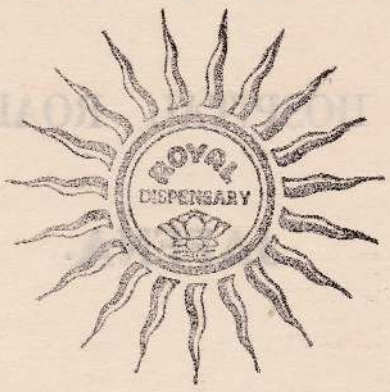
THE JAFFNA MEDICAL JOURNAL

VOL. XIV NO. 1 MARCH 1979



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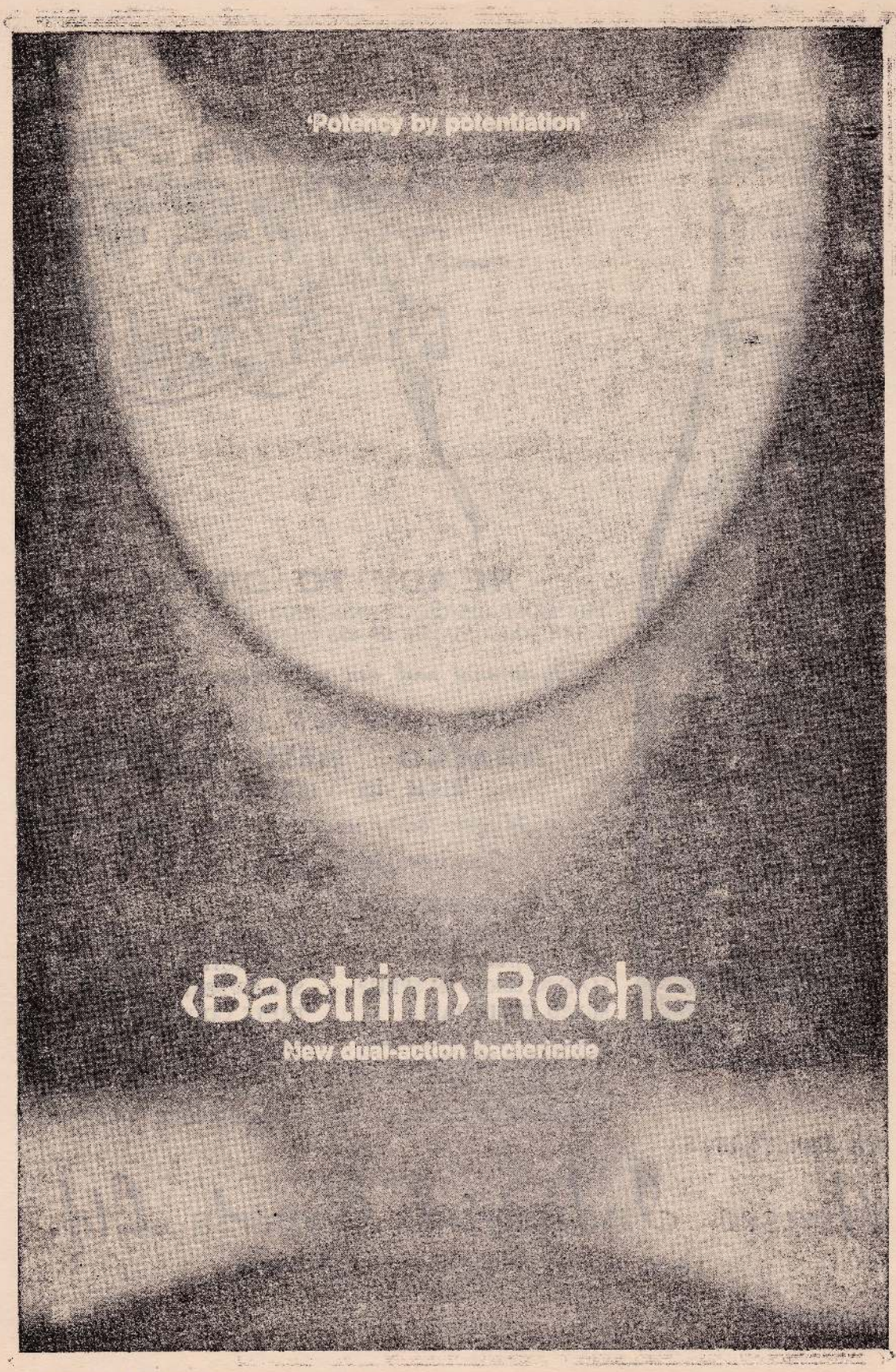

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THE JAFFNA MEDICAL JOURNAL

(THE OFFICIAL PUBLICATION OF THE JAFFNA MEDICAL ASSOCIATION)

VOL. XIV, No. 1 MARCH 1979

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Editorial....

A Medical School in Jaffna

On the 8th of October 1978 the medical school at Kaithady, Jaffna was ceremonially declared open by the Hon. Minister of Education Dr. Nissanka Wijeratne in the presence of a large gathering of university teachers, doctors and members of the public. The minister in his address recalled the long tradition of learning in the peninsula and said that the present inauguration was not a new beginning but a continuation of medical education from our historic past. He reminded the audience of the medical school founded in 1848 at Manipay by the American Missionary Dr. Samuel F. Green and said that the distinguished doctors who graduated in this school served both in Sri Lanka and abroad. A policy decision made then shifted the scene of medical education to Colombo, and the medical school in Colombo was founded in 1870. The minister described the present context as "home coming" rather than a new start.

The history of medical education and medical practice in Jaffna is of importance and interest to all. Before the advent of Western medicine there existed in the island, indigenous systems of medicine known as Ayurveda and Siddha systems the latter being of South Indian origin.

Like the Greeks who believed that the art of healing originated from Appolo, our ancients believed in the divine origin of their knowledge and believed that Brahma the source of all knowledge blessed them with this system of medicine.

In Jaffna during the reign of Varodaya Segarajasekaran in the 14th contury A. D. a Tamil Medical text book "Segarajasekaram" was compiled. In its section on anatomy the author describes the dissection of dead bodies killed in battle. In the 15th century another medical work "Pararajasekaram" was compiled under the patronage of King Pararajasekaran. This is a comprehensive work on Ayurveda and Siddha systems.

The medical school founded in Jaffna marks an important phase in the development of our health services. The establishment of several autonomous universities is a land mark in the history of education in this country. The Jaffna Medical Association welcomes this policy of the government.

Editorial . . . .

Psychiatric Services — Some thoughts about planning

THE Mental Health Act of 1959 paved the way for the practice of Psychological Medicine in General Hospitals of the United Kingdom. Although similar legislation has still not been passed in Sri Lanka, similar Units have been in existence for more than a decade in many parts of the country. General Hospital practice and its impact on the service as a whole deserves careful examination, so that a policy and a plan can be formulated for the future.

Practice in General Hospitals of Sri Lanka is largely influenced by the absence of a long term Mental Hospital for the area where certification and compulsory treatment is possible. In practice the Units in General Hospitals admit and treat patients who are devoid of insight to give valid consent for treatment. This is usually done with the approval of well meaning relatives who are grateful for having spared them of the difficulties of taking the patient to M. H. Angoda. However the legal risks involved are not inconsiderable and this position is likely to continue until the proposed Mental Health Act is passed in Parliament.

Apart from the population discussed above, a new category of patients are seen in General Hospitals. While the former group with mainly disordered or disturbed behaviour reach the Psychiatrist through Courts, Police and Social Agencies, the latter group are always referred by the General Medical Services. They are likely to remain undetected and untreated in the absence of treatment facilities in the General Hospital.

It has now been realised that a relatively high proportion of the population at large suffers from psychiatric disorder and only a small fraction of it comes to the attention of the Psychiatrist. Therefore, it is not surprising that the hopes of our Medical Administrators to reduce congestion at M. H. Angoda by opening the Provincial Units is likely to remain unfulfilled though steps are certainly in the right direction. Recent reports suggest that approximately 10 % of patients admitted to a Medical Unit are referred for psychiatric consultations (Crisp A. H.1969). In view of the association of psychiatric and somatic illnesses especially in later life the need for double diagnosis in medicine has been stressed (Martin Roth 1974). While undetected depressions, anxiety states and the diseases of the elderly form the bulk of referrals from the Medical wards opinions are frequently sought in psychiatric complications of chronic physical disease. The management of "parasuicide" and alcoholism have become a major problems in

these General Hospital Units. Increasing demands are made on the time and effort of the Psychiatrist in the treatment of sexual disorders. Shortage of beds, over crowded clinics, lack of trained staff and equipment pose major problems to the practising Psychiatrist.

Though many of these shortcomings are common to the rest of the Health Services, some at least can be remedied without additional expenditure. A practical plan has to be formulated without further delay.

Planning and organization of Psychiatric Services in a developing country is beset with many difficulties. Much information has been gained in the recent past on the epidemiology and natural history of many mental disorders. In a country where trained Psychiatrists are few in number, priorities have to be clearly worked out and the point prevalence rate alone is of little significance in planning (Giel R and Harding TW 1976). It is futile to adopt planning strategies applied in more developed countries like the United Kingdom.

Due to the long tradition of government sponsored welfare services in Sri Lanka both in the sphere of Education and Health, expectations of our people are different from other underdeveloped countries. The methods applied in these countries are inappropriate in Sri Lanka.

Prevalence rate of psychoses in Sri Lanka varies within very narrow limits from rates obtained in other parts of the world (Wijesinghe C Petal 1975). The period prevalence rate (in a study lasting for six months) which was expected to approximate to point prevalence rate for Schizophrenia was 3·8/1000 (Wijesinghe C Petal 1975). Schizophernia is a disorder that causes much concern to the family and society due to its disorganising and disruptive nature and chronic course. Treatment with phenothiozines causes substantial beneficial effect in course and outcome and therefore should receive the highest priority in the course of planning.

Much of the treatment of patients suffering from neuroses has necessarily to be undertaken by the general practitioner and the District Medical Officer. The main priority in this area is to recognise the large proportion of patients attending curative services with physical symptoms without an organic cause so that further inappropriate investigation and treatment can be limited (Giel R Harding TW 1976).

It has been pointed out that 94.5% of patients suffering from active psychiatric disorder were not receiving any attention (Wijesinghe C Petal 1975). Shortages and interruptions in the supply of drugs to diagnosed patients contitutes a serious problem in follow-up clinics. Patients are compelled to travel as much as 50—75 miles in some instances to obtain their maintenance doses from specialist clinics in Provincial Hospitals. It is suggested that a restricted list of essential psychotropic drugs should be allocated to District Hospitals and Peripheral Units. District Medical Officers and Medical Officers in charge of Peripheral Units should be entrusted with the care of patients discharged from psychiatric wards or clinics in Provincial Hospitals. It is hoped that in the course of time a system of referral will develop between the District Hospital and the specialist clinics resulting in better case detection.

Planning is essentially an exercise in obtaining a long term view of development. The population for the District of Jaffna

is 701,603 (Census Population of Sri Lanka General Report 1971). Approximately a hundred beds are available for the treatment of mental illness. Patients from Mannar District and Vavuniya District each with a population of 77,786 and 95,243 respectively make use of the meagre facilities available in Jaffna. The facilities available are inadequate to treat even the number of patients with acute and subacute functional psychoses that seek treatment, let alone the number prevalent at any point of time.

A long term target should be fixed for at least one Psychiatrist per 100,000 general population. In the United Kingdom as mentioned by Parkes, DHSS recommend a ratio, of 1 per 60,000 general population as minimum requirement to provide a satisfactory service. Adverse effects of psychiatric morbidity on the family and society and on the economic life of the community needs no further elaboration here.

If optimal development is to take place within a reasonable period of time the teaching Departments in our Medical Schools should be given the facilities to train not only undergraduates but also postgraduate students in Psychiatry. Also it is from such Centres can we hope to have the large number of personnel trained to carry out psychiatric social work in the community.

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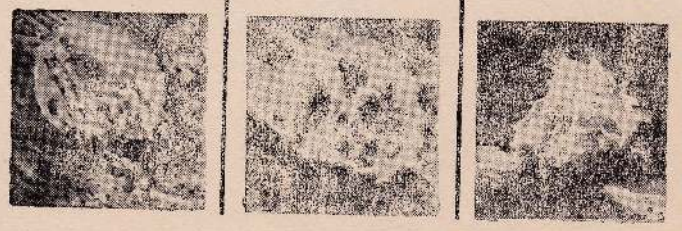

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Presidential Address — 1976 — 1977

CARDIAC SURGERY IN A PROVINCIAL HOSPITAL

R. NATKUNAM, F. R. C. S.

LADIES AND GENTLEMEN,

ONE year ago you entrusted me with the Presidentship of the Jaffna Medical Association. The obligation to give a Presidential Address that goes along with this honour did cast a grim shadow but there was still twelve months more for that and I felt very elated. These twelve months have rushed past, unfortunately a part of this period has been fritted away in an unpleasant and indolent trade union action and now I am faced with the ordeal of giving the lecture. I can do no better than follow the worthy example of the Presidents before me and review in outline some aspects of my work in this hospital.

Even pre-historic man knew how vital an organ was the heart. It was the bull's-eye for the hunter. The ancient Greeks regarded it as a centre of life and the seat of intelligence, and any interference with this organ was a sacrilege. A somewhat fatalistic attitude towards heart disease persists to this day. In the development of cardiac surgery, perhaps the most significant advance came in 1628 with William Harvey's discovery of the blood circulation. Then the 19th century brought great changes in surgery; anasthesia, antisepsis and asepsis were giant steps taken during this period. In 1904 Ferdinand Sauerbruch solved the problem of pneumothorax which had, till then, been the barrier, to any thoracic operation. Once this barrier was conquered thoracic surgery became the most vigorously growing field of surgery in the 20th century. This was made possible by almost the feverish activity of scientists in various disciplines and has resulted in the use of X-rays electrocardiography, cardiac catheter studies, angiocardiography and echocardiography so that it is now possible to make a precise anatomical diagnosis in cardiac disorders. The range of operative intervention upon the heart has been vastly increased by the development of the heart lung machine by Gibbon in 1953. Thus, has the old order changed although, we in Jaffna, unaided by many of these sophisticated instruments, have had to rely for diagnosis more on that ancient, nevertheless important, art of simply observing the patient!

Of over 600 major operations performed in the Thoracic unit in the five year period ending in December 1976, 189 operations were cardiac surgical procedures.

SLIDE I

Disease	No. of Cases
Mitral Stenosis	131
A S D	32
P D A	18
Miscellaneous	8
Total	189

The miscellaneous group included 4 with Fallot's tetralogy on whom Blalock procedures were done, 2 patients in whom fixed rate induction-coil pacemakers were inserted, one patient who had a repair of an aorto-pulmonary window and one other with persistent pericardial effusion associated with myxoedema on whom a pericardiectomy was done. These cases were presented at several clinical meetings of this Association during the course of the last five years.

Mitral Stenosis

Next to operations for carcinoma of the oesophagus, mitral valvotomy has been the commonest surgical procedure performed in this unit. The stenosis was the result of rheumatic heart disease i. e. we had no congenital mitral stenosis.

SLIDE II

Age	Under 20 yrs.	21 to 40 yrs.	Over 40	Total
Females	36	30	4	70
Males	33	19	3	35
Total	69	49	7	125

There are two features worthy of note in this slide. Firstly the sex distribution. The greater predilection of the female to the disease is a feature noted in the West. Paul (1966) demonstrated that in the mitral stenotic child both sexes were equally represented but stated that the characteristic female dominance, typical of the West, was seen in his adult patients. In Jaffna, the distribution of the disease has been roughly equal in the two sexes and this feature was common to the child as well as the adult. A second point worthy of note is that critical mitral stenosis requiring surgical relief has a peak incidence in the second decade. This again contrasts with the findings in the West where the peak incidence is in the 4th decade.

While rheumatic fever itself afflicts children of the same age group both in the East and in the West, rheumatic carditis with critical stenosis develops much more rapidly in the economically poorer countries. The reason for this accelerated evolution of the pathologic process in these latter countries is not known. In passing I would like to state that poor growth is a feature of the mitral stenotic child. A

satisfying aspect of surgery in the child is the improvement in growth resulting from the improved cardiac output consequent to the valvotomy. Poor growth should therefore be considered an indication for early surgery so that the full growth potential of that child may be realised before it passes the growing age.

Pregnancy and Mitral valvotomy

Twelve of the patients in this series had their valvotomy performed during pregnancy. Our policy has been to manage patients who are symptomatic for the first time during the pregnancy (i. e. those who were symptom-free before the pregnancy) with bed rest, digitalis, salt restriction and diuretics and advise surgery only if these measures fail. In the group that had been symptomatic even before the onset of pregnancy, surgery is the treatment of choice. The procedure is well tolerated by both the mother and the foetus and we have had no hesitation in advising surgery.

Mortality

Three of our patients died—a mortality rate of 2.3%. One man of 45 died as a result of a tear of the left atrium which resulted in uncontrollable bleeding while the other two, a girl of 11 years and a woman of 54 years died 2—4 days after surgery. Both these patients had severe pulmonary hypertension. The operative relief of mitral stenosis by closed mitral valvotomy has proved one of the most satisfying intracardiac operations. Severe pulmonary hypertension, a heavily calcified valve and the production of serious mitral incompetence are factors that affect the results unfavourably.

Atrial Septal defect

It is in the treatment of A. S. D. that we most felt the lack of adequate facilities to investigate and assess the cases. Defects in the atrial septum include the ostium secundum defect, the ostium primum defect and the endocardial cushion defect. The secundum-type defects are among the most common cardiac malformations. We have had 32 cases coming up for surgery. 19 of them were females while 13 were males. Their ages ranged from 8 years to 43 years. In the majority of cases diagnosis was based mainly on clinical findings of a pulmonary systolic murmur, fixed widely split second sound in the pulmonary area together with the characteristic X-ray and E. C. G. findings. Cardiac catheterization was done in only a few cases. In 4 cases the clinical diagnosis of septum secundum defect was proved wrong at operation. One of them turned out to be a V.S.D., another an aorto-pulmonary window while 2 others were found to have septum primum defects.

Morbidity and Mortality

Of the 27 patients who are alive today, 1 patient has recurrence of symptoms. This patient, a girl of 19, was catheterized and was found to have a re-opening of the defect. At the initial operation she had a septum secundum defect with anomalous drainage of the right inferior pulmonary vein into the right atrium. She will need a reoperation with closure of the defect using a patch. Two of the patients in the septum secundum group died in the early post-operative period. Both had severe pulmonary hypertension. One patient died on the second post-operative day following on an air embolus.

Any child who on routine examination is found to have the signs of an ASD should be investigated and if the pulmonary to systemic flow is found to be more than 2 : 1, surgery should be advised. The risk in this group is minimal.

SLIDE III

Defect	No. of Cases	Mortality
Septum Secondum	26	2
Sinus Venosus defect	3	1
Septum Secondum with Anomalous Pulmonary Venous drainage	1	1
Septum Primum defect	2	2

Persistent Patent Ductus Arteriosus

P. D. A. is the result of the abnormal persistence into post-natal life of a ductus arteriosus. It is more common in girls than in boys; we had 14 girls and 4 boys. In all these patients it was possible to do a ligation of the ductus. We had no deaths in this group. One girl was clinically diagnosed as having a P. D. A. was, at operation, found to have in addition a pulmonary stenosis and a V. S. D.

Since the complications of even asymptomatic P. D. A. are serious (infective endocarditis and pulmonary hypertension) and because the mortality of the operation is low, surgery is advised in all cases.

Fallot's Tetralogy

There were 4 patients in this group on whom a Blalock operation of pulmonary artery to subclavian artery anastomosis was done. We lost one of these patients due to congestive cardiac failure. These patients ranged in age from 2 to 20 years. Though the current thinking on this condition is against the performance of this procedure especially in those over 4 years when a total correction would be the operation of choice, I have done this procedure because we have no facilities to do any better. The patients have benefited in that they became less dyspnoeic, and their p.c v dropped to near normal levels. (80% to 50% in one patient.)

With that I conclude the survey of the types of operation that had been done in the Thoracic unit of the Jaffna Hospital in the last 5 years. Having to perform these procedures with limited staff, poor facilities for investigation and assessment have caused great anxiety. This type of anxiety is well described by Ian Aird in his book "The training of a surgeon". He states "the surgeon's anxieties are particularly acute, his disappointments singularly oppressive. In moments of decision too, most surgeons feel a curious loneliness. When all appropriate advice has been taken from his colleagues and when all aspects of the problem have been discussed with his own staff, it is the surgeon himself who must make the final decision and in making it he necessarily stands apart from his colleagues in a kind of loneliness". Ian Aird continues to state "a certain resilience to disappointments is a requisite in surgery but resilience should not be cultivated too

far. An insensitive bouyancy is apt to blunt the conscience of the surgeon, a conscience to which he must always feel answerable". I again quote from Rudolph Matas on the surgeon's conscience. Speaking of failures he says "these are cruel moments. If in the course of an operation, the surgeon is assailed by violent emotions, the rapidity with which they follow each other and the physical exertion in which he is engaged absorb all his energy and suffice to divert his mind from all other preoccupations. These violent and often terrible experiences do not possess, however, the intensity of the more deliberate reflections which come on after the tragedy is over. Then the merciless, self-imposed cross examination begins, in which the surgeon, standing before the bar of his own conscience, asks himself what part of the disaster may be attributable to his own sins of commission or omission, or if the outcome of the tragedy is merely the result of an accident, that which no man can possibly alter or divert".

I have had these anxieties and depressions but I have not been overwhelmed by them because the burden has been shared by the enthusiastic anaesthetists and house-officers with whom I have had the pleasure of associating in this work. The devotion to duty of the nurses and the physiotherapists attached to the unit has also been a source of strength.

In conclusion let me thank you most warmly for inviting me to give this lecuture and for listening to me patiently.

MANAGEMENT OF OBSTRUCTIVE JAUNDICE ANALYSIS OF 16 CASES

M. GANESARATNAM, F. R. C. S., (Eng.)
General Surgeon

AND

R. GANESHAMOORTHY, F. F. A. R. C. S., (Eng.)
Anaesthetist

From the Departments of General Surgery and Anaesthesia, Jaffna General Hospital

Introduction :

JAUNDICE is conveniently classified into pre-hepatic (haemolytic), hepatocellular and obstructive types. The former two are termed 'medical' jaundice and treated conservatively. Obstructive jaundice can be either intra-hepatic or extra-hepatic. Intra-hepatic obstructive jaundice (due to cholestasis, enzyme defects, primary biliary cirrhosis etc.) is also treated conservatively, but a few of these cases, with dilated bile ducts are amenable to hepato-intestinal by-pass operation. Extra-hepatic obstructive jaundice is almost always treated surgically.

At times it is difficult to differentiate extra-hepatic from intra-hepatic obstructive jaundice. In the diagnosis of extra-hepatic jaundice, the following facts are important :

(a) history not suggestive of infective hepatitis.

(b) history suggestive of pancreatic carcinoma or gall stones.

(c) absent history of medication that causes intra-hepatic cholestasis e.g. : phenothiazines, methyl-testosterone.

(d) past history of cholecystitis or cholelithiasis.

(e) prolonged jaundice of intermittent nature.

(f) palpable gall bladder.

(g) radiological evidence of calculi in the gall bladder area.

(h) liver biopsy, a fall in the serum bilirubin level in response to steroids and SGOT/SGPT ratio.

Oral cholecystogram and intravenous cholangiogram cannot be done when the serum bilirubin level is more than 4—5 mg%. In better equiped hospitals, ultra-sound scanning, fibreoptic retrograde cholangiogram, scanning after rose-bengal, percutaneous transhepatic cholangiogram and coeliac axis angiogram are done to determine the type and site of obstruction.

The probelms of surgery in obstructive jaundice are :—

(a) **Maintenance of adequate perfusion and oxygen supply to liver during**

Anaesthesia. This is maintained by maintaining the blood pressure near normal and also by preventing anoxia during surgery.

(b) **Excessive bleeding due to lack of clotting factors**

1. Vitamin K dependent — Prothrombin, Factors VII, IX and X.
2. Non Vitamin K dependent—Fibrinogen and Factor V.

These factors are synthesised in the liver. Vitamin K is not absorbed due to the absence of bile salts in the intestine in patients with biliary obstruction and the liver damage [8] following biliary obstruction hinders the synthesis of all these factors.

Deficiency of Vitamin K dependent factors are improved by parenteral Vitamin K. In better equipped Hospitals stored clotting factors and fresh frozen plasma are available for use. The alternative is fresh blood transfusions.

(c) **Post operative renal failure**[1]

This can be minimised by i. v. Mannitol during surgery which maintains an adequate urine output.

(d) **Hepatic Coma:**— Building up the glycogen in the liver minimises hepatic damage during surgery. This can be achieved by dextrose and Insulin prior to surgery.

(e) **Ascending Cholangitis**[5]

(f) **Wound dehiscence and incisional hernia**[6]

(g) **Post operative pancreatitis**[1,9]

Materials and Methods:

TABLE I: CAUSES OF JAUNDICE

Causes of jaundice	Number of patients
Carcinoma of pancreas	6
Carcinoma of common bile duct (case 9)	1
Stones in the common bile duct	5
Obstruction at the lower end of common bile duct (case 6)	1
Amoebic abscess of right lobe of liver (case 7)	1
Chronic cholecystitis with an enlarged lymph node compressing the common bile duct (case 10)	1
Congenital stenosis of common bile duct with superadded cholecystitis (case 14)	1

TABLE II: AGE AND SEX DISTRIBUTION

Age (years)	Causes of jaundice	Total number F	M
0—10	Congenital stenosis of common bile duct (case 14)	1	nil
11—20	none		
21—30	Chronic cholecystitis with an enlarged lymph node obstructing the common bile duct (case 10)	1	nil
	Obstruction at the lower end of common duct (case 6)	nil	1
31—40	Amoebic abscess of right lobe of liver (case 7)	nil	1
41—50	Carcinoma of common bile duct (case 9)	nil	1
51—60	Carcinoma of pancreas	1	1
	Stones in the common bile duct	1	2
61—70	Carcinoma of pancreas	1	3
	Stones in the common bile duct	nil	2
		5	11

Sixteen patients with an age range of 7 to 70 years, and a male / female ratio of 2·2: I, who were operated in one surgical unit at Jaffna General Hospital, during 1978 are presented. Table I lists the causes of jaundice in this group, and Table II shows the distribution of the cases by age and sex in relation to the cause of jaundice. Taking the sixteen patients as a group, jaundice (100%), abdominal pain (85%) and intermittent fever (81%) were the most common symptoms. However, while 5 / 5 patients in the gall stones group had both abdominal pain and fever, 2 / 7 patients with carcinoma had neither of these symptoms, and one other did not have fever. (Table III). On clinical

TABLE III: CLINICAL MANIFESTATIONS

Clinical manifestations	PERCENTAGE OCCURRENCE IN THE series	carcinoma group	stones group	others
Number of patients*	100%(16)	100%(7)	100%(5)	100%(4)
(a) Abdominal pain	85 (14)	71 (5)	100 (5)	100 (4)
(b) Intermittent fever	81 (13)	57 (4)	100 (5)	100 (4)
(c) Lump in the right hypochondrium Enlarged liver	63 (10)	86 (6)	20 (1)	75 (3)
Palpable gall bladder	31 (5)	71 (5)	nil	nil
At surgery: Large gall bladder	56 (9)	100 (7)	nil	50 (2)
Small gall bladder (chronic cholecystits)	38 (6)	nil	100 (5)	25 (1)
Normal gall bladder (case 7)	6 (1)	nil	nil	25 (1)
(d) Pruritus	38 (6)	43 (3)	20 (1)	50 (2)
(e) Anorexia & loss of weight	38 (6)	86 (6)	nil	nil
(f) Past history of similar attack	38 (6)	nil	80 (4)	50 (2)
(g) Duration of jaundice (weeks) 1—3	25 (4)	nil	60 (3)	25 (1)
4—6	31 (5)	43 (3)	20 (1)	25 (1)
7—12	44 (7)	57 (4)	20 (1)	50 (2)

* Number of patients is given within brackets.

examination a palpable lump in the right hypochondrium was the most common finding. Of the patients with carcinoma 6/7 (86%) had an enlarged liver, and 5/7 (71%) had an enlarged gall bladder, while in the patients with gall stones, only 1/5 (20%) and 0/5 (00%) had englarged liver and gall bladder respectively. Three of the 4 patients in the 'others' group had an enlarged liver but none had clinically palpable gall bladder. The operative findings showed a minor false negative in the patients with carcinoma, in that all seven were now seen to have enlarged gall bladders, in 2 of whom the enlargement was obscured by the enlarged overhanging liver. All 5 patients with calculous obstruction had contracted gall

bladders. (Table III). Two patients, one with an unidentified obstruction at the lower end of the common bile duct and the other with a right lobe amoebic liver abscess (Table I), had prominent veins in the right side of the chest and abdomen; in both the dilated veins were no longer visible after surgery. Six patients complained of pruritus (Table III), all of whom had serum bilirubin levels over 15mg%. Four others with levels over 15 mg% denied any pruritus. Anorexia and loss of weight were symptoms only in 6 patients in the carcinoma group. A past history of attacks of abdominal pain with intermittent fever was present in 4/5 patients with calculous disease and in 2 others who had acalculous chronic cholecystitis. The latter two patients had enlarged lymph nodes adjacent to the common bile duct, (Cases 10&14). The average duration of jaundice in this series was 7·1 weeks.

TABLE IV: LABORATORY INVESTIGATIONS

Investigation	Carcinoma group 7	Gall stones group 5	Liver abscess 1	others 3
Pre operative serum bilirubin mg%	8·4—30	1·6—22	1·8	7·2—22·4
Serum alkaline phosphatase K. A units	17·5—60	24—60	54·5	33—60
Prothrombin time (secs) (control 20—22 secs)	18—35	18—25	22	20—28
Radiological evidence of gall stones	nil	5	nil	nil
pancreatic clacification	1	nil	nil	nil
Blood urea mg%	25—70	25—65	30	not done
Serum bilirubin after surgery	1·5—9·3	1—2·4	not done	0·6—8

(Table IV). The serum bilirubin was below 5 mg in 2, between 5 and 15 mg in 4, and 15 to 30 mg% in 10. The serum alkaline phosphatase was estimated in 13 patients; 5 had levels below 30 K. A units, the lowest level being 17·5, four had levels between 30 to 60 and four over 60. The prothrombin time was prolonged in 8 patients.

Abdominal radiographs were taken in all 16 patients; 5 had evidence of calculus or calculi in the common bile duct and one patient with pancreatic calcification was shown to have a carcinoma of the pancreas.

A percutaneous liver biopsy was carried out in three patients in whom the diagnosis was obscure, but the reports were inconclusive in all three.

Pre operatively all patients had:

(a) vitamin K 10 mg i.m daily for 5 days or more except case 14, who had 5 mg daily.

(b) 50—100 gm of glucose with 25—50 units of soluble insulin i. v. for 3 days.

(c) mannitol 20%, 250 ml one day prior to surgery (except case 14), and urine out-put was measured.

(d) antibiotics either ampicillin or cortrimoxazole.

(e) bleeding, clotting and prothrombin times done.

(f) no premedication.

In the theatre, all the patients had a vein cut-down and the bladder catheterised prior to surgery. They were pre-oxygenated for 3 minutes with 100% oxygen. Anesthesia was induced with a sleep dose of thiopentone sodium (150—300 mg) and trachea intubated after suxamethonium chloride (50 mg). They were ventilated with nitrous oxide and oxygen (6 : 3), and the muscle relaxant used was gallamine 80—160 mg. Pethidine 50—100 mg was given i. v, to supplement nitrous oxide. Arterial blood pressure and urine out-put were monitored at frequent intervals. Mannitol 20%, 250 ml was infused in all patients except in case 14. Five patients with carcinoma required blood transfusion during surgery, whereas the others had no blood transfusion. The action of gallamine was reversed with atropine and neostigmine.

At surgery, 6 patients had a hard growth in the pancreas with a distended common bile duct and gall bladder. In one of these, the lower common bile duct and cystic duct were infiltrated by the growth, and choledocho-duodenostomy was necessary[1]. Four others had cholecysto-jejunostomy with entero-anastomosis. The sixth patient, the only hospital death is presented in more detail :

Case 8; M 52 yrs, had a large growth in the pancreas. He bled excessively, developed hypotension, and surgery was confined to cholecystostomy and biopsy of the growth. He died on the second post operative day due to hepato-renal failure. Biopsy report was that of an adenocarcinoma.

In the patient with carcinoma of the common bile duct : **Case 9; M 49 yrs,** the gall bladder was found distended with 'white' bile and contained four calculi, which were not seen in the roentogram. Exploration of the dilated common bile duct yielded two fleshy masses, which at biopsy were found to be adenocarcinoma. The sphincter of Oddi did not offer any resistance to dilators. Cholecysto-jejunostomy with entero-anastomosis was done after the removal of caluli. He made a slow recovery and the serum bilirubin dropped from 15·4 mg% to 9·3 mg% on the ninth day after surgery. He was found to be free of icterus and pruritus after 3 months.

The five patients with gall stones underwent exploration of the common bile duct, removal of calculi, dilatation of sphincter of Oddi with Bake's dilators and cholecystectomy. T—tubes were not used in three of these patients.

The other four patients are outlined below: **Case : 6 M 30 yrs;** presented with jaundice, abdominal pain and intermittent fever. He had a large palpable liver. He was thought to have an intrahepatic biliary obstruction and was treated with a course of steroids in the medical unit. Since the response to steroids was not satisfactory and the liver biopsy report was inconclusive laparotomy was done. At surgery, a large liver and hidden underneath

a large tense gall bladder along with a dilated common bile duct was found. Two litres of bile were aspirated from the gall bladder. The pancreas was found to be normal and there were no stones in the common bile duct or gall bladder. Cholecysto-jejunostomy and entero-anastomosis were carried out, as the patient's condition precluded further exploration. He made an uneventful recovery and was discharged on the tenth day after operation. He is now in good health and is being followed in the clinic. The cause of obstruction in this patient is not clear but may be due to chronic pancreatitis or stricture of the common bile duct.

Case 7: M 31 yrs; presented with abdominal pain, intermittent fever and jaundice of 3 months duration. He had a large palpable liver and an X-ray of chest did not reveal an elevated right dome of the diaphragm. At operation he had a large abscess in the right lobe of the liver, from which 3500 ml of greenish-yellow pus were drained. The abdomen was closed with a drain in the abscess cavity. He was treated with amoebicidal drugs and discharged 15 days after surgery.

Case 10: F 22 yrs; presented with jaundice, intermittent fever and abdominal pain. She complained of pruritus and gave a past history of a similar attack four years earlier. At surgery she was found to have chronic cholecystitis with an enlarged lymph node compressing the common bile duct. Cholecystectomy with removal of the lymph node was done, and the patient made an uneventful recovery. Biopsy of gall bladder and lymph node revealed chronic cholecystitis and reactive changes respectively. Jaundice in cholecystitis without calculi has been described in the past [16].

Case 14: F 7 yrs; presented with abdominal pain, intermittent fever, enlarged liver, and gave a past history of jaundice an year earlier. She developed jaundice one week later and the liver kept on enlarging till its lower margin was felt in the right iliac fossa just prior to surgery. At surgery, a large liver and a distended gall bladder with absent cystic duct were found. A large lymph node was found adjoining the common bile duct which was narrow. The common hepatic duct was found dilated. Cannulation of the common bile duct was not possible even after the removal of the lymph node. A piece of gall bladder was taken for biopsy and cholecysto-jejunostomy with entero-anastomosis was done. The biopsy was reported as chronic cholecystitis and the gland showed reactive changes. This patient is asymptomatic after surgery.

Results:

One patient (case 8) in this series died on the second post operative day. All the others left hospital after 10 to 19 days (average 13·8) of stay after the operation.

Immediate post operative serum bilirubin levels were higher in patients who had prolonged jaundice. It was over 4 mg% in 6 patients who had jaundice for 6—12 weeks. These patients became anicteric subsequently.

Case 2; who had biliary obstruction due to stones was ventilated for 8 hours post operatively, due to delay in the recovery of muscle power.

Excessive bleeding, uraemia, hepatic coma and ascending cholangitis did not occur in the fifteen survivors. Case 8,

who had an inoperable carcinoma of pancreas died of excessive bleeding and post operative hepato-renal failure.

Three patients had mild wound sepsis. There was no incidence of wound dehiscence or incisional hernia[6]. None developed a biliary fistula or pancreatitis

Discussion :

It is now well known that liver function deteriorates with prolonged obstructive jaundice, and post operative renal failure is directly related to the depth of jaundice[1]. Further, biliary obstruction may lead to biliary cirrhosis and ascending cholangitis which increase the risk of surgery. It is now recognised that 4 weeks is the critical time at which hepatocellular function starts to deteriorate[8]. In this series, we found that patients with jaundice of greater than 6 weeks duration took a longer time to become anicteric, which is probably due to hepatic damage. One of our patients with jaundice of 3 months duration, awaiting surgery was postponed by one week due to non-availability of blood. His condition deteriorated during this week, and following surgery died on the second day due to a combination of excessive bleeding and hepato-renal failure.

It is vital that an early diagnosis of the type of obstruction is made in patients with jaundice, if surgery is to be safe. Surgery in intra-hepatic obsruction should be avoided, as it may precipitate liver failure, which carries up to 42% mortality[8]. Early diagnosis of the type and site of obstruction is difficult in our hospital which lacks facilities for special investigations such as; percutaneous trans-hepatic cholangiogram, ultrasound scanning, fibreoptic retrograde cholangiogram, splenic venogram, coeliac angiography and operative cholangiogram. However, the diagnosis of extra-hepatic obstruction can be made clinically, without the above special investigations, with an accuracy of about 80% by experienced clinicians[8]. In our series, the diagnosis of extra-hepatic obstruction was made mainly on clinical grounds together with a few routine investigations, with an accuracy of 94%. We had to resort to a laparotomy to determine the cause and site of obstruction. Of the 16 patients in our series 15 were found to have extra-hepatic obstruction at surgery, and the other patient was found to have a large abscess in the right lobe of the liver.

The features of extra-hepatic obstruction are enumerated in the introduction. At surgery 9 patients had large distended gall bladders, although only 5 of these were palpable pre operatively. The liver was found enlarged in four patients (2 cases of carcinoma of pancreas & 2 cases of lower common bile duct obstruction) in whom the gall bladder was found enlarged at operation, but not clinically palpable. The enlarged liver over-hung the large distended gall bladder obscuring it away from the palpating fingers. In one of these patients (case 6) the gall bladder contained two litres of bile, although it was not clinically palpable. Thus a large liver in a patient with jaundice which does not fit into the 'medical' type, should arouse the suspicion that there may be an "occult distended gall bladder" which would signify an extra-hepatic obstruction.

In our series the duration of jaundice was longer in those belonging to the carcinoma group, probably due to late

diagnosis. This feature has been described by Balasegaram [9] in whose series, the curative resection was indicated in only 2% of patients 86% of our patients with carcinoma had anorexia and loss of weight, symptoms which should arouse the suspician of carcinoma. Hypertonic duodenography has been advocated by Balasegaram for early diagnosis of carcinoma.

In our series, we had five patients with alkaline phosphatase levels between 17·5—30 K. A. units, although it is generally accepted that the alkaline phosphatase level is above 30 K. A units, in obstructive jaundice. This agrees with the findings in other series[8]. We find that the alkaline phosphatase levels do not bear any relationship to the levels of serum bilirubin.

Only 10--33% of gall stones are radio-opaque. However, in our series all patients with obstruction in the common bile duct due to stones, had radio-opaque calculi. This poses the question whether many cases of obstructive jaundice due to radio-translucent stones are being missed. This needs further study.

It is our experience that surgery in jaundiced patients is safe, if sufficient precautions are taken to prevent excessive bleeding, post operative renal failure and hepatic failure. It has been suggested that the risks are minimal if operation is undertaken within six weeks of jaundice [8] We agree with the above, as we did not have any morbidity or mortality in patients with jaundice of less than 6 weeks duration. Even in patients with prolonged extra-hepatic jaundice, surgery with the above precautions (enumerated in materials and methods) is indicated as they would deteriorate without surgery. Five of our patients had jaundice of 3 months duration and we were able to prevent these complications in four. One of these patients who recovered, had 'white bile' at surgery, indicating no biliary secretion.

Drug administration has to be judicious in these cases because of their impaired liver function. Arterial hypotension and hypoxaemia have to be avoided during anaesthesia to prevent deterioration of liver function. Volatile anaesthetic agents are avoided because of the possibility of these or their metabolites causing liver damage. Gallamine is preferred to tubocurarine, because of its shorter duration of action and its excretion to a larger extent by the kidneys. Adequacy of liver perfusion during anaesthesia can be achieved by maintaining; (a) arterial pressure near normal, (b) urine out put above 30 ml. per hour, (c) the skin warm and (d) rapid filling of skin capillaries when emptied by digital pressure. The cause for the post operative ventilatory failure in one of these patients (case 2) is obscure. This was probably a partial neuro-muscular block due to gallmine. In this patient serum sodium and potasium were within normal limits.

The operative procedure was confined to a palliative internal by-pass operation, in cases of carcinoma, as the growth was either not resectable (4 cases) or the patients unfit for radical surgery (3 cases). Internal by-pass surgery was also done in two others with benign lower common bile duct obstruction. All our 9 patients in whom internal by-pass surgery was done, recovered from surgery. This contrasts with the series by Balasegaram[9] where 10 of the 16 patients died within two weeks of internal by-pass surgery. This may be due to the type of internal by-pass. We performed cholecysto-jejunostomy with entero-anastomosis in all

patients except one, in whom we had to do a choledocho-duodenostomy. Balasegaram did not attempt cholecysto-jejunostomy, but was inclined to do choledocho-jejunostomy (II patients), cholecysto-gastrostomy (2 patients) and choledocho-duodenostomy (3 patients). It is our belief that cholecysto-jejunostomy with entero-anastomosis, and choledocho-duodenostomy as described by Johnson and Rains[11] are safe and reliable, as we had no post operative mortality or morbidity following these procedures.

Balasegaram advocated permanent T-tube drainage as the mortality was high in his series with internal by-pass surgery. We believe that T-tube drainage carries an increased mortality and morbidity. This is in agreement with the findings of other authors, that T-tube drainage is associated with an increased incidence of ascending cholangitis, stricture formation and biliary fistulae.[11, 13, 14] Balasegaram had 10 deaths out of 49 patients who underwent permanent T-tube drainage within 3 weeks of surgery.

In three of the five patients with stones in the common bile duct (cases 12, 15 and 16), no T-tubes were used, but a drain was placed in the gall bladder bed after the closure of the common bile duct. This has been described earlier by Collins and Redwood.[15] The sphincter of Oddi was dilated in all these patients. They were ambulant from the second day, as there were no tubes to disturb them. These patients had prophylactic antibiotics as they fall into the high risk group for infection as classified by Keighly et al.[10] The possibility of calculi left behind exists (4 to 10% in most series), but the chances of these calculi being washed into the duodenum may be greater, if the bile has only one passage to pass through. If jaundice recurs, it could be safely treated surgically by trans-duodenal sphinterotomy as described by Aubrey and Edward in 1978.[6] All our patients recovered without any complication.

Although Irvin et al[6] described an incidence of wound dehiscence and incisional hernia of 27·1% in the 48 patients, we had no wound dehiscence or incisional hernia. We are unable to pinpoint why we did not get these complications, as the incision and closure were similar to that used in their series.

Summary:

Fifteen of the sixteen patients with obstructive jaundice who were subjected to surgery had extra-hepatic obstructive lesions, the only exception being a case of amoebic liver abscess. The diagnosis of extra-hepatic obstruction was established almost entirely on clinical and haematological data. Radiology was confined to plain X-rays of the abdomen, facilities for retrograde contrast radiography and angiography being unavailable. Three patients had preliminary percutaneous liver biopsies the results of which were inconclusive. It is our view that surgery should be undertaken once extra-hepatic obstruction is diagnosed, in hospitals where facilities for more sophistigated investigations are unavailable.

Acknowledgements:

We are grateful to Dr. Jega Pasupati, Dr. M. Kanagarajah, Dr. S. Sivakumaran and Dr. P. Arulanantham who referred these patients, and to Mr. Rudra Rasaretnam for his invaluable advice.

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THE MANAGEMENT OF ABDOMINAL TRAUMA

A. VIJAYARAGAVAN, M. B., F. R. C. S. (Eng.)
Surgeon, Accident Service, Colombo General Hospital.

Summary

ABDOMINAL injury is classified into gunshot, penetrating and closed. The gunshot injuries are considered separately from the other penetrating wounds as they are all explored routinely as visceral damage is invariable. Though both penetrating and closed injuries are managed on the principle of selective conservatism marked differences exist between them. Blunt injury caused by road traffic accidents and falls from a height may be associated with injuries to head, chest, spine and the limbs and for this reason enjoys a higher mortality. The abdominal injury itself is usually confined to a single viscus, unlike in the case of stab wounds where even when the wound is single the tendency is to produce multiple injury. The criteria for exploration are laid down, techniques of repair described and the results discussed.

Introduction

Abdominal injuries account for less than 5% of admissions to the Accident Service. However, they are consistently the most difficult group to treat as, often, it is not easy to decide whether to explore the abdomen; and on exploration, of the confrontation by the unexpected.

Classification

Though abdominal injuries are usually classified as open (penetrating) and closed (blunt) it is more useful to recognise gunshot injuries as a special type of the open variety. A preliminary requisite in the management of abdominal trauma is to establish the need for exploration of the abdomen: that is, the presence of visceral damage must be established. The idea of selective surgery for abdominal injuries has now been pretty well established. This consists essentially of thorough initial examination and continued close observation of all patients with abdominal injuries and submitting only those with definite evidence of visceral damage to surgery. This applies to both open and closed injuries. However, in the case of gunshot injuries visceral damage is invariable. In a series of 15 consecutive cases of gunshot injuries studied in an eight-month period in 1971, it was noted that all had visceral injuries.[1]

In contrast to open injury which focuses attention to the abdomen from the very start, the unmarked abdomen in blunt trauma especially when associated with injuries to other systems such as head, chest and pelvis, tends to be missed resulting in delay in the diagnosis of the abdominal lesion. Even when the abdomen alone is involved, local injury to the abdominal wall tends to confuse the correct interpretation of the clinical signs of tenderness, guarding and rigidity.

Mechanism

In open injuries the viscera are directly damaged by the offending weapon or missile. In closed injuries the solid organs usually suffer from the direct trauma that is applied to the abdominal wall being transmitted to the viscus. Splenic ruptures

may sometimes be due to contre-coup injuries. Direct crushing of the bowel against the bony vertebral column causes injuries varying from contusion to laceration to full thickness tears. Two other mechanisms have been described in the case of bowel. Sudden compression of the bowel in one place may lead to rapid movement of intestinal contents away from the site of compression leading to a rise in intraluminal pressure and rupture. This is especially so if movement of intestinal contents is restricted by adhesions, kinks etc. Intestinal rupture may result from falls from a height. When this happens the rupture occurs at the junction of the mobile and fixed parts, for instance the duodeno-jejunal area or the ileo-caecal area. There is no direct impact on the abdominal wall and the tear is due to sudden deceleration, the mobile part of the bowel continuing to move tears away from fixed portion which has ceased to move.

Management

A thorough examination of the whole patient is first carried out especially in those who are unconscious or have multiple system injuries. A clear airway is maintained, if necessary by oro-tracheal intubation or tracheostomy. Chest injuries take priority in the treatment schedule. Blood and air in the pleural space must be evacuated by intercostal tube and any significant flail must be stabilised. Resuscitation of the collapsed patient is commenced pari tpasu with the examination. One or more venous cutdowns are performed in the upper limbs in all cases of suspected intraperitoneal haemorrhage. It is important to observe this small point as haemorrhage from the IVC or the iliac veins will preclude flluid replacement via the lower limb veins. Infusion is commenced with normal saline while blood is sent for emergency direct testing. The need for transfusion with undirect-tested group O Rh negative blood seldom arises.

The policy followed at the Accident Service is one of selective surgery for abdominal injuries. The exploration of the abdomen in all cases of abdominal injury would lead to a large number of unnecessary laparotomies. In a consecutive series of 226 patients with penetrating injuries of the abdomen studied and published elsewhere[6], 70 patients were not explored and recovered uneventfully. Of those submitted to laparotomy 10% did not have visceral injury. Had all 226 patients been operated on the negative laparotomy rate would have been in the region of 40%.

Diagnosis of Visceral Injury

Open wounds of the abdomen may not penetrate the peritoneal cavity. Procedures adopted to establish penetration, such as probing of the wound and X-ray after the injection of radio-opaque dye[2,3] are no longer carried out as it was shown that not only was the demonstration of penetration by the above methods inconclusive due to muscle spasm, obliquity of the tract etc., but more important, the presence of penetration was not synonymous with the presence of visceral damage.

The detection of visceral injury depends largely on clinical acumen; laboratory and radiological investigations being helpful in only a small proportion of cases. The one investigation that proved helpful in doubtful cases was peritoneal lavage.[4] The four quadrant abdominal paracentesis[5] proved unhelpful producing not only false negatives but also false positives, due to aspiration of blood from a retroperitoneal haematoma due to a fractured pelvis. Paracentesis was

employed in 17 patients yielding a true positive in 9, false positive in 1, false negative in 3, and a true negative in 4.

It has been our experience that radiology was not helpful except in a few specific instances. Erect films of the chest are done routinely for all closed injuries of the abdomen for evidence of fractured ribs, haemo or pneumothorax, ruptured diaphragm and pneumoperitoneum. It has been our experience that free gas has only occasionally been demonstrated in cases of ruptured bowel, and the absence of free gas therefore does not preclude it.

Intravenous pyelography is carried out in all patients suspected of renal injury and those presenting with haematuria after trauma. This provides information helpful in localising the side of the injury, the nature of the injury as well as the presence of a normal kidney on the opposite side.

A small barium meal has occasionally clarified a doubtful traumatic rupture of the diaphragm.

Another simple radiological procedure that has proved useful on rare occasions is the venogram to delineate the iliac veins in patients with fractured pelvis due to runover accidents who present with shock resistant to resuscitation. This is performed by injecting 20 ml of Urografin into the long saphenous vein at the ankle and taking two films—the first when 15 ml has been injected and the other as soon as the plate has been changed.

By and large the decision whether to operate is made on the clinical findings. The abdominal cavity contains hollow as well as solid viscera (including blood vessels). Injury to the former causes peritonitis due to soiling of the peritoneum with its contents. Injury to the latter causes haemorrhage. Thus the clinical signs looked for are those of peritonitis and haemorrhage.

The pulse, blood pressure, pallor and sweating are observed. The local signs that were assessed are given in Table 1. It is seen that abdominal guarding was the single most useful sign. Abdominal distension was rarely present without visceral damage, but usually was a late sign. The same applies to absent bowel sounds. Long before these signs manifested other signs such as guarding were significant enough to make the diagnosis of peritonitis. The final assessment is however made on the entire clinical picture.

Technique of Exploration

Vertical incisions cannot be surpassed for rapid and wide exposure. Our tendency is to make more midline than paramedian incisions. Exploration through transverse extension of an open abdominal wound is not recommended as viscera at some distance from the injury may be damaged and extension of transverse incisions is difficult. If blood wells out on opening the peritoneum no time must be wasted in attempting to suck out the blood as continuing haemorrhage makes this difficult to achieve quickly. The three common sources of massive intraperitoneal haemorrhage are the spleen, liver and the mesentery. Lacerations of the liver and spleen are easily palpated and this should be carried out through the sea of blood. If these are negative the mesentery is inspected next. The tears that cause haemorrhage are situated parallel to the bowel and tend to devascularise the segment of bowel situated opposite the tear.

Other sources of bleeding are the kidneys and the great vessels.

The presence of purulent fluid denotes ruptured bowel. If bile staining is noted the duodenum must be closely inspected for injury. Bile staining commonly occurs with perforations of the upperjejunum and will occur in the rare injuries to the gall bladder and bile ducts.

Large quantities of clear fluid in the peritoneal cavity usually signify an intraperitoneal rupture of the bladder.

Technique of Repair

Splenic injuries require splenectomy. The following technique permits rapid removal of the spleen even in conditions of poor visibility occasioned by the presence of blood. While the left edge of the wound is retracted strongly upwards the palm of the left hand is placed over the diaphragmatic surface of the spleen and retracted medially. The lieno-renal ligament which is thereby put on the stretch is divided and the spleen delivered gently into the wound. The short gastric vessels are then tied off and the splenic pedicle ligated after pushing the tail of the pancreas away from the hilum.

Liver injuries vary in their severity from small incised wounds which have ceased bleeding at the time of the operation and which require no treatment to large stellate fractures of the right lobe with avascular blocks of tissue which require lobectomy.

If haemorrhage is controlled by compression of the hepatic artery and portal vein in the lesser omentum, ligation of the lobar branches of these vessels may be performed. The resultant atrophy of the corresponding lobe is in time compensated for by hypertrophy of the opposite lobe. If haemorrhage is not arrested by compressing the free border of the lesser omentum it means that bleeding is from the main hepatic veins and these cases require a major hepatic resection. Most liver lacerations however are not very deep and bleeding can be controlled by deep suures placed parallel to the laceration.

Renal injuries when unaccompanied by other visceral injury present with haematuria with no abdominal signs. In these patients treatment is conservative if no significant extravasation is seen in the IVP.

When seen at laparotomy the perirenal haematoma must be explored and as much renal tissue as possible must be conserved by resorting to suture or partial nephrectomy.

Hollow visceral lesions occured frequently. Perforations of the small intestine are closed in two layers in the transverse axis after trimming any devitalised tissue from the edges. In most cases large bowel too was similarly treated. Extensive contusions as occur in gunshot wounds contraindicate simple closure. These require exteriorisation or primary resection.

The entire length of the small bowel must be closely inspected, particular attention being given to perforations at the mesenteric border as perforations into the leaves of the mesentery are easily missed. Surgical emphysema of the mesentery was noted in one patient near the bowel edge and this led to the identification of a tear at this site. In other cases a haematoma at this site may be the only pointer.

Injuries to duodenum and pancreas are easily missed even at laparotomy especially if confined to the retroperitoneum. The presence of bile staining or haematoma around the duodenum is an indication for exploration of the duodenum and the

pancreas as often both suffer injury together. The standard Kocher manoeuvre is carried out together with exploration of the lesser sac through the gastro-colic omentum. Minor injuries of the pancreas which do nor disrupt the duct require haemostasis and suture. Major injuries of the tail and body require distal pancreatectomy. Major injuries of the head which are often associated with disruption of the duodenum require pancreato-duodenectomy, but fortunately these injuries have been extremely rare in our experience.

Isolated duodenal perforations are closed transversely. A gastro-enterostomy is added only if significant narrowing is thereby caused.

Incidence of Visceral Injury

This is shown in Table 2. The small bowel is seen to be the most frequently involved viscus in all injuries. The injuries in stab and gunshot wounds are often multiple unlike in closed wounds where the rupture is often single. Splenic injuries were present in only 10 patients with closed abdominal injury. This is in contrast to most published series. Hepatic injuries necessitating resection were associated with a high mortality. Injuries of the genito-urinary system were rare except for ruptured urethra.

Mortality

Blunt abdominal injury has the highest mortality. (Table 3) The severity of the abdominal injury may be obscured by the presence of concomitant injuries. Two patients, one with intestinal rupture and another with splenic injury were missed due to associated unconsciousness. Gunshot injuries had the best result but their numbers were small. In a previous series[1] the mortality was 13%.

Discussion

According to the principle of selective surgery for abdominal trauma, exploration is carried out only if the presence of intra-peritoneal mischief can be established. Careful assessment of the patient at repeated intervals is often required. This principle of 'wait and see' can only be accepted if it does not cause any mortality or increased morbidity in the patients treated conservatively. That this condition is satisfied has been shown[6 7]. From the point of selection for surgery the gunshot injuries are the easiest to treat as they are all routinely explored. But gunshot wounds produce some of the most bizarre intra-peritoneal injuries that taxes surgical skill to the utmost. Blunt trauma poses the greatest difficulty and is associated with the highest mortality. This is because these patients may have associated injuries to other systems. Table 4 shows that the mortality increases as associated injuries increase. Abdominal injury alone is associated with a mortality of 14% but the mortality rises to 80 to 100% when injuries to head, chest and long bones occur. The overall mortality is 27% compared to 7·9% for stab wounds.

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TABLE I

Relationship of Clinical Sign to Presence of Visceral Injury

Clinical Signs	Stab Injury (226)		Gunshot (13)		Closed (111)	
	With visceral injury	Without visceral injury			With visceral injury	Without v/i
	146	80			71	40
Abdominal Guarding	106	27	11		54	18
Abdominal Distension	16	0	4		23	5
Flank Dulness	6	0	2		5	1
Bowel Sounds Present	73	64	13		18	23
Bowel Sounds Absent	11	0	4		10	3

TABLE II

Incidence of Visceral Injury

Viscus	Stab	Gunshot	Closed
Small Bowel	51	8	30
Liver	41	6	19
Stomach	35	2	5
Large Bowel	22	3	9
Vascular	21	3	5
Retroperitoneal haematoma	17	1	18
Spleen	14	2	10
Pancreas	10	1	1
Kidney	9	2	6
Duodenum	6	1	1
Urinary Bladder, Ureter, Urethra	4	0	13

TABLE III

Abdominal Injuries - Mortality

Type	Total	Died	Mortality
Stab	226	18	7·9%
Closed	111	30	27%
Gunshot	13	0	0%

TABLE IV

Associated Injuries with Closed Abdominal Trauma

	Number	Died	Mortality
Abdominal alone	63	9	14%
Abdominal and Chest	9	6	67%
Abdominal, Chest and Head	1	1	100%
Abdominal, Chest and Orthopaedic	11	6	55%
Addominal, Head, Chest and Orthopaedic	5	4	80%
Abdominal and Head	5	2	40%
Abdominal, Head and Orthopaedic	3	0	0%
Abdominal and Orthopaedic	14	2	14%
Total	111	30	27%

DIARRHOEA EPIDEMICS IN JAFFNA

C. WIJAYARAJAH, M. F. C. M., D. P. H., M. B. B. S.
Regional Epidemiologist, Jaffna.

K. VELAUTHAPILLAI, D. B., D. P. H., M. B. B. S.
Public Health Bacteriologist, Fever Hospital, Angoda.

A. NATKUNAM, D. C. P., M. B. B. S.
Pathologist, G. G. H., Jaffna.

Acute diarrhoel diseases constituté one of the greatest social evils — not only do they kill young children living in developing countries but they also retard the growth and impair the quiality of life of those who survive. The problem is overwhelming and complex as it involves deep-rooted behavioural factors in addition to the more obvious social and economic inequalities. Despite wide-spread awareness of this problem, there has been a lack of definite commitment to deal with it.

JAFFNA in recent times has become the victim of exotic diarrhoeal infections. Cholera El Tor was, reported in Sri Lanka for the first time during a diarrhoea epidemic in Jaffna in October 1973. Thereafter the disease rapidly spread throughout the rest of the Island. The Cholera epidemic was followed by an outbreak of dysentery in Jaffna in 1976. Shigella dysenterae 1 (Shigella Shiga) was isolated for the first time in Sri Lanka during the outbreak in April 1976. The geographical distribution of these cases was almost the same as that in the Cholera epidemic and the majority belonged to the low socio-economic group. Subsequently dysentery outbreaks were reported in other parts of the Island with the isolation of Shigella dysenterae 1.

DYSENTERY OUTBREAK IN JAFFNA

Materials and method

Stools and rectal swabs from every dysentery patient admitted to the General Hospital Jaffna was plated primarily on desoxycholate citrate agar (Difco), Salmonella Shigella agar (Difco) and Mac Conkey agar (Difco). Samples were also cultured on Selenite broth (Difco) for enrichment. From Selenite broth plating was done secondarily on Salmonella-Shigella agar (S. S. agar) and Bismuth sulphite agar (Difco). The plates were incubated overnight at 37°C. Lactose non-fermenting colonies were picked into Kligler medium (Difco) and on to urea medium (Christenson). The pickings that were urease negative and hydrogen sulphide negative were

tested with polyvalent antisera for Samonella and Shigella. The strains that produced hydrogen sulphide and non-urease producers were tested for Salmonella only with polyvalent antisera. Those strains that gave positive reactions with polyvalent-antisera were identified serologically with typing antisera. Indophenol oxidase test (Ewing and Johnson, 1960) and test for motility using Cragie tubes were additional tests employed.

The following tables give the results of bacterial isolation from diarrhoeal stools done at the Pathology Laboratory G. G. H. Jaffna during 1976.

TABLE I

Results of specimens processed for Cholera

Month	No. of Samples	+ ve for Ogawa	+ ve for Inaba	Total + ve	+ ve for N. A. G.
Jan.	94	0	1	1	9
Feb.	146	1	1	2	18
March	235	1	0	1	31
April	289	6	6	12	53
May	385	5	5	10	31
June	374	1	5	6	16
July	272	1	0	1	3

TABLE II

Results of specimens processed for Entero-bacteriaceae

Month	No. of Samples	+ ve for entero-pathogenic E coli	+ ve for Salmonella	+ ve for Shigella	+ ve for Plesio-monas	Yersinia-entero colitica	TOTAL
Jan.	5	2	0	0	0	0	2
Feb.	27	1	0	0	0	0	1
March	135	17	5	2	0	0	24
April	174	14	2	11	0	0	27
May	385	9	8	48	7	1	73
June	374	15	16	32	9	1	73
July	272	19	9	45	1	0	74

TABLE III

Serotype of Shigella Isolated

Month	Sh. dysenteriae 1	Sh. flexneri	Sh. boydii	Sh. Sonnei
Jan.	0	0	0	0
Feb.	0	0	0	0
March	0	0	2	0
April	10	0	1	0
May	7	18	4	18
June	4	10	15	3
July	7	8	19	11

TABLE IV

Percentage of Isolation

Jan.	Sample insignificant
Feb.	3·6%
March	17·9%
April	14·9%
May	18·9%
June	19·5%
July	27·2%

TABLE V

Antibiotic Sensitivity Pattern of Shigellae

	Tetra-cycline	Neomy-cin	Erythro-mycin	Ampicil-lin	Chloram-phenicol	Bactrim	Furozoli-dine
Sh. dysenteriae	R	R	S	S	R	S	S
Sh. flexneri	R	S	S	R	S	S	S
Sh. boydii	R	S	S	R	S	S	S
Sh. sonnei	R	S	S	S	S	S	S

TABLE VI

Summary of Bacterial isolations from diarrhoeal Stools done at the Pathology Laboratory G. G. H. Jaffna from January to December 1976.

I.	No. of Stool specimens examined for Cholera and Non-agglutinable vibrios	3422
	No. positive for Cholera	43 (1·25%)
	No. positive for N. A. G.	175 (5·11%)
	Of the 43 positive for Cholera vibrio :—	
	No. positive for Ogawa	24 (55·8%)
	No. positive for Inaba	19 (44·2%)
II.	No. of school specimens examined for Entero-bacteriacea	2999
	No. positive for Enteropathogenic E. coli	144 (4· 8%)
	No. positive for Salmonellae	62 (2· 0%)
	No. positive for Shigellae	239 (7· 9%)
	No. positive for Plesiomonas	26 (0·86%)
	No. positive for Yersinia entero / colitica	3 (0·10%)
	No. positive for Edward Cellatara	1 (0·03%)

Of the 239 specimens positive for Shigellae, serotypes were as follows :—

No. positive for Sh. dysenteriae	56 (23·43%)
No. positive for Sh. flexneri	54 (22·17%)
No. positive for Sh. boydii	74 (30· 9%)
No. positive for Sh. sonnei	55 (23· 0%)

Interpretation of the results

There has been some controversy as to the aetiological agent causing the dysentery outbreak because a large variety of organisms have been isolated.

In diarrhoeal diseases of infective origin, organisms that circulate in the community cause disease in susceptible individuals if the faeco-oral contamination chain goes on unhampered. Epidemics of dysentery occur due to endemic organisms when there is a breakdown in health standards due to variation in seasonal factors. The introduction of a new organism causes a severe outbreak as there is no immunity in the community for such an organism. New pathogens introduce new clinical features as was seen in this outbreak. As the new organism is superimposed on the endemic flora it is not unusual to isolate more than one type of organism. In children endemic strains are seen to cause a severe disease as the epidemic strains, for the

children do not possess any immunity. In hospital admissions more epidemic strains are isolated initially. With passage of time when newly introduced organism becomes endemic the frequency of isolation of all serotypes will be the same unless by floral competition one type replaces the other (Tables II & III).

In epidemics due to a new organism the first observation is a new clinical syndrome. This gets correlated subsequently by the identification of a new organism Hence for practical purposes the new syndrome should be thought of as due to a new organism. Shigella dysenterae I is a new strain to Sri Lanka This is the only Shigella species which produces an exotoxin in addition to endotoxin produced by all Shigellae. This is probably responsible for the severity of the symptoms as well as other sequelae in these cases. The isolation of the organism, the epidemiological evidence and the clinical syndrome point to the fact that the dysentery outbreak was due to Shigella dysenterae I.

The Shiga Problem

The isolation of Shigella dysenterae I during the dysentery outbreak highlights the dangers of infection with this organism. There had been 1057 diarrhoeal admissions to the paediatric unit G. G. H. Jaffna during the period December 1975 to May 1976 with 33 deaths during the height of the outbreak. Besides children, adults were also affected.

It was observed that typically the disease had two phases. In the first phase there was watery diarrhoea, abdominal cramps and fever whereas the second phase was characterised by stools which contained little or no faecal matter and consisted mainly of blood and mucus; during the second phase fever was often absent. Because amoebiasis was endemic the disease was initially treated with amoebicides with poor response.

As in Cholera, effective treatment depends on rehydration, with careful monitoring of electrolytes. Shigella species penetrate the intestinal epithelial cells but rarely invade beyond the lamina propria. However it has been suggested that Shigella Shiga has considerable invasive potential and in one third of the cases in Los Angeles positive blood cultures were obtained (Ref. 3). This bacteriaemia may be important in the pathogenesis of haematological complications and warrants antimicrobial therapy. In contrast bacillary dysentery due to Shigella sonnei, Shigella boydii and Shigella flexneri is usually mild and the use of antimicrobial drugs is not recommended.

In recent decades the distribution of Shigella dysenterae I has been mainly in the middle East, parts of Asia and areas of Central America. In 1969 and 1970 a large epidemic occurred in Gautemala, El Salvador and Mexico accompanied by a high mortality. In Gautemala alone there had been 112,000 cases with 13,500 deaths (W. H. O.) which were caused by multiple drug resistant Sh. dysenterae 1. In Bangladesh recent outbreaks due to Shigella Shiga have produced a severe fulminating dysentery especially in infants and children.

In our country only a proportion of cases of dysentery are investigated bacteriologically. It would be advisable however to investigate at least the following types of cases —

(a) The clinically severe patients.
(b) Diarrhoeas with new clinical features.
(c) Cases that present during an epidemic.

Recent advances in knowledge and techniques.

Intensive research activities stimulated by the current cholera pandemic have made available new knowledge about diarrhoeal diseases. This includes :

—The discovery that a single method—using oral rehydration fluid—can treat most cases of dehydration from watery diarrhoea including cases of cholera, in all age groups

—The recognition of the role of new viruses and bacteria which makes it possible to identify aetiological agents in more than 80% of diarrhoeas; this is a reversal of the situation present a few years ago when 80% of the cases remained undiagnosed and were called "acute undifferentiated diarrhoeas"

—The under-standing of the pathogenesis of most of the diarrhoeas, opening up possibilities for improving treatment and prevention, including development of vaccines

—The demonstration that the provision of safe drinking water supplies alone is not sufficient to prevent acute diarrhoeal diseases

—The finding that travellers' diarrhoea, though caused by a variety of diarrhoel pathogens, is commonly due to enterotoxigenic E coli, in which case judicious use of antibiotics may have a preventable role

—The demonstration that aluminium adjuvanated whole-cell cholera vaccines may provide better protection in children and longer protection in adults than the generally available vaccine, justifying their further study.

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LESIONS OF THE ANTERIOR MEDIASTINUM

J. CANAGARAJAH, M. B., B. S. JEGA PASUPATI, M. R. C. P.
Y. PARAMSOTHY, M. B., B. S. RUDRA RASARETNAM, M. B., F. R. C. S.

From the Departments of General Medicine and Thoracic Surgery, Jaffna General Hospital.

Summary:

IN the brief period of 16 months 10 patients with anterior mediastinal lesions were seen at this hospital. Three presented with myaesthenia gravis, two of whom had hyperplasia. All three were benign, 2 were improved after surgery and there was one operative death. In the non-myaesthenic group were 7 patients with tumours, all of whom had pressure symptoms and 2 had superior vena caval obstruction. Five of them underwent surgery, all tumours being large and weighed between 570 and 1250 grams. Four were malignant. Four were excised completely and one partially. There were 3 hospital deaths, within 2 days, and one other patient succumbed 12 months later. The only long term survivor is the patient with a benign terato-dermoid. Two other patients with large anterior mediastinal masses were orthopnoeic but refused surgery and have been lost to follow up.

Introduction:

Hospital medical practice in Ceylon is faced with the problem of a large number of patients who attend the outpatients department and restricted facilities in the way of personnel, equipment and time that is available to deal with them. This must inevitably lead to diagnosis being made at time when the disease is so far advanced that it is almost obvious and the necessity for admission incontravertible. Early diagnosis is sometimes possible, particularly in those lesions which are common in the particular locality, familiarity and experience then being of most help to the clinician rather than a process of deductive reasoning being based on education and awareness of the literature. By all accounts, mediastinal cysts and neoplasms are relatively uncommon. Daniel, Diveley, Edwards et al (1960), and Sabiston and Scott (1952) had a hospital admissions incidence of 1 : 3000 and 1 : 3400 respectively for all mediastinal tumours while a previous report from this country gave the incidence as 1 : 19500 (Paul, Rasaretnam and Yoganathan 1970). In this report is presented an account of 10 cases of anterior mediastinal lesions, 7 of which were neoplasms and the other 3 being cases of myaesthenia gravis without thymoma, for the purpose of drawing attention to these lesions. Our incidence of hospital admissions for just anterior mediastinal tumours is 1 : 6361, a figure which is considerably greater than the total incidence of all mediastinal tumours quoted from Colombo. All 10 of our patients were symptomatic, and if the three cases of myaesthenia gravis are excluded, the other 7 cases had very advanced tumours presenting in almost a terminal phase.

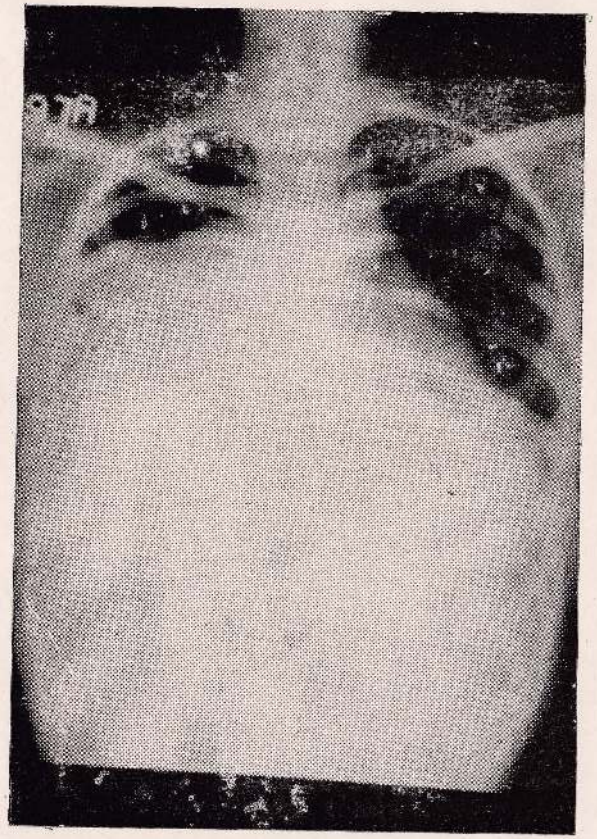


FIG. I

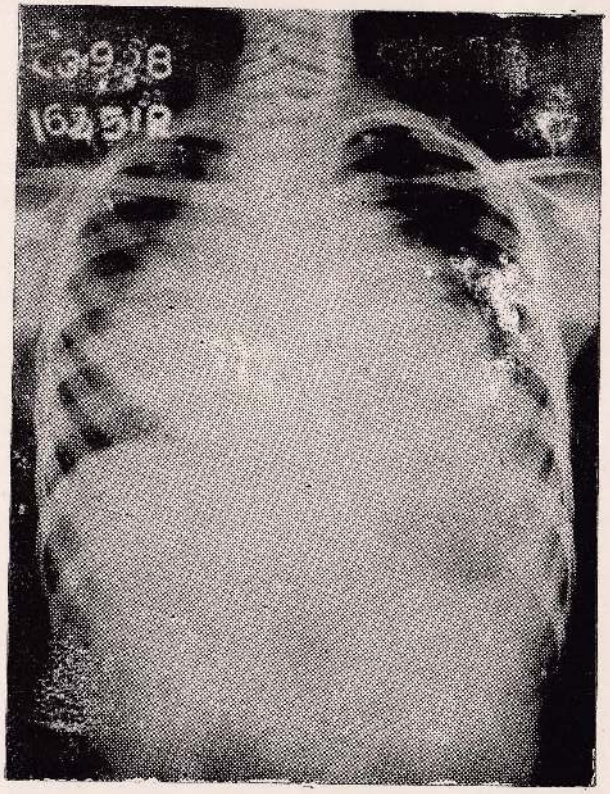
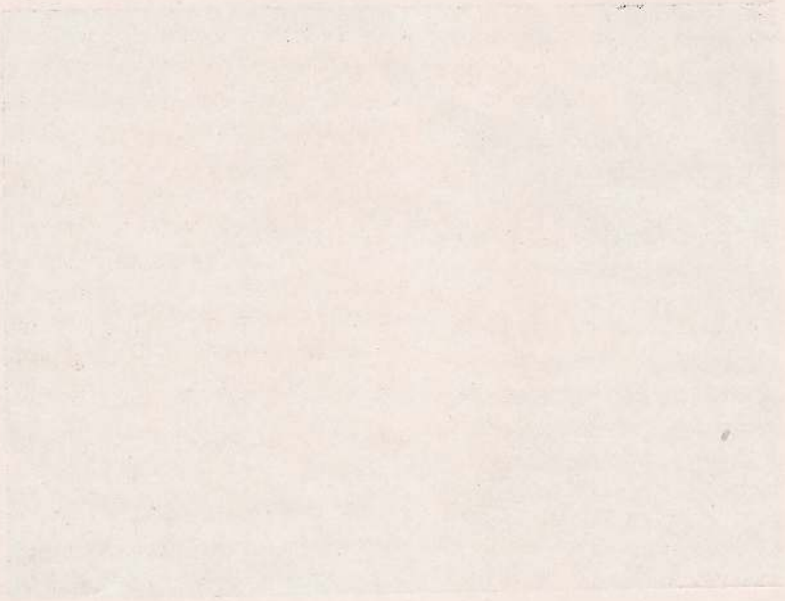
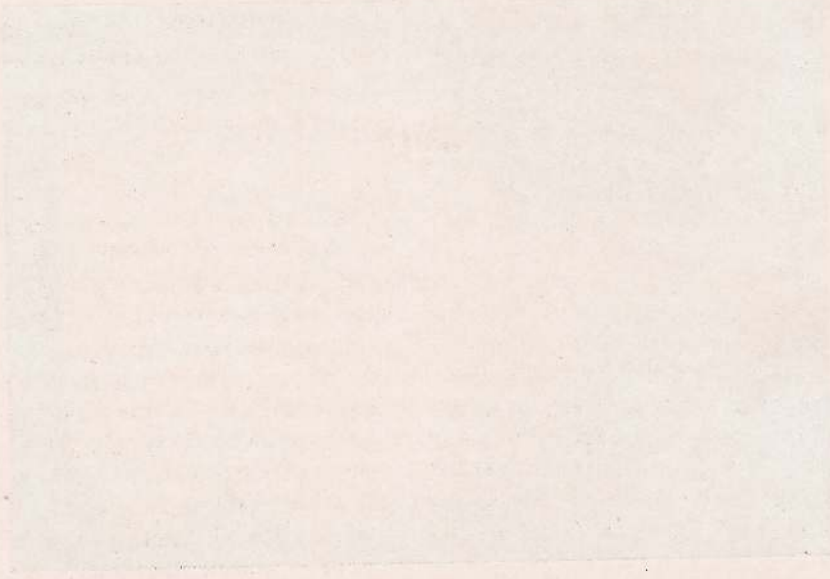


FIG. II





Case Material and Results

Ten patients with anterior mediastinal lesions were admitted to the Jaffna General Hospital during the period 1st September 1977 to 31st December 1978. (Table 1). There were 7 cases of tumour without hormonal effects and 3 cases of myaesthenia, 2 of whom had hyperplasia while the third had an atrophic thymus. These two groups of patients are considered separately in view of their different presentations and prognosis.

ANTERIOR MEDIASTINAL TUMOURS: There were 6 females and one male, with an age range of 11 to 70 years. Three each were below 20, and over 50 years, respectively. All 7 patients presented with fever, cough chest pain and generalised weakness. Two patients had superior vena caval obstruction, and 2 others who had malignant thymomas presented with stridor. Four patients underwent surgery here and one at another hospital, while 2 patients aged 55 and 70 years refused consent for operation and have now been lost to follow up.

TERATODERMOIDS: There were two patients who had teratodermoids, one of which was malignant. The 18 year old girl with a benign teratodermoid (Case 1) had a large neoplasm occupying the whole of the right pleural cavity (Figure 1) with compression of the right lung. A tumour weighing 1135 grams was excised, and at follow up 8 months later she was in excellent health. Histological examination showed a benign terato-dermoid, with myxomatous tissue, muscle, glands with columnar epithelium, embryonic vascular tissue, sebaceous glands, epidermal cysts, cartilage, bone, adipose tissue anp cystic spaces lined by papillary columnar epithelium.

Case 2. 50 year old female had been treated some years previously by hysterectomy for carcinoma of the cervix and had also had a lump in the breast excised subsequently but no details of this were available. She presented with one months history of loss of weight and anorexia together with cough, fever and chest pain. She was a diabetic and was under treatment for hypertension and hypothyroidism. Prior to surgery a left pleural effusion was aspirated, and a friable malignant tumour was removed piecemeal from the anterior mediastinum. She died in the immediate post-operative period. Histology showed evidence of a malignant teratodermoid.

THYMIC NEOPLASMS: There were 3 cases of malignant thymoma (Cases 3, 4 & 5), all of whom presented with histories of cough, fever, dyspnoea and chest pain of 2 to 4 months duration, and two of them had developed stridor by the time of admission. All 3 had in the course of their illness sought previous medical attention, both of the western and ayurvedic varieties. In the earlier phase of their illnesses symptoms were non-specific and presumably there was a paucity of physical signs. Chest Xrays had not been taken in any of these cases prior to the present admission. One of the patients, a 11 year old girl had complained of increasing dyspnoea which had apparently been diagnosed as asthma for which she was treated successively by both allopathic and ayurvedic systems, and was then brought to this hospital on account of progressive deterioration and the development of asthma. Chest Xray (Figure 2) revealed a large anterior mediastinal tumour which proved to be a malignant thymoma. In two of the 3 patients the tumour was resectable while one was so extensive

due to infiltration of the sternum, pericardium and ribs that only biopsy was possible. Histological examination showed tumours of lympho-epithelial lymphoid and spindle cell varieties. The patients with the latter two histological varieties succumbed within 48 hours of the operation from respiratory insufficiency; while the girl with the lympho-epithelial tumour developeed pulmonary and thoracic cage metastases within three months which were unresponsive to both radiotherapy and chemotherapy and she died 12 months after operation.

MYAESTHENIA GRAVIS : Three male patients aged 26, 28 and 57 years had been under treatment in the Medical Clinic for periods varying between 6 weeks in the eldest to 9 years in the 26 year old. One patient had mainly occular symptoms, but latterly had complained of tiredness at the end of the day. The other two patients had muscular weakness. In all three patients there was a recent deterioration on medication and hence their consideration for surgery. There was incipient respiratory failure in one patient, and the response to Neostigmine was poor. All three patients were assessed by a physician with a particular interest in the disease. Two patients had been on Neostigmine for periods of 9 years and 1½ years, the indication for operation being a recent deterioration while on medication. The third patient whose illness was of shorter duration had rapid progression of symptoms coupled with inadequate response to Neostigmine. In all 3 cases the thymus together with the surrounding fat was excised after a sternal splitting incision. There was no evidence of local infiltration, unlike in the cases of malignant thymoma as described above. All 3 patients were extubated soon after operation and appeared to have adequate ventilation. The two younger patients had a marked improvement in respiration immediately after operation requiring only a single dose of Neostigmine on that day, compared with 4 or 5 doses as their pre-operation requirement. The dosage of Neostigmine was assessed on clinical grounds, assessing the adequacy of ventilation and pupillary size for which reason Pethidine was preferred to Morphine as an analgesic. The reduced dosage seemed to be adequate for 48 hours after which both patients were gradually restored to their previous dosage. In spite of requiring medication at the same level both these patients felt symptomatically better and there was objective improvement in muscle power and in occular movements. Review one and twomonths post-operatively both patients maintained their improved state and were back at work. The third patient appeared to maintain a steady state with just adequate ventilation but he sustained a sudden cardiac arrest 40 hours post-operatively.

Discussion

The symptoms from anterior mediastinal tumours are non-specific and signs are virtually non-existent in the early phases. However in the cases presented in this paper symptoms were severe and physical signs of an intra-thoracic lesion obvious albeit not pathognomonic. It is now well established that the diagnosis should be made radiologically at the pre-symptomatic phase. Ringertz and Lidholm as long ago as 1956 found that 61% of their cases were diagnosed while still asymptomatic. While a correct tissue diagnosis can only be predicted with about 80% accuracy, it is yet of only academic interest in sofar as all mediastinal tumours must be excised. and further treatment with cytotoxic drugs or radiotherapy

can then be determined. Thymic neoplasms, teratodermoids and retrosternal thyroids account for over 90% of all anterior mediastinal neoplasma, and only rarely are other mesenchymal neoplasma seen in this situation. Lymphomas and lymphangiomas too occur rarely, but are more commonly situated in the middle mediastinum. Thyroids and thymic neoplasms are perhaps more commonly seen in the superior part of the anterior mediastinum but when sufficiently large may occupy the whole of the anterior mediastinum or may project into one or other of the pleural spaces, as may teratodermoids. Two of our cases were so large that it occupied virtually the whole of a pleural cavity. The presence of calicification within the tumour is of some diagnostic value, occuring more commonly in teratodermoids. Radioisotope scanning with I^{131} is useful in the diagnosis of retrosternal thyroids, which are usually associated with cervical thyroid enlargement, the retro-sternal component being a prolongation of the inferior pole which commonly passes downwards anterior to the left innominate vein and then occupies the anterior mediastinum; when it passes down behind the innominate vein the retrosternal thyroid is liable to project to a greater extent into the pleural cavity and is situated in the posterior mediastinum.

The 80% incidence of malignancy in anterior mediastinal neoplasma in our cases is high, and must be a reflection of the diagnosis and admission of patients for surgery. No doubt there must be a large number of patients with benign neoplasms still walking the streets of Jaffna, the non-specificity of their symptoms and paucity of physioal signs in the early stages obscuring the diagnosis purely because routine chest Xrays were not taken. Whether malignancy supervenes with time in a previously benign tumour is unknown. The incidence of malignancy for all mediastinal tumours reported from Colombo was only 29% (Paul, Rasaretnam & Yoganathan 1970), while slightly higher figures were reported from other countries. Considering thymomas alone, Batata, Martini, Huvos et al (1974) had a 66% malignancy rate, while Weissberg, Goldberg and Pearson (1973) quote a figure of 47%. Malignancy in thymomas is judged on the presence of local infiltration and metastases rather than on histological criteria, and hence must be an operative decision. Histologically four main cell types are recognised: Lymphoid, lymphoepithelial, epithelial and spindle cell varieties. Benign tumours were completely encapsulated while malignant tumours were either non-encapsulated or incompletely encapsulated and invasive (Batata et al 1974.) It is difficult to predict behavioral pattern on histological criteria but in an analysis of 46 thymomas seen at the University of Michigan Medical Centre over a period of 34 years it was found that the epithelial variety showed the greatest tendency to aggression. All authors are agreed that surgical removal of a thymoma whether benign or malignant offers the best prognosis. (Weissberg, Goldberg and Pearson 1974: Batata et al 1974; LeGolvan and Abell 1977: Gerein, Srivastava and Burgess 1978). None of the patients with benign thymomas in their collected reviews had metastases at a later date, while both transpleural seeding and haematogenous metastases were subsequently seen in the malignant cases. There is no clear evidence that either pre-or post operative radiotherapy or chemotherapy confers significant benefit.

Thymomas have been associated with variety of other clinical entities; red cell

aplasia, hypogammaglobulinaemia, thrombocytopenia, endocrine abnormalities such as hypothyroidism, hypercalcaemia and Addison's Disease. Only one of the patients in our cases who had thrombocytopenia had any of these syndromes.

Myaesthenia gravis is a disease that is still poorly understood; and yet most authorities agree as to the value of thymectomy in these patients. However neurologists do not advocate thymectomy in all myaesthenics. The presence of occular symptoms alone is not regarded as sufficient to warrant thymectomy, and it is probably common ground that thymectomy should ideally be performed in patients who are either unresponsive to, or show escape from cholinergic medication. The best response is seen in young females with a myaesthenic history of less than one year. Patients without thymomas tend to fare better than those with neoplasms. Obviously malignancy carries an increased risk. Paradoxically, myaesthenia gravis has appeared for the first time a short while after thymectomy (Weissberg et al 1973). Thymectomy for a patient with myaesthenia gravis was first carried out by Sauerbruch in 1912, and the procedure was subsequently made rational by Keynes (1946) and Blalock (1939). Until recently the operation has been associated with a not insignificant morbidity and mortality mainly due to post-operative respiratory problems. Fraser, Simpson and Crawford (1978) reported 64 patients managed with no deaths in the immediate post-operative period and only 6 late deaths, attributing their success to team work by neurologist, anaesthetist and surgeon. Medication was continued pre-operatively omitting only the last dose due before operation and subsequent doses being ordered by the neurologist assessing the degree of weakness and avoiding cholinergic crises by assessing pupillary size. In the immediate post-operative period there is a reduced requirement for medication and in some centres cholinergic drugs have been witheld for periods up to 72 hours. Where respiration has been inadequate, intermittent positive pressure ventilation has been used temporarily and if the necessity for it is greater than 72 hours then tracheostomy has been advised. Jaretzki, Bethea, Wolff et al (1977) drew attention to the presence of accessory lobes to the thymus and even the presence of thymic tissue in the fat surrounding the gland and hence advocated adequate exposure by both median sternotomy and cervical incision so as to obtain complete clearance. However, because of the increased morbidity with this operation some surgeons have used a transcervical approach (Genkins, Papetes, Horowitz et al 1975; Kark and Kirschner 1971). While the whole thymus gland can be excised transcervically, accessory thymic rests may not be reached, but there is no clear evidence that such remnants cause persistence or deterioration of myaesthenia. Improvement in over 90% of their patients after thymectomy have been reported by Fraser et al (1978) and Papatastas et al (1971).

By and large the patients in our report have been treated late, and in the case of the neoplasms it has been associated with an 80% mortality within the year. Late diagnosis has been due to a variety of causes not the least important of which is adequate time, personnel and facilities for the investigation and management of the large number of patients attending the hospitals. More particularly, the lack of routine chest radiology in patients with non-specific symptoms must mean that a large number of patients with curable lesions are being missed. The purpose of this paper is to draw attention to the

Case No.	Age : in yrs.	Sex :	Duration of illness	Symptoms	Signs	Xray findings & other data	Operative findings	Result
Mediastinal tumours								
1	18	F	2 mths	fever, cough, dyspnoea, chest pain, generalised weakness	dyspnoeic, mediastinal shift to left, PN dull over the rt. chest, and AE reduced	opacity almost filling the whole rt chest. Ant: mediastinal tumour in lat: view	Benign teratodermoid. wt: 1132 grams	Alive 1 yr.
2	50	F	1 mth	anorexia, loss of weight, cough evening pyrexia	anaemia, left pleural effusion	Xray: opacity over left chest. diabetes, myxoedema, hysterectomy for Ca Cx	Malignant teratodermoid Operation elsewhere	Died 2 hrs-post opn.
3	55	F	2 mths	fever, cough, chest pain, dyspnoea	febrile, PN dull, & AE reduced over rt chest, orthopnoeic	Xray: opacity over lower rt chest, with ant: mediastinal tumour on lat: view	refused operation	lost to follow up
4	11	F	3 mths	cough, dyspnoca, wheezing, hoarseness, stridor, dysphagia	stridor, orthopnoea increased area of precordial dullness, hard lump just palpable in suprasternal notch, JVP elevated liver 4 FB's	Xray: anterior mediastinal tumour projecting slightly into both pleural cavities. Thrombocytopenia	malignant lymphoid thymoma wt: 570 grams tracheostomy for IPPV	died 48 hours post opn.
5	42	M	4 mths	cough, dyspnoea, dysphagia hoarseness	orthopnoea, stridor, anaemia. hard was felt in the mass suprasternal region	Xray: widening of mediastinum due to ant; mediastinal tumour	inoperable malignant thymoma - spindle cell	died 10 hrs post opn.
6	16	F	6 mths	cough, dyspnoea, chest pain, generalised weakness	anaemic. mediastinal shift to the right, PN dull & AE reduced on the left	Xray opacity entire left chest due to ant: mediastinal tumour	malignant thymoma, lymphoepithelial wt: 1250 grams	died 12 months post opn.
7	70	F	6 mths	swelling of face & neck, hoarseness, dysphagia	SVC obstruction, cervical and left axillary lymphadenopathy	Xray: widening of mediastinum.	refused operation	lost to follow up
Myaesthenia gravis								
8	26	M	9 yrs	weakness of occular muscles, dysphagia, tired-	bilateral complete ptosis and external ophthalmop-	Xray: N.A.D.	Thymic hyperplasia wt: 20 grams	improved

					orthopnoeic	stinal tumour on lat: view		up
4	11	F	3 mths	cough, dyspnoca, wheezing, hoarseness, stridor, dysphagia	stridor, orthopnoea increased area of precordial dullness, hard lump just palpable in suprasternal notch, JVP elevated liver 4 FB's	Xray: anterior mediastinal tumour projecting slightly into both pleural cavities. Thrombocytopenia	malignant lymphoid thymoma wt: 570 grams tracheostomy for IPPV	died 48 hours post opn.
5	42	M	4 mths	cough, dyspnoea, dysphagia hoarseness	orthopnoea, stridor, anaemia. hard was felt in the mass suprasternal region	Xray: widening of mediastinum due to ant; mediastinal tumour	inoperable malignant thymoma - spindle cell	died 10 hrs post opn.
6	16	F	6 mths	cough, dyspnoea, chest pain, generalised weakness	anaemic. mediastinal shift to the right, PN dull & AE reduced on the left	Xray opacity entire left chest due to ant: mediastinal tumour	malignant thymoma, lymphoepithelial wt: 1250 grams	died 12 months post opn.
7	70	F	6 mths	swelling of face & neck, hoarseness, dysphagia	SVC obstruction, cervical and left axillary lymphadenopathy	Xray: widening of mediastinum.	refused operation	lost to follow up
Myaesthenia gravis								
8	26	M	9 yrs	weakness of occular muscles, dysphagia, tiredness at end of day	bilateral complete ptosis and external ophthalmoplegia	Xray: N.A.D.	Thymic hyperplasia wt: 20 grams	improved
9	28	M	1½ yrs	weakness of occular muscles, dysphagia, weakness of all 4 limbs	bilateral ptosis, weakness of all 4 limbs	Xray N.A,D.	Thymic hyperplasia	improved
10	57	M	1½ mths	weakness of occular muscles, weakness of limbs, dyspnoea and increasing respiratory insufficiency	bilateral complete ptosis, external ophthalmoplegia, weakness of limbs, poor respiratory movements of chest wall	Xray N.A.D.	Atrophic thymus wt: 20 grams	Died 48 hours post opn.

PN—percussion note

AE—air entry

Table I—Clinical details of the 10 patients

mediastinum. Acknowledgement: It is with pleasure that we thank Dr. S. Sivakumaran M. D., M. R. C. P., and Dr. R. Natkuam F. R. C. S. for allowing us to include the two patients admitted under their care.

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PULMONARY ALVEOLAR PROTEINOSIS — A CASE REPORT

D. CHANMUGAM, M. D., F. R. C. P. (London)
Department of Medicine,
Faculty of Medicine, University of Ceylon (Colombo).

ROSEN, Castleman and Liebow in 1958 described a hitherto unrecognized disorder of the lungs which they called Pulmonary Alveolar Proteinosis. They were struck by the characteristic appearance histologically where most of the pulmonary alveoli were filled with a granular eosinophilic material which gave a positive reaction to Periodic Acid Schiff stain. The disease commonly occurs in young and middle age men with the most important symptom being dyspnoea. In 20% of cases the onset is an acute febrile illness. Radiographic changes are always present and vary, commonly there is diffuse lung shadowing radiating from the hilum mimicking pulmonary oedema. Since the original description many cases have been reported from all over the world. The first case from Ceylon is reported below.

Case Report

N. J. a 56 year old Muslim house wife was admitted with a four month history of progressive shortness of breath of insidious onset accompanied by cough productive of varying amounts of sputum sometimes purulent. She was seen for these symptoms at the chest clinic from where she was referred to me. She did not have any other symptoms. At no time did she cough up blood. There was no family history of respiratory illnesses; she had never been exposed to any industrial pollution neither had she taken any drugs. On admission (No. 164123) she was of average build, centrally cyanosed and breathless at rest. There was no finger clubbing or cervical lympadenoapthy. The relevant physical signs were confined to the chest and consisted of bilaterally diffuse coarse crepitations (crackles). Apart from moderate polycythaemia (Haemoglobin concentration =16·7 grams dl) the results of routine investigation were normal. The chest X'ray (Fig. 1) revealed bilateral varying densities more on the right side and more towards the hilum. Pulmonary function studies (Dr. A. T. W. P. Jayawardena) did not reveal any airways obstruction. As B. Friedlander was grown from the sputum (Prof. T. E. D. Chapman) she was treated with parenteral Ampicillin and intermittent oxygen through a nasal catheter and postural drainage. Though there was a marked improvement in the cough the dyspnoea and radiographic changes were unaltered. An open lung biopsy (Dr. R. Natkunam) was done and reported (2612 u/78) as showing the changes of Pulmonary Alveolar Proteinosis (Prof. M. Yoganathan and Dr. G. Vijeyaratnam). A trial of steroids was tried without any improvements. Her subsequent progress was steadily downhill and she died three months after admission in respiratory failure.

Discussion

Many of the earlier case reports were in individuals who were exposed to industrial dusts and it was therefore thought then

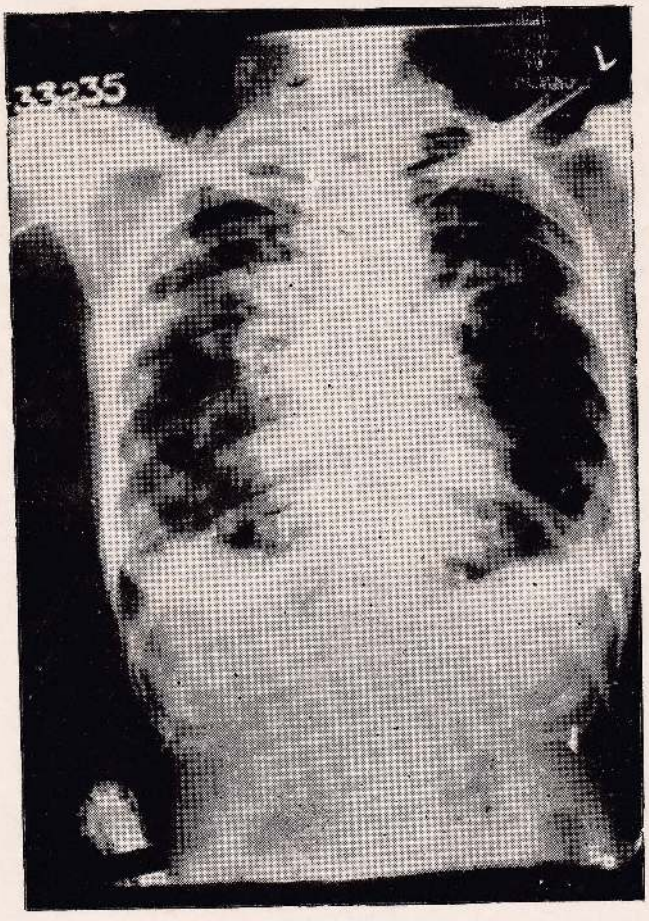

FIG. 1

that this represented a new pneumoconiosis. But later studies showed that the material filling the alveoli was composed of phospholpids which were identical to the surfactant found in normal lungs. This is a highly active surface lipid layer lining normal alveoli and essential for normal alveolar architecture. Too little of this material results in alveolar collapse and atelectasis such as is seen in the respiratory distress syndrome of the newborn. Excess amounts of this lipid material may be the causative feature in Pulmonary Alveolar Proteinosis producing a reduced lung volume with impaired gas transfer resulting in progressive pulmonary insufficiency. Cough with productive sputum may be result of secondary infection including fungi. Other features which are not specific are polycythaemia, cyanosis, increased serum lactic dehydrogenase abnormal arterial gas tensions and markedly reduced lung compliance and diffusion capacity. The abnormal radiographic appearances are not diagnostic and clinically Pulmonary Alveolar Proteinosis is difficult to distinguish from pulmonary oedema, fibrosing alveolitis, sarcoid or even chronic infections. The diagnosis is unequivocally established by lung biopsy where on microscopy large groups of alveoli filled with granular acidophilic material frequently containing laminated round or ovoid bodies and acicular clefts. From the walls of the alveoli large mononuclear cells can be seen rising. Rosen et al (1958) believed that these septal cells form early in the disease, fill the alveoli, develop the PAS positive granules and then disintegrate producing the characteristic alveolar contents. Since lung biopsy either closed or open involves considerable risk to the dyspneic patient Vidone, Hoggman et al (1966) have shown that sputum examination alone can be utilized to show the presence of the characteristic material and this has been used by Costello, Moriarty et al (1975) successfully in the diagnosis of Pulmonary Alveolar Proteinosis.

Davidson and Meleod (1969) reviewing 139 cases showed that the prognosis generally was poor but that spontaneous improvement had been noted, and in some of the patients the disease had been self limiting (Dobson and Karlish, 1975) Steroids and antibiotics have been used extensively without any consistently significant improvement. Since the use of pulmonary lavage successfully by Jenkins, Teichener et al (1975) this would seem to offer the best chance to the patient. The lavage which is usually done under general anaesthesia with normal saline alone or fortified with heparin or acetyl cysteine may have to be repeated several times. But it should be noetd that several authors who have used lavage successfully admit that recovery in their cases may have been fortuitous.

The aetiology of this condition remains obscure. Rats exposed to silica dust have been shown to develop a similar histological appearance. In one of their cases Costello, Moriarty et al (1975) showed that the same specimen of lung obtained by needle biopsy showed both lipoid pneumonia and Pulmonary Alveolar Proteinosis, and they postulated that lipoid pneumonia was a forerunner of the latter. Of great interest is the work of Vijeyaratnam and Corrin (1973) who have described alveolar proteinosis in rats given a chemical agent IPRINDOLE. The relevance of this in the human disease remains to be established.

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A CASE OF GENERALISED MORPHOEA

M. NADARAJAH, M. R. C. P.
Consultant Dermatologist,
General Hospital, Jaffna.

P. MAYURANATHAN, M. B., B. S.
House Physician,
Dermatology Uuit,
Genenal Hospital Jaffna.

Mrs. R GANESWARAN, M. B,, B. S.
House Phvsician,
Dermatology Unit,
General Hospital Jaffna.

Place :
Dermatology Unit,
General Hospital, Jaffna.

Introduction :

MORPHOEA (Syn. Localised Scleroderma) is an uncommon disease involving the skin and its appendages, in contrast to systemic sclerosis (Syn. Generalised Scleroderma) where the systemic manifestations of the disease are more prominent. Morphoea had been divided into further sub groups by the morphological characteristics of the lesions A guttate and plaque types have been identified. Here the lesions are small, superficial with a smooth surface, and Ivory colour. While enlarging, they may show a violaceous border. Lesions of linear type are known to occur predominently on the extremities and scalp (Dilley et al, 1968). Segmental Morphoea affects large areas of skin on the face and one or more extremities and may result in atrophy of the underlying subcutaneous fat and muscles leading to contractures. Generalised Morphoea is the term used to describe very extensive cases showing a combination of the four types described above (Christianson et al, 1956 Driessen et al 1966).

Generalised Morphoea is a rare condition predominantly affecting the female in which there is sclerosis of the skin occuring in a wide-spread manner, usually starting in the trunk. This condition, as mentioned above, is marked by the absence of systemic manifestations. A causal factor has not been identified, so far, in this disease (Wilkinson, Arthur Rook and Ebling 1972).

A case of generalised morphoea is descrided below :

Case Report :

Miss. V, a sixteen year old school girl from Mullaitivu (Vavuniya District) came to our Clinic on the 7th of July, 1978 with a complaint of enlarging disfiguring patches involving the abdomen and Chest for the last one year. Her illness started as a small patch of dark, thickened area in the skin overlying the left anterior iliac spine and a similar patch over the upper half of the right breast. These patches enlarged circumferentially and 2 months later, new lesions appeared on the right side of the lower abdomen and overlying the upper part of the left breast. After a further period of two months, more lesions appeared over the shoulders, upper portion of the back, of chest in front, over the upper chest area, along with a ribbon-like, elongated area of similar discoloration on the dorsum of left mid-fore-arm. Main symptoms that

troubled her were the disfigurement caused by the patches, absence of sweating in the affected areas and and an occasional pricking pain lasting a few minutes involving her breasts. She was otherwise normal.

She is the 4th child in a sibship of 3 boys and four girls. Her parents are living and are apparently in good health. Her father and her elder sister have Icthyosis involving mostly their legs. Her father had suffered from pulmonary tuberculosis and got well after a full course of anti-tuberculous therapy. None of her close relatives are affected by any other skin ailment, birth deformities or psychiatric illness. She attained menarche at 13 years of age. Her menstrual flow and cycles were normal. She had had recurrent attacks of malaria for about 4 years, which occured once a month and this was treated with chloroquine and primaquine. For the last one year, she is free of malarial fever.

She was dark complexioned girl of above average build for her age, height 154 cm. weight - 44¾ kg. with a keen intellect. In the affected areas, skin was found to be hyperpigmented, dry and indurated with a smooth waxy surface. In the front of lower abdominal wall, an oval area 6"×8" in diameters of affected skin was seen. However, in the centre of the lesion, the umbilicus and a circum umbilical area of 1" width was spared (figure I). On either side, this lesion extended round the waist to form a band 4" in width. Similar areas were seen, involving both breasts, circumscribing the areola in a circle of 7" diameter. The skin of the areola and nipples were spared (figure I). On the back an oval patch, 2"×6" in the midline was identified. Rounded patches over both shoulder prominences with a dimension of 3" diameter were also seen. A linear area of affected skin was identified, 8"×½", overlying the middle area of left forearm, on its dorsal aspect. A smaller lesion in the scalp area, near the hairline, overlying the left temporal region (2" in diameter) was identified. Small patches were noted in the skin behind the earlobes, and overlying the mastoid areas. No definite border was identified in any of the above lesions. There were no atrophy of muscles, or contractures associated with these lesions. She had mild Icthyotic skin involving both legs below the knees. Her other systems were found to be normal,

The following investigations were done :

Hb 11·6 g. %, blood picture - within normal limits; WBC 10, 200 per cu- m. m. N_{71} L_{27} E_2 ; ESR 18 m. m. 1st Hour; Urine full Report - was normal; Serum proteins - 7·2 g %, Albumin 3·6 g %; Globuiin — 3·6 g% L. E. cell test — Negative; Fluorescent antibody test for serum Antinucleoprotein factor — Negative; VDRL - non reactive; Rheumatoid activity - Negative; Chest 'X' Ray - showed bilateral cervical Ribs.

Biopsy of the skin in the affected area showed : a normal Epidermis with thickened dermis. Collagen in the dermis was thickened and arranged in closely packed areas of homogenisation, through the full thickness of dermis. There were scanty skin appendages and blood vessels in the section. A single sweatgland was found to be closely "bound down" and embedded in the homogenised collagen. Patchy areas of inflammatory exudates (mainly lymphocytes) were identified in the area surrounding the sweatgland,

around blood vessels and in the upper dermis. (Fig III)

Discussion:

A diagnosis of Localised scleroderma was considered in this case due to the characteristic appearance of hyperpigmented, indurated smooth patches of the skin with intact sensation and of long duration. Confirmation of the diagnosis was sought by the examination of skin biopsy. Remarkable features in the biopsy were the presence of thickened and closely packed dermal collagen from the subcutaneous fatty layer to the papillary layer, hugging the latter closely, the absence of fatty tissue around the sweatgland, and the normal epidermis. These features point to a late sclerotic stage in the disease process (Lever, 1975). The distribution of Sclerotic skin was interesting. Although it involved areas surrounding the umbilicus, areola and nipples, the latter areas were remarkably spared. High consideration was given to this feature by Christianson et al (1956), in their description of generalised morphoea. An absence of systemic symptoms: normal propulsive movement in the oesophagus evidenced by screening, negative antinuclear antibody test and an insignificant elevation in the erythrocyte sedimentation rate were additional features supporting a diagnosis of localised scleroderma. The presence of large plaques of affected skin, segmental lesions and linear lesion in our patient entitled the localised sclerotic lesions to be referred as Generalised Morphoea (Christionson et el, 1956).

Arthralgia and Raynaud's phenomenon were noted by Christionson et al, 1956 to be associated with generalised morphoea in a proportion of cases. The linear lesion in the left forearm is not associated with arthralgia in our patient. Absence of Raynaud's phenomenon is noteworthy in our case. Absence of atrophy of muscles and contractures of muscles and tendons in the areas underlying the scalp lesion and the linear lesion of the forearm are worthy of note (Hickmenn et al, 1964) Skeletal anomalies especially congenital unilateral atrophy of upper and lower extremities and spinal coloumn deformities were associated with generalised morphoea (Christianson et al, 1956) However, an association between the presence of cervical ribs and generalised morphoea was not documented Our patient illustrates the presence of bilateral cervical ribs in a case of generalised morphoea (figure II). An absence of symtpoms in the upper extremities, inspite of cervical ribs and generalised morphoea is remarkable in our case. As noted in the study by Christianson et al 1956, our patient too had a naevus in her addominal wall (figure I). Symptoms of neuroses in cases and their relatives were noted in the same study. This association in lacking in the case illustrated here. Normal menstural cycles in our case contrasts with some of the cases studied by Christianson et al (1956). Further, the large family and the absence of miscarriages amongst her close relatives is striking against the conclusion drawn in the same study by Christianson et al.

All the available types of treatment are unsatisfactory and spontaneous recovery is not uncommon in generalised morphoea. One regime of therapy advocates the use of chloroquine for about 3 months (Domonkos, 1971). It is noteworthy that our patient had recurrent attacks of malaria for about 4 years and was on chloroquine therapy, once a month. However, she did not develop malaria after the onset of

her skin ailment. Corticosteroid therapy does not offer any lasting benefit; but the patient feels better and the joint symptoms may be ameliorated when prednisolone 10—15 mg. taken daily (Domonkos, 1971). In the absence of joint symptoms systemic corticosteroid therapy was not considered in this case. Equivocal results were obtained by Jansen et al, 1968, in his study with azathioprine A high protein diet with liberal vitamins supplements, warmwater baths and general exercise were the only therapeutic measures meted out to this patient. However the fact that the disease becomes inactive in 3-5 years and has a good prognosis, is indeed gratifying (Fleischmajer et al, 1972)

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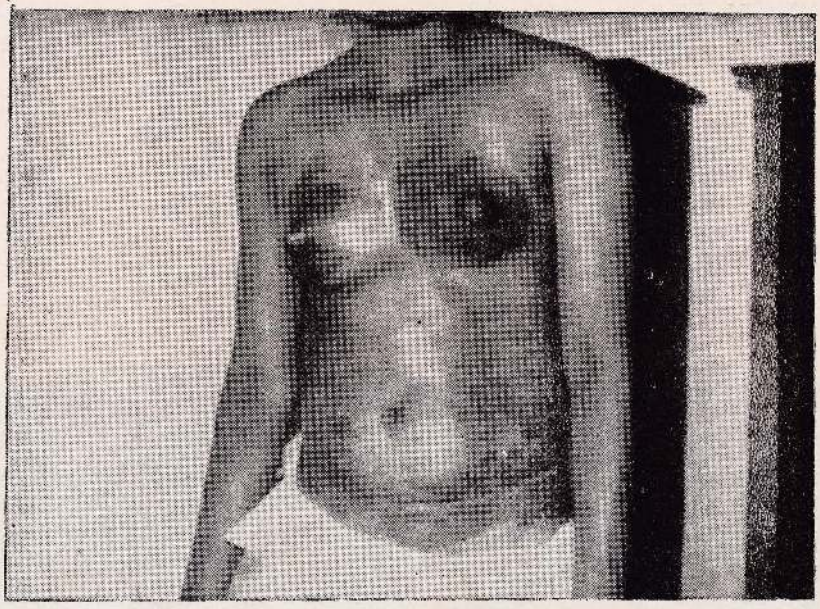

FIG. I

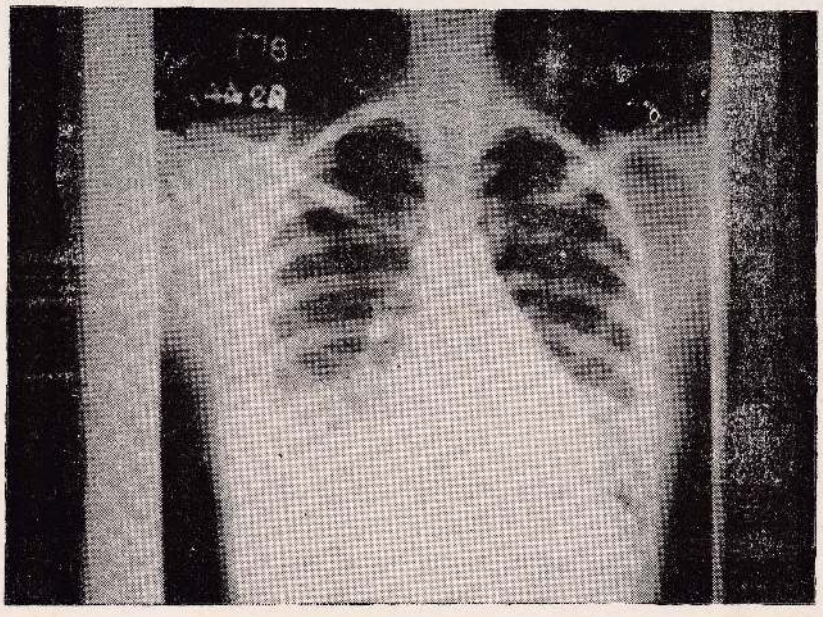

FIG. II

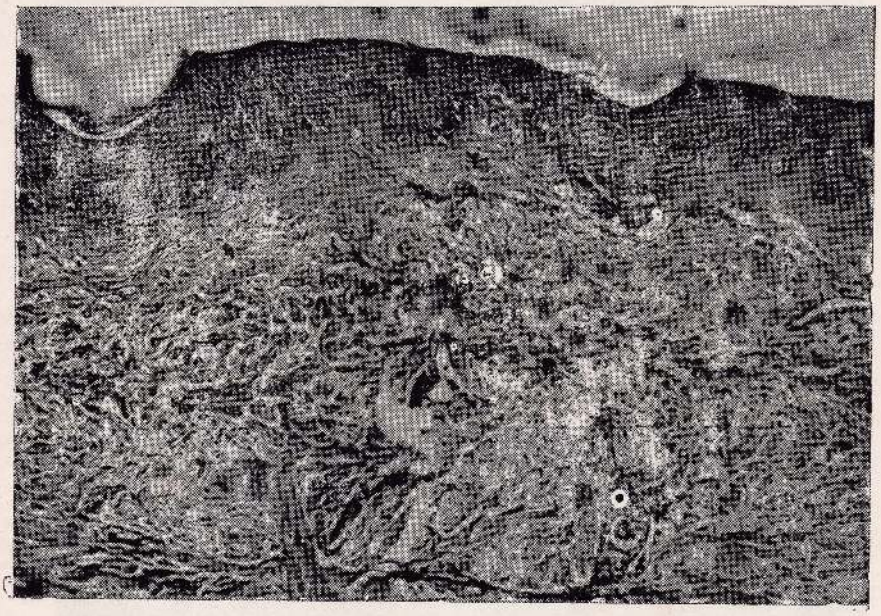

FIG. III

SICK SINUS SYNDROME — CASE REPORT

P. MAYURANATHAN, M. B., B. S.
House Physician,
Dermatology Unit,
General Hospital, Jaffna.

S. SUNTHARANATHAN, M. B., B. S.
House Physician,
Medical Unit — B,
General Hospital, Jaffna.

Venue :
Medical Unit — B
General Hospital, Jaffna.

Summary :

A case of sick sinus syndrome with an unusually prolonged history of symptoms and having a congenital aetiology is presented here.

Introduction :

Sick Sinus Syndrome is a heterogenous group of electrocardiographic abnormalities occuring as a result of sinoatrial node (SANode) dysfunction. These electrocardiographic abnormalities may be one or more of the following :

(i) Persistent severe and unexpected sinus bradycardia (ii) Cessation of sinus rhythm (sinus arrest) for short intervals during which no other (escape) rhythm arises or somewhat longer periods with replacement of sinus rhythm by an atrial or junctional rhythm (iii) long periods of sinus arrest without the appearance of a new pacemaker resulting in total cardiac arrest (ventricular arrhythmias may then follow) (iv) Chronic atrial fibrillation or transitory atrial fibrillation, often but not always accompanied by a slower ventricular rate (which is not due to digitalis) (v) Inability of the heart to resume sinus rhythm following cardioversion for atrial fibrillation and (vi) episodes of SANode exit block which are not related to drug therapy. The aetiological factors that are related to the development of sick sinus syndrome are not clear, but sick sinus syndrome had been identified in wide and totally different clinical situations. These clinical situations range from Congenital, Inflammatory, Ischaemic and rheumatic conditions to surgical injury and Infiltrative diseases of the Atria (Ferrer, 1973).

Sinoatrial node is a specialised structure situated in the posterior aspect of the right atrial wall near the opening of the superior venacava. Some of the cells of SANode have an inherent automaticity and discharge at a faster rate than any other cell in the heart. Any extraneous influence that alters the rate of depolarisation and repolarisation of the nodal cells will alter the rate of electrical impulse initiation in the SANode and hence, the rate of the heart. SANode is richly innervated with sympathetic and parasympathetic fibres. Under normal circumstances, the heart rate and therefore cardiac output is adjusted by the nervous and humoral mechanisms to fulfil the demands of the body.

In Sick sinus syndrome there is a dysfunction in the initiation and conduction of electrical impulses in the SANode, in response to varying demands by the body. As a consequence, on the one hand there is hypoperfusion of brain and cardiac muscle causing intermittent and life- threatening symptoms and in the other, there is congestion in the Pulmonary circulation leading to symptoms of Pulmoary Oedema.

Case Report :

Mrs. R., a 57 year old school teacher, having 4 children was in average health until 4 years ago. Four years ago, while she walked a distance of half a mile, one day, she developed tightening chest-pain in the retrosternal area accompanied by excessive sweating. After resting a while, the pain disappeared. Henceforth, this pain was reproducible by similar exertion. She curtailed her activities in order to avoid the pain and did not obtain treatment. For the last 1½ years she noticed a feeling of giddiness, and light headedness appearing on and off, in an irregular fashion. It was not related to exercise. The day prior to admission, while eating a meal, she developed pricking pain in the throat and retrosternal area which after few seconds enveloped her entire chest. She also noticed tightness of the chest, difficulty in breathing and excessive sweating. Slurring of speech accompanied the above symptoms. The following day on admission she had dull ache in the retrosternal area, giddiness and unsteadiness of gait.

She was recognised as a "sick girl" in her school career and was exempt from games as moderate exertion precipitated "choking attacks" for her. During such an attack, she found difficult to breathe, a tightness in the chest, inability to stand or lie flat and needed to be propped up and supported. The entire ordeal lasts 20 minutes and would die off warranting no further treatment. She was free of these attacks for the last one year. Till 10 years of age she was subject to "drop attacks". She had anaemia complicating her last pregnancy and child birth, 15 years ago. Her menopause was eleven years ago. There was no family history of significant illnesses.

On examination, she was comfortable, of average build and afebrile. She was not cyanosed. Her pulse was irregular and was about 60 per minute. Pulse volume and tension were satisfactory. Her jugular venous pressure was not elevated, Blood pressure recorded was 140/100 m. m. Hg. Her heart was clinically normal. Examination of her other systems too did not reveal any abnormality. On investigating : an electrocardiogram showed Sinus bradycardia with irregular rhythm; chest- "X" Ray-normal cardiac size with mildly congested lower Pulmonary zones; SGOT- 02 units per ml; Serum cholesterol 280 mg.%; Blood urea 40 mg.%; urine full report-Normal. Patient was advised bed rest and diazepam 10 mg, twice a day and hydrochlorothiazide 50 mg, once daily were instituted. She became pain-free but her giddiness and light headedness recurred.

Discussion :

The presence of persistent, severe Sinus bradycardia of about 50 per minute, normal cardiac enzyme studies and symptoms of poor coronary and cerebral blood flow of many years duration strongly suggested a possibility of sick sinus syndrome in this case. Sinus arrest for relatively long periods of time with regular

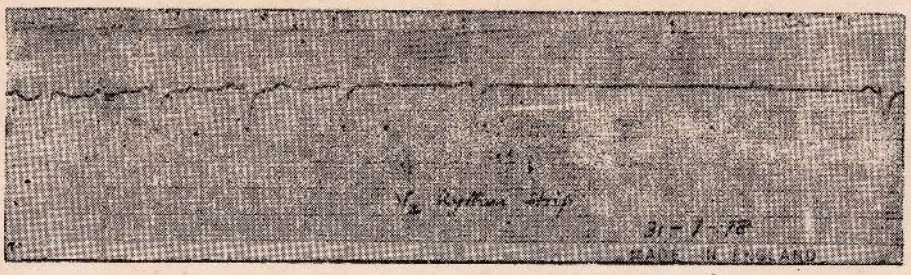

FIGURE I

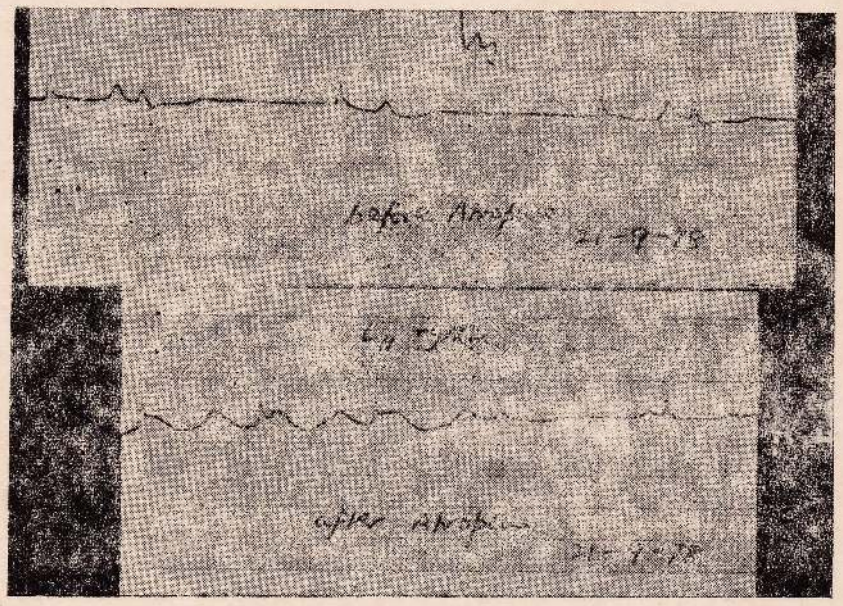

FIGURE II

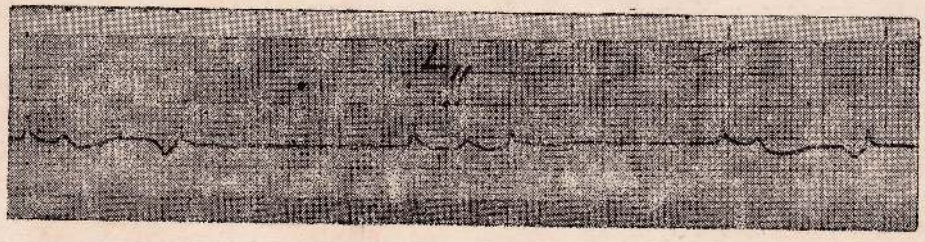

FIGURE III

Figure I

Figure II

Figure III

junctional impulse initiation was another feature noted in the electrocardiogram. During follow-up, a subsequent ECG. (Figure I) revealed the combination of tachycardia followed by sinus bradycardia. This combination represents delayed SANode recovery time (Ferrer, 1973). The diagnosis of sick sinus syndrome rests upon the degree of overdrive suppression. This is best expressed as a percentage of the Control rate (Mandel et al, 1971). At a resting cycle length of 1800 m. sec., a pause of 3200 m. sec. (Figure I) (i e.) a percentage increase of 78% (or 178% of control) is diagnostic of sick sinus syndrome, in our patient. Further, an intravenous injection of atropine sulphate (1 mg) did not increase the sinus bradycardia to a sinus rate of well-over 90 per minute (Figure II) (Rosen et al, 1971). Frequent long pauses, like that as seen in Figure 1 (a pause of 3 seconds) with no lower pace maker activity would lead to serious hypoperfusion of vital organs, causing symptoms. These long pauses were frequent after a period of overdrive of SANode. On the other hand, junctional rhythm took over (Figure III) when SANode failed, in the absence of SANode overdrive. These features conform to the finding that there is extranodal involvement of the same disease process in case. of sick sinus syndrome (Ferrer, 1973).

The presence of symptoms of Ischaemic heart disease for four years, sustained elevated blood pressure in spite of slow rhythm and an increased serum cholesterol are additional features seen in this case. Hence, there is an increased vulnerability towards precipitation of myocardial infarction in this patient, should a tachyarrhythmic episode occur. A ventricular demand pace maker was sought as remedy for her.

Ferrer (1973) in her extensive study of sick sinus syndrome included familial sinus node disease without other illness, a familial syndrome of heritable Q-T interval prolongation with arrhythmias and an isolated fibrotic local lesion of unknown cause as associated with sick sinus syndrome. A congenital predisposition is hence associated with some of the cases of sick sinus syndrome. In our case, although a family history is not forthcoming, congenital aetiology is thought of due to the absence of any other associated illness and the recent onset of symptoms of Ischaemic heart disease. Her respiratory distress episodes makes one ponder as to whether the dysrhythmic illness dates back to her second decade of life. Further, the "drop attacks" during her 1st decade of life could easily be infact stokes-Adams attacks (Ferrer, 1968), Natural History of Sick Sinus Syndrome although is prolonged, usually expands over a decade or so. Occurrence of symptoms of sick sinus syndrome over four or five decades is quite an unusual feature. Although the risk of asystole in the absence of a stable escape rhythm for a long period of time casts doubt over the existence of the disease for such prolonged periods of time, the presence of clear-cut history of respiratory distress symptoms without any other associated illness makes one to associate the symptoms with existing sick sinus syndrome. Since, the prognosis of Sick Sinus Syndrome is variable, as there are many people living comfortably with 'sick sinus' (Kaplan, 1978) for long periods, a congenital aetiology would be more appropriate in this case. However, we hope that further studies would elucidate the beclouded aetiological aspects of sick-sinus syndrome, as in the case discussed herein future.

Acknowledgement :

Our thanks are due to Dr. S. Sivakumaran, MD., M. R C. P. for permission to publish this case from his unit.

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TYPHOID FEVER WITH COMPLETE HEART BLOCK — A CASE REPORT

P. KULENTHIRAN, M. D., M. R. C. P.
Consultant Physician,
General Hospital, Jaffna.

Present Address:
General Hospital,
Batticaloa.

P. MAYURANATHAN, M. B., B. S.
House Physician,
General Hospital Jaffna.

Place:
Medical Unit,
General Hospital, Jaffna.

Summary

WE report here a case of typhoid fever, who developed persistent heart block with atrio-ventricular dissociation and was maintained on long-acting Isoprenaline Therapy.

Case Report

A 38-year old House-wife who was well until mid-July of 1974 developed continued fever. She had loss of appetite and generalised body-aches accompanying the fever. After five days of illness, she was admitted to General Hospital, Jaffna. On admission, she was found to be febrile, temperature being 101°F, midly anaemic and with a regular pulse of 68 per minute. Blood pressure was 120/70 m. m. Hg. Her jugular venous pressure was not elevated. On percussion, her heart was found to be enlarged. An ejection systolic murmur best heard in the mitral area was not conducted to the neck or axilla. Respiratory system was clinically normal. She had no palpable lymph nodes, Her liver, spleen and kidneys were not papable. There was no neurological abnormality. The following investigations were done. Haemoglobin 9·0 g. 100 ml. (60%); E.S.R. 130 mm. 1st Hour; S.G. O.T. 22 Units per ml; SAT-Report-S.typhi 'H' 1:200, S.typhi 'O' 1:50, para A 'H'- Negative Urine full Report - Albumin in traces, Red cells (3-5) per field, Pus cells (5-10) per field, granular casts were present in few numbers. On the third day of hospital-stay, therapy with chloramphenicol was started, On the 6th day, her pulse rate was 52 per minute and rhythm regular. The following day temperature was recorded at 98.6°F and prednisolone 30 mg, a day was instituted. The pulse rate dropped further to 48 per minute. An electrocardiogram taken at this stage showed bradycardia with sinus rhythm and prolongation of PR-Interval to 0·22 seconds. Four days later, a second electrocardiogram showed evidence of complete heart block with a heart rate of 40 per minute (Figure 1). After 17 days of hospital stay, she was discharged on a minimum "tailed-off" dose of prednisolone. After a few weeks at home, on resuming house-hold duties she developed syncopal attacks and reported to our Clinic. She was re.admitted and was started on Saventrine 30 mg. three times a day. Her pulse rate rose from 35 per minute to 60 per minute (Figure 2). She was discharged on Saventrine 30 mg. twice a day and was free of syncopal attacks, Presently, i. e., after four years of her initial illness she is still maintained on the same drug and is in reasonable health.

Discussion

Due to the availability of effective antityphoid chemotherapy, uncomplicated typhoid and paratyphoid fevers seldom pose therapeutic problems. In the untreated case, during the first week of illness when there is septicaemia and toxaemia due to liberated endotoxins, any organ in the body may suffer damage. The cardiac muscle may be affected during the early stages of illness leading to a myocarditis with bradycardia. Microscopically, during this phase cloudy swelling of the cardiac muscle with focal necrosis has been observed. In severe cases inotropic cardiac failure may ensue culminating in death (Muir, 1964). Occasionally, typhoid fever may be complicated by the appearance of endocarditis set up in the process of septicaemia (Muir, 1964). Temporary or permanent heart block is not recorded as a complication of typhoid fever. This case shows the development of complete heart block in the course of typhoid fever. This heart block was permanent.

Cloudy swelling and focal necrosis may involve any part of myocardium, the conducting tissue of the heart being no exception. The involvement of the latter, may lead to focal fibrosis and a permanent heart block. Our case, probably represents this stage in Pathology,

Complete heart block embraces many entities. These result from block to the normal electrical conduction of pacemaker impulses at varying levels. Distal to the level of block a new pace.maker takes on the function of initiation of excitation impulses. A block at the AV-nodal level with a new pace-maker at the bundle of His level (i. e.) distal to the block, might occur. Block may occur in the bundle of His with new pace-maker situated distally in the bundle. Complete AV-block distal to the bundle of His with subsidiary pace-maker situated at a point within the bundle branch-Purkinje system (with the resultant abnormal QRS - complexes in ECG) may also result. Persistent acquired complete heart block is most often of the last type. However, in our case the block is either in the AV - node or in the bundle of His as evidenced by normal QRS - complexes. His - bundle tracing is necessary to differentiate between AV-Nodal block and bundle of His block. Junctional pace-makers generally increase their discharge rates in response to exercise or after administration of atropine, provided that the autonomic innervation of the conducting tissue is intact. These patients may remain asymptomatic for relatively long periods (Damato 1975).

In general, persistent complete heart block necessitates an artificial pace-maker, But an Intra-His bundle block may leave a pace-maker still responsive to direct stimulation by Isoprenaline. The abolition of symptoms with long-acting Isoprenaline therapy is enough justification to avoid an artificial pace-maker. This patient did not have any relapse of symptoms while on Saventrine (30 mg. twice a day) for the last four years.

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MICROPERFORATIONS OF THE CERVICAL OESOPHAGUS

J. CANAGARAJAH, M. B., B. S.
House Surgeon, Thoracic Unit.

N. SABANAYAGAM, M. B., B. S.
House Surgeon, General Surgery.

Jaffna General Hospital.

Summary :

THREE cases of microperforations of the cervical oesophagus in each of whom spontaneous healing occurred are presented. Two were caused by indirect trauma while the third was due to a foreign body. The lesser degree of local tenderness than was expected was a useful guide to management.

Introduction :

Perforations of the oesophagus are surgical emergencies associated with a high morbidity and mortality due mainly to delay in effecting definitive repair. Most writers advocate repair of the perforation and drainage of the contaminated space (Sawyers, Lance, Foster, et al 1975; Triggiani and Belsey 1977; Janssen 1976)... Sawyers et al (1975) had a mortality rate that was increased fourfold when the repair was delayed for more than 24 hours. Triggiani and Belsey emphasized the importance of an aggressive surgical approach for perforations of the thoracic oesophagus in view of the problems that might otherwise accrue from untreated mediastinitis. As the oesophagus shows a propensity for spontaneous healing with time, perforations involving the cervical segment may be managed with drainage of the contaminated tissues of the neck alone as the problems of mediastinitis do not necessarily occur in these patients (Triggiani and Belsey 1977).

Case Reports :

CASE 1. A 29 year old man who fell off a moving tractor and struck the front of his chest on the ground complained of pain in the chest and neck and pain on swallowing. He had neither cough, nor haemoptysis. There were no signs of external injury. He was mildly dyspnoeic and in pain, and there was extensive bilateral surgical emphysema of the neck with mild tenderness in the same region. The cardiovascular and respiratory systems were nomal. A radiograph of the chest and neck showed deep surgical emphysema confined to the cervical region. There was no mediastinal emphysema, nor pneumothorax. The lung fields were normal. A presumptive diagnosis of oesophageal perforation was made and he was treated conservatively with avoidance of oral feeding, intravenous therapy and antibiotics. Two days later he was symptom free and the emphysema was no longer evident. On the 7th post-injury day he was discharged from hospital but was readmitted on the 10th day with a history of a single bout of haematemesis after forceful vomiting just after a meal. Pain was absent. At oesophagoscopy the following day an old linear haematoma 20 mm long and 2—3 mm wide at 18 cms was seen in the four o'clock position. The rest of the oesophagus was normal. A repeat radiograph of the chest and neck was normal. His recovery was uneventful.

CASE 2. A 28 year old man presented with a complaint of painful swelling of the neck and difficulty in swallowing which occurred immediately after a road traffic accident when as a rear seat passenger he was suddenly thrown forwards. There was a momentary loss of consciousness and on recovery he noticed the above symptoms. On clearing his throat, he brought up a little blood. His general condition was fair. There was extensive bilateral surgical emphysema of the neck with mild to moderate tenderness in the same region. The cardiovascular and respiratory systems were normal. Radiography of the neck showed deep surgical emphysema with forward displacement of the oesophagus by a column of air (Fig. 1). A diagnosis of oesophageal rupture, probably a microperforation, was made and he was treated conservatively for 48 hours by which time symptoms had abated. Oesophagoscopy demonstrated a submucosal haematoma at 21 cms in the 11 o'clock position. It was 7 mm long and 4 mm wide. The surgical emphysema had disappeared by the third day and his subsequent recovery was uneventful.

CASE 3. A 20 year old female was admitted with a history of having swallowed the pointed half of a rusty safety pin one month previously. Soon after the accident she brought up some blood. She was treated by her family doctor with injections presumably antibiotics, for 5 days following which the pain disappeared. Two weeks prior to admission she again complained of chest pain and on "coughing" brought up blood. A firm linear foreign body was felt in the subcutaneous tissue on the left side of the suprasternal notch. There was no surgical emphysema. Tenderness was present only over the foreign body. The rest of the clinical examination was normal. A radiograph of the neck showed a pin lying slightly to the left and anteriorly in the lower neck At operation through a low transverse cervical incision a linear foreign body 4 cms long and encased in the sheath of fibrous tissue was found with the pointed end just deep to the cervical fascia and medial to the left anterior jugular vein while the other end was directed posteriorly at the root of the neck. A fibrous tract extended beyond the blunt end of the foreign body towards the oesophagus. There was no evidence of oesophogeal leak. The fibrous tract and the foreign body were excised. She made an uneventful recovery.

Discussion:

Perforations of the oesophagus may occur in the cervical, thoracic and abdominal portions. Cervical perforations may follow endoscopy. penetrating trauma of foreign bodies and rarely due to blunt trauma. Thoracic perforations may occur with all the above causes and also following paraoesophageal surgery and spontaneously (Boerhaave Syndrome). Perforations of the abdominal segment are usually associated with paraoesophageal surgery, and endoscopy and only rarely are spontaneous. Pain is the commonest sympton of oesophageal perforation and the location of which is dependant upon the site of the oesophageal tear. When the perforation involves the cervical oespohagus it is accompanied by pain and resistance of the neck to passive movement, together with subcutaneous emphysema and tenderness, the extent of the latter being an useful. index of the degree of tissue contamination. This sign is obviously lacking in thoracic lesions. Perforations of the

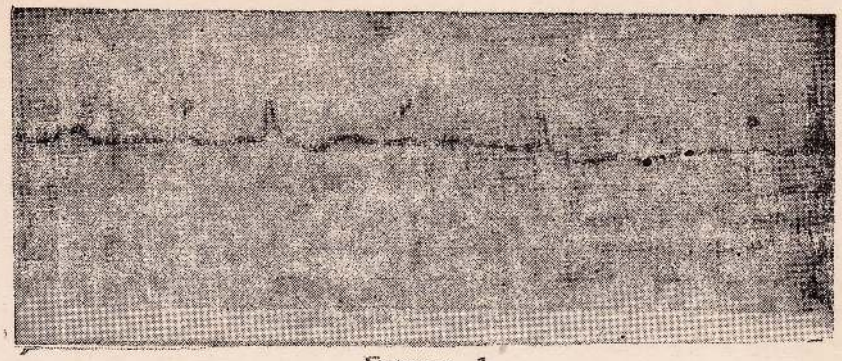


FIGURE 1

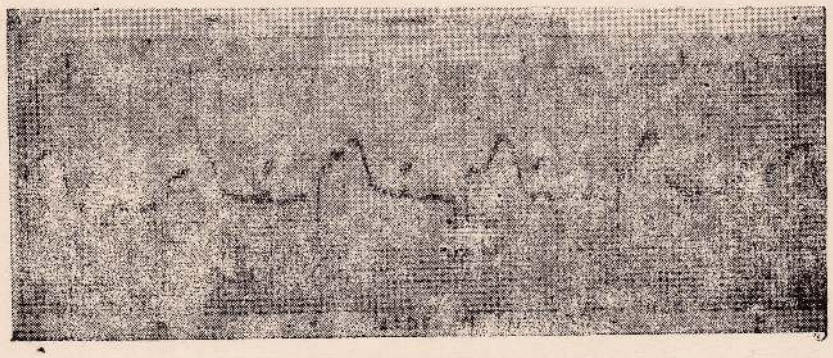


FIGURE 2

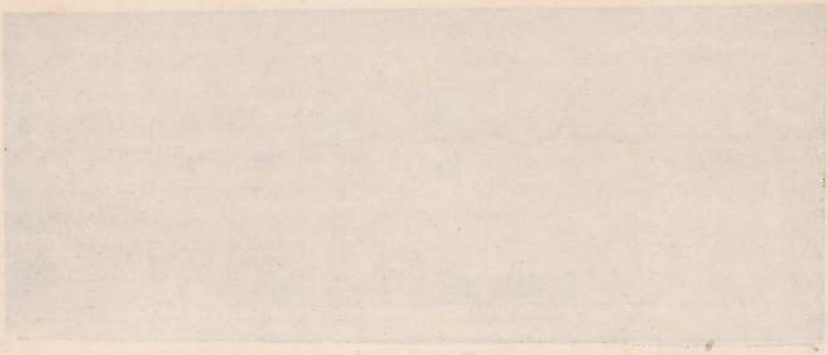


Figure 1

Figure 2

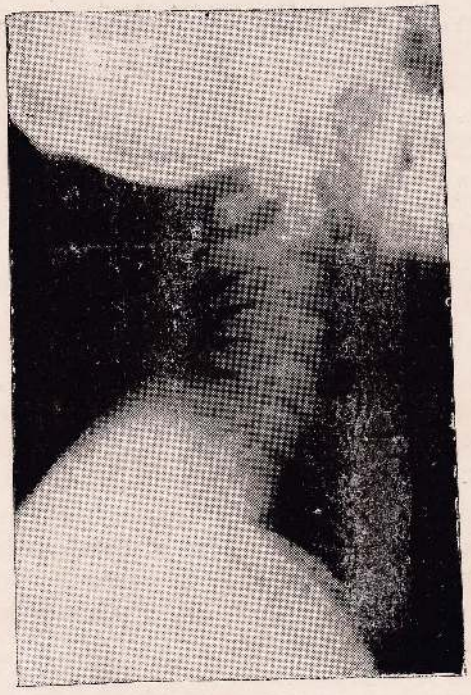


FIG. I (a)

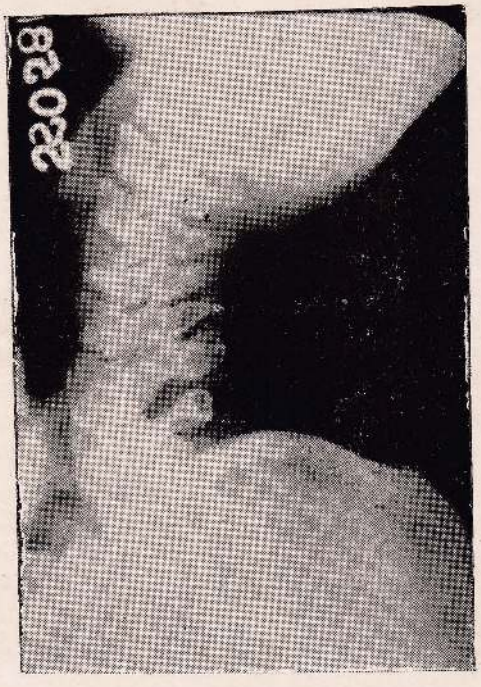
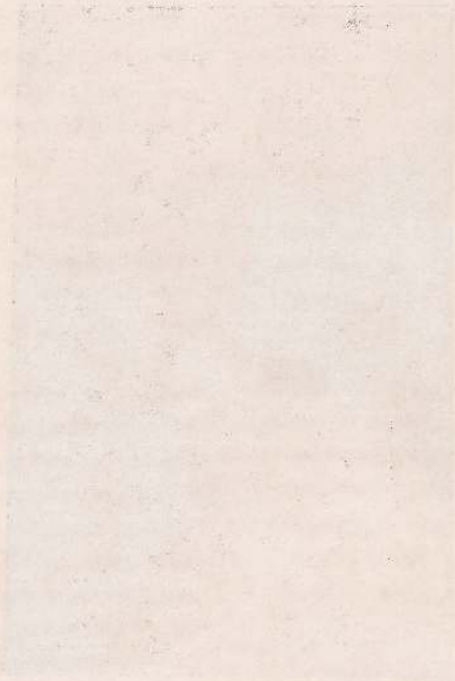
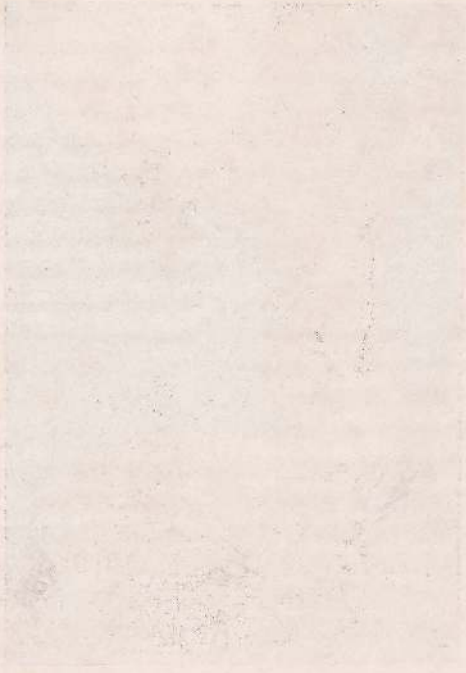


FIG. I (b)





thoracic oesophagus which cause mediastinal emphysema may extend to surgical emphysema of the neck, but the toxicity associated with mediastinitis should cause no confusion in the diagnosis. Perforations of the subdiaphragmatic oesophagus are less common; such lesions will almost inevitably lead to peritonitis. The close relationship of symptoms to either trauma, or a history of either forced swallowing or vomiting in cases of spontaneous perforation should point to the diagnosis. Very occasionally spontaneous rupture may occur without such a recognisable event as is illustrated by the case of the young dental surgeon who sustained a rupture of the middle third of the oesophagus while watching what he thought was an amusing television programme (Triggiani and Belsey 1975).

In the presence of cervical oesophageal perforations, radiographs of chest and neck may demonstrate widening of the superior mediastinal shadow, subcutaneous emphysema of the neck and an increased distance between the trachea and the vertebral bodies (Fig 1). When the lesion is in the chest rediographs will show widening of the entire mediastinal shadow, emphysema in the mediastinum or subcutaneously in the neck together with hydrothorax and or pneumothorax. Contrast radiography is a useful method of identifying the site and extent of the perforation but the failure to demonstrate a leak does not exclude a perforation (Berry & Ochsner 1973). If clinical suspicion of oesophageal perforation exists then an attempt must be made to visualise it by oesophagoscopy ; (Carter and Hinshow 1965).

Spontaneous rupture which follows emesis is associated with forceful extrusion of gastric juice into the mediastinal pleura with resultant mediastinitis. The pleural outpouring of fluid in result to contamination added to gastric juice losses may amount to several thousand millitres in only a few hours with the development of hypovolaemic shock. (Sawyer et al 1975) Microperforations are spared most of these complications but should mediastinitis occur, drainage will be necessary.

The mechanism of rupture in spontaneous perforations is dependant on violent contraction of the abdominal muscles or an attempt to swallow forcefully with a failure of relaxation of the cricopharyngeal and cardioesophageal sphincters resulting in a sudden rise in intra-oesophageal pressure (Derbeand Mitchell 1956). In two of the cases presented in this report we believe that the mechanism of rupture must be as described above, 1or even though there was a history of indirect trauma to the chest, it is likely that the suddeness of the incident caused contraction of the two sphincters after the patient swallowed air resulting in a microperforation of the oesophagus. The lesser degree of tenderness than would have been expected for the extent of surgical emphysema indicated a spontaneous closure of the perforation and hence the lack of exploration. Oesophageal perforations should ideally be treated as emergencies by repair, but minor variations occur with the site and nature of the perforation, and the interval between rupture and time of recognition. The delay in repair of oesophageal perforations has been associated with a high mortality. Most of the published account deal with perforations of the thoracic oesophagus where mediastinitis has inevitably been present. The first successful repair of a spontaneous perforation was reported by Barrett in 1946. Subsequent reports have confirmed the

necessity for early repair and drainage of contaminated tissue. In these reports the tears varied between 4—20 cms and it is obvious that unless closure was effected there would be continued contamination of the tissues. In the case of microperforations, there is a minimal transmural breach which would initially allow the escape of air and perhaps a little fluid. Spontaneous healing rapidly occurs preventing further extravasation. Perforations of the cervical oesophagus which do not communicate with the mediastinum carry a better prognosis provided the cervical tissues are drained and oral feeding avoided. Microperforations, although rare have been described (Gardner 1949 : Nanson and Walker 1953, Naclerio 1957), and in one unusual case a second spontaneous microperforation of the cervical oesophagus occurred after an interval of 16 years. (Russell and MacDonald 1968). Although most cases of spontaneous perforation have been associated with vomiting, a lesser number have followed forced swallowing of even soft foods such as steamed fruit pudding (Davidson 1964) or a Swiss roll (Nanson and Walker 1953). In two of the cases presented in this report there was a history of minor blunt thoracic trauma which in itself is unlikely to have caused rupture of the cervical oesophagus. It seems more reasonable to assume that they are in fact cases of spontaneous microperforations due to sudden swallowing of air.

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"CENTRAL RETINAL ARTERY OCCLUSION WITH RETAINED CENTRAL VISION"

T. S. SHANMUGALINGAM, M. B. B. S. (Ceylon)
Senior House Officer,
Eye Hospital, Colombo.

Summary :

THE central retinal artery (CRA) is an end artery, occlusion of which invariably results in blindness. A rare case of CRA occlusion with retained macular function with 6/6 vision is presented and discussed.

Introduction :

The CRA, a branch of the ophthalmic artery, enters the optic nerve about one cm. proximal to the nerve head, runs in the substance of the nerve and divides into superior and inferior branches each of which inturn divides into a nasal and temporal branches. These branches supply the inner layers of the retina. The outer layers of the rods and cones and the outer nuclear layers are supplied by the choroid-cappilaries. The only anastomosis of the CRA, is with the ciliary system (circle of Zinn) and is normally of capillary dimensions. This can be of three types :

(a) Branches running forwards and anastomosing on the surface of the disc. If they are congenitally enlarged are called cilio - retinal arteries (CIRA). Pathological enlargement may be seen in occlusion of the central retinal vessels.

(b) Branches running directly into the substance of the nerve. If congenitally enlarged are called optico ciliary vessels and are seen pathologically in optic neuritis and glaucoma.

(c) Branches running into the pial sheath, called chorio — vaginal veins. Pathologically may be seen in myopia.

The percentage of population having CIRA appears to be increasing with the advancement in the techniques of detection (16·7% Lang and Barret — 1888; 16·4% Salzmann-1912; [1] 20% Mcleod et al 1960; [2] 49·5% out of 1000 consecutive cases studied by Justice and Lehmann — 1976[3]). The CIRA when present, arises from temporal edge of the disc by a hook shaped origin which is diagnostic and it always supplies the macular area. Independent macular blood supply may be received in one of the following ways :

(a) When a (CIRA) is present

(b) When there is a macular branch of the CRA which arises proximal to the narrowing at the lamina cribrosa.

(c) When there is a branch from the choroidal vessels running through the disc to supply the macula.

Case Study :

Mrs. A. M., a 33 year old female attended our clinic at the Eye Hospital, Colombo, at 9-30 a. m. on 27-7-78 with a history of sudden onset of loss of vision

of the right eye (R/E) from about 7-30 a.m. on the same day. She also gave a history of loss of field of vision and intact central vision. Prior to this she was in perfect health, except for an extraction of the left lower second pre—molar under local anaesthesia at the Dental Institute three weeks ago. She had not been using contraceptive pills. On examination she was conscious and rational tongue pink, pulse 72 / minute, regular, of good volume, and present in all four limbs equally. The carotid pulsation was normal, BP. 120/80 other systems were unremarkable. Examination of the eyes showed the visual acuity to be of 6/6 in the L/E and 6/36 in the R/E. The R/E was 6/36 with pin hole. The pupils were of normal size, the right pupil reacted sluggishly to direct light but exhibited brisk consensual light reflex. The L/E reacted normally. Fundoscopy of the R/E showed an interesting finding of cattle truck appearance or fragmentation of the blood column in the R-inferior temporal artery, which arose nearer to the superior nasal branch. The other arteries were thin and pale. The veins were of normal appearance. The macula was oedematous and a cilio-retinal artery was noticed. The left eye also showed a CIRA. Thus a diagnosis of CRA occlusion with partial occlusion of the R/inferior temporal artery in a patient with CIRA was made.

The patient refused admission and hence investigations and treatment were done at the clinic itself. Digital ocular massage was given for about fifteen minutes and the following investigations were ordered.

1. WBC/DC.....8600; N-58; L-36; E-06;
2. ESR.....1 st hr. 06mn; 2nd hr. 15 mm;
3. Urine sugar..... nil
4. Blood cholesterol ..... 245 mg%.

She was on the following treatment:

a—Prednisolone ... ...
10 mgs t. i. d.

b—Acetazolamide (Diamox)
500 mgs stat and t. i. d.

c—Cinnarizine (Stugeron).....
25 mgs. t. i. d.

On 29-7-78 : She had a visual acuity of 6/6 on both right and the left eyes. She also had a tunnel vision on the R/side. Fundoscopy of the R/E showed a pale retina, thinned out arteries and a normal coloured retina between the macula and the disc, while the rest of the retina was pale. The cattle truck had disappeared, and also the macular oedema. The CIRA was reddish and seen very clearly. The field was plotted, and it showed a central field which corresponded with the normal area of retina (fig: 1 and 2). The same treatment was continued.

On 11-9-78 : The visual acuity (V/A) was 6/6 on both right and left sides. Fundoscopy showed the retina to be of normal colour indistinguishable from the CIRA area, and the vessels appeared normal. But the disc was definitely paler than the L/Side and the CIRA with a hook shaped origin was seen very clearly against the pale background. A number of new vessels were seen at the nasal edge of the disc-pathloogical enlargement of the cilio retinal anastomosis. There were streaks of whitish scarred areas along the inferior temporal artery, about two disc diameters away from the disc, margin. The central field plotted remained almost the same.

On 27-9-78: The central field remained the same and V/A was 6/6 in both eyes.

Discussion:

Acute occlusion occurs from four basic causes.

1. Vaso—obliteration
2. Embolus
3. Raised extra vascular pressure
4. Spasm

The patients usually present with a history of sudden onset of loss of vision. This may occur when the patient suffers a hypo - volaemic state or be the first symptom of a major thrombotic phenomena. The V/A is usually reduced to hand movement. Within 15 minutes of a total occlusion swelling of the retinal neurones occurs and the typical cloudy swelling develops within 2 to 3 hours. Irreversible changes in the ganglion cells have been reported to occur as early as 3½ hrs. after the occlusion (Krell - 1968[1]) and cell degeneration occurs after 16 hrs. Clinically, the larger arteries are reduced to threads, the smaller arteries disappear. The veins are little affected. Blood columns in the vessels usually the veins may show cattle truck appearance, with the variation of the intra — ocular pressure. Such appearance in the arteries as in our patient suggests partial occlusion of the arteries. The retina looses its transparency and becomes milky white In Complete occlusion, after sometimes the macula appears as a "cherry red spot", due to the choroidal reflex. Later the disc becomes pale and the picture is complete. In our patient, the macular oedema explains the 6/36 vision. When the oedema subsided the V. A. improved to 6/6. It could be observed that the field plotted correspond to the area of retina spared (Fig: 1 and 2).

Investigations:

The investigations should be aimed at finding the cause of the arterial obstruction. In our patient from the basic investigations available, the cause of the occlusion was thought to be arterial spasm. True arterial spasm as a cause of acute occlusion is still debated. Associated conditions are migraine, quinine overdosage, and trauma to the orbital skeleton. Propagated spasm along orbital arteries may also occur, as a rare complication of dental anaesthesia of the upper jaw. In these cases abnormal arterial supply of the face and the orbit are suspected. (Ffytche. T. J. — 1974 [4])

Treatment:

The purpose is to restore the retinal circulation, which is achieved by increasing the retinal perfusion, dislodging the embolus, and or overcoming the spasm if present. The perfusion pressure of the retinal cappillaries is the difference between the arterial pressure and the intra—ocular pressure (Melcod et al 1960[2]) A variety of procedures like laying the patient flat, cooling the limbs, systemic drugs "rebreathing" retro - bulbar injections of vasodilater drugs like tolazoline, papaverine, procaine, Acetyl choline, aminophylline, and nicotinic acid were tried and/or suggested. Surgically parcentesis of the anterior chamber was also recommended. Ffytche. T. J. (1974[5]) suggests the following regime in all cases of CRA occlusion within the first 48 hours.

a. lay the patient flat;
b. I. V. Acetazolamide (Diamox) 500 mgs stat.
c. apply ocular massage for 30 mts.

Diamox 500 mgs and the ocular massage reduces the I. O. P. from 15 mmHg to

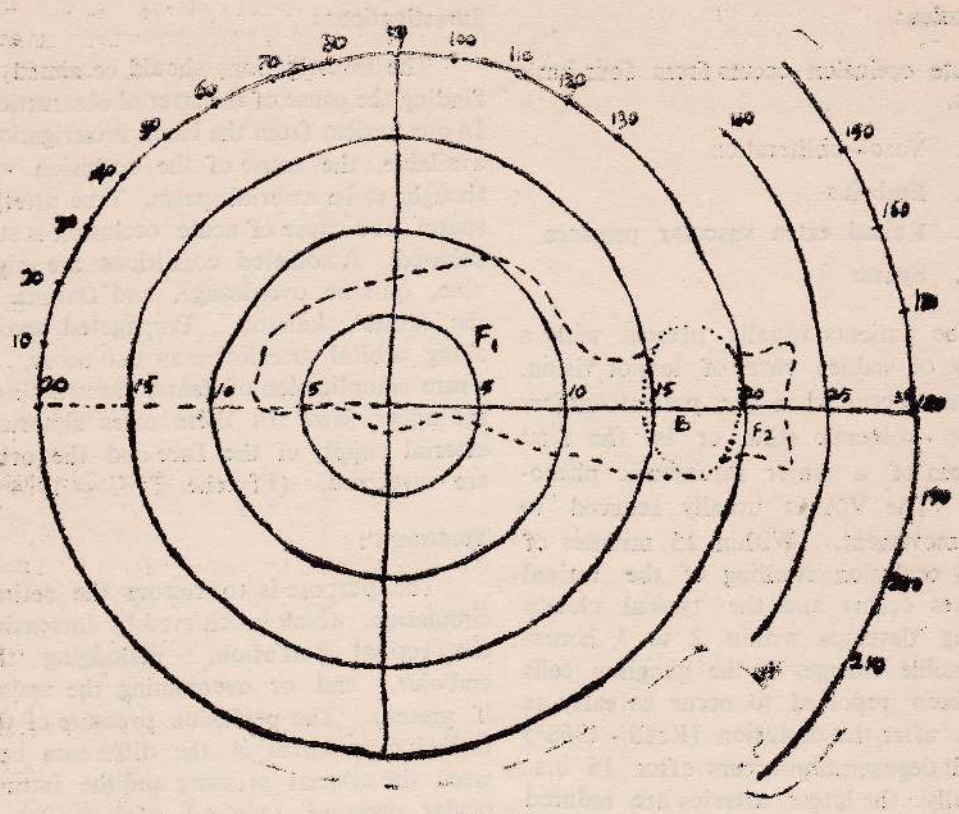

FIG. 1

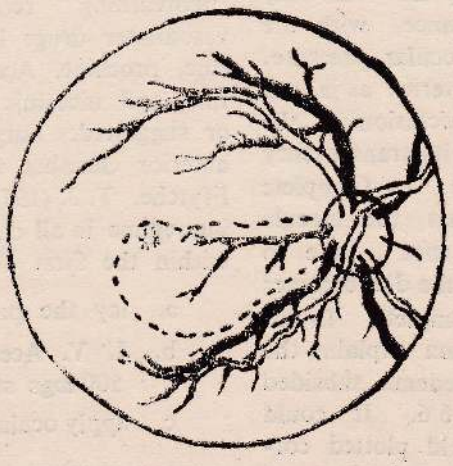

FIG. 2

5 mmHg. In ocular massage alone an average increase of 80% of retinal volume flow was recorded. Firm massage with compression of the globe for upto 15 seconds at time is suggested by Ffytche.

In our patient a variety of reasons could explain the preservation of central vision.

1. The presence of the Cilio—retinal artery
2. Early medical attention (the patient managed to come within two hours because she happened to be a relative of an attendent)
3. Timely ocular massage (the only procedure that we could do)
4. Partial occlusion of one branch, as shown by the "cattle truck"
5. Drugs; Diamox 500 mgs. oral stat Injn, is not available with us) and Stugeron and prednisolone.

In conclusion I would like to quote Professor W. S. Foulds, who said "It is important not to mislead oneself into thinking that therapy being given was having a beneficial effect when infact, spontaneous improvement was occurring" (Foulds W. S. 1974[6]),

Acknowledgement :

I would like to thank Dr. R. E. Selvarajah, my teacher, for his encouragement and advice, Dr. S. Kugathasan for his valuable assistance and the M. S. Eye Hospital, for granting permission to publish this case.

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NITRATES IN HEART DISEASE — A SHORT REVIEW

D. CHANMUGAM, M. D., F. R. C. P. (London)
Department of Medicine,
University of Ceylon (Colombo).

IT is now just over 100 years since nitrites were first used and it is remarkable how it has stood the test of time notwithstanding doubt as to the details of its mechanism of action. It is the purpose of this short review to discuss briefly the common nitrates and their uses.

Haemodynamic Effects. All pharmacological properties are shared by the nitrite ion and large number of organic nitrate esters. The basic action is to relax smooth muscle. This action which is not specific affects all smooth muscle irrespective of innervation. The result is complex depending on reflex effects of arterial and venous vasodilation producing variable falls in systemic and pulmonary capillary wedge pressure and increase in the heart rate. Despite its reputation as a coronary artery dilator most studies show that even when injected directly into sclerotic coronary arteries nitrites do not increase coronary blood flow. A redistribution of blood from well perfused healthy myocardium to poorly perfused heart muscle has been demonstrated, The drug also favourably alters the imbalance between myocardial oxygen supply and demand. The final effect will depend on multiple interacting variables eg. the dose of nitrate, its metabolism, left ventricular systolic and diastolic wall tension, heart rate, contractility of the myocardium, severity of coronary artery narrowing and the extent of coronary artery dilatation produced by local hypoxia.

Nitroglycerin (Trinitrin, Glyceryl trinitrate). In spite of the complexity of its action about 75% of patients with angina pectoris benefit from Nitroglocerin which is the same compound used in explosives. The drug comes in 0·15 mg, 0·3 mg, 0·4 mg. or 0·6 mg tablets. The tablets which deteriorate rapidly on exposure to light must be stored in dark brown glass (not plastic) tightly sealed containers preferably in a refrigerator and replaced every 3—6 months. The patient must carry no more than a weeks supply on him. The tablet must be taken sublingually and not orally. For acute angina the patient must cease whatever activity that brought on the pain, assume the sitting position and immediately dissolve one tablet, to start with of 0·15 mg. strength, under his tongue. The duration of action of the drug varies with the strength of the tablet from 15 minutes to a few hours. The smaller dose may be replaced with a larger tablet if there is no relief in 1-2 minutes. If the drug is not active there will be no burning sensation under the tongue. The standing or supine position should be initially avoided as the former may result in syncope and the latter an increased venous return. Transient headaches are common and if there is excessive dizzinsss the patient should lie down with his feet propped up 1 foot and

reduce subsequent doses. If there is no relief of pain the dose should be repeated every 3 minutes for a further 3 consecutive doses. If there is still no relief or if the pattern of angina is different from the usual the patient should seek admission to the nearest hospital immediately. A patient may use up to 60 tablets per day. The best results are obtained in angina pectoris due to coronary sclerosis. In idiopathic hypertrophic subaortic stenosis (hypertrophic outflow obstructive cardiomyopathy) angina should be treated with propanolol. Nitroglycerin is contraindicated in glaucoma and increased intracranial pressure and should not be used in angina due to anaemia, aortic stenosis, hypotension, tachyarrythmia and routinely in acute myocardial infarction. In a patient with predictable angina, nitroglycerin can be used prophylactically; to be taken a few minutes before the usual activity which the patient knows will bring on angina.

Apart from its use in angina nitroglycerin has been used in the treatment of chronic congestive cardiac failure and even acute myocardial infarction. In the former the arteriolar dilating property of nitroglycerin is thought to be the effective factor. Nitroglycerin has been shown to limit infarction size in animals and man but their use in these situations should be used with great caution and only in units where sophisticated haemodynamic monitoring is available. More wide use of coronary arteriography has shown that infarction has been seen to occur in young patients with patent coronary arteries and also as a result of arterial spasm specially in patients with Prinzmetal's variant angina. Nitroglycerin may have a role to play in the management of these cases.

The long acting nirates.

Preparations with a longer action have an obvious appeal. The drugs commonly available are isosorbide dinitrate (Oral and sublingual) erythrityl tetranitrate (oral and sublingual) and pentaerythrityl tetranitrate (oral only). The non metabolized nitrate is the only pharmacologically active part and as no radioactive material could be found at any time in the plasma of human volunteers after ingesting radioactively labelled nitrates due to the very rapid hepatic metabolism many doubted the usefullness of nitrate given orally. More recent work has shown that during the drug's rapid transit in the blood to the liver some of the drug is immediately taken up by the cells lining the blood vessels and it is this rather than the presence of the drug in the blood stream which is responsible for the pharmacological action of nitrates.

The drug for which most reliable haemodynamic data are available is isosorbide nitrate and these indicate the action of this drug is similar to that of sublingual nitroglycerin but later in onset and longer in duration. Oral therapy (5 mg, 10 mg, 20 mg tablets) in 5-30 mg doses four times a day, up to a maximum of 360 mg. / day provide effective anti-angina prophylaxis and can even improve ST segment changes. The effect can be seen in 15 mts and is dose dependent. In an individual patient the rate of hepatic degradation must be kept in mind. Two other forms of isosorbide are available. They are the sublingual form (2·5 mg; 5 mg tabs) and the chewable (5 mg tabs,). The sublingual form has a faster onset of action than the oral form (5-10 mts) and a more certain duration of action (1-2 hours). It is given as 2·5 mg-10 mg

every 2-3 hours. The chewable form has an even more rapid onset (5 mts) but the duration varies from patient to patient. It is usually given as 5 gm chewed every 2-4 hours.

With regard to the other two drugs pentaerythritol and erythrityl tetranitrate data regarding speed of onset and duration are not so reliable and are given below in Table 1.

Nitroglycerin ointment available for two decades is gaining popularity now. The haemodynamic effects of the drug absorbed through the skin are those of the sublingual drug expect that the onset is delayed from 15-60 mts. and the action lasts 3-6 hours. The drug is available as 2% ointment and is thinly applied over a skin surface usually the chest and then covered with a plastic wrap. Typical doses are ½-2 inches square every 4 hours, This form has been used successfully in angina refractory to other long acting nitrates, unstable and nocturnal angina.

All these long acting nitrates have been also used in patients with refractory cardiac failure but the selection of cases which will respond demands haemodynamic monitoring. The smallest dose should be initially used and stepped up slowly to tolerance or desired side effect. If undesirable side effect follow, they may decrease with continued usage or another preparation may be tried. If nitrates are being withdrawn they should be withdrawn slowly as abrupt cessation has been known to precipitate infarction.

This brief review attempts to indicate why and how nitrates have continued to be useful in the treatment of ischaemic heart disease.

Drug	Tablets	Conventional dose	Onset	Duration
Erythrityl	Oral 5, 10, 15 mg tabs sublingual 10 mg tabs chewable 10 mg tabs	5—30 mg three times a day	unknown likely delayed	long (probably same as Isorsorbide).
Pentaerythritol Tetranitrate	Oral 10mg, 20 mg tabs	10—30 mg 3—4 times/day	unknown likely delayed	long
	oral 30, 40mg tabs	1 tab orally every 12 hours on an empty stomach	delayed	long
	oral 80 mg timed disintegrating caps	1 cap orally every 12 hours on an empty stomach	delayed	long
	oral 80 mg sustained action tabs	1 tab orally every 12 hours on an empty stomach	delayed	long

Table 1. There are many commercial preparations where the above active ingredients are combined with other drugs usually sedatives.

TRANSIENT GLOBAL AMNESIA — A CASE REPORT

T. GANESWARAN, M. B. B. S., M. R. C. Psych. (Eng.), D. P. M.
Psychiatrist,
General Hospital, Jaffna.

S. HIMAYAKANTHAN, M. B., B. S.
House Physician,
General Hospital, Jaffna.

Department of Psychiatry, Jaffna General Hospital

Introduction :

THE syndrome transient global amnesia was first described by Fisher and Adams in 1958. This clinical syndrome occurs in middleaged or elderly people with dramatic suddenness, to clear spontaneously within a few hours of its onset. The characteristic features are a memory loss for recent events with an inablity to register new impressions. The patients retain their personal identity and show no abnormality in behaviour, apart from anxiety, and no evidence of impaired perception (Brain 1969). The retrograde (amnesia which occurs shrinks rapidly and the resulting) amnesia is entirely that of attack itself. Though more than a hundred cases have so far been described the pathophysiological mechanisms are still obscure. Focal epileptogenic discharge and transient impairment of the vascular supply to vital memory centres has already been implicated in its causation (Stein 1975). A case seen in Sri Lanka is reported.

Case Report :

A 52 year old female was admitted to General Hospital, Jaffna on 21-9-78 with a history of giddiness. She has lost her memory for all events on the day prior to admission. Her daughter who accompanied the patient said that her mother was quite well until 8 a. m. on the previous day, when she set out to visit her sister, a nun teaching in a Convent. On that morning she had done all her household work and did not display an anxiety or worry nor did she complain of headache or giddiness. After she left home she had borrowed some money from one of her friends and walked about half a mile to the bus stop. She travelled by bus after paying the correct fare but left her umbrella in the bus when she reached the school where her sister worked. The journey in the overcrowded bus was a strenuous one lasting 3 hours. As soon as she reached the school around 11 a. m., she complained of giddiness. She was seen by a doctor who gave her injection of Vitamin B Complex. She

continued to complain of giddiness and therefore on the same day late in the afternoon she was taken home by car and asked to rest in bed. While lying in bed later that evening she suddenly began to wonder as to why she was wearing a dress which she normally wears when she leaves home to visit her friends or relations. She had complete loss of memory for all events for a period of about 8 hours since she set out on her journey that morning. Her sister and daughter who were questioned said that the patient was aware of her personal identity and there was no suggestion of automatism. She was a widow with 8 children. Her husband died 4 years ago. There was no previous history of hypertension, diabetes mellitus, cerebrovascular disease or psychiatric disorder. Environmental stresses were non contributory. On admission pulse was 78/minute and regular. B. P. 110/80 mm of Hg. Clinical examination of heart, lungs, abdomen and central nervous system revealed no abnormality. ESR was 15 mm in the first hour. Hb was 11·3 gm%. Fasting blood sugar was 105mg%. ECG, EEG, X-rays of Skull, Spine and chest were all normal. Within 24 hours of admission she was free of her attack of giddiness, but the amnesia for a period of about 8 hours from 8 a. m. on 20-9-78 persisted and was permanent. At subsequent follow-up symptoms did not recur.

Discussion :

Either due to the non recurring nature of the illness, or due to the unawareness of the patient of his illness at the time of the attack the clinical syndrome of transient global amnesia may be missed. This is probably the first case to be reported in Sri Lanka. Patients suffering from disturbances of memory are often referred to the psychiatrist when emotional factors are suspected in their causation.

Memories are said to have two important subjective properties namely, "time ordering" and personal identity and personal identification (Suarez 1976). All events are perceived in a time dimension, that is, some events are percieved as having happened before other events. Further, an individual can distinguish past events which are part of his immediate personal experience from past experiences and events conveyed to him from secondary sources (Suarez 1976). Loss of memory may be for all events of a certain period of a patient's life—temporal amnesia, in contrast to categorical amnesia (Nielson 1955), where a category of events are banished from memory irrespective of its place in the time dimension, Amnesia shown by patient described in this report is of the former variety. Emotional factors were non contributory.

In common with previous cases where variable precipitating factors were reported (Fisher and Adams—1958) the attack appears to have been precipitated by moderate or severe physical effort namely a strenuous jonrney by bus and a long walk. The etiological factors and triggering mechanisms have been discussed by stein in 1975. Cerebral ischaemia leading to infarction and epilepsy or transient arterial spasm without cellular changes have been implicated tn its causation. Obstruction of the cerebral blood flow to the memory centres due to embolus, arteriosclerosis or imflammatory vasculitis, hypertension hypercoagulable states have all been mentioned as possible causes (Stein 1975). The Hippocampal region and the medial dorsal nucleii of the thalamus appear to

be the sites of transient impairment of vascular supply (Stein 1975). The patient described probably suffered from such episode.

Acknowledgement :

Authors thank Dr. S. Sivakumaran, Physician G. H. Jaffna and the Medical Superintendent G. H. Jaffna, for granting permission to publish this case.

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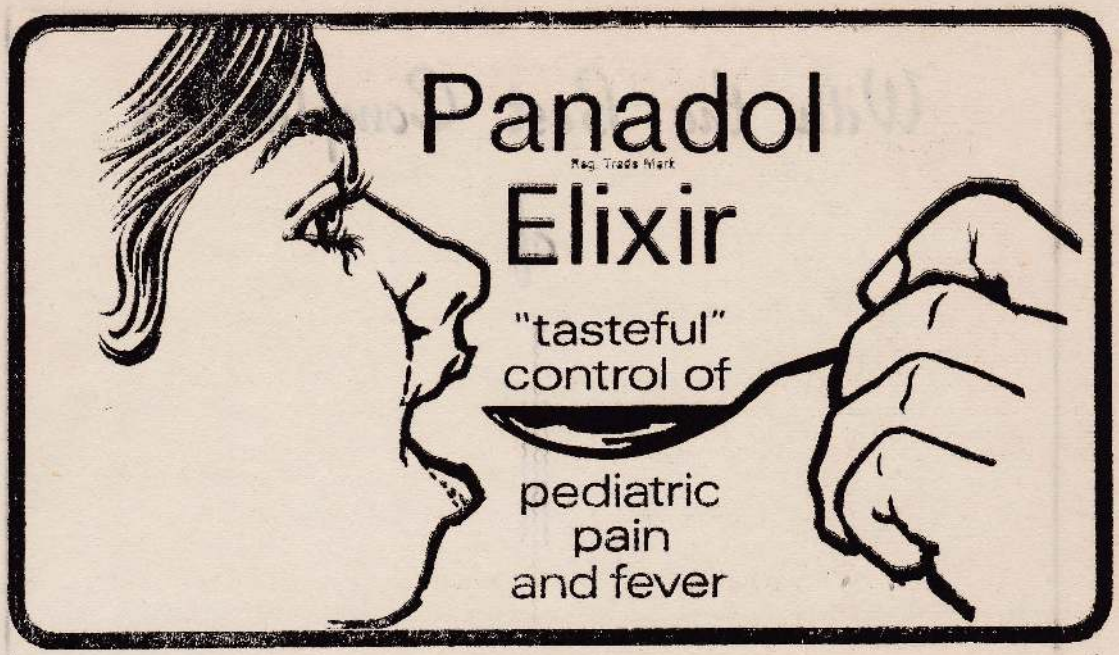
Medical News

Jaffna Medical School. On September 1978 the Minister of Education, Mr. Nissanka Wijeratne delivered the Inaugural Address at the ceremony concerned with the establishment of a new medical school, in Jaffna. The ceremonies were held in the hall of the Medical Faculty of The Jaffna Campus at Kaithady where the pre-clinical departemnts of Anatomy Physiology and Biochemistry are situated. Seventyfive students were admitted to the new medical school, and when they pass the second M. B. will continue their clinical studies at the Jaffna General Hospital. Together with the Galle Medical School the two schools increase the number of institutions of medical education to four in this country.

Annual Clinicical Meeting of the Jaffna Medical Association : A two day meeting will be held on December 8th and 9th, 1979 at Jaffna. Members of the medical profession who are interested in reading short papers of 15 minutes duration are invited to communicate with the Organising Secretary Dr. R. Devarajan, Library, General Hospital, Jaffna before 15th October 1979. Details may be obtained from the Organising Secretary.



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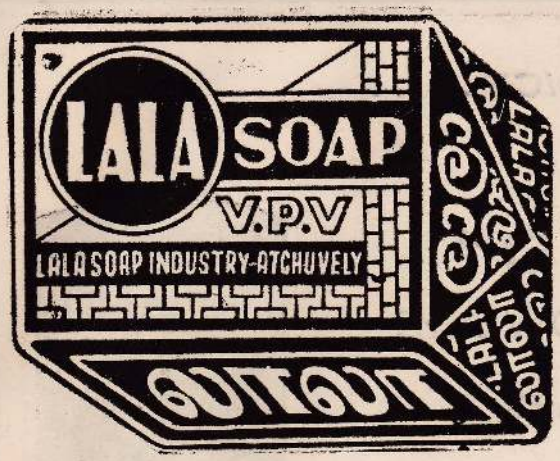

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for the Editors, Medical Journal, General Hospital, Jaffna, 27 / 79