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VOL. IV

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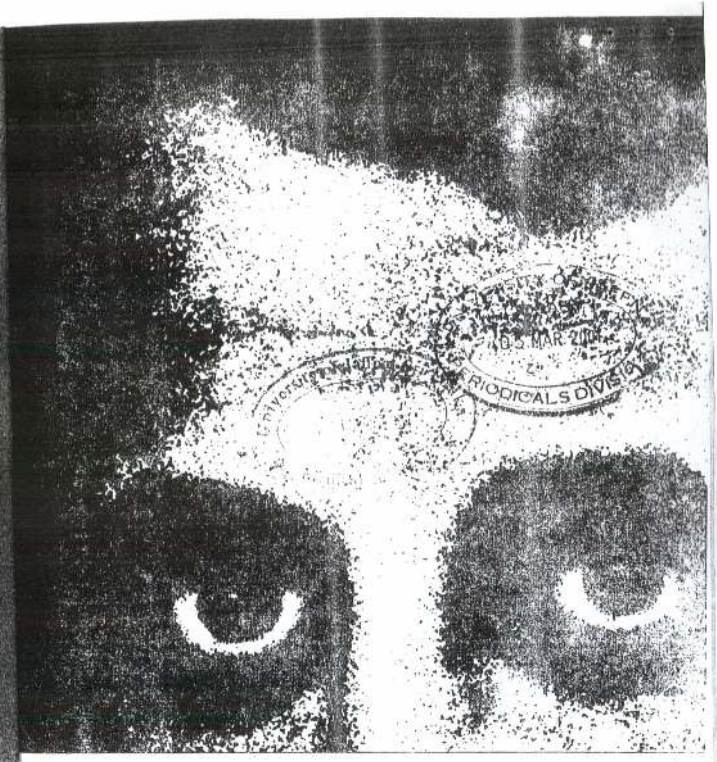

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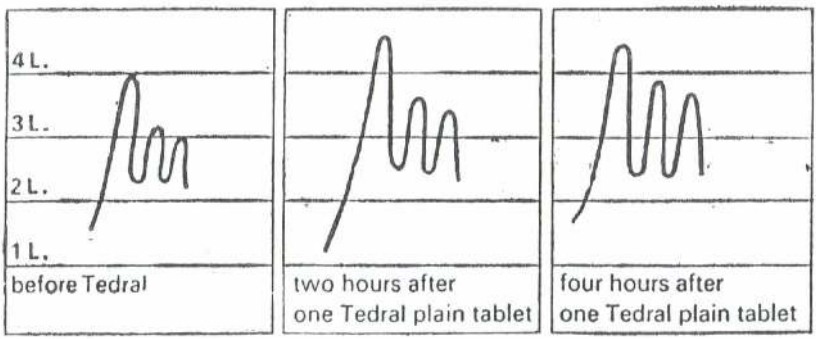

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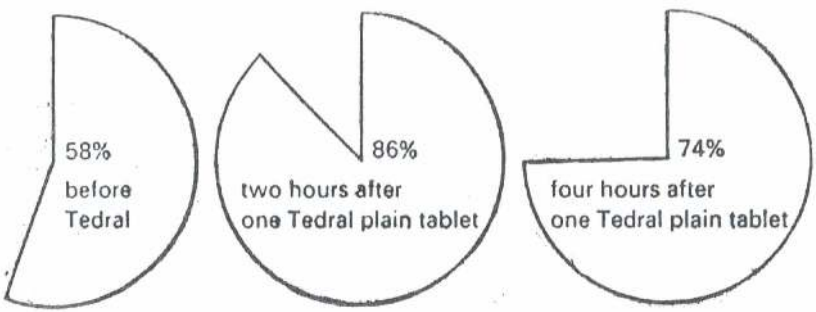

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VOL. IV **MAY / AUGUST, 1967**

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EDITORIAL NOTICE

Original articles and papers of interest to the Medical Profession are invited.

Scripts submitted for publication in the journal should be sent to *Dr. A. Gabriel F.R.C.S., Hony. Editor, General Hospital, Jaffna.* Papers should be prepared in typescript, double-spaced with 1½" margins, and submitted in final form so as to reduce the necessity for later corrections. The author (s) should retain a carbon copy for reference.

Authors should give their names, professional qualifications and their present post or place in which the work was carried out optionally.

The title should be precise and, if possible, brief. If the title given is unsuitably long to head subsequent pages, a short title should be indicated for this purpose.

Tables and Illustrations should fit within the page size of the journal $9 \times 7\frac{1}{2}$" and be kept separate from the text. Glossy photographs should be supplied unmounted and drawings should be finished suitably for reproduction. Descriptive legends should be typewritten and annexed to the appropriate figures which should be serially labelled.

References in the text should bear the author's surname and the year of publication both within parentheses, *e.g.* (Smith 1960).

When the author's name is part of a text sentence, the form to be used is exemplified thus: ..... "as Fernando (1 45) observed,....."

If the reference is to joint authorship, all their names should be indicated on first appearance in the text thus:

Fernando, Perera and Pieris, (1962).....

In subsequent references in the text, the form should be Singham *et al*, (1960).

References should all be listed at the end of the text in alphabetical order and not numbered. The Hansard system should be followed here too, with modification as follows:

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Editorial

JOURNAL OF THE JAFFNA CLINICAL SOCIETY

WE recently read in the press that ten thousand more nurses are to be recruited shortly to ease the present shortage of this category of staff in our medical services.

This news leads us to think about patient care in our own hospital in general.

In the first place, we must be clear in our own minds about what we mean by "Patient Care".

This consists of 1. Medical care i. e. Examination, Diagnosis and Treatment of the patient's disease. 2. Patient Comfort which is largely a province of the nursing profession and other paramedical personnel.

This is certainly an arbitrary division made only for the purpose of clearer thinking. It is very well known that there is considerable interplay between these two aspects and the people concerned with one aspect should pay adequate attention to the other, for the good of the patient.

We need not enlarge upon medical care because we think we have progressed fairly rapidly in this direction by turning out more doctors and specialists and providing greatly improved ancillary services. e. g. We see that more peripheral units are being staffed with at least one doctor which was not so a few years ago.

Coming to Patient Comfort, we must define it. Patient Comfort is the attention given to the mental and physical requirements of a patient, apart from the mere administration of drugs or the performance of surgical procedures upon a patient.

How many of our nurses and doctors sit down and talk to a patient about his work or his family? This is indeed rare. It is rare because we have no time for this sort of thing or no mind for it. No time because of the large numbers of patients in the wards, no mind for it because the large majority of us are guilty of treating what should be a vocation as a job being done to earn a living, doing no more than what the book of rules require.

We have rarely seen in our experience a doctor or nurse asking a patient "Are you comfortable?" "Can I get you anything?" unless of course the patient happens to be a relation or a known party.

This may indeed be due to lack of time in a busy overcrowded ward but it is in a large measure due to our narrow mindedness and selfishness as a society and due to poor basic medical and nursing training. Not poor basic training in the skilful giving of injections, cold sponging or assisting or performing surgical procedures but in social relationships. It is futile to talk about the lamp of

Nightingale and the Oath of Hippocrates if one cannot render expired air resuscitation to a person of the opposite sex who has been dragged out of a well, drowned and apnoeic, just because society will frown upon the procedure.

The answer to the problem of poor patient care in Ceylon as a whole and Jaffna in particular is not only to increase the number of medical and paramedical personnel but also to reorientate our mental and social attitudes to each other as medical men and women and towards all those who will come under our care as patients.

Let us start this change of heart and attitude right here in our own medical and nursing schools and let society follow our lead. We should not be so bound by social rules and regulations or such a high sense of our position that we should find it difficult to spare a moment to ask a poor sick patient "How are you today?"

*PULMONARY THROMBOEMBOLISM — DIAGNOSTIC PROBLEMS

By

S. ANANDARAJA, M. D. (Cey.) M.R.C.P. (Lond.) M.R.C.P. (Edin)

Physician, General Hospital, Jaffna.

I have had the unpleasant experience of being present at post-mortem examinations of patients who had been under my care, who had apparently died suddenly in whom no obvious cause was detected during life, when pulmonary embolism was revealed as the fatal factor. In my defence I can only state that my colleagues also missed the diagonosis and I quote the figures of Smith who in 1958 estimated that the diagnosis of pulmonary embolism was missed in 70% of cases during life.

On glancing through the case notes of my patients who had ended on the postmortem slab, it was quite apparent that there were always warning signs of the impending catastrophe which during life did not appear serious enough to warrent a more thorough inquiry as to their significance. Evidence such as persistant tachycardia, tachypnoea, attacks of breathlessness, fleeting chest pain and anxiety were ignored when no causative factor was apparent.

It is with these diagnostic problems of pulmonary thrombo-embolism (PTE) that I shall deal in this article.

Pulmonary embolism may result from a blood clot fat, air, foreign body or fragments of a malignant tumour being impacted in the pulmonary blood vessels. The source of the embolus may arise in the heart or the systemic veins.

Clots arise from the heart in auricular fibrillation, congestive cardiac failure, myocardial failure and myocarditis.

In the case of myocardial infarction, septal infarcts are usually to blame.

Thrombosis occuring in the systemic veins have various aetiological factors which may be briefly summarised as follows :

I. LOCAL VENOUS INJURY
   - Inflammatory
   - Traumatic
   - Chemical
   - Infiltrative-malignant tumours

II. SLOWING OF THE BLOOD FLOW
   - Congestive cardiac failure
   - Local obstruction

III. INCREASED TENDENCY TO CLOTTING
   - Post-Operative-prolonged operations-Traumatic pelvic, cardiac lower abdominal especially if cardiac failure is present
   - puerperium
   - polycythaemia
   - haemoconcentration
   - enteric fevers e.g. typhoid
   - Tissue breakdown e. g. Malignancy

and following myocardial Infarction

In Britain, pulmonary thrombo embolism has been increasingly recognized as one of the comonest acute chest conditions in hospital practice. Short in 1952, estimated that the incidence of pulmonary embolism was higher than that of lobar pneumonia. The condition is unfortunately too often missed in its early stages. It is still a major cause of death in a distressingly large proportion of hospital admissions. The mortality rate can be reduced by treatment, but

* Based on a paper read before the Jaffna Clinical Society in November, 1965.

this demands early diagnosis. The incidence of PTE in Ceylon appears to be less than in Britain. It does however occur with sufficient frequency to warrant a serious consideration of this problem.

Goodwin in 1963, described two main clinical groups (1) Obvious source of embolism was present and there was obstruction of the large arteries and the condition was characterised by pleuritic pain and haemoptysis (2) Occlusion of the smaller arterioles and microarteries without an obvious source.

The clinical features here resemble those of idiopathic pulmonary hypertension. There is however considerable overlap as patients in group (2) May develop occlusion of the larger arteries and present with the signs and symptoms of the group (1) patients.

The microembolisation occuring in the second group is said to be silent until evidence of pulmonary hypertension developed with the onset of exertional dyspnoea, syncope and chest pain. In this paper, I shall mainly confine myself to the problems attenendent on the early diagnosis of the first type. By defiaition, the second type is silent until the late stages when, with the onset of pulmonary hypertension, it is clinically indistinguisable from idiopathic pulmonary hypertension unless it is associated with features which occur when the large arteries are occluded.

The clinical presentation of PTE is often confusing and confident diagnosis is difficult. The lack of pulmonary embolism consciousness is also a contributory factor to delayed diagnosis. The difficulties of diagnosis are due to several features. There is a group of patients in whom there is a tendency for one clinical feature to so dominate the overall picture that other features are obscured, resulting in a confident yet mistaken diagnosis. For example the outstanding feature in a particular patient may be a persistent tachycardia with the result that a diag of cardiac arrythmia is made or eve thyrotoxicosis.

Another group presents difficulties account of the initial transient natur the early symptomatology with the abs of physical signs and positive inv gational result. Further, there are leading silent intervals in which the pat is apparently well and symptom free betw intervals of episodes of embolisation. another group, diagnostic difficulties a when the presenting symptomatology extrathoracic and when there is no obv source of embolism and no aetiological fac for venous thrombosis. It is indeed fortunate that the classical features of P such as cough, haemoptysis, pain and tac pnoea do not appear in combination i sufficient persentage of patients, early in onset to be of diagnostic value. It is fortunate that there are no diagnostic la ratory criteria which are of value in early stages. Electrocardiography and X- examination may prove of little val Diagnosis rests on a good history and ca ful physical examination.

There are certain important features wh have been neglected in the usual textbo descriptions. I believe that in certain ca these features could be of crucial diagnos value.

In order to bring these features of t diagnostic difficulties of PTE into relief, am here presenting an analysis of 100 ca of PTE.

This study is based on 100 patien admitted to Whipps Cross Hospital, Lo don, betwee 1957 and August, 1963. I ha excluded those patients who have develop PTE after surgery, as in these patients t diagnosis presents no problem.

This series does not include all patien admitted to this hospital with pulmona embolism, as only a proportion of the ca notes of these patienss were available.

Sex incidence: Thecondition was equally distributed between the sexes—49 males and 51 females. Textbooks usually give a higher incidence in females.

Seasonal Incidence: There appears to be no seasonal variation of any significance in this series. It has however been claimed that a higher incidence occurs in spring and autum.

Age: The youngest in this series was 20 years and the oldest 65 years of age.

Predisposing conditions in order of descending frequency are as follows:

CARDIAC DISEASE

25 patients had cardiac involvement of some severity; 12 patients had rheumatic heart disease. It is well established that the incidence of PTE in rheumatic heart disease is high. (Ball, 1956; Goodwin, 1963). The source of the embolus is the deep veins of the legs in patients in cardiac failure. Sluggish blood circulation combined with imobilisation are factors of importance.

Atrial fibrillation is often present and here the thrombus may originate in the right atrium. Though the left side of the heart is mainly damaged in mitral stenosis, post mortem examination has shown that in atrial fibrillation 90% of the cases have thrombi in the right atrium as well. Atrial fibrillation would have been present for about one year. Thrombosis in the pulmonary arteries, may occur in severe valvular disease.

There were four patients with hypertensive heart disease. Eight patients had ischaemic heart disease.

It is worth remembering that patients with myocardial infarction may develop PTE, without congestive cardiac failure, as there may be mural thrombi. Furthermore the combination of bed-rest and increased coagulability of the blood following on cardiac infarction predisposes to deep vein thrombosis.

CHRONIC BRONCHITIS

There were 14 cases with a history of chronic bronchitis. In these patients, local changes occuring in the pulmonary vessels may play a significant part, especially when pulmonary hypertension is present. Immobility in bed-ridden patients probably plays its part. The role of secondary polycythaemia, often associated with chronic pulmonary disease and increased viscosity of the blood may be significant. There is however some dispute and conflicting evidence as to the incidence and importance of polcythaemia in chronic bronchitis.

I am inclined to believe that the role of chronic bronchitis in the pathogenesis of PTE is of much greater importance than the figures in this series indicates. The clinical picture of recurrent pulmonary thromboembolism may so simulate an acute exacerbation of chronic bronchitis that the diagnosis of PTE is never entertained. The greater majority of these patients eventually succumb. Post mortem examinations are rarely made on known chronic bronchitics thought to have died of respiratory failure.

NEOPLASMS

Malignant tumours were present in eleven cases. These occured in the bronchus, colon, uterus, prostate, caecum, stomach and the bladder. There was no predominance of any particular site. These patients had no other significant predisposing causes to PTE. One of these patients had thrombophlebitis migrans. There was no patient in this series with carcinoma of the pancreas, which is the site conventionally associated with thrombophlebitis migrans.

VARICOSE VEINS AND SUPERFICIAL THROMBOPHLEBITIS

There were 12 patients, all of whom were females. In the past it used to be taught that superficial thrombophlebitis was seldom

responsible for PTE. It is now however being increasingly recognised that it is not so and futhermore patients with superficial thrombophlebitis are quite likely to have thrombosis of their deep veins as well.

NEUROLOGICAL DISEASE

Neurological diseases predisposed to PTE in 11 patients. Six patients had hemiplegia following cerebrovascular accidents. Byrne and O'Neil (1952) reported an increased incidence of PTE in hemiplegies. In this series, three had parkinsonism and one had syringomyelia. Ten of these patients were not bedridden at the time of the thrombotic episode.

A fact worth mentioning is that deep vein thrombosis occured on the paralysed side in the hemiplegics.

TRAUMA

Trauma was responsible for deep vein thrombosis in four patients.

PREGNANCY

Two patients were pregnant at the onset of PTE in this series. There were no other predisposing causes. This is surprising as one would not expect venous thrombosis and PTE in a state of hyperkinetic circulation. For example PTE is hardly ever encountered postoperatively in thyrotoxic patients who have had throidectomy.

DEHYDRATION

This was responsible for deep vein thrombosis followed by PTE in one patient who was in the terminal stages of anorexia nervosa.

CORTICOSTEROID THERAPY

Cortico steroid therapy of over one year's duration was probably responsible for deep vein thrombosis and PTE in a patient with a skin disease.

No obvious predisposing causes were present in by far the largest group of patients. There were 27 males and 8 fe in this group which from a diagnostic of view is of great importance. In patients, especially if clinical evidenc deep vein thrombosis is absent, the diagnosis of PTE is likely to be missed. is worthwhile stressing this feature of One should be prepared to make the nosis on available clinical evidence, and to exclude it purely due to the lack of obv sources for the emboli. Obesity has blamed for most ailments. In this se there appeared to be no predisposition the development of PTE in the obese. 15 patients in this series were conside to be overweight.

Clinical Features : The classical pres tation of PTE with a combination of ch pain, cough, haemoptysis, tachypnoea tachycardia is not so common as is imagi in the early stages. In the present ser this combination occured in less than third of the patients in the early stag These features, however, are comm enough in the latter stages with recurr PTE or with massive embolism. Howev death not infrequently intervenes bef the full clinical picture unfolds itself. O has to be prepared, herefore, to diagn the condition on certain significant, scanty, features in the early stages.

Signs and Symptoms : Tachypnoea a difficulty in breathing is of cardinal impor ance. This was recorded in 81 out of 1 patients. In 8 of these patients this was t sole presenting feature. The respirato rate is between 30 to 40 per minute. T breathing is shallow. This feature may g unnoticed in patients with chronic bron chitis or heart disease. Close questioni may however reveal that these episodes ma have occured in the past and are not nece sarily related to effort or posture and already present, may become more sever The chronic bronchitic usually gets sho of breath on exertion or if in respirator

failure has it continuously. In a patient without any apparent cause for tachypnoea this feature is of the greatest importance.

When tachypnoea occurs with other features it helps to confirm the diagnosis. Marshall and Allison produced experimental proof to show that there need be no increase in central venous pressure to account for tachypnoea. Weidner and Light have shown that complete dennervation of the lung will decrease the tachypnoea showing that the vagal reflex was the main underlying mechanism.

Chest Pain: Pleuritic pain occured in 64 patients and was often associated with tachypnoea. A point of importance about pain was that it was often bilateral and occurred successively at different sites. A significant number of patients had pleuritic pain unilaterally though there were bilateral radiological and clinical signs. Pleuritic pain may contribute to the tachypnoea as the patient tries to breathe shallowly to prevent the pain. Tachypnoea however is not solely due to the pleuritic pain.

Sub-sternal pain: Soreness or tightness occured in 20 patients. This resembles that due myocardial ischaemia only superficacially. Substernal pain due to PTE is often transient and unrelated to effort while in infarction it lasts much longer and in angina pectoris it is usually related to effort. Occasionally the pain is of extremely severe crushing nature and prolonged and in these patients the condition was often fatal and postmorte examination showed massive embolism in the pulmonary arteries. Pain of the cardiac type however occured in 18 patients and in some was associated with substernal pain. In these patients the diagnosis may be difficult. Paraesthesia, pain and heaviness of the left arm has been complained of. Others have pain radiating to the neck and back. Here too however a history of effort is not always present. Goodwin states that patient with pulmonary hypertension are able to carry on moderate activity till suddenly seized by a feeling of suffocation. The differentiation from primary cardiac pain will depend often on the presence of other features.

Pain of a cardiac type has been attributed to lowered left ventricular output with reduced coronary blood flow. It has also been attributed to reflex coronary vasospasm. However, both the myocardial and the pulmonary vessels are supplied by the vagus and it is concievable that pathology at these two sites should produce pain of similar distribution.

Cough: was a significant feature in only 48 patients. It was usually of a dry painful, unproductive type. The absence of cough in over 50% is well worth stressing.

Haemoptysis: again is a feature that deserves mention mainly because it is absent in over 50% of cases. In this series it occured only in 39 patients.

The heamoptysis may vary from slight staining of the sputum to frank severe and recurrent bleeding. The latter feature is usually late and occurs in recurrent embolism with infarction and lung destruction. Goodwin (1963) states that when profuse haemoptysis occured and when there was a source of emboli detectable, one of the larger pulmonary vesseles is always blocked.

Bronchospasm with wheezing occured in 11 patients, who had no history of chronic bronchitis as asthma. It is possible that this too is mediated by a vagal reflex mechanism.

Extrathoracic symptoms: The diagnosis of PTE can be very difficult when the patient's presenting symptoms are related to systems outside the chest. The true diagnosis is often obscured for a considerable time as these extrathoracic manifestations dominate the clinical picture. These features are usually not mentioned or sufficiently stressed in popular teaching.

Abdominal pains was a prominent features and often the main complaint in 24 patients. The pain was either epigastric or in either hypochondria. Occasionally, there is pain in the flanks. The pain is accompanied by nausea and vomitting. Occasionally, nausea and vomiting dominate the picture. Occasionally the pain may be misleading in that it may be related to food.

Flatulence and distension were prominent features in 8 patients while acute intestinal obstruction was simulated in 2 patients.

Diahorrea of a persistent type occured in 5 patients and 2 patients had retention of urine. Three patients with diahorea and both patients with urinary retention had pelvic vein involvement.

Two patients had signs and symptoms simulating an acute intestinal obstruction and died before contemplated lapsotomy was done. One of these had thrombosis involving the inferior vena cava. In the other patient there was no intra-abdominal pathology. The severity of these abdominal symptoms has been such that on admission to hospital some of them had been diagnosed as cholecystitis, duodenal ulcer perforation and acute intestinal obstruction. The onset of abdominal pain with or without nausea or vomiting is of special diagnostic significance in the chronic bronchitic in whom the diagnosis in the early stages can be extremely difficult. Though diaphragmatic pleurisy can explain some of the abdominal symptoms, other evidence of diaphragmatic involvement was not always present in these patients, Furthermore, patients with diaphragmatic pleurisy due to other thoracic causes do not appear to suffer from such persistent and acute abdominal symptoms, other evidence of diaphragmatic involvement was not always present in these patients.

The pulmonary vasculature is supplied by the vagus and these abdominal symptoms may be explained on the basis of the theor of central misrepresentation.

The classical symptom so often describe as diagnostic, " the call for the bed pan " prior to death occurd only in 4 patients This desire to have a motion is the result of a vagal reflex. It has been shown experimentally that when the pulmonary artery pressure reaches twice the normal value daefacation occurs and this can be prevented by vagotomy. Symptoms of PTE followed daefacation in 4 patients, here the responsible factor may be the valsalva manoeuvre.

Faints 12 patients gave a histor of fainting or dizzy spells. In som the fainting was associated with exertion Faintness as opposed to shock or collaps does not carry with it a bad prognosis treatment is instituted early. There wer patients who had a history of fainting befor any other manifestation of PTE. This i probably due to a central reflex. When i occurs late in the condition recurrent em bolisation and progressive hypertension wit fall of left ventricular output and subsequen cerebral ischaemia is responsible. Here th prognosis is usually bad.

NEUROLOGICAL MANIFESTATIONS

Frank neurological symptoms occured i 11 patients. 5 had epileptiform seizures 5 had transient hemiplegias and one ha transient aphasia. These symptoms resembl those seen in idiopathic pulmonary hyper tension and may be the presenting feature of micro-embolisation but they do also occu in frank PTE. In two of our patients epileptiform seizures were the presentin features and in the others these neurologica manifestations followed on progressiv embolisation. All these patients had P. M examinations and no intracranial lesion were demonstrated. Drowsiness and a con fusional state are common terminal event They were recorded in a further 11 patients

Mental changes: This is a very strikin feature of thromboembolism. And it

surprising that it is hardly ever mentioned in the literature. Anxiety and depression with restlessness was a striking feature in 21 patients in this series. It occurs early and sometimes before onset of pain or tachypnoea. In the absence of any physical signs in the chest, when marked anxiety is accompanied by any other features such as palpitation, tachypnoea or tachycardia and when the patient complains of pain here and there in the chest or abdomen and when there is also a history of dizzy spells in the past, it will not be surprising if a diagnosis of anxiety neurosis is made. Very often a patient would have been already labelled a hypochondriac. In one patient in this series the psychiatrist was consulted and toxic confusional state diagnosed. Very often the patient would have been labelled by the nursing staff as a troublesome and grumpy type. If inquiry into the past history of such patient reveals no neurotic tendencies it is worth paying serious attention to this form of presentation. In my opinion this anxiety state is of great diagnostic value, especially when the predisposition to P.T.E. is present.

Remissions and relapses : with sometimes long silent intervals is another significant feature. In this series a history suggestive of recurrent embolisation was obtained in 16 patients in which the history dated back for periods varying from a weeks to six years. When complete remission of symptoms occur it is likely that recanalisation of thrombi may be a contributory factor. Not only do the intensity of the symptoms vary but ECG changes and cardiac size on radiology will also show variations.

Physical Signs of Deep Vein Thrombosis. Clinical signs of D.V.T. may often be absent. In this series only 48 patients had any clinical evidence of deep vein involvement. Homan's sign was positive in less than half the patients with other evidence of D.V.T. Bedford places the incidence of a positive Homan's sign in D.V.T. at 25%. A positive Homan's sign in the absence of other evidence of D.V.T. was not detected in this series. The usual signs to be expected are a slight swelling, duskiness and increased warmth of one leg. The superficial veins are fuller and empty slowly when the limb is elevated. Tenderness may the detected in the calf or the sole of the foot.

CARDIAC SIGNS

Perhaps the most constant sign of diagnostic value was persistent tachycardia. 69 patients had a pulse rate of over 100. Of these 21 had a pulse rate between 110 to 130 and 24 had pulse rates over 130.

In 4 patients the pulse rate was so elevated that a diagnsois of primary cardiac arrythmia was made and treated as such.

Persistent tachycardia may be the only positive physical sign of value and in the absence of significant cardiac disease should be a pointer to a posibility of PTE.

Palpitation due to extrasystoles are a common feature. Auricular fibrillation is invariably present when rheumatic heart disease is in the primary condition. But occasionally auricular fibrillation may come with the onset of PTE, in the absence of rheumatic heart disease. On auscultation, triple rhythm, systolic murmur in the pulmenary area and a split pulmonary second sound may be heard. In this series 18 patients had triple rhythm. Systolic murmurs were detected in 20 patients and the split pulmonary second sound in 6. A Graham Steele murmur may be occasionally heard and a friction rub may be heard over the base of the distended pulmonary artery. Evidence of right ventricular failure is frequently present with a raised JVP and an enlarged liver.

Temperature : The temperature chart is often helpful. The onset of persistent low grade pyrexia may indicate deep vein thrombosis and PTE. A temperature between

99° and 101°F was present in 37 patients. Kinsey and White in 1943 pointed out that the on-set of fever in congestive cardiac failure usually meant pulmonary infarction. Perhaps a feature of greater diagnostic value is the occurance of a subnormal temperature. This was recorded in 15 patients in all of whom there was pulmonary embolisation of a significant degree.

CYANOSIS

Central cyanosis was present in 34 patients of whom six had chronic bronchitis and four had rheumatic heart disease. Where embolisation had been the main factor, cyanosis was commoner in the presence of multiple emboli of which there were 17 cases. Five patients with single large emboli had central cyanosis.

It would thus appear that central cyanosis is an indication of fairly severe pulmonary vascular involvement. It is not a feature of early small embolisation. Central cyanosis may be due to the development of Pulmonary A—V shunt or when pulmonary hypertension is marked due to shunting of blood through the fossa ovalis. Pulmonary oedema which is often present may be a contributory factor. In some cyanosis may only be peripheral, and in some pallor is a marked feature.

Peripheral Cyanosis And Shock: Peripheral Cyanosis was present without evidence of shock in 18 patients, Sweating was a prominent feature. The onset of shock is usually ominous. In this series only two patients presenting with shock survived. Occasionally, sweating alone occurs without shock and this was a feature in five patients. Daley et al in 1948 showed increased action potentials in the cervical sympathetic chains when emboli lodged in the pulmonary vessels in experimental animals. This sympathetic stimulation may not only explains the sweating but also explains other features such as palpitations and the anxiety syndrome which is so often seen.

Jaundice: occurred in six patients and in four in whom the serum bilirubin was done it was elevated. In all these patients infarction was seen on post-mortem examination.

Lung Signs: Physical signs in the chest were detected in sixty nine patients. In thirty there were bilateral signs while in thirty four there were only unilateral signs. Unilateral signs were commoner on the right. The usual signs found were crepitations, diminished breath sounds and impaired percussion note at the bases. Pleural rubs were frequently heard. It is important, if one were to detect pleural rubs to get the patient to breathe deeply to the point of pain. The classical signs of consolidation are not usually found as the usual textbook descriptions would have us believe. The diminshed breath sounds and impaired percussion note is often due not to underlying consolidation but to an elevation of the diaphragm which is a common radiological finding in these patients. Pleural effusion is usually detected clinically in the later stages and may be massive persistent and recurrent, especially when associated with primary heart disease or pulmonary infarction. The pleural effusion is usually blood stained but sometimes it is straw coloured.

INVESTIGATIONS

Radiography: Perhaps the most characteristic features of pulmonary embolism is the presence of bilateral lower zone involvement. In the early stages there is diaphragmatic involvement often associated with streaky or mottled shadowing in the lower zones. Occasionally, the appearance may resemble that of consolidation or isolated rounded or oval shadows may be seen. The presence of bilateral pleural effusion in the sameX-ray or when serial X-rays shows the effusions alternating from side to side should alert one to the diganosis. Horizontal diaphragmatic shadows are often seen and these are associated with an elevated diaphragm. These may be due to sgmental atelectasis.

Prominence of the hilar pulmonary shadows, sudden tapering of an enlarged pulmonary artery or the absence of vascular shadows in a segment or lobe is highly suggestive. In these cases, tomography may be of value.

Other radiological features which may occasionally be noted are (1) Abscess at the site of arterial occlusion (2) Calcification of the thrombus (3) Varying size of the right heart and (4) Prominence of the pulmonary hilar shadows. In our series, seventy four patients were X-rayed and of these bilateral lower zone changes were seen in thirty, the right lower zone changes in twenty seven and left lower zone changes in thirteen. Hilar prominence was noted in twenty two and diaphragmatic elevation in ten. Normal X-ray was reported in four and in only one patient were there upper zone changes.

E. C. G. In pulmonary hypertension with right ventricular hypertrophy there is often T-wave inversion over the right praecordial leads. If right ventricular dilatation occurs, right bundle branch block may be present and sometimes may be the only indication to PTE, and is transient. In the case of PTE, this T-wave inversion may be seen as far as V-5. The T-wave is accompanied by a deep S-wave. If the larger pulmonary arteries are involved, dominent R-waves are seen over the right praecordial leads.

Right ventricular strain of an acute nature occurs in PTE, resulting in right axis deviation so that deep S-wave in lead I and Q-wave and an inverted T-wave in lead 3 may occur. Lead AVF may also show an inverted T. Right atrial strain may be evidenced by tall P-waves.

The clockwise rotation produces deep S-waves in V 5. and T-wave inversion is seen often up to V 3 but on occasions as far as V 5.

The limb lead patterns may be mistaken for posterior myocardial infarction. However, ST elevation does not occur. Furthermore there are no Q-waves in lead 2 and a reciprocal depression in lead AVL or Q-waves in AVF.

Blood W. B. C. / D. C. The white cell count is usually raised in the presence of pulmonary infarction, though a normal count did not exclude an infarct.

E. S. R. Usually raised though, again, a normal value does not exclude pulmonary embolisation.

Serum glutamic oxalacetic transaminase values may be elevated but a normal value does not exclude the diagnosis.

Blood Urea. May be raised but is not of diagnostic value.

Serum lactic dehydrogenase. This is probably of more value than the above investigations. It was not done in any of the patients in this series. The levels rise in 24 hours, reach a maximum in 48 hours and falls to normal levels in 10 days.

DEATHS

There were 62 deaths in this series of which 33 were females and 29 were males. Of these, PTE was diagnosed before death in 41 patients. Post mortem examinations were done on 57 patients.

Post-mortem findings: Multiple embolisation was found in 22 patients, multiple infarcts in 8 patients and ingle infarcts in ten patients.

In this series 24 patients were found to have massive emboli. They were situated sitting astride the bifurcation of the pulmonary artery or in either pulmonary artery was more often the site of impaction than the left.

When only unilateral pathology was present the right side appeared to be more often invoved than the left and in all the post-mortems reported there was no men-

tion of upper zone involvement. The predominant involvement of the lower zones may be due to the greater pulmonary blood flow to the lower lobes.

Deep vein thrombosis was found in 36 patients. Of these it was detected during life only in 17 patients. Pelvic vein thrombosis, often associated with femoral vein involvement was found in 8 patients. Of these two patient had pelvic neoplasms. Inferior vena cava thrombosis was detected in two patients.

The right atrium was the source of embolism in one patient. No obvious source of embolism was detected in 6 patients. In the balance six patients, no post mortem reports were available.

TREATMENT

Forty patients had adequate anti-coagulant therapy. Of these five patients died, i. e. 88% recovery rate. Sixty patients had no anticoagulant therapy. Of these four survived and were discharged but not followed up. Fifty-six patients who had no anticoagulant therapy died. Thus there were 93% death rate in untreated cases.

SUMMARY AND CONCLUSION

In this paper I have attempted to draw attention to some of the features of pulmonary thromboembolism involving the larger pulmonary arteries which may help in early diagnosis. Patients with recurrent small emboli are claimed to present first clinically with pulmonary hypertension. The chief features of which are extreme fatigue and progressive dyspnoea during exertion and occasionally dyspnoea follows exertion. Left ventricular output is lowered and features of cardiac ischaemia such as anginal pain and of cerebral ischaemia such as aphasia, hemiplegia or epileptiform seizures may occur. These patients have pale cold extremies, slight peripheral cyanosis, small pulse, raised jugular venous pressure with characteristic flicking "a" waves. Auscultation may reveal presystolic third heart sound, an ejection systolic murmur in the pulmonary area split P2, the diastolic murmur of pulmonary incompetence and the aa-systolic murmur of tricuspid incompetence. All these features develop also in patients with frank PTE, if they live long enough, untreated. I suspect that many patients who present with features of pulmonary hypertension and are supposed to have had silent embolisation, would if carefully questioned, give a history suggestive of recurrent embolisation, such as attacks of unexplained spasmodic dyspnoea, fleeting pains in the chest, bronchospasm and occasional fainting spells. Anxiety may be a prominent feature. No source of embolus is detected in these patients. The most likely site is the pelvic venous plexus.

If patients who present with such transient and indefinite clinical features date their symptoms to a difficult labour and delivery or to suggestive surgical procedure, the diagnosis must be strongly suspected and treated before the onset of obliterative pulmonary hypertension.

Early diagnosis must be made and this is only possible if we do not consider microembolisation as being always silent. In patients with frank embolism too, early features such as anxiety, fleeting pains attacks of fainting and spasmodic tachypnoea do occur and are probably due to earlier microembolisation. Here, however unless treatment is started early, occlusion of larger vessels soon occurs and the full blown clinical picture evolves rapidly. In my opinion, sudden massive embolisation without previous embolism is uncommon. A careful retrospective inquiry into the history of these patients who were diagnosed as massive pulmonary embolism on P. M. examination, has always revealed features suggestive of previous smaller embolisation.

A perusal of the stated causes of death certification in this series of patients would

give a wrong impression of the incidence of sudden massive embolism for another reason.

Patients have been treated for pneumonia pleural effusions or congestive cardiac failure and these have apparantly shot off a massive *embolus which has killed them.* The truth is that in these patients' the primary illness was the result of embolisation and massive embolism was the final and total episode.

It is worth recalling that the largest group in this series i.e. 35%, had no obvious predisposition to PTE and that clinical evidence of deep vein thrombosis was absent in 52%. There appeared to be predominance of males in this group with no predisposition—27 as compared to the 8 females. Deep vein thrombosis has been reported to follow even minor trauma such as sprains. This may explain the male predominance in this group.

In conclusion, I would like to draw your attention once again to the figures quoted earlier regarding survival. 80% of those treated with anticoagulant therapy survived while 93% of those who were untreated succumbed. Pulmonary thromboembolism is therefore an eminently treatable condition especially if the diagnosis is made early.

ACUTE OSTEOMYELITIS

By

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ACUTE Osteomyelitis is today considered a curable disease, and one not presenting any particular problem. But, having treated over forty patients during the last two years, I feel that though we have reduced mortality to zero, morbidity is still very high. The reason for this at least in most cases, rests with the patient and his adherence to the quacks.

Early diagnosis and prompt treatment is very important if the morbidity is to be reduced in the disease and the purpose of this paper is to review this disease with particular reference to early diagnosis and treatment.

Pathology: Osteomyelitis means inflammation of the cortex and medulla of bone. The term does not emphasise the initial focus of infection in the metaphysis of a long bone and almost implies infection of the shaft. Nathan Smith (1804) said "Inflammation is confined to the shaft of long bones." The term is actually indicative of those cases, where the infection has invariably spread to the shaft when the doctor sees his patient and is unfortunate in that it fails to emphasise the all important early phase of the disease.

In the vast majority, the offending organism is the Staphylococcus aureus with an occasional Streptococcal and very rarely a Pneumocceal infection. Acute Osteomyelitis is a local manifestation of a blood stream infection, which is usually transient and which is secondary to primary sources of infection such as furuncles, abrasions or upper respiratory tract infection. In 50% of cases an obvious primary source may no be found (Wandsworth 1948).

The upper end of the femur and lower end of the tibia are the commonest site involved Increased liability to trauma and being th site of maximum growth may be part of th explanation for this predilection.

The first skeletal manifestation of th disease is commonly localised in a singl metaphysis of a long bone. The primar source of infection is NOT in the shaft, t which the infection may spread subsequentl

Anatomical Features: The long bone of th growing skeleton is divided into metaphysi epiphysis and diaphysis. Hent (1937) state "metaphysis represents the bone mo recently formed from the epiphyseal carti age and is therefore more vascular, mo delicate, more susceptible and less immun than the older bone of the shaft which is mo dense and compact. The marrow of th shaft which occupies the medullary cavit is generously provided with cellular el ments while the marrow of the metaphys which fills the interstices between th trabeculae of the cancellous bone, presen a paucity of phagocytic cells.

Since Lexer in 1876, by intravenous in jections of living Staphylococcus aureus i young rabbits produced multiple abscesse near the ends of long bones, various expl nations have been put forward for this eart localisation to the metaphyses.

Hobo studied the vascular arrangemen at the end of long bones. Metaphyse branches of the nutrient artery gradual

become thinner as they approach the metaphyseal region. Just behind the epiphyseal cartilage, they loop back and open into large venous sinuses. There is a marked slowing of the blood stream. This, with a paucity of phagocytic elments, renders the bacteria at a definite advantage.

As the circulation in the metaphyseal side of the epiphyseal plate becomes cut off from the epiphyseal vessels by the age of one year, secondary involvement is not common in children.

Role of Trauma: 50% of the cases usually give a history of trauma. The injury is usually in the form of a mild sprain. This presumably, causes a small haemorrhage in the metaphyseal region, providing a good nidus for the organism to settle and thrive.

As the metaphyseal cortex is thin especially so near the epiphyseal disc, infection within the cancellous metaphysis may spread into the subperiosteal space. As the periosteum is attached firmly to the entire circumference of both proximal and distal epiphyseal plates, involvement of joints does not usually occur. This does occur however in the proximal metaphysis of the femur which is intracapsular and therefore the joint is involved directly.

Thus, in the earliest phase a "small abscess" remains localised to the metaphyses, for a few days. This is called the stage of acute haematogenous metaphysitis.

Progress of Infection: The metaphyseal vessels thrombose, pus invades the cortex, the periosteum is lifted off the cortex, disrupting all vacular connections and leads to cortical necrosis and sequestration. The spread of infection down the shaft is from subperiosteal space via the Haversian canals.

Diagnosis and Clinical Criteria: There has never been the slightest doubt about the tremendous usefulness of early diagnosis. Platt (1928) said "Acute Osteomyelitis must be given the pride of place in the clinical conciousness of the practitioner." Hent (1937) wrote "The incidence of chronic Osteomyelitis will only be reduced when the lesion is attacked during the stage of "acute metaphysitis" before perforation of the thin metaphyseal cortex." Alteimeir and Wandsworth (1948) in evaluating the results of treatment by penicillin in 71 cases concluded. "The results were excellent, with almost complete healing of bone, with minimal bone damage, when Pencillin was started within three days of the onset of symptoms.

Acute Osteomyelitis need not be a crippling disease, if the diagnosis is made early and the lesion treated properly. This is illustrated by the following case:—

K.R. was admitted with a history of trauma of 3 days duration. Clinicaliy, he was tender over the lower end of radius. X'ray showed no abnomality. At operation on the same day, pus was evacuated by drill holes. He made an uneventful recovery, and left hospital in a fortnight. He was on Penicillin for three weeks and the E. S. R. was back to normal in three weeks. He has no complaints since then.

Pyrah (1933) Harris (1960) Trueta (1954) found that more than 50% of the cases came only about 4 to 5 days after the illness and Harris (1960) found that only in 25% of cases was the correct diagnosis made at the first examination.

One wonders why a correct diagnosis is not made in time. There are several factors for the delay. The main reasons are as follows:—

(1) **Treatment by quacks.** Well over 75% of our cases came to us after treatment by quacks, when part if not the whole shaft was involved. Trauma takes the patient to the fracture specialist of the "Ayurvedic world" and this treatment appears to be

"Pathu" and "Magical Oil" for traumatic pain. Such people are doing a dis-service to the community. They make a diagnosis of fracture or sprain and carry on this "treatment." It is when the patient's condition deteriorates that the patient comes to us. Whereas early diagnosis means a cure usually in three weeks, these patients end up with pathological fractures and a high risk of chronic Ostemyelitis. This is well illustrated by the following case:—

Patient R.A. aged 6 years was admitted on 20-4-65 with osteomyelitis of upper Tibial Metaphysis. At operation, on same day, a large Subperiosteal abscess was evacuated. Patient was on Pencillin for five weeks and on 26-5-66 and 7-8-66 sequestrectomy was done and the wound healed. This wound broke down and a sequestrectomy had to be done five months later before complete cure took place.

II. Diagnosis of "sprain" made: A history is obtained in 50% of cases. This is a veritable trap. The injury is usually a minor one and the patient is apparently well for a few days.

III. Diagnosis of Rheumatism or Poliomyelitis: We have not had any cases where this confusion arose, but when in doubt as Wilkinson (1951) wrote "It makes very little difference if a patient with Rheumatic fever or poliomyelitis is initially treated with pencillin, but, it makes a great difference both to leg and limb if a patient with Osteomyelitis is given salicylates.

IV. Confusion between Osteomyelitis of the end of a long bone and Septic Arthritis:

This arises particularly around the knee. Sympathetic reactionary effusion in the knee joint is not uncommon in infection around the lower end of femur. Presence of localised tenderness in Osteomyelitis and the fact that the knee can be moved in this condition clinches the diagnosis.

V. Patient reported late for treatment: In the early stage the constitutional upset may not be much and local pain very little. Hence the patient may ignore the condition for a few days causing a serious delay in the diagnosis, leading to severe chronic Osteomyelitis.

Diagnosis: The clinical picture of a child who is pyrexial ill and toxic, who resents interference, complains of pain in a limb and presents swelling, and tenderness near the end of the affected long bone is well known.

But one must bear in mind that the temperature may be below 100° or even normal. W.B.C, E.S.R. and X ray changes may not be abnormal for sometime. Blood culture if negative does not preclude the diagnosis.

The most important sign is marked localised tenderness over the end of a long bone. Trueta (1954) found it in each of his 10 cases. Even in an unco-operative child with patience, one can find a localised tender spot.

If the disease is kept in mind throughout and the point of localised tenderness sought near the metaphysis of a long bone, mistakes in diagnosis can usually be avoided.

TREATMENT:

The advent of Pencillin as a potent systemic antibiotic was seized upon by the entire surgical world. As the "be all and end all" of the treatment of osteomyelitis. Florez (1943) reporting the results of treatment in his first 15 cases of serious infection treated by systemic pencillin 5% of which were cases of Acute Osteomyelitis said "with adequate a dose of Pencillin Osteomyelitis could be controlled without surgical interference," Agerholm and Trueta (1947) reporting on 30 cases had a 100% cure rate. Trueta however admitted that in cases with abscesses on admission, surgery was necessary.

Antibiotics and surgery are both required in practically all cases.

Plan of Management:

(1) On admission the diagnosis is entirely clinical. X'rays are done to confirm Osteomyelitis, or exclude other bone diseases. Blood culture, W.B.C./D.C. E.S.R. are usually of no value and may mislead the inexperienced.

(2) Antibiotic Therapy: We still use Penicillin in spite of the Resistant Staphylococcus aureus. This is changed 48 to 72 hours later when culture and Antibiotic sensitivity reports are available.

(3) General condition of the patient may require attention. Anaemia, dehydration may require transfusion.

Metaphyseal decompression should be carried out as soon as is practicable. There is nothing to gain and everything to lose by waiting.

An analogy between acute metaphysitis and acute appendicitis is not out of place and is very apt. The tragic complications of both diseases are due to 'perforation'. As much as accepted treatment of acute appendicitis is surgery before perforation, so also the best treatment of acute metaphysitis is decompression before rupture into the subperiosteal space. A subperiosteal abscess should be considered analogus to peritonitis and be must regarded as failure to institute early treatment.

Trueta (1946, 1954) advocated waiting for twenty four hours before surgery is considered in acute cases. There are serious objections to this line of treatment.

After twenty four hours of antibiotic treatment, it might not be so 'straight forward' to make a decision for or against operation. The average (really over 90%) cases of osteomyelitis presenting in this country on admission have passed the stage in which resolution with only antibibiotics is likely and valuable time is lost.

On the other hand the advantages of immediate operation are many:

It is based on a sound pathological fact that the lesion is localised to the metaphysis in the early stages. This is decompressed before it has a chance to spread. It prevents or minimises the thrombosis in the venous sinuses and nutrient artery. This instantly relieves the pre-operative excruciating pain. The relief of pain is very constant.

It affords an early opportunity of obtaining a sample of pus before the patient has received any appreciable amount of antibiotics.

If one follows the policy of immediate operation in every case of osteomyelitis, negative explorations are bound to occur. But this does no harm. In our experience there has been no case of negative explorations, as patients hardly come to us at an early stage.

Guttering of the bone is preferred by some to drilling the bone when the condition is more advanced than that of a very early metaphysitis.

At times, intramuscular abscesses cause a periosteal reaction and present as cases of osteomyelitis. We have had four of these, in which one showed definite periosteal reaction. At operation drilling showed no pus. These wounds heal and present no further problem.

Post-operative Management:—How long should antibiotics be given?. We usually give antibiotics for two weeks, if all goes well with the wound and the patient in general. In lower limbs post-operative non-weight bearing is also important particularly when the X-ray shows patchy destruction of the bones without much newbone formation. In these cases once the wound is

healed, the limb is immobilised in a plaster cast as long as is necessary.

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THE CARE OF THE PATIENT ON A RESPIRATOR

By

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THERE are many circumstances in which a patient may require the use of a respirator.

It may only be for a short period as in the treatment of emergencies such as electric shock, drowning or poisoning e.g. with Barbiturates, salicylates or for far longer periods as in the treatment of poliomyelitis, Tetanus, Diptheria, Acute Infective Polyneuritis, Crush Injuries of the chest ("Stove-in" chest) and in head injuries. Respirators are also used in anaesthesia for controlled ventilation of the lungs and in the treatment of post-operative respiratory insufficiency from any cause resulting from surgery or anaesthesia.

It should be realised by those who are assigned the task of managing these patients that they are dealing with ill human beings who have a poor cardio-respiratory reserve and function and that they are easy victims of infection. Their lives depend on constant medical supervision and devoted care.

STERILITY

The patient should be nursed in a clean room and all sterile precautions taken. Masks should be worn by all who enter the room. The person attending on the patient directly should scrub and wear sterile gloves especially when attending to the tracheostomy wound and during the aspiration of secretions from the airway.

THE TRACHEOSTOMY

If a patient is to be kept on a respirator for any length of time, a tracheostomy has to be done for three reasons viz.

(1) It is easy to suck the airways free of secretions. If secretions are allowed to collect in the airways, these may get inspissated and form a nidus for infection. Also, these secretions may block the airway and prevent adequate inflation of the lungs.

(2) It is easy to give these patients intermittent positive pressure respiration (IPPR) without inflating the stomach.

(3) The tracheostomy tubes used in this sort of work are provided with inflatable cuffs which when blown up, fit the tracheal wall snugly and prevent the entry of saliva, mucus, blood and vomit into the respiratory passages.

The tracheostomy wound should be covered with dressings soaked in an antiseptic solution such as Chlorhexidine 1% solution. This has been found to be very effective in the prevention of wound infection in many centres. Swabs from the wound should be taken as often as possible and sent for culture and antibiotic sensitivity. One should not handle the tracheostomy wound unnecessarily especially with bare unclean hands.

The tracheostomy tube should be changed every three or four days, depending upon the amount of crusting and obstruction that is encountered.

The cuff of the tracheostomy tube should be deflated every three or four hours for about five minutes at a time. This is preferably done during one of the suction procedures. The pharynx should be cleared *before* the cuff is released. Secretions can collect between the glottis and the tracheostomy tube cuff. It is therefore very import-

ant to release the cuff and apply suction at the same time during this procedure.

HUMIDIFICATION

Normally, the air we breathe is warmed and moistened in its passage through the nose and nasopharynx. This capacity to warm and moisten the air is lost once the patient has a tracheostomy. This will result in drying and crusting of secretions as mentioned earlier. Therefore, the air being pumped into the patient's lungs must be warmed and moistened by passing the air through a humidifier. There are several types of humidifiers available. The best ones have a thermostat which controls the temperature automatically. The temperature of the inspired air is usually kept between 30°C and 40°C. The required temperature of the water can be set on the humidifier. It is important to see that the hose leading to and from the humidifier is not loose in order to avoid loss of inspired volume, due to leaks in the circuit.

CONNECTION TO THE RESPIRATOR

There are a large number of respirators in use in Intensive Care Units all over the world. They vary widely in cost and complexity and also in case of maintenance and repair. Examples are the Radcliffe series, Barnet, Beaver, Cyclator, Engstrom, Blease Pulmoflator, The Drager Poliomat, Smith-Clarke, Bird etc.

Every hospital worthy of its name should be equipped with two or three of these respirators particularly because of the number of conditions which may require its use especially as an emergency, e.g. One respirator could be kept in the out-patient department, another in the operating theatre and a third in a Thoracic Unit. All three should be freely available to any ward in the hospital whenever it is required.

The doctor and nurse looking after the patient should be thoroughly familiar with the respirator used in their place of work. Indeed it is important for all doctors and nurses to know this if adequate measures are to be taken in the treatment of these diseases. The writer's experience is that this knowledge is sadly lacking and attempts to get people interested in this aspect of nursing care have been unrewarding so far.

The nurse must be familiar with the working of the respirator because she is the person who will be in continuous attendance on the patient. This applies to the Houseman too because he will be summoned by the nurse when things appear to be going wrong.

Before the respirator is ready for use it should be carefully checked for leaks in the circuit and for any electrical faults. One should see that the manually operated part of the respirator is functioning and ready in order to be prepared for a power failure. If the machine is driven by compressed air, then spare cylinders should be kept ready for use.

After the patient is connected up to the respirator, it is important to see that the respirator is shifted to the opposite side of the patient every twelve hours or so in order to avoid prolonged pressure on one part of the tracheal mucosa all the time. During a suction procedure, an assistant can empty the water traps of the respirator and also check and replenish the water in the humidifier.

Once the patient is connected to the respirator one should auscultate the chest and check that the air being pumped in is entering both lungs equally and evenly and that the airways are always free of secretions.

MEASUREMENTS

(1) Tidal Volume

The volume per inflation should be about 400ml to 500ml in the average

adult and proportionately less in the children and infants.

(2) Minute Volume

This is the volume of air being pumped in per minute. This is about 10 to 12 litres per minute in adults.

Both these values can be measured on the volumeter provided on the respirator (usually a Wright or Drager respirometer)

These volumes should be measured at regular intervals and if there is a progressive fall in the volumes so measured, there may be a leak in the circuit or there may be respiratory obstruction. e.g. secretions, over-inflated trachy tube cuff, trachy tube in bronchus. This should be attended to at once.

The volumes of the air required to adequately ventilate each patient is given on a chart called the Radford Normogram relating Age, sex, weight, body temperature to ventilatory requirements. In actual practice however it has been shown that the values given by the normogram are less than 50% to 100% of the volumes actually needed by these patients.

(3) The pressure of inflation

In some machines like the Radcliffe respirator, the pressure of inflation can be set by moving a set of weights up and down the arm connected to the inflating bellows. The pressure indicated on the manometer on the machine is the pressure at the mouth in centimetres of water. The pressure in the airways and in the alveoli lower down is less than this.

If it is found that the minute and the tidal volumes show a progressive fall over a certain period, do not increase the inflation pressure without checking for airway obstruction.

Always clear out the airway of secretions and check the cuff of the tracheostomy tube and also its position. Usually, it is found that with tracheal toilet alone the respiratory volumes revert to normal and no increase in pressure is required. This way of checking applies to all respirators although individual variations in operation and adjustment may be encountered. It is therefore important to know how a particular respirator in a respiratory unit is worked.

MONITORING OF PARAMETERS

When a patient is on a respirator it is important not only to see that the patient is being ventilated properly but also that all physiological values are as normal or near normal as possible.

(1) The general condition of the patient should be continuously watched 6g Is he conscious, semi-conscious or comatose? Is there any evidence of dehydration? Check blood pressure, pulse rate and volume, colour of the mucous membranes. Anaemia? Cyanosis? In some centres the E.C.G. is continuously monitored by an oscilloscope as is the E.E.G.

(2) The tidal volume and the Minute Volume.

(3) The pH of the blood, the arterial pO_2 the pCO_2 and the standard bicarbonate of the blood should be measured regularly. The aim is to keep the arterial pCO_2 at 40 mm Hg. It is sometimes mistakenly thought that if the arterial pCO_2 is kept at 40 mm Hg, then all is well. But there are other factors such as metabolic acidosis (e.g. due to anoxia, diabetes mellitus, renal failure which are not allowed for in this concept. Even if the pCO_2 is at or near 40mmHg, the pH and the standard bicarbonate value may not be normal. The respiratory volumes required to keep the pCO_2 normal in one patient may not be the same as that required in another patient because one has to compensate for any metabolic acidosis by an increased ventilatory volume (i.e. create a compensatory respiratory alkalosis) and if this measure is still inadequate, actively give drugs like

Sodium bicarbonate iv or trishydroxymethyl amino methane (THAM) to overcome the metabolic acidosis. This is why it is important to measure the pH and the standard bicarbonate of the blood as well as the pCO_2 of the blood.

However, it has been found that a reasonable *overall* index of respiratory inadequacy is the standard bicarbonate value of the blood. If this is over the range of 24 to 31m. eq/100 ml, then there is a *tendency* to alkalosis and the reverse means a *tendency* to acidosis due presumably to overventilation and underventilation respectively, other causes of alkalosis and acidosis being previously corrected.

FEEDING

Intravenous drips must be avoided but this may not always be possible e.g. patients with traumatic injuries may require drips. Generally, an intragastric tube passed nasally is usually adequate. Saline, dilute milk, Casilan, Whole milk may be given in that order prior to moving on to a normal diet.

BLADDER CARE

This depends upon the type of case one is treating. If the patient is passing urine normally, it is best not to interfere with a catheter. In other cases where there is a loss of bladder control, catheterisation may be essential. The urine output should be charted and a fluid chart maintained.

Skin Care

This is very important. The patient should be nursed on each side for two to three hours and then prone for another three hours. The limiting factor in this case will be the poor circulatory state of the patient.

The Environmental Temperature

This should be around 70°F for reasonable comfort in this country.

Conclusion

The foregoing is a *general account* of the management of a patient on a respirator in any disease or condition which requires such treatment. In detail however there are variations with each disease and also varying practice in different Intensive care units. What is common to all units is the following of a set plan of treatment and unremitting devoted medical and nursing care.

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ABO BLOOD GROUPS AND DIABETES MELLITUS

an analysis of 300 Tamil diabetic patients

By

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THE discovery of blood groups have had their impact on medicine in various ways. Blood transfusion is practised on a large scale. They have important medico-legal applications. (Fernando 1958). Blood groups are proving to be of great value in anthropological studies, in the evaluation of relationship between races and their migrations. (Mourant 1954, Weiner 1946). Study of inheritance of blood groups have contributed to a large extent in the understanding of human genetics. During recent years it has been realised that blood groups which were considered harmless variations and little affected by selection are in fact closely associated with considerable differences in susceptibilities.

Since the attempt of Alexander (1921) to establish an association between blood groups and diseases, numerous studies have been made in this field. In this article a relationship between group A and diabetes mellitus has been demonstrated. No other reports of studies in this field in Ceylon are available for comparison.

Source of material

The entire material was obtained from diabetic patients being treated at the Government General Hospital, Jaffna. All patients studied were Tamils. These patients were diagnosed as diabetics at one of the two medical clinics run at the above mentioned institution and were either continuing treatment at one of these clinics or had been referred for routine therapy to the diabetic clinic. The control group for the study was obtained from the analysis of blood groups of 9000 Tamil patients and blood donors at the same institution. These results have been published in this Journal in August 1966. Vol—VI part II.

Results

Table I. Distribution of blood groups in the diabetic patients.

	A	B	AB	O	Total
Males	43	61	5	62	171
Females	34	36	3	56	129
Total	77	97	8	118	300
Percentage Distribution	25.67	32.33	2.67	39.33	

Table II. Percentage distribution of the ABO groups in the diseased and control populations.

Total No. examined		A%		B%		AB%		O%	
Diseased	Control	Diseased	Control	Diseased	Control	Dis.	Con.	Dis.	Con.
300	9000	25.67	20.56	32.33	31.31	2.67	5.36	39.33	42.77

As could be seen from table 11 there is an excess of Group A in the diseased population. Application of the X^2 (chi squared) test of significance to these figures shows that there is a significant difference in the distribution of the blood groups in the two groups. X^2 for 3 degrees of freedom is 8.7166 which is significant at the 5% level. Comparing the relative proportions of group A and the rest taken together X^2 for one degree of freedom is 4.7265, which is again significant at the 5% level. Comparing group O and group A, X^2 for one degree of freedom is 4.065 which is also significant at the 5% level.

Hence it would seem that there is a significant excess of group A among the diabetic patients. This finding corresponds to the findings of McConnell, Pyke and Roberts (1956). In the present study sub-divisions into age and sex groups have not been attempted due to the comparatively small numbers. It is intended to do a more detail study when a larger series has been gathered and the results would be published in this journal in due course.

Historical account and comments

The first paper that was published on this subject of blood groups and diseases was by Alexander (1921) who concluded that there was a higher percentage of group B and AB among patients suffering from various types of carcinoma. Buchanan and Higley (1921) conclude that there was no association between blood groups and diseases. They based their conclusions on the study of 2446 patients who were classified into 17 disease groups. It was subsequently shown that they had not correctly interpreted their figures, and from those same figures it was shown that 3 of the disease groups considered namely chronic ulcers (almost all Peptic), jaundice and pernicious anaemia showed a distinct association with blood groups. (Roberts 1957)

Further investigations of blood groups and diseases appear to have been on the lines of discovering a possible change of blood groups due to disease.

The next significant positive finding was that of Ugelli (1936) who established the association between group O and peptic ulcer.

Though sporadic investigations were being carried out in this field the greatest contribution was in 1953 by Aird Bentall and Fraser Roberts. They investigated the blood groups of 2854 patients suffering from carcinoma of the stomach and found that there was a significant excess of group A and a deficiency of group O.

Further these authors along with Woolf (1954) established statistical procedures for the analysis of figures in this field of investigations. Due to the small numbers that were available at each centre it became necessary to combine data from different centres and from different countries. Simple addition of frequencies would not yield a correct combined estimate or lead to valid tests of significance. Hence it became necessary to weigh the differences in blood group incidence between the diseased and the control series taking into account the differences in the incidence of blood groups in the normal population for the different areas.

Aird, Bentall, Mehigan and Fraser Roberts (1954) showed a statistically significant association between group O and peptic ulcer. In that series 1015 patients with gastric and duodenal ulcers were examined but there was no significant difference between the two types of ulcers.

Subsequently with a larger series it has been shown that the association between group O and duodenal ulcer is closer than between group O and gastric ulcer. (Roberts 1957).

Stimulated by these positive findings investigations were being carried out on

these and other diseases. No variations were obtained in the series for carcinoma of the breast, bronchus, colon, and rectum from the controls. Conflicting findings were also reported. Wallace (1954) in Scotland, Billington (1956) in Australia found no significant excess of group A in carcinoma of the stomach. These same investigators found no significant differences between controls and patients with gastric ulcer. However there are larger number of workers who have agreed with the findings of Aird et al than those who have reported contradictory findings.

Most of the reports on gastric ulcer and carcinoma of the stomach were statistically examined by Fraser Roberts with the mathematical technique of Woolf of combining data from different areas and he showed a significant excess of group O in peptic ulcer and group A in carcinoma of the stomach in the over all analysis.

A number of other diseases too have been studied. Patients suffering from pernicious anaemia show an unduly high percentage of group A.

Bronchopneumonia studied by Struthers (1957) showed a significant reduction in group O and an increase in the groups A, B and AB. These findings have been contradicted by others.

Speedby (1959), Stewart and Krut (1962) have found a deficiency of group O in patients with coronary heart disease.

Other diseases like pituitary adenoma, portal cirrhosis, rheumatic fever, toxaemia of pregnancy and hypertension have been studied but contradictory findings have been reported.

Besides diseases fertility, sex ratio, age of onset of menarche and other conditions have been studied. Kirk, Kirk and Stenhouse (1953) in their series claimed to have shown a highly significant trend in fertility. Group A women being more fertile than women in group O between the ages 25 and 35 and group O women being more fertile before and after this period. This finding too has been contradicted on various grounds (Edwards 1957).

Following the discovery of association of particular blood groups with certain diseases it became obvious that there were certain advantages or disadvantages attached to the possession of certain genes for the respective blood groups. Hence blood groups may be said to have selective value.

From the classical work of Bernstein, Fisher and others it was clear that there was a polymorphism as far as the blood groups were concerned and amply displayed by the ABO system of blood groups. (Polymorphism just means that normal human chromosomes contain one or the other of a pair of alleles, neither being rare or may contain one or the other of a series of multiple alleles of which all or some have appreciable frequencies—(Roberts 1963). Most populations have appreciable amounts of the genes A, B and O with rare exceptions like the absence of gene B in the South American aborigines. This polymorphism that has existed for centuries necessarily implies a balanced polymorphism and could exist because of its advantage to the species. It must depend on the dynamic interplay of selective forces and it is no mere accidental concentration of genes of neutral or of almost neutral selective value (Roberts 1963).

The distinct susceptibilities of certain blood groups to particular diseases hence would contribute to this balanced polymorphism.

A number of explanations for the association of blood groups with diseases have been put forward.

Anti-bodies of the blood group system have been implicated but this does not hold much grounds when it is realised that the

anti-bodies present in groups A and B are both present in group O and hence it is very unlikely that they have differential actions in the individuals with different blood groups.

H substance found in individuals with group O has been incriminated in the mechanism of causation of peptic ulceration.

This substance is found in a large measure in the intestinal secretions of the secretor group of individuals. However the H substance is also found in smaller quantities in individuals of other blood groups and no definite evidence is available to show that the H substance which is a mucho polysaccharide plays a role in the production of peptic ulcer.

Similarly the group specific substance A and B may be incriminated in either the direct production of the disease or may afford a protective action against any particular disease. This again appears rather unlikely for in the case of association of group A with carcinoma of the stomach viewed with the knowledge of chemical carcinogenesis mucopolysaccharides could hardly be blamed for the causation of carcinoma.

It has also been postulated that major genes may also produce additional effects, though geneticists have usually conceived of genes as having a single specific effect. However with the accumulation of knowledge in the field of genetics this viewpoint is gaining grounds.

The pattern of distribution of blood groups in western Europe and Britain in general approximately is group O 46%, group A is 42%, group B 9%, and group AB 3%. (Koch 1957). Afro-Asians differ in that group B shows a higher incidence. Tamils follow the pattern in the Afro-Asian countries (Hills 37, Seneviratne 41, Koch 53, Fernando 58, Joseph 66). It would hence be interesting to observe whether there is a higher frequency of group B associated with disease in the Eastern races or whether the Western pattern prevails. I have not been able to obtain any series done in the Afro-Asian people, most of this type of work having been done in the Western countries. Koch (1957) stated that studies were being undertaken in Ceylon but the Author learns that these studies were not completed for any reliable conclusions to be made. In the author's present series it would be observed that among the Tamils despite the fact that Group B is the predominant group there was a significant excess in group A among the diabetics which corresponded to findings of McConnell Pyke and Roberts in a series done in Liverpool. This observation would thus favour the hypothesis of additional effects of major genes accounting for the association of certain blood groups with particular diseases.

Summary

The frequency of ABO blood groups among 300 Tamil patients suffering from diabetes mellitus has been studied and comparisons made with control series.

A significant excess of group A is observed among the diabetics. In spite of the excess of group B in the control population the diseased group showing an excess of group A, which corresponds to the disease pattern in the Western countries where group A is the predominant group, would indicate that perhaps the association of blood groups with diseases is due to an additional influence of the particular major gene responsible for the blood group.

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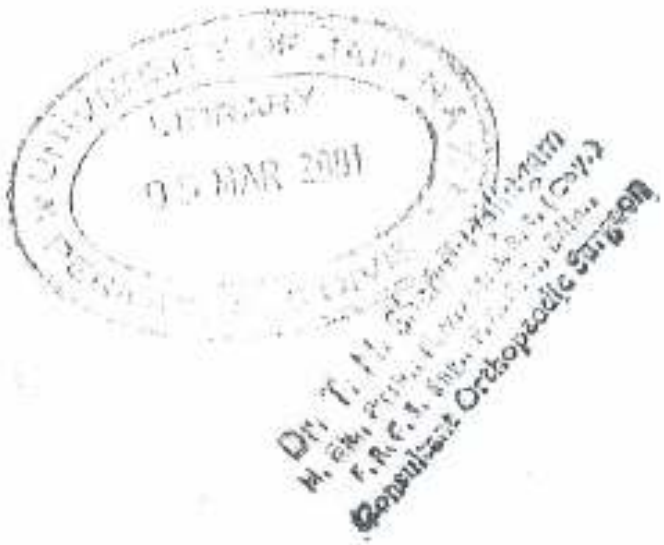

CORNEAL GRAFTING

A CASE REPORT

By

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PATIENT S. S., 38 years of age of Uruthrapuram, a father of nine children, complained of defective vision affecting both eyes. His story was the sad one so often heard in our clinics. Nine years ago he was struck in one eye by a paddy stalk. He received native treatment for this eye injury. The result was that he was left with a blind eye with a whiteness of the black of the eye (leucoma). Six years later he lost the sight of the other eye after a similar course of events. Once again he was left with the same type of corneal opacity. He never sought eye treatment at any State Hospital.

On 9-1-67 this man was admitted to the Eye ward for the treatment of these bilateral corneal opacities. He was blind and miserable and had to be led about by someone.

On examination his vision was found to be very poor. Vision Right eye=counting fingers at 2 ft. Vision Left eye=counting fingers at 1 foot. He did not have nystagmus or strabismus. The cornea was scarred to the same extent on both sides. The right corneal opacity was slightly vascularised. There were three or four superficial blood vessels running into the opacity from the periphery. There were no deep blood vessels. The iris and the pupil were visible hazily under powerful light and with the use of the corneal microscope. The anterior chambers were of normal depth. The iris was not adherent to the back of the cornea. The thickness of the cornea was difficult to assess. The state of the lens and even less so, the state of the vitreous could not be reliably guaged. Projection was good in both eyes and it was concluded that the retinae of the two eyes were functioning well and that sight could be restored if a successful corneal grafting operation were done. It was decided to graft one eye, the right eye.

Other Investigations:

The blood pressure was 130/80.

The WBC & DC was 7,600/ cmm.

Polymorphs	—	73%
Lymphocytes	—	19%
Eosinophils	—	8%

Urine report — Sugar, nil.
Albumin, nil.
Deposits, few epithelial cells.

Conjunctival smear—No pathogens were isolated.

The patient was judged clinically to be of normal health apart from his visual disability.

On 15-1-67 a non-penetrating (lamellar) corneal grafting was done on the right eye. The technique used is described below.

TECHNIQUE OF OPERATION

Premedication.

Phenobarbitone gr. 2 was given at 7 A.M.

Pethidine 75 mg IM and, Largactil 25 mg IM were given at 7-30 A. M. (Chlorpromazine.)

Anaesthesia.

Local. (a) Cocaine hydrochloride 4% surface.
(b) Xylocaine 2%, 2.5 ml Facial akinesia.

(c) Xylocaine 2%, 1.25 ml Retro-ocular.

(d) Xylocaine 2% lid infiltration.

The eye was steadied by the application of sutures through the four rectus muscles. The two mattress overlay sutures crossing each other at right angles were then applied. The eye was then closed and the cutting of the graft from the donor eye was started. The method used for cutting the graft was that invented by Garber. All that is required for cutting a lamellar graft is a sharp razor blade and two strips of stiff sterile carboard. Special care was taken to retain the half of the disc cut from the donor cornea, bearing the epithelium. The corneal disc was cut (punched out) using a 7 mm Francheschetti trephine. The next step was to cut out a similar disc from the cornea of the patient's right eye (the graft bed in the recepient eye). This was done by trephining with the same trephine used on the donor cornea, to a depth of 0.5 mm. The disc was then cleanly split off from the patient's eye using a medium sized Desmarse's knife. After the disc of diseased cornea was removed, it was seen that a few scattered dots of deep opacities were left in the deeper layers. However a large part of the pupillary zone was clear. The split donor disc of cornea was then placed on the recepient's graft bed, care being taken to place the graft with the epithelium on the outside. The sutures which had been drawn aside to permit trephining were now gently drawn over the graft and tied. Locking sutures were placed at the intersections. The eye was dressed with chloramphenico l ointment and, both eyes were padded.

Post-Operative Care

Injections of Penicillin, 500,000 units IM twice a day for 6 days. Sulphadimidine 2 tablets three times a day. Pethidine 75 mg on the night of the operation day.

On the third post-operative day, it was noted that the pupil was small and, it was suspected that the patient had developed an iritis. Atropine 1% in oil was instilled. The patient was also put on oral steroid therapy (prednisolone 10 mg twice daily). On the 4th day the pupil was still undilated. On the 5th day the pupil was still undilated and it was thought to be the result of adhesions of the pupil to the lens (posterior synechiae). On the 6th day the pupil had not dilated. The graft was in its place but as the sutures and fibrin around the sutures were preventing a clear view of the iris and pupil it was decided to remove the sutures. On the 7th day the sutures were removed though it was too early to do so. After removal of the sutures it was seen that the pupil was irregular and semidilated. On examination with a loupe it was seen that the adhesions were old ones probably formed during the time of his eye injury, and treatment. There were no signs of an active lesion in the iris. There were a few corneal opacities present. Local steroid therapy was commenced. Prednisolone was reduced to 5 mg tds. On the 8th day atropin was discontinued. On the 13th day the corneal opacities had become smaller and slightly less dense.

On the 17th day Vision in the operated eye was 6/24 with —2D spherical and —1D cylindrical at 180 deg. On the 3rd day microscopic examination did not reveal any vascularisation of the graft. The pupil was clear of exudates. The sight in the right eye was 6/6 part (Tamil Test Type) with the same glass as the one tried earlier. The patient was able to read N5 (near vision) without a glass. The patient insisted on going home to see his family after three years of sightlessness. In fact the last child had died soon after birth during this period and the patient had never seen the child.

The unaided sight in the operated eye (right eye) at the time of discharge from hospital was 6/24 distance vision and N5, (normal) near vision. The patient promised

he would be back for further observation on this eye and for surgery on the left eye.

COMMENTS

The commonest eye operation done in Ceylon is cataract extraction in one form or other. The same cannot be said of corneal grafting or keratoplasty. Two factors have made this operation a comparative rarity. One drawback has been the difficulty of obtaining suitable donor material, meaning fresh human eyes or preserved cornae. The other has been paradoxically enough the non-availability of cases suitable for corneal grafting. In short we have an abundance of complicated cases of corneal opacities which have gone even beyond the reach of corneal grafting. It will therefore be evident that we must aim at the proper treatment and prevention of corneal diseases before we can even think about corneal grafting. The quack treatment of disease of the eye is an enormous calamity in this country. A full discussion of these problems is beyond the scope of this article.

It is the comparative rarity of this operation and the excellent visual results obtained that have prompted me to report this case, which is the second of a similar operations (three operations) done by me recently in Jaffna. The eye used on this patient was donated by the Gampaha Eye Donation Society. The eye was that of a little girl 1½ years of age. She died of gastro-enteristis complicating Kwashiokor. Enucleation was done within one hour of death on 13-1-67 at 7 p.m. The eye was received by me on the 14th night at 9-30 p.m., at the railway station. On the 15th morning the eye was used at about 8 a.m. The cornes was used for grafting 38 hours after the death of the child.

Mode of Preservation and Transport

The cornea was sent in sterile liquid paraffin in a small glass jar with a wide mouth and well fitting glass stopper. The glass jar was placed in a large ice flask surrounded by polythene bags containing ice cubes. The flask was sent by the express train—the Yarl-devi. On opening the flask on 15-1-67 the eye was seen to be well preserved and the cornea was found to be slightly cloudy.

Thanks are due to the parents of the little child who died an untimely death but gave a fresh lease of life to a poor man and his large family. I must also thank the secretary of the Gampaha Eye Donation Society, Mr. Siriwardene for sending us the eye for grafting. I must also thank the theatre staff and ward nurse for the valuable help given to make the operation a success. It was something new to them but they were eager to learn and help.

MANAGEMENT OF A PATIENT IN "IRREVERSIBLE" SHOCK

CASE REPORT

By

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PATIENT K. B. B. 9367VOR age 32 years was admitted to ward 13R of the General Hospital Kurunegala on 3-3-67. The admitting officers notes read "Severe anaemia complicating pregnancy. Swelling of the body. Pregnancy six". On admission her Blood Pressure was 100/70 mm Hg. Hb 25%.

Between admission and 27-3-67, the date of delivery, she was treated to a Hb % of 55. The birth of a baby weighing 7 pounds was uneventful. The placenta was expelled normally. The placenta looked incomplete on examination. She had postpartum haemorrhage. A blood transfusion was given at 4-30 a. m. There was no response to treatment. An emergency hysterectomy was decided on. The operation commenced at 5 a. m. on 28-3-67. A subtotal hysterectomy was done. A piece of placental tissue was adherent to the wall of the uterus. Two pints of blood were transfused during the operation. At the end of the operation, she was conscious and rational and all reflexes had returned. She had a tachycardia and a very low Blood Pressure at the end. No record of the immediate post-operative condition was made. She was removed to the recovery bay of the operating theatre at 6-30 a. m. Between 6-30 a.m. and 8-30 a.m. she received two pints of cold blood rapidly, one pint of Macrodex and two pints of 5% Dextrose, to restore her blood volume.

At 8-30 a. m. her blood pressure was unrecordable, pulse rapid and of low volume. She was covered with sweat, very restless, confused and dysponeic. Morphine 8 milligrammes and promethezine 12 mg. were given intravenously. This made no difference to the clinical condition, she remained restless, blood pressure not recordable, covered with sweat, limbs cold and cyanosed.

As she was still very restless at 10-45 a. m. she was given Morphine 10 mgm intravenously. This made no improvement to the clinical condition. Around 11 a.m. her condition deteriorated, she was semiconscious and confused, had an ashen countenence and was very dyspnoeic. The radial pulse was not palpable, blood pressure could not be recorded, extremeties were cyanosed and very cold. Oedema of the whole body was observed Intravenous therapy looked useless at this stage. She had progressed gradually to an irreversible stage of shock.

At 12-30 p.m. Promazine 20 mgm diluted in distilled water was given intravenously until the extremeties were flushed and warm to the palpating hands and the flow indicated by the pulse monitor increased. About ten minutes after Promazine she was quiet. Her breathing was less laboured and she looked comfortable.

At 2-30 p m., she was quiet, looked comfortable, respirations regular and easy and extremeties were warm. No cyanosis, no sweating. Oscillations of the pulse monitor were satisfactory, indicating adequate blood flow in the periphery. Systolic blood pressure

was 95 mm Hg. Pulse was 120 per minute, regular and the volume had improved. Promazine 15 mgm was given intravenously slowly at 2-45 p. m.

At 4-20 p.m. the patient was quiet with a systolic blood pressure of 95 mm Hg. Promazine 10 mgm was given slowly intravenously because the pulse monitor showed a fall in the peripheral blood flow. Thereafter, respirations were regular, no cyanosis, no sweating. Extremeties were warm and the pulse monitor showed satisfactory blood flow. Pulse was 125 per minute volume and full. The patient was fully conscious and rational and comfortable at this stage. She was despatched to the ward with the following instructions:

(i) 1 pint of 5% Dextrose to be followed by 1 pint normal saline very slowly intravenous for the next twelve hours.
(ii) Record and chart, respiratory rate, pulse, systolic blood pressure every half hour.
(iii) Continuous oxygen inhalation via nasal catheter continuous.
(iv) Promazine 10 mgm I.V. slowly at 7 p.m.

The record read as follows:

5-30 p.m. systolic blood pressure 90 mm Hg. pulse 128 p.m. respiration 28 p.m.
6 00 p.m. systolic blood pressure 90 mm Hg. pulse 128 p.m. respiration 28 p.m.
6-30 p.m. systolic blood pressure 90 mm Hg. pulse 128 p.m. respiration 28 p.m.
6-50 p.m. Blood pressure 90/70 mm Hg. pulse 140 per minute.
7-00 p.m. Promazine 10 mgm I.V.
7-30 p.m. systolic blood pressure 95 mm Hg. pulse 136 p.m. respiration 28 p.m.
8-00 p.m. systolic blood pressure 95 mm Hg. pulse 128 p.m. respiration 28 p.m.
8-30 p.m. systolic blood pressure 95 mm Hg. pulse 128 p.m. respiration 28 p.m.
9-00 p.m. systolic blood pressure 95 mm Hg. pulse 126 p.m. respiration 24 p.m.
10-00 p.m. Promazine 20 mgm, Morphine 12 mg n, I.V. slowly.
Patient catheterised and 12 ounces of urine removed.
12-30 p.m. systolic pressure 90 mm Hg. pulse 128 p.m. respiration 24 p.m.
11-00 p.m. systolic pressure 90 mm Hg. pulse 128 p.m. respiration 24 p.m.
11-00 mn. systolic pressure 95 mm Hg. pulse 124 p.m. respiration 22 p.m.
1-00 a.m. systolic pressure 100 mm Hg. pulse 120 p.m. respiration 22 p.m.
2-00 a.m. systolic pressure 100 mm Hg. pulse 120 p.m. respiration 20 p.m.
3-00 a.m. systolic pressure 105 mm Hg. pulse 120 p.m respiration 22 p.m.
5-30 a.m. systolic pressure 110 mm Hg. pulse 116 p.m. respiration 22 p.m.
29-3-67 Morning Temperature 98.8 degrees F.
8-10 a.m. Blood pressure 110/80 mm Hg.
Peristalsis present
Lungs aerating and clear
Passed 10 ounces of urine.

From 29-3-67 she made an excellent recovery without any complications.

The wound healed well and she was discharged home on 16-4-67 with the baby.

COMMENTS:

Anaemia complicating pregnancy is a very common condition one meets. It is an accepted fact that one of consequences of severe anaemia in pregnancy is the ease with which circulatory shock follows slight trauma and haemorrhage. In severe hookworm anaemia complicating pregnancy, fatal obstetric shock following normal delivery was increased in patients with haemoglobin concentration of 30% or less; the degree of shock depending on the severity of the anaemia. This patient was admitted with a haemoglobin percentage of 25. She was treated for this condition and reached a haemoglobin percentage 55 of before delivery. Unfortunately for her, she had post-partum haemorrhage and underwent a major surgical operation. She was transfused 5 pints of blood at various intervals, after delivery and in the post-operative period. As anticipated she was in a state of peripheral circulatory shock at the end of the operation. The causative factors being (a) Anaemia and its effects on the heart leading to circulatory failure, (b) hypovolaemia in anaemic cardiac failure (c) effects of emergency surgery and anesthesia on an anaemic patient, (d) rapid cold blood transfusion.

In the immediate post-operative period she was treated for hypovolaemia and received two pints of cold blood rapidly and one pint of Macrodex. Her state of shock did not respond to blood volume replacement. Morphine and Promethazine did not improve her condition. Between 6-30 a.m. and 12-30 p. m. efforts to resuscitate her failed. Adrenergic amines and hydrocortison were with-held.

She gradually drifted to a state of irreversible shock, not responding to blood volume replacement, narcotics and oxygen inhalations. During the latter stages she was semiconscious and confused, extremeties cold and fully vasoconstricted without any blood flow as shown by the absence of oscillations of the pulse monitor (Cotel-Keating). Blood pressure was not recordable, radial pulse was not palpable. This picture was one of the "classical shock syndrome" in typical cold hypotension. The other features were oedematous hands and feet. She was cyanosed on oxygen.

This irreversible state was detected in time. Assessing the patient's physiological state, there was no doubt that she was in metabolic acidosis arising out of prolonged vasoconstriction. Her laboured breathing and irational behaviour was due to the rise in blood lactate levels.

At this stage dilating the vascular space was considered to be the only line of treatment at the time. No biological measurements were possible in the hospital. Therefore it was taken for granted that the patient was in metabolic acidosis as judged by the clinical state. Promazine (Sparine) was preferred to Chlorpromazine because of less toxic effects. The drug of choice Phenoxybenzime was not available. Fractional doses of Promazine were given until the extremities were warm and a good blood flow shown on the pulse monitor strapped to the tip of the index finger. This state was maintained for a few hours without the addition of narcotics or fluids. Two hours after dilating the vascular space, she showed signs of improvemennt. She was quiet, breathing was not laboured, no cyanosis, no sweating, and a systolic blood pressure of 95 mm Hg. was recorded.

From 2-30 p.m. to around 10 p.m. she was maintained on I. V. Promazine given just enough to keep her extremeties warm when these showed signs of becoming cold.

At 5-30 p.m. she was returned to the ward and it will be seen from the record of the blood pressure, pulse and respiration that

she had she gradually improved. The following morning she had a stable blood pressure of 110/80 mm Hg. Of particular note was, that in spite of a prolonged period of shock, she had passed 22 ounces of urine in under 24 hours.

One of the reasons for a patient drifting into an irreversible state is due to the prolonged vasoconstriction produced physiologically by the patient and pharmacologically by the individual treating the patient. Vasoconstriction in the skin and viscera is a physiological response to blood loss and shock. Vasoconstriction leads to anoxia of the tissues and viscera, anaerobic metabolism of the mitochondria takes place. Blood lactate level rises and a metabolic acidosis results. If this is not corrected the patient eventually dies.

An irreversible state can be recognised when the fluids administered intravenously do not produce any improvement in the clinical condition of the patient. At this stage the patient looks pale and oedematous, a physical sign not seen earlier. The haemodynamics of this condition lie in the altered response of the precapillary sphincters which control the opening and closing of the capillaries to blood flow. The precapillary sphincters controlling the microcirculation open and close in response to metabolic products of glycolysis, and the oxygen needs of the tissues. As metabolic acidosis increases the precapillary arteriolar sphincter looses its tone and does not respond to the normal humoral mechanisms. The sphincter at the venule end retains its tone. Blood and fluids given at this stage accumulate here, engorge the capillaries and fluid seeps into the extracellular space depleating the blood volume.

The rationale of dilating the vascular bed and restoring the blood flow seems the most reasonable solution in resuscitating such patients. Blood flow to the tissues and viscera are improved and maintained. The liver and kidneys are not deprived of oxygen. Aerobic metabolism at the mitochondrial level takes place. In this patient once adequate blood flow was established and maintained she was left to recover gradually by her own physiological processes.

Blood pressure is not an index of blood flow. Shock exists and persists when there is inadequate tissue perfusion. Shock can be attributed to (a) deterioration of cardiac function, (b) inadequate blood volume and or (c) enlargement of the vascular space. By failing to understand the haemodynamic derangement, it is indeed very tempting to reverse the most easly recognised sign, the lowered systolic pressure with adrenergic amines. Although this results in a "temporary improvement", it aggravates the low tissue perfusion and the blood lactate rises.

The use of hydrocortisone is gaining favour in the management of patients in shock and indeed there has been clinical evidence of improvement. The improvement in these patients in shock may be due to the inotropic effect on the heart, an adrenolytic effect or a stabilization of the cell membrane. Hydrocortisone has a place in the treatment of patients in shock who do not show improvement to blood volume replacement. The recommended dose of hydrocortisone is 40 to 60 mgm per Kg body weight, given intravenously, repeating one fourth of this dose every six hours until the desired effect is obtained. It will be seen that the limited finances of a hospital in this country will not permit one to treat even a single patient with hydrocortisone.

One has to consider the transfusion of cold blood rapidly, as another possible cause of persistant shock not responding to blood

volume replacement. Prolonged vaso-constriction and lowered blood pressure may be the result of giving cold blood rapidly. To prevent this state confusing the issue, warming of blood via a very long polythene tubing is recommended. This would not have contributed very much to this patients irreversible state of shock.

The scientific approach in the management of shock requires monitoring the central venous pressure (c v p), treating the lowered cardiac output, measuring the pH, determining the blood lactate level and correcting them with the necessary drugs. This procedure could not be undertaken in this hospital. The only guide to blood flow was the pulse monitor—(Cotel Keating.)

SUMMARY:

The outline of the management of irreversible shock is described. The traditional practice of using adrenergic amines and hydrocortisone is discussed briefly. The modern practice of vasodilation in treatment of circulatory shock is based on the understanding of the haemodynamic derangement. The case demonstrates a simple approach in the management of shocked patients. Scientific methods were not available. A pulse monitor is a useful guide to the state of the peripheral circulation and tissue blood flow.

ACKNOWLEDGEMENTS:

I wish thank Dr. Ratnaike, Obstetrician for permission to report this case history.

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MANAGEMENT OF A CASE OF 'TOFRANIL' (IMIPRAMINE) POISONING

CASE REPORT

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'Tofranil' (Imipramine) is a drug used in the treatment of mental depression, especially as a substitute for electroconvulsive therapy, when the latter is contraindicated for various reasons.

It is available in 25 mgm tablets or as ampoules of 2 ml containing 25 mgm for intra muscular injection. The dose for adults is 25 mgm to 50 mgm t.d.s. Side effects include dryness of the mouth, giddiness, nausea, headache, tremor, fatigue sweating, skin rashes, constipation, urinary retention, agitation and a motor disorder which is seen in elderly patients.

This drug is frequently prescribed and has an attractive colour and if left around the home by careless relatives it could be taken in large amounts by children with resultant poisoning. There is a possibility of encountering such cases in the future.

The following is a report of a case of Imipramine poisoning in a child and its successful management.

Patient S. 1½ years, Male (BHT 15768/1978) of 29-5-67) was admitted to the Children's Ward with the history of having ingested a "large number" of pink tablets which were being taken by another member of the household. This was identified by the Out-door Dispensary and later by the relatives' clinic card as 'Tofranil'.

There was no previous history of febrile or afebrile convulsions or of epilepsy. The child had been leading a perfectly healthy life since birth. On examination, the child was very drowsy, interspersed by sudden bouts of crying, rapidly followed by cramp like spasms and breath holding. Both eyes were rolled up during these attacks.

The BP was 120/90; Pulse rate 140/min and of small volume. The heart sounds were normal.

The lungs were clear. There were no other abnormalties.

A stomach wash was done and the washings were pink in colour and contained undigested grains of rice.

The child was thereafter kept in the 'head low' position. Penicillin 1 megaunit and Streptomycin 0.5 Gm were given by intramuscular injection in order to cover any respiratory complication.

Soon after the stomach wash, the convulsion was repeated; this time there were jerky clonic movements of all four limbs and there was opisthotonos. Both pupils were dilated and fixed. The frequency and

duration of the convulsions rapidly increased and the respirations became jerky and shallow.

Paraldehyde 1.5 ml im was given along with Oxygen by a conical mask. A further dose of Paraldehyde 1.0 ml im was given half an hour later followed by Sodium gardenal 30 mg im after a further half an hour.

As the fits were still not controlled and there were now poor respiratory efforts with secretions in the lungs, the child was referred to the Department of Anaesthesia for further treatment.

The problems were " 1. To secure control of the airway and maintain adequate respiration, especially during the convulsion.

2. To control the frequency and severity of the fits 3. To maintain an adequate circulation and prevent circulatory failure.

The child was transferred rapidly to the operating theatre in the 'tonsil' position (head low, hips raised, lateral) with Oxygen inhalation by mask. In the theatre, the pharynx was cleared of secretions and the child then turned supine. A Magill size 1 endotracheal tube was passed after laryngoscopy and connected to a T-piece adaptor. One limb of the adaptor was connected to a Boyle's machine and Oxygen at 4 litres/min delivered to the patient. By occluding the open end of the T-piece it was possible to inflate the lungs with oxygen and synchronise each inflation with an inspiratory effort of the patient. Intermittent suction of the airways was carried out via the endotracheal tube by a gauge 00 polythene suction catheter.

In order to control the fits, it was at first envisaged to give Thiopentone Sodium iv followed by suxamethonium chloride (Scoline) iv and fully control the respiration. But since Sodium gardenal had been given only a short while earlier and further barbiturate may have led to circulatoty depression, a gaseous mixture of Nitrous Oxide 3/litres/min and Oxygen 2 litres/min was run in from the Boyles machine and assisted respiration continued. The idea was to supplement the effects of Paraldehyde and Barbiturate given earlier with this gaseous Mixture. The fits ceased about 20 minutes after intubation of the trachea. The patient was watched for a further hour after cessation of fits, on Oxygen alone and then returned to the ward.

The temperature showed a tendency to rise but was kept down below 99'F by physical methods. The airway was kept clear by intermittent suction and respiration assisted during the night.

There was a gradual fall in blood pressure from 120/90 to 90/70. A cut down on a limb vein was done and a drip of 5% Dextrose was given (1120ml in 12 hours). In retrospect, we think that the latter figure was in fact the patient's normal BP and the value 120/90 was a raised BP due to the effect of the drug. It was necessary to repeat the Paraldehyde 1.0 ml only once in the night as the child was restless but there were no fits at all.

After about 16 hours of the ingestion of the tablets, the child passed urine. Soon afterwards the child opened its eyes and was able to recognise the mother. Respiration was now normal and as the child was straining on the endotracheal tube and coughing, this was removed after pharyngeal toilet. The temperature was normal.

The blood urea was 18 mg/100 ml and liver function normal. There were no neurological complications.

After further observation, the child was discharged after five days.

COMMENT

In cases of convulsions of this sort, the aim should be to

(1) Prevent anoxic episodes, keeping the airway clear and protected and rendering respiratory assistance, should this be required.

(2) Control the frequency and intensity of the convulsion.

(3) Support the patient's circulation

(4) Control the pyrexia which occur in such cases.

These measures were taken in this case as outlined.

It is much safer to carry out gastric lavage in severely comatose patients after the trachea has been intubated. Even if gastric lavage is carried out in the head low, lateral position, there is a possibility of aspiration of gastric contents and this probably happened in this case. Again during a fit, there is a possibility of gastric contents being forced into the unprotected airway by the violent muscular contractions of the fit. Similar aspiration has been demonstrated during the fasiculations of skeletal muscle after injected Suxamethonium in anaesthesia.

It would have been ideal to give this patient Thiopentone sodium rectally (as suppositories) but these were not available. We had to use Sodium gardenal im.

As to the part played by Nitrous Oxide and Oxygen in this case, it is difficult to assess. Nitrous oxide is a very mild anaesthetic and if it did anything here, it probably acted as a supplementary agent.

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Thanks are due to Dr. M. Visuvaratnam, M.D. M.R.C.P. for calling us in so promptly, Dr. H. A. D. Weerasooriya, M.R C.P. D C.H. for agreeing to the publication of this case and to the nurses of the Children's ward and the Department of Anaesthesia who helped with this case.

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CLINICAL SOCIETY NEWS

Our former assistant secretary, Dr. A. E. A. Joseph has gone on transfer as lecturer in forensic medicine at the University of Ceylon in Peradeniya.

The Annual General Meeting was held in April and the above office bearers were elected. The meeting was followed by a symposium on "Acquired Heart Disease". The following spoke:- Dr. H.A.D. Weerasooria, Dr. S. Anandarajah, Dr. M. Visuvaratnam, Dr. B. A. Mills, Dr. B. W. Rasiah and Dr. S. J. Stephen. A dinner followed at the Palm Court Hotel.

The second meeting was an illustrated lecture on "Renal Hypertension" by Dr. K. Jeyasingham M. S. F.R C.S., Lecturer in Surgery, University of Ceylon.

The third meeting was an interestieg illustrated talk by Dr. S. J. Stephen M.S. F.R.C.S. entitled "A critical review of 100 cases of carcinoma of the oesophagus".

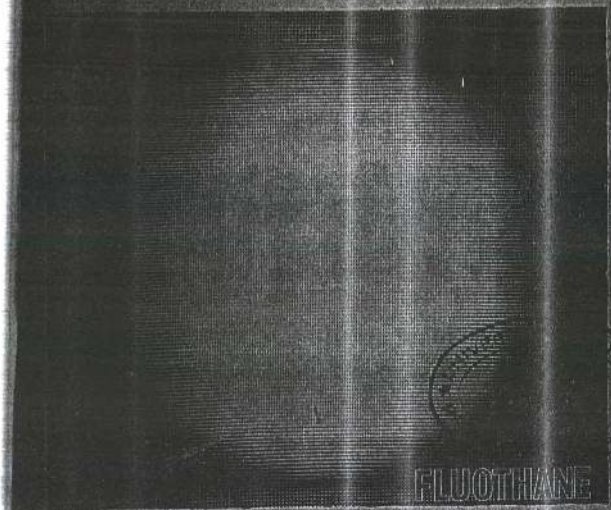
The fourth meeting of the Society was addressed by two guest speakers Dr. P. R. Anthonis F.R.C.S. on "Post-Cholesystectomy symptoms" and Dr. B. S. David M.D.M.R.C.P. on "The horrors of starvation". Both talks were illustrated by slides.

Dr. Anthonis is Surgeon at the General Hospital, Colombo and Dr. B. S. David is Paediatrician, General Hospital, Colombo South. The guests were entertained to dinner at the Jaffna Rest House.

In the early part of the year, the clinical society received a gift of books from the United States Information Service. The books were handed over to the society by Mr. John Russel, Director, U.S.I.S., at a tea party in the Nurses' Lecture Hall. Later Mr. Russel was shown round the Jaffna Hospital. We thank the U.S.I.S. and Mr. Russel for this gift.

We wish to convey our sympathies to Dr. A. Gabriel on his recent bereavement.

M. Ramanathan,
Honorary Secretary.



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