonia content of blood is responsible for the neurological manifestations.

CLINICAL FEATURES AND DIAGNOSIS :

I. General Deterioration of Health.

Poor appetite, indigestion, flatulence, difficulty in storing glycogen, and mobilising glycogen, no manufacture of protein. The patient shows a steady downhill during the course of the disease.

II. Jaundice.

Failure to excrete bilirubin by the liver. This may be present from the beginning of the disease as in hepatitis and in biliary cirrhosis. The jaundice is not present in cirrhosis liver because there is balance between necrosis and regeneration. But when jaundice sets in when it is previously absent, it indicates the impending failure.

III. Low Grade Fever.

The temperature may go up to 100° — 101° F. The low grade fever is explained in various ways.

1. The bacteria by passing the liver barrier may be responsible.
2. Response of heat regulating centre to the products of Necrosed and autolysed Liver cells.
3. The B. Colic septicaemia especially in cirrhosis liver patients.

IV. Fetor Hepaticus.

It is a sweet slightly-faecal odour detected in the breath. It has been likened to the smell of mice or to a freshly opened corpse. It is of intestinal origin. It was originally considered as a grave sign, but is now known to occur in patients as a transient thing in people who are not in coma. This is one of a very good and early signs to recognise hepatic failure in a patient suffering from liver disease. The odour is due to mercaptan and related compounds. This can occur not only in cases hepato-cellular disease but also in cases with extensive portal and systemic anastomosis. Another way when it can occur is following an operation of porto-caval anastomosis.

V. Neurological signs.

The cerebral changes are diffiuse and all parts of C. N. S. may be involved. Changes are mental as well as motor disturbance of the Central Nervous system.

(a) Mental changes.

Changes in behaviour and personality with confusion and disorientation. Sometimes screaming followed by stupor may be present Delirium, unconsciousness and the patient goes into a coma. The coma fluctuates and it is unwise to give poor prognosis. The mental changes are due to retention of bile acid in blood. The coma is due to poisoning by the nitrogenous compound from the gut. The biochemical disturbance is a complex one though ammonia plays a significant role. There is some interference in the ammonia binding mechanism of the brain.

(b) Disordered motor activity.

Flapping tremors of the outstretched hands, similating the beating of the bird's wings. This may spread to the whole body and there is complete incoordination. Rapid irregular movements of the fingers, flexion, extension, at metacarop-phalangeal joints. There is tremor of the hand

which is more common than the flexion extension movements. The tremor may not be obvious : unless we ask the patient to put out his arm in front and hence may be missed if not carefully looked for. Speech may become disordered. When the patient is in coma, the legs are semicrossed and flexed at knee. Pupils are dilated. There may be rigidity and the tendon reflexes are exaggerated. Meningism may be present and occasional epileptiform convulsions may occur. The neurological manifestation may occur as a transient one or in the chronic form. But very often occurs as a terminal type of event in a fulminent hepatitis.

Of course there are changes in E.E.G. are those which need not be discussed here.

VI. Circulatory Changes.

Circulatory changes may be present. There is a hyperdynamic circulation. The cardiac output increases. A high output state and warm extremities due to Sodium chloride retention. There may be left ventricular enlargement. But the changes are not helpful in the diagnosis or the recognition of the syndrome.

VII. Endocrinal Changes.

These are definitely not a feature of a acute failure but are due to a chronic hepatic insufficiency in a longstanding case. The changes are due to failure of the damaged liver to inactivate the hormones especially the oestrogens and as a result of which capillary angiomata, gynocomastia, testicular atrophy and also infertility in woman may occur.

VIII.

Other changes like deranged pigment metabolism and also clubbing which cannot be properly explained.

PROGNOSIS :

When hepatic failure complicates, hepatitis has a worse outlook. 90% mortality rate. But if the attack is got over as the functional capacity of the liver is very great the patient may recover to normal and one has to be reasonably hopeful about the prognosis.

When it complicates cirrhosis and the precipitating factors are obvious, the prognosis is good as alchoholic and infections can be controlled on the other hand if it develops following a haemorrhage the outlook is bad. Insidious or chronic form of failure has got a better outlook and if the patient is not jaundiced and the symptoms due to intestinal auto-intoxication the patient can be rescued from the impending danger.

Treatment :

A. To prevent an acute hepato-cellular failure in a patient suffering from the disease.

B. Treatment to be instituted when the failure has set in.

A. TO PREVENT THE COMA :—

1. Resist the use of drugs that are detoxicated by the liver.

(a) *Sedatives* :

More often we are confronted with the problem as to what drug to use for the sleep of the patient or sedation. The answer will be that no drug should be used to sedate him. Especially the problem will arise when the patient has a severe gastro-intestinal bleeding. More so when the doctor as well as the patient get frightened when there is a severe bout of haematemesis, our hands to be tightly tied to use morphia in these cases as it is an absolute contra-indication. Pethidine,

Paraldehyde and barbiturate group of drugs are used best by withholding them from giving to the patient especially when there is a gastro-intestinal crisis. If still one wants to give some drug it can be paraldehyde 10—15 c.c. per rectum, or potassium bromide or the half the usual dose of phenobarbitone may be given. Of the three the safest will be potassium bromide, the least safe will be phenobarbitone half the dose.

(b) The next important thing is methionine, choline, the lipotrophic factor and a high protein diet. These should be avoided.

(c) *Diuretics* :

Ammonium chloride — This is used along with mersalyl to reduce the oedema and ascites in cirrhosis liver patients. Though it may be argued that the ammonium chloride in the small dose to pricipitate hepatic coma, it may however be avoided.

Diamox — It is also unsafe to use this for the purpose of diurisis in patients with liver diver disease as this can also precipitate an acute failure of liver.

Ammonia exchange resins — These are used more to prevent the sodium retention in the body in patients who do not want to restrict his salt intake. Anyway restricted salt intake is preferable than to get an acute episode with the use of resins.

(a) *Ascites Tapping* :

Paracentasis abdominalis, often and with a sudden relief of pressure can precipitate the failure in an occasional case. It is wiser to tap once or twice and restrict the sodium intake than to tap frequently and be a constant strain on the liver toproduce more protein dhe to a drainage of proteins from the ascitic fluid. Probably the plasma protein goes down with tapping and not following restricted salt intake. Tapping to be performed only occasionally.

(e) *Gastro-intestinal haemorrhage* :

Due to rupture of the oesophageal varies.

Immediate transfusion is necessary. The infusion may have to be given for a prolonged period especially if it is a ooze. Fresh blood is always preferable as it supplies all the Coagulation factors which may be deficient in patients with liver disease.

B. TREATMENT TO BE UTILISED WHEN THE FAILURE HAS SET IN :—

1. Elimination of potentially toxic nitrogenous substance from the Gastro-intestinal tract.

Diet :- When the patient has neuro logical manifestations, proteins to be withheld. When the symptoms are brought under control proteins can be given. The increase should be gradual.

25 grams protein per day for one week and add 25 gms. per week.

During the initial period adequate calories should be supplied in the form of carbohydrates and fat. Otherwise endogenous proteins break-down will occur. If the patient is cooperative 2000 calories can be given by mouth as 20% glucose plus fruits or intragastric drip of 20% glucose in water 2000 c.c. formed 1600 calories. 1 gram of potassium chloride added per day divided into 4 meals and 20 gram increase in alternate days. In un cooperative cases 20% glucose in water 2000 c.c. I. V. drip.

It is better to sterilize the gut with a broad spectrum antibiotic as long as one week is essential. For this purpose chlor-tetracycline or oxy-tetracycline may be used. Parenteral are not as effective as oral. But when the patient is comatose it will be difficult to administer the anti-biotic by mouth at the required interval. So the routine followed will be 250 mgms. of Achromycin is given as a I. V. drip along with glucose saline twice daily. This parenteral therapy will have a double fold action. One on the intestinal bacteria ; but this may not be as effective as oral therapy but on the bacteria which are absorbed from the gut and circulate in the blood stream without being filtered by the liver.

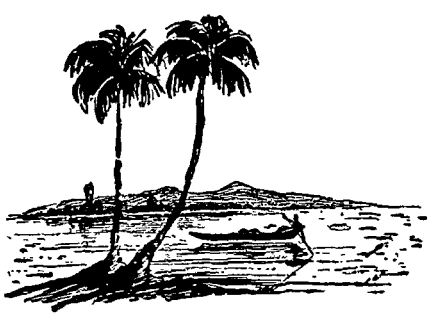
To the drip Vit. B-12 — 100 to 200 mgms. Vit. C., 200-500 mgms. may be added. Other vitamins as B. complex and (water soluble) Vit. K. also can be added.

During the acute phase ACTH 20 units may also be given as a drip.

DRUGS OF DOUBTFUL VALUE :

Glutanic acid : 20 grams a day in divided does in 500 c.c. of Glucose solution I. V. as a drip may be employed with a view that glutanic and combines with ammonia to form innoccous glutanic.

Testosterone is also given for correcting the protein metabolism that occurs in hepatic failure.



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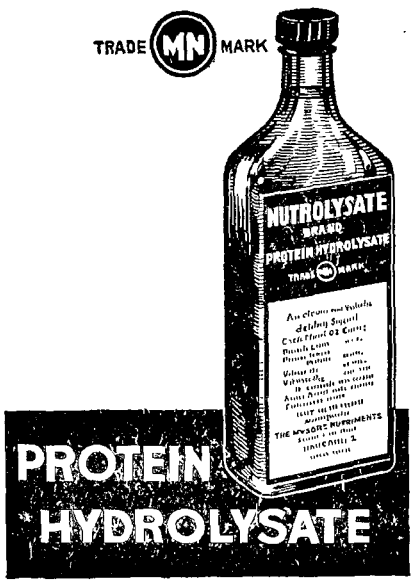

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Blepharochalasis

DR. P. RAMASAMY, M. B. B. S., D.O.

Blepharochalasis an exceedingly rare condition is characterized by stretching and atrophy of the tissues of the upper eye lids consequent on chronic or recurrent oedema of the lids followed by herniation of the orbital fat into the palpebral compartment.

The malady occurs usually at puberty affecting both sexes equally. It may be congenital and may be transmitted hereditarily as a dominant characteristic. A concatenation of events take place in the evolution of this disease. To start with there are intermittent oedematous swellings of the upper lids which are usually painless. Each attack leaves behind certain amount of laxity of the structures of the lids. Ultimately a permanent bagginess of the upper lids occurs whose skin become thined out and wrinkled and in extreme cases may hang over the lid margin in loose folds producing a kind of ptosis called PTOSIS ATONICA. Though all the tissues of the lids suffer damage in this disease, the skin and the septum orbitale seem to have borne the brunt of the attack. The septum orbitale gets thined out and destroyed, allowing the orbital fat and some-times a portion of the lacrimal gland to herniate into the palpebral compartment. Now the skin which is already wrinkled and thrown into folds gets distended and ballooned by the herniated fat. In extreme cases the lids become so much weighed down that the palpebral fissure gets narrowed and vision interfered with. This is the stage of PTOSIS ADIPOSA or FAT-HERNIA.

AETIOLOGY:- The aetiology of this condition is unknown. More than one factors are indicted. They are the inherent weakness of the structures of upper lids, liability of the upper lids to vasomotor and trophoneurotic changes and toxins, both exo and endo, bacterial and non - bacterial. Probably all of them have a hand to play in the production of this disease in more than one combination. As an extreme rarity the condition can be produced by the same mechanism in nephritis, uucompensated cardiac lesions, thyrotoxicosis and myxoedema.

I have come across only two cases so far both more or less during the same period about four months back.

Case I:- A girl, age 18, unmarried, came to me on 16-9-56 with painless bilateral swelling of the upper lids of four years duration.

History :- Four years back she had redness of both the eyes with slight discharge, marked blepharospasm and swelling of both the upper lids. The condition was probably kerato conjunctivitis. She had treatment at the Goverment Hospital and when the condition subsided it left behind certain amount of laxity of the upper lids which remained unabated for three years.

Last year she had painful cord like swelling on the medial side of the left thigh and leg with temperature and flexion deformity at the hip. She was admitted in the Government Hospital on the surgical side (and the condition) (lymphangitis or thrompophlebitis with psoas spasm) was treated with some injections, tablets, mixture and tractions to the affected limb. While under treatment she took a day's mixture of three ounces as one single dose and got severe reaction with swelling of the face, eyelids and ulceration of the buccal mucous membrame and the gums. This time when the reaction subsided it left behind an increased amount of bagginess of the upper lids which is very slowly increasing in size since then.

Clinical findings :- Had symmetrical swelling of the upper lids, painless, not pulsating, skin over which is streched and slightly discoloured movements of the lids and the eye balls normal; no proptosis; no skin disease on the face; no mask like and expressionless face associated with myxoedema.

Swelling was soft in consistancy, did not pit on pressure, could not be reduced by pressure, not warm, not tender, no pulsation felt; preauricular glands not palpable; deep palpation did not reveal any pathological lump inside the orbit.

Fundi and vision were normal on both sides.

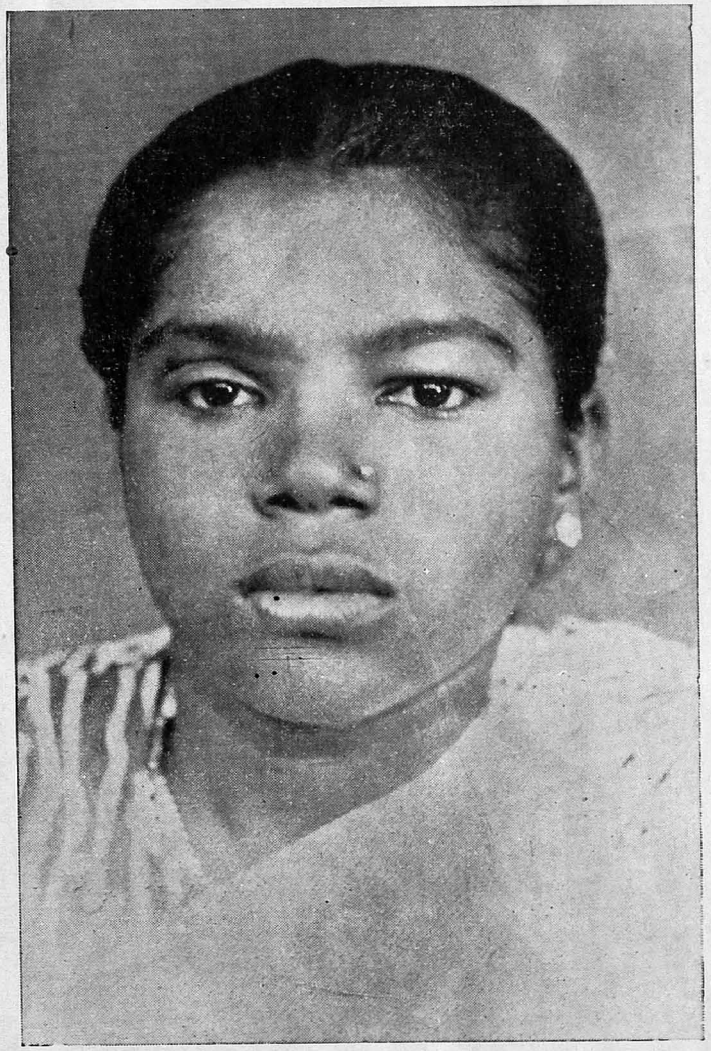
General examination revealed cardiovascular system, respiratory system, and central nervous system were normal; liver and spleen not palpable; lymph glands not enlarged anywhere in the body.

Investigations:- Urine did not contain albumin, sugar, cast or R. B. C. B. P. normal; B. K. negative; blood smear picture showed slight eosinophilia; (blood picture was examined to rule out leukeamic infiltration of the lids); Xray of the lateral view of the skull was taken to rule out pituitary tumours as few cases are on record that such swellings of the upper lids occured in pituitary tumours. The sella turcica was found normal in this case.

I have operated on this girl on the right side and she is awaiting operation on the left side (see picture). Details of the operation are mentioned under treatment.

Case 11 :- A lady of thirty years, married, came to me with

Blepharochalasis



Case 1.

After operation on the right side

symmetrical painless swelling of the upper eye lids of about fifteen years duration; she was very vague about the history of her condition. Clini cal findings were just the same as in the previous case. She was not willing for operation.

Treatment :- The only effective treatment is plastic surgery consisting of excision of the redundant skin, removal of the herniated orbital fat and herniated portion of the lacrimal gland and reinforcement of the septum orbitale by darning with fine silk. During the operation care should be taken not to cut the tendon of the levator palpabrae superioris,not to remove too much of the redundant skin, not to cut the ducts of the lacrimal gland while excising the herniated portion of the gland and not to approximate the tarsal plate too close to the orbital periosteum while darning the septum orbitale. Careless cutting of the levator tendon which results in ptosis and too much removal of redundant skin or too close approximation of the septum orbitale to the periorbita resulting in lagophthalmos are serious mistakes as both ptosis and lagophthalmos are much more serious and disfiguring conditions than blepharochlasis. A local block anaesthesia by injecting novacain along the upper orbital margin close to the periosteum helps a lot to achieve the desired ends of the operation than the usual local infiltration anaesthesia employed in lid surgery as during the operation the normal consistancy and motility of the lid are maintained by the block anaesthesia, helping the surgeon to identify and avoid cutting of the delicate levator tendon and even if the tendon is inadvertantly cut, to know that it is cut at the time of the operation itself so that repair (suturing) of the cut ends of the tendon undertaken then and there. It also helps the surgeon to put trial sutures while darning the septum orbitale and before excising the redundant skin and see and confirm that the patient is able to close the lid normally, all the same achieving the desired aesthetic result, the only consideration for which these patients seek treatment usually.

SUMMARY

A rare disease by name blepharochalasis is described and case notes on the two cases seen so far given; in one case which was operated the details of the operation described and the importance of certain steps in the operation emphasised.

(The Case which was operated was presented at the Erskine Hospital Clinical meeting on 9th January, 1957.)

Fig. 1. X 40 (H & E)

MOLLUSCUM—SEBACEUM

Low magnification of almost the entire growth, showing the massive central keratinisation with epithelial proliferation.

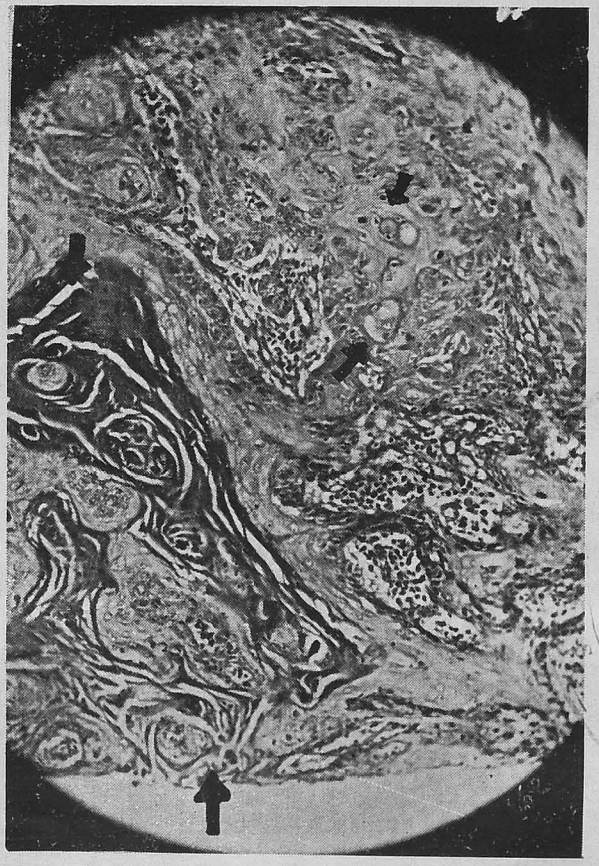


Fig. 2. X 320 (H & E)

MOLLUSCUM—SEBACEUM

A part of the central keratinisation with pseudo-epitheliomatous out growths into the corium, with a number of cell nests.

Fig. 3. X 360 (H & E)

MOLLUSCUM—SEBACEUM

The central mass of Keratin extending deep into the dermis as branched and invaginated crypts filled with keratin material. (marked x)

Molluscum Sebaceum

(A CASE REPORT)

FROM THE DEPARTMENT OF PATHOLOGY, MADURAI MEDICAL COLLEGE

DR. V. C. ANGULI and DR. K. ARUMUGHAM.

Molluscum sepaceum, Kerato acanthema, self healing squamous cell carcinomata of the skin and molluscum pseudocarcinomatosum are various terms applied to a group of solitary or multiple benign tumours of the skin which initially grows rapidly, reaching a maximal size 1—1.5 cms. diameter in 8—10 weeks. They appear as firm projecting umbilicated nodules with horny centres, which in the past have been diagnosed as carcinomatous lesions both clinically and histologically. These tumours have been found to retrogress spontaneously and slowly there after in about 6 months or less. During regression there is a gradual flattening of the lession and finally the horny plug falls of, leaving a depressed scar. The incidence is equal in both sexes. The lesions have occurred on the face, on the ears, behind the ears, on the face and fore arms.

Although most dermatologists and many clinicians are aware of this condition from times long, there had been an astonishing neglect of this subject till 1936, when (3) H. Mac Cormac and R. W. Scarff published a short account of this benign tumour for the first time. Later in 1953, (4) Rook and Whimster published a definite account of this condition in 29 cases. In 1953, (2) Four-acres and Whittick have classified and extended the original work. They pointed out many similarities between Molluscum and spontaneously healing epithelioma of the skin. (1) Beare, however, who has studied the largest series of 76 cases, recognises molluscum sebaceum as a specific entity, distinct from the so called self healing epithelioma of the skin because of the multiplicity of lesions and the familial incidence in the latter condition.

Distinction from genuine epithelioma of the skin is often difficult on purely histological examination of especially small biopsy pieces. The history of rapid growth without regional lymph node enlargement and the gross appearance of the lesion are important guides for diagnosis. Clinically, distinction from carcinoma is not always possible and hence biopsy of lesion is an essential confirmatory aid. The co-operation of both the clinician

and the pathologist is needed for confident diagnosis.

The salient feature in the histological appearance is stated to be massive central keratinisation extending in continuity into the proliferative downgrowths of the epithelium.

It has become obvious that molluscum sebaceum has no aetiological relation with molluscum contagiosum. The true nature of the lesion is undecided. Originally it was thought to be of sebaceous gland origin but all recent evidence indicates to a direct epidermal origin, while the cycle of of rapid growth followed by spontaneous regression is suggestive of virus infection, there is no positive proof for this view.

It is of interest to note that the majority of the cases reported are from the united kingdom. A few cases are known to have been reported from Spain. The lack of reports from other countries is an important factor in the study of geographical distribution. Report ofits occurence in other countries would be welcome for a good deal of study remains to be done, regarding its nature, aetiology etc. After what has been said, it is obvious that the occurrence of a case the first one from this institution warrants reporting :—

Case Report :- A fairly well nourished woman Mrs. R. aged 38 years reported to the surgical wards of the Governmet Erskine Hospital, Madurai for an ulcerative mass of over two months duration over the right gluteal region. The lesion started as a papule and grew rather rapidly during thc first 4 — 5 weeks. Later it became ulcerated. Clinical examination revealed an oval indurated mass of 2. 5. cms. in diameter. The ulceration corresponded to a depressed central area. The mass was raised one cm. above the skin surface. The regional lymph nodes were not enlarged. A clinical diagnosis of an epitheliomatous ulcer was made and an excison biopsy was done by Dr. C. K. P. Menon. The post operative period was uneventful and the wound healed to clean depressed scar.

The excised mass was spherical 3 x 2 cms.) and was covered with normal skin all round on the surface except on the summit which was raw, rough and papilliferous. Although the everted edges of a malignant ulcer was not present, the roughened ulcer in the centre with induration all round was suspicious. The cut section presented an uniform solid greyish white appearance and the depressed summit extended down as an irregular crater.

Microscopic Appearance : A number of sections were studied. The outer boundary of the tumour is covered by stretched epithelium. Low magnification fields show that the tumour is composed of a number of invagination or crypts, (Fig. 1) filled

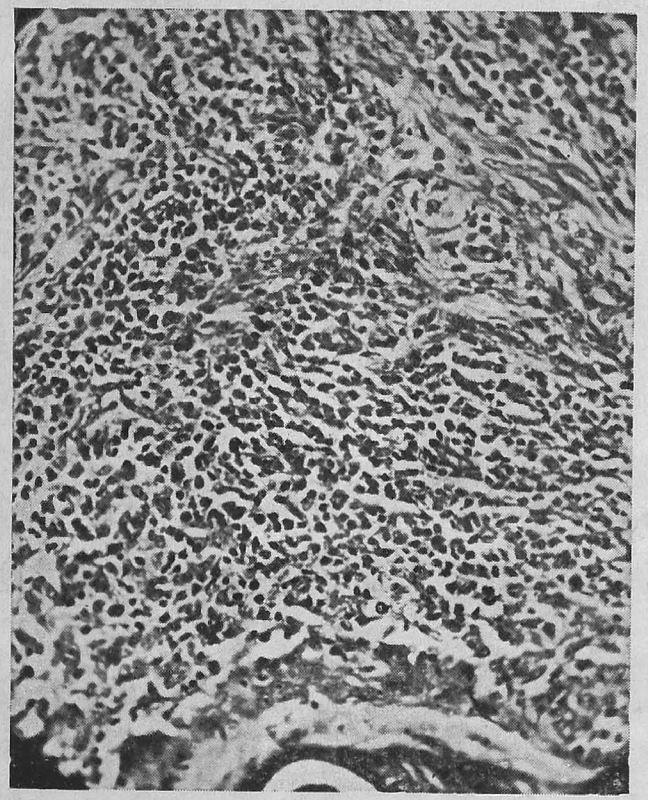


Fig. 5 X 600 (H & E)
MOLLUSCUM SEBACEUM

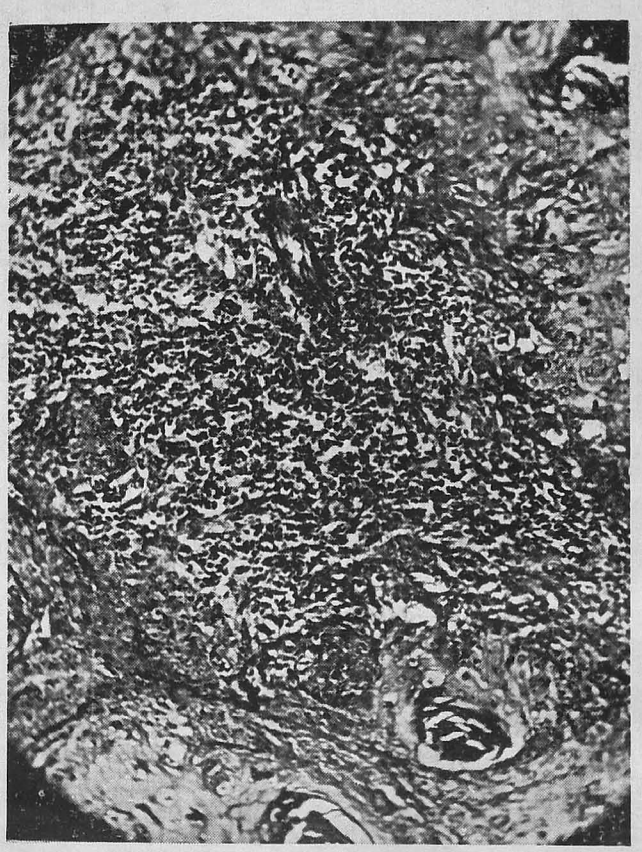


Fig. 4. X 360 (H & E)
MOLLUSCUM SEBACEUM

Both fields show diffuse infiltration with inflammatory cells in the corium — polymorphs, eosinophils and lymphocytes — invading the epithelial outgrowths from the keratin filled crypts.

with masses of keratin material. These crypts are lined by markedly hyperplastic epithelium which has given rise to warty projections both into the crypts and into the dermis as penetrating cords of cells. These growths into the corium present a pseudo epitheliomatous appearance with production of numerous cell nests. (Figs. 2 and 3) These keratin filled crypts appear to communicate with a larger space corresponding to the central ulceration. From the deeper aspect, the hyperplastic epithelium forms irregular keratinised cords into the deeper dermis where there is diffuse infiltration with polymorphs, eosinophils and lymphocytes (Figs 4 and 5). There is no histological evidence of frank malignancy in any of the sections studied. The rest of epithelium shows slight acanthosis. There are no inclusion bodies. With above finding a histological diagnosis of molluscum sebaceum was made.

The histological anatomy is suggestive of primary occurrence of an implantation like epidermal cyst on which numerous papilliferous growths develop as a result of warty hyperplasia of the epidermal epithelium, resulting finally into a cyst like arrangement, filled with keratin. The above surmise fits aptly as regards the site of the lesion of this case.

Summary :

1. A brief account of the condition with reference to nomenclature and literature is given.

2. A report of a case with histological findings is detailed.

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Acknowledgment :

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5. Mr. K. Yadavakrishnan, Madurai Medical College, Madurai for his secretarial assistance.

6. Mr. Rajayya, Artist, Madras Medical College, Madras for his valuable aid in taking micro-photographs.

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Odd Uses for Old Tools

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INTRODUCTION :

IT has always been interesting to study the properties of a drug which have been termed side-actions and other uses. In pharmacological classification, we come across several instances where compounds of varying chemical composition and biological properties are grouped together and called by a common name, according to the particular type of action. e.g. antihistaminics, antimalarials, antirheumatics, antiamoebics, etc.

There is a tendency for us to focus our attention on the action which is prominent probably at the moment and miss any other actions which the drug may possess. An antihistaminic may possess an antiadrenaline, antiacetylcholine or analgesic action. Again, take the drug Rauwolfia — for a long time it was used in the indigeneous medicine for the treatment of epilepsy (Roy's pills). Recently attention was drawn to the beneficial effects of Rauwolfia in hypertension. While evaluating the drug in hypertension, workers noticed a sedative effect on the brain. This was considered to be an unwanted side-effect. Later, the same unwanted side effect turned out to be a blessing, in the treatment of mental cases, maniacal conditions being effectively controlled.

Sometimes the other actions have caught the attention of enquiring people and have been the starting points of new approach to therapy of diseases. For example, some workers noticed that there was remission of rheumatoid arthritic conditions during pregnancy and in jaundice. They traced it to the steroids and ultimately pitched upon the adrenal cortical steroids. Thus the adrenal cortical steroids and their pituitary stimulator (ACTH) have come to be used in the therapy of rheumatoid arthritis.

In going through the literature about antimalarials, a similar thought struck me and I set out to find the "Other Therapeutic Uses" of the group of drugs which go by the name 'Antimalarials'. There are a number of antimalarial drugs in use, the chief ones being the cinchona group of alkaloids, pamaquin and its homologues, pentaquin, chloroquin and camoquin, mepacrine, paludrine and Daraprim. They have different chemical structures cupriene of quinine, aminoquinoline of pamaquin group, aminoacridine of mepacrine, biguanide of paludrine and pyrimethamine of Daraprim. Such a variegated group has one property in common, namely, antimalarial action. Of course, they act on the malarial parasite in different ways. When we study these compounds, we can pick up some common "Other actions or other uses" of one or more of these antimalarials.

In 1953 I expressed the above view in a paper read at the All India Pharmaceutical Conference at Madras. When your Secretary requested me to address you, I thought that the above would be a good subject for a talk. Personally, I wanted to determine if any other new

additions have been made to the "Other uses". I was not surprised to come across more than half-a-dozen new applications coming to light since 1953.

The other clinical applications of antimalarials. I have gathered and grouped under four headings :

Disease Condition	Antimalarials used
I. *Heart Diseases* :	
(a) Cardiac arrhythmias	Quinine, QUINIDINE, Mepacrine
(b) Angina pectoris	Quinine, Qunidine
II. *Dermatological conditions* :	
(a) Chronic lupus erythematosus	Quinine, MEPACRINE, CHLOROQUIN, Daraprim
(b) Light sensitivity, sunburn	Chloroquin
(c) Scleroderma	Chloroquin
(d) Cutaneous leishmaniasis	Mepacrine, Chloroquin
III. *Parasitic Infections* :	
(a) Amoebiasis	Quinine, Mepacrine, CHLOROQUIN, Camoquin
(b) Giardiasis	MEPACRINE, CHLOROQUIN, Paludrine, CAMOQUIN, Acranil.
(c) Taenia saginata	MEPACRINE, CHLOROQUIN
(d) Hymenolepis nana	Acranil
(e) Paragonimus westermani Clonorchis sinensis	CHLOROQUIN
(f) Oxyuris vermicularis	Mepacrine
(g) Trichomonas vaginalis	Mepacrine
IV. *Miscellaneous conditions* :	
(a) Polycythaemia vera	Daraprim
(b) Epilepsy	Mepacrine
(c) Bronchial asthma	Phthalamaquin

NOTE :- Drugs found to be of definite usefulness are given in bold letters.

I. CARDIAC CONDITIONS :—

(a) *Arrhythmias of Heart* : Of these, the tachycardias are of special interest to us. These are further divided into sinus tachycardia, paroxysmal auricular tachycardia, AURICULAR FIBRILLATION, AURICULAR FLUTTER and ventricular tachycardia. Sinus tachycardia is a physiological one due to relaxation of vagal tone over the sinoauricular node or the pace maker of the heart. Proxysmal tachycardia is due to aberrant foci in the auricles starting rapid impulse production. Auricular fibrillation and flutter have been previously supposed to be due to a circus movement in the auricular muscle, a band of excitable muscle tissue always presenting itself before an impulse and thus perpetuating it. This theory is in disrepute now. In fibrillation the heart beats about 450 - 500 times per minute and in flutter, about 300 times per minute. Ventricular fibrillation is always final and ends in cardiac arrest.

In the treatment of auricular fibrillation and flutter, quinidine reigns supreme. Quinidine is a dextro-rotatory isomer of the cinchona alkaloid, Quinine. It is interesting to recall how this drug came to be introduced in the therapy of arrythmias of heart. In 1927, Wenckebach noticed that some of his patients with auricular fibrillation when treated for malarial attacks, with large doses of Quinine, recovered from malaria and curiously enough were cured of their fibrillation. Wenckebach's discovery of the action of Quinine in atrial fibrillation soon led to the use of Quinidine, the dextro-rotatory isomer of Quinine, in the arrhythmias. Quinidine is much more effective than Quinine and is therefore preferred for the therapy of Atrial Fibrillation, atrial flutter and premature systoles. Some physicians in the West still employ Quinine Hydrobromide for the treatment of premature systoles. Others use the so-called Wenckebach pills (Quinine 0.1 Gm. and Strychnine 0.1 mGm.). It is the Quinine that exerts beneficial effects while Strychnine has no pharmacodynamic actions useful in the treatment of premature systoles. Quinine has also been used to slow the tachycardia of hyperthyroidism.

Quinidine has now become the mainstay in the treatment of fibrillation. It completely abolishes fibrillation, in some cases causing a reversal to normal or in some it may merely slow the rhythm without reverting to the normal rate. Anyway, the depressant action of quinine and quinidine are responsible for their action in fibrillation.

Dosage :- Quinidine is given as follows : 0.2 Gm. initially to test for idiosyncrasy, from next day 0.4 Gm. five times daily for about five days when the desired effect will be manifest and then followed by a maintenance dose of 0.2 — 0.6 G. daily. If within five days results are not favourable, it is better to stop the drug. Quinidine is useful mainly in auricular fibrillation of recent origin.

Recently Mepacrine has been introduced in the therapy of auricular fibrillation with some good success. Mepacrine has been found to inhibit a number of enzyme systems., e.g. yellow enzyme system of Warburg, Choline esterase, etc. It has been suggested that such inhibition of choline-esterase may be responsible for the use of this drug in cardiac arryhthmias. In therapeutic range it restores normal sinus rhythm in dogs with experimental atrial fibrillation, probably by a mechanism similar to that of Quinidine; on this basis, the drug has had trial in

Cardiac arrhythmias in man. It is also being reported to prevent ventricular fibrillation induced by epinephrine in dogs under chloroform anaesthesia.

As with quinidine, it is found to be most useful in cases of auricular fibrillation of recent origin associated with non-valvular cardiac disease. In cases of chronic fibrillation with or without heart disease, it is regrettable that it is of no more value than quinidine. Only in these cases therapy is lacking a good drug. Quinidine is of no use and may be dangerous, as it is likely to precipitate a thrombo-embolic phenomenon with fatal results.

Dosage :- 0.3 — 0.6 G. of Mepacrine are given (10 c.c. of 1%) intramuscularly. There is no known chemical relation between Quinidine and Mepacrine to explain the similarity in action.

(b) *Angina Pectoris* : In a recent well controlled study by Riseman & others (1955) using placebos, various antimalarials and known coronary vasodilators (like nitroglycerine etc.) it was shown that quinine and quinidine were very effective in producing relief. Probably the iso-quinoline group is responsible for the vasodilator action and hence the benefit in angina.

II. DERMATOLOGICAL CONDITIONS

Some of the antimalarials have been found to be useful in some dermatological conditions. *Chronic lupus erythematosis,* a dermatoses affecting usually the skin of the face and hands, especially the exposed parts, (it may co-exist with rheumatoid arthritis), responds very well to therapy with some of the antimalarials (QUININE, MEPACRINE, CHLOROQUINE & DARAPRIM). Many other drugs also have been in use such as P. A. B. A., A. C. T. H., Cortisone, Procaine, gold, Vitamin A, oestrogens, and androgens, with varying results.

Quinine has been in the run for a long time. It has been combined with iodine in the treatment of this condition. Quinine sulphate is given orally in 0.5 Gm. doses thrice daily for 5-7 days and the skin lesions are touched with Tincture of Iodine once daily. After a week's rest, the course is given again. During the period of rest the crusts are removed.

Recently Mepacrine has been introduced in the treatment of lupus erythematosus. A number of workers claim very good success with such therapy. The lesions disappear with the skin condition returning to normal. Mepacrine therapy is only empirical. It may possibly act by reducing the light sensitivity of the skin or it may act similar to cortisone or it may antagonise adenyl compounds. It may be given in 300 mgm. daily doses orally, till the skin is stained and then, 100 mgm. daily as maintenance dose.

Duration of treatment varies with clinical response. Very few toxic reactions have been reported. The staining of the skin by the drug to an ungainly extent as to appear jaundiced, is a great drawback. This staining is due to the drug and not to any jaundice. The drug produces exacerbations after initial subsidence in some cases. It may be controlled by continuance of therapy. It produces hyperkeratoses in some people.

It was mentioned that one drawback of mepacrine was its staining property. Recently it has been replaced by chloroquin in the treatment of lupus erythematosus. Chlorquin is equally, if not

more, efficient and less toxic than mepacrine. It does not stain the skin. Dosage of 0.5 Gm. daily for two weeks, followed by 0.25 Gm. daily is sufficient to suppress and control the disease. Careful repeated blood counts should be done as it may occasionally cause bone marrow depression.

Daraprim has also been tried in chronic lupus erythematosus. The dosage is 25 mg. a week initially, then raised in two steps to 25 mg. daily, for 2-3 months. Some improvement has been reported.

It is apparent that it is not the antimalarial properties of these drugs that exert the beneficial action but something else associated with their chemical conformation. Quinacrine and chloroquin have somewhat similar structural formulae whereas quinine and daraprim are not closely related to them structurally.

Other skin conditions reported amenable to therapy with antimalarial drugs are *light, sensitivity, sunburn* and *sclêroderma.* This problem is much more acute amongst the fair-skinned races. With increasing vogue of sunbathing and outdoor activities, light sensitivity has become more frequent. Any substance that will increase tolerance to sunlight should therefore be a boon. Mepacrine (HCl) prevents recurrences in photosensitised persons and has been used successfully in solar dermatitis. Chloroquin may be added to ordinary table salt as a possible preventive against the ill effects of sunlight in sensitive persons.

It is reported that patients with *scleroderma* who had not responded to all other usual treatments, were successfully treated with chloroquin. The mode of action is not known. Possibly by being deposited in the skin, it may act on the collagen fibres directly. Or it may influence the adrenal or pituitary mechanism through its accumulation in the liver. In treating scleroderma, it is said, that chloroquin is easier to administer, relatively more economic and less toxic, even after months of use, than ACTH or Cortisone.

Strangely, *cutaneous leishmaniasis* (oriental sore) is reported to have been cured with cicatrisation, by the administration of Chloroquin, in a dosage schedule of 0.75 Gm. twice the first day, and 0.5 Gm. twice the second and third day and then 0.5 Gm. every 5th and 7th day until healed.

It has been both contended and denied that local infiltration therapy with mepacrine is useful in cutaneous leishmaniasis.

III. PARASITIC INFECTIONS :—

(a) *Amoebiasis* : Amoebic infection of human beings occurs due to a protozoa, entamoeba histolytica. This protozoa occurs in a cyst form and an active motile form called trophozoite. Man ingests the cysts which undergo transformation to trophozoites in the gut and there multiply, causing ulceration of the mucous membrane, diarrhoea and dysentery. They invade the intestinal wall and get disseminated all over the body through circulation, getting deposited mainly in the liver. Here it causes hepatic amoebiasis ranging from hepatitis to hepatic abscess limited to the liver boundary or may burst into the chest cavity and cause lung complications. When environmental conditions become unsuitable to the parasites for multiplication and thriving, the amoeba get encysted and are passed in the stools. It is only the cysts which are infective when ingested by another person. It is

very interesting to note that malarial parasites are also protozoa called plasmodia. There are a number of drugs used in the therapy of amoebiasis like emetine, vioform, chiniofon, aureomycin etc.

Quinine has been known to be a general protoplasmic poison. It can kill low grade organisms like paramecia. Quinine was used in the treatment of amoebic dysentery in the past. Brookes (1917) reported the efficacy of quinine orally in doses of 2 Gm. Fletcher (1924) advocated the use of slow rectal injections of 1-2 litres (1 : 2000 to 1 : 1000 solutions) of quinine for local action. Of course, these are out of use now. Still they give us an indication of the progress in therapy.

Recently other antimalarial drugs have come to be used in the treatment of amoebiasis. Mepacrine is useful only against trophozoites causing dysentery. It is effective neither against the forms that have invaded systemically nor against the encysted forms in the gut. Dose :- Mepacrine is given in doses of 100 mgm. thrice daily for 10 days.

Chloroquin exhibits unique therapeutic value in extra-intestinal amoebiasis in man (i.e. amoebic hepatitis, amoebic abscess of liver, pulmonary amoebic abscess, etc.). The property of its getting concentrated in the liver determined its use in the hepatic amoebiasis. However, it is not useful against intestinal amoebiasis. Therefore, it should be used with an intestinal amoebicide, even though in some cases there are no indications of intestinal lesions and only signs and symptoms of hepatic involvement. Clinical response to chloroquine in patients with hepatic amoebiasis is usually as prompt and complete as that to emetine and the drug has proved effective in some individuals failing to respond to emetine. Chloroquin, like emetine, is not always curative and therefore, adjuvant medical and surgical measures may be necessary. Amoeba do not develop tolerance to chloroquin. The conventinal course of chloroquin phosphate for extraintestinal amoebiasis in adults is 1.0 Gm. daily for two days, followed by 0.5 Gm. for two to three weeks. The course can be repeated or alternated with emetine therapy. The two drugs, however, should not be prescribed at the same time because of severe toxic effects which may follow.

The efficacy of chloroquin in amoebiasis suggests that in halogenated oxyquinoline amoebicides it is the quinoline nucleus per se and not the iodine content of the molecule that is important ; indeed, there is no direct relation between the dose or the efficacy of these compounds and their iodine content.

Similar to chloroquine, Camoquin is effective in amoebic hepatitis and not useful in intestinal ameobiasis.

(b) *Giardiasis* : Giardia intestinalis, usually considered a pathogenic organism, may occasionally be found in the faeces of a healthy person. Giardiasis is quite common especially in children. Symptoms usually are anorexia, flatulence, abdominal pain and attacks of diarrhoea. Clinical diagnosis is confirmed by microscopic examination of the faeces which reveals organisms in the vegetative or cystic form or both.

Giardia lamblia infection of the intestinal tract is amenable to treatment with mepacrine, 100 mgm. three times daily for 5-7 days. ACRANIL, a mepacrine derivative is also said to be useful in giardiasis.

Although mepacrine is almost specific in this condition, it may produce toxic symptoms. Konar and associates (1953) at Calcutta tried Camoquin. It was given in a single dose of 3 tablets of 0.2 Gm. for adult, 2 tablets for patients aged 5-15 years and 1 tablet for children under 5 years. With this dosage schedule toxicity was less and stools were free from the parasite.

Chloroquine also has been reported to be of value in human giardiasis. Paludrine too has been tried with some benefit in this conditions.

(c) *Taenia Saginata*: Mepacrine and chloroquin have been reported to be useful in the treatment of taenia saginata infestation (beef tapeworm) perhaps replacing the old male fern therapy. Mepacrine like male fern is not lethal to the tapeworm, but it will cause the scolex temporarily to detach from the intestinal wall and the worm can then be expelled by a purge. Before administering the drug it is essential to empty the intestine of its solid contents, by restricting the patient's diet to fluids for two days, on each of which a laxative is given. On the third day, the dose of mepacrine (1 Gm.) is given and later a purgative. As mepacrine is likely to irritate the stomach, it is advised to give sodium bicarbonate solution in addition or else to give the drug through Ryle's tube directly into the intestines.

Chloroquine also has been reported to have been useful in taeniasis.

(d) Two other trematode diseases, common in China, due to Clonorchis sinensis which affects the biliary tracts and Paragonimus westermani, which mainly affects the lungs, which for a long time have defied treatment, have of late been successfully treated with chloroquine, given over a period of several weeks.

(e) *Hymenolepis Nana* (*Dwarf tapeworm*) : Mepacrine may be of limited value in this infection. However, it is not effective in children. Recently ACRANIL, a compound closely related to mepacrine, has had preliminary trials with some success in the treatment of this condition, as well as in giardiasis.

(f) *Oxyuriis Infection* : Mepacrine has received a favourable trial in human infection with oxyuris vermicularis.

(g) *Trichomonas Vaginalis* : Mepacrine has been able to control the infection.

MISCELLANEOUS CONDITIONS :-

(a) *Polycythaemia Vera* : Isaacs (1954) reported Daraprim to be useful in this condition. In experimental animals it causes aminopterinlike reactions and lowering of red and white cells in the peripheral blood. Dose :- 25 mg. daraprim once daily after breakfast until RBC count approached normal and maintained indefinitely at 12.5 mg. daily and discontinued if the fall was rapid. No toxicity was noticed. If this finding is confirmed, it will really be a step forward in the therapy of this usually bane condition.

(b) *Bronchial Asthma* : Phthalamaquin, a derivative of 6-methoxy-4-amino quinoline, which is closely related to the antimalarial chloroquine, is claimed to have both bronchodilator and antihistaminic properties. It is said to have the following characteristics (C. F. Geschickter 1955). It is concentrated in the respiratory tissues where it is cumulative.

It can be given by all routes. Tolerance is rare. This will open up new avenue for the treatment of bronchial asthma. Dose :- 3-5 mg./kg. body weight daily for several months.

(c) *Epilepsy* : Mepacrine has been reported to be of some value.

CONCLUSION :-

Thus we see that many of the antimalarials are slowly finding place in the therapy of many unrelated disease entities other than malaria. Perhaps they may prove as versatile as cortisone. I will not be surprised if, in the course of the next few years, the other uses of these antimalarials become more numerous. Possibly, by studying the accidental, stray or unaccountable usefulness in other disease conditions, we may get an insight into the workings of the therapeutic mechanisms.

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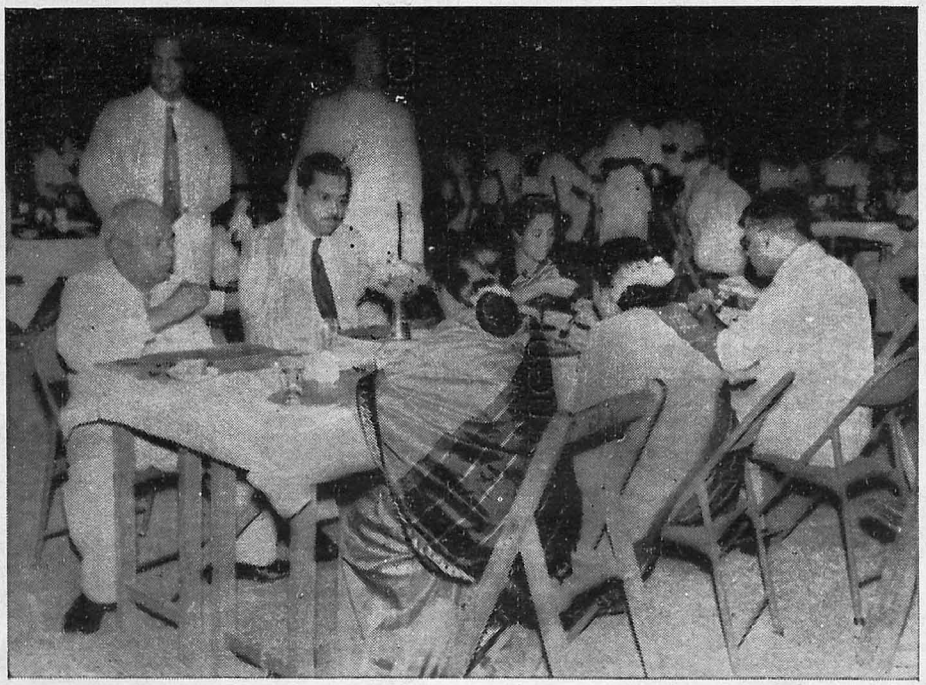
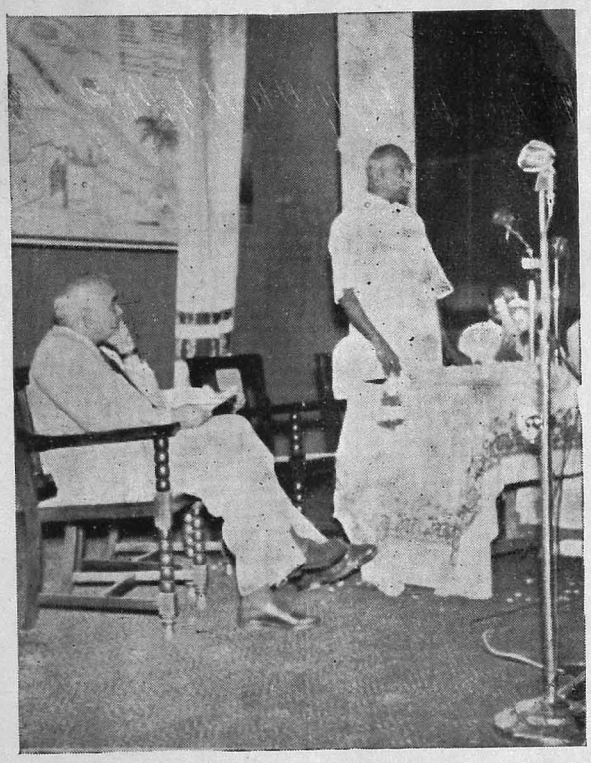


"Surgical Approach."



INAUGURATION

OF

MADRAS STATE EMPLOYEES' INSURANCE SCHEME

THE MADRAS STATE EMPLOYEES INSURANCE SCHEME

★

The State Insurance Scheme was inaugurated in Madurai on the 28th. October 1956. The Scheme, which is working so very successfully in Coimbatore and Madras, provides number of benefits to the industrial worker. There are at present six Govt. Dispensaries, manned by eleven Asst. Surgeons, situated in the thickly populated labour areas of the city, in addition to the four Mill Dispensaries also in the scheme. About sixty beds are provided in the Christian Mission Hospital exclusively for the inpatient treatment of the labourers, and Specialists are appointed by Govt. for each branch in the Erskine Hospital, where the workers are referred to by the Medical Officers of the dispensaries for specialist examination and treatment. The insured person gets cash benefit for sickness (in addition to medical benefit), disablement, maternity and dependent's benefit in the event of death of the worker due to or as a result of employment injury. At present the scheme covers only the worker, but after April of this year it is going to be extended to the families of the workers also. About 28000 workers of this city are now enjoying the benefits of this scheme.

Dr. S. Bhuvaneeswaran M.B.B.S.

THE MYSORE INDUSTRIAL & TESTING LABORATORY, LIMITED

Malleswaram, BANGALORE—3

The Industrial & Testing Laboratory was started in 1931 by the Government of Mysore mainly to supply, as far as possible, the pharmaceutical requirements of the Government Medical, Public Health and other Departments and also to have a well equipped Testing Laboratory to analyse Government purchases.

The Laboratory progressed and expanded till 1945 when the Government felt that further expansion could be accelerated under private management—the Government actively participating in the management.

The Mysore Industrial & Testing Laboratory, Limited was formed in 1945 and are the successors to the Government Industrial and Testing Laboratory. The Capital of the Company is Rs. 30 lakhs and the Issued Capital is Rs. 15 lakhs. Rs. 9,37,500/- only being called and paid up. The Government of Mysore nominate the Chairman and the two Directors on the Board of Management.

The Laboratory Manufactures

All Galenioals, B.P., & B.P.C., & Fine Chemicals

GLANDULER PREPARATIONS

Reticulin Brand Liver–Extract (Oral & Injectables)

INJECTABLES

WELL-TESTED AND POPULAR SPECIALITIES

Vitoferrin, Vitomalt, Sedatin, Linctus Codeine co.
Gripe Syrup, Extract of Malt with Cod Liver Oil

ANTISEPTICS AND DISINFECTANTS

Iteol-3, Lysol and Phenyle (A & B grades)

MALT

Extract from Ragi and containing all the growth promoting factors present in the germinating Ragi–Production Capacity half a ton per day

and a

WIDE RANGE OF TABLETS

Arthrophen, Aspirin, Bevitamin, Gluconate-D, Cevitamin, Milk of Magnesia, Retifolin, Somnitol, Throat Menthalic, Vitamin B. Complex etc. etc.

The Laboratory has all the necessary up-to-date testing and manufacturing equipment and is also staffed with competent and experienced chemists

The Manufactory is situated in BANGALORE and has a Sales Depot at No. 35/37 Thambu Chetti St. Madras, and Selling Agents at Hubli and Hyderabad

Erythroblastosis Foetalis

(A CASE REPORT)

FROM THE DEPARTMENT OF PATHOLOGY, MADURAI MEDICAL COLLEGE

DR. V. C. ANGULI and DR. P. NATARAJAN.

IT is the most important condition among the antenatal, neonatal and congenital diseases. Diamond and Levine have established that hydrops foetalis, icterus gravis and the haemolytic disease of the newborn are all manifestations of the same condition produced by the same pathological process namely primary intra-vascular red cell destruction caused by specific maternal agglutinins reaching the foetus before birth — in varying degrees of severity. The diagnosis of the condition may be made during the antenatal period with certainity only some times ; in other cases it may be suspected, while at birth the suspicion may be confirmed or refuted. Antenatally the diagnosis may be foretold radiologicallly or serologically. Characteristic changes in the X-ray typical of hydrops foetalis may be evident about the 32nd week of pregnancy and the mother can in such instances be prepared for the outcome, for hydrops foetalis is a uniformly fatal condition. On the purely clinical side, hydrops foetalis may result from maternal diabetes or congenital syphilis.

The following is a brief account of simple facts about iso-immunisation for an understanding of the serological data :

Although recent trends in haematology have led to the discovery of new blood groups in man, from a practical point of view, a knowledge of the roles of the three blood group antigens A, B and Rh in clinical medicine is sufficient to get at almost all the problems of blood group incompatibility in man. Rh is commonly spoken as if it is a single antigen ; in fact, the red cells contain a complex mixture of Rh antigens. It is essential to know that persons who are immunised to any one of the Rh antigens lack the so called 'D' factor and it is to this factor they become sensitised or immunised. Most Rh anti-bodies have the specific anti 'D' factor. The convention to-day is a D negative person is called Rh negative and a D positive person is called Rh positive. This is advantageous because Rh is more familiar term than D. Every individual must inherit either Rh positive D or the Rh negative factor 'd' from each of his parents with the resulting combinations as DD, Dd or dd. The first two would give rise to a Rh positive individual. The value of the finer distinction of "Dd" combination will be appreciated in the following instance :— In a Rh immunised Rh negative woman, who has lost children from haemolytic disease of the newborn if her husband is Dd, there is every chance of having a child born, healthy and alive, in the 'dd' combination, namely Rh negative and therefore unaffected by the maternal Rh negative antibodies. This is of academic interest.

Two kinds of Rh antibody have been established : One agglutinates the Rh positive cells suspended in saline known as bivalent antibodies and the other agglutinates Rh positive cells suspended in serum or albumin known as the uni-

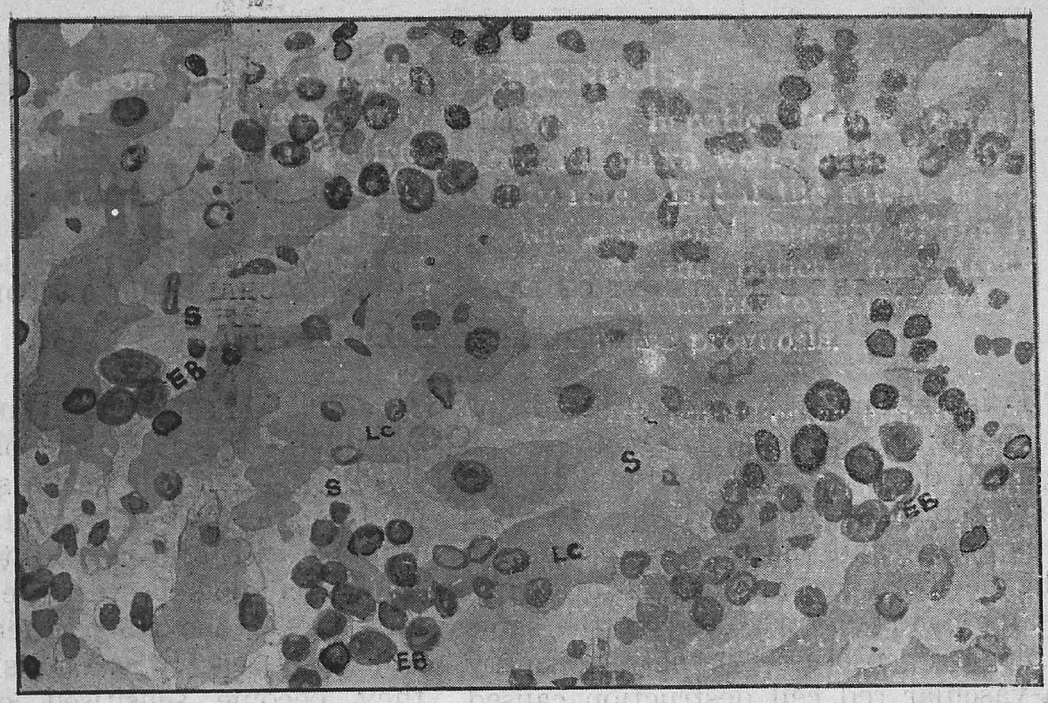


Figure. 1.

Liver in Erythroblastosis : Shows foci of erythropoesis, (EB), dilated sinusoids (S) and distortion of liver cords (Lc).

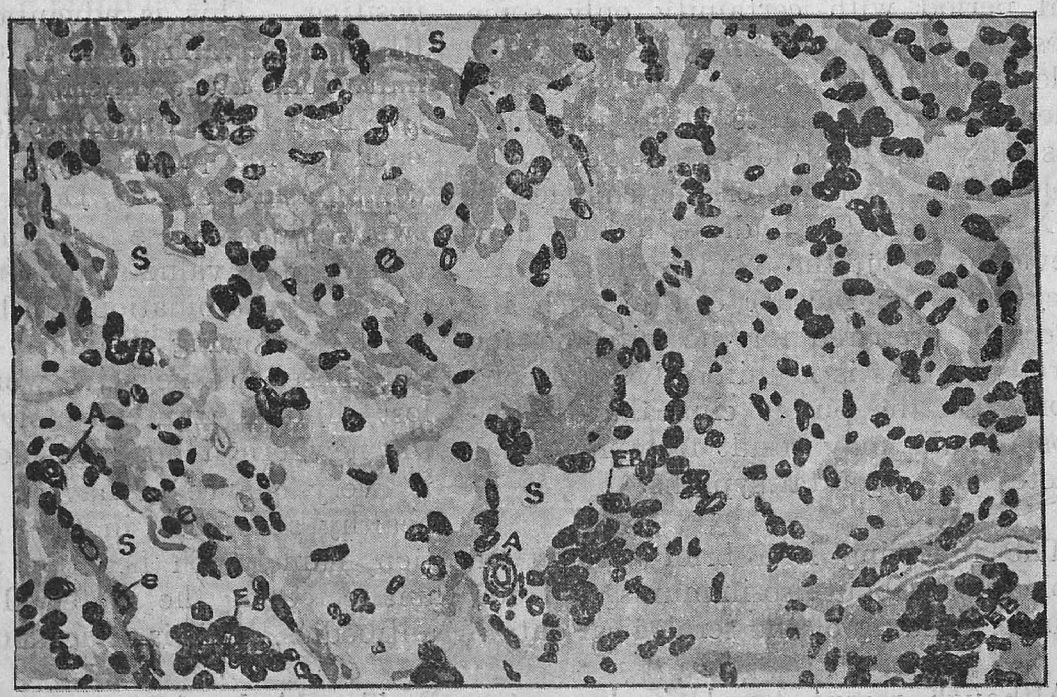


Figure. 2.

Spleen in Erythroblastosis : Sinusoids (S) distended by erythroblasts (EB) and lined with prominent endothelial cells (e). Malpighian bodies could not be distinguished. Few arterioles (A) are found in the vicinity of the sinusoids.

valent or blocking antibodies. Serologically, the following factors are of significance in predicting an affected infant.

It is difinitely known that the incomplete univalent antibodies pass the placenta readily while the bivalent ones, which are active in saline fail to pass. Davidsohn and Stern have presented evidence that the presence of higher amounts of antibodies in albumin than in saline dilution, during the last four weeks of pregnancy is likely to affect the infant.

The above account is an out come of study of he condition following an autopsy examination of a still born child referred from the Maternity wards of the Government Erskine Hospital, Madurai. The dead child was the first born of the mother 19 years old an Anglo-Indian, who was married a year ago. No ante-natal serological examination was done either for Rh or for evidence of luetic infection. The cause of still birth was thought to be due to congenital syphilis and the infant was autopsied four days after death.

It was full term foetus weighing 6 lbs. and presenting marked cyanosis. There was no evidence of maceration and the abdomen was slightly distended. There were no external manifestations of congenital syphilis. About 12 ounces of straw coloured transudate were present in the peritoneal cavity.

All the viscera showed varying degree of congestion. The spleen showed slight enlargement. The weights were within normal limits. The brain was semi solid in consistency, however, there were no localised bile staining marks in the region of the basal ganglia or the cerebellum. The umbilical cord was not examined.

The placenta was not available for examination.

The following is a brief account of histopathological study of the organs :—

THE LIVER : Extra medullary erythropoises was the dominant feature. Large, circumscribed foci of immature red cells — most of them are erythroblasts and normoblasts — were seen scattered diffusely throughout the liver parenchyma. The erythropoises appeared regular and set in more numerous foci than in the liver of a newborn infant. The sinusoids were dilated and were lined by prominent Kupfer's cells containing pigment. The liver cells showed compression atrophy with moderate displacement of the cords. There was increase of periportal fibrous tissue.

THE SPLEEN : The striking feature was the presence of large proliferative endothelial cells lining the sinusoids, a number of them containing erythropoetic centres. Lymphocytes were scanty — a mere cuffing of the arterioles here and there. Haemosiderin laden histiocytes were present throughout the splenic pulp.

THE LUNG : The alveoli showed expansion. A number of cells with large nuclei were present in the inter-alveolar-tissue. These represent the immature cells, for increase of such circulating monocytic cells other than immature red cells is unknown in intrauterine life.

THE BRAIN : There was no deposition of bile pigment in the nerve cells, myelin sheaths and interstrial tissue of the brain stem and cerebellum.

THE KIDNEY : Distinct erythropoitic centres were present specially in the

sub cortical regions — than in the region of the pyramids.

The above findings suggest a wide spread extra medullary erythropoises — a reaction referable to a severe degree of intra-vascular haemolysis.

Subsequent to the autopsy examination, the grouping of the blood of the parents was done a month after the delivery. The mother was Rh negative and the father Rh positive the grouping done against anti D-serum.

The above report offers a post-mortem diagnosis of a mild form of hydrops foetalis. There had been prolonged severe intrauterine haemolysis, the foetus had become profoundy anaemic and hydropic before birth and was still born. There was no time for jaundice or Kernicterus to develop. One can expect bile staining of the basal nuclei, if the child had lived for 36 hours.

REMARKS :

The percentage incidence of Rh negative persons is said to be low in our country, giving the general impression that the occurrance of erythroblastosis foetalis is very rare amongst us. There has been no scientific assessment of the population regarding 'Rh'. With the geometrical progression of increase in our country's population, it is reasonable to state that one should expect complex combinations in the blood groups of the people with a probability of the "dd" combination individuals also. This would mean that erythroblastosis foetalis is not an uncommon condition but the true incidence is missed because thorough antenatal serology is not done in most of the cases. Though both antenatal and postnatal diagnoses require adequate laboratory facilities, it is more important for the practitioner to include this disease in his differential diagnosis of similar conditions.

This is a condition where the latest methods of treatment have resulted in a appreciable decrease in the mortality from haemolytic disease. More attention is focussed towards the prevention of the disease than modification of treatment of kernicterus.

In every suspected case, besides serological examination of the parents, the placenta and the child's blood (from the umbilical vein) must be examined for the evidence of the disease. The saline titre of the anti Rh antibodies in the mother is no criterion of the severity of the disease in the foetus.

While it is geenrally known that more than one pregnancy may be required to induce an effective level of anti-Rh agglutinins in the mother, it is difficult to reconcile with the occurrance of hydrops foetalis in first pregnancy as in the above case. The variability in the reaction of Rh negative mothers to produce antibodies is perhaps due to the differences in the antigenicity of of the DD and Dd combinations in the father — more in the former and mild or nil in the latter.

SUMMARY :

1. A brief account of the isoagglutination with reference to the Rhesus factor is stated.

2. A case of hydrops foetalis with complete autopsy findings is reported.

3. A few general remarks pertaining to the case are offered.

REFERENCES :

1. Diamond L. K. (1947) *Proc. Roy. Soc. Med.* 40 : 546.

2. Davidsohn and Stern K. (1948) *Americal J. Clin Path.* 18--690.

3. Levine P. (1941) *Americal J. Clin Path.* 11--898.

ACKNOWLEDGMENT :

Our grateful thanks are due to 1. The Principal, Madurai Medical College, Madurai for permitting us to publish this report.

2. Mr. K. Yadavakrishnan, Madurai Medical College, Madurai for his secretarial assistance.

3. Mr. A. G. S. Sundaramurthy, Artist, MDU. Medical College, for his assistance.

LIST OF CONTRIBUTORS

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Dr. P. NATARAJAN M.B., B.S., Tutor in Pathology, Madurai Medical College, MADURAI.

Retention of Urine

GYNAECOLOGICAL ASPECTS.

DR. K. S. KRISHNAN, M.B.B.S., F.I.C.S., Madurai.

Retention of urine in the female is not as common as in the male because the female urethra is short and has not got the anatomical curvatures and the constrictions that characterise the latter. Barring post-operative retention, acute and complete retention are comparatively rare. From the point of view of the diagnosis and treatment, as in the male neurological causes such as cord lesions, disseminated sclerosis and tabes dorsalis have to be excluded before thinking of gynaecological causes. In the chronic cases where there is a large amount of residual riune there is risk of infection. A large cystocele is an instance in point.

Anatomy

The female urethra is 4 to 5 cm long. It extends downwards and forwards from the neck of the bladder and ends in the external urethral orifice in front of the vagina. It is closely adherent to the anterior vaginal wall. In its course it pierces the triangular ligament. Within the layers of this ligament there is a voluntary muscle surrounding it, acting as a compressor of the urethra. The st of the urethra proximally is surrounded by voluntary muscular fibres, as in the male. Normally the female urethra should admit a 28 French urethral dilator. Herman considers that 29 French (diameter 9 m.m.) should go in easily. In the elderly woman, due to fibrosis, the maximum diameter of the normal urethra may be 7 m. m. (21 French). If inflamation is superadded, it may be much less I have seen, in a cadaver, an urethra admitting easily the thumb.

Causes of Obstruction in the Urethra.

Meatal strictures occur after ulceration and fibrosis due to syphilis, chancre and lymphopathia venereum. In the last, it is often associated with considerable scarring of the adjacent parts of the vagina and vulva and the anorectum. Occasionally a calculus, usually originating in the upper urinary tract may be lodged in the urethra. It is rare for a stone to be formed in a urethral diverticulum. The calculi when present can be palpated and usually easily expressed by digital pressure through the vagina, in most cases. Pedunculated tumours of the bladder have very occasion-

ally been a cause of retention. A benign fibrosis in the region of the neck of the bladder in the female has been known to produce an obstruction to the urinary flow (like the medium bar in the male). Transurethral resection is done for the condition.

Malignant Growths Causing Obstruction :–

A carcinoma of the neck of the bladder may cause obstruction at the proximal end of the urethra. Extension of malignant growths from the adjoining parts, vulva and cervix may infiltrate the urethra and obstruct it.

Pressure on the Urethra from Without :–

By far the most common causes of retention are an impacted gravid retroflexed uterus and a fibroid. The retroflexed uterus giving the symptoms is usually 14 to 16 weeks pregnant. The organ rises up from the pelvis after the fourth month of pregnancy and so this condition will not be seen after that period. Two swellings will be palpated, one the distended bladder as a midline tumour above the symphysis pubis and another the gravid uterus per vaginum. Occasionally, a pelvic abscess or a haematoma or a pelvic ovarian cyst, occupying the pouch of Douglas may also produce the same symsptoms and make diagnosis difficult. After the bladder is emptied with a catheter, a re-examination of the pelvis will reveal the exact condition. Fibromyomata on the anterior or posterior wall of the uterus may produce impaction of the organ in the pelvis and cause similar symptoms. Novak mentions two cases of acute retention following a failure to relieve the desire to void when it was first experienced. The cause in the first case was a gravid uterus with a myomatous nodule in the anterior wall and in the second, a spinister, a myoma found in the anterior wall. The patients, with such tumour masses within the pelvis causing acute obstruction to the urinary flow, present certain signs. The cervix is drawn up and may even come to be placed behind the symphysis pubis. So there is an elongation of the anterior wall of the vagina. The anterior fornix is so displaced that it cannot be reached with the examining finger. The external urethral meatus retracts into the vagina and only a slit may be visible. Consequently catheterisation will be difficult. Treatment consists in passing and fixing a catheter. Only very slow decompression of the bladder will lead to a gradual return of the uterus to its usual position–anterversion and anteflexion. When an indwelling catheter is used, calcium Mandelate must be given orally. The modern tendency is not to use an indwelling catheter but to pass a catheter when there is the urge to micturate. When these fail, correction of the retroverted uterus in the knee-chest position must be done. Myomata, of course, are remo-

ved surgically at the earliest possible time after the patient is relieved of the retention.

Post-operative causes of retention of urine:–

Operative procedures such as abdominal and vaginal plastic operations cause a reflex acute retention of urine. Repair of enteroceles, by interfering with the nerve supply of the bladder may cause retention for a short time. Frequent catheterisation or continuous drainage of the bladder will have to be resorted to. At Johns Hopkins, before the patient leaves the operation table 30 c.c. of 0.5% aqueous solution of mercurochrome is instilled into the bladder. Craig who instituted the routine believed that the mercurochrome caused a chemical irritation of the bladder resulting in an earlier desire to void. This instillation once a day is suggested even if there is repeated catheterisation or there is an indwelling catheter (Novak). Mercurochromie disinfects the residual urine.

Psyhchiatric Causes :–

Hysteria among them may produce acute retention of urine, though it is rare nowadays. One should guard against making the mistake of diagnosing a pregnancy or an ovarian tumour, in such cases. Treatment is psychiatric.

Reference: Novak E. and Novak E.R. Text Book of Gynaecology IV edn. Williams and Wilkins and company. Baltimore 1952.

Meetings held for the year 1956—57

Date	Speaker	Subject	
3rd March '56	Dr. P. Arunachalam, M. D. D. M. R., T. D. D.	Some Fundamental Aspects of Kidney Disease.	Tea
31st March '56	Dr. V. Krishnamurthy	"Radium"	Tea
21st April '56	Dr. C. S. Sadasivam, M. S.	Some Common Surgical Pediatric Problems and the Infant.	Dinner
26th May '56	Dr. K. P. Ganesan, M. D.	Cerebro Vascular Accidents	Dinner
	Symposium		
23rd June '56	Dr. C. K. Padmanaba Menon M. S. F. R. C. S. (Eng) on Surgery		
	Dr. N. G. Pandalai, M. D., D. T. M. F. R. C..P. on Bacteriology		Dinner
	Dr. K. Ramachandra, M. D. on Medicine	Lower Respiratory tract infection	
	Dr. K. A. Kalyanam, M. B. B. S., D. M. R. on Pathology		
26th July '56 Picnic At. Cumbakarai	Dr. V. Sreenivasan, M. R. C. P.	Treatment of Comas	Lunch
18th August '56	Dr. K. C. Nambiar, F R. C S. (Eng) F. R. C. S. (Edin)	Low Back Ache	Tea & Dinner
22nd September '56	Dr. K. Ramachandra, M. D. Dr. K. N. Vasudevan, M. S.	Medical Aspects of Diarrhoea	Tea
27th October '56	Dr. Augustus Asirvatham, B. A., M.S.	Minor Surgical Problems	Tea
24th November	Dr. A. Krishnaswamy, M. B. B. S., D. M. R.	Principles of Radiation therapy	Tea
22nd December	Dr. H. A. Satya Joseph, M. B. B. S., M. R. C. P. D. T. T. M. & H.	Bacterial (other than tuberculous (infections of kidney	Tea
12th January '56	Dr. V. Sreenivasan M. Sc; M. B. B. S. Ph D.	Electro-phoresis As An Aid in Diognosis	Dinner

Annual Report for the Year 1956—57

DR. K. A. RAMALINGAM M.B.B.S.

Mr. President, Ladies and Gentlemen,

It is with very great pleasure that I present this annual report of the Madura Branch of the Madras State Branch of the Indian Medical Association.

It is indeed gratifying that during the year under review, our Association had carried out all its functions in a very smooth manner and the record of its activities is no less imposing than in the previous years.

Membership :

We are legitimately proud that our membership during the year has risen from 180 to 230, and that we have the largest membership among all the State Branches. But there are still a number of medical men and women, who are not members of our Association. I take this opportunity to appeal to all such medical men and women to join our association so as to make it a fully representative body.

We are also proud to have many eminent surgeons and physicans in our ranks.

Meetings :

We have been holding meetings egularly every month and the attendance at these meetings has been uniformly very good. As a matter of fact, the present Association Hall built to accommodate 80 members is now found to be very inadequate for the growing membership of the Association ; and the Office-bearers have already embarked on plans to collect donations for the purpose of erecting a new and bigger hall in the adjoining lawn. The need for a bigger hall is so real and immediate that I appeal to all the members of the Association to contribute generously towards this building fund and thus help the office-bearers in their efforts.

At the monthly meetings, we have had the privilege of being addressed by learned lecturers ; some of whom have attained pre-eminence in some branch of Medical Science or other. A few of our members, who are general medical practitioners, have been heard to remark about the too academic nature of some of the lectures and discussions, having no direct bearing on problems of every-day Practice. But I feel, that in these days of nuclear fission and radio-active isotopes, we are likely to be left in the background if we confine ourselves only to day-to-day problems instead of trying to keep in touch with the recent advances in Medical Science.

After a lapse of four years, we had a picnic-cum-monthly meeting at Cumbakarai, a place about sixty miles away from Madurai and known for its beautiful waterfalls and refreshing climate. Those of our members who attended, had an opportunity to relax and to take part in competitive games and sports which formed a special feature of the occasion.

During the year, I was able to arrange six open-air Dinner meetings during which the members had the opportunity to freely mingle and fraternise with each other. My thanks are due to all the members who, by their unstinted enthusiasm, contributed to the success of these dinner meetings. My thanks are also due to Messrs. The South Indian Manufacturing Co., Madurai, The Voltas & Company, and Ravison Pharmaceuticals, and Squibb, who so generously acceded to my requests and provided us with those grand dinners.

Our monthly meetings were almost always made more interesting by the screening of instructive films on topics of medical interest. My thanks are due to the various Pharmaceutical companies who made those films available, and also to the Health Department of the Madurai Municipality for the ready and generous loan of their projector and operating personnel.

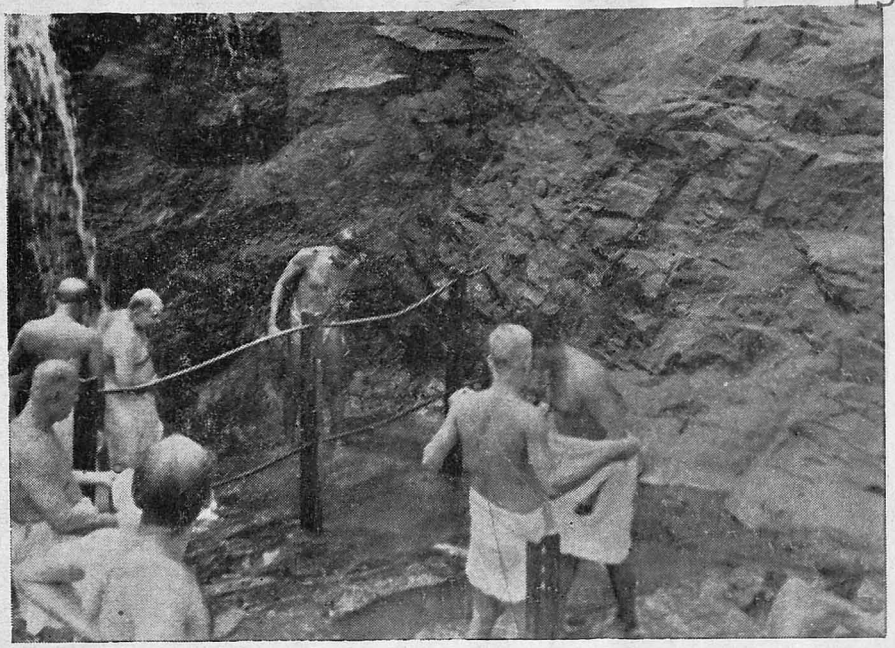
A happening of note during the year was the holding of a Provincial Council meeting at Madurai in the month of August, after a lapse of a number of years. Our Provincial Council members have been attending the Council meetings very regularly and we are very thankful to Drs. G. A. Naidu, Abdul Sathar, K. Gopal, and S. Nataraja Pillai for attending all the meetings and voicing the views of our branch so effectively.

Higher Examinations :

It gives me very great pleasure to record on this occasion the achievement of three of our members in the field of higher studies. I refer to Dr. K. A. Kalyanam who has obtained the Fellowship of the Royal College of Surgeons, Edinburgh, and to Drs. S. Shanmugham, & P. Krishna Menon who have been successful in the Primary Fellowship examination of the Royal College of Surgeons London held recently in Ceylon. On behalf of the Association, I congratulate them most heartily on their brilliant success. It is my sincere hope that more and more of our members will strive for and achieve success in higher examinations thus bringing great honour and prestige to our Association.

Journals

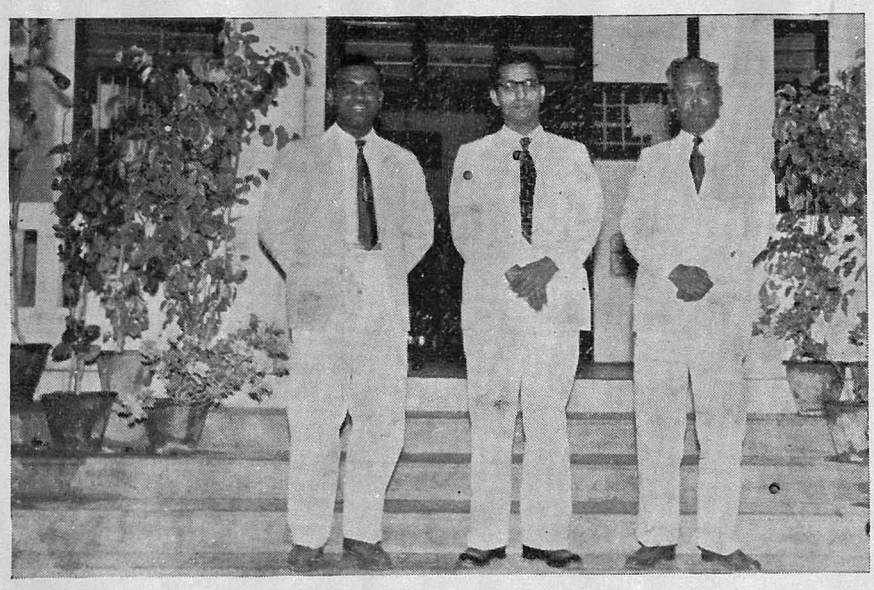
Our Association is subscribing to the following journals : British Medical Journal, British Journal of Ophthamology, British Journal of Surgery, Surgery, Gynaecology and Obstetrics, The Lancet, Practitioner and Antiseptic. We realise that the members



Spot the surgeon at the Cataract.

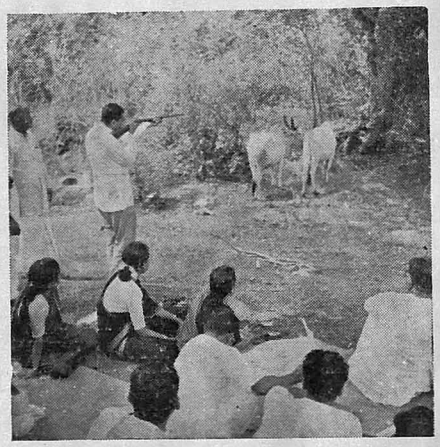
To the Health of Dr. Vethachalam.

Photo : Dr. S. Bhuvaneshwar



Dr. K. P. Ganesan with our Major & " Minor "

" Au Revoir "



Bull's Eye at Cumbakarai

Photo : Dr. S. Bhuvaneshwar

are not deriving the maximum benefit from these journals by keeping them in our Association hall, where the members can go and read them. We also realise that it will be far more useful to the members if only the journals can be circulated among them. But at present we are handicapped by the fact that we do not have sufficient staff to look after the work of circulating these journals in addition to the routine work of the Association.

Quacks and Quackery :

As an Association we have always been against quacks and quackery in any form. And our Association launched such an active campaign against a quack in Periakulam that he had to leave the place. The very fact that there was only one such instance shows how indifferent we are towards this widely prevalent evil. On behalf of the association, I appeal to all the members of our branch to do their duty by bringing to the notice of the Association specific instances of quackery that they may come across so as to enable the Association to take steps to eradicate this evil.

Appeal for Funds :

Once again, I appeal to the members to contribute generously and help the office-bearers to build a new and bigger Association hall. I also appeal to them for funds for the purpose of converting the land behind the present hall into a well-laid out and beautiful garden.

I took up the office of Secretaryship with all its great responsibilities, in a spirit of humility and service. I have endeavoured to enhance the prestige of our Association and to bring about a spirit of greater fellowship among the members. What little success I might have achieved is entirely due to the kind co-operation, extended to me by each and every member of the Association. My special thanks are due to Dr. S. V. K. S. Thangarajan who by his untiring efforts, has brought out this souvenir with such unique features. My thanks are also due to the Office-Staff who faithfully carried out all duties entrusted to them.

R. Seshan, G.D.A., F.C.A.,
CHARTERED ACCOUNTANT

4. Chockappa Naidu Street,
Madurai 21—1—57

To
The Honorary Secretary,
The Madurai Medical Association,
Madurai.

Sir,

1. ACCOUNTS OF THE ASSOCIATION FOR THE YEAR ENDED 31--12--56.

I enclose the cash receipts and payments account of the Association for the year ended 31st December 1956 and report on the accounts as follows :—

2. Cash on hand and the banks and in fixed — deposits : I have verified the cash on hand and in banks and found the same in order.

3. INVENTORY : An inventory for moveables and a register for books are kept and written to date.

4. ARREARS OF SUBSCRIPTION : The arrears of subscription amounted to Rs. 999—8—0 of which Rs. 72/-, I am informed, have been since received ; advance subscription amount to Rs. 33/-. Of the arrears, Rs. 432—8—0 pertain to deceased, removed, resigned and transferred members. During the year couple members have been charged at a rate of Rs. 3/- per mensem.

5. DONATIONS :

(a) *Silver Jubilee donations* : As per my previous report silver jubilee donations promised but unpaid Rs. 135/-. Received in the account year Nil.

(b) *Building fund donations* : Received in the account year Rs. 10/- from a new member. Donations promised but unpaid Rs. 2761/-. These donations arrears are long overdue.

(c) *Rajkumari Amirt Kaur Reception Receipt* : In my previous report I stated that a cheque of Rs. 15/- which was received was not collected and was taken off the books and that the uncashed cheque was not shown to me ; No further particulars are available regarding this cheque.

6. OUTSTANDING LIABILITIES AND ASSETS.

(a) Property taxes : Municipal property tax paid up to 30-9-56.
(Municipal receipts for taxes paid for the two half years ending 31-3-56 (Rs. 118-12-0) were not made available during audit).

(b) Electric bill for December 1956 payable.

(c) Salaries paid up to December 1956.

(d) Contribution to Indian Medical Association paid for the members for the half year (first) 1956-57 and for 13 members for the second half year 1956-57.

7. Amount due to the association or amounts prepaid as on 31-12-1956.

(a) Prepaid subscription in respect of One Magazine up to March 1957
FourMagazines up to May 1957
One Magazine up to September 1957.

(b) Cycle license paid to 31-3-1957.

(c) Arrears of subscription — please see para 4 supra.

(d) 1957 Souvenir payments—Rs. 9-15-9.

(e) Due from Secretary — Rs. 35/-.

(f) Interest on fixed deposit for the year ending 31-12-56.

8. Comparison of accounts.

	Year 1956	*Year* 1955
Income receipts	Rs. 5452	Rs. 3432
Revenue expediture	Rs. 5732	Rs. 3662

Increase in income receipts due to increase in subscriptions ; as also due to souvenir advertisement receipts and donations for dinner which were absent in the year 1955.

Increase in expenditure due to rise in almost all the expenses and emergence of new expenses such as election expenses and building repairs as compared to the year 1955.

9. Nineth State Madras Medical Conference Souvenir Arrear :

Arrears due from one advertiser (Santex Chemical Industries Baroda) in the above Conference Souvenir not yet received. This arrear is long over due.

10. Value of assets and property of the Association as on 31-12-56.

(a) Building and furniture :

Cost of expenditure on building and furniture as on 1-1-56	Rs. 30,320-14-1
Addition during the year — chairs, table and platform.	483-14-0
Cost of expenditure on building and cost of furniture as on 31-12-56.	
Total	Rs. 30,804-12-1

(b) Cost of equipment as on 1-1-56 228- 2-0

Addition of cycle during the year 212- 8-9

Total Rs. 440-10-9

The above values are without any depreciation written off as also not taking into account the value of the cycle sold during the year.

11. General :

(a) 1956 *Souvenir Advertisement arrear* : Arrear due for half page advertisement from Standard Pharmaceuticals Works Ltd., Calcutta. Steps may be taken to collect this arrear. One advertisement in the Souvenir is stated to be free.

(b) *Interest on fixed deposit* : Interest receivable for two half yearly rests ending 31-12-56 has not been received and recorded in the books.

(c) *Clerk's security deposit* : I suggest that this deposit of Rs. 50/- map be invested separately.

(d) *Property tax* for the Association building remains to be Rs. 59-6-0 per half year and the Municipality may be requested to reduce the tax, the nature and object of the Association being explained to it.

(e) *Members and subscription Register* : Members may be requested to sign the subscription register on paying the subscription so that there need be no complaint of any member paying his or her dues and not being credited with the amount.

(f) *Double debit for a single payment* : While a payment for a printing

bill of Rs. 16/- is recorded in election expenses account, a similar amount has again been debited for the same bill in printing charges account though I am informed the printer has been correctly paid Rs. 16/- only. This excess debit of Rs. 16/- to printing charges account has to be — rectified and Rs. 16/- have to be credited to the accounts.

(g) *Clerk and his appointment*: The Governing Body has by its proceedings dated 10-4-56 decided to appoint Mr. Krishnamurthi the present clerk at Rs. 50/- per mensem. But Mr. Krishnamoorthi has been paid for the period of his service from 13-3-55 to 31-3-55 at the rate of Rs. 40/- per mensem. This payment is to be approved.

Whenever a person is appointed, the date from which his service is to run may also be kindly minuted.

(h) *Payments*: The payments are supported either by vouchers or certificates of payments. For certain payments bills or receipts are available when both should be available ; in certain cases acknowledgement of payment for sums exceeding Rs. 20/- have not been stamped.

(i) *Conclusion*: The books have been well kept and I was given the necessary information by the Secretary, Treasurer and the clerk Sri Krishnamoorthy.

Yours faithfully,
K. Seshan.

Reply To Auditor's Report.

Subjects for moving in the general body Item 4. arrears of subscription of Rs. 432—8—0 from deceased, removed and resigned members. The General body approves of writing off this debit Rs. 432-8-0 since it is not possible to collect them.

Item 5 (a) **Sivler Jubilee Donation** (1953) unpaid Rs. 135/-. The General Body approves of writing off the unpaid amount of Rs. 135/- due from Silver Jubilee Donation.

Building Fund Donation

Unpaid amount of Rs. 2761. The General Body approves of writing off the whole amount of Rs. 2761, since it is found it is impossible to recover.

Secretary's reply to the Auditor's report :-

The Auditor's report is thoroughly correct and his remarks of non-collect arrears accruing from Silver jubilee donation and building fund donation is a matter of great concern to me. I find it is not possible to collect them due to lapse of long time. Regarding the Rajkummari Amrit Kaur reception receipt of Rs. 15, I enquired my predecessor and found for no-body's fault it is missing. The remark on double debit for a single payment of Rs. 16, it is not a wise policy to pursue the matter further.

THE MADURA MEDICAL ASSOCIATION, MADURAI.

Cash Receipts and Payments Account for the Year ended 31st December 1956

RECEIPTS

			Rs.	As.	Ps.	Rs.	As	Ps.
A	**Opening Balances**							
	(1) Cash on hand with the Treasurer	...	50	3	9			
	(2) Cash on hand with the Secretary	...	15	0	0			
	(3) Building Fund Investment in S. B. A/c. with the Indian Bank Ltd.	...	42	8	2			
	(4) General Fund investments							
	(*a*) in current a/c with the Indian Bank Ltd.	...	817	2	1			
	(*b*) in S. B. a/c with the Indian Bank Ltd.	...	217	12	4	1142	10	4
B	**Income Receipts during the year**							
	Subscriptions	...	4174	4	0			
	Sale of list of members	...	32	0	0			
	1956 Souvenir advertisement Receipts	...	515	0	0			
	Donations for Dinner	...	658	7	0			
	Interest on fixed deposit and Savings Bank accounts	...	41	4	0			
	Miscellaneous	...	13	12	0			
	Building Fund Donation	...	10	0	0			
	Receipt from ex-secretary	...	6	14	0	5451	9	0
C	**Suspense Receipts**							
	Flood Relief Donation	...	30	0	0			
	Security Deposit received from new clerk	...	50	0	0	80	0	0
D	**Capital Receipts**							
	Sale Proceeds of old cycle	...				35	0	0
E	**Transfer**							
	Transfer of Balances of Nineth Madras State Medical Conference	...				1854	1	0
	Total	...				8563	4	4

PAYMENTS

			Rs.	As.	Ps.	Rs.	As.	Ps
A	**Revenue Payments**							
	Salaries and Bonus							
	To clerk	...	688	8	0			
	To Gardener	...	415	0	0			
	To extra salary to ex-clerk	...	63	0	0			
	To Bonus to staff	...	72	0	0	1238	8	0
	Socials and Meetings	...				1266	9	6
	Building Repairs	...				249	9	9
	Subscription to Medical Magazines	...				355	0	0
	1956 Souvenir expenses	...				180	0	0
	Election expenses	...				142	14	3
	Contribution to I. M. A.	...				1626	0	0
	Printing charges	...				205	15	6
	Miscellaneous	...				58	12	9
	Postage	...				121	4	6
	Lighting charges	...				29	15	4
	Property Taxes	...				178	2	0
	Audit fees	...				30	0	0
	Stationery	—				49	4	0
	Total Revenue Payments	...				5731	15	7
B	**Capital Expenses**							
	Purchase of cycle	...	212	8	9			
	Chairs and Table	...	391	0	3			
	Platform and mat	...	92	13	9	696	6	9
C	**Suspense Payments**							
	Flood Relief Fund Donation collected remitted	...	350	0	0			
	Refund of security Deposit to ex-clerk	...	50	0	0	400	0	0
D	**Advances and Prepaid Expenses**							
	Advance to Secretary	...	35	0	0			
	1957 Souvenir Payments	...	9	15	9	44	15	9
	(1) Cash on hand with the Treasurer	...	36	0	8			
	(2) Cash on hand with the Secretary	...	15	0	0			
	(3) Building Fund Investment in S. B. A/c. with Indian Bank Ltd.	...	43	5	0			
	(4) General Fund Investment in							
	(*a*) Current a/c. with the Indian Bank Ltd.	...	358	8	1			
	(*b*) In S. B. a/c. with the Indian Bank Ltd.	...	221	15	6			
	(*c*) In current a/c. with the Pandyan Bank Ltd.	...	15	1	0			
	(*d*) In fixed deposit in Pandyan Bank Ltd.	...	1000	0	0	1689	14	3
	Total	...				8563	4	4

Examined and found corrct subject to my report of date.

Madurai
21—1—1957

R. SESHAN
Chartered Accountant

BUDGET FOR THE YEAR 1957.

INCOME	Rs.	As.	Ps.	EXPENDITURE	Rs.	Aa.	Ps.
Subscriptions :				**Central Fund Contribution**			
160 Resident Members	3840	0	0	206 Plus 25 New members	1,848	0	0
45 Non-Resident ,,	405	0	0				
				Salaries			
Expected New Members :				Clerk 55 / 12	660	0	0
20 Resident members	480	0	0	Garden 30 / 12	360	0	0
5 N-Resident	45	0	0	Bonus	85	0	0
Interest	60	0	0	Souvenir Expenses 1957	800	0	0
Souvenir 1957	1,500	0	0				
				Maintenance of the Building :			
Building Fund Donation	1,000	0	0	Tax, Electricity, Repair Etc.	300	0	0
				Journals	350	0	0
				Social	250	0	0
				Postal	80	0	0
				Printing	80	0	0
				Typewriter (New)	400	0	0
				Stationery	40	0	0
				Audit Fees	40	0	0
				Furniture Repairs	20	0	0
				Cycle repairs	23	0	0
				Library Hall Construction Ist stage.	2,000	0	0.
Total Rs.	7,336	0	0	Total Rs.	7,336	0	0