K. Ganeshamoorthy.

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Contents

BENADRYL EXPECTORANT

key factors for effectiveness and patient acceptance

BENARDYL PROPERTIES	BENADRYL BENEFITS
● ANTITUSSIVE	— Effectively brings down cough counts
● NON-NARCOTIC	— Safe for prolonged use
● ANTISPASMODIC	— Relaxes bronchial tree
● ANTIHISTAMINIC	— Relieves distressing symptoms of nasal stuffiness, sneezing watering of eyes
● SURFACE ANAESTHETIC	— Reduces throat irritation through local anaesthetic action

BENADRYL FACTOR

blended with medicaments to provide

Soothing, demulcent, mild anaesthetic action on irritated mucus membrane of the throat

Aids liquefaction and removal of mucus secretions

- ELEGANCE — Rich look; evenly consistent
- TASTE — Pleasant, refreshing unmatched

BENADRYL EXPECTORANT

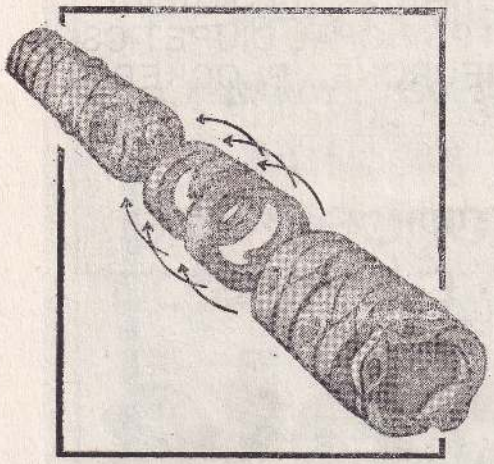
Superior-safe cough reliever

in all age groups

Minipress* (prazosin HCl)

the logical choice

meeting your priorities in antihypertensive therapy



- Effective when used alone
- Effective when used in combination with other agents
- Logical adjunct to beta adrenergic blockers
- Controls blood pressure safely without compromising hemodynamic functions
- Combines long-term antihypertensive efficacy with excellent toleration

Minipress

Convenient Dosage Regimen

Start: 0.5 mg b.i.d. or t.i.o. — Initial dose for all patients †

Progress: 1 mg b.i.d. or t.i.d. — Review and adjust according to response after 4–7 days

Add: thiazide — A thiazide diuretic may be added if the response to dose of 1 mg b.i.d.–t.i.d. is incomplete. It is inadvisable to increase the dose of Minipress beyond 2 mg t.i.d. before adding a thiazide diuretic

2 mg b.i.d. or t.i.d. — Review and adjust according to response after 4–7 days

1-2 weeks††

Final Titration: Usual maintenance dose in most patients will be in the range of 3 to 9 mg daily, given in divided doses b.i.d. or t.i.d.

A few patients will require 12 or 15 mg and in rare instances up to 20 mg daily, divided b.i.d. or t.i.d.

PRECAUTIONS

A very small percentage of patients have responded in an abrupt and exaggerated manner to the initial dose of Minipress (prazosin hydrochloride). Sudden loss of consciousness, lasting for a few minutes or in very rare instances up to one hour, has been reported. This reaction, believed to be due to postural hypotension in abnormally responsive individuals, usually occurs within 30 to 90 minutes of the initial dose and is associated with premonitory symptoms of dizziness, weakness, and sweating. The effect is self-limiting and in most cases does not recur after the initial period of therapy or during subsequent dose titration steps.

This exaggerated response appears to be related to dose but not to severity of hypertension. The clinical experience to date indicates that the incidence and severity of early hypotensive symptoms can be reduced by a few initial dose and cautious increases during the first one to two weeks of treatment.

As when instituting therapy with any effective antihypertensive agent, the patient should be advised how to avoid symptoms resulting from orthostatic hypotension and what measures to take should they develop. The patient should be cautioned to avoid situations where injury could result should dizziness or syncope occur during the initiation of Minipress (prazosin hydrochloride) therapy.

† The doctor is advised to consult full prescribing information.

†† Dosage should usually be adjusted at intervals of one to two week. More rapid adjustments can be made when quick reduction of pressure is indicated.

*Trademark

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Editorial

Dwindling Resources and Rising Expectations

For the past twenty years at least the public of this country has been assailed with the news that we have the best medical service in Asia. The majority being denied the opportunity to travel abroad have been in no position to make any assessment but it has been the common lot of many to be told that treatment cannot be given, or operations carried out for shortages and deficiencies of various sorts. This certainly holds for the hospitals outside Colombo, and it may even hold for those in the capital. These deficiencies exist not due to wanton neglect, but on account of the rising cost of drugs, hospital equipment and consumable items. To compound matters there has been an exodus of trained medical personnel, starting with the doctors in the mid sixties as a trickle, soon to become a flood. Later the nurses and ancillary medical personnel such as laboratory technicians and radiographers followed suit leaving a void as no substantial increase in the training grades had been achieved and today shortages of every variety exist in the medical services. In the last three years two new medical schools were established in this country increasing the intake of students from 250 to 400, but even if they should all graduate, is there any guarantee that these new doctors will man the posts in the department? It is time then, to take stock and implement measures that will attract people to stay in the hospital and health services.

The most serious discrepancy is one of finance, affecting both the Exchequer and the public servants. The relatively low salaries in this country and a rapidly rising cost of living with an inflation rate of approximately 50% makes it virtually impossible to exist without an additional source of income, and only a handful enjoys that privilege. It is not a question of luxuries, but of bare necessities that confront the personnel in the hospital service and can one expect the long hours of financially unrewarding work that is essential for the success of hospital treatment from these individuals if they must be concerned about the next meal for the children at home? On the otherhand, the salaries not only in the affluent west and the oil rich Gulf States but even in most countries of Africa and the Pacific are five, ten, twentyfold or more than those paid here, and it is not unreasonable for a man who finds it impossible to provide just adequate living for his family to consider employment abroad. One has only to consider the high salaries paid to the United Nations Volunteer Doctors working in this country to bring the matter closer to home. It does not seem that there will be a shortage of such situations for the next twenty years at least and hence unless there is a substantial increase in salaries the exodus is almost certain to increase. But it is not only financial considerations that results in this exodus. Dissatisfaction with working conditions, amounting almost to frustration that prevents those skilled personnel in the health service from using their skills and becoming mere ciphers in the bureaucratic machine is equally important and the exodus is greatest from the provincial hospitals where facilities of all kinds are notoriously short.

With the intention of increasing our medical output two new medical schools were established in the last three years but one cannot impart knowledge by legislation. Every medical student is aware of the better facillities in Peradeniya and Colombo and who can blame them for wanting their share of the cake? Being taught as they are in the euphemistically titled Teaching Hospitals where dictum, but not the example is to be followed, for they are often at variance, and always to the patients' cost and aware through the literature of a different world of medicine and medical education abroad surely even the most optimistic planner will realise that the fledgling graduates are liable to follow the beaten path.

The remedy is no easy task. Funds from the Exchequer are limited. However their utilisation can be used to better purpose. Facilities in the four medical schools and their associated hospitals in the way of buildings, personnel and equipment must be upgraded to a standard of medical education and patient care that is in keeping with the needs of the society and the aspirations of the personnel in the service. The tutory style of education so common in universities in Asia breed mediocrity, and worse still deny the brighter students of the opportunity to progress. It then goes without saying that these four centres should encourage research, both basic and clinical, and should be the referral centres for the hospitals within their perimeter. Naturally it will cost money, but it will be money well spent for the four medical university centres will provide the training ground for both under—and postgraduate students and will also provide the attraction of posts of distinction. None of this can be achieved overnight but instead of drifting it is time to consider the necessity for a National Health Policy and plan for its phased implementation.

As far as Jaffna is concerned the shortcomings in the "Teaching Hospital" are so numerous that even the stoutest must blanche at their recitation. The spirit is wlling but can it withstand the ravages of frustrated skills? Not for long. It is time for a definite plan for the development of the health services centred round a new Jaffna General Hospital which will function as the main teaching centre for under—and postgraduate students in this region. That word is used in a purely geopraphical sense for one hopes that medical students and doctors from all parts of this country will choose to work in the Jaffna General Hospital of the future.

INTENSIVE CORONARY CARE

K. S. de Silva MD (Cey.), FRACP., MRCP., MRCPE. [1]

DEVELOPMENT

CORONARY heart disease (CHD) causes up to one third of deaths in the adult male in Western societies and a means of reducing this devastating death toll is urgently needed. Ideally the main thrust of effort in this field should be directed at the primary prevention of the underlying pathology, coronary atherosclerosis. Much research in this area has uncovered the relevance of various coronary risk factors in its aetiology which is now considered multifactorial, and the near certainty of further aetiological factors yet to be discovered is apparent. It is also agreed that efforts at primary prevention should be instituted early in life, possibly in early childhood before atherosclerosis has progressed to any degree. Until prevention of the disease becomes a practical possibility, the best hope to reduce the death rate from CHD would clearly be to provide optimal care after the episode of acute myocardial infarction (AMI) has occurred and thereby salvage lives.

Prior to the advent of Coronary Care Units (CCU's) in 1962, the mortality of patients suffering AMI and fortunate enough to reach the hospital alive was 30-35%. With specialised CCU care this mortality has now been reduced to 10-15%f The benefits of providing for a specialised area within the hospital catering purely for AMI patients and nursed by specially trained staff became apparent when more information concerning the disease itself became available. A survey in 1961 (Melzer et al.) showed that the majority of deaths (47%) in the acute phase were due to arrhythmias, while 43% of deaths were due collectively to shock and cardiac failure. Only 10% were due to embolism and cardiac rupture. It was also known that ventricular fibrillation (VF) was the cause of 80% of arrhythmic deaths, and Zoll in 1956 had already demonstrated that VF could be terminated by an externally delivered electric shock. It was left to Kouvenhoven it al. in 1960 to devise the simple procedure of cardiopulmonary resuscitation (CPR) by closed chest cardiac massage and mouth to mouth ventilation before the concept of intensive coronary care became a reality. It was reasoned by its first proponents that arrhythmic deaths could be reduced and hopefully prevented by the continuous observation of AMI patients in a special facility by specialised personnel, with facilities for urgent defibrillation immediately at hand. Developments in the electronic field and the advent of continuous monitoring equipment brought the plan to reality, and the implementation of the scheme became finally possible with the realisation that specialised nurses rather than doctors could be relied upon to identify and interpret arrhythmias, and then act upon their judgement in the emergency situation.

THE BASIC SCHEME

The basic components of the intensive coronary care scheme consist of the following :

1. The patient who is admitted with suspected AMI to a specialised CCU facility.

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[1] *Consultant Physician.*

2. The attachment of monitoring equipment to the patient and the establishment of an intravenous line.
3. The specialised nurse in constant attendance, whose function it is to monitor the patient's progress closely, identify warning arrhythmias and other adverse features, take emergency action as dictated by her own judgement and keep her medical superiors abreast of the patient's progress.
4. The Physician who although not constantly present, is kept informed of patient events by the CCU nurse and assumes overall responsibility for the patient's care.

There has been continuing debate regarding patient selection to the CCU, and more recently several studies (e. g. Hampton's in Nottingham) are reported to show that satisfactory home care is feasible in certain selected subgroups of patients who are considered to be at good risk. The consensus of opinion and the author's personal view is that this reasoning is fallacious, since the course of AMI being so unpredictable, a good risk patient can only be identified in retrospect. It is common experience that a perfectly stable patient on admission may quite unexpectedly develop a life-threatening arrhythmia at any stage in his progress, although admittedly, complex ventricular tachyarrhythmias are more common in extensive infarctions and early in the course of AMI. The prudent policy would therefore be the admission of all patients suspected of having sustained AMI as far as the CCU bed situation permits, priority being given to the younger age groups rather than the elderly. It is inevitable that with this policy some 30% of admissions would be found ultimately to suffer from conditions other than AMI (usually severe angina or the intermediate coronary syndrome). The duration of care in the CCU in most units averages 3-4 days but longer stays are inevitable when complications arise. Since late hospital deaths are a source of significant worry, the ideal situation would be a system of subacute coronary care in an area adjoining the CCU. This facility is not available in most hospitals and recourse must be made to admission to general medical wards in most cases. If facilities are available, the ambulant patient may continue to be monitored telemetrically with transmission of data to a central monitor in an effort to detect unexpected arrhythmias.

Acute coronary care is essentially a system of specialised nursing. It can rightfully be stated in fact, that the morbidity and mortality of patients in CCU's is in direct relation to the quality of nursing provided and the competence of its nursing staff. Nursing duties and responsibilities include the continuous assessment of the patient's clinical state by direct observation and ECG monitoring, one of the fundamental objectives of the latter being the anticipation of serious rhythm disturbances by detecting warning arrhythmias (see later). Depending on specific CCU policy, certain standing orders concerning emergency drug administration may be carried out in these circumstances and emergency defibrillation performed in the appropriate situation. Every nurse, and indeed all personnel concerned must be proficient in the technique of cardiopulmonary resuscitation to maintain the circulation until corrective measures are applied in the event of cardiac arrest. In addition to their specialised duties, the CCU nurse must provide high level general nursing care, and identify and manage emotional problems so common in AMI patients by providing for a calm, unhurried and confident atmosphere, in addition to constant reassurance to allay the fears and misapprehensions of patients under their charge. It cannot be emphasised too strongly that no other

element of coronary care is more important than the role of the specialised CCU nurse.

PRACTICAL MANAGEMENT

Details of this subject are beyond the scope of this review and only some basic principles of care during the acute coronary attack will be discussed.

The mortality from AMI is highest in the early hours following the onset of the major symptom (50% mortality in 2 hours). Ideally therefore, ECG monitoring should begin in the patient's home as soon as the diagnosis is made and should continue during his transport to hospital. The patient should be kept under constant observation until his admission to a CCU, the prerequisites during this phase being pain relief, reassurance, and the detection and treatment of rhythm disturbances. On arrival at the emergency area in hospital, admission procedure should be such that the patient is immediately transferred to the CCU without any delay en route.

Pain Relief

Morphine still appears to be the analgesic of choice in most AMI patients. It is best given intravenously in most cases with severe pain, and may be repeated as necessary. It may be combined with Atropine in cases with bradycardia. Hypotensive reactions may be countered by raising the foot of the bed, and vomiting, by the administration of anti-emetics. Pentazocine (50-60 mg. I.M. or slowly I.V.) is a useful alternative but carries the relative disadvantage of raising pulmonary vascular resistance, although its potential for use by paramedical personnel parenterally in the pre-hospital phase probably outweighs this. The anxiety-allaying action of opiates is an undoubted benefit. Another approach in the management of continuing pain is the slow I.V. administration of beta-adrenergic blocking drugs (e. g. Propranolol in a dose of 0.1 mg./kg. body weight given slowly), and contraindications to its use must of course be excluded. This method instituted early in the course of AMI has been found effective in limiting infarct size experimentally and has been advocated in the clinical situation. It is clearly effective in the relief of cardiac pain in addition. Oxygen therapy is also useful in this context and is routinely used in most centres during the first 48 hours. In cases of unstable angina (acute coronary insufficiency) not responding to intensive medical management including aggressive beta-blocker therapy, it has been the practice particularly in certain centres in the U.S.A. to proceed with intra-aortic balloon pulsation, emergency coronary angiography, and if the circumstances are deemed feasible, emergency coronary artery bypass surgery (CABS). This aggressive attitude is expected to improve morbidity and mortality in an unstable situation and salvage viable myocardium.

Cardiac Arrhythmias

Cardiac rhythm disturbances encountered during the early stages of AMI may be rapid or slow, intermittent or persistent and either atrial or ventricular in origin. In the CCU there are two principal reasons for treating them: a) because they may be associated with haemodynamic deterioration leading to possible increase in infarct size, or b) because they may presage more serious and potentially lethal dysrhythmias. Studies by Lown et al. have shown the significance of certain forms of ventricular ectopic activity in foretelling the development of more serious and complex ventricular tacchydysrhythmias. Their suppression when identified is obviously indicated. The contrary philosophy, is the prophylactic administration of anti-arrhythmic drugs in all patients admitted with AMI, in view of the recognised fact that some 25% of primary

ventricular fibrillation (VF) occurs unheralded without any preceding warning signs. This concept has led to the development of prophylactic drug regimes by Lie et al. using I.V. Lignocaine, and by Anandaraja using I.V. followed by oral Disopyramide (personal communication), amongst others. It would appear that their results in the prevention of primary VF are equally, if not more, impressive.

1. Ventricular ectopic beats (VEB's).
   These are of almost constant occurrence in AMI. VEB's conforming to the Lown criteria (more than 5/minute salvoes, multifocal VEB's and R-on-T VEB's) should be suppressed. Lignocaine is the drug of first choice for this purpose, given as an I.V. bolus of 1 mg./kg. body weight followed by an I.V. infusion of 1-4 mg./minute. Smaller doses are used in cardiac failure and the elderly. Pharmacokinetic studies by Harrison et al. suggest that a second bolus at 30-45 minutes helps to maintain a therapeutic plasma level of the drug ab initio and thereby prevents breakthrough arrhythmias. Other drugs which may be used include Mexiletine (a Lignocaine analogue), Procainamide, beta-blocking drugs, and Disopyramide.
2. Ventricular Tachycardia (VT).
   Initial therapy depends on the presence or absence of haemodynamic deterioration. If associated with cardiovascular collapse or severe hypotension, immediate defibrillation is required. Otherwise, drug treatment on the lines described for the treatment of VEB's may be instituted. In all cases predisposing factors, e.g. hypoxia, acidosis and hypokalaemia should be corrected.
3. Idioventricular Tachycardia.
   This relatively benign dysrhythmia is now recognised to be quite common in AMI. It is non-paroxysmal in nature and the idioventricular focus discharges at a relatively slow rate of 50-120/minute. It may either be observed without the institution of specific drug therapy, or the attack may sometimes be terminated by accelerating the sinus pacemaker with I.V. Atropine.
4. Ventricular Fibrillation (VF).
   Treatment is immediate unsynchronised DC shock with a 400 joule discharge in the setting of cardiac arrest. A second shock may be given if the first is unsuccessful. Thereafter if reversion has not been achieved, CPR is instituted and Lignocaine 100 mg. by I.V. bolus together with I.V. Sodium bicarbonate (e.g. 100 meq. stat.) is given. Further attempts at electrical reversion are made, Lignocaine and Sodium bicarbonate being continued as necessary. A beta-blocker I.V. may be useful in resistant cases, and occasionally success may ensue by converting fine VF to coarse VF by I.V. Adrenaline 1 mg. and repeating the DC shock.
5. Atrial Arrhythmias.
   Occasional atrial ectopic beats (AEB's) are of no consequence. Frequent AEB's may presage atrial fibrillation (AF) and may be treated with Digoxin Disopyramide, a beta-blocker, Procainamide or Quinidine. Atrial flutter is rather uncommon and responds often to Digoxin and a beta-blocker, although a low energy DC shock (10-100 joules) is more effective. Atrial fibrillation is often paroxysmal and tends to terminate spontaneously. Its occurrence is often an adverse prognostic feature, and AF is recognised to be associated with older age, atrial overload due to cardiac failure, atrial infarction and acute pericarditis. If

haemodynamic embarrassment is evident, reversal with DC shock is indicated. Otherwise the ventricular response may be controlled with Digoxin and/or a beta-blocker.

6. Bradyarrhythmias.

These are commonly associated with inferior wall infarcts, and a significant sinus bradycardia of under 45-50/minute may be treated cautiously with incremental doses of I.V. Atropine since bradycardic rhythms are potentially unstable. First degree A-V block does not require treatment, and second degree A-V blocks (Mobitz Type I) often respond to Atropine, as does complete A-V dissociation complicating inferior infarcts. An Isoprenaline infusion in low dosage may be employed with benefit in the latter situation. Unresponsive blocks especially if complicated by Stokes-Adams' episodes, severe hypotension or cardiac failure or much ventricular ectopic activity, require temporary transvenous endocardial pacing. This prophylactic measure would usually be instituted earlier in His-Purkinje blocks complicating extensive anterior wall infarcts, although there is some doubt whether the ultimate prognosis in these patients is materially improved. Bilateral bundle branch block, bifascicular block (e. g. RBBB with left anterior fascicular block), and other E.C.G. evidence of trifascicular disease would commonly constitute indications for prophylactic transvenous pacing.

Left Ventricular Failure (LVF) and Cardiogenic Shock

More than 90% of the total mortality from AMI is now due to advanced LVF and cardiogenic shock, since sudden arrhythmic deaths are now effectively combated with present methods. Present approaches which are now making some headway in the control of pump failure include its early detection by a combination of clinical, electrocardiographic, radiological and haemodynamic methods, improved concepts in therapy based on knowledge gained from haemodynamic assessment, the treatment of cardiogenic shock with intra-aortic balloon counterpulsation (IABC), and appreciation of the importance of trying to limit infarct size. The advent of the Swan-Ganz catheter has brought haemodynamic monitoring to the bedside. Its use however, is restricted to well equipped and well staffed units with a high turnover of patients, so that sufficient experience is gained with the technique. The possibilities of technical error and resulting fallacious data are high when the method is used by the occasional enthusiast. It is recognised that the most useful estimate of left ventricular function is the left ventricular end-diastolic pressure (LVEDP). The Swan-Ganz catheter enables an indirect assessment of this to be gained by estimating diastolic pressures in the main pulmonary artery and in the wedged position. Studies by several workers have shown that the LVEDP is elevated in the majority of AMI patients. Sequential studies have also demonstrated that changes in left heart filling pressures may be predicted by parallel changes in an elementary E. C. G. measurement, namely the P terminal force. These P wave alterations measured in the supine position in lead V1 from a standard 12 lead cardiograph, provide a useful, simple and non-invasive tool for assessing LV function in these seriously ill patients, and since they occur even earlier than conventional clinical and radiological methods, more effective therapy becomes possible. Insight gained from haemodynamic monitoring methods has also rationalised the drug therapy of acute LVF in these patients. Vasodilator therapy, utilising drugs acting on the arterial and venous sides of the

circulation reducing impedance to LV ejection and LV preload respectively, has revolutionised therapy in acute LVF, especially in the situation of papillary muscle rupture causing acute mitral incompetance (MI). Drugs used for this purpose include I.V. agents (Sodium nitroprusside, Phentolamine) and oral drugs (Prazosin, Isosorbide, Trinitrin) and-ideally their administration should be monitored by estimations of systemic arterial pressure, cardiac output and LV filling pressure. LVEDP measurements also confirm the diagnosis of hypotension and shock-like states due to hypovolaemia, which requires controlled I.V. infusion for its correction. Interventricular septal rupture complicating acute anterior wall infarction and papillary muscle rupture leading to acute MI must be identified. The prognosis in general is poor in these cases, and is only improved if the acute situation could be stabilised sufficiently until surgery is performed in early convalescence.

Routine treatment is instituted with I.V. Frusemide, fluid and salt restriction when LVF supervenes, but the status of digitalisation is controversial. It is of undoubted benefit when AF is present, but there is debate presently whether the drug is indicated in sinus rhythm, particularly in the long term. Positive inotropism is undesirable in AMI in view of its potential for infarct extension. Regardless of the therapy employed, review of all drugs and in particular Digitalis should be undertaken at follow up, since prolonged therapy with this drug may in fact be needless after compensation is regained.

The status of routine anticoagulation is also debatable. Definite indications for doing so would include leg vein thrombosis, pulmonary or systemic embolism or a previous history of these conditions, cardiac failure, and cardiogenic shock. Extensive varicose veins would also probably be a relative indication. Prophylactic therapy (e.g. subcutaneous Heparin 5000 I.U. 8 hourly) could be stopped when the patient is mobile and long-term anticoagulation is no longer recommended. The importance of adequate hydration and leg movements while in bed, should not be underestimated.

IABC is the technique that has gained the widest acceptance as far as mechanical circulatory assistance is concerned, in the management of cardiogenic shock. The mortality still remains distressingly high with a poor long term prognosis, whatever measures are undertaken. The limitation of infarct size is a commendable goal since long term prognosis depends largely on the amount of remaining viable myocardium. Attention to the details of management outlined previously will help to stabilise the patient with acute myocardial ischaemia and thereby limit the extension of infarction. Specific modalities include the early administration of I.V. beta-blocking drugs.

REHABILITATION

This should commence while the patient is in the CCU, by continued reassurance and providing the patient with an understanding of his condition, and later by advice and discussion concerning realistic goals. Early mobilisation in uncomplicated cases is recommended, walking being allowed after his return from the CCU to subacute care, and discharge from hospital being considered about 10-12 days after admission. The presence of complications would obviously prolong the patient's stay. A more recent innovation is the performance of a low level exercise stress test prior to discharge and at intervals thereafter, the results of which go far towards influencing therapy. The limits of prescribed exercise both isometric and dynamic, could be determined by objective criteria, and a rational basis for anti-arrhythmic therapy directed towards

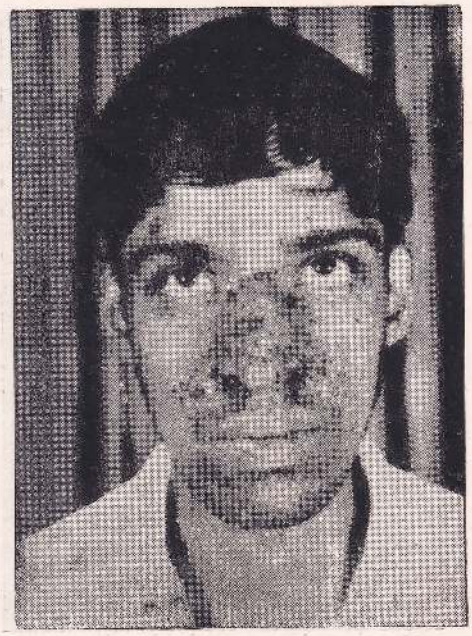
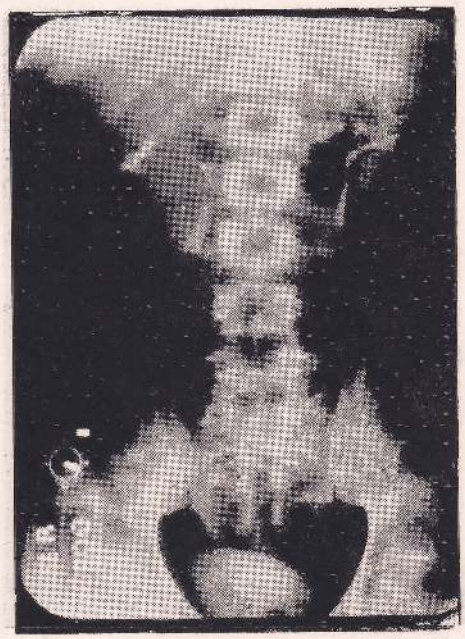


Figure 1



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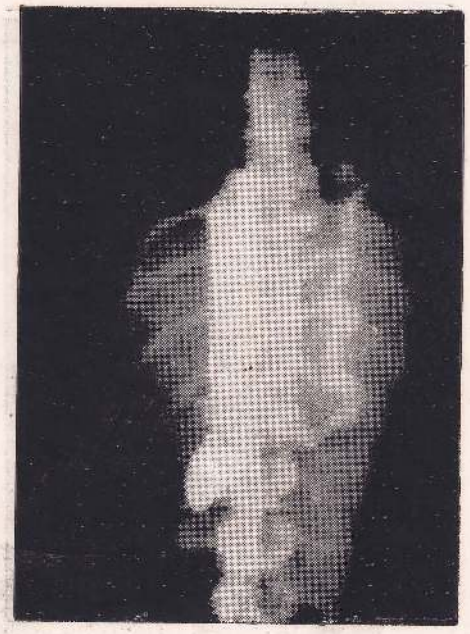

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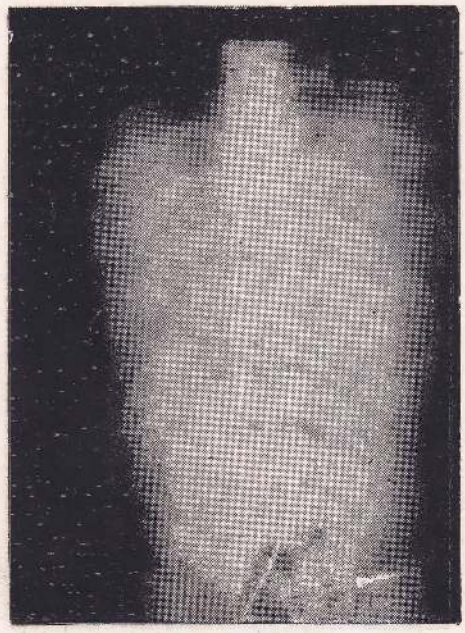

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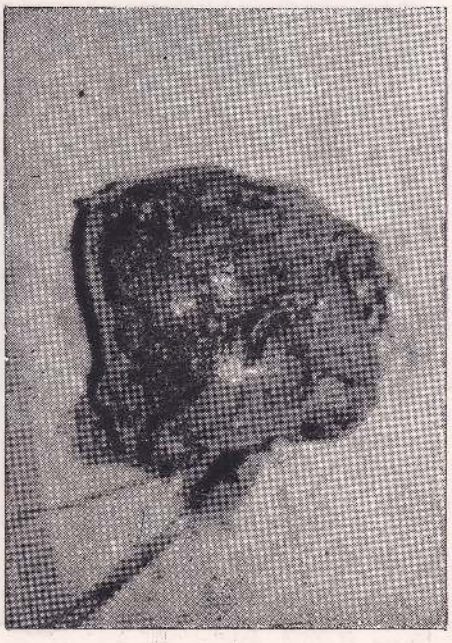

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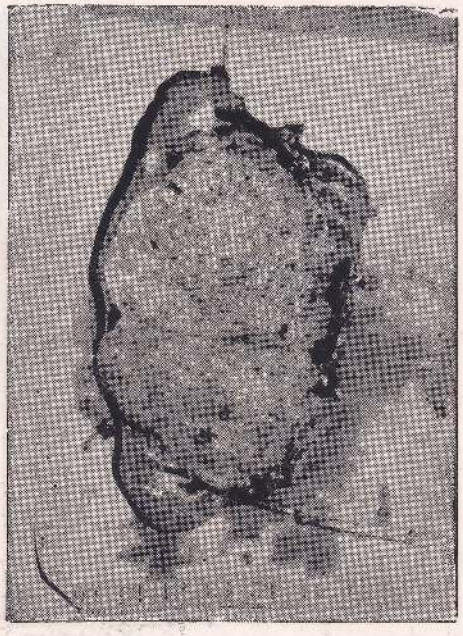

Figure 6

exercise-induced arrhythmias would be available. Not the least advantage of post-AMI stress testing, is the demonstration to the patient (and his spouse) of his ability to undertake physical exercise without problems to a degree perhaps greater than was previously considered possible, a realisation of undoubted psychological benefit. Graded physical retraining under gymnasium conditions is in vogue in many centres, and although conclusive evidence concerning the reduction in long term mortality is lacking, its undoubted psychological benefits and morale boosting potential are apparent.

Post-AMI mortality is highest in the first year (15%), and recent studies show that continued therapy with beta-blocking drugs go far towards reducing this as far as the incidence of sudden cardiac death is concerned. Similar results have been obtained with the anti-platelet uricosuric drug Anturan in a dose of 200 mg. q.i.d. for the first seven months. The incidence of non-fatal reinfarction however does not appear to be significantly influenced by these drugs. It has been demonstrated in several recent trials that Asprin therapy does not provide any significant benefit in this regard. Attention to coronary risk factors seems obviously important even after the event, but stopping smoking has been the only factor to receive statistical confirmation in reducing post-AMI mortality (especially sudden cardiac death) in data derived from large scale studies. Control of blood pressure seems obviously important, and the use of beta-blocking drugs for this purpose would be logical in view of its other advantages in this context. Common sense dictates that an optimum body weight should be maintained, but no real evidence exists that strenuous attempts at normalising serum lipids either by means of strict dietary measures or lipid lowering drugs confers any special advantages. A moderate life style with reasonable levels of physical activity should be encouraged, and the continued care of uncomplicated patients should be the responsibility of the family doctor Specialised after-care would be necessary when complications either exist or supervene, and then, conditions requiring further investigation and perhaps surgical treatment should be sought. These would include increasing post-infarction angina, intractable ventricular dysrhythmias and cardiac failure, the latter two conditions being sometimes secondary to surgically correctable ventricular akinetic areas or aneurysms.

RENAL HAMARTOMA (ANGIOMYOLIPOMA) WITH TUBEROUS SCLEROSIS

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Hamartomatous renal lesions are frequent in patients with tuberous sclerosis, a disease characterised by the classical clinical triad of mental retardation, epilepsy and adenoma sebaceum. However, unless the surgeon is familiar with the neurocutaneous manifestations of tuberous sclerosis and its association with renal hamartomas, the true nature of the renal lesion is often misdiagnosed as a carcinoma of the kidney, until the histopathological examination has been completed. (Olurin et al, 1971). Although renal hamartomas have been well documented in the literature, we wish to report this case as it presented several unusual features not often described in the literature.

Case report:- H.P.V.P., a 24 year old male presented to the Colombo General Hospital in November 1973, complaining of a progressively enlarging painful lump in the right upper abdomen of about 2 years duration. He had experienced intermittent mild upper abdominal and right flank pain for over 8 years. He had several bouts of fever with chills, and noticed some nocturnal frequency and dysuria but no haematuria. His weight had remained steady and there were no symptoms referable to his cardiovascular or respiratory systems or the gastrointestinal tract.

His past history was significant in that he had epileptiform fits from the age of 4 years, the fits occuring quite frequently upto the age of 10 years after which he had no fits. A nodular rash on the face distributed over the nose, cheeks and the nasolabial folds, was present from childhood and had become progressively worse over the years (Fig. 1). The patient had started schooling only at 12 years of age and left school after completing grade 5.

His parents were alive and well and there was no history of epilepsy or adenoma sebaceum in them. A younger brother of the patient is said to have had fits in childhood, while the youngest brother had died in infancy following a convulsive seizure.

When seen in November 1973, he was a well nourished male of below average intelligence. There was no oedema of the feet or lymphadenopathy. The jugular venous pressure was not raised. The nodular erruption on his face was present. The B.P. was 130/80 mm Hg. The heart and lungs were clinically normal. Abdominal examination revealed a nodular, hard, ballotable, non-tender mass in the right lumbar region, extending from the costal margin forwards to almost the umbilicus. The liver was palpable 2 finger breadths below the costal margin but was smooth and non-tender. Free fluid was present in the abdomen. There were several prominent tortuous veins coursing upwards on the anterior wall of the abdomen and the chest. Bilateral varicoceles were present.

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There were *cafe-au-lait* patches scattered all over his body.

His haemoglobin was 8.2 gms%, PCV 33%, RBC 3,730,000 cmm, WBC count was 8,400 with a differential count as follows:- Neutrophils 35%, lymphocytes 30%, and eosinophils 35%. The serum electrolytes were normal and the blood urea was 50 mgm%. Serum Calcium was 9.9 mgm%. The ESR was 12mm in the first hour. His blood group was O Rh negative, as it was with all the surviving members of his family. The total serum bilirubin was 1.3 mgm/100 ml, thymol turbidity 5 units and zinc sulphate turbidity 30 units. Urine culture was sterile, and the electrocardiogram was normal.

A plain radiograph of the abdomen revealed a soft tissue shadow in the right upper abdomen. The chest X-ray was normal. The intravenous urogram showed a normal left kidney. The appearances of the right side were 'those of a large renal cyst' (Fig. 2) Quite in contrast to this, the selective renal angiogram revealed a large, very vascular, space occupying lesion, involving the lower pole and the hilar regions of the right kidney (Fig.3) In view of this finding and the clinical features of inferior vena caval obstruction, the renal lesion was thought to be a hypernephroma, and in order to assess the extent of inferior vena caval invasion, a cavogram was performed. This revealed a dilated right renal vein emptying into a grossly dilated inferior vena cava. There were other dilated collateral veins seen in the film (Fig. 4). Several attempts at getting the Seldinger catheter into the right atrium failed due to an obstruction encountered at the level of the diaphragm. Close scrutiny of the cavogram films, however, showed no space occupying 'tumour' tissue within the lumen of the vena cava. A sleletal survey revealed no obvious metastatic deposits, but showed areas of patchy sclerosis in the skull, lumbar vertebra and the iliac bones.

On 5-1-1974 the right kidney was explored via a thoraco-abdominal approach There were large venous channels on the chest and abdominal walls and on the surface of the diaphragm. The peritoneal cavity contained a large quantity of straw coloured fluid. A large, rather knobbly tumour was found to occupy the middle and lower parts of the kidney with oedematous cellular tissue beneath Gerota's fascia. There was no tumour tissue to be felt within the lumen of the renal vein or of the inferior vena cava, both of which were grossly dilated. A solitary, hard, pale yellow 'nodule' about 2.5cm in diameter was found on the inferior surface of the right lobe of the liver. A radical nephrectomy was carried out. No attempt at excision of the liver 'nodule' was made, since it was felt at that time, that the prognosis would not be altered by such a procedure. The post-operative course was uneventful and the patient left hospital on the fourteenth postoperative day. When seen six weeks later he was quite well but still had dilated collateral veins on the chest and abdominal walls.

Histopathology:- The tumour measured 15cmx 12.5 cm x 10cm and weighed 730 grams. (Fig. 5). On bisection the tumour was well cicumscribed and showed a speckled cut surface with whorled areas, bluish white in colour and firm and fibrous in consistency. (Fig. 6). There were no friable or necrotic areas and no haemorrhages. Histologically, the tumour showed an admixture of adipose tissue, smooth muscle, sheets of collagenous connective tissue and numerous blood vessels showing distortion of their lumina. There were no features of malignant change in either the cells or their nuclei. The line of demarcation between the renal parenchyma and the tumour was well defined and there was no infiltration of the kidney by the

tumour. The appearances were those of a hamartoma (angiomyolipoma) of the kidney. (Figs. 7 & 8).

Following the histological report, a search was made for other manifestations of the tuberous sclerosis complex. Examination of the ocular fundi revealed a unilateral high myopia in the left eye, with a large pale excavated optic disc, consistent with the developmental defects associated with tuberous sclerosis. There were no phakomata. The EEG showed no definite abnormality of ictal significance Further skeletal x-rays revealed irregular thickening of the cortices of the tibia, the metacarpal and metatarsal bones. There was a single cystic lesion in the proximal phallanx of the left index finger. (Fig. 9) A liver scan (antero-posterior projection) showed generalised enlargement of the liver and of the spleen. Histological examination of a 'nodule' from the facial erruption showed sebeceous glands and excessive number of hair follicles, compatible with adenoma sebaceum.

Discussion.

Tuberous sclerosis is an uncommon congenital disorder first described by Bourenville and Brissaud in 1880. It derives its name from the potato-like tumours, composed predominantly of neuroglial tissue, found in the brains of those affected by this malady. In the fully developed tuberous sclerosis complex described by Moolten (1942), similar lesions are found in other organs and tissues such as skin, heart, lungs, liver, eyes, kidneys, and pancreas. Bones are frequently involved in the process. The majority of reported cases have been from mental institutions. In the European institutions for epileptics and feeble minded, Ross and Dickerson (1943), estimated the incidence at approximately 0.6%. Both sexes appear to be equally affected, the age incidence varying considerably.

Mental retardation and epilepsy are the commonest neurological manifestations of tuberous sclerosis and are the result of the diffuse gliosis found in the brain. These lesions occassionally undergo calcification and are sometimes demonstrable radiologically. The striking cutaneous manifestation is adenoma sebaceum, a greasy nodular erruption with a butterfly distribution over the nose, cheeks and the nasolabial folds. Cafe-au-lait patches, shagreen patches and subungual fibromata are other cutaneous manifestations. In about 50% of cases multiple small nodules, the so called "rhabdomyomas" may be found in the heart and these may give rise to electrocardiographic abnormalities and even symptoms of heart failure. The lungs often contain multiple cysts. Our patient presented several neurocutaneous manifestations but there were no obvious cardiac or pulmonary lesions. The characteristic ocular lesions in tuberous sclerosis are phakomata which are round or elliptical, white or grey areas, usually found separate from the optic disc. (Moolten 1942). Other ocular changes include optic atrophy and perioptic nerve scarring. The unilateral high myopia and the large pale excavated disc seen in the left eye of our patient are changes sometimes seen in patients with tuberous sclerosis. Osseous manifestations of tuberous sclerosis have been well described by Beck and Hammond (1957). These usually take the form of sclerotic areas associated with the presence of small cysts in the phalanges, as was demonstrated in our patient. The other organ most affected is the kidney.

The renal lesions, as well as those found in other affected organs and tissues are essentially hamartomatous tumours. The term hamartoma was coined by Albrecht in 1904 to denote "tumour like malformations in which there is only abnormal mixing of the normal components of the

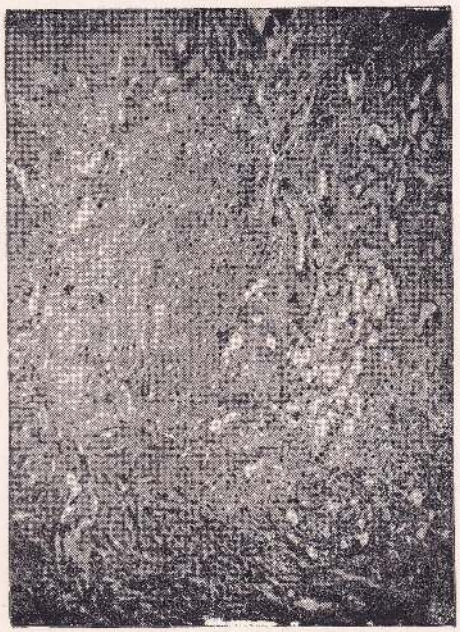


Figure 7

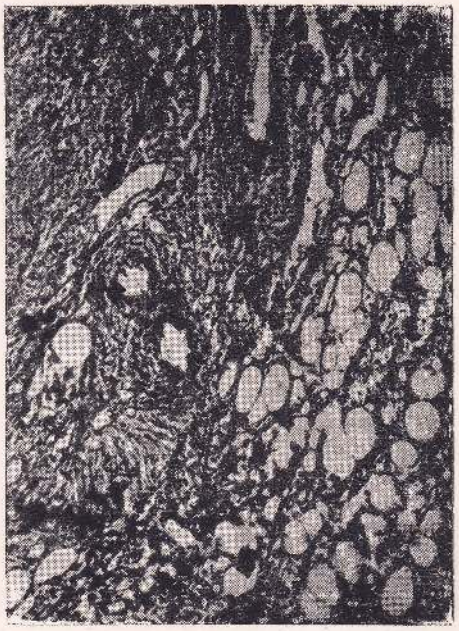


Figure 8

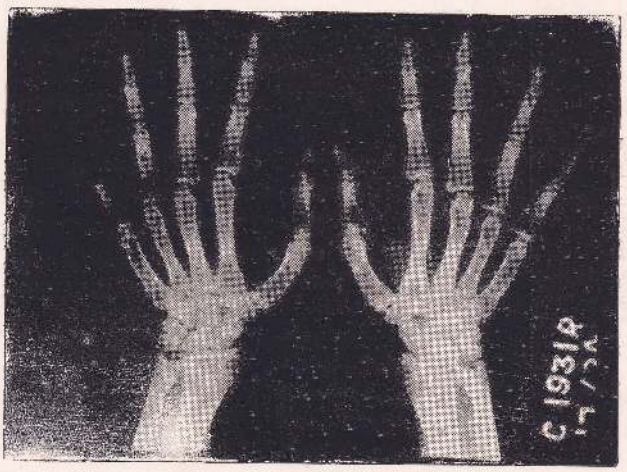
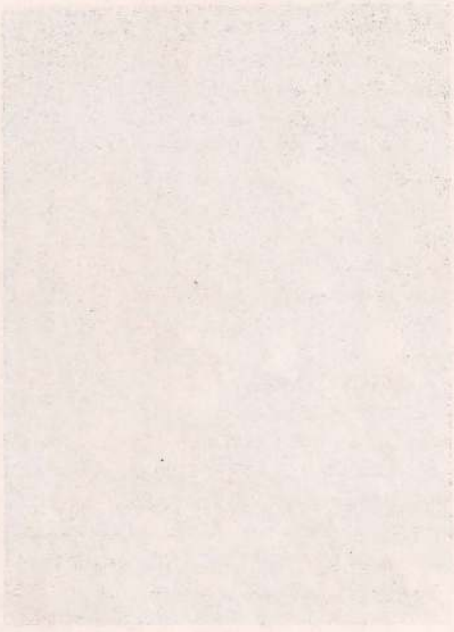
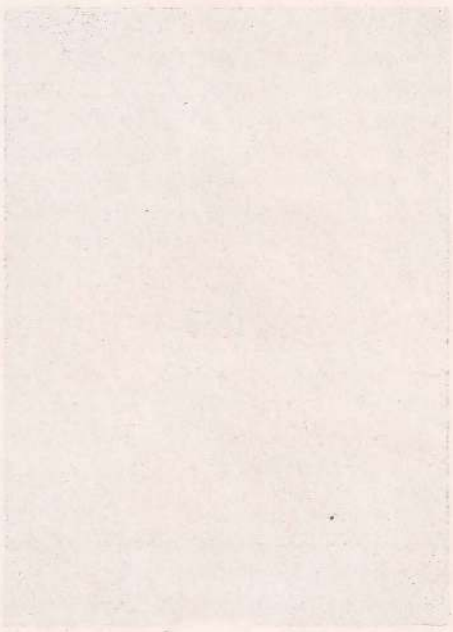
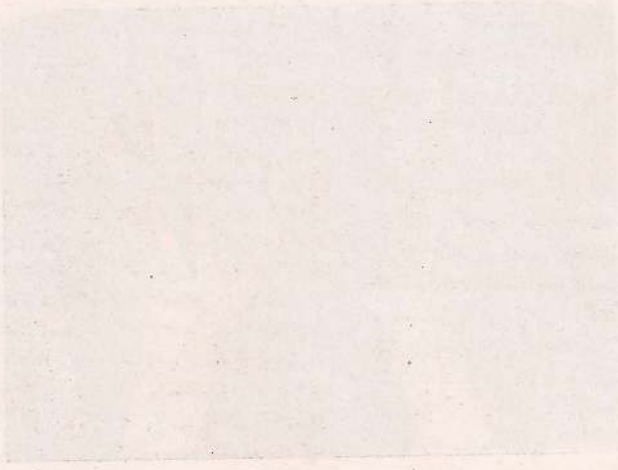


Figure 9







organ in which they occur". They have been reported in most organs of the body, the lung been the most common site.

Hamartomatous renal lesions have been found to occur in two different clinical situations. Those found in association with the tuberous sclerosis complex and those which are seen independent of any manifestations of tuberous sclerosis. When they occur in association with tuberous sclerosis, these tumours are usually small, bilateral and asymptomatic. They rarely reach clinically significant proportions, since most of these patients succumb in their early lives to intercurrent infections, status epilepticus, intracaranial or visceral tumours or sudden heart failure. Le Brun et al (1955) in their survey of the literature, found an incidence of 60%, while according to Critchley et al (1932), they occured in 80% of patients with tuberous sclerosis. However, tuberous sclerosis as such is rarely encountered in surgical practice and unless the surgeon is familiar with its clinical manifestations, the diagnosis can easily be missed.

In the much rarer situation, where renal hamartomas occur independent of tuberous sclerosis, the tumours usually reach symptomatic proportions, are frequently unilateral and solitary, though smaller lesions may be present within the kidney. Some sf these patients, however, have been known to develop the stigmata sf tuberous sclerosis long after the diagnosis of the renal hamartoma.

The commonest presenting symptom of these patients with hamartomatous renal lesions, have been abdominal or loin pain, of varying grades of severity, and either intermittent or continuous. Other symptoms include abdominal swelling loss of weight, haematuria, anaemia and rarely circulatory collapse and shock from haemorrhage which may be intratumoral, intraperitoneal or retroperitoneal. The signs of inferior vena caval obstruction, as was present in our patient, have, so far as we are aware, not been described in the literature. Although these tumours present no characteristic clinical features to differentiate them from other renal tumours, Price and Mostofi(1965) feel that those cases presenting with an abrupt onset of severe abdominal or flank pain, associated with either a tender palpable mass or with tenderness without a mass in the kidney region, especially when combined with radiological evidence of a renal tumour, should be strongly suspected of having a renal hamartoma.

As with clinical features, there are no characteristic urographic or angiographic appearances peculiar to renal hamartoma. The clinical and radiological features referable to renal hamartoma are very much like those seen in renal carcinoma and other abnormalities such as polycystic disease, lesions which are not infrequently associated with tuberous sclerosis (Inglis, 1954)

Angiomyolipoma is one of several varieties of hamartoma that may be found in the kidneys of patients with tuberous sclerosis. Peron and Gray (1960) describe cases with tuberous sclerosis in which there were hamartomas in the kidney, liver and the pancreas, in addition to the glial nodules in the brain. On the basis of these findings we believe that the 'nodule' found in the liver of our patient which we mistook for a metastatic deposit, is a separate hamartomatous lesion.

Histologically these tumours are non encapsulated and are composed of a mixture of fat, smooth muscle, and b.lood vessels. Whether they are truly benign or possess malignant potential remains unsettled. While it is generally considered to be a benign lesion (Allen and Risk, 1965; Le Brun et al, 1955; Price and Mostofi (1965) there are some who feel that these tumours can undergo malignant change (Riches

1964). There are very few cases on record where venous invasion has been found. Peron and Gray (1960) found one case in which the tumour had invaded and caused rupture of a large intratumoral vein, while Berg (1955) reported one with invasion of the renal vein. However, Price and Mostofi (1965) in a review of 129 cases found no evidence of venous invasion in any of their cases. In the case we report, there was both clinical and angiographic evidence of inferior vena caval obstruction, yet at operation we could find no invasion of either the inferior vena cava or the renal vein, by the tumour. The caval obstruction appeared to be purely extrinsic in origin and in the light of the finding of a probable second hamartoma in the liver, and the persistence of the collateral veins despite the nephretomy, we are inclined to postulate the presence of a third hamartomatous lesion in the liver causing the caval obstruction. However the possibility of a congenital constriction of the inferior vena cava cannot be ruled out as similar developmental defects such as subaortic stenosis, subnormal development of the aortic arch and cardiac defects have been described in association with tuberous sclerosis (Moolten, 1942).

Most patients with these tumours usually undergo nephrectomy, since they are not always correctly diagnosed preoperatively. However, where the diagnosis is suspected clinically, treatment should be conservative, wherever possible, since they have a clinically benign course. Their removal becomes imperative when by virtue of their size, the patient is unable to carry on a normal life, or they give rise to complications such as spontaneous haemorrhage threatening life. Where conservative surgery is undertaken, wide excision should be carried out, since local recurrence is known to occur with incomplete excision. Immediate frozen section biopsy, as suggested by MacQueeny et al (1964), seems a more rational procedure when conservative surgery is contemplated. The prognosis is good when only one kidney is involved. However, if the tumours are bilateral, multiple and large, they often carry a poor prognosis from eventual renal insufficiency.

Summary

A renal hamartoma with tuberous sclerosis in a 24 year old male, is described. Several unsual features not commonly encountered with this condition have been emphasised. The literature available to us has been briefly revieved.

Acknowledgements

We wish to thank Dr. Leslie Jayasuriya, FRCS, consultant surgeon, for allowing us to publish details of the patient admitted under his care.

Figure 1: Photograph of patient's face showing the characteristic adenoma sebaceum distributed over the nose, cheeks and the nasolabial folds.

Figure 2: Intravenous Urogram showing the appearances of a large right renal cyst and a normal left kidney.

Figure 3: Selective right renal angiogram showing a large very vascular space occupying lesion involving the lower pole and the hilar region.

Figure 4: Inferior Vena cavogram showing the grossly dilated IVC and dilated collateral channels.

Figure 5: Photograph of the tumour after removal.

Figure 6: Cut section of the tumour showing clear demarcation from the rest of the renal parenchyma.

Figure 7: Microscopic appearance of the tumour showing an admixture of adipose tissue, smooth musle, connective tissue and blood vessels.

Figure 8: Microscopic appearance under a higher magnification showing clearly the adipose tissue, blood vessels and conective tissue.

Figuve 9: Radiograph of both hands showing a single cystic lesion on the proximal phalanx of the left index finger.

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IDIOPATHIC SPLENOMEGALY IN SRI LANKA

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Summary

Eight patients with gross splenomegaly without obvious cause together with their clinical, haematological and radiological findings are described. Three were from areas where malaria was endemic. The liver histology revealed absence of hepatic sinusoidal lymphocytosis and lymphocytic infiltration of the portal tracts although Kupffer cell hyperplasia was seen in two patients. Scintiscan confirmed that the splenic enlargement was due to reticulo-endothelial hyperplasia. Available evidence points to a splenomegaly of undetermined aetiology.

Introduction

Splenomegaly of variable proportions is not uncommon in tropical countries. In recent years there has been considerable interest in the Tropical Splenomegaly Syndrome (TSS) which consists of the following: persistent marked splenomegaly in adults, raised serum levels sf IgM and a positive response to prolonged antimalarial therapy. There is no single feature either in the liver or spleen which can be regarded as specific but a combination of hapatic sinusoidal lymphocytosis (HSL) and reticulo-endothelial hyperplasia are usual. Nagaratnam et al (1973) described two patients with TSS in Sri Lanka.

Eight patients with large spleens without HSL and other characteristics of TSS and without any other known cause to account for splenomegaly are presented.

Patients and Methods

Eight patients with large spleens (more than 7 cms below the left costal margin) and no obvious cause were studied. The study included history and physical examination. Routine haematological methods were used in the determination of haemoglobin, haematocrit, white blood cell count, platelet count and blood film smears. Other investigations included plasma proteins and empirical liver function tests and transaminases. Electrophoresis for haemoglobins revealed no abnormal haemoglobins. Marrow aspirations from the sternum were stained with Leishman's stain. Iliac crest biopsy was done in three patients. Serological studies for malarial antibodies were done in six patients. Liver biopsy specimens were obtained with a Vim Silverman needle after appropriate blood coagulation studies. Spleno-portogram was done in four patients, chest radiograms in all and Barium swallow for oesophageal varices in four. A scintiscan using Technitium colloidal sulphur was done in two patients.

Results

Clinical Profile: There were five males and three females. Their ages ranged from 41-62 years with a mean of 52 years. Three were from malarial areas and of them two had malaria. There was no history of alcoholism, haematemesis or jaundice. Nutrition was good in all except two (Cases 1 and 3). All were afebrile when first seen, five presented with abdominal discomfort, and distension. Two had breathlessness on exertion and ease of

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fatigue. One had repeated attacks of left hypochondrial pain due to perisplenitis.

The liver was palpable in all but one, ranging from 2 cms to 7 cms (mean 4.5) The consistency was firm, the surface smooth and non tender. The spleen was palpable in all varying from 7 cms to 17 cms (mean 9.5) below the left costal margin firm and non tender except in one, where it was tender during the attacks of perisplenitis. There were no clinical signs of portal hypertension nor stigmata of chronic liver disease.

Laboratory Findings: Haematological: The haemoglobin values ranged from 5.2-13.0 gm/100ml (mean 9.5). The haemocrit 19-36%. The total white blood cell count ranged from 1,750-8,000/cmm. All except one had a normal differential count (Case 1). The platelet count varied from 92,000-410,000/cmm. The reticulocyte count was below 1% in all. Malarial parasites were not seen in blood smears. The marrow was normal in six, hypocellular in one and hypercellular in the other. Erythropoiesis was normoblastic in all. No lymphocytic infiltration was seen in any. Free iron was seen in one. The erythrocyte sedimentation rate was below 12 mm/hr in all except in one where it was 120 mm/hr. This patient was admitted with a history of fever with a cold of 3 days duration. The serum bilirubin was normal in all (below 0.8 mg/100ml). Routine liver function tests revealed no abnormalty. The plasma proteins ranged from 6.6-7.8gm/100 ml with a normal albumin/globulin ratio. The malarial antibody determination was weakly positive in Case 3 (1/40) and Case 4 (1/20) and negative in the other four.

Histological Appearance: The histology of the liver was normal in five. Two had slight fatty change with Kupffer cell hyperplasia and the other haemozoin pigment. The lobular architecture was maintained in all. There was no sinusoidal

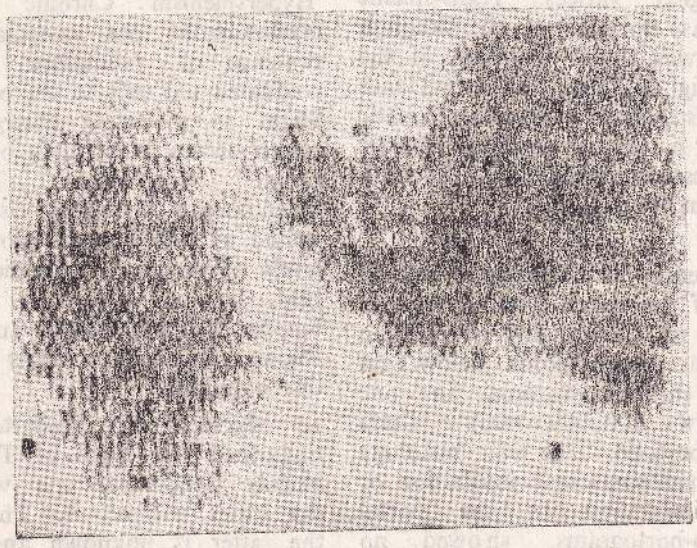

Fig 1 Spleno-portogram. There is no evidence of portal obstruction. The intra hepatic portal vascular pattern is not that of cirrhosis. The portal vein is wider than normal but this is probably a physiological widening to accocodate the flow.

Fig 2 Scintiscan. Shows hepatomeagly, with diffuse concentration of radioactivity throughout the liver Marked enlargement of the spleen.

lymphocytosis and the portal tracts were normal.

Radiological: Teleradiogram of the heart showed no enlargement of the cardiac shadow in any of the patients. The lung fields were clear. Barium swallow revealed no oesophageal varices. Trans-splenic portogram was normal in all although in one there was slight dilatation of the portal vein (Fig 1). There was no evidence of portal obstruction. The intrahepatic portal vascular pattern was normal. Hepatic scanning indicated concentration in both liver and spleen (Fig. 2).

Discussion

Eight patients with moderate to marked enlargement of the spleen without obvious cause showed no abnormality other than anaemia in four (Haemoglobin below 8gm 100ml) and thrombocytopenia in one (Platelets below 100,/000/c.mm). There was no reticulocytosis. The liver histology revealed no hepatosinusoidal infiltration or lymphocytic infiltraiion of the portal tracts. Spleno-portograms showed no abnormality. Scintiscan depicted high concentration of radioactivity in the spleen indicating the splenic enlargement was due to reticulo-endothelial hyperplasia rather than to congestion. Three of the patients were from malarial areas of whom two had had attacks or malaria.

Large spleens associated with anaemia, reticulocytosis, leucopenia and thrombocytopenia are usually ascribed to 'Hypersplenism'. Chronic leukaemia or myelosclerosis are associated with the presence of premature leucocytes and nucleated red cells in the peripheral blood. Slight to moderate enlargement of the spleen occurs in cirrhosis of the liver with portal hypertension. The Tropical Splenomegaly Syndrome is often characterised by raised serum globulin, increased levels of malarial antibodies, elevation of IgM, hepatic sinusoidal lymphocytosis, Kupffer cell hyperplasia and hypertrophy. Malarial infection is highly associated with this condition.

Marsden and Hamilton (1969) distinguished two types of Tropical Splenomegaly Syndrome, one with HSL and the other without it. The aetiology of the latter is unknown and it occurs in malarial areas. Boyer et al (1967) suggested that non-cirrhotic portal fibrosis may be responsible. It was reported in Hong Kong (Cook et al., 1963) and

Calcutta (Basu and Aikat, 1963) that gross splenomegaly presented a problem not yet clarified. Severe liver disease is common in these areas and there is evidence to suggest that splenic enlargement may preceed the hepatic disorder.

The correlation between lymphocytic infiltration of the hepatic sinusoids and splenomegaly is not invariable. Even in malarial areas TSS occurs without HSL. In New Guinea HSL was seen in some villagers with splenomegaly and also in other villagers without splenomegaly.HSL was found in many of the liver biopsy specimens in Uganda and New Guinea (Marsden et al. 1967). Sagoe (1970) demonstrated that lymphocytic infiltration was not limited to the liver but also involved the peripheral blood and marrow of patients with this syndrome in Nigeria. In our cases the marrow and blood showed no increase in lymphocytes and the liver histology was normal.

In this study, although the immunoglobulin levels and the response to antimalarials were not determined, other features sf TSS were lacking. Thus there exists in Sri Lanka, patients with marked splenomegaly the cause of which is not well determined.

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ACUTE INVERSION OF THE UTERUS CASE REPORT

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Acute inversion of the uterus, though rare, is a life saving emergency. About 75% of all the inversions are due to mismanagement of the third stage of labour and are preventable. The incidence in the United Kingdom is one in 27,000 deliveries, in the United States of America one in 20,000 deliveries and in India one in 8500 deliveries. The mortality in acute inversion is over 40% and increases when the time interval between inversion and reposition increases. Prompt diagnosis and early repositioning of the uterus are of utmost importance to avoid complications and thereby save the life of the patient. Any delay in repositioning brings about neurogenic shock, due to excruciating pain caused probably by the pull on the ovaries and the infundibulo — pelvic ligaments, haemorrhage from placental site and increase in bulk sf the inverted uterus due to entrapment of fluid.

Case Report

A 30 year old multigravida was admitted to the obstetric ward, General Hospital, Jaffna, at 7.10 a.m. on 12-7-80 with a history of retained placenta after a full term normal delivery of her third baby at 5.30 a.m. in a Maternity Home. Her two previous pregnancies had been normal. On admission, the patient was in a state of shock, but not collapsed. Her tongue was pale with a low volume pulse, rate of 110 per min. and systolic pressure 70 mm. of mercury. The diastolic blood pressure was unrecordable. The heart and lungs were normal. The abdomen was soft and the usual smooth, round, well contracted uterus was not felt. Instead, a tender dimpled mass was felt at the level of the pubic symphysis. On gentle pelvic examination the placenta was found to be hanging out, partly separated, and was removed with ease. The uterus was found to be inverted into the vagina. A diagnosis of Stage II Acute Inversion of the uterus was made and immediate steps were taken to combat shock, alleviate pain, and to replace fluid loss. Hydrocortisone 1000mg i.v. and morphine 1/4 gr. was given. Asaline infusion was started. At 7-30 a.m. when the patient was taken to the operating theatre, the was of low volume and 120 per min. with a systolic blood pressure sf 50 mm. of mercury. At 7-40 a.m. the systolic blood pressure was 60 mm. of mercury when blood transfusion was started. At 8-10 a.m. just before general anaesthesia it was 70mm of mercury. Under general anaesthesia manual correction was attempted first, and a successful repositioning was achieved by a combination of manual and hydraulic methods. With the right hand in the uterine cavity ergometrine 0.5 mg. i.v. was given and the hand was removed when the uterus was hard and well contracted A syntocinon infusion of 10 units (2%) in one pint of normal saline was started. The proceedure was completed in twenty minutes. At 8-50 a.m. the systolic pressure was 90 mm. of mercury and diastolic pressure still not recordable. At 9.00 a.m. the blood pressure was 100/60 mm. Hg. and the patient's condition improved. At 4.00 p.m. the pulse was 86 per min. and blood pressure 90/60 mm. Hg. which maintained until discharge. She had crystalline Penicillin 1 mega unit b.d., Stretomycin 1 G daily and a dose of Tetanus Toxoid were also given. The

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next day she developed a temperature of 100.2 °F which subsided on the third day. She had no other complications and was discharged on the fifth day on ampicillin for a further two days.

Discussion:

A fundal insertion of the placent and atony of the uterus are two causative factors for acute inversion of the uterus. Atony may occur in normal labour or when some inhalation anaesthetics such as chloroform or halothane are used. Under these circumstances, pulling on the cord, pressing on the fundus, a wrongly applied Credes' method and sudden increase in intra abdominal pressure after the second stage may precipitate inversion. A short cord, or a cord wrapped round the neck several times may produce the same effect in a precipitate labour while the patient is standing. Spontaneous inversions have been reported.

Three stages have been described in the process of inversion. Stage I where the fundus bulges into the uterine cavity but does not protrude through the os; Stage II where the inverted fundus protrudes through the cervix and lies within the vagina; Stage III where the inverted body passes completely through the os and lies outside the vulva; the latter is easily diagnosed but the prognosis is bad. Stage II is the commonest of the presentations.

The diagnosis does not present much difficulty when a careful and complete examination is carried out. The uterus in such cases will be felt in the lower abdomen, almost at the level of the pubic symphysis with a tender dimple at the fundal area. On pelvic examination., which is essential, a large uniform lump is felt with the cervix all round its base. This may be differentiated from a prolapse after partus, where the os is at the centre of the lump with blood oozing out, and the absence of the cervix at the base of the lump. Rarely a large fibroid polyp is mistaken for inversion. In inversion the general condition of the patient is not affected immediately and this is the most appropriate time to reposition it. But the patient's condition deteriorates rapidly due to shock and haemorrhage and she usually proceeds into a state of collapse. Unexplained collapse or shock in a post partum patient may be due to acute inversion of the uterus.

Acute inversion diagnosed at the time of occurence should be repositioned immediately by the manual method, or by a combination of manual and hydraulic methods. Morphine 1/4 gr. intramuscularly or 1/6 gr. intravenously is a valuable aid before manipulation. If late, and the patient's condition is not satisfactory, as in this case, any attempt at inflicting pain, including a pelvic examination, may be dangerous and repositioning should be carried out under general anaesthesia. The methods available for repositioning are: 1) manual method, 2) O'Sullivan's Hydraulic method, 3) Bimanual repositioning at laparotomy, 4) Incision of the cervical ring vaginally and 5) incision of the cervical ring at laparotomy. As a pre requisite, in all these methods,the placenta is stripped off before attempting repositioning. In the manual methodthe uterus is grasped in the hand, with the inverted fundus resting on the palm and squeezed back into its proper position. The part inverted first is replaced last. Counter pressure is maintained upon the abdomen with the other hand throughout the manoeuvre. O'Sullivan's method involves the filling of the vagina with a warm antiseptic solution or warm isotonic saline therby increasing the fluid pressure in the vagina. This is achieved by pushing the terminal end of the tube up to the posterior fornix, after filling the Douche can and tubing with the warm sterile solution, with the

help of the hand inside the vagina and making the vaginal introitus airtight. The introitus is made airtight by approximating the two labia minora anteriorly over the hand inside the vagina. Care is taken to see that the Douche can is full until end of the manoevure so that no air is trapped in the system. The can is raised to a maximum of one metre above the level of the patient. Once the hydraulic pressure inside the vagina is raised, the uterus goes back to its porper position. At laparotomy the round ligaments are drawn, with Allis forceps, in stages towards the inverted fundus until the uterus is repositioned which is aided by manipulation vaginally. By the vaginal approach, the bladder is dissected and pushed up by the Spinelli method, the ring and lower part of the inverted uterus (i.e. the part near the cervix) are incised and repositioning achieved. This may also be done by the Kustner method where the cervical ring is divided posteriorly. At laparcto my the cervical ring is incised by Haultain's procedure where the incision is made in the posterior part and repositioning done. The incised part is stitched up once repositioning is complete. Ergometrine 0.5 mg. intravenously given once repositioning is complete, in all the above methods.

As the majority of the inversions are due to mismanagement of the third stage, any attempt to remove the placenta either by controlled cord traction or Credes' method is made on a well contracted uterus. Therefore ergometrine should always be given immediately after the second stage, irrespective of the expulsion of the placenta, as a retained placenta is not so dangerous or urgent as an acute inversion of the uterus.

A recurrence after repositioning may be due to incomplete repositioning and a recurrence in the succeeding pregnancies has not been reported. Above all, prevention eleminates the risk of serious consequences.

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CEPHALIC TETANUS

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Summary

Two cases are reported of cephalic tetanus which presented with unilateral facial weakness and signs of generalised tetanus. The facial involvement was characterized by the presence of paralytic as well as spastic features, which helped in differentiating it from other causes of facial palsy. Views on the pathogenesis of this unusual condition are discussed.

Introduction

Since the first description of the clinical entity of cephalic tetanus by Sir Charles Bell in 1836, there have been several reports of this condition from various parts of the world (1, 2, 3.) Vakil *et al* (1) defined cephalic tetanus as a variety of tetanus associated with paresis or palsies of one or more of the cranial nerves. Involvement of the muscles supplied by the facial nerve has been the commonest manifestation, (1, 4) although third, fourth, sixth and twelth cranial nerve palsies have also been reported (3, 5).

We report two cases of cephalic tetanus seen within a period of four months at the General Hospital, Kandy. In these patients, the predominent feature was the presence of a unilateral facial palsy.

CASE REPORTS

Case **1.** A 12 year old boy was transferred from a Base Hospital to a surgical unit in Kandy, on 26-9-1977, following a penetrating injury in the left supra-orbital region, caused by a fall on a pointed piece of wood the previous day. The wound was about 4 cm deep and heavily contaminated. On admission the patient was drowsy and restless, but no other neurological abnormalities were found. The fundi were normal, there were no cranial nerve palsies and the tendon reflexes were symmetrical with flexor plantar responses. There was no neck stiffness. Kernig's sign was negative. The other systems were clinically normal.

Active immunization against tetanus was initiated, along with systemic antibiotics namely penicillin, chloramphenicol and sulphadiazine. Local trauma to the eye was treated with topical chloramphenicol. On 29-9-77, he was less drowsy but was found to have neck rigidity. Two days later the patient developed trismus, abdominal rigidity and generalised rigidity of limbs. However the most striking finding was a left sided lower motor neurone facial weakness. A lumbar puncture performed with a view to excluding menginitis produced normal cerebrospinal fluid (C.S.F). Serial skull radiographs failed to reveal any basal fractures.

A tentative diagnosis of tetanus was made and treatment commenced with diazepam, chlorpromazine and anti-tetanus serum (A.T.S.). A gastrostomy was performed to facilitate feeding,. However he went on to develop generalised spasms during the next 24 hours. Respiratory embarrassment caused by repeated spasms necessitated tracheostomy. His condition further deteriorated with development of

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tachycardia and he succumbed to the illness two days later.

Case 2. A 14 year old boy was transferred from a District Hospital on 25-01-78, following a scalp injury in the left frontal region caused by a stone, eight days previously. There had been no loss of consciousness or bleeding from the ears or nostrils. On the fourth day following injury he developed difficulty in opening the mouth, drooping of the left upper eye lid, pain and stiffness in the back of neck. He was transferred with a provisional diagnosis of tetanus. On admission he was alert and conscious. The trismus was marked and in addition he had a *risus sardonicus*, mild neck rigidity and abdominal rigidity. The striking abnormality was a left sided lower motor neurone facial palsy as evidenced by absence of voluntary wrinkling of the forehead, inability to close the eye fully, rolling of the eye ball upwards on attempted closure (Bell's sign) and deviation of the mouth to the opposite side. (Fig. 1) Despite these paralytic features, the left eye was half closed at rest. He appeared to have spasm of the facial muscles on the opposite side as shown by increased supra-orbital wrinkling and a prominent naso-labial fold, giving the impression of a 'partial risus' The rest of the nervous system including the ocular fundi was normal. There were no abnormalities in the other systems clinically. Examination of C.S.F. and skull radiography revealed no abnormality. Treatment was commenced with A.T.S. diazepam and chlorpromazine. The antibiotic used was crystaline penicillin. There was no necessity for wound toilet as the wound had healed satisfactorily. This clinical picture remained unchanged for a week and he then began to show signs of improvement.

Electromyographic (E.M.G.) studies were carried out one week after admission and were repeated a week later. Facial muscles were sampled using a concentric bipolar needle electrode and the action potentials were amplified and dis-

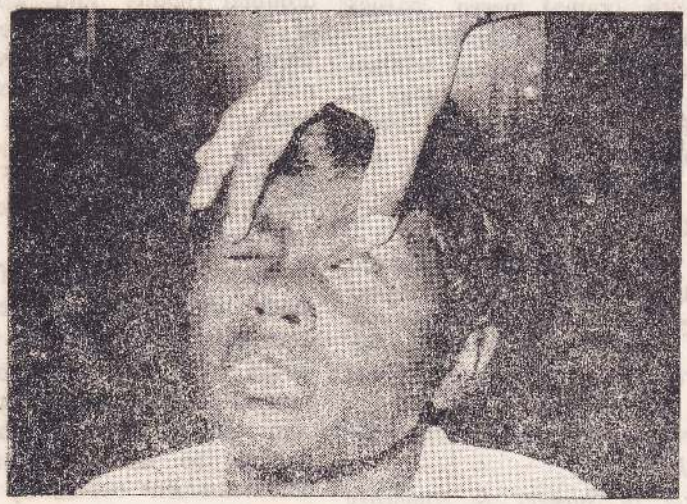

Fig. 1. Case 2 showing facial weakness on the left

played on an oscilloscope with simultaneous auditory monitoring. The striking abnormality was continuous resting motor unit activity in the facial muscles on both sides. Muscles sampled from the limbs and the trunk too showed similar resting motor unit activity. No fibrillation or fasciculation potentials were seen, but on the left side of the face where the paralysis was evident, the recruitment pattern on maximum voluntary contraction was reduced. The facial nerve conduction time measured by stimulating the facial nerve anterior to the tragus and recording the action potentials from the *orbicularis oris* revealed no difference on the two sides. An attempt to study the effect of facial nerve blockade on the E.M.G. was not successful due to technical reasons.

Discussion

Despite active immunization programmes, tetanus continues to cause considerable morbidity in many parts of Sri Lanka. While the generalised disease continues to be the most frequent and easily recognised, several variants have been described. Cephalic tetanus is one such variant, which is considered to be rare, the reported incidence being less than 1% of all cases of tetanus (6). It usually follows wounds of the head, face and neck (4). In both our patients the culpable injury was confined to the upper region of the head and to the side on which the cranial nerve palsy was observed. In the first patient, a basal fracture or meningitis damaging the facial nerve had to be considered in the differential diagnosis. In the second patient, exclusion of a Bell's palsy was the diagnostic difficulty. However subsequent development of the classical features of generalised tetanus, the normal C.S.F. and lack of radiological evidence of skull fractures facilitated the diagnosis.

The unusual feature of the facial palsy was the presence of paralytic as well as spastic features in the muscles affected. This phenomenon was more apparent in the second patient who showed marked ptosis presumably due to increased tone in the *orbicularis oculis* muscle on the paralysed side. However, on volition the patient was unable to close the eye completely, and on attempted closure the eyeball was seen to roll upwards, a sign of lower motor neurone facial palsy. As stated earlier, the same muscles showed continuous motor activity at rest. Electromyography failed to show any features of denervation such as fasciculation or fibrillation potentials. This again helped to exclude a pure paralytic lesion such as Bell's palsy or a traumatic facial palsy. The continuous resting motor unit activity observed in our patient is a recognised feature of tetanus (7). Vakil *et al* (1) have made observations similar to ours in cases of cephalic tetanus.

Differing views have been expressed as to the pathogenesis of the facial weakness in tetanus. These include mechanical compression of the seventh nerve in the stylomastoid canal under the influence of tetanus toxin (8, 9), affection of the end organs of nerve fibres by the toxin (10), neuritis due to tetanus toxin (4) and inactivation of the anterior horn cells of the facial muscles (11). However many of these theories fail to explain the combination of spastic and paralytic features, which happens to be the cardinal feature of cephalic tetanus.

In an attempt to explain this complex phenonenon, Vakil *et al* suggest interference of motor unit activity at the central synaptic level involving both facilitatory and inhibitory pathways. Whatever the pathogenesis may be, cephalic tetanus has presented some interesting diagnostic problems. These two cases

reemphasize that the presence of a motor cranial nerve palsy, seventh nerve palsy in particular, in a patient suspected of having tetanus should not necessarily detract from a definite diagnosis of tetanus. It is worthwhile to consider the possibility of cephalic tetanus in patients who present with motor cranial nerve palsies particularly when associated with neck rigidity and other ill defined muscular symptoms.

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POST STREPTOCOCCAL ACUTE GLOMERULE-NEPHRITIS—A REVIEW

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This review attempts to high light recent developments in the study of Post Streptococcal Acute Glomerulo Nephritis (PSAGN).

The streptococcus. The structure of the cell wall is important in its precise identification. Division of beta haemolytic streptococci into 18 Groups (A-H, K-T) is based on the serologic reaction to the group specific polysaccharide found inside the outer protein layer. In the outer protein layer two antigenically different proteins are identifiable M and T and these are used to sub type the organism. Streptococci are identified by their cultural appearances on blood agar and grouped and typed on their agglutination reactions dependent on their cell wall carbohydrate and protein (M and T) content. Group A streptococci which are 96% beta haemolytic commonly cause pharyngitis and pyoderma. Antibiotics have little effect on the duration of streptoccocal sore throat but reduce effectively and promptly the complications of otitis media, pharyngeal and cervical abscess. Systemic antibiotics are helpful in limiting and eradicating the organisms in the skin.

More important than the clinical infection of the skin and throat are the delayed complications of acute rheumatic fever and PSAGN. Acute rheumatic fever can follow about 18 days after the onset of pharyngitis by any type of Group A streptococci. Therefore antimicrobial prophylactic therapy against this organism is indicated to prevent recurrences in every patient who has had acute rheumatic fever. PSAGN on the other hand may follow Group A infection of the skin and throat but only by certain M types (and T types in those cases which cannot be M typed). The risk of nephritis is higher after infection by type 49 of the skin (25%) than the throat (5%). The onset of symptoms is about 3 weeks after onset of pyoderma and 10 days after pharyngitis. Several extra cellular products of the streptococci have been identified. These include enzymes such as streptolysins S and O, hyaluronidase, streptokinal desoxyribonuclease (DNAase) and Nicotinamide adenosine dinucleotidase (NADase) and erythrogenic toxins. Streptolysin S is non immunogenic but the O lysin which is responsible for the surface haemolysis evokes an antibody response which is more after a throat infection than a skin infection. This is because in the skin the lysin O is bound by cholesterol and other lipids. However the antibody response to DNAase is more with the skin than the throat infections. If the presence of antibodies to several of the above mentioned antigens are sought, a positive serological response will usually be found The streptozyme test using sheep red cells coated with five antigens is a useful screening test to identify these antibodies with few false positive and false negatives. Clinical Manifestations and diagnosis. During the streptococcal infection haematuria may be present but this disappears before the development of nephritis. The latent period before PSAGN develops is about 10-21 days. An interval

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shorter than 4 days is more in favour of an exacerbation of already existing glomerulonephritis rather than PSAGN. The usual presenting features are those of an acute nephritic syndrome-haematuria, oedema, some degree of oliguria and hypertension. Sometimes there may be additional cardiovascular changes sf cerebral cedema and pulmonary congestion. Haematuria either microscopic or macroscopic occurs in more than 90% of patients. Fluid retention occurs in every patient and is related to the reduction in glomerular filtration rate causing retention of sodium and water. All the clinical features eg. hypertension and even acute renal failure are related to this retention and emphasize the importance of limiting dietary sodium. For the diagnosis there should be (i) evidence for a streptococcal infection by cultures of throat or skin and serological evidence as discussed above (ii) abnormalities in the examination of the urinary sediment for RBC, WBC and casts. Proteinuria should be present but should not exceed 2-3 G/24h. If no urinary abnormalities are noticed PSAGN is unlikely although there are recorded cases of biopsy verified PSAGN without urinary abnormalities (iii) low levels of serum complement fractions. Total serum complement is low during the active stage of the disease and is reflected by a low C 3 level. Complement levels return to normal by 3 weeks. Persistently low levels indicate some other renal lesion.

Differential Diagnosis. Firstly the other causes for an acute nephritic syndrome should be considered. These may be divided into the primary renal diseases like membrano proliferative glomerulonephritis and the systemic diseases with renal manifestations like Henoch Schonlein purpura, systemic lupus erythematosis. Next, other infections, both bacterial and viral which can cause acute glomerulonephrtis should be considered. These include preumococcal pneumonia, typhoid, brucellosis, ECHO virus, Coxsackie, Measles, Chicken pox, Mumps, and Esstein Barr virus. This can be very difficult and hence the importance of establishing the occurrence of a preceeding streptococcal infection. Where cardiovascular features predominate in the presence of minimal or even absent urinary abnormalities the diagnosis of PSAGN is very difficult and may require verification only by renal biopsy.

Pathology and pathogeneis. PSAGN is a sequclae of immune complexes deposited in the glomeruli. The evidence for this is (i) low levels of complement in the early clinical course (ii) the presence of circulating immune complexes (iii) demonstarations by immunofluorescence of granular deposits of Immunoglobulin G and complement in the glomeruli. This deposition is responsible for the characteristic mesangial and epithdial cell proliferation seen in the affected glomeruli on light microscopy. The glomerular capillary basement membrame is unaffected On electron microscopy electron dense deposits are always seen, usually in the sub epithelial layers.

These deposits are predominant in the first 3-6 weeks. In patients in clinical remission who have had repeat renal biopsies these deposits had disappeared. The nature of these deposits is not certain. So far no one has showed the presence of streptococcal antigens in the electron dense deposits or in the circulating immune complexes. The glomerular damage is believed to result from lysozomes released from polymorphs drawn to the site of the antigen antibody reaction and from direct action of activated complement components.

Treatment. No form of therapeutic intervention has been shown to acclerate the healing of the renal lesion. The importance of salt and water retention has

been mentioned and attention to the fluid intake is important and may be all that is required until the clinical manifestations subside. At this stage bed rest is no longer necessary. Mild degrees of hypertension should be treated to avoid cardiovascular complications. Diuretics are indicated if there is circulatory overload before congestive cardiac failure and pulmonary oedema develop. If either of these, or acute renal failure develop dialysis should be started. Dialysis may have to be prolonged and there are cases reported of complete recovery after months of dialysis.

Prognosis. The great majority of children and also adults with PSAGN recover spontaneously and completely. What percentage of cases become chronic? It is difficult to answer this question as it is first necessary to define what constitutes chronic glomerulo nephritis. Is it a single abnormal urinalysis or an elevated blood urea or even an abnormal renal biopsy? Or must it include a very sensitive test of renal function eg. the urinary sodium output after saline infusion? How often must these tests be carried out and what should be the period of follow up in patients with acceptable evidence of PSAGN before complete cure is pronounced?

Differences in the criteria mentioned above has led to dissimilar results recorded in different reviews. One prospective study in Trinidad where epidemics of PSAGN in association with streptococcal skin infections occur and where also the endemic incidence is high may provide the answers.

PERFORATED PEPTIC ULCER BRIEF REVIEW OF THE LITERATURE AND AN ANALYSIS OF 28 CASES

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Summary: A retrospective analysis is made of 28 cases of perforated peptic ulcer treated surgically in one unit over a two year period. Only 60% of the patients presented with typical symptoms and signs, the remaining 40% presenting atypically, mainly due to delay in arrival at this unit. The delay also accounted for 17% of the patients undergoing surgery more than 72 hours after the perforation. The need for resuscitation, while present in all cases of peritonitis, is increased in patients presenting with shock. The surgical management was confined to closure of the perforation. The overall mortailty was 20% for operative cases and its rate was proportionately higher in cases of chronic peptic ulcer perforation, in comparison with patients with acute ulcer perforation.

Introduction :

There is an increasing incidence of perforated peptic ulcer particularly in urban areas which shows a direct association with both stress and the usage of anti-inflammatory drugs such as asprin, butazolidine and corticosteroids (1-3). The perforation of either gastric or duodenal ulcers result in peritoneal soiling with irritative fluid which causes an internal chemical burn, and which in turn is followed by a marked outpouring of peritoneal fluid, initially sterile but which usually becomes secondarily infected with time. The resulting bacterial peritonitis may occasionally resolve, or become localised forming either subphrenic or pelvic abscesses, or more usually leads to generalised peritonitis which ends fatally. The necessity for definitive treatment in the latter group of patients is obvious. The characteristic features of peptic ulcer perforation are sudden onset of severe abdominal pain and abdominal regidity (4). If left untreated, the rigidity passes off with time, and abdominal distension due to ileus, with dehydration and shock as the consequences of continued peritoneal fluid losses are the presenting features of patients who are seen late in the course of the illness. About 60-80% of cases of perforated peptic ulcer have free gas in the peritoneal cavity which may be demonstrated radiologically as gas under the diaphragm in the erect posture. Donavan et al (6) confirmed the diagnosis and the extent of the leak by the oral administration of Hypaque, a water soluble contrast medium. This procedure, termed a gastroduodenogram, is also useful in identifying those cases where spontaneous sealing of the ulcer has occurred.

Therefore in the treatment of peptic ulcer perforation it is necessary to consider (1) the peritonitis, (2) the perforation and (3) the complications of the ulcer.

Peritonitis is an immediate and serious threat to life and its management takes priority. Fluid loss into the peritoneal cavity may amount to as much as 5 litres (4) and its replacement must obviously be a prime consideration. Intravenous infusions of electrolytic solutions are instituted at a rate governed by the estimate of fluid

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depletion which increases with time. Nasogastric aspiration and antibiotics are used to keep the stomach empty and control bacterial infection respectively.

Occasionally, the perforation may seal spontaneously, but more usually it fails to do so allowing continued leakage and soiling sf the peritoneal cavity. Those patients with typical symptoms in whom both symptoms and signs resolve spontaneously, probably have ulcers which have become sealed and are best treated non-operatively with nasogastric suction intravenous fluids and antibiotics. In th, majority of patients there is a pregressive worsening of both symptoms and signs and this group of patients require surgical intervention and repair. Between these two groups of patients is however a third group, less well defined and in whom the allocation to either of the two protocols of management outlined above is difficult. It is in this middle group of patients in whom a gastroduodenogram would be most useful in demonstrating those with "leaking" ulcers in whom surgical closure is advisable, while those with sealed ulcers as demonstrated radiologically, could then be treated conservatively (6).

Peritonitis in these cases is merely one of the complications of a peptic ulcer, and once treatment for it is instituted it is necessary to consider the treatment of the ulcer itself. In recent reviews it has been found that the natural history of the ulcer can be predicted (5-10). Analysis of large series of patients by these authors has shown that only 10 to 20% of patients with perforations of acute ulcers need subsequent surgery for persistence of ulcer symptoms whereas 50 to 70% of patients with perforations of chronic peptic ulcers have required a second operation. This difference in the natural history of patients with acute and chronic peptic ulcers has a bearing on the type of operation that is needed at the time of presentation with perforation, since the majority of such patients will require surgical intervention for the perforation itself. Most authors agree (6-10) that patients with perforations of acute ulcers, and poor risk patients with chronic ulcer perforations are best managed by closure of the ulcer alone (as the surgical procedure), while perforations in chronic ulcers in patients who are in reasonable condition should undergo closure of the perforation together with definitive surgical treatment for the ulcer. The definitive surgery that is most favoured is either Truncal Vagotomy and drainage, or Parietal Cell Vagotomy (6-11). A few surgeons however still hold to the older form of treatment,advocating simple closure of the perforation as an immedate procedure in those patients requiring surgery, and reserving definitive ulcer surgery only for those patients in whom persistence of symptoms at follow up demand it. (12)

Meterials and Methods.

During the 2 year period September 1977 to September 1979, 28 patients with perforated peptic ulcers were treated surgically in one general surgical unit at Jaffna General Hospital. This paper is based on a retrospective analysis of these cases; 3 patients who left after operation and against medical advice have been untraceable and have not been considered further in relation to the post-operative analysis.

The patients' ages ranged from 18 to 74 years, 70% being in the fifth and sixth decades. There was a male preponderance in a ratio of 3:1. Fiftythree percentum of the patients gave a history of dyspepsia and abdominal pain suggestive of previous peptic ulcer disease and 10% of the patients stated that they had recently taken drugs such as Asprin and Phenyl Butazone, well known as harmful to the gastric mucosa. Sudden onset of severe abdominal pain was the presenting complaint in 60%

of the patients, 10% complained of dyspnoea and 28% stated that they had not passed urine on the day of admission. Three patients (10%) did not give a history of abdominal pain, and did not have abdominal pain at the time of admission. Abdominal rigidity was present in 60%, and abdominal distension in 46%. Sixteen per centum were in a state of collapse at the time of admission. An erect radiograph of the chest was taken in only 10 patients, 7 of whom had free gas under the diaphragm. Operation was carried out within 12 hours of the perforation in only 21%, and within 24 hours in 68% of the patients. There was a delay of over 72 hours between perforation and operation in 17% of the patients due mainly to delayed admission to this hospital. It was the policy to offer operative treatment to all patients with perforated peptic ulcers irrespective of the time of admission.

Resuscitation was carried out with intravenous fluid therapy with 5% Dextrose and electolytic solutions, and nasogastric aspirations. Antibiotics were given to all 28 patients. The 16% of patients admitted in a state of collapse also had catheterisation of the urinary bladder and mannitol was administered to promote the secretion of urine. The operative procedure was confined to simple closure of the perforation with chromic catgut sutures and overlay omental patches in 27 patients. In one patient the edges of the perforation could not be approximated and hence the ulcer was excised and pyloroplasty performed. The peritoneal cavity was routinely washed out with saline and an intraperitoneal drain brought out through a separate stab incision was left in situ in those cases where pus was present. Routine post-operative management included intensive chest physiotherapy, and serum electolyte measurement when abdominal distension persisted. In these latter group of cases if the abdominal distension persisted after the correction of electrolytic disturbances (when present) Metclopropamide was prescribed. Four patients had troublesome distension, 2 of whom improved after Metclopropamide (16).

There were 4 cases of post-operative wound infection and one burst abdomen, none of whom had evidence of a residual intraperitoneal abscess. There were 5 deaths (20% mortality), 3 being in patients with perforation of chronic peptic ulcers. Three of the deaths occurred in patients whose operation was carried out more than 72 hours after the perforation. One death was due to recurrent perforation on the 8th day, and another to massive haematemesis on the eleventh post-operative day.

Discussion.

The threat to life from an unsealed peptic ulcer perforation, is due to the ensuing peritonitis. The overall mortality which was about 25% in 1940 has improved to the region of 11% in recent years due mainly to earlier diagnosis, physiologically based resuscitation, and earlier surgery (1, 4, 10, 13). In the underdeveloped countries these three factors remain at risk for various reasons, not the least of which are patients preference for an alternative system of medicine which having no surgical speciality can offer no benefit to patients with perforated peptic ulcers, the relative scarcity of specialised facilities i.e. surgery, difficulties with transport from rural areas, and in a small percentage of cases who delay seeking medical attention and have advanced in their disease to a stage in presentation that is not commonly encountered in the standard texts on the subject, thereby posing diagnostic difficulties not only to the lone medical officer in a rural outpost but even to surgeons in general hospitals. With this in view I have reviewed my experience of 28 cases treated at the Jaffna General Hospital

which is the only General Hospital for the entire Northern Province, serving a population of close to a million people, and those who are furthest away having to travel 80 to 100 miles for treatment at this institution.

Diagnosis:

The majority of patients with perforated peptic ulcers have the typical features of sudden onset of severe abdominal pain, usually epigastric, and becoming progressively worse, some having in addition a previous history suggestive of peptic ulceration, a rigid abdominal wall and tenderness all over the abdomen so that the diagnosis in countries like the United Kingdom, the United States and those of Western Europe can confidently be made on clinical grounds alone. In my series only 60% of patients gave a history of sudden abdominal pain and a like number had abdominal rigidity so that a significant minority of 40% presented atypically. It is possible that these unusual presentations were due to (i) delayed presentation resulting in confused histories, the initial rigidity of peritonitis wearing off with time and the patients thus presenting as very ill individuals with abdominal distension; and (ii) atypical symptoms, as was present in 6 patients (21%), due probably to the advanced stage of the disease consequent upon delay to an extent unencountered in the developed world and hence dealt with scarcely, if at all, in the literature. These unusual presentation merit a little further examination. Three patients (10%) in my series did not have abdominal pain, and the abdomen in each although distended and silent was lacking in rigidity. Two of them were in a state of shock. All three presented late and the interval between perforation was more than 48 hours which probably accounted for the death of 2 of them. This mode of presentation is probably a variant of the subacute perforation described in Bailey and Love's Short Practice of Surgery where the patients had minimal pain and in whom minimal rigidity was present. All 3 patients in this series who did not complain of pain were admitted more than 48 hours after the onset of symptoms and it is probable that the initial onset of mild pain was either forgotten by the patient or even overlooked by the house surgeon in the taking of the history. Three other patients (10%) had troublesome dyspnoea together with abdominal pain and were unable to lie flat. Anseline (1), and Donovan et al (6) have discussed patients presenting with dyspnoea, but the subject is not described in the standard texts. Each of them described one patient in whom dyspnoea was a prominent feature. Donovan's patient improved immediately after relief of the tension pneumoperitoneum by the use of an 18 guage needle, suggesting that the dyspnoea was due mainly to intra-abdominal tension. In the 3 patients presented here the dyspnoea was no longer present after surgery, probably due to the release of tension at that time. Being an unusual symptom dyspnoea is not a well recognised feature in these patients and it needs to be borne in mind in the atypical case. The final atypical presentation was in one of the patients in whom admission was delayed over 48 hours and who presented without the characteristic abdominal pain, but did have anuria for 2 days. The referral note with this patient stated that she was sent on account of retention of urine! In addition to the other features mentioned, she was also in shock. Resuscitation with large volumes of electrolytic solutions and later surgery resulted in a satisfactory outcome notwithstanding the pre-renal failure which was present. Such a presentation of renal failure with peptic ulcer perforation is not referred to in the current standard surgical texts or in the references used in the preparation of this paper, and it is

undoubtedly due to the late presentation. It is an awareness of these various atypical presentations which will enable earlier diagnosis and admission to hospital for definitive treatment, which will result in a reduction of the mortality rate of 20% shown here.

Resuscitation:

The need for resuscitation in every case of peritonitis is obvious but it is the scale of resuscitation that varies. In those societies where there is a greater awareness of medicine and its derangements, better transportation and lesser distances of travel to hospitals, such patients presenting with shock are thought to have large leaking ulcers, extensive peritonitis and a higher mortality rate (4). Large quantities of intravenous fluids are required, to replace the fluid loss into the peritoneal cavity resulting in a depletion of both vascular and extracellular fluid compartments initially, and later similarly affecting the cellular compartment too and at this stage being associated with derangement of the sodium pump mechanism. It not only compounds the clinical picture but also enhances the mortality rate. Five patients (18%) in this series were admitted in a state of shock, not so much due to the size of the ulcer as the delay in presentation and needed more aggressive resuscitation. In one of these patients there was 4 litres of fluid in the peritoneal cavity! Following resuscitation and surgery, three patients recovered, one died and one patient left hospital against advice on the second post-operative day. None of these patients would have survived without both aggressive resuscitation and surgical repair as they all had leaking ulcers.

Operative treatment:

It is well established that with perforated peptic ulcers the mortality rate increases with the delay in surgery, and particularly so after 12 hours (1). The overall mortality rate in my series was 20%, but in those patients undergoing operation after a delay of 72 hours the mortality increased to 75%. Only 21% of these patients underwent surgery within 12 hours of perforation accounting for the high mortality. The blind, non-surgical management of patients with perforated ulcers has usually ended in disaster (4, 6) and while most authors report mortality rates of up to 74% with non-operative management, a few have reported series of either low, or even no mortality (1, 6, 8), indicating that in carefully selected patients in whom spontaneous sealing off of the perforation has occurred a non-operative regimen may be followed with acceptable results. Kay and his colleagues (10) found that perforations of acute ulcers, i.e. patients with ulcer symptoms of less than 3 months, tend to spontaneous sealing and that on the other hand large chronic ulcers when perforated would never show that tendency. It is obvious then, that it is this latter group that will always require surgical repair. Management of the former group calls for fine judgement bearing in mind that a failure in conservative management will inevitably result in a delay in surgical treatment and thereby carrying an additional risk. In my series, non-operative management was carried out only in those patients whose clinical condition suggested spontaneous closure and in whom continuous improvement was manifest, and this group carried no mortality. It is not analysed in the figures presented in this paper as confirmation of diagnosis was not obtained. All other patients underwent surgery. Donovan et al (6) selected for conservative management only those patients in whom a gastroduodenogram demonstrated a sealed ulcer perforation. This facility was not available at the Jaffna General Hospital, and a conservative policy of management had to be based on clinical evaluation, and only when it was strongly in favour of spontaneous sealing

was surgery witheld. Of the 28 patients who underwent surgery, in only one was the ulcer found to have been sealed off. A similar policy of clinical evaluation was used by Ti and Young (8) in Malaysia with good resuts.

There is still some controversy as to the type of surgery to be carried out when operating for perforation. Drury et al (12) advocate simple closure of the ulcer-perforation initially and definitive surgery to be later employed only in the case of recurrent ulcer symptoms. Others advocate closure of the ulcer with immediate definitive surgery in selected cases (5, 6, 7, 8, 9, 11). In the case of those large chronic ulcers that cannot be closed by simple sure, and also when malignancy is suspected definitive surgery has to be performed at the first operation. This may take the form of excision of the ulcer and pyloroplasty in duodenal ulcers, or gastrectomy for gastric ulcers. However the over-riding consideration must be what is best for the individual patient and delay in presentation causing fluid and electrolyte disturbances increases the mortality,which would be further enhanced with prolongation of operation time. Since late presentation was the order of the day with the patients in this series the policy adopted was of simple suture in 27/28 patients, and ulcer excision and pyloroplasty in only one patient in whom simple closure could not be achieved. Two of the five deaths are attributable to direct complications of the ulcer itself, recurrent perforation and bleeding in 2 patients with chronic ulcers. Whether the addition of definitive surgery for the ulcer at the initial operation would have spared them these complications is conjectural for they had the additional hazard of physiological fluid and electrolyte losses. An alternative would have been to use Cimetidine post operatively, known to be effective in ulcer healing (14, 15), but at that time the drug was not available in Jaffna.

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ACTINOMYCOSIS OF THE LUNG

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Actinomycosis of the lung

Summary: Two cases of pulmonary actinomycosis in whom the diagnosis was made at a stage of development of sinuses in the chest wall are presented. Both responded to prolonged Benzyl Penicillin therapy. The clinical and radiological features of the disease are briefly reviewed

Introduction: Actinomycosis is a disease of world-wide distribution the incidence of which has recently been falling presumably due to the widespread use of Penicillin. It is a chronic systemic disorder caused by Actinomyces israelii a common commensal organism in the mouth which may become pathogenic and cause tissue invasion, and affecting the face, neck, thorax and abdomen. The thoracic form of the disease accounts for about a quarter of all cases and usually occurs after aspiration of A. isrealii into the bronchus. Symptoms are inconspicuous in its early stages, the diagnosis being usually made at the stage of chronic sinus formation by the demonstration of the typical "sulphur" granules, and culture of the organism. Two cases of pulmonary actinomycosis seen during the last 5 years at the Chest Clinic, Kandy, are presented.

Case Material

Case 1. An 18 year old male agricultural worker was first seen at the Chest Clinic, Kandy on 8th September 1975 with a complaint of irregular pyrexia and left sided chest pain both of which had been present for the past 6 weeks. There was no history of cough. He had 2 carious teeth in the lower jaw and the signs of consolidation over the left lower chest. He also gave a history of contact with tuberculosis. Sputum examination for acid fast bacilli was negative, as was the Mantoux test. A diagnosis of unresolved bacterial pneumonia was made and he was treated with chloramphenicol 250 mgms 6 hourly for 9 days without improvement. On the 25th September his original symptoms still

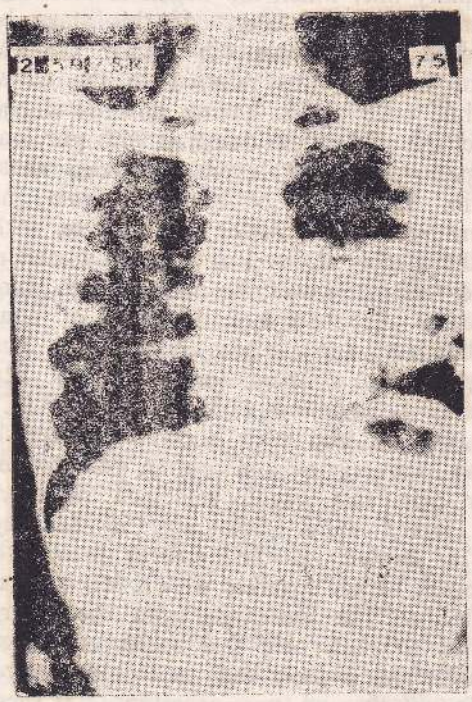

Fig. 1. Case 1. Xray of chest of 25-9-75 showing: high left leaf of the diaphragm, sli ghtcrowding of lef. 4th, 5th and 6th ribs and a confluent opacity in left mid and lower zonest

Chest Clinic, Kandy

[1] *Consultant Physician*

persisted and he had in addition a tender swelling 3 cms above the left nipple. A radiograph of the chest showed elevation of the left hemidiaphragm, slight crowding of the left 4th, 5th and 6th ribs and a confluent opacity in the left mid and lower zones (Fig. 1). He now sought treatment from an ayurvedic physician and a week later a sinus developed in the area of the chest wall swelling, which began to discharge pus. After a further 7 days a second swelling developed below the nipple and shortly afterwards showed sinus formation. Ten weeks later a thirp painful swelling, 9 cms in diameter appeared over the 7th left rib in the anterior axillary line, which too began to discharge pus a fortnight later. At this stage he sought advice from the Kandy General Hospital and was referred to the Chest Clinic once again. having now developed a cough of 3 weeks duration with purulent expectoration and severe left sided chest pain. Induration of the skin from the sternum to left anterior axillary line, with 3 discharging ulcers, 3 to 6 cms in diameter, and tenderness over the affected area were present. He still had the signs of consolidation over the left lower chest. The erythrocyte sedimentation rate was 126 mm in the first hour, white cell count (WBC) 10,800/ cu mm with normal differential. The Mantoux test and sputum examination for acid fast bacilli remained negative. A repeat chest radiograph in January 1976 showed all the features present 4 months earlier together with a left pleural effusion. The discharge from the ulcers failed to show acid fast bacilli on direct smear and aerobic and anaerobic culture was negative on two occastons. It then transpired that the ulcers were cleaned daily with Savlon and when this was stopped a pure growth of Actinomyces israelii was isolated on culture of the pus. He was treated with Benzyl Penicillin 2 million units 6 hourly for 6 weeks and half the dose for a further six weeks. Ten days after commencing penicillin therapy his symptoms had abated and two months later the ulcers healed. A chest radiograph in April 1976 showed crowding of the left 2nd to 6th ribs with periosteal thickening of the 4th, 5th and 6th ribs, an elevated left hemidiaphragm, opacity in the left mid and lower zones and residual effusion (Fig. 2). One month later the crowding of the ribs and the pulmonary opacity were less prominent. Periosteal reaction was still present, but the hemidiaphragm was less elevated than previously. He was

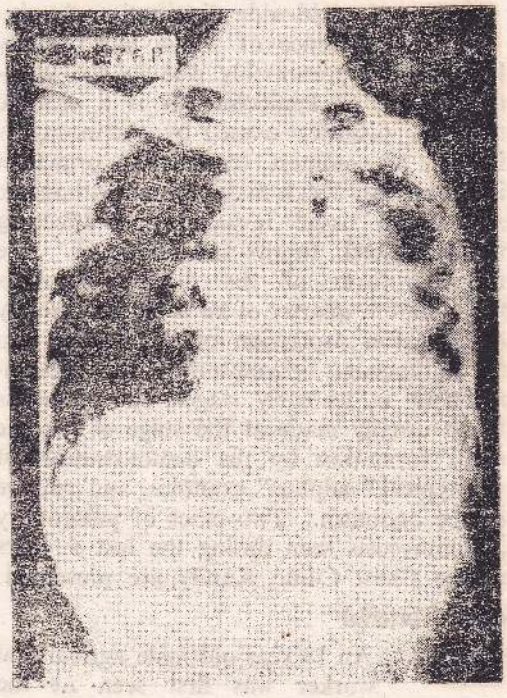

Fig. 2 Case 1. Xray of the chest of 5-4-76 showing; high left leaf of the diaphragm, crowding of the left 2nd to 6th ribs, periosteal thickening of the left 4th, 5th and ribs, pleural effusion and opacity in the left mid and lower zones.

discharged after 3 months penicillin therapy and remained well when reviewed one month later.

Case 2. A 48 year old male agriculutural worker was seen at the Chest Clinic, Kandy on 5th May 1978 complaining of 4 painful swellings with discharging sinuses over the anterior chest wall, and a cough with mucoid expectoration, the total duration of symptoms being 4 months. The discharging sinuses were each about I cm in diameter. Clinical examination revealed a resonant percussion note with vesicular breath sounds over the chest. His ESR was 101 mm in the first hour and the WBC 9300/cu mm with normal differential. Sputum was negative for acid fast bacilli, and culture of the pus yielded Actinomyces israelii. A radiograph of the chest on 24th April 1976 showed crowding of the left 5th to 8th ribs with increased density of the lateral portions of the 4th to 7th left ribs and widening of the 7th and 8th. The left hemidiaphragm was elevated and streaky markings were present in the left lower lung. He was treated with Benzyl Penicillin 2 million units 8 hourly. Pain and tenderness disappeared after one week, and the ulcers healed by the end of the second. After 3 weeks the penicillin was reduced to one million units b.d for a further 9 weeks and when reviewed was making satisfactory progress

Discussion.

Thoracic actinomycosis is a chronic suppurative disorder caused by the rod shaped bacterium Actinomyces israelii which though a commensal organism in the mouth becomes invasive in the presence of any devitalising process. The presence of carious teeth and an unkempt mouth are usually associated with clinical actinomycosis. Symptoms are often lacking in the early stage of the pathological lesion and clinically and radiologically it has to be differentiated from pneumonia, lung abscess, empyema, tuberculosis, nocardiosis, other fungal infections and bronchial carcinoma (1, 2). The infection charcteristically spreads across tissue planes frequently causing pleural effusions which rapidly progresses to empyema, periosteal reactions of the ribs and infiltration of the chest wall with the development of sinuses from which the charcteristic "sulphur" granules are obtained, at which stage the diagnosis may easily be made (1, 3). The Actinomyces israelii was long considered to be a fungus but is now classified under the bacteria (4). It is a microaerophilic filamentous bacterium which is cultured with difficulty. Antibiotics and local antiseptics should not be used prior to obtaining a culture. Normally present in the mouth, thoracic actinomycosis results from aspiration into the bronchi (5). The bovine form of disease is caused by A. bovis and although the human disease is common in agricultural workers there is no evidence of contagious spread from cattle to man (6).

The pulmonary lesion is a chronic suppurative process of insidious onset. Later, cough, with mucopurulent or blood stained sputum, pleurisy and increasing systemic upset with night sweats and weight loss may be present (1,5). Characteristic of the disease is the tendency to transgress across fascial planes resulting in the early development of pleural effusions which rapidly progress to empyema, periosteal reactions and the development of chest wall sinuses (1,7) features which are much less evident in nocardiosis. The incidence of actinomycosis in this country is not known although Cooray (8) reporting on 5 cases seen between 1954 and 1960 stated that it was not uncommon. In a search of the literature however no further reports were found.

Young males are most commonly affected (9). While the diagnosis in the early stages may be difficult, the development of multiple sinuses and the presence of the characteristic sulphur granules should indicate the correct diagnosis which may be confirmed by the culture of the organism. Other laboratory tests are of less diagnostic importance. The ESR is commonly elevated (5), and while the WBC and differential count in the two patients reported here were within the normal range, Riddell (5) found a neutrophil leucocytosis.

The radiological features of thoracic actinomycosis (7) were well illustrated in the two cases presented here and have been described in the text. The raised hemidiaphragm is however not so well documented.

The diagnosis becomes very probable with the demonstration of suphur granules and confirmed by culture sf the organism. Slade et al (2) found that fixing the sputum in Bouin's fluid was an aid to the demonstration of the characteristic sulphur granules in the stained material and advocated its use in diagnosis. The treatment of this disease has been dramatically changed with high doses of penicillin for prolonged periods, doasage varying from 6 million to 20 million units daily (1, 3, 10). Tetracycline (11), and Streptomycin (12) ans Isoniazid (13, 14) have also been successfully used.

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PENETRATING INJURY OF THE HEART

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Summary: A patient who had sustained a penetrating injury of the heart survived due to the combined temporary beneficial influence of cardiac tamponade and the avoidance of a rise of intrapericardial pressure beyond the critical point because of a pericardio-peritoneal communication. The diagnosis, which in retrospect should have been made earlier was only made when the signs of intraperitoneal haemorrhage forced attention from a general surgical unit.

Introduction. Penetrating cardiac wounds have been known since ancient times but were then regarded as being invariably fatal. In the seventeenth and eighteenth centuries some doubt regarding this conclusion arose after the discovery of healed ventricular scars at autopsy in patients who had in addition precordial scars and following this the more aggressive surgeons attempted to save the lives of the acutely injured by repair. Sir Stephen Paget in 1896 wrote "the surgery of the heart has probably reached the limits set by Nature to all surgery; no new method and no new discovery, can overcome the natural difficulties that attend a wound of the heart" (1). The same year Rhen performed the first successful repair of a cardiac wound in man. By 1909 Peck reviewed 160 cases treated by surgery with a mortality of 64% (2). There was a gradual improvement and by 1941 Elkin (3) had a reduced operative mortality of 42%. Napolean's surgeon, Larrey treated a wounded heart by pericardial aspiration, in 1829 and showed that these injuries were not necessarily immediately fatal although his own patient died 68 days after injury and 23 days after operation of suppurative mediastino-pericarditis (4). In view of the mortality rate of around 40% Blalock and Ravitch in 1943 and Ravitch and Blalock in 1949 reported reduced mortality rates by pericardiocentesis alone (5,6). Subsequently initial pericardiocentesis for the relief of tamponade with penetrating cardiac injuries and reserving thoracotomy and cardiorraphy for its recurrence or continued bleeding became the preferred policy in many centres (7-9). This latter policy implies that close monitoring is maintained for at least 48 hours. It has been reported that between 65 and 81% of all penetrating heart injuries succumb before admission to hospital (1, 10). In this country stab wounds of the chest are distressingly common, occur throughout the country although its incidence is perhaps highest in those areas of greatest population density along the southwest coast. However, where it is frequent treatment by virtue of familiarity is also better than in the areas of low incidence. Given that the majority of these patients will have to be treated by general surgeons whose work load carries an in-take admission rate of approximately 40 patients on each Casualty day, and the shortages of nurses and monitoring facilities, penetrating cardiac injuries should ideally be treated by immediate thoracotomy and cardiorraphy. In the patient reported below there was a delay of over 5 hours to operation presumably due to unawareness of its necessity on the part of the house staff, and although the result was fortuitously gratifying, this report is made

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for the purpose of drawing attention to both the injury and the need for surgery.

Case Report: On the 3rd October 1973 a 30 year old man was admitted to the Casualty Surgical Ward at Kurunegala General Hospital at 4 p.m. with a history of having been stabbed half an hour previously..... There was an incised wound 2.5 cms long in the fourth left intercostal space just lateral to the sternal border. He was unconscious, without radial pulse or recordable blood pressure. An intravenous infusion was commenced with Normal saline and continued with 5% Dextrose and he was also given Hydrocortisone 200 mgms as a bolus and with each unit of intravenous fluid; half an hour after admission the systolic blood pressure was 70 mm Hg. Three hours later the pressure was still only 80 mm Hg and he had now developed mild abdominal distension with tenderness on the left. The presence of abdominal signs of haemorrhage was the indication for calling for a surgical opinion. At laparotomy five and a half hours after admission about one and a half litres of blood was found in the peritoneal cavity, and blood was seen to be ejected with each heart beat through a 4 cms long incised wound in the tendinous portion of the diaphragm. A separate thoracotomy through the 5th left intercostal space showed a further one litre of spilt blood in the pleural cavity due to a penetrating wound on the inferior border of the right ventricle, which was sutured with interrupted linen. A 2.5 cms long incised wound on the dorsal surface of the liver was also sutured. During the pre- and intraoperative periods he had received a total of 4 litres of intravenous fluids including 750 mls of blood. His post-operative period was complicated by bronchopneumonia, slight haemorrhage from the thoracotomy incision on the 4th day, and pericardial effusion which yielded only 20 mls on aspiration 8 days after operation. He was discharged from hospital 28 days after admission.

Discussion: The diagnosis of a penetrating wound of the heart depends primarily on a high index of suspicion which was lacking in this case. It should be suspected with all precordial wounds and it may also occur with more remote chest wounds, epigastric and even neck wounds. Posterior chest wounds have only rarely been associated with heart injuries. The clinical features depend to a large extent on the competency of the pericardium in controlling haemorrhage; when competent there is a progressive increase in both intrapericardial volume and pressure, but not in direct linear relationship, resulting in reduced ventricular filling in diastole, and low cardiac output due to tamponade, while when free bleeding from the pericardial to either pleural, or less commonly, peritoneal cavities occurs, low cardiac output results from hypovolaemia. Beck's classical triad of falling arterial pressure, raised central venous pressure and quiet heart sounds will then only be seen in the presence of tamponade, but even then the triad is present in less than a third of all cases. The fall in arterial pressure is a late sign and quiet sounds depends so much on the environment and the observer, both of which are often upset with the admission of these patients. The raised central venous pressure should arouse suspicion but it has to be differentiated from that which occurs with hyperpnoea. With free bleeding however, just the low cardiac output, poor volume pulse and hypotension in spite of transfusion should indicate haemorrhage and whatever its origin, the treatment demands early thoracotomy. The latter features were present in this case, but perhaps the increasing confinement of general surgeons to the abdomen has resulted in a failure

of proper education of the junior staff. Penetrating cardiac injuries may occur but rarely in the experience of a general surgeon, but a successful outcome depends on both correct diagnosis and treatment. The use of hydrocortisone in this case is difficult to justify for it has not been shown to have been of any value. The result, though fortuitous, was gratffying, and the case is reported to perhaps draw the attention of the junior surgical staff in this country to the occurence of these cases.

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HOMICIDAL POISONING

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Homicide by poisoning has been known from time immemorial. Its incidence was high during the latter part of the last century and early part of this century, but the number has appreciably decreased due to the combined effect of the restrictions exercised by the law enforcing authorities in the acquisition of drugs and poisons on the one hand and due to the advancement in the techniques used in the detection of poison and drugs in the human tissues on the other. A murderer who intends committing murdrer by poisoning plans elaborately. "A perfect poison" is one that is tasteless and odourless and is easily soluble in water. It should produce symptoms of poisoning mimicking, after some interval, those of a natural disease. Very few poisons possess all these properties. Table (I) gives a list of some of the notable trials of murder by poisoning. It will be observed that in most of these cases the victim is usally the spouse. In most instances where were murders were committed by practising doctors, the homicidal agent used is either a drug or a poison. Table (II) Doctors in particular are in a position to commit murder by poisoning and escape detection, as dangerous drugs and poisons are easily accessible to the members of the medical profession. No one is watching or questioning them. When their object has been achieved, they can authorise the disposal of the body by issuing the necessary death certificate. One should

Table I

CASE	RELATIONSHIP TO THE ACCUSED	HOMICIDAL AGENT IDENTIFIED AT POST-MORTEM EXAMINATION
Rex. v. Crippen	Wife	Hyoscine
Rex. v. Armstrong	Wife	Arsenic
Rex. v. Seddon	Miss Barrow (friend)	Arsenic
Radford	Wife	Arsenic
Rex. v. Greenwood	Wife	Arsenic
Reg. v. Maybrick	Husband	Arsenic
Rex. v. Wilson	2nd husband	Phosphorus
	3rd husband	Phosphorus
Reg. v. Merrifield	Mrs. Ricketts (friend)	Phosphorus
Reg. v. Barlow	Wife	Insulin
Reg. v. Chapman	1st wife	Antimony
	2nd wife	Antimony
	3rd wife	Antimony
Rex. v. Pritchard	Wife	Antimony

Table II

CASE	CAUSE OF DEATH
Rex. v. Dr. Crippen	Hyoscine poisoning
Rex. v. Dr. Pritchard	Antimony poisoning
Reg. v. Dr. Palmer	Strychnine poisoning
Rex. v. Dr. Lamson	Aconite poisoning
Reg. v. Dr. Neillcream	Strychnine poisoning
Rex. v. Dr. Smithurst	Antimony poisoning
Rex. v. Dr. Ruxton	1st victim strangulation 2nd victim Head injuries

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not however run away with the idea that many murders are committed by doctors. There are over three thousand doctors in Sri Lanka and over seventy thousand in Eng'and and Wales but only a handful of professional murders had been recorded in these countries during the past fifty years.

Table (III) contains the list of common homicidal agents.

Table III

POISON
Arsenic
Antimony
Thallium
Cyanide
Phosphorus
Barbiturate
Chloroform
Hyoscine
Strychnine
Insulin

Arsenic. Despite the severe restrictions placed on the acquisition of arsenic and the ease with which it can be detected in human tissues, it still enjoys a prominent place as a homicidal agent. Notable trials for murder by arsenic poisoning are those of Mary Blandy, Mandeleine Smith, Mrs. Maybrick, the Seddons, Armstrong and Greenwood.

Arsenic is a gastro-intestinal irritant; vomiting is almost invariable, but seldom delayed for more than twelve hours, and is associated with a sense of constriction in the throat. Abdominal pain and diarrhoea occur within five to twentyfour hours and the stools resemble 'rice water'. Some complain of muscle cramps specially in the calves. In fatal cases, the patient rapidly goes into a state of shock and peripheral vascular collapse. Pigmentation of the skin, hyperkeratosis, peripheral neuritis, mental instability and delirium are features of chronic arsenic poisoning. Lethal dose is two to five grains. Arsenious oxide, being testeless and odourless, can be administered without arousing the suspicion of the intended victim. Arsenious oxide is sparingly soluble in cold water so Mandeleine Smith used hot chocolate to dissolve the arsenic. Porridge or gruel is also a good vehicle as it can hold considerable amount of poison without exciting suspicion. Potassium arsenite is another compound of arsenic which is commonly used as a homicidal agent. Mrs. Maybrick and the Seddons used potassium arsenite from fly papers. Simpson in 1949 (1) reported a case of arsenical poisoning that occurred under the very nose of the doctors and the staff and the poisoner nearly got away with it. Gordon Radford, a laboratory technician poisoned his ailing wife with arsenic, while she was a patient in a sanatorium. He carefully introduced some arsenic in pies which he sent to his wife. When Mrs. Radford became sick after eating one of the pies, she reamarked to one of her friends who was regularly visiting her, about her husband's extra-ordinary kindness since of late. She however requested her to get one of the pies examined for poison. Her friend sent the pies to the Medical Superintendent with a covering letter. The letter went into the secretary's "in-tray" and the parcel was placed on the Medical Superintendent's table. It was a Saturday after-noon and when he saw a nice pie, thinking it was from a friend, took it home for lunch. Before he could finish eating it, he became sick. He carefully retained the remaining part of the pie and on Monday morning when he read the letter sent by Mrs. Radford, he realised why it had been sent to him. The pie was analysed and found to contain 3 grains of potassium arsenite. Mrs. Radford died 3 days after eating the pie.

Sectional analysis of her hair revealed that she had been systematically poisoned over a period of three months. Radford when he realised that he was being sus-

pected, poisoned himself by taking prussic acid.

A doctor (4) once administered a lethal dose of prussic acid to his 5 year old daughter and then took his own life. A research chemist (5) murdered his mother who was suffering from cancer by giving her potassium cyanide and thereafter he too ended his life by taking the same. In Malaya according to Gimlette (6) potassium cyanide was used with honey for the purpose of committing murder. The under surface of the knife used for cutting a water melon was smeared with the poison and it was so arranged that the intended victim got the lower portion smeared with the poison while the assailant ate the upper half.

Strychnine

Strychnine is rarely prescribed but its use for poisoning continues. It has been described as the bitterest substance known, in spite of which it has been used as an instrument of murder. There are many ways of masking the taste of strychnine. It is possible to administer strychnine in alcohol without arousing the suspicion of the victim. It can be introduced into foods which have bitter taste. It can be given as pills. Strychnine stimulates the higher centres and produces a picture clinically indistinquishable from that of tetanus. In the famous case of Rex. v. Palmer (7) strychnine pills were given to Cook by Palmer. Thereafter Palmer persuaded 80 year old Dr. Bamford to issue a medical certificate for "apoplexy" but the intervention of Cook's father resulted in an inquest and autopsy. The autopsy was performed by a local doctor and an undergraduate. Palmer who was allowed to be present at the autopsy deliberately jostled the operator at the very moment he was dissecting the stomach causing thereby the spilling of some of its contents. In spite of all these flaws in the medico—a legal procedures, Palmer was found guilty by the Jury.

Hypoglycaemic agents

Symptoms of hyper insulinism begin when the blood sugar falls to the region of 50-60 mg/100ml. Initial symptoms of hypoglycaemia are hunger, fainting, pallor, headache and numbness of face and extremities. Further lowering of the blood sugar produces dysarthria, diplopia, muscular twitching, convulsions, coma and death. Fortunately, murdrr with hypoglycaemia agents is rare. The first recorded case of homicidal poisoning by insulin was that of Regina. v. Barlow. Details of this case were reported by Birkinshow at-al in 1958 (8) Barlow, a state registered nurse married the deceased woman in 1956. In May, 1957 he lost his job when his wife was also expecting a baby. On the 3rd of May, 1957, she was found dead in the bath. When the doctor arrived at 11-30 p.m. he saw the deceased lying on the right in the bath. The water had been drained and the body was warm and wet. According to Barlow she took off her pyjama and went for a bath at about 10-00 p.m. and in the meantime he had dozed off. When he woke up at 11-20 p.m. he found her lying face downwards in the bath. He pulled the plug, drained the bath and gave her artificial respiration. Surprisingly the sleeves of Barlow's pyjama were not wet while the pyjama of the deceased was soaked in sweat. This aroused the suspicions of the police. Post-mortem confirmed that death was due to drowning. As no poison could be detected in the internal organs of the deceased, the body was re-examined on May 8th and four puncture marks were detected on the buttocks. Barlow suggested from the witness box that his wife might have injected insulin herself. It is not easy for one to inject into both the buttocks. The layout of the house was

such that no third person could have entered the house at that time without the knowledge of Barlow. Barlow had access to insulin and he had knowledge about its ill effects. When he gave instructions to his colleagues he had commented that insulin is the best drug available to commit a perfect murder. What precisely happened is known only to Barlow. The probabilities are that after she was rendered unconscious by injecting insulin she was carried into the bath and the scene was so arranged to stimulate accidental drowning. Barlow was found guilty of murder and sentenced to life imprisonment.

Barbiturates Toxic symptoms may occur following a single dose. Death occurs due to depression of respiratory and circulatory functions. Barbiturates are used widely in the treatment of anxiety states, sleeplessness and other psychiatric conditions. The posibility of committing murder by administering barbiturates must always be kept in mind. In the Armstrong case (9) Terrance Armstrong aged 6 months, son sf a naval berth attendant died suddenly. The doctor in attendanc ecould not give a death certificate and an autopsy was ordered. At the sutopsy there was nothing to account for the death but a red "berry" was discovered near the right tonsil and similar red "berries" were found in the stomach. The stomach contents were analysed and 1/3 grain of seconal was extracted. Further 1/50th grain of seconal was extracted from the vomit stained pillows. These "berries" were actually parts of seconal capsule. A verdict of death due to seconal poisoning was returned. As no other evidence was forthcoming John and Janet Armstrong, the father and mother of the child remained free for about an year. During this period the Armstrongs quarelled and drifted. Janet Armstrong applied for separation and maintenance but her application was disallowed, and she walked out of the courts in tears. Inspector Gates who investigated Terrance Armstrong's death was present in Court at that time and asked Janet Armstrong whether she had anything to tell the police. She hesitated for a moment and then volunteered a statement which resulted in the canviction of John Armstrong.

Other Agents

Aconite is one of the most deadly poisons known. It produces a tingling sensation numbness of the tongue and mouth followed by constriction of the throat and difficulty in swallowing. The case of Dr. Lamson (10) is the only known instance of homicidal use of aconite in England.

Atropine

Atropine is rarely used as a homicidal agent even by persons who have access to the drug. Withaus (11) cited a case where a nurse is said to have added belladona to milk drunk by the senior surgeon of her hospital. She was acquitted owing to inadequate evidence to cannect her with its administration.

Hyoscine hydrobromide was used by Dr. Crippen (12) to kill his wife. Willcox isolated 2/5th of a grain of hyoscine hydrobromide from the viscera about 6 months after burial. Dr. Crippen had purchased 5 grains of hysocine hydrobromide 12 days prior to the death of his wife. He lied about her disappearance and he was caught when he attempted to excape the law by ocean liner to America with his girl friend.

Chloroform if used inadvertantly can cause death due to ventricular fibrillation. Adelaide Bartlett (13) was charged for the murder of her husband by pouring liquid chloroform down his throat while he was asleep. She

was acquitted owing to insufficient evidence to connect her with the crime.

In conclusion most of the poisons used for homicidal purposes produce symptoms similar to those of many known natural diseases where even the most skilled medical personnel are apt to be misled. One should therefore look for poisoning especially when the diagnosis of a natural disease is in doubt.

Phosphorus is not an ideal homicidal agent. It has an unpleasent odour and a disagreeable taste, but these however do not prevent it from being used as a homicidal agent. It produces liver damage and mimics infective hepatitis of viral origin. Poisoners use various vehicles to mask its taste and odour. In the Merrifield case it was beleived that the vechicle was rum and sugar or black-currant jam. In Wilson's case (2) Mary Elizabeth Wilson was found guilty of the murder of her second husband Oliver James Leonard and her third husband George Lorence Wilson by administering yellow phosphorus in the form of beetle poison in cough mixture.

Thallium has many but fortunately not all the properties of a "perfect posion". Salts of thallium are easily soluble in water and are odourless, tasteless and colourless. The symptoms occur after an interval of 12 hours giving enough time for the criminal to cover his tracks. When a patient presents with polyneuritis and alopecia, one should think of thallium poisoning but unfourtunately these symptoms may be delayed as long as 10 days.

Antimony

If not for the notable cases Rex. v. Chapman and Rex. v. Pritchard the amount of space devoted to antimony poisoning in forensic testbooks would be very small indeed. Chronic rather than acute poisoning is common as it is usually administered in small doses at frequent intervals rather then one large dose. The clinical picture superficially resembles natural illness. The outstanding features of antimony poisoning are nausea, vomiting, thirst, diarrhoea, and muscular cramps. The case of Chapman (3) is worth mentioning as it is historic and intructive. Even the most skilled medical personnel of Guys Hospital at that time were deceived. In July, 1902 a woman named Maud Marsh was admitted to Guys Hospital with a history of recurring bouts st abdominal pain and diarrhoea. A diagnosis of tuberculous peritonitis was made and she was treated for almost a month in Guys Hospital. She remained well for about two months after which, symptoms recurred and after 12 days of treatment by a local doctor, she died. A private autopsy was performed and antimony was identified in the material submitted for snalysis.

Sir Thomas Stevenson repeated the analysis on the orders of the coroner and antimony was isolated in all parts submitted for analysis. Maud Marsh was living with a person named Chapman prior to her death. Investigations revealed that Chapman had purchased an ounce of tartar emetic five years back at Hastings. Further investigations revealed that sometime after this purchase, his first wife Isabella Spinks had died and 2 years later his second wife Bessie Taylor met the same fate. The cause of death in the case of Isabella was put down to tuberculosis while Bessie Taylor was reported to have died of intestinal obstruction after a brief illness of nausea, vomiting, and abdominal pain. Large amounts of antimony were found in each of the two bodies. Chapman was found guilty and executed..

Cyanide. In cyanide poisoning death comes on with dramatic suddenness. Muscular twitching, vomiting and frothing at the mouth may precede unconsciousness. Cyanide is a rare choice for homicide,

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ILEOSIGMOID KNOT

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Summary

A case of ileosigmoid knot is reported. In this condition a loop of ileum and the sigmoid colon are twisted around each other in a knot. This is like a double volvulus with two closed loop obstructions. Gangrene follows quickly with the tightening of the knot and early surgery is essential.

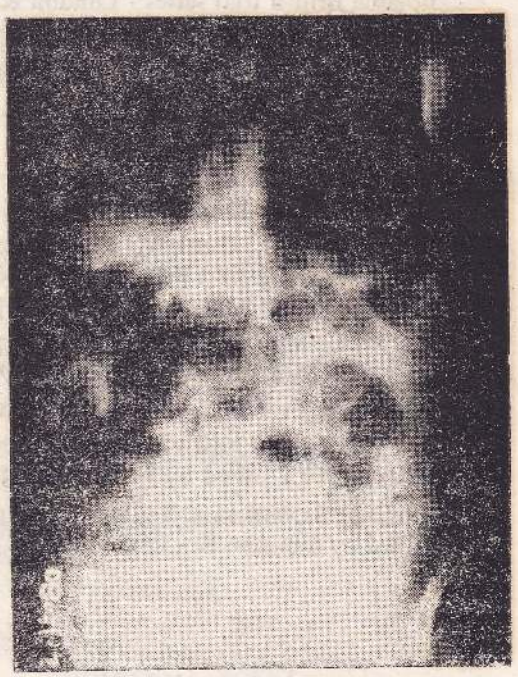
Case Report

A 65 years old male was admitted to the General Hospital, Jaffna at 12.30 p.m. on 2.11.80. On 1. 11. 80 at 8 p. m. the patient had rice meal with vegetables. At 10 p.m. he developed sudden, severe, colicky hypogastric pain. There was no radiation or shifting of this pain, which aggravated with passage of time. The pain became continuous after 4 a. m. After this the patient did not remember anything. He had absolute constipation and did not pass urine. He vomited once.

On examination, the patient was found to be in severe pain, drowsy, pale and dehydrated. His pulse was 120/ minute, regular and feeble. Blood pressure was not recordable. There was slight abdominal distension, generalised tenderness and guarding. Bowel sounds were absent. Rectum was empty and there was no blood on the finger. A mass which was displaced by the examining finger was felt in the pelvis. Cardio-vascular and respiratory systems were normal.

Jaffna General Hospital

1 *Consultant Surgeon*



Plain X'ray of abdomen showed: (Fig 1)

(1) A large distended loop of bowel (sigmoid colon) with limbs directed downwards into the right lower quadrant with absent haustration and smooth convex border.

(2) There were air and fluid levels in the small intestine mainly in the left side displaced by the pelvic colon.

(3) The caecum and proximal colon contained faces and little gas, shown as mottling.

The patient was resuscitated with 200 mls. of N. saline, 500 mls. of 5% Dextrose, intravenous hydrocortisone 400 mgs. and chloramphenicol and taken up for laparotomy at 4.30 p.m. During operation, a further, 2000 mls. of N. saline, 500 mls.

of Hartmann's solution, 500 mls. of molar lactate, 250 mls. of mannitol and 500 mls, of blood were infused. In all he received over 5000 mls. of I. V. fluids and 500 mls. of blood before and during operation. At exploration through a right paramedian incision, the pertioneal cavity contained blood stained fluid. The ileum was wrapped round the sigmoid colon causing gangrene of 5 inches of sigmoid colon and 7 feet of small intestine, 6 feet from the duodeno-jejunal flexure. Resection of both loops of gangrenous bowel segments and end to end anastomosis of the small bowel and left iliac colostomy with closure of the rectal stump was done. Colorectal continuity was restored through an oblique left iliac muscle cutting incision. The patient had an uneventful recovery and left hospital on 5.12.80.

DISCUSSION

(1) Mechanism of the knot. Abnormally mobile small intestine with a long mesentery wraps round a sigmoid colon with a mesocolon, that is long and poorly attached. The loop of small intestine wraps round the base of the sigmoid colon from right to left and passes beneath itself forming a half knot. With peristalsis more ileum passes round the sigmoid causing constriction of the blood supply to both the loops, viz. ileum and sigmoid colon1. At laparotomy 80% have gangrenous bowel. (2) There are two closed loops—sigmoid colon and ileum. The caecum and proximal colon in between the loops is a vacuum and is not distended with gas but contains faeces. Ingestion of high bulk diet in the presence of an empty small bowel is believed to be responsible for the knot. Ileo—sigmoid knot occurs in communities that eat a high bulk diet like the Ugandans1 and Afghans (3) during the Ramazan. The sigmoid colon wrapping round the ileum has also been reported1.

(2) Radiology

Radiological findings which are of diagnostic value are, grossly dilated loop of sigmoid colon with limbs directed downwards into the right lower quadrant, air and fluid filled loops of small intestines, proximal large bowel which is filled with faeces but containing little air and medial deviation of the distal descending colon(3). The last featrue was not seen in this case.

There is early gangrene in both the loops. In the case reported obstruction in the sigmoid colon occured first (hypogastric pain) and strangulation occured at 4 a.m., when the pain became continuous. As there is no prospect of spontaneous resolution or of successful conservative management, surgery is urgent and is the only method of saving life. There is early effusion of blood into the peritoneal cavity and into the infarcted gut wall and lumen. Peritonitis develops secondary to bacterial penetration of the infarcted bowel wall. Rapid resuscitation with intravenous electrolyte solutions, hydrocortisone and wide spectrum antibiotics and blood help to reduce the high mortality of 40% to 50%1. At laparotomy untying the knot is difficult and hazardous as it may cause repture of the gangrenous loops. The ileum is divided after clamping at the point of entry and exit. End to end anastomosis of the small intestine and terminal colostomy or Paul Mickulicz's exteriorisation of the sigmoid colon immediately and colorectal anastomosis after two to three weeks gives the best results.

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VOLVULUS OF THE SMALL INTESTINE

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Summary:

Three cases of volvulus of the small intestine, each of which was caused by a different mechanism, are presented. Delay in diagnosis, made resection necessary in two. The clinical features of small bowel volvulus are analysed.

Introduction:

One of the less common causes of intestinal obstruction is volvulus of the small intestine, particularly the variety that occurs unassociated with malrotation of the midgut. Most of the published case reports have been from Russia, Scandinavia, and India where a bulky diet of course grain has been incriminated as a causative factor. The present report consists of three cases of volvulus of the small intestine, each caused by a different mechanism.

CASE REPORTS

Case 1 H.S.F., a 34 year old male was admitted to the Kandy General Hospital on 29th April 1965 with a history of generalised abdominal pain of three days duration. The pain was constant, and during the three days he had also suffered from anorexia, nausea and constipation. On examination he was well nourished and not clinically anaemic. There was no cervical lymphadenopathy. Cardiovascular and respiratory systems were normal. There was moderate abdominal distension, but the abdominal wall was quite soft to palpation. There was no tenderness. Bowel sounds were normal.

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He was treated by gastric suction and intravenous fluids. A plain X'ray of the abdomen showed dilatation of the small intestine. Laparotomy the same day through a left paramedian incision showed no free fluid. There was clockwise rotation of the base of the mesentery of the small intestine causing a volvulus of that segment of the bowel, which had undergone two and a half turns. There was no malrotation and the ascending colon was fixed in its usual position. The volvulus was untwisted, the height of the mesentery from the posterior abdominal wall to bowel wall was now found to be eighteen inches. His post-operative recovery was complicated by bronchopneumonia and he was discharged from hospital on the 20th day.

Case 2 P. C. D. A. a 58 year old man was admitted on 10th July 1966, with a history of sudden onset of pain in the left side of the abdomen at 7 a.m. Later, the pain was felt over the whole abdomen. It was described as being pricking in nature, and its intensity had not changed. There was no radiation of pain, and none was felt at the shoulder tip. Half an hour after the onset he had vomited twice, the vomitus consisting mainly of mucus. Three hours after the onset of symptoms he had noticed abdominal distension. His bowel habits had been regular, and he had a bowel action shortly before the onset of the pain. Two years previously he had developed upper abdominal pain which was aggravated by the taking of meals, and had persisted for six months. On examination, he appeared to be comfortable, and not anaemic. The tongue was coated. There was moderate abdominal distension with reflex guarding in the upper abdomen and slight tenderness in

the left iliac fossa. No masses were palpable. No definite diagnosis was made and he was treated conservatively. The following morning there was severe abdominal distension with guarding and tenderness in all areas. Laparotomy carried out soon afterwards showed blood stained free fluid in the peritoneal cavity, due to a double volvulus:- a volvulus of the sigmoid colon on its mesocolon, and a secondary volvulus of a loop of small bowel around the stem of the sigmoid volvulus. Both were non-viable, but were reducible fairly easily. Sigmoid colectomy with end to end colo-rectal anastomosis, and resection of 2½ feet of ileum with end to end anastomosis was carried out, and the abdomen closed with a corrugated rubber drain brought out through a stab incision in the left iliac fossa. He was kept on intravenous fluids and gastric suction for three days and then made an uneventful recovery. Sutures were removed on the 12th day and he was scheduled for discharge the following day. That night however he fell out of bed, but no abnormality was found at that time. His condition however deteriorated rapidly and he died on 24th July. Post mortem examination showed the cause of death to be a rupture of the ileum and suprarenal haemorrhage.

Case 3 W.M.T.B. a 48 year old male was admitted on 27th October 1966 with a history of sudden onset of severe abdominal pain the previous night. It was a continuous generalised abdominal pain, with colicky exacerbations. He had vomited once just prior to admission. There was no history of haematuria. Bowels were normally open three times per day, but stools contained neither blood nor mucus. No abnormality was found on clinical examination, the abdomen being soft, and without guarding of tenderness. During the evening and night he complained of severe abdominal pain, and on the latter occasion generalised abdominal tenderness was found. The following day there was a marked deterioration. The pulse was rapid and thready, and the abdomen distended. Marked guarding and tenderness on light palpation was present and a diagnosis of peritonitis was obvious. At laparotomy he was found to have a gangrenous mid small bowel due to volvulus. A small hiatus about 2 cms long was present in the mesentery of the mid small bowel, and a knuckle of ileum had passed through this and become strangulated. With this loop acting as a fulcrum the small intestine had undergone clockwise rotation through 360 degrees, and the whole volvulus was now gangrenous. In the apparently normal but distended segment of proximal jejunum six diverticula were present along the line of mesenteric attachment, each the size of an olive. Subtotal small bowel resection was necessary leaving about 3 feet of small intestine which was anastomosed end-to-end. Post operative recovery was complicated by bronchopneumonia and mild wound sepsis and he was discharged on the 16th day.

Discussion:

The diagnosis of volvulus of the small intestine is seldom made pre-operatively resulting in delay in definitive treatment and a high incidence of intestinal gangrene. Sudden onset, with pain, vomiting and abdominal distension have been described as the characteristic features (3). However, in many cases, the early features are non-specific, and the diagnosis of an intra-abdominal condition requiring surgical intervention is often overlooked (4, 5). Vomiting, although described as a typical feature is often so mild that the possibility of upper intestinal obstruction is not considered. In most cases, the pain is described as being continuous, with colicky exacerbations being noted less commonly. Abdominal distension occurs some hours

after the onset of symptoms, and even during the early phase of its presence, the abdomen remains soft and relaxed. Guarding and tenderness develop later, and all too often, as happened twice in the cases reported here, the necessity for exploration is only made at this stage, and the presence of a long segment of gangrenous bowel is so surprising that one is baffled by the rapidity with which such a serious state could develop in a patient in whom only a matter of hours earlier the possibility of serious intra-abdominal pathology was hardly entertained. The early symptoms are probably due to a partial volvulus causing intermittent intestinal obstruction. Woods' description (6) "the affected loop was evidently swinging like a ship at anchor, partially folding over and then swinging free again" seems to explain the features in the difficult cases.

Volvulus of the small intestine may be primary or secondary, depending on the presence of a causative mechanism. The primary variety is unusual and only a few cases have been recorded in the literature. In all three patients described in this paper the volvulus was secondary to some other factor, each of which is itself uncommon. McKechnie and Priestley (7) in reviewing 37 episodes of small intestinal volvulus at the Mayo Clinic over a 25 year period found peritoneal adhesions to be the commonest cause. Diverticulosis of the small intestine has been estimated to occur in 1.3 per cent of patients (8). Walker (9) considered them to be acquired abnormalities, occurring on the line of mesenteric attachment at points of weakness where the blood vessels and nerves entered the intestinal wall. Aird (10) however postulated a developmental error—"parallel vacuolation theory". Most cases of diverticulosis are asymptomatic, and require no treatment. Munyaradzi and Wapnick (11) collected eleven cases of jejunal diverticula with small bowel volvulus from the literature and added one of their own. Seven further cases from previous reports, and one from this paper must be added to that list (3, 12) bringing the total up to twenty. Unlike the previously reviewed cases, the present one required resection of the volvulus and on account of the short segment of small intestine remaining, the diverticular bearing area had to be retained.

Fifty cases of transmesenteric herniae were reviewed by Cutler & Scott in 1944 (13) when they presented two of their own cases in whom volvulus of the afferent and efferent limbs of the strangulated loop had occurred. The mesenteric defects vary considerably in size and may be as small as 1.5 cms diameter. Recently Sharma, Bhargava and Galviya described a small bowel volvulus in a loop that had herniated through a mesenteric defect 16 inches x 12 inches. (14). The mechanism of volvulus in the case reported here was different in that only a knuckle of bowel was incarcerated in the mesenteric hernia, and this acted as the fulcrum around which volvulus of almost the whole of the small intestine had occurred.

Double volvulus of small and large intestine has to be differentiated from the condition of ileo-sigmoid knotting, a condition which again is common in Russia, Scandinavia and Africa (15). The latter condition is caused by a loop of ileum wrapping itself around the base of a passive sigmoid colon, ensnaring it in a knot which is difficult to unwrap. On the other hand, the double volvulus is a rare condition in which the volvulus of the pelvic colon occurs initially, and as it displaces the small intestine from the upper left quadrant of the abdomen a loop of the latter is swept around the pedicle of the sigmoid volvulus into the left para-colic gutter and undergoes volvulus as it does so. Reduction is easily achieved, but resection of both loops may be necessary (16).

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