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Editorial

The Annual General Meeting

by A.A. Sandosham

VIEWED FROM ANY ANGLE, it can be safely asserted that the Eleventh A.G.M. held in Penang from 2nd to 4th April, 1971 was a grand success. The arrangements, which had been in the hands of an Organising Committee, with Mr. Peter C. Vanniasingam as Chairman and Dr. Chee Chin Seang as Secretary, were excellent with all the meetings being held in the airconditioned comfort of the Mandarin Hotel and the Association owes a debt of gratitude to our hosts in Penang. The attendance was good, many members having travelled up to Penang with their wives to join their counterparts in the Northern Branch. As expected, the Informal Night at which members and their wives let their hair down, was a resounding success. The Annual Dinner and Ball was well patronised with covers laid for more than 350 and was graced by His Excellency the Governor of Penang and his Consort, the Minister of Health and the Chief Minister and their wives, the Joint-President of the Commonwealth Medical Association and representatives of the Indonesian Medical Association and various sister professional organisations. The social gatherings and entertainment arranged for the ladies were well attended and much appreciated. The competitions in tennis and golf were well organised and the large audience benefited much from the high standard of the papers presented and the discussions at the Forum held as part of the Scientific Session.

The Annual General Meeting itself was a lively affair and the Annual Report of the outgoing Council, drawn up by the Hon. Secretary, Dr. Pius Martin (who was unanimously re-elected), was discussed page by page. There were many questions requesting clarification and amplification, sometimes by members who had not taken the trouble to read documents that had been circulated in advance. There were those who felt that the Council could have achieved more and acted more speedily.

As mentioned by Dr. V. Thuraisingham in his Incoming President's Address, there have been rumblings of discontent among the younger members of the Association but few had come forward with constructive suggestions and offered their services to do preparatory work and help make a case or find a solution. He appealed to them to join the Association and contribute not only their weight in numbers, but also their energy, minds, aspirations and enthusiasm for the good of the profession, remembering that our Association is not a Trade Union empowered to negotiate terms and conditions of service with the employer. Despite these criticisms, it was evident to the meeting that the Out-going Council had done a splendid job during its year of office by increasing ordinary and life membership, maintaining a high standard of professional ethics, having representatives on numerous Boards and Committees, organising clinical and scientific meetings, publishing regularly the Medical Journal of Malaya, the MMA Newsletter and the Public Health Society Bulletin, organising the successful 5th Council Meeting of the Commonwealth Medical Association and generally looking after the interest of the profession in the country. On the proposal of Dr. T. Satchithanandan, the President-Elect, the General Meeting passed heartily and unanimously a resolution proposing the adoption of the Annual Report and congratulating the Out-going Council, particularly the President, Dato (Dr.) Keshmahinder Singh, for a job of work well done.

One problem that was discussed at length was the incorrect image of the Association that was projected to the profession, the public and Government by misleading newspaper headlines and the over-emphasis of critical comments taken out of context from our publications like the MMA Newsletter or talks given by MMA officials at Conferences, Seminars and Forums. Sometimes, wrong statements are attributed to the Association or its officials without their retraction when the mistake is pointed out. Even if corrected, the mischief has been done and it can be very upsetting to the Association and other parties concerned. Dato (Dr.) Abdul Majid bin Ismail suggested that much of our trouble arose from a lack of liaison and dialogues with the press, the Ministry of Health and others involved. The problem is how to achieve the desired result. An appeal was made to members to use their influence and lobby the right people and explain the policies and activities of the Association to avoid misunderstandings in the future.

Substantial changes have been effected in our Constitution which will come into force with their ratification by the Registrar of Societies. The name of the Association has been changed to Malaysian Medical Association, thus permitting the opening of membership to doctors in East Malaysia. Steps will be taken to start negotiations with the Sarawak and Sabah Medical Associations to finalise future relationship. In keeping with the democratisation of the presidential election procedure recommended by the A.G.M. last year, it is now possible for any member to nominate a candidate although the latter should be from one of the three different regions in rotation, and every member will now have the right to vote him into office by postal ballot on the lines of the system adopted in electing members to the Medical Council. While this procedure will result in casting the net more widely and make every member feel he has a say in the election of the President, there is the possibility of having too many names on the list and, judging by past experience, a relatively smaller proportion of doctors, especially among the isolated private medical practitioners, taking the trouble to send in their ballot papers. If all members are not aware of this and do not put in the extra effort to get ballot papers duly filled and posted, there is a real danger of this important office being held, year after year, by Government officers from the country's big hospitals where there is a concentration of members easily approachable by the energetic canvasser.

In the past, we have had the services of private medical practitioners like Dr. S.G. Rajahram, Dato (Dr.) R. Sathiah, Dato (Dr.) A.W.E. Moreira, Dr. R.F. D'Costa, Dr. Tan Chee Khoon, Dr. J.B.A. Peter and Dato (Dr.) Keshmahinder Singh, many of whom have made substantial contributions to the advancement and progress of our Association. It is to be hoped that this will be kept in mind and that all members, including the private medical practitioners, will make a determined effort to vote and make the democratising process a success, resulting in the election to the most coveted position in our Association a person of the highest calibre, be he in the public or the private sector.

Coma and Consciousness

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Coma and Consciousness

ALL TOO OFTEN, patients with coma or lesser alterations of consciousness present to the physiciar with an inadequate or an unreliable history. The comatose patient, hence, is a challenge in diagnosis and therapy. Having grappled and reversed potential life-threatening situations, the physician then proceeds with an orderly rational approach to the diagnosis and treatment of the precipitating factors responsible for the comatose state.

The purpose of this discussion is to define coma and discuss briefly its pathophysiology. In addition, we hope to present clinical entities that could produce coma and describe a practical approach from the point of view of physical examination, neurological examination and investigations towards an adequate management of these patients.

In order to understand and approach rationally the problems of coma, one must first comprehend the levels of awareness and the need for categorising the various "levels of consciousness". We cannot, therefore, limit our discussion to coma, but rather must define and discuss impaired "levels of consciousness".

One should not attempt to define consciousness in pure biological terms, as it is that process which utilises sensory perception via exteroceptors, memory, intellect, attention and comprehension, all of which help to produce an appropriate response. Coma is then an extreme lack of consciousness in which a state of vegetative existence prevails. In other "levels of consciousness," superficial reflexes, such as corneal and gag reflexes, may be present. All responses may be absent or often abnormal. The latter may take the form of decerebrate posturing elicited, say with mere touch pain or other exteroceptive stimuli.

Between these two extremes lie arbitrarily delineated categories. If one reviews the literature discussing this subject, one sees that most authors have judicially avoided definitive categories and many terms have been used synonymously. For the purpose of this discussion, the realm between coma and consciousness can be categorised in a manner that is meaningful and can be communicated as follows:— (Table 1)

Stupor or semi-coma is that state of relative unresponsiveness to environment or specific sensory stimuli, in which superficial reflexes are intact, and deep reflexes may or may not be elicited. No response is obtained on questioning alone although not uncommonly an inappropriate or unintelligible grunt may be heard. However, appropriate but

TABLE I

1) Coma
Vegetative state.
In lighter planes, superficial reflexes such as corneal and gag reflexes persist.
No response to verbal stimulation.
No response or abnormal response to painful stimulus such as decerebrate posturing.

2) Semi-coma or Stupor
Superficial reflexes intact.
Appropriate, but primative response to painful stimulus (i.e. flexion withdrawal).
No response to verbal stimuli.
May or may not respond to pain with unintelligible utterings.
Can be of psychogenic origin, such as catatonic stupor.

3) Somnolent
Reflexes intact.
Tends to return to sleep-like state; totally inattentive to environment unless continually subjected to direct sensory stimulation.

4) Lethargy
Reflexes intact.
Disinterested in environment.
Responds appropriately to minimal direct stimulation.

5) Confusional States
Alert or lethargic-responds to minimal stimulation.
Reflexes intact.
Responses are inappropriate; may confabulate or hallucinate.
At times, difficult to distinguish from dysphasia.

6) Conscious
Alert, oriented.
Responds appropriately to environment and specific sensory stimulation.

primitive responses, such as flexor withdrawal, are elicited to painful stimuli. Catatonic stupor, a classical example of psychogenic stupor, should be entertained in the differential in these instances. Sometimes patients are described as somnolent. This is characterised by a persistent sleep-like state in which the patient is totally inattentive to the environment unless stimulated repeatedly.

Lethargy, on the other hand, is best described as a disinterest and lack of attention to the environment. However, in this state the patient responds readily and often correctly to minimal direct stimulation, including questioning. Definition of commonly used terminology will not be complete unless confusional states are also included. This category is used to distinguish the confused, disoriented, and often hallucinating patient from the conscious one, because consciousness implies appropriate responses to sensory stimulation. Whereas the conscious patient is always alert, the confused or delirious patient may be alert lethargic, or somnolent. A dysphasic patient often gives the impression of being confused and unless this is borne in mind, errors in diagnosis are likely to result.

The physiology of consciousness and sleep, as well as the pathophysiology of coma, remains poorly understood despite numerous publications on the subject.[1]

Consciousness is attributed to the integrity and proper functioning of the periaqueductal grey area,[2,3] and the reticular formation. Both these areas, and especially the latter, are physiologic systems that defy precise anatomical delineation. The reticular formation has been shown to extend from the lower medulla to the thalamus with its pathway within the central portions of the brainstem. In 1949, Moruzzi and Magoun identified the ascending reticular activating system (ARAS) which has been shown to extend from the midbrain to the thalamus and to have definite but diffuse cortical connection.[4,5]

Subsequent experimentation of their thesis revealed that interruption of the ascending reticular activating system in any way led to coma. That sleep and coma were similar in that they were both due to a failure of the ARAS to activate the cerebrum, thus found support. However, both electroencephalography and single neuron recordings within the reticular formation, demonstrate an increased activity of these cells rather than electrical silence during sleep. As a result, current opinion that physiological sleep is an active, rather than a passive process,[6] seems plausible. Although the "principal" and "sustaining" projections of the ARAS are diffuse and varied, the reticular formation proper is housed compactly within the brain stem. It is therefore understandable why small mass lesions, hemorrhages, infarctions in the brain stem can compromise its

functional integrity and produce profound coma.[7,8] On the other hand, hemispheric lesions not causing brain stem compression do not result in altered consciousness. However, metabolic disturbances from infection,[9] drugs,[10] endocrinop athies[11,12] or various system dysfunctions may result in coma due to impairment secondarily of the metabolism and thus function of the neurons and supporting glial cells of the cortex.[13,14] However, here again it is difficult not to implicate the reticular formation in some way or even disregard it.

The clinical conditions which can lead to alterations of brain function are varied and include trauma, mass lesions intracranially, endocrine disturbances, exogenous toxins and failure of extracranial organ systems resulting in either primary or secondary metabolic disturbances of the neurones and glia.

The classification and discussion, as well as the approach to diagnosis and therapy, can therefore best be delineated if these disorders are classified into two basic groups. The first category includes primary disorders of the brain and its vasculature. These include trauma, cerebral vascular lesions, neoplasms, infectious disease, metabolic disorders, seizure disorders and psychogenic alterations. (Table II)

These can again be divided into three groups:

1) Those which impair the function of the reticular formation with the brain stem by a disruption of this system by way of compression and or displacement or by impairment of its circulation as a sequelae to thrombosis or hemorrhage.
2) Those which interfere with neuronal and glial cell function, e.g. infections of the brain and meninges, toxins, system electrolyte and nutritional deficiency states, primary metabolic disorders of the brain (see Table III).
3) Those of psychogenic origin notably catatonic states, severe depressive reactions, and hysteria.

Because of the wide variety of entities involved, the clinician faced with the problem of a patient with altered consciousness must approach the problem logically and efficiently. All too often, when one is faced with a deeply comatose patient, the feeling of inadequacy and insecurity results in a dramatic array of assorted laboratory tests and administration of drugs based on illogical diagnosis and without a comprehensive examination. It must be kept in mind that proper diagnosis and treatment of the comatose patient often results in his return to a functional life.

Its recognition and treatment should thus be an emergency in the true sense of the word in view of this potential reversibility. There is, for instance, no

TABLE II
PRIMARY BRAIN DISORDERS AFFECTING CONSCIOUSNESS

1) Trauma
- Concussion
- Contusion
- Epidural hematoma
- Subdural hematoma
- Depressed skull fracture
- Penetrating wound

2) Cerebral Vascular Lesions
- Cerebral artery thrombosis
- Basilar artery thrombosis or embolus
- Cerebral embolus
- Brain stem hemorrhage
- Intracerebral hemorrhage
- Cerebellar hemorrhage
- Subarachnoid hemorrhage
- Ruptured aneurysm
- Arteriovenous malformation

3) Neoplasms
- Gliomas
- Meningiomas
- Neuromas
- Metastatic lesions

4) Infectious Diseases
- Meningitis
- Encephalitis
- Meningoencephalitis
- Abscess
- Cerebral
- Empyema
- Subdural
- Epidural

5) Metabolic Disorders of the CNS
- Jacob-Creutefeldt disease
- Picks disease
- Schilders disease
- Leukodystrophies
- Progressive myoclonic epilepsy
- Huntingtons Chorea

6) Seizure Disorders
- Status epilepticus
- Post-ictal states

7) Psychogenic States
- Hysteria (conversion reactions)
- Depressive reactions (severe)
- Catatonia

TABLE III
SYSTEMIC DISORDERS AFFECTING CONSCIOUSNESS

1) Metabolic Disorders
- Hypoglycemia
- Insulin induced
  - exogenous
  - endogenous
- Alcoholic induced
- Vitamin deficiency
- Thiamine
- Pyridoxine
- Niacin
- B1
- Alkalosis and acidosis
- Hepatic failure
- Porphyria
- Renal failure
- Electrolyte imbalance
- Fever

2) Exogenous Toxins
- Alcohols
- Barbiturates
- Tranquilisers
- Hallucinogenic drugs
- Hypnotics
- Salicylates
- Carbon monoxide
- Cyanide
- Anticholinergics
- Heavy metals

3) Endocrine Disorders
- Hypopituitism
- Hyper & hypothyroidism
- Hyper & hypoparathyroidism
- Adrenal insufficiency
- Pancreatic dysfunction

4) Cardiorespiratory System
- Cardiac dysfunction
- Myocardial infarction
- Congestive heart failure
- Hypersensitive carotid sinus
- Valvular disease
- Arrhythmias
- Pulmonary
- Hypoventilation
- Airway obstruction

TABLE IV
PROBABLE DIAGNOSIS RELATED TO ONSET OF COMA

1) Acute
- Trauma
- Cerebral vascular lesions
- Basilar artery thrombosis or embolus
- Brain stem hemorrhage
- Intracerebral hemorrhage
- Subarachnoid hemorrhage
- Cerebral hemorrhage
- Neoplasms
- Hemorrhage into pre-existing tumor
- Insulin coma
- Acute adrenal insufficiency
- Carotid sinus sensitivity
- Cardiac arrest
- Hysterical coma

2) Sub-acute
- Trauma
- Subdural hematoma
- Cerebral vascular hematomas
- Cerebral thrombosis
- Cerebral embolus
- Subarachnoid hemorrhage
- Neoplasms
- Hemorrhage into pre-existing tumors
- Infectious disease of the CNS
- Seizure disorders
- Systemic metabolic disorders
- Exogenous toxins
- Endocrine disorders
- Cardio-respiratory system dysfunction

3) Chronic
- Trauma
- Subdural hematoma
- Neoplasm
- Infection of the CNS
- Brain abscess only
- Metabolic disorders of the CNS
- Systemic metabolic disorders
- Pulmonary disorders
- Psychogenic disorders

substitute for an adequate history as the basis of approach to the treatment of the comatose patient. If nothing more, the rate of alteration of consciousness to the level of coma manifested should be ascertained. This is because the rate of transformation from consciousness to coma provides a clue to a

rational appraoch to diagnosis and treatment. Table IV indicates how these rates of alterations from consciousness to coma help predict the line of aetiologic probabilities in the genesis of the coma, and thus help to both prognosticate and treat these patients meaningfully.

It is an accepted fact that trauma can result in instantaneous coma. However, it must be remembered that particularly subdural hematomas may not cause neurological deficits or altered sensoria for days to months after the injury. In this context, it may be stated that concussion is usually not a significant therapeutic problem since, by definition, it is a trauma-induced transient coma lasting no more than a few minutes with full recovery and no structural damage.[15,16] However, these patients should be observed for possible development of subdural hematomas. Hospital observation is advised anywhere from 24 to 48 hours.

Cerebral vascular lesions of the brain stem,[17] whether they be infarctions or hemorrhages, are capable of producing instantaneous coma. It is indeed rare for a cerebral thrombosis or an embolus to produce acute coma. Coma from a cerebral thrombosis, when it occurs, takes hours to days to develop because it is the resultant cerebral edema that leads to secondary compression of the reticular system. Subarachnoid hemorrhage, on the other hand, will often produce coma in seconds to hours, especially if it is accompanied by ventricular extension of the hemorrhage. Massive intracerebral hemorrhage must be included in the acute category, but smaller hemorrhages usually produce neurological deficits with lesser states of altered consciousness.

A pre-existing tumor may result in rapid or sudden coma when hemorrhage occurs into it. This is unusual but we have seen it associated with glioblastomas and oligodendrogliomas. Injudicious use of spinal puncture in a patient with an intracranial mass lesion, be it tumor, intracerebral hemorrhage or a subdural hematoma, may result in precipitous coma as a result of transtentorial herniation with resultant brain stem compression.[18]

Infections of the central nervous system usually result in altered levels of consciousness accompanied by clinical evidence of an infectious process and coma may take hours to develop in untreated cases.

Seizure disorders rarely produce coma except during the actual ictal period. This transient period is due to the bombardment of the reticular system by an abnormal large volley of electrical impulses. The post-ictal state is most often one of lethargy and somnolence accompanied by confusion and amnesia. Occasionally, however, the post-ictal state may be one of stupor.

Primary metabolic and psychogenic origins of profoundly altered levels of consciousness usually have a compatible history of a chronic nature. The systemic disorders produce coma with appropriate prodromes. However, rarely an acute onset of coma may be seen following cardiac arrest, adrenal insufficiency, or hypoglycemia. However, it should be kept in mind that hysterical coma may be precipitated suddenly in an appropriate situation from which the patient needs complete escape.

The clinical evaluation of the patient centers around four essential parameters in addition to the history. These are:

1) Type and character of the coma;
2) Pupillary responses;
3) Oculomotor signs; and
4) Patterns of respiration.

These evaluations are only of relative value and in profound coma, there are usually no eye signs of value. Such parameters are of greatest value when the patient has a limited alteration of consciousness.

The degree of alteration of consciousness as alluded to previously can be summed up as follows: The lighter stages are usually of supratentorial origin and may be (1) primary brain dysfunction, (2) secondary to toxic substances, or (3) as a result of disruption in cerebral metabolism from extracranial system failure. These situations are of a subacute and chronic nature. Profound alterations of consciousness are either due to primary organic brain stem pathology, which is of an acute nature, or are secondary to diffuse cerebral impairment, with subsequent brain stem compression representing an end stage in a subacute or chronic process.

Pupillary responses provide clues as to the origin and depth of central nervous system dysfunction. The pupils are evaluated as to size, and response to light and accommodation. The ciliospinal reflex characterised by a dilatation of the pupil as a result of painful stimulation to the ipsilateral side of the neck should also be elicited. When it is intact, it mitigates against the likelihood of a severe brain stem destruction.

Pupillary signs in coma are of value in terms of providing clues as to its possible origin. Cortical anoxia, atropine and scopolamine produce widely dilated pupils unresponsive to light stimulation. Posterior fossa hemorrhages, pontine hemorrhage, and

opiates produce pinpoint pupils. Pupillary responses are also of value in localising brain stem lesions. Midbrain (tectal) lesions obviate the light reflex and the pupils are round and 5-6 mm. in diameter. They tend to fluctuate in size spontaneously and the ciliospinal reflex remains intact.

With midbrain lesions involving oculomotor nuclei, the pupils are in midposition and unresponsive to all stimuli. Pontine lesions and compressive lesions of the posterior fossa tend to produce pinpoint pupils. One should be aware that as a general rule, pupils are reactive in metabolic coma. Although about 12 per cent of the normal population exhibit unequal pupils, anisocoria should always arouse suspicion of localised central nervous system pathology.

The third parameter is that of the oculomotor responses which result from vestibular stimulation.[19] Stimulation of the semicircular canals induces the vestibulo-ocular reflexes. This can be accomplished by two simple methods: 1) Doll's eye maneuver; 2) Caloric stimulation.[20]

The doll's eye maneuver is described as such because it is reminiscent of the mechanical movement of the eyes of a doll, i.e. when the reflex is intact, sudden rotatory movement of the head stimulates the semicircular canal initiating the reflex and induces conjugate deviation of the eyes in the opposite direction. Caloric response is most readily elicited by irrigating the external auditory canal with ice water for about 30 seconds. The intact reflex system produces nystagmus with the fast component away from the irrigated ear.

The caloric test is of significance in evaluating the integrity of the brain stem and is of exceptional value in helping to separate the organic from the psychogenic coma. In the deeper stages of cortical depression, the reflex from caloric stimulation and the doll's eye response become exceptionally brisk. With brain stem involvement, the reflex becomes perverted and the response depends upon the level of involvement of the brain stem.

Although several forms of respiration associated with central nervous system pathology have been described,[21,22] there are three basic types which are of value in the evaluation of the comatose patient. These are: 1) Cheyne-Stokes Respirations (CSR): 2) Central Neurogenic Hyperventilation (CNH); and 3) Ataxic Breathing.

Cheyne-Stokes Respiration (CSR) is a regular pattern of alternating periods of respiration and apnea. The respirations increase in depth to a point, then decrease in depth in a regular pattern. This type of respiratory pattern is most commonly seen with deep lesions of the cerebral hemispheres or diencephalon which are often bilateral.

Central Neurogenic Hyperventilation (CNH) is characterised by deep, regular, rapid respirations without alteration in depth or rate and there are no periods of apnea. This respiratory pattern denotes dysfunction at the ponto-mesencephalic levels. Unlike in metabolic acidosis, (Knussmaul breathing), these respirations have a forced expiratory component to them.

The third form of respiratory pattern is that which has been labelled as ataxic breathing. This type of respiration is irregular in rate and depth; there is, in addition, no pattern to the rhythm or periods of apnea. Ataxic breathing indicates medullary dysfunction and is a sign of impending respiratory arrest. Reversible coma, associated with ataxic respirations, demands respiratory assistance and a tracheostomy is therefore recommended.

Laboratory Evaluation

There are several laboratory examinations which are of value in determining the origin of coma and are of invaluable assistance to the physician in reaching an accurate diagnosis. These are presented here only in terms of indications and relative value of such examinations.

Basic blood values to be drawn immediately when one is faced with a comatose patient and the etiology is obscure include:

1) BUN;
2) Blood Sugar;
3) Serum Electrolytes;
4) CBC; and
5) Alcohol and barbiturate levels.

These basic studies, as well as a routine urinalysis, serve as a guide to possible extracranial system dysfunction and certain exogenous intoxicants as the precipitating factor of the coma.

Other useful studies to be utilised include:

1) ECG;
2) Echoencephalogram;
3) Brain scan;
4) EEG; and
5) Spinal puncture.

ECG

The electrocardiogram should always be performed in patients with alterations of consciousness since failure of cardiac output induced by myocardial infarction, arrhythmia, chronic heart failure, and valvular disease is a common precipitating factor of alterations of consciousness, especially in patients

over the age of 50. It must be kept in mind, however, that intracranial pathology, especially trauma and subarachnoid hemorrhage, produce alterations in the ECG, varying from arrhythmias to ischemic patterns.[23]

Echoencephalogram

The echoencephalogram utilises ultrasound to detect shifts in midline structures and is of particular value in cases of traumatic origin. However, it must be kept in mind that this study is only as good as the technician performing the procedure and negative results never rule out intracranial mass lesions.

Brain Scan

The use of isotopes to ascertain intracranial pathology is of relative value in terms of definitve diagnosis. Positive scans are most commonly seen with tumors, hematomas and ischemic lesions. Again, a negative study does not rule out an intracranial pathology. One must also be cautioned that a positive scan will be seen from scalp trauma and it often remains positive for six to eight weeks[24] after the injury.

EEG

The electroencephalogram is invaluable in detection of intracranial pathology, especially with diffuse cortical depression. The electroencephalogram is often able to separate cortical dysfunction due to primary[25] brain pathology from systemic disorders and drug ingestion. The EEG is also of great value in separating the organic from the psychogenic origins of alterations of consciousness.

Spinal Puncture

Lumbar puncture of the subarachnoid space, when indicated and if carefully performed, has a definite place in the diagnosis and management of comatose patients. The usual precautions will have to be undertaken. Fundoscopy is of paramount importance to rule out papilloedema. In those cases where intracranial mass lesion are suspected, be they tumor, hematoma or cerebral edema from any cause including infarction, the risk of herniation is always present. It is our experience that rises in intracranial pressure that have occurred slowly over a period of weeks to months, e.g. a neoplasm is more dangerous from this point of view than cases, say, of subarachnoid hemorrhage. This may be a function of the moulding that occurs in the region of the brain stem from a chronic increase in pressure above the region of the brain stem.

Further examination of the cerebrospinal fluid is indeed of great value in the diagnosis of primary demyelinating disorders and other metabolic disorders of the central nervous system as well as in central nervous system syphilis. However, in these situations, the puncture can be performed selectively after one is sure that the patient is stable and mass lesions have been ruled out, thus avoiding the risk of acute herniation of the brain stem. In all cases with papilloedema, with suspected mass lesions, it is recommended that mass lesions be ruled out by means of EEG, brain scan, or contrast studies prior to the examination of the cerebrospinal fluid.

When spinal puncture is performed, it is necessary to obtain all available information at the time, as indicated by the patient's clinical state. This includes:

1) Carefully measured opening and closing pressures when the patient is relaxed, and after care has been taken to be sure that the patient's knees or a pillow are not compressing the abdomen, since this will give falsely high readings.
2) Specimen of cerebrospinal fluid is to be examined for: a) Cell count and morphology; b) Protein; c) Seriology; d) Colloidal gold and e) Sugar concentrations.
3) In addition, in cases of suspected infection, CSF should be examined for: a) Smear; b) Routine culture and sensitivity; c) India ink preparations; d) TB and fungal cultures and also Giemsa stains for possible malignant cells, as the latter not uncommonly produce a carcinomatous meningitis with altered sensorium.

In those cases where cerebrospinal fluid sugar content becomes crucial in an infection, a blood sugar estimation done at the time of the lumbar puncture could help to interpret the results.

4) In cases of suspected primary central nervous system metabolic disorders, a specimen should be sent for gamma globulin estimation where this study is available.
5) The Queckenstedt test (jugular compression) should not be done as a routine during lumbar puncture. It only serves to increase the venostasis within the brain and aggravates the patient's condition further. Its use is not recommended in the investigation of patients in coma.

In the initial management of the comatose patient, the function of the physician in the face of coma must be to prevent a deterioration of an already serious situation. This requires primary attention to the airway. The insertion of a plastic oral airway is recommended in all patients in coma even if they

exhibit a stable respiratory pattern associated with adequate pulmonary ventilation and oxygenation. When poor ventilation, cyanosis or ataxic bleeding is present, nasal tracheal or endotracheal intubation is indicated, followed by tracheostomy when the patient has stabilised. In deep coma with poor ventilation, respiratory assistance may be indicated. Often in these cases, external ventilatory assistance is advisable with a manual mask resuscitator prior to the intubation. Also, prior to intubation, 0.4 to 0.8 mgms. of Atropine sulphate administered intravenously is recommended to minimise vagal reflexes during intubation. After an airway has been established, pharyngeal and tracheal suction should be undertaken and repeated as frequently as necessary to obtain patency or the tracheobronchial tree. In addition, the patient should be maintained on his side or prone unless a cuffed endotracheal or tracheostomy tube is in place. This ensures drainage of the respiratory tract, and offsets the chances of an aspiration.

In addition to pulmonary ventilation, hemorrhage and shock are the other problems which can produce death and irreversible brain damage most readily in these patients.

Hemorrhage outside the nervous system, needless to say, must be controlled and blood replacement instituted. A patient himself in shock with spontaneous respiration is always a suspect for an extracranial cause for the shock. If hemorrhage is not overt, then four quadrant abdominal taps are recommended to exclude an intra-abdominal bleed. Hypotension accompanying alterations of consciousness can be due to drug ingestion, cardiac dysfunction, dehydration, or anoxia. When necessary, vasopressors are indicated and it is recommended that systolic pressure not be taken over 100 to 110 mm. of mercury initially.

The remainder of the management can be divided into two groups: 1) Traumatic and 2) Non-traumatic induced coma. (Tables V and VI)

Coma of traumatic origin requires initially supportive management with attention directed to the airway and hemorrhage, as outlined above. The necessity for a general physical evaluation cannot be overstressed. Blood sugar, electrolytes, CBC, and urinalysis should be obtained, whenever able in these patients, in order to have a base line prior to anesthesia and surgery. Intravenous fluids should be started with Ringer's Lactate (whole blood when indicated). An echoencephalogram is of value and may delineate the site of pathology.

When otorrhea or rhinorrhea is suspected, the

TABLE V
CLINICAL MANAGEMENT OF TRAUMA INDUCED COMA

Primary evaluation and management

1. Maintain airway
2. Control hemorrhage
3. Maintain patient on side to prevent aspiration
4. History
5. General physical evaluation
6. Neurological evaluation
7. Laboratory studies as indicated
8. IV fluids
9. Foley catheter to drainage
10. Type and cross-match for whole blood
11. Echoencephalography
12. Skull and cervical spine X-rays

Secondary evaluation and management

1. Neurosurgical evaluation
2. Radiographic examination
3. Radiographic contrast studies as indicated
4. Conservative management or surgery as indicated

TABLE VI
CLINICAL MANAGEMENT OF COMA NOT OF TRAUMATIC ORIGIN

Primary evaluation and management

1. Maintain airway
2. Reverse shock
3. Maintain patient on side to prevent aspiration
4. History
5. General physical evaluation
6. Neurological evaluation
7. ECG
8. Laboratory studies as indicated by history – BUN, CBC, blood sugar, urinalysis, ETOH and barbiturate levels, electrolytes, liver profile, blood gases.
9. Spinal puncture when **infection is suspected**
10. Initiation of therapy directed at systemic disorders affecting brain function

Secondary evaluation and management

1. Skull X-ray
2. EEG
3. Brain scan
4. Echoencephalogram
5. Radiographic contrast studies when indicated
6. Spinal puncture when indicated (see text)

drainage should be tested with "Dextrostix", for the presence of sugar. A positive test would indicate a cerebrospinal fluid leak. Neurological evaluation should be performed to ascertain the degree of injury and level of function. When the patient is stable, skull and cervical spine X-rays are recommended in all cases of coma of traumatic origin. These studies should be accomplished with the least amount of movement possible. Supportive measures should be maintained, vital signs checked frequently and neurosurgical evaluation obtained as soon as possible.

Coma of non-traumatic origin requires the same initial supportive measures, as well as a general and neurological evaluation. History is the most pertinent guide to the initial "work-up". When only a limited history is available, it is wise to obtain a urinalysis and blood for CBC, electrolytes, glucose, BUN, and levels of common exogenous toxins, e.g. barbiturates and alcohol. Blood gases and a liver profile are often of value.

After blood is drawn, it is recommended that intravenous fluids be started with 5% dextrose and water and that 50 c.c. of 50% dextrose solution be given to reverse hypoglycemia if present. The intravenous fluids should be changed in accordance with the results of the electrolyte determinations. Gastric lavage is indicated only if ingestion has occurred within four hours. In patients with no gag reflex, lavage should be done only after a cuffed endotracheal or tracheostomy tube has been inserted and the cuff inflated.

As outlined earlier in this discussion, spinal fluid examination at this time is recommended only when infection is strongly suspected or when the history and examination findings point to a subarachnoid hemorrhage without focal findings. Otherwise, lumbar puncture should be postponed until the patient is stable and intracranial mass lesions have been definitely ruled out by means of EEG, brain scan, echoencephalogram and contrast studies.

In summary, this discussion has been directed at: 1) Defining the terms used to describe the alterations of consciousness in a manner that can be communicated meaningfully; 2) Presenting a brief discussion as to the pathophysiology and clinical conditions responsible for the problem; 3) Providing a useful, rational guide to the initial evaluation and management of the alterations of consciousness.

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Environmental Health Aspects of Metropolitan Planning and Development

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THE RELATIONSHIPS BETWEEN health and sanitation on the one hand and city and regional planning on the other are not readily understood or appreciated by planners.

As far back as 1948, the First World Assembly adopted a resolution recommending collaboration with the United Nations Social Commission, and United Nations Economic Commission for Europe, and other regional commissions of the United Nations with special reference to the hygiene of housing, is an endeavour to obtain adequate representation in any international scheme for town and country planning or for the improvement of housing. There is a constant need for co-ordination and co-operation between the various agencies responsible either directly or indirectly with urban planning and development.

The various factors related are –

(i) Vocational training for building trades and improvement of productivity in the construction industry; co-operative housing and workers' housing;

(ii) Problems related to co-operative housing and workers' housing;

(iii) Improvement in housing, also through the development and use of forest products in building and through the assistance of home economists in plans for family housing;

(iv) Studies on the social aspects of housing and of urban development; and

(v) School building research and training programme for officers connected with development programmes.

Perhaps no problem facing mankind at the present time is of greater concern than that of uncontrolled urban development. The population is increasing at a rate never before experienced and this is occuring at a particularly serious rate in all the developing countries. The greatest pressure is being felt in urban and metropolitan areas due to the huge rural-urban migration.

This rapid increase in the urban situation is not necessarily due to the fact that people like living in them, but because of their apparent efficiency as centres of industrial production.

Before the end of this century, the world's population will have more than doubled. Since only 8-10% of the population will be able to produce sufficient food the rural-urban drift will continue and the vast majority of the 3,000 million new people added to the present population of the world will be urban dwellers. The problem of the cities will continue to become more and more serious with the passage of time.

Urbanisation has its good points as well as its bad ones. It affords opportunities for improving the standards of living, education, housing, social satisfactions, and public health.

But uncontrolled and unplanned development and encroachment on space (in terms of land, air and water) will lead to an increase in the danger of a spread of disease, and the threat to health from noise, overcrowding, and the general degradation of man's physical, psychological and social environment.

In spite of this potential danger, there remains the real hope that mankind, in the years to come, can

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achieve great advances. For the first time ever, man is equipped with knowledge and material to control his environment. He can use recent advances in science and technology to the benefit of the human race. Never before have so many people and so many nations been concerned about human welfare and the human condition.

The problems to be faced in social and economic development are tremendous, but man has the ability to be equal to the challenge.

Because of its complex and varied nature, urban development requires numerous agencies to work together to achieve the common objective of a well planned situation. Due consideration must be given to public health, safety, convenience and welfare, and to the development of the aesthetic, economic, social and cultural potential of metropolitan areas.

Our world is in the midst of a fundamental transformation, from agricultural and rural life to a highly urbanised way of life, with industry as a chief source of livelihood.

The movement of people from country to town is not a new phenomenon, but the rate at which this is happening is causing alarm.

The economic and technological resources needed for the job at hand are staggering. In India, for instance, up to U.S. $22,000 million will be required in a period of 25 years, to house new inhabitants of cities with over 100,000 people. Similar figures for Malaysia are not available. The cost of improvement to the traffic system in Kuala Lumpur alone is estimated to cost U.S. $50 million and if this can be taken as an indication of the extensive costs involved in urbanisation programmes, one can well imagine the cost of improving the total environment in urban areas. The sudden and unprecedented pouring of rural people into unprepared urban areas within developing countries may reach catastrophic dimensions within the next decade. Because of the low earning capacity, poor nutrition, and crowding of the people, the danger of disease becomes especially menacing in the tropical and sub-tropical regions of the world.

It creates, in fact, a far greater possibility of disaster than the Industrial Revolution of a century ago. What makes for even graver danger is, first, the climatic differences between the temperate and tropical or sub-tropical zones of the earth and the different disease vectors associated therewith, and, secondly, but not necessarily in a secondary sense, the educational unpreparedness of the emerging countries.

Although it might appear that technical knowledge is available to deal with any mass disease situation that may arise, the fact is that there are not enough adequately educated people at the present time to meet even the every day problems of the growing communities of the developed countries, let alone the developing countries.

It cannot be denied that the health and sanitary aspects of urban areas are fairly well known, and we may even conclude that there need be no further investigation. This is, in fact, not so. The most important consideration is money. Costs are high and funds are usually limited. What we must search for is not merely a method of control, but a feasible method of control; a solution, therefore, must be within the economic and operational reach of the people to be protected or within the financial and administrative capacity of the country concerned, or, failing this, within the operational capacity and willingness of the international community.

Although it may be argued that scientific discoveries will, ultimately, make all things possible, the implementation of a scientific discovery is a technological undertaking, calling for understanding of the use of man, money, and materials. Indeed, it is doubtful if technology can ever overtake science.

Planning for environmental health

It is most gratifying to note that there is better appreciation of a rational approach to social and economic development. There is an acceptance of long-range economic, social and physical planning.

The lack of proper guidelines is the basis of the inherent difficulty faced by planners in the complex task of thinking constructively about the metropolitan area of the future. It is important to think of the metropolitan area as a coherent whole and to recognise the interplay of social, political and economic factors which must be taken into consideration.

It is not often appreciated that the health agencies have an important responsibility in planning. If planners are to develop effective programmes, they must rely on the environmental health profession. There is no more effective way of justifying planning standards than to relate them directly to health standards. The problem faced by the planners is, therefore, in the final analysis, an environmental health problem.

The establishment and maintenance of a healthful environment, particularly in urban areas where environmental problems are most acute and require the bringing together of the social, biological and physical sciences concerned with the health aspects of man's relationships with his environment, involve safe-

guarding water, air, food, conveyances, dwellings and the recreational and living environment. It involves not only the control of the quantity and quality of the basic necessities, but also, importantly, the control of waste by-products, whether solid, liquid or gaseous. These by-products, if left uncontrolled, would lead to widespread disease.

A study of the public health problems associated with metropolitan development and urbanisation, particularly in the developing nations, reveals that the more immediate problems are to be found in the environmental health field. This is confirmed by recent studies and surveys carried out in various parts of the world by the United Nations and its specialised agencies specially. These problems pertain to the inadequacy of water supplies and of waste disposal, the unhygienic condition of housing and of the residential environment, the poor selection of sites and layouts for residential neighbourhoods and industry, and the pollution of the environment by noxious chemical and microbiological agents. Other problems closely related to environmental health include zoning, urban renewal, land use, subdivision regulations, school and recreational facilities, and vehicular traffic. These activities are all of great concern to public health administration.

Objectives

While the objectives of both planners and environmental health workers is to improve the health and well being of the people, it is of particular significance that they both lay emphasis on prevention rather than cure. This common purpose arouses the expectation that the urban population, through the positive and co-operative action of planners and public health officials, will have an urban environment in the future.

Community environmental planning must reach beyond the concept of simple disease prevention towards the long-range goal of comfort, efficiency, and the promotion of well-being. Sometimes these objectives are partially included within the meaning of aesthetics, but it is surprising how clearly the general public sees them as "health-related".

Conservation of Resources

To assure future generations of their birth right, immediate steps must be taken in many parts of the world to control gross pollution of air, land and water. Pollution is a major destroyer of resources. Even today, in many areas, pollution by faeces and sullage water is a direct menace to health. In other places, mine wastes, large-scale industrial dereliction, the indiscriminate dumping of garbage and refuse, erosion, sand and gravel pits are important factors contributing to destruction of resources.

Water pollution is perhaps the easiest form to recognise. Although water is a resource to be shared by industry, power, transport, agriculture, and communities as well as individuals, far too often self-interest prevails and irreparable damage results.

Air must be increasingly regarded as a world resource to be conserved and utilised in perpetuity.

Lines of Action

The possible lines of action for more effective environmental health planning programmes fall into three main categories:

(1) By the citizens and citizen groups;
(2) By institutions of higher learning, research centres and professional organisations.
(3) By governmental and intergovernmental agencies, including international groups.

Urban communities are, in fact, social systems and subsystems that develop and change as a result of the interplay of intricate, at times almost bewildering, combinations of forces or influences – technological, social, economic and intellectual. Though it is only too well-known that changes occuring in one part of a system leads to changes in other parts of the system, yet there are far too many examples of public administration throughout the world where public administrators and their non-official allies are pressing ahead with their own programmes with little heed or concern for their by-products or side effects. This is particularly true of re-housing projects where relatively large numbers of individuals, families and businesses are moved without sufficient thought being given to the side effects of such moves.

Organising for Action

Planning is useless if means are not provided for its implementation. These means normally include legislation, finance, organisation, professional co-operation, and public understanding and support. No universal formula for planning urban and metropolitan areas can be suggested.

In conclusion, I wish to say that the problems arising out of urban living will continue to increase and the challenges must be met with sound planning and determined efforts, so that the great advantages accruing from urbanisation will far outweigh the disadvantages.

In this way, we will reduce the environmental hazards to a minimum and make urban living a thing to be cherished.

Renal Diseases in Pregnancy:
A clinical review and appraisal

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THE HUMAN FEMALE presents with the problem of higher risk of developing renal tract infections than her male counterpart. This has been primarily incriminated to the alterations in the renal tract resulting from the effects of pregnancy.

Renal Function in Pregnancy — A Physio-Anatomical Consideration

The physiological and anatomical changes that occur in the cardio-vascular and renal systems during pregnancy and puerperium can confuse the picture sufficiently to render it difficult to evaluate renal function tests in these patients. Further, the close proximity of the parturient canal to the renal tract renders the latter highly susceptible to infection, with resultant impairment of renal function, during or following pregnancy.

The salient physio-anatomical considerations with reference to renal functions in pregnancy are as follows:

(i) The progressive haemo-dilution, occurring after the first trimester of pregnancy, results in the fact that the level of blood urea during pregnancy remains much lower than in a non-pregnant adult. Blood urea concentrations of 15 to 25 mgm.% are the normal expected values during pregnancy and early puerperium, whereas a blood urea level of 30 mgm.% which is normal for a non-pregnant patient, is abnormal during pregnancy.

(ii) In pregnancy, the renal blood flow levels are increased by as much as 40 to 50% above the non-pregnant value of about 1 to 1.2 litres per minute. This will result in increased urinary output, which is one of the physiological causes for increased frequency of micturition in pregnancy. Further, the haemodilution and increased urinary output can contribute to difficulties in the interpretation of the results of renal clearance tests, i.e. urea clearance, inulin clearance and creatinine clearance tests, in pregnancy.

(iii) The significance of albuminuria in pregnancy can be difficult to evaluate as this could be due to toxaemia of pregnancy, renal tract diseases, postural albuminuria, or to contamination of the urine by the increased vaginal discharge (normal or abnormal) in pregnancy and puerperium.

(iv) In pregnancy, the renal threshold for glucose is lowered, and the evaluation of the significance of glycosuria is thereby rendered difficult.

(v) In pregnancy, especially after the fourth month, there is progressive dilatation of the renal collecting systems, viz calyces, renal pelvis and ureters, resulting in about 3-fold increase in the storage capacity, and subsequent urinary stasis. This renal dilatation is the result of two main factors, viz: (a) compression effect of the gravid uterus upon the ureters at the pelvic brim, and (b) passive relaxation effect on the ureteric musculature by progesterone and "relaxin" like substances. The dilatation of the renal tract

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results in urinary stasis, which can predispose to ascending urinary tract infection.

(vi) The increased lymphatic connections between the bladder and kidneys, and between the colon and the kidneys, are incriminated as factors predisposing to the spread of infection from the bladder and colon (E. Coli) to the kidneys.

(vii) The short length of the female urethra (in contrast to the male) results in a close anatomical proximity of the urine in the bladder to the skin of the external genitalia, perineum, vaginal and anorectal contents. This state increases the risk of the urine in the bladder becoming infected from pathogenic bacteria from these secondary sources.

(viii) The extremely close proximity of the urethra, bladder and ureters to the parturient canal, especially the vaginal and the gravid lower uterine cavity, renders the lower renal tract highly susceptible to trauma during the intrapartum period. Such trauma is especially liable to occur during manipulative vaginal delivery, such as forceps, ventouse and destructive operations, Caesarean section delivery and prolonged labour. Trauma, in its turn, will predispose to renal tract infections.

Routine Screening of Urine in Asymptomatic Pregnant Patients

(i) **Albumin:** Routine testing of urine for albbuminuria in pregnant patients is undertaken at all antenatal visits, as a screening test for toxaemia of pregnancy. However, the presence of albuminuria could indicate urinary tract infection or other forms of renal pathology. It is also essential to exclude postural albuminuria and albuminuria from contamination by vaginal discharge before evaluating the significance of albuminuria.

(ii) **Glycosuria:** Routine testing of urine for glycosuria in pregnant patients is undertaken at all antenatal visits, as a screening test for diabetes mellitus. However, glycosuria could be physiological, such as would be the result of lowered renal threshold ("renal glycosuria") or lactosuria of late pregnancy and puerperium. Incidence of physiological renal glycosuria and lactosuria have been reported to occur to as high a frequency as 30 to 35% of pregnant patients. Hence, to confirm the diagnosis of diabetes mellitus, the detection of glycosuria should be followed-up by a glucose tolerance test.

(iii) **Pus cells:** In most clinics, the presence of more than five pus cells per micro-litre or per "high-power field" of urine is considered as diagnostic of urinary tract infection and is used as a screening index.

(iv) **E. Coli:** E. Coli is the commonest organism being held responsible for more than 95% of urinary tract infections in pregnancy, (Pinkerton et al, 1967). However, most pregnant women excrete some E. Coli in their urine, even without evidence of urinary tract infection. Pinkerton et al (1967) advocated the use of the T.T.C. (Triphenyl Tetrazolium Chloride) Test, as a screening test, to detect the presence of a significant number (100,000 or more per ml. urine) of E. Coli in the urine that will constitute evidence of urinary tract infection. However, this test does not seem to have come into wider useage despite its strong advocacy by Pinkerton et al (1967) at the Fifth World Congress of Gynaecology and Obstetrics, held in Sydney in September 1967.

Renal Diseases in Pregnancy

In Table I is presented the common types of renal diseases that may be seen in pregnant patients. Of all the renal diseases seen in pregnancy, pyelonephritis, both acute and chronic, and acute renal failure are the most commonly associated renal complications of pregnancy. In fact, pregnancy and its complications can predispose, in their turn, to pyelonephritis and acute renal failure in the obstetric patient. A fuller discussion of their inter-relationships is not possible in this review paper. The other six renal conditions, listed in **Table I**, are relatively unrelated to the pregnancy state, and are coincidental associations. However, such cases invariably create their individual problems in diagnosis and management, discussion of which is again limited in this paper. Further, there is little doubts that the pregnancy state can have adverse effects on the pre-existent renal disease process, as well as the fact that the pre-existent renal disease state can have adverse effects on the pregnancy.

Acute Renal Failure in Obstetrics and Gynaecology

In obstetrics and gynaecology, there seems to be a higher predisposition to the occurrence of acute renal failure. In **Table II** (above) is presented a comprehensive list of all the common clinical causes of acute renal failure that can be encountered in obstetrical and gynaecological practice. Severe and prolonged state of shock, irrespective of the cause, can readily result in acute renal failure from renal ischaemia, due to inadequate renal perfusion. Fulminating septicaemia, especially following septic abortions, fulminating toxaemia of pregnancy including eclampsia, and severe concealed accidental haemorrhage are, by far,

the three major causes of acute renal failure in modern obstetrical and gynaecological practice. Fulminating infections of the renal tract and severe intravascular haemolysis from mis-matched blood transfusions, bacterial septicaemia (Cl. Welchii) and chemical agents, are at present less commonly encountered in good obstetrical and gynaecological practice. Acute urinary tract obstruction and nephrotoxic agents are rarely encountered as causes of acute renal failure in present-day obstetrical and gynaecological practice.

Pathology of Acute Renal Failure

The pathology of acute renal failure, irrespective of the clinical aetiology, may be schematically presented as shown in **Figure 1,** which is self-explanatory. In obstetrics and gynaecology, acute renal circulatory insufficiency is the commonest and most important of the mechanisms of acute renal failure; and acute tubular necrosis, which is usually secondary to a state of acute renal circulatory insufficiency, is the second common mechanism of acute renal failure. However, most of the obstetrical and gynaecological aetiological factors of acute renal failure can also become operative by directly precipitating acute tubular necrosis. Acute bilateral cortical necrosis is a rare pathological cause of acute renal failure, and is classically seen in cases of severe concealed accidental haemorrhage (Bourne et al, 1962 and MacGillivray, 1950).

It is not possible to discuss the management of acute renal failure in full in this paper. The salient aspects of the management should inco-operate the following:-

(i) Treat and alleviate the underlying cause of acute renal failure.
(ii) Maintenance and control of fluid and electrolyte balance.
(iii) Control of blood non-protein nitrogenous levels (blood urea etc.).
(iv) Prevention of infection (provision of good nursing).
(v) Implementation of renal dialysis (haemodialysis or peritoneal dialysis) when the indication arises.

Chronic Nephritis and Pregnancy

The occurrence of pregnancy in a patient with chronic nephritis is a rare association; but when it does arise, the outlook for both the mother and the foetus are extremely poor.

The management of patients with chronic nephri-

TABLE I
RENAL DISEASES IN PREGNANCY

(1) Pyelonephritis in Obstetrics
  (a) acute
  (b) chronic
(2) Acute Renal Failure in Obstetrics
(3) Acute Nephritis (Bright's Disease)
(4) Nephrotic Syndrome
(5) Chronic Nephritis
(6) Renal Tuberculosis
(7) Polycystic Disease of the Kidneys
(8) Renal Calculi

TABLE II
RENAL FAILURE IN CLINICAL AETIOLOGY OF ACUTE OBSTETRICS AND GYNAECOLOGY

1. Shock
  (i) Neurogenic.
  (ii) Oligaemic.
  (iii) Bacteraemic.
  (iv) Anaphylactic.
2. Septicaemia.
3. Fulminating PET/Eclampsia.
4. Concealed Accidental Haemorrhage.
5. Fulminating Renal Infection.
6. Intra-Vascular Haemolysis
  (i) Blood Groups
  (ii) Bacteria
  (iii) Chemicals
7. Acute Urinary Tract Obstruction.
8. Nephrotoxic Agents.

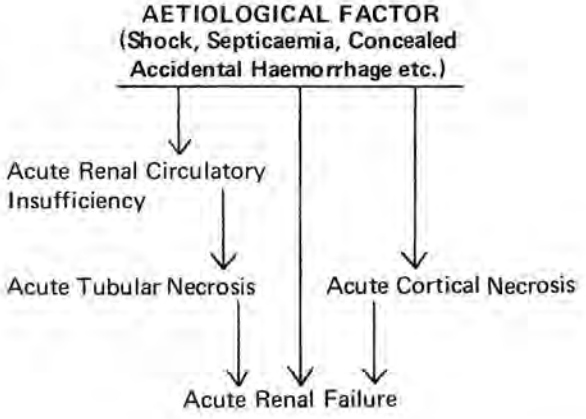

TABLE III
CHRONIC NEPHRITIS
Differential Diagnosis between PET and Chronic Nephritis with pregnancy

1. Definite history of having had acute nephritis.
2. Albuminuria in early months of pregnancy (before the 20th week)
3. Albuminuric retinitis constantly present.
4. Repeated urine examinations will reveal:
   (a) Consistently low S.G. below 1010.
   (b) Persistent albuminuria throughout pregnancy.
   (c) Presence of Hyaline, and occasionally granular and epithelial casts.

TABLE IV
CHRONIC NEPHRITIS
Pre-pregnancy Renal Investigations

1. Intravenous pyelography *
2. Repeated Blood Urea Investigations
3. Urea-concentration Excretion Test
4. Water Concentration Test
5. Microscopy of the Urine.

TABLE V
CHRONIC NEPHRITIS
Findings in Favour of a Successful Pregnancy

1. The absence of Albuminuria.
2. A normal or slightly elevated B.P.
3. A Blood Urea not exceeding 30 mgm.%
4. A Urea-Concentration of over 2Gm%.
5. A constantly high S.G. of the urine – over 1010.

tis and pregnancy constitute a problem even in the best hands. In order to attain maximal maternal and foetal salvage in these cases, the medical and obstetrical skill should be of the highest standard. In addition, their care involves excellent team-work between obstetrician, physician, biochemist and nursing personnel.

The salient aspects of the problem of chronic nephritis and pregnancy can be presented in summary-form in the undermentioned four tables (**Tables III, IV, V and VI**). These tables are self-explanatory.

TABLE VI
CHRONIC NEPHRITIS
Findings in Favour of an Unsuccessful Pregnancy

1. Persistent and/or severe albuminuria.
2. Continued and increasing hypertension.
3. Blood Ureas exceeding 40 mgm.%.
4. Urea-concentration of less than 2Gm%.
5. A persistently low S.G. of Urine of 1010 or less.
6. Increased retinal changes.
7. Supervention of severe PET or even mild PET in very early pregnancy (24th week).

Summary

1. A review of renal function in pregnancy, on a physio-anatomical basis is presented.
2. The significance and pitfalls, involved in the routine screening of pregnant patients for renal pathology, are discussed.
3. The pattern of renal diseases encountered in the pregnant patient, is tabulated.
4. The problem of acute renal failure in obstetrics and gynaecology is comprehensively reviewed and appraised.
5. The salient aspects of the relatively rare clinical problem of chronic nephritis and pregnancy are tabulated.

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The ABO Blood Group Frequency Distribution of Kuala Lumpur based on a Blood Donor Sample

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Penang.

Introduction

THE ABO BLOOD GROUP frequencies of the non-aboriginal population of Western Malaysia has been studied by Simmons et al[1] (1950), Schebesta[2] (1952) and Poon Wai Lum and Amarasingham[3] (1968). The studies in all these cases have been on small numbers. The ABO group frequencies of the population of Singapore has been more thoroughly studied by Allen et al[4] (1947), Gibson-Hill[5] (1953) and Yeoh[6] (1960) and in 1962 K.T. Chan[7] (1962) published data on a large-scale survey of blood donors in Singapore. The population of Western Malaysia is composed mainly of Malays, Chinese and Indians. The ethnic background of the non-aboriginal population of Western Malaysia is similar to that of Singapore. It is expected that the ABO blood group distribution among the various races in Western Malaysia would be similar to that of Singapore.

Aim

The purpose of this study is to establish the ABO group frequencies among Malays, Chinese and Indians of Kuala Lumpur, the capital of Western Malaysia, using a large sample of normal healthy people. The results would be useful in the study into the possible association of various diseases to blood groups in the different races living here and also in evaluating the value of blood group evidence in crimes where blood grouping results are involved.

Procedure

(a) Materials and Methods

The records of the Blood Transfusion Unit, General Hospital, Kuala Lumpur were examined and all blood donors registered between May 1961 and August 1970 were taken as the sample in the present series. The donors may be regarded as normal adults for the reasons given below.

(b) Acceptance of Donors

A blood donor is accepted by the Blood Transfusion Unit on the following criteria:—

(i) Donors must be between 18 and 60 years of age.
(ii) Donor's weight must be more than 100 lbs.
(iii) Donors should have no recent history of having malaria, jaundice or other serious illness.
(iv) Their haemoglobin level must be over 85%.
(v) Prior to the donation, the doctor assesses the donor to make sure that he is physically fit to donate blood.

(c) Determination of Blood Group

The blood is separated into cells and serum and both these entities are used to determine the blood group. The cells are tested against commercially prepared Anti-A and Anti-B sera, and the blood group result is cross-checked by testing the serum against standard A cells and B cells on a tile.

TABLE Ia

Year by year distribution of ABO blood groups among the three major locally domiciled ethnic groups in Kuala Lumpur.

YEAR	MALAYS								
	Total No. of Groupings Done	ABSOLUTE NUMBERS				PERCENTAGES			
		O	A	B	AB	O	A	B	AB
1961 (May–Dec.)	1,113	510	250	284	69	45.8	22.5	25.5	6.2
1962	1,496	640	375	395	86	42.8	25.1	26.4	5.7
1963	1,340	534	308	396	102	39.85	23.0	29.55	7.6
1964	1,483	636	353	377	117	42.9	23.8	25.4	7.9
1965	1,476	685	332	376	83	46.4	22.5	25.5	5.6
1966	1,217	509	300	343	65	41.8	24.65	28.2	5.35
1967	1,582	776	372	345	89	49.1	23.5	21.8	5.6
1968	1,624	754	399	366	105	46.4	24.6	22.5	6.5
1969	1,765	751	460	453	101	42.55	26.05	25.7	5.7
1970 (Jan.–Aug.)	1,460	655	360	361	84	44.85	24.65	24.7	5.8

YEAR	CHINESE								
	Total No. of Groupings Done	ABSOLUTE NUMBERS				PERCENTAGES			
		O	A	B	AB	O	A	B	AB
1961 (May–Dec.)	714	349	171	164	30	48.9	23.95	22.95	4.2
1962	998	472	234	232	60	47.3	23.45	23.25	6.0
1963	1,005	461	262	241	41	45.8	26.1	24.0	4.1
1964	935	457	231	209	38	48.8	24.7	22.4	4.1
1965	871	429	209	202	31	49.25	24.0	23.2	3.55
1966	619	295	148	153	23	47.7	23.9	24.7	3.7
1967	697	326	182	163	26	46.8	26.1	23.4	3.7
1968	620	297	156	143	24	47.9	25.15	23.05	3.9
1969	713	325	184	171	33	45.6	25.8	24.0	4.6
1970 (Jan.–Aug.)	414	196	102	94	22	47.4	24.6	22.7	5.3

Criteria for Ethnic Division

The ethnic group chosen for study are the major race groups residing in Kuala Lumpur (Western Malaysia) and the criteria for ethnic sub-division adopted in the present survey is as follows:–

(1) Malays: All Malays of Western Malaysia.

(2) Chinese: All Chinese from any part of China.

(3) Indians: Northern and Southern Indians, Pakistanis and Ceylonese.

Results

In all, a total of 29,040 blood groups were available for analysis, comprising 14,556 Malays, 7,586

TABLE Ia

YEAR	INDIANS							
	Total No. of Groupings Done	ABSOLUTE NUMBERS				PERCENTAGES			
		O	A	B	AB	O	A	B	AB
1961 (May–Dec.)	549	220	127	168	34	40.1	23.1	30.6	6.2
1962	793	327	153	257	56	41.2	19.3	32.4	7.1
1963	637	255	139	193	50	40.0	21.8	30.3	7.9
1964	709	306	147	204	52	43.2	20.7	28.8	7.3
1965	701	296	141	224	40	42.2	20.1	32.0	5.7
1966	593	242	146	156	49	40.8	24.6	26.3	8.3
1967	838	369	184	237	48	44.0	22.0	28.3	5.7
1968	807	374	156	229	48	46.35	19.3	28.4	5.95
1969	752	357	144	210	41	47.5	19.15	27.9	5.45
1970 (Jan.–Aug.)	519	227	113	152	27	43.7	21.8	29.3	5.2

TABLE Ib

The distribution of ABO blood groups among the three major locally domiciled ethnic groups in Kuala Lumpur. (1961 to 1970)

Ethnic Origin	Total No. of groupings done	Absolute Numbers				Percentages			
		O	A	B	AB	O	A	B	AB
Malays	14,556	6450	3509	3696	901	44.3	24.1	25.4	6.2
Chinese	7,586	3607	1879	1772	328	47.5	24.7	23.3	4.3
Indians	6,898	2973	1450	2030	445	43.1	21.0	29.4	6.4

TABLE II

Gene frequencies of the three Major Ethnic groups in Kuala Lumpur

Ethnic Group	p	q	r
Malays	16.21	16.98	66.82
Chinese	16.04	15.21	68.76
Indians	14.51	19.60	65.89

Chinese and 6,898 Indians. The results are summarised in **Tables 1a and 1b** which gives the absolute numbers in each ethnic and blood group and the corresponding percentages.

The ethnic distribution of the donor sample is compared to the ethnic distribution of the general population of Kuala Lumpur in **Table VI.** From it, it will be seen that the majority of donors are Malays, although they form the lowest percentage of the population of the Kuala Lumpur district. It has been

TABLE III
The ABO group and Gene frequencies of Malays in this series compared with other investigations in Singapore and Malaysia.

Series	Place	Population	Authors	Numbers	Percentages				p	q	r
					O	A	B	AB			
1.	Kuala Lumpur West Malaysia	Malays	Present Series 1970	14,556	44.3	24.1	25.4	6.2	16.21	16.98	66.82
2.	West Malaysia	Malays	Poon Wai Lum & R.D. Amarasingham 1967	616	36.86	25.16	31.98	6.0	17.837	22.027	60.136
3.	Singapore	Malays	K.T. Chan, 1961	5461	38.42	20.07	29.23	7.29	17.70	20.26	62.04
4.	Perak	Malays	Schebesta, 1952	44	30.09	20.45	43.18	2.27	—	—	—
5.	Singapore	Malays	Gibson-Hill, 1953	42	40.48	16.67	40.48	2.38	—	—	—
6.	Singapore	Malays	Allen & MacGregor 1947	1963	40.45	26.54	25.67	7.34	18.65	18.11	63.24

noted that the reason for this is that they are the ones who come forward to donate more willingly. They also form the majority of large donor groups such as the Police, Army and the Dusun Tua Youth Camp. The percentage of Chinese donors is very small compared to the population percentage.

The gene frequencies were calculated by the formulae of Fisher's method which were published by Dobson and Ikin in1966 and Roberts in 1948. The results are summarised in **Table II.**

The formula is given below:—

$$p = \frac{t-s}{v} \quad q = \frac{u-s}{v} \quad \text{and } r = \frac{s}{v}$$

where $s = \sqrt{O}$

$t = \sqrt{O + A}$

$u = \sqrt{O + B}$

and $v = t + u - s$

in which O, A, and B are the actual number of cases in Groups O, A and B in the sample.

Discussion

(a) The year-to-year variation was found to be slight in the distribution within each group.

(b) The three ethnic groups were found to have different frequency distribution. The Chinese have a relatively high group O frequency with about the same A and B group frequencies. In the case of the Indians, there was a significantly large distribution of Group B over Group A, 29.4% against 21.0%. In the case of the Malays the difference of Group B over Group A was a much smaller amount, a difference of 1.3%.

(c) **Tables III to V** give a comparison of the ABO group frequencies found in the survey compared with the investigation of other workers on similar ethnic groups in Western Malaysia and Singapore. The results indicate that there is not much variation between the Chinese and Indian ethnic groups living in these territories. The frequency distribution of the Malays show differences particularly with the B group where the percentage was found to be 25.4%, which is rather lower than that of the Poon Wai Lum and R.D. Amarasingham series and the K.T. Chan series which were 31.98 and 29.25% respectively. It is, however, very similar to that found by Allen and Macgregor which was 25.67%.

Summary

The ABO group distribution among the main race

TABLE IV

The ABO and Gene frequencies of Chinese in this series compared with other investigations in Singapore and Malaysia.

Series	Place	Population	Authors	Numbers	Percentages				p	q	r
					O	A	B	AB			
1.	Kuala Lumpur W. Malaysia	Overseas Chinese	Present Series 1970	7,586	47.5	24.7	23.3	4.3	16.04	15.21	68.76
2.	West Malaysia	Overseas Chinese	Poon Wai Lum & R.D. Amarasingham 1967	940	45.85	23.29	25.54	5.32	15.409	16.776	67.755
3.	Singapore	Overseas Chinese	K.T. Chan, 1961	15,262	43.53	25.99	24.99	5.48	17.363	16.741	65.896
4.	Singapore	Overseas Chinese	Allen & MacGregor, 1947	624	43.11	24.04	27.72	3.13	15.87	18.09	66.04
5.	Malaya	South Chinese	Simmons et al, 1950	250	46.4	25.2	20.8	7.6	16.5	13.9	68.1
6.	Singapore	Overseas Chinese	Yeoh, 1960	1,000	44.30	25.5	26.5	3.70	16.79	17.39	65.82

TABLE V

The ABO group and Gene frequencies of Indians in this series compared with other investigations in Singapore and Malaysia.

Series	Place	Population	Authors	Numbers	Percentages				p	q	r
					O	A	B	AB			
1.	Kuala Lumpur W. Malaysia	Indians, Pakistanis & Ceylonese	Present Series 1970	6,898	43.1	21.0	29.4	6.4	14.51	19.60	65.89
2.	West Malaysia	— do —	Poon Wai Lum & R.D. Amarasingham 1967	826	34.87	22.03	35.84	7.26	16.308	24.931	58.761
3.	Singapore	— do —	K.T. Chan, 1961	5,000	39.02	21.02	33.60	6.36	14.983	22.700	62.317
4.	Singapore	Northern Indians	Allen & MacGregor 1947	1,478	34.30	24.97	32.61	8.12	18.22	23.04	58.74
5.	Singapore	Southern Indians	Allen & MacGregor 1947	389	31.88	76.73	34.45	6.94	18.70	23.62	57.68

TABLE VI

Distribution of Donor samples and population in the District of Kuala Lumpur among the three major locally domiciled Ethnic groups. (Adjusted to the criterion for ethnic division adopted in the study)

Ethnic Group	Population*		Donor Sample	
	Number	Percentage	Number	Percentage
Malays	124,815	16.79	14,506	50.13
Chinese	493,765	66.42	7,586	26.12
Indians	124,815	16.79	6,898	23.75

*Estimated population for year 1968 — information from Department of Statistics, Malaysia.

in Kuala Lumpur, West Malaysia, has been determined using a large sample from data collected from the Blood Transfusion Unit of the Kuala Lumpur General Hospital.

Acknowledgement

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An Outbreak of Hongkong Influenza in a Youth Camp in West Malaysia

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IN JULY, 1968, an epidemic of A2/Hongkong influenza occurred throughout West Malaysia and subsided towards January, 1969. However, it did not abate altogether but continued to appear in 1969 both sporadically, mainly among the urban population, and in small isolated outbreaks in institutions, including the University of Malaya. The causal agent in all the cases investigated in Kuala Lumpur was found to be the same Hongkong/68 influenza virus strain.

Towards the end of the first week of April, 1970, another outbreak of acute respiratory disease (ARD) was observed at the National Pioneer Youth Corps Training Centre at Dusun Tua, Ulu Langat, Selangor. The health authorities and the Institute for Medical Research (IMR) were called in to investigate and A2/Hongkong influenza was again confirmed as being responsible. Shortly after, the outbreak spread outside the camp limits to involve schoolchildren and their contacts in Selangor. The same virus was isolated again.

This paper describes in detail the investigation of the influenza outbreak in the Dusun Tua camp.

The National Pioneer Youth Corps Training Centre

The Dusun Tua camp is situated along the Langat River in a valley and is 16 miles by road from Kuala Lumpur. The river forms its western and southern boundaries and towards the north is a swamp and a padi field. There were rubber trees and jungle to the east but these were cleared in August, 1969 to enlarge the camp.

About 3,000 youths, aged between 16 to 25 years, are enrolled four times a year. The first batch was recruited in October, 1969. They come from all the states of W. Malaysia and stay in the camp for the whole duration of their training programme, which is three months. The staff, instructors and others form a separate group of about 200 individuals, most of whom come to the camp daily from Kuala Lumpur and Kajang.

Medical care for staff and trainees is provided for by one medical officer and four medical assistants. The personnel make full use of the facilities provided and it can be assumed that all affected by the influenza outbreak came forward for treatment.

Occurrence of the cases

The influenza outbreak started on 6 April, 1970, reached its maximum proportion within four days and was over in approximately three weeks. The attendance at the clinic more than doubled during the first week of the outbreak as is shown in **Figure 1.** The increase was due almost exclusively to acute respiratory disease. Prior to the epidemic only 3% of the population reported daily for various complaints.

FIGURE 1
CLINICAL ATTACK RATE OF INFLUENZA
AT THE DUSUN TUA YOUTH CAMP, APRIL 1970

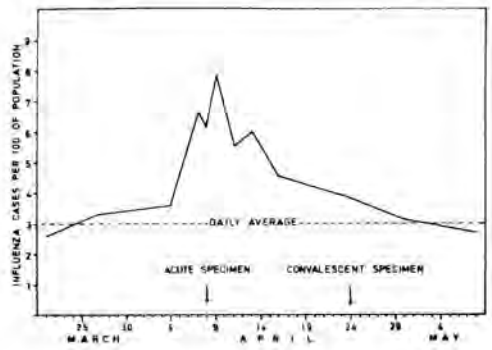

Fig. 1

During the first week of the outbreak, more than 6% with an influenza-like illness were seen daily. An estimated total of 2,800 persons was affected during the three weeks of the outbreak, giving a clinical attack rate close to 80%. More than 120 badly ill persons had to be hospitalized in the sick-bay for periods varying from two to seven days.

TABLE 1

Presenting symptoms of 200 patients with influenza at the Dusun Tua Youth Camp.

Symptoms	No. of patients	Rate %
Fever	180	90
Cough	150	75
Headache	90	45
Weakness/Malaise	40	20
Chills & Rigors	40	20
Chest-pain	39	20
Coryza	31	15
Giddiness	30	15
Sore-throat	28	15
Myalgia	24	12
Abdominal pain	12	6
Vomiting	10	5
Ocular pain	6	3
Profuse perspiration	5	3
Nausea	3	2
Arthralgia	4	2
Diarrhoea	1	0.5

On 13 April, when the epidemic was settling down, one patient turned up with typical 'flu-like symptoms. He was treated as an outpatient like all the other 'flu cases. Later, however, he developed neurological symptoms suggestive of meningo-encephalitis and went into coma III.

Throat swabs and paired sera were taken for investigation. No influenza isolate was obtained from the throat specimen, neither was the patient positive for influenza, serologically. Subsequently, however, arbovirus studies proved him to be a case of Japanese encephalitis.

Clinical Observations

The onset of the disease was abrupt in most cases with headache, nasal stuffiness and fever as the prodromal symptoms. The duration of the illness was from three to five days in uncomplicated cases. In others, it lasted up to ten days. The clinical features were studied in 200 patients. Half of these were the acutely ill cases which were hospitalised and the other half consisted of 100 outpatients selected at random. An analysis of the main symptoms is given in **Table 1.**

The most frequently encountered feature noted in 90% (180) of the cases was fever and this was associated with chills and rigors in 20% (40) of the cases. The temperature on admission ranged from 100° F to 103° F and the highest recorded during the first two days of the illness was 105°F. The fever subsided within three days in most cases but lasted up to one week in those with complications. Although cough was the second most frequent symptom affecting 75% (150) of the cases, it was mild and non-productive. About 20% (39) had sub-sternal chest pain accentuated by this dry cough. Almost 50% (90) had headache, mainly frontal, although a few had retro-orbital pain made worse by upward and lateral movements of the eye-ball. Sore-throat and coryza did not feature very prominently in this outbreak. Diffuse myalgia was more common than arthralgia. Many complained of faintness and giddiness – some actually collapsed and fainted on the field during physical training.

On examination, a large proportion presented with flushed faces and a hot dry skin but a few perspired profusely. Watery eyes with conjunctival injection

was a common feature. Some had injection of the pharyngeal wall and tonsils as well. Evidence of bronchiolitis and pnuemonitis, in the form of scattered rhonchi, wheezes and moist rales was found in 35–40% (77) of the cases. A few had clinical evidence of pulmonary consolidation. These chest complications accounted for almost half of the cases admitted. No other complications were noted and there were no fatalities.

Recovery was complete in three to four days in uncomplicated cases. Antibiotics were used for cases with secondary chest-infection. Recovery in these cases took almost ten days. Convalescence was prolonged by post-infection asthenia, malaise, anorexia and depression.

MATERIALS AND METHODS

Collection of specimens

Two throat swabs were taken from each of 8 acutely ill patients during the early phase of the disease and placed in 5 ml. of chilled Hartley's broth containing a final concentration of 625 units of penicillin and 125 ugm. of streptomycin per ml. Paired (acute and convalescent) sera were collected from 6 patients for serological investigation by haemagglutinin-inhibition (HI) tests. Two patients were bled during the acute phase of the disease but were not available when the convalescent specimen was due. All the specimens were transported in thermos flasks containing ice.

Virus isolation and identification of isolates

Each throat swab specimen was inoculated into six 10-day-old embryonated chicken eggs. The volume of inoculum was 0.2 ml., about 3/4 of which was introduced into the amniotic and 1/4 into the allantoic cavity. Allantoic and amniotic fluids were collected after three or four days of incubation at 35 C and tested for the presence of haemagglutinating (HA) agents by a spot test. This consisted of 0.25 ml. of 0.5% fowl RBC added to 0.25 ml. of amniotic or allantoic fluid. A cell control made up of 0.25 ml. of the fowl RBC and 0.25 ml. of 0.85% phosphate buffered saline was also set up. Readings were taken after the mixtures were allowed to stand at room temperature for 45–60 minutes when the control cells had settled to a button.

Specimens with a negative spot test were given one more amniotic/allantoic passage before they were disregarded. Those showing a positive spot test were titrated by the HA test. Further passages were performed to increase low titres.

Isolates were identified by the HI test (Davenport and Minuse, 1964) using 8 haemagglutinating units of virus. Each isolate was tested against the following hyperimmune rooster sera, some of which were supplied by WHO and some prepared in this laboratory from virus seeds also supplied by WHO:

Polyvalent A	A2/Taiwan/1/64
Polyvalent B	A2/Malaya/302/54
A2/Hongkong/8/68	B/Singapore/3/64
A2/Malaysia/1/68	

Serology

Paired sera of the 6 patients were tested for rise in HI antibodies. All sera were inactivated at 56 C for 30 minutes prior to treatment with Receptor Destroying Enzyme (RDE) to remove non-specific inhibitors. The method adopted was that recommended by the WHO International Influenza Center for the Americas.

The sera were tested in the HI test against the following virus strains:

The respective patient's isolate	A2/England/878/69
A2/Malaysia/1/68	A2/Taiwan/1/64
A2/Hongkong/8/68	B/Switzerland/265/67

RESULTS

Identification of the isolates

HA agents were isolated from all the eight throat swabs collected and high HA titres were attained by all the isolates after the first or second amniotic/allantoic passages.

The eight isolates were identified as Influenza Type A2 viruses and were found similar to the 1968 Hongkong strain (Table 2).

Antigenic comparison of a representative Dusun Tua strain (F2/70) with other A2 strains showed that of the current (1968–69) viruses, F2/70 is more closely related to A2/Hongkong/68 and A2/Malaysia/68 strains than to A2/England/69 strain, which has been reported by Dr. H.G. Pereira of WHO World Influenza Centre as representing a "drift" from the prototype Hongkong strain in its antigenic characteristics **(Table 3).**

TABLE 2

Typing of Influenza Isolates of the Dusun Tua patients, by HI test

Rooster Antisera	HI Titre* and Antigen							
	F1/70	F2/70	F3/70	F4/70	F/5/70	F6/70	F7/70	F8/70
A2/Mal/1/68	160	80	80	160	160	160	80	320
A2/HK/8/68	640	160	320	320	320	640	160	320
A2/TW/1/64	<10	<10	<10	<10	10	<10	<10	10
B/S'pore/3/64	<10	<10	<10	<10	<10	<10	<10	<10
A2/Mal/302/54	<10	<10	<10	<10	<10	<10	<10	<10
Polyvalent A	1280	320	640	640	640	1280	320	640
Polyvalent B	<10	<10	<10	<10	<10	<10	10	<10
HA units used	8	8	8	4	4	8	8	4

* Titre expressed as reciprocal.

TABLE 3

Antigenic Comparison of the Dusun Tua Isolates with other A^2 Influenza Strains

Virus	HA Units used	Rooster Antisera				
		A2/TW/1/64	A2/HK/8/68	A2/Mal/1/68	A2/Eng/878/69	F2/70
A2/TW/1/64	8	*640*	<10	10	40	<10
A2/HK/8/68	8	<10	*640*	160	80	640
A2/Mal/1/68	8	<10	640	*320*	80	640
A2/Eng/878/69	8	<10	80	80	*320*	80
F1/70	8	<10	640	160	80	320
F2/70	8	<10	160	80	80	320
F3/70	8	<10	320	80	80	320
F4/70	4	<10	320	160	80	320
F5/70	4	<10	320	160	80	320
F6/70	8	<10	640	160	80	640
F7/70	8	<10	160	80	40	160
F8/70	4	<10	320	320	80	320

Serological findings

Paired (acute and convalescent) sera obtained from six patients who yielded influenza isolates were tested for rises in HI antibody titre. The remaining two patients with isolates were not tested, because only single serum specimens were obtained from them. The paired sera of the patient (M.N.) initially suspected as an influenza case, but later found to be suffering from Japanese encephalitis, were also tested for influenza antibody rises.

All the six influenza patients did not show any residual antibodies to A2/HK/68 virus in their acute specimens but showed marked antibody rises (16– to

TABLE 4

Rises in Haemagglutin-Inhibiting Antibody among Influenza Cases at the Youth Training Camp, Dusun Tua

Patient	Serum	Patient's Isolate		HI Titre* and Antigen									
				A2/Mal/1/68		A2/HK/8/68		A2/Eng/878/69		A2/Taiwan/1/64		B/Switz/265/67	
		Titre	Rise	Titre	Rise	Titre	Rise	Titre	Rise	Titre	Rise	Titre	Rise
Z.A.	Acute	<10	128	<10	128	<10	>512	<10	128	20	64	<10	1
	Conv.	640		640		>2560		640		1280		<10	
S.G.	Acute	<10	16	<10	32	<10	16	<10	16	20	8	20	1
	Conv.	80		160		80		80		160		20	
C.T.	Acute	<10	32	<10	64	<10	64	<10	64	<10	64	20	1
	Conv.	160		320		320		320		320		20	
Y.B.	Acute	<10	64	<10	32	<10	64	<10	32	20	16	10	2
	Conv.	320		160		320		160		320		20	
Y.A.	Acute	10	32	<10	64	<10	64	<10	64	40	8	<10	1
	Conv.	320		320		320		320		320		<10	
M.B.	Acute	10	32	<10	32	<10	32	<10	32	10	8	<10	1
	Conv.	320		160		160		160		80		<10	
M.N.	Acute	No		40	1	40	0	20	0	20	1	<10	1
	Conv.	Isolate		40		20		10		20		<10	
H.K.	Units Used	8		8		8		8		8		8	

* Titres expressed as reciprocal, (+) Case of laboratory-confirmed Japanese encephalitis

greater than 512-fold) against their own isolate, A2/Mal/1/68, A2/HK/8/68 and A2/Eng/878/69 viruses **(Table 4)**. The rise against A2/TW/64 virus was less marked (8– to 64-fold). No significant rises were detected against the B virus. M.N. did not show any rise at all to any of the influenza strains and was therefore not similarly infected as his fellow inmates of the camp.

Discussion

From past experiences, when a significant antigenic change in the current influenza virus strain takes place, resulting in a new variant, major epidemics occur but soon after, because of acquired immunity to the new virus, the outbreaks peter out and usually do not make their reappearance until another "shift" occurs.

In W. Malaysia, the Hongkong 'flu virus caused a nationwide epidemic in August, 1968. All age-groups were affected but the majority of the patients were adults, both young and old.

Unlike previous epidemics, however, where the new variant strain ceased to be active after a few months, the Hongkong 'flu virus persisted in the general population, especially in Kuala Lumpur, for more than two years after the 1968 epidemic. The serological results **(Table 4)** of the infected youths in the Dusun Tua camp showed that none of the influenza patients had any appreciable residual HI antibody to A2/HK/68 virus prior to the infection in 1970, and preliminary findings of a post-Hongkong influenza survey (still in progress in this laboratory) seem to indicate poor antibody response to the A2/HK/68 virus in the general population. However, this does not necessarily explain the continued presence of the infection in the country as it has been found (Rapmund et al., 1959) that circulating antibodies in the blood are not a reliable index of protection from clinical disease, as persons possessing little or no demonstrable antibody may escape infection to which others with relatively high antibody titres are susceptible.

Antibodies produced locally at the site of infection (e.g. those found in saliva, sputum and nasal washings in respiratory diseases) have been shown to reflect better the extent to which an individual is protected against infection (Mann et al., 1968; Cate et al., 1966 and Smith et al., 1966). As it is not known whether or not those infected after the 1968 epidemic in Malaysia had antibodies in their respiratory secretions, the reasons for the persistence of the A2/HK/68 virus in the Malaysian population remain obscure.

Summary

This paper describes an influenza outbreak in April, 1970, at the National Pioneer Youth Corps Training Centre, Dusun Tua, Selangor (16 miles from Kuala Lumpur). A2/Hongkong virus, closely related to the prototype strain, was isolated from all the eight patients investigated and was serologically proved responsible for the infections.

The outbreak represented yet another of the several minor outbreaks in Kuala Lumpur caused by the same virus which had persisted for more than two years after the initial epidemic in 1968. No obvious reasons for this could be given.

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Metabolic Changes in Protein Malnutrition

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ONE OF THE MAJOR CONCERNS in the world today is the adequacy of food supply in relation to population growth. The prevalance of protein malnutrition and its serious consequences are well recognised today in many of the developing countries. For man, the most important changes are those due to insufficient intake of protein or intake of protein of poor quality. Among the more general signs of protein malnutrition in human beings are poor growth, lack of resistance to infection and a high mortality rate.

Individuals who have a marginal nutrient intake may have a low level of vitality and health and these may eventually result in subclinical nutritional deficiency symptoms. Often, the severe deficiency state will become obvious only when they have prevailed for a long period of time. It is important to identify these conditions in the early stages before the tissue changes have advanced to a point where they cause irreversible damage.

One of the objectives in studying the metabolic changes in protein malnutrition is to be able to detect the existence and also to measure the extent of protein deficiency. Persons who have subsisted on insufficient intakes of protein undergo progressive biochemical changes which precede the clinical manifestations of malnutrition. The present paper will consider some changes which may be detected in vivo and which can serve as methods for evaluating protein nutrition in population groups.

The sequence of biochemical changes expected to occur in the organism when it is subjected to conditions of protein lack or protein restriction are (a) reduction in the metabolic expenditure of body proteins which can be considered as a process of metabolic adaptation; (b) decrease in the so-called protein reserves with some tissues suffering more than others; and (c) decrease in protein moieties with key metabolic functions such as enzymes, resulting in highly abnormal biochemical and physiological function.

Biochemical methods may be employed to test three aspects of protein nutrition:-

1) The relative adequacy of dietary intake
2) Metabolic changes due to tissue malnutrition
3) Depletion of body stores of protein

The biochemical methods used for the above purposes involve quantitative determination of nutrients or related metabolites in such tissues as blood and urine. The interpretation of results requires a knowledge of the metabolism of amino acids and protein, including their storage in the body, the possibility of synthesis and the mode of excretion.

The primary function of proteins in diets is to supply amino acids. The quality of a protein depends upon its amini acid composition. In adult animals only amino acids are absorbed, there being no appreciable transfer of proteins across the intestinal wall. There is intimate mixing of nitrogen derived from the food with that derived from the catabolism of tissue proteins and the plasma amino acid pattern represents

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the resultant of the two processes. Calorie intake has a substantial influence on protein utilisation, in that calorie restriction results in substantial increase in nitrogen excretion due to utilisation of tissue proteins for energy. Conversely, addition of carbohydrates or fat to calorically inadequate diets produces increased nitrogen retention.

Adaptive enzyme changes

It is known that in man as well as in the rat, the level of protein intake influences the activity of some enzymes concerned with amino acid metabolism. These changes may reasonably be regarded as adaptive and part of a compensating mechanism, since their effect must be to economise on amino acids when supplies are short.

In rats on a low protein diet, there is an increase in the activity of the amino acid activating enzymes and a decrease in the activity of the urea cycle enzymes in the liver. The effect of these two changes in rats would presumably be that, on a low protein diet, an amino acid entering the liver would have a greater chance of being incorporated into protein and a smaller chance of being degraded to urea than in those on a normal protein intake. Thus the organism adjusts to a low protein intake not by a reduction of overall nitrogen turnover, but by an alteration or diversion of metablic pathways such that a smaller proportion of the available nitrogen is excreted and a larger proportion is used for protein synthesis.

We must visualise that a large proportion of the amino acids liberated by catabolism may be resynthesized to proteins in the same cell, without ever entering the blood stream. In the ideal state, there would be complete re-utilisation with no loss or wastage at all.

Reduction in metabolic expenditure of nitrogen

The output of nitrogenous substances in the urine has long been know to vary with the nitrogen intake. Nitrogen excretion in urine is high when protein intake has been high and vice versa. In normal subjects, urea may account for 80 – 90% of the total urinary nitrogen. Changes in urea excretion account for the observed changes in urinary excretion of nitrogen, since other urinary nitrogen constituents remain stable for all practical purposes under these conditions. The excretion of endogenous urea fluctuates with the size of the nitrogen metabolic pool, so that in general urea excretion is high when the reserves are maximum and low whn they are depleted. When individuals live on chronically sub-optimal intakes of protein, their metabolic expenditure of body proteins is reduced.

In malnourished subjects, total urinary nitrogen is low and the amount of urea nitrogen both absolute and relative is reduced. A low proportion of urea nitrogen might be regarded as evidence of protein depletion. Changes in the ratio of urinary urea to total nitrogen or creatinine essentially reflect variations in urea excretion, since the relative amounts of other urinary nitrogenous compounds are not altered significantly in malnutrition. Platt (1954) measured the urinary excretion of urea of children and lactating women of different nutritional and socio-economic conditions and found the ratio of urea nitrogen to total nitrogen to be markedly lower in the groups with poorer nutrition.

With spot samples of urine, the total nitrogen or urea nitrogen may be related to creatinine. The urea nitrogen/creatinine nitrogen ratio is an approximate index of dietary protein related to muscle protein stores. An index of 30 or lower in a random sample is indicative of malnutrition. Arroyave (1965) found the urea/creatinine ratio to be from 8 – 9 in preschool children of low socio-economic status and around 15 in children of the same age group belonging to the upper socio-economic group.

Serum Albumin

One of the immediate consequences of a reduced protein intake is a decrease in protein turnover. When protein intake is altered, changes in the fractional and absolute rates of albumin catabolism may occur without corresponding changes in serum albumin concentration. In malnourished infants, the fractional catabolic rate of plasma albumin is decreased. The synthetic rate is closely dependent on the protein intake. When the protein intake is reduced, there is an immediate fall in the rate of albumin synthesis, followed after a lag period of several days, by a fall in the rate of catabolism. There is also a transfer of albumin from the extravascular to the intravascular pool. These are mechanisms which tend to preserve the constancy of intravascular mass when the synthetic rate is reduced by a low protein diet.

The simple measurement of serum or plasma total protein concentrations will not reveal early stages of protein deficiency. Serum albumin is synthesised at the expense of other tissue proteins. When the deficiency is severe, there is detectable drop in total protein concentration. It appears that although a distinct fall in albumin concentration occurs only when the clinical picture ensues, some less evident decreases occur earlier. A fall in albumin concentration might be regarded as a sign of incipient exhaustion of protein reserves. It is to be appreciated that the possibility of plasma volume changes makes the interpreta-

tion of plasma protein concentration a difficult question.

In addition to albumin, other plasma protein fractions may also be reduced in malnutrition. Lack of transport protein in the plasma causes a marked depression in levels of plasma vitamin A. The concentration of growth hormone has been shown to respond rapidly to changes in dietary protein intake. Growth hormone produces a fall in the catabolic rate of albumin. Children recovering from malnutrition show decreasing levels of growth hormone 1 – 2 weeks after refeeding.

Muscle proteins and Creatinine

The body's ability to synthesise serum albumin is affected relatively late and the primary effect of protein lack is depletion of muscle tissue. When the amino aicd supply of the diet is inadequate, the body utilises proteins of some tissues to maintain the amino acid pool at a minimum, but yet compatible with the synthesis of other proteins of key metabolic importance.

Skeletal muscle proteins are a significant endogenous source of essential nitrogen under conditions of dietary protein restriction. Skeletal muscle forms only about 25% of the body weight in infants against 45% in adults. Muscle forms the largest protein reservoir of the body (20% protein). This reservoir is rather easily depleted and other tissues are spared at its expense. Although the overall calculated turnover rate of muscle proteins is slow, muscle is composed of many kinds of protein, covering a whole range of metabolic activities, some of which undoubtedly have a faster turnover than others. It is known that glucocorticoids exert a catabolic effect on the muscle and an anabolic effect on the viscera. This would make muscle a source of amino acids for enriching the amino acid pool and promoting protein synthesis in organs such as the liver (Waterlow, 1969).

Decreased muscle mass is a characteristic of children suffering from long-term protein malnutrition. Biochemically, restricted protein intake is reflected by a low urinary excretion of creatinine per unit of time. The muscle mass can be estimated by measuring the urinary excretion of creatinine. Estimation of the decrease in muscle mass is necessary for evaluating loss of reserve body proteins as maintenance of skeletal muscle mass is compatible only with adequate protein intake. Standard, Wills and Waterlow (1959) showed a statistical correlation between changes in creatinine excretion and muscle mass as estimated from skinfold thickness and limb circumference. Creatinine excretion is roughly proportional to body muscle mass in man and the daily excretion varies almost linealy with the body weight.

Borsook and Dubnoff (1947) demonstrated that 98% of the body creatine, the precursor of body creatinine, is in muscle. The rate of creatinine excretion must depend upon the amount or concentration of creatine in muscle and the rate at which it is irreversibly converted to creatinine. Creatinine for practical purposes is not affected directly by variations in protein intake.

The measurement of creatinine output is, in fact, of great value in the assessment of protein nutrition. It gives information about one of the most important reservoirs of protein in the body. According to Clark et al (1951), the creatinine coefficient (mg. creatinine/24 hrs/Kg body weight) is 22 (range 14.2 – 32.0) from birth to 24 months of age and 25 (range 16.3 – 36.2) in subjects aged between 2 years and 18 years. Arroyave and Wilson (1961) proposed that to relate creatinine excretion to body length, rather than weight provides a more sensitive index of the loss of muscle mass as the former index is unaffected by adipose tissue.

Plasma amino acid pattern

Children maintained experimentally for even a few days on a nitrogen free diet, show alternations in concentration of plasma-free amino acids – particularly valine, leucine, isoleucine and cysteine, this change being accompanied by an increase of some of the non-essential amino acids. There is good correlation between plasma amino acid levels and proportionate weight dificits, if this is not more than 30%. As body protein deficits are made good, fasting amino acid nitrogen increases.

In protein malnutrition, there is usually a fall in total amino nitrogen and most of the essential amino acids, particularly the branched chain amino acids. However, phenylalanine and lysine are much less affected. There is also a fall in some of the non-essential amino acids, particularly tyrosine. The concentration of most of the non-essential amino acids, notably glycine, alanine, proline, histidine, serine and aspartic acid, are well maintained or even increased.

In laboratory practice, a one dimensional chromatography is done on a fasting sample of serum, and it is usual to express the plasma amino acid pattern as a ratio of the non-essential to the essential amino acids. (Whitehead's ratio).

$$\text{Whitehead's ratio} = \frac{\text{glycine, serine, glutamic acid, taurine}}{\text{leucine, isoleucine, valine, methionine}}$$

According to Whitehead (1964) the ratio in healthy

children is less than 2. The ratio is increased in protein malnutrition and is high in kwashiorkor.

Mechanism of changes

Levels of non-essential amino acids are maintained simply because their carbon skeletons can be manufactured in the body. They compete with the dwindling amounts of essential amino acids for nitrogen made available by transamination. The concentration of lysine is maintained because it is not involved in transamination. The altered pattern of amino acids in plasma may lead to a distortion of the intracellular amino acid pattern and this, in turn, may affect the rate of protein synthesis.

Enzymes in protein malnutrition

The lack of amino acids is expected to interfere also with enzyme synthesis as a primary factor. Definite and consistent decreases in some enzymes have been found in severe protein malnutrition and alterations in amino acid metabolism have been demonstrated in kwashiorkor. They are probably due both to widespread metabolic changes, possibly caused by enzyme defects and to immediate inadequacy of dietary protein.

Hydroxyproline excretion

The results obtained by Widdowson and Whitehead (1966) in rats undergoing protein depletion suggested that significant rises in amino acid ratio did not occur as early as changes in hydroxyproline excretion.

Hydroxyproline peptides are excreted in the urine as a by-product of collagen metabolism. The amount of hydroxyproline excreted is closely related to the rate of growth. During growth, there is not only an increase in the amount of collagen, but also, as in bone, a continuous remodelling, so that catabolism of collagen accompanies synthesis. The measure of hydroxyproline excretion is a measure of the turnover rate of collagen. Increased excretion of hydroxyproline during growth results from the presence of increased amounts of metabolically active soluble collagen in tissues. Determination of hydroxyproline excretion is particularly valuable, since it is a dynamic measurement, which gives information about rates rather than absolute amounts and it is an estimate of growth rate.

Picou, Alleyne and Seakins (1965) showed that hydroxyproline excretion was generally reduced in undernourished children. Malnourished children excreted 14.6 mg/24 hours and normal children 32.7 mg/24 hours. This may be a useful index of growth failure, before there are significant changes in body weight. In the absence of growth, there is a reduction in the size of the soluble collagen pool. In malnutrition, the amount of collagen in the body is relatively increased and that of the cellular protein decreased. Collagen is stable in some tissues than in others and it does not share in the general depletion of body proteins.

Whitehead (1965) related the amount of hydroxyproline to that of creatinine in random specimens of urine. In normal children, the rate of hydroxyproline excretion decreases with increasing age, as the rate of growth falls off, while that of creatinine increases. Whitehead found that the reduction in the hydroxyproline index in malnourished children was statistically related to the weight deficit. He showed that the measurement of total urinary hydroxyproline was of value in assessing the nutritional status of communities of children. Those living on diets deficient in either protein or in the total calories excreted subnormal amounts of hydroxyproline.

$$\text{Hydroxyproline Index} = \frac{\text{mM hydroxyproline}}{\text{mM Creatinine/1/kg body wt}}$$

The index for normal children is between 2.0 and 5.0 (mean 3). Clinically malnourished children have indices between 0.5 – 1.5 (mean 1) and in marginally undernourished children, the index ranges between 1.0 and 2.0 (mean 1.5). Abnormal values are found in children deficient in both protein and total calories.

It is evident from the above that an understanding of the metabolic changes in protein malnutrition can contribute significantly to the assessment of nutritional status during the early stages of protein deficiency.

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Filariasis in a Rubber Plantation in Negeri Sembilan, West Malaysia:

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PREVIOUSLY, FILARIASIS has not been documented in the state of Negeri Sembilan, West Malaysia. Although isolated cases of elephantiasis have occasionally been reported, there has been no published confirmation of whether these cases originated in the state.

Following the recognition of a number of cases suggestive of filarial elephantiasis on a rubber and oil palm estate in the Port Dickson area, the Senior Medical and Health Officer, Negeri Sembilan, requested that a filarial survey of the estate be made by the Filariasis Division, Institute for Medical Research, Kuala Lumpur. This survey was performed in May 1970.

Description of the Site

Located in the Port Dickson district of the state of Negeri Sembilan, Sua Betong Estate lies some five miles from the coast, 10 miles southeast of Port Dickson town and 30 miles southwest of Seremban (see map). The estate contains 7,123 acres of rolling hills with several low-lying areas, some 400 acres in total, which become freshwater swamps during the rainy season. Most of the estate is planted in rubber, with about 12 percent devoted to oil palm. About one-fifth of the estate is bordered by jungle, the remaining portion surrounded by other rubber estates.

The estate is split into four geographical "divisions", (three agricultural and one factory and processing). Each division has its own residential areas. The workers' accomodations consist of ground level buildings without window screens.

The estate personnel, including dependents, numbered 2,165 at the time of the survey. Ethnically, about two-thirds were Indians, less than one-third were Chinese, and there were a small minority of Malays. The various ethnic groups were relatively evenly distributed among the four divisions and among the various occupations.

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Fig 1

Map of West Malaysia showing the location of Sua Betong.

Methods and Materials

Working in three independent units, a research team from the Filariasis Division, Institute for Medical Research, examined the estate personnel at several sites in or near housing divisions. All work was done between 1900 and 2300 hours on two successive days. Name, age and sex were recorded for each subject. Ethnic group was judged from the names, which are highly characteristic.

Employing a graduated capillary pipette fitted to a modified Sinton's pipette, a finger prick blood specimen of 20 cmm. was made for each subject. Films were dried overnight, then stained one hour with Giemsa (Revector, Hopkin & Williams Ltd., Chadwell Heath, England) using 35 drops of 7.6% concentrate per 100 ml. buffered distilled water, pH 7.2. The number and species of microfilariae were recorded for each film. The same films were assessed for malarial parasites by the Division of Malaria Research, Institute for Medical Research, Kuala Lumpur.

No studies of vectors or of microfilarial periodicity were attempted, and few clinical examinations were performed.

Results

Thanks to the fine cooperation of the Sua Betong Estate management and staff, 1,866 (87.1%) of the 2,165 persons on the estate were examined. Of these, 903 were males and 963 were females. Ages ranged from 20 days to 85 years. Ethnically, 65 per cent were Indian (non-Muslim), 30 per cent Chinese, and 5 per cent Muslims (mostly Malays but with a few Muslim Indians). The great majority were workers and families but some supervisory personnel were included. Among the 1,866 subjects, 96 (5.1 per cent) were positive for microfilariae, all morphologically consistent with the sub-periodic strain of **Brugia malayi.**

Microfilarial densities tended to be quite low as shown in **Table 1.** The highest was 100 microfilariae per 20 cmm.

The microfilarial carriers were unevenly distributed among the four geographical divisions as shown in **Table 2.**

As recorded in **Table 3,** microfilariae were found in persons in all age groups. The youngest positive was one year, the oldest 85 years. Proportionately, more males than females were found to be microfilariae carriers. Statistically, the difference in the proportions of microfilarial carriers among the two sexes is highly significant (chi square – 12.1, $P < 0.005$). This difference, however, is not noted in the children below 10 years of age.

The microfilarial prevalence rates vary among the three ethnic groups as shown in **Table 4.** The difference between the microfilarial rates of the Indians and Chinese is statistically significant (chi square – 9.2, $P < 0.005$). However, there is no significant difference between the proportions of microfilarial positives among the (non-Muslim) Indians and the Muslims (chi square – 1.34, $P = 0.25\pm$).

Search for malarial parasites revealed six positives among the 1,866 smears examined. This represents a prevalence rate of 0.3 per cent. Four of the parasites

TABLE I

Microfilarial density (per 20 cmm.) distribution among 96 infected subjects, Sua Betong Estate, Negeri Sembilan, West Malaysia.

	1–9 mff	10–19	20–29	30–39	40–49	50–59	60 & over
Number of subjects	60	19	8	3	1	2	3

TABLE 2

Distribution of microfilarial carriers among the four geographical "divisions" of Sua Betong Estate, Negeri Sembilan, West Malaysia.

Division	Number examined	Number positive	Percent positive
South	637	26	4.1%
Sg. Ujong	588	33	5.6%
Dodum	327	31	9.5%
Factory	314	6	1.9%
All divisions	1866	96	5.1%

were identified as **P. vivax,** one **P. falciparum;** one remained unidentified.

Discussion

As the data show, infection with sub-periodic **Brugia malayi** occurs in all age groups and in all ethnic groups among the personnel of the Sua Betong Estate, although microfilarial densities are generally quite low.*

* As a result of these findings the entire estate population, excluding infants and pregnant women, received treatment with diethylcarbamazine, 5 mg/kg body weight, once a week for six weeks.

The overall prevalence, 5.1 per cent, determined from a single blood specimen of 20 cmm. for each subject, is probably a considerable underestimate. Edeson (1959) demonstrated that where microfilarial loads were low, as in the present case, 25 per cent or more of the carriers may be missed if only 20 cmm. films are examined. However, 20 cmm. films remain useful if large numbers of subjects are surveyed in an untreated area.

Although clinical examinations were not generally performed on the subjects, several cases of minimal elephantiasis involving the feet and ankles were observed. In addition, some fifty cases, mostly mild, of filarial-type elephantiasis had been seen by the estate's medical staff. The degree of morbidity in terms of episodic adenolymphangitis and fever among estate personnel is unknown.

The finding that significantly more men than women have patent filarial infection is interesting, particularly as it is not noticed in children under 10 years old. This may reflect a difference in exposure, due to different occupational patterns or sleeping habits. Rubber tapping, begun about dawn, however involves roughly as many men as women. Men rather than women are apparently more apt to sleep outside the houses at night.

The differences of microfilarial prevalence in the various geographical divisions may reflect differences in exposure to mosquitoes. "Factory division" workers, who have the lowest prevalence, spend much less occupational time in the rubber forests. The reasons why Dodum division has the highest microfilarial rate are not presently clear. It has much less swampy area than the Sg. Ujong division.

The significantly lower microfilarial prevalence rate among the 557 persons of Chinese origin is difficult to explain. Most of these people have

TABLE 3

Distribution of microfilarial carriers by age group and by sex among 1,866 persons surveyed at Sua Betong Estate, Negeri Sembilan, West Malaysia.

Age group	Number of males examined	No. males positive	Percent males positive	Number of females examined	No. females positive	Percent females positive	Total number examined	Total number positive	Percent positive
0–4	131	3	2.3	137	3	2.2	268	6	2.2
5–9	159	6	3.8	165	10	6.1	324	16	4.9
10–14	154	17	11.0	161	4	2.5	315	21	6.7
15–19	105	7	6.7	126	4	3.2	231	11	4.8
20–24	60	9	15.0	80	3	3.8	140	12	8.6
25–29	53	4	7.6	49	1	2.0	102	5	4.9
30–34	69	3	4.4	69	0	0	138	3	2.2
35–39	34	2	5.9	45	1	2.2	79	3	3.8
40–44	41	3	7.3	28	0	0	69	3	4.4
46–49	21	2	9.5	29	1	3.5	50	3	6.0
50–54	23	1	4.3	39	3	7.7	62	4	6.5
55–59	23	2	8.7	27	3	11.1	50	5	10.0
60 and over	25	4	16.0	13	0	0	38	4	10.5
TOTAL	903	63	7.0	963	33	3.4	1,866	96	5.1

occupations similar to those of the other employees and share the same living quarters. They are relatively evenly distributed among the geographical divisions. Greater susceptibility of one racial group than another is not a demonstrated feature in filariasis. The living habits of the estate's various ethnic groups, such as use of mosquito nets, burning pyrethrin coils, or sleeping outdoors are not known.

Entomological studies to identify the important filarial vectors were outside the scope of the present survey; however, such work is planned by the Division of Entomology of the Institute for Medical Research.

Since the sub-periodic form of **B. malayi** naturally infects a number of animals as well as man, and may have an animal reservoir (Laing et al, 1960), a search for animal carriers in the region might be useful. According to a member of the estate management, leaf monkeys **(Presbytis spp.)**, which are known to be reservoirs elsewhere in West Malaysia, are found on the estate.

Sua Betong Estate is surrounded by similar rubber growing areas. It would seem likely that filariasis reaches beyond the estate boundaries. However, at present the extent of **B. malayi** infection and the degree of public health problem it presents in the

TABLE 4

Comparison of Microfilarial positive subjects by ethnic group, Sua Betong Estate, Negeri Sembilan, West Malaysia.

Group	Number	Number positive for microfilariae	Percent positive
Indian	1,218	77	6.3%
Chinese	557	16	2.9%
Muslim*	91	3	3.3%
Total	1,866	96	5.1%

* Mostly Malays but includes a few Indian Muslims.

surrounding region and in the state of Negeri Sembilan at large is unknown. Further investigation is recommended.

Summary

1. The status of filariasis in the state of Negeri Sembilan, West Malaysia, is unknown but has been assumed to be insignificant.
2. A total of 1,866 persons, representing 87.1 per cent of the total living on a rubber estate in Negeri Sembilan, were surveyed for microfilariae. An overall microfilarial rate of 5.1 per cent was found. All sub-periodic form of **Brugia malayi**.
3. The microfilarial carrier rates were unevenly distributed: men had a higher rate than women, ethnic Chinese had a lower rate than the other groups, and markedly different rates were found in different geographical divisions within the estate.

Acknowledgements

We wish to acknowledge the interest and assistance of the C.M. & H.O. and S.M.O.H. Negeri Sembilan; the Estate Division Manager, Kumpulan Guthrie Sdn. Bhd., Mr. J. Jefferies; the Sua Betong Estate Manager Mr. Samuel; the Estate Visiting Medical Officer, Dr. D.R. O'Holahan; and the Director of I.M.R., Dato (Dr.) Abu Bakar. We especially thank the staff of the Filariasis Division, I.M.R. for their able technical assistance. Finally, we acknowledge the assistance of Mr. Yap Loy Fong, Division of Malaria, I.M.R. for examining all slides for malarial parasites.

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A Survey of Ocular Injuries at the University Hospital

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Introduction

THE AREA AROUND the University Hospital is becoming industrialised with a rising population so that a survey of ocular injuries to identify main causes and to assess visual loss would be desirable.

Methods and Materials

This series of 335 accidents involving the eyes were seen at the Eye Clinic of this hospital over a period of 2½ years (October 1967 – March 1970) – the percentage of accident to new cases was 9.1% – see **Table I.** The injuries were nearly all uniocular – many of the damaged eyes had more than a single structure involved.

The orbit was X-rayed in cases with history of penetrating injury to exclude radio opaque I.O.F.B. The fundus was examined for any retinal lesions where possible. Gonioscopy to look for any damage to the filtering angle was done.

Results and comment

Sex: The ratio of males to females was 285:51 i.e. nearly 6:1 and this ratio rises higher in the industrial group. Even in the home, males are injured in a ratio to 2:1 to the females.

Age: Excluding the children, most of the victims belonged to the 15 – 35 age group (with a peak at the 20 – 25 age group). This economically active group accounted for 226 out of 335 i.e. 70% **(see graph).**

More than half the corneal F.B. and nearly half the superficial corneal lesions were seen in industry. **(Table III and IV).** Agricultural injuries (lallang, branch of trees, etc.) also contributed to one-quarter of the superficial corneal lesions.

Corneal perforations (42 cases) are associated with severe damage and a corresponding high incidence of severe visual loss. There are three main groups:-

(a) children accounted for 17, mainly resulting from sticks and stones
(b) industries 15, usually from handling wires
(c) vehicular accidents 10, caused by broken glasses.

The visual prognosis is better if there is no associated traumatic cataract or other serious injury. A number of the corneal scarring and perforations could be helped by corneal grafting.

The total number of traumatic cataracts is 44 – 36 from perforation and intraocular foreign body and 8 from blunt injuries **(Table III).** If the corrected vision after cataract extraction (or spontaneous absorption)

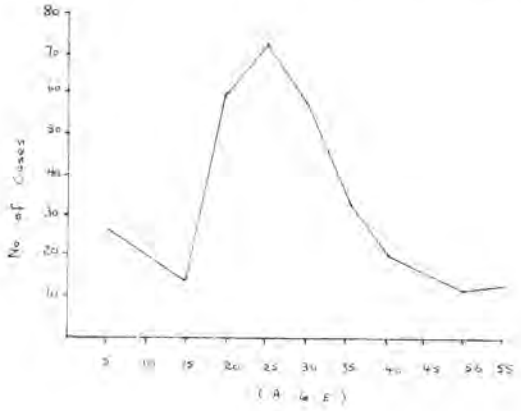

TABLE II
Classification of Accidents

Class	No. of Cases	Percentages of total
1. Industrial	157	47%
2. Home	39	11.5%
3. Sticks & Stones	38	11.2%
4. Vehicular	29	8.5%
5. Agricultural	27	8.3%
6. Sports	24	7.5%
7. Assault	15	4%
8. Firecracker	6	2%
	335	100%

TABLE I
Incidence

Year	Ocular Injuries	Total of New Cases	Percentage of injuries
1967 (Oct – Dec)	19	265	7.2
1968	128	1,014	12.5
1969	142	1,909	7.4
1970 (Jan – Mar)	46	383	12.0
Total	335	3,671	9.1

TABLE III
Nature of All Injuries

	No. of Cases
1. Corneal foreign body	87
2. Corneal (non-penetrating)	48
3. Conjunctival	46
4. Cut corneas with perforation	42 (26 with traumatic cataract)
5. Lid and adnexa	35
6. Hyphaema	23
7. Intraocular foreign body (I.O.F.B.)	17 (10 with traumatic cataract)
8. Retinal	10
9. Iris & ciliary body	9
10. Traumatic cataract (non-perforating)	8
11. Muscle paresis	4
12. Optic atrophy	3
13. Ruptured globe	3
Total	335

is better than 6/12, the patient was advised corneal contact lens (11 out of 19) to restore binocular single vision; however, a number could not afford contact lens.

Intraocular Foreign Body: Nearly all 14/17 occurred in industries – usually the result of the use of a hand hammer. Two-thirds of these have associated traumatic cataract. 50% of I.O.F.B. have visual acuity of less than 6/60. One quarter (4 out of 17) with between 6/18 and 6/60, only one-quarter are better than 6/12 and even this may deteriorate with time due to late complications.

Flash burns (Table IV): All 17 resulted from acetylene and oxyacetylene welding. The victims were usually bystanders and sometimes the welders themselves. Flying metal particles may cause pitting of the glass and so, obscure vision and this may lead to the goggles being removed. For casual visits to welding shops, spectacles of ordinary glass will absorb sufficient U – V light to prevent flash burn. Ideally, the welder should be separated from their neighbours by light proof screen.

Chemical Burns (Table V): There was only one case of severe visual loss and another of moderate loss

TABLE IV
Industrial Injuries

Nature of Industrial Injury	No. of Cases
1. Corneal foreign bodies	51
2. Superficial corneal	22
3. Lids and adnexa	16
4. Welding (flash burns)	17
5. Perforations	15 (with 7 traumatic cataract)
6. Intraocular foreign body (I.O.F.B.)	14 (with 8 traumatic cataract)
7. Chemical	13
8. Hyphaema	4
9. Retinal	2
10. Optic atrophy	1

TABLE V
Chemical Burns

Caustic soda	9	Industry	13
Acid	5	School	7
NH3	4	Home	5
Lime and cement	3	Assault	2
Miscellaneous	6		
	27		27

TABLE VI
Slight to Moderate Degree of Visual Loss 6/18 – 6/60

Cause	No.
1. Corneal scarring	11
2. Perforation	6
3. I.O.F.B.	4
4. Traumatic cataract	2
5. Retinal	2
6. Optic nerve damage	1
Total	26

TABLE VII
Gross Impairment of Vision 6/60

Cause	No. of blindness	Percentage of Incidence
1. Perforations	13	35%
2. Corneal scarring	8	22%
3. I.O.F.B.	8	22%
4. Traumatic cataract	2	7%
5. Ruptured globe	3	9%
6. Hyphaema	2	7%
7. Optic atrophy	1	3%
	37	

6/24 – both victims of assault – resulting in corneal scarring. All others had better than 6/12 vision. The good recovery was due to the awareness of the importance of washing the eye thoroughly – before it can do serious harm. Most of these cases had only conjunctival burns – a few with minimal corneal scarring.

Hyphaema: This is usually the result of blunt injury – missile inflicted (stones and sticks) in children and often may be associated with perforation. Lack of parental supervision may be a factor. Children do not realise these things.

Badminton makes up nearly 50% of the sports injury (11/24). This usually results from mishap with own partner (doubles) getting the shuttle-cock or the racquet. Most of the victims end up with hypnaema. The wearing of splinter proof glasses gives considerable protection. Turning around to look at one's partner during the game should be kept to a minimum. Apart from one serious injury (commottio retinae) from hockey, the others only received superficial lesion.

Home Injuries (11%) caused mainly by bumping into things, chopping firewood, combs, baby's fingers, falls in bathroom and striking against objects, splashing soda, hot cooking oil, hot water, etc. These largely caused superficial lesion.

Vehicular Accident (8.5%) (Table II). More than half (17) were superficial. One-third (10) were perforations and 2 optic atrophy. Safety belts could have

prevented the head being thrown forwards towards the wind-screen. In the motor cyclist, the wearing of protective goggles would have saved three perforating injuries.

Assault (4%) mainly caused by the fist, but the bone gives sufficient protection to the globe. The use of weapons, mainly sticks, has caused such serious injuries as commottio retinae, cataract and dislocated lens. Assault by chemicals have already been touched on.

Firecracker injuries: Only six were seen during the festive seasons of 1968 and 1969. Only two had hyphaema and the rest only superficial lesions. The suggestion that a tape recorder be used may be a safer alternative.

Visual results: Perforations, corneal scarring and I.O.F.B. head both the list of visual losses **(Tables VI and VII)**. This rather alarmingly high incidence of gross impairment, 11% of the total 37/335, is partly due to a number of serious injuries being referred to us from various parts of West Malaysia.

Accident prevention is improving in the industries. Guards on abrasive machines, presses and cutting devices, and eye guards are reducing the accidents but there are still innumerable tasks which require the use of a hand hammer. The high proportion of industrial eye injuries are caused by lack or failure of wearing suitable eye protection. Even when such protection is available, the workman is not infrequently reluctant to make use of it – he is unfamiliar with the injury.

Prevention of accidents: Propaganda and protection should be more readily available. They reduce wastage from industry. The insurance companies could spend some of the money it hands out as compensation in providing propaganda – tailored to the individual firm.

Conclusion

1. Seventy percent of the injuries occurred in the youthful age group of 15 – 35 years.

2. Males outnumber females in the ratio of 6:1 (even so to a lesser degree in the home). Females are more careful and less accident prone than the males doing the same work.

3. Industrial accidents accounted for 47% of the injuries – 157 out of 335.

4. Many of the damaged eye had more than a single structure involved as shown by perforating injury – 26 out of 42 showing traumatic cataract. More than one-quarter of all traumatic cataract can be helped to have binocular vision with contact lens.

5. One interesting feature is the spontaneous absorption of cataractous lens with hardly any trace of the lens. This rare feature (in a Western eye) occurred in three Chinese and two Malays, all under the age of thirty.

6. Perforation 35%, corneal scarring and I.O.F.B. 22% each, are the three commonest cause for gross loss of vision. If corneal grafting is freely available, a number of corneal scarring could be salvaged.

7. Sticks and stones account for over 10% of cases (38), mainly occurring in children. Children do not realise these things and explanations have to be given to them more than once.

8. Most industrial and some of the other accidents could be prevented if anti-accident propaganda and protection are more readily available.

Acknowledgements

We wish to thank the Medical Records Department of this hospital for helping with tracing these cases and Miss Yap for her assistance in the preparation of this paper.

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Isolation of *Plesiomonas Shigelloides* in Malaysia

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Introduction

FERGUSON AND HENDERSON, in 1947, reported the isolation of a motile, anaerogenic paracolon organism which they designated as C27, sharing common antigens with **Shigella sonnei.** In Ceylon, Schmid, Velaudapillai and Niles in 1954 reported the study of 12 C27 strains, 4 of which were from domestic animals (sheep, goat, cow and polecat). Bader (1954) described a bacterium, the O antigens of which were related to those of **Shigella sonnei** and assigned the micro-organism to the genus **Pseudomonas** on the basis of its polar monotrichous flagellation. But Ewing and Johnson (1960) suggested that the C27 organism was more closely related to the genus **Aeromonas.**

Habs and Schubert in 1962 (cited by Eddy and Carpenter 1964) suggested that a new genus should be created for this organism and proposed the name **Plesiomonas** (meaning neighbour monad, i.e. to **Aeromonas).**

The epithet shigelloides (shigella-like) was employed by Bader (1954) because the organism possessed common antigens with **Shigella sonnei.** The name **Plesiomonas shigelloides** has been retained despite the finding that only some of the strains have antigens common with shigella organisms, because the specific epithet was validly published according to the Bacteriological Code.

Cooper and Brown in 1968 isolated 38 strains of **Plesiomonas shigelloides** from the faeces of 36 children and 2 adults in Australia and they used the name **Plesiomonas shigelloides.**

The current paper describes the isolation of such an organism from the faeces of a 35-year-old male Chinese tin mine worker, admitted to the medical unit of the University Hospital in Kuala Lumpur.

Clinical features

The patient had a one-week history of a high, dry, remittent fever associated with chills, rigors, nausea and vomiting. For the first four days of his illness, he was constipated but subsequently he passed watery yellow stools once or twice a day.

There was no past history of significance. However, two months previously, his mother had a similar illness which lasted for 10 days.

On physical examination, he was febrile with a temperature of 39.8° C, was dehydrated and mildly jaundiced. His blood pressure was 140/70 mm.Hg.

The abdomen was mildly distended with tenderness over the left paraumbilical region. The liver was enlarged to 6 cms. and the spleen to 1½ cms. below the right and left costal margins.

There was no significant lymphadenopathy. The other systems were normal. His haemoglobin was 14.4 gm/100 ml., white blood count 6,600/ml., with a differential count of neutrophils 35%, lymphocytes 59%, monocytes 4%, atypical lymphocytes 2%. His serum bilirubin was 2.5 mg/100 ml. with 0.6 mg. conjugated and 1.9 mg. unconjugated. The urine did not contain any bile or urobilinogen.

In view of the above findings, a provisional

diagnosis of typhoid fever was made, and the patient started on chloramphenicol therapy.

Methods of Isolation

The patient's faeces were cultured bacteriologically 7 times during his stay in hospital. The faeces were plated on MacConkey agar and deoxycholate-citrate agar and incubated at 37°C for 18 hours. Part of the specimen of faeces was also cultured in selenite F medium for 18 hours and then was plated onto MacConkey and deoxycholate-citrate agar.

All cultures were examined for non-lactose fermenting colonies and these were subcultured onto Kligler iron agar slopes which were incubated at 37°C for 18 hours. On inspection after incubation, the results were an acid butt and alkaline slope with no gas or hydrogen sulphide production – a reaction similar to that given by the shigella group of organisms. Accordingly, a slide agglutination with **Shigella sonnei** antiserum gave a very strongly positive reaction. But subsequent biochemical reactions did not confirm the organism as a **Shigella sonnei.** These suggested the organism to be **Plesiomonas shigelloides,** and this was confirmed by Dr. Cooper in Adelaide. The organism was isolated only from the first two of the 7 specimens of faeces examined. No organism of the dysentery or enteric group was isolated.

Bacteriological Features

Plesiomonas shigelloides was a motile gram negative rod which formed smooth, colourless and convex colonies, 1–1.5 mm. in diameter with entire edge on deoxycholate-citrate medium after incubation for 24 hours. It was a facultative anaerobe growing well both aerobically and anaerobically on blood agar as well as on nutrient agar. In Kligler iron agar, the reaction given was similar to that of shigella organisms – acid butt, alkaline slope with no gas or hydrogen sulphide being produced. The following table gives the bacteriological features of the organism.

Serology

The organism gave a strong positive slide agglutination reaction and agglutinated in dilutions of up to 320 in a tube test with **Shigella sonnei** phase I & II antiserum (Burroughs Wellcome).

Bacteriological features of Plesiomonas shigelloides

TEST	RESULTS
Motility	+
Catalase	+
Cytochrome oxidase	+
Gas in glucose	–
Acid in glucose (1 day)	+
Acid in lactose (1 day)	–
Acid in lactose (3 days)	+
Acid in sucrose (1 day)	–
Acid in maltose (1 day)	+
Acid in mannitol (1 day)	–
Acid in dulcitol (1 day)	–
Acid in inositol (1 day)	+
Acid in salicin (1 day)	–
Acid in xylose (1 day)	–
H2S (Kligler)	–
Indole	+
Methyl Red	+
Voges Proskauer	–
Citrate utilisation	–
Urease	–
Malonate utilisation	–
Phenylalamine deaminase	–
Lysine decarboxylase	+
Arginine decarboxylase	+
ONPG reaction	+

Antibiotic Sensitivity

Sensitivity to antibiotics was tested on plates of Oxoid sensitivity agar using MAST antibiotic discs. The organism was found to be sensitive to ampicillin, cephaloridin, streptomycin, tetracycline, chloramphenicol, neomycin, kanamycin and trimethoprim.

Discussion

Cooper and Brown, in their paper, discussed the similarities as well as the difference between **Plesiomonas shigelloides** and **Aeromonas.** A characteristic feature of this organism is that it sometimes has **Shigella sonnei** antigens. Not all the isolates possess this feature, but some of them may have other shigella antigens.

In Japan, Honi, Hayashi, Maeshima, Kigawa, Miyasato, Yoneda and Hagishara in 1966 (cited by Cooper and Brown 1968) described 10 strains which had

Shigella dysenteriae type 7 antigen. Cooper and Brown in Australia found that only 4 of their 38 strains had **Shigella sonnei** antigens and one had **Shigella felexneri** type 6 antigens. Eddy and Carpenter (1964) found that out of their 21 strains, only 12 possessed the **Shigella sonnei** phase I antigen.

Thus the majority of the strains do not have any shigella antigens. Therefore an organism giving shigella-like reaction in Kligler iron agar and not agglutinated by shigella antisera must be tested for **Plesiomonas shigelloides**. A presumptive identification can be made if it is also motile, oxidase positive and mannitol negative.

The question that cannot be answered categorically is whether this organism gives rise to a dysentery-like illness.

Our patient was provisionally diagnosed as a case of enteric fever but was never confirmed bacteriologically or serologically. However, he made an uneventful recovery after chloromycetin therapy, and no **Plesiomonas shigelloides** or dysentery or enteric type of organisms were isolated subsequently.

No other cause of his illness was elucidated. Blood cultures, 4 specimens, were negative; Widal and Weil-Felix titres were not significant.

Are we entitled to say that the cause of his illness was **Plesiomonas shigelloides**? Cooper and Brown isolated this organism from infants suffering from mild as well as severe diarrhoea. They believe that this organism can be the cause of enteritis especially in infants, although the organism may be found in the absence of enteritis. Some of the C27 strains of Eddy and Carpenter (1964) also came from human patients, mainly with diarrhoea.

Other workers in Ceylon and in Japan have isolated the organism from domestic animals, healthy adults as well as from children and adults suffering from diarrhoea. This is reminiscent of the food poisoning type of Salmonella which can be isolated from a variety of domestic animals, reptiles, healthy human beings as well as from people suffering from gastro-enteritis.

It may well be, as Cooper and Brown pointed out, that possession of shigella antigens may confer on them the ability to produce illness. It is hoped that as more medical microbiologists become aware of this organism, more evidence will be brought forward for the pathogenicity or otherwise of **Plesiomonas shigelloides.**

It is believed that this is the first reported isolate in Malaysia and further work on it is in progress.

Summary

Plesiomonas shigelloides was isolated from the bowel of a patient with pyrexia and diarrhoea. The morphology and biochemical reactions of the organism are described and its significance discussed.

Addendum

After preparing this manuscript, a further 8 strains of **Plesiomonas shigelloides** were isolated from patients with diarrhoea.

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Necrotizing Enteritis

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BECKERMANN (1946), from Germany, first described a number of cases of severe illness of acute onset with abdominal pain and slight rigidity of abdomen associated with profuse diarrhoea, vomiting and mild fever. These cases were often fatal and were due to necrotic inflammation of several areas of the intestine, especially in the jejunum.

Epidemic outbreak of necrotizing enteritis had been described from New Guinea, Murrell (1967). Sporadic cases had been described from the United Kingdom, Greville Young, (1949); the United States of America, Patterson and Rosenbaum (1952); Australia, Goulston et al (1965); Indonesia, Gan et al (1962); Thailand, Headington et al (1967) and Uganda, Wright (1967). The association of the disease with pork eating has been described by Murrell (1967). A case is described here which had the features of necrotizing enteritis. Causative factors of the disease are discussed.

Case Report

A 12-year-old-female Chinese was admitted with a history of colicky abdominal pain associated with passage of mucus and loose watery stool, "red" in colour for 3 days, and vomiting of 2 days' duration. She had been perfectly healthy prior to her present illness and no one else in the family had a similar illness. On examination, she was well built but pale, dehydrated, conscious and co-operative. Pulse was 148 per minute regular, BP — 95/65 mm Hg. She was febrile with a temperature of 99°F. There was no neck stiffness. Respiratory, cardiovascular system and central nervous system were normal. Relevant physical findings were confined to the abdomen which was grossly distended and diffusely tender. Bowel sounds were infrequent. Liver dullness was obliterated. Rectal examination was normal. A diagnosis of acute gastroenteritis with paralytic ileus was made.

Investigation

Haemoglobin was 13.1 G/100 ml, WBC 3400 per μl, with neutrophils of 53 per cent, lymphocytes 44 per cent and monocytes 3 per cent, erythrocyte sedimentation rate was 6 mm/hr. (Westergren). Blood urea was 48 mg/100 ml, sodium 128 mEq per litre, potassium 3.1 mEq per litre. A random blood sugar was 108 mgm/100 ml; urine analysis was within normal limits. An electrocardiogram revealed atrial tachy-cardia with a heart rate of 180 per minute. A straight X-ray of the abdomen revealed dilated small bowel, with multiple fluid levels; the appearance was suggestive of paralytic ileus.

Blood and stool cultures and blood for Widal-Weil-Felix were done after admission. She was treated with chloramphenicol 500 mgm 6 hourly I.M., and her dehydration was corrected by intravenous therapy. Her condition progressively deteriorated after admission. She developed high fever, bleeding per rectum and severe abdominal distension. Because of the massive bleeding per rectum, she was then referred to the surgeon.

Laparotomy was carried out on the 4th June 1970. Abdomen was markedly distended and on opening the peritoneum greenish fluid was seen.

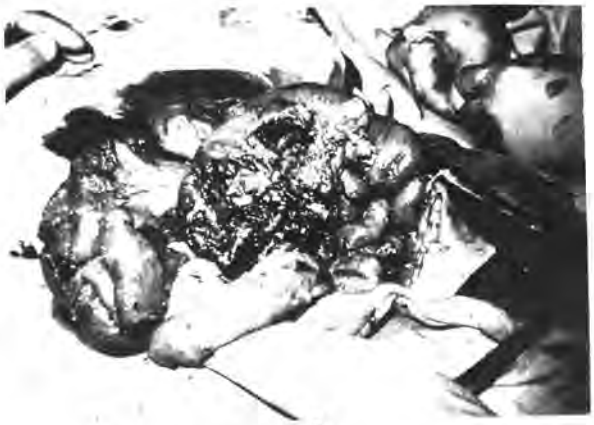

Fig 1
Portion of the small bowel with gangrenous necrosis and perforation on the second laparotomy.

Isolated segments of small bowel extending from the jejunum to the ileum was haemorrhagic in appearance. About 1½ feet of small bowel, just 2 feet proximal to the ileo-caecal junction, was markedly haemorrhagic in appearance, with some spotty dark coloured areas. No perforation of any part of the bowel was detected. The whole of the small intestine and, to a smaller extent the large bowel, was distended. The arterial arcade of the small bowels was patent. As it was impossible to define the area of impending gangrenous bowel and in view of the widespread involvement of the whole of the small gut, no resection of the small intestine was done but decompression of the bowel was carried out to relieve the abdominal distension.

Post-operatively, she developed a high fever, partly controlled by penbritin. Soon after, she developed intermittent colicky abdominal pain, requiring frequent doses of pethidien for relief. Brown loose stools persisted. Because of the persistence of colicky abdominal pain, and the provisional diagnosis of mechancial obstruction, laparotomy was again carried out on 17th June 1970. Almost the whole segment of ileum, about 2 feet from the ileo-caecal junction, was necrotic and perforated. Multiple perforations of the jejunum were also found **(Figs. 1 and 2)**. The necrotic and non-viable small intestine was resected and anastomosed.

On the 20th June, 1970, because of the discharge of faecal material from the drainage sites, laparotomy was again carried out. Further perforations proximal to the previous anastomosis and at other sites were detected. Again non-viable gut was resected and perforation closed. On 22nd June 1970, because of

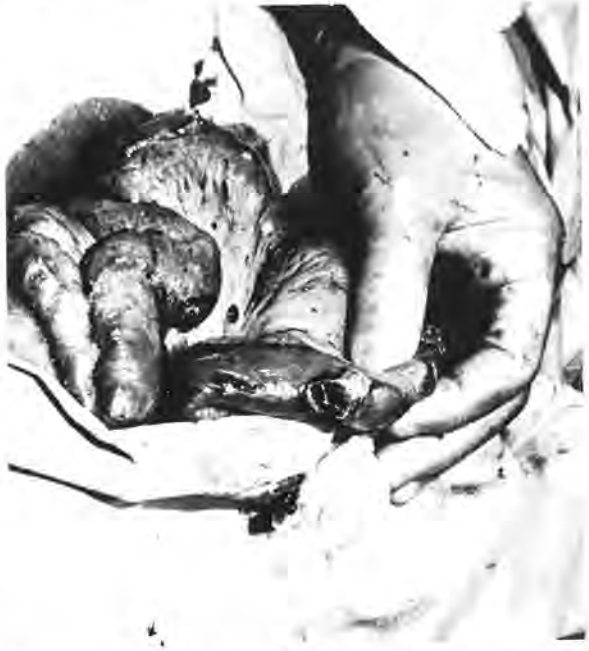

Fig 2
Appearance of the small bowel on the third laparotomy, showing the punch-out appearance of the perforation.

further discharge of faecal material through the drainage sites, laparotomy again was carried out and further perforations were detected. Resection of non-viable gut was carried out. However, the patient's condition deteriorated soon after the fourth laparotomy and died. Widal and Weil-Felix tests were available on the 2nd week and both were normal. No pathogen was isolated from stool culture.

Histological examinations of the resected gut revealed evidence of an early organising fibrinopurulent peritonitis involving the entire serosal surface. The luminal surface of the small intestine contained only a few focal patches of mucosa. Some of these consisted of only a few intestinal glands with the surface epithelium sloughed off. The submucosa was moderately thickened, the lymphatic vessels were dilated and there were a moderate to marked increase in histiocytes, plasma cells and lymphocytes.

In areas of necrosis, there were also a large number of polymorphonuclear leucocytes. The necrosis of the mucosa and submucosa in some areas extended into the inner layer of smooth muscular wall, showing degeneration of the smooth layer without accompanying inflammation. The micro-organisms seen on special stain consisted of a mixture of gram-positive rods; no gram-negative rods were seen. The histologic picture in all three specimens was that of a non-

specific necrotizing enteritis with marked submucosal oedema with early fibro-proliferative activity.

Post-mortem examination findings were mainly confined to the abdomen which revealed evidence of diffuse fibrino-purulent peritonitis. Numerous foci of necrosis were found throughout the small intestine and proximal colon. Numerous fibrous and fibrinous adhesions were found between all peritoneal surfaces. The peritoneal cavity contained thick yellow white purulent exudate. The mesentery was thickened, haemorrhagic and contained numerous large firm lymph nodes. The lungs were heavy and congested with focal areas of atelectasis. The heart was dilated and numerous subendocardio-petechial haemorrhages were found, particularly in the left ventricle.

Conclusion The findings were that of necrotizing enteritis of obscure aetiology.

Discussion

Since the first description of the disease from Germany by Beckermann (1946), similar cases have been reported from other parts of the world. Different authors have used different names to describe what appears to be the same disease. Greville-Young (1949) used the term acute jejunitis, whereas Patterson and Rosenbaum described it as enteritis necroticans. Goulston et al preferred to use the term ulcerative jejunitis.

Although there still exists a greal deal of controversy about the terminology of this disease, there is also no uniformity about the aetiology of the disease. Oakley thought it to be due to Clostridium Welchii type F., whereas Schutz believed it to be due to Cl. Welchii type A., Murrell et al postulated Cl. Welchii type C. to be the cause of this disease. Although most of the authors tend to link this disease to the infection by some strains of Cl. Welchii, Greville-Young was of the opinion that this is a variant of Crohn's disease. Kravetz and Brazenas thought it to be due to a virus and related this disease to enteritis gravis which is sometimes associated with infectious hepatitis. In the case reported here, there was no evidence of Clostridium Welchii infection. The culture of the stool and intestinal contents of post-operative material did not grow Cl. Welchii. However, antibody titre was not estimated against Cl. Welchii.

Although the terminology of the disease and the causative mechanism of necrotizing enteritis have not been firmly settled, it is important to recognise the condition, as early diagnosis and correct management may reduce the mortality and morbidity. In the tropics, it is important to differentiate this disease from the Salmonella enteritis and in children, it can simulate intussusception.

The surgical treatment of this pathological state is the correction of dehydration, electrolytes imbalance, hypovalaemic shock and resection of necrotic gut. However, in the above reported case, the disease process at the time of laparotomy was widespread involving multiple segments of the small gut, without well demarcated or obvious gangrene of the small bowel. Post-mortem findings revealed more extensive involvement than at first realised, in that the disease process extended throughout the whole gastrointestinal tract, involving patches of the oesophagus, stomach and colon.

Whether radical resection of the diseased small bowel, or any portion of the small bowel that had necrosed mucosa, with or without normal seromuscular layered coat during the first operation, would have helped remained uncertain. However, from the study of this case, it is felt that earlier resection is strongly recommended, especially when the area of involvement is not extensive and this might prevent the occurrence of perforation.

Summary

A case of necrotizing enteritis in a young Chinese girl is described here. The aetiology and the management of the disease is discussed.

Acknowledgement

We wish to thank Dr. M. Somasundaram, Senior Lecturer in Medicine, for allowing us to investigate the patient under his care and to Che Rohani for secretarial assistance.

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Angiographic Features of Sturge-Weber Disease:
Report of a Case

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ENCEPHALOTRIGEMINAL ANGIOMATOSIS or Sturge-Weber Disease, in its complete form, consists of a facial naevus in the distribution of the trigeminal nerve and at least one of two other major features of the syndrome, namely, an intracranial angioma or an angioma of the choroid of the eye (Normal 1963).

Other associated clinical manifestations include convulsions, hemiparesis, hemiatrophy, mental retardation, bulpthalmos and congenital glaucoma. The pathologic-anatomical change intracranially is in essence a capillary venous angiomatosis of the leptomeninges. In all cases, the affected area becomes atrophic, with deposition of calcium and/or ferruginous material in the cortex and sub-cortical tissues (Lichtenstein 1954). The role of radiology lies in the demonstration of the abnormalities using plain skull X-rays, pneumoencephalography and cerebral angiography.

Allan Sturge (1879) was credited with being the first to describe the condition clinically, but in 1922, Weber published a radiograph of the characteristic double curvi-linear shadows outlining the cerebral sulci which have since become the hallmark in radiological diagnosis. Pneumoencephalography is used primarily to show or confirm cerebral atrophy. The changes include ventricular enlargement and dilated cerebral sulci on the affected side. In advanced cerebral atrophy, the basal cisterns are also widened.

Although Moniz and Lima had used cerebral angiography to investigate this condition in 1935, relatively few cases have since been studied by this technique, primarily because of the reluctance on the part of investigators to subject children to angiography.

However, Poser and Taveras (1955) stressed that this procedure was equally safe in children and adults. The same authors later (1957) collected 35 cases studied by cerebral angiography from the literature and added 15 cases of their own. From that combined series they concluded that angiographic findings in encephalotrigeminal angiomatosis were not exclusively confined to the presence of venous angiomata although such vascular anomalies were most frequently seen, accounting for 46% of positive findings of the 50 cases reviewed.

Other angiographic features, which may be demonstrable in this syndrome, include arteriovenous malformations, cerebral arterial thromboses, cerebral atrophy and anomalies of the cerebral veins and dural sinuses. It is primarily in connection with the latter appearances that the author wishes to present the following case.

Case Report

C.Y.C., a 14-year-old male Chinese patient, was admitted to the medical ward of the University Hos-

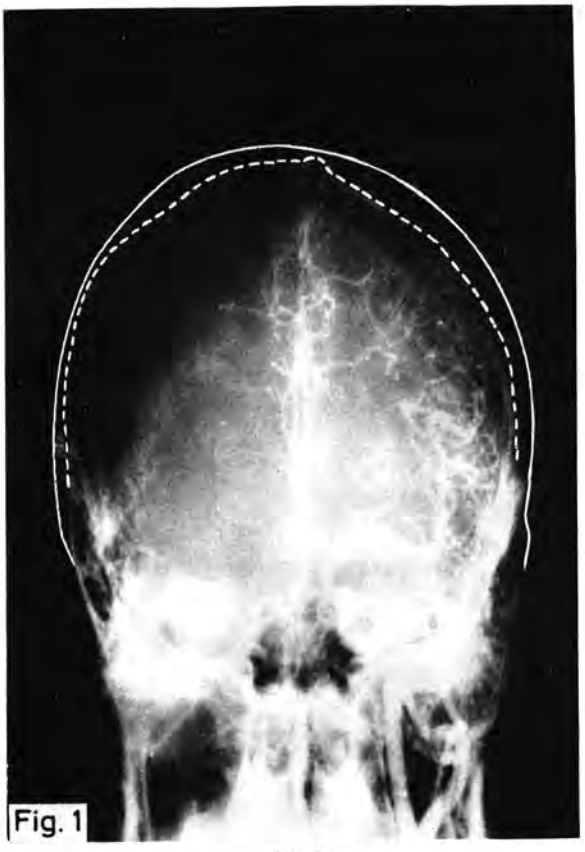

Fig 1
Minor shift of anterior cerebral artery to the left. Compensatory vault thickening indicated by continuous and dotted lines.

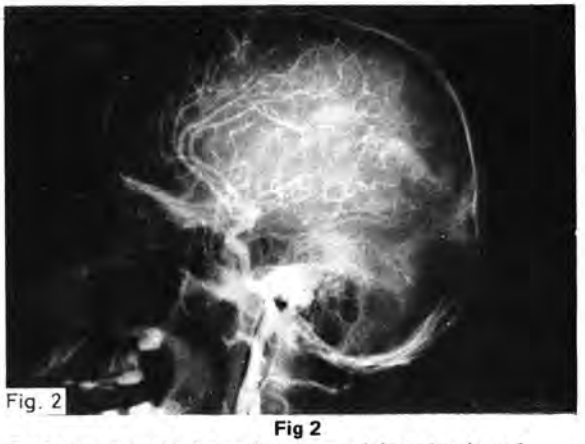

Fig 2
Evidence of cerebral atrophy: large sylvian triangle and rather low position of sylvian point. Slight increase in curvature of anterior cerebral artery.

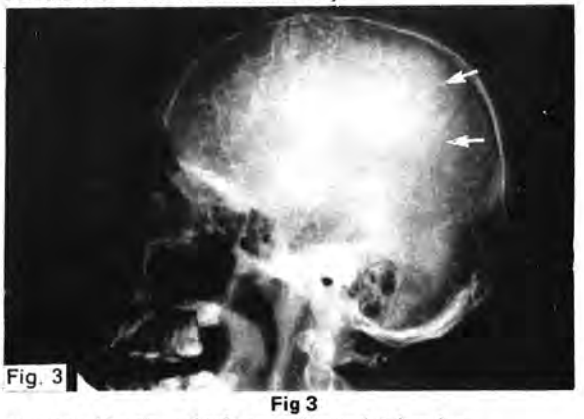

Fig 3
Non-opacification of left posterior parietal region, indicating cerebral atrophy (arrows).

pital with a history of generalised fits since the age of 1 year. The onset of the seizures was always preceded by an aura, described as a feeling of anxiety. Each fit would last about 2-5 minutes, occurring approximately once every 3-4 weeks. He had a port-wine mark on the left side of his face which had been present since birth. According to the parents, his milestones of development had been delayed. In the four months prior to admission, he was noticed to be aggressive and "destructive".

Examination showed him to be a well-built but mentally retarded patient. He had a port-wine naevus on the left temple, cheek, both lips and both eyelids.

Central Nervous System

The fundi appeared normal. The cranial nerves were intact. Motor power in the limbs was grade IV on the right side and grade V on the left. Electroencephalography suggested atrophy in the left posterior parietal area. The provisional clinical diagnosis was Sturge-Weber Disease.

Radiological Examination

Plain skull X-rays showed no sign of intracranial calcification. There was however a reduction in size of the left half of the cranial cavity with compensatory thickening of the skull vault.

A left carotid angiogram done under general anaesthesia showed the following abnormal features:-

(i) The arterial phase showed a 5 mm. shift of the anterior cerebral artery to the left side **(Fig. 1)**. Evidence of cerebral atrophy was further strengthened by the presence of a large sylvian triangle and downward displacement of the sylvian point in

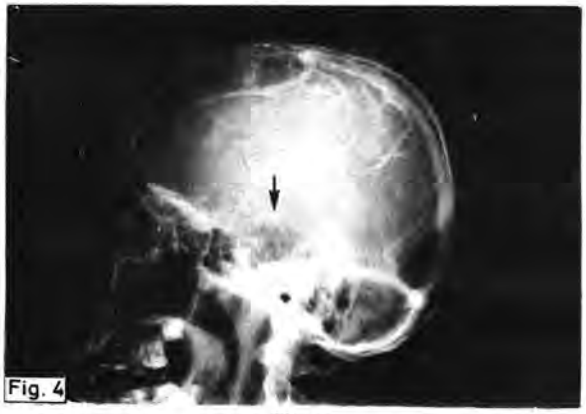

Fig 4
Small venous anomaly (arrow) and large left basilar vein. Partial agenesis of superior sagittal sinus with prominent draining veins. No evidence of filling of internal cerebral vein.

the lateral view **(Fig. 2)**. In the late arterial and capillary phase, the posterior parietal region was not opacified, indicating focal atrophy in that region. **(Fig. 3)**

(ii) The venous phase revealed a small venous angioma in the left temporal region **(Fig. 4)**. In the late venous phase, an enlarged tortuous basilar vein was seen coursing upwards and backwards to join the posterior portion of the vein of Galen. There was no evidence of filling of the internal cerebral vein or its tributaries. A partial agenesis or thrombosis of the superior sagittal sinus was noted. The superficial cortical veins draining into the superior sagittal sinus appeared numerous and prominent.

In view of the above findings, cross compression was performed to fill the venous system on the right side. The venous phase in this series showed a rather small thalamostriate vein emptying into the right internal cerebral vein **(Fig. 5)**. The left internal cerebral vein was again not visualised.

Discussion

Despite the intensive study by Poser & Taveras (1957), few recent workers have used cerebral angiography as a routine in investigating cases of Sturge-Weber Disease in its complete or incomplete forms. A reveiw of the available literature in English since 1957 shows rather disappointingly the paucity of positive findings in cases subjected to cerebral angiography

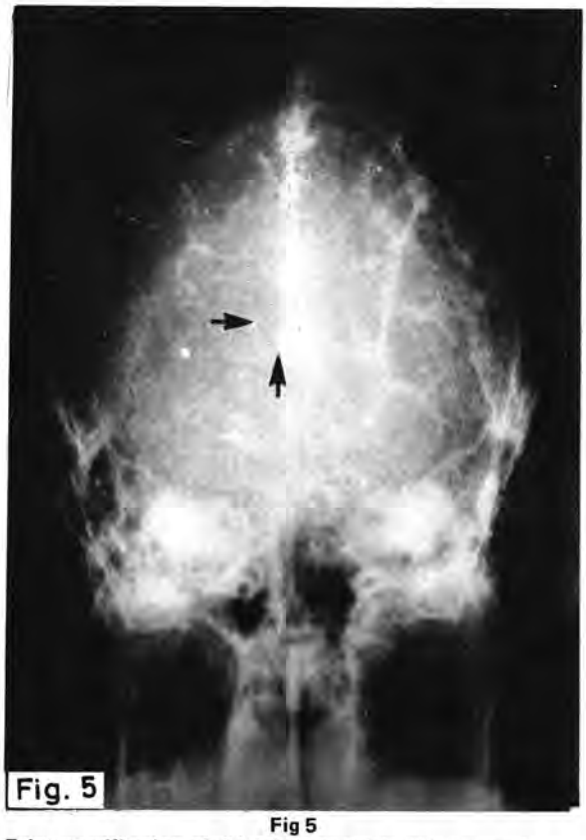

Fig 5
Faint opacification of right thalamostriate vein (arrows) and right internal cerebral vein on cross compression. Left internal cerebral vein not visualised.

(Table 1). A classic form of this condition was reported in detail by Nellhaus, Haberland and Hill (1967) who considered the angiogram to be negative although there was obvious evidence of hydrocephalus and cerebral atrophy.

In their reports, Peterman, Hayles, Dockerty and Love (1958), Falconer and Rushworth (1960) and Rimon and Katila (1966) did not state the criteria they used in classifying the angiograms as abnormal or otherwise. The technique employed was also not described. It is possible that these authors might have used stricter critera in their radiological interpretation of abnormality than those advocated by Poser and Taveras, who also emphasised the use of rapid serial films to achieve more complete visualisation of the capillary and venous circulation. Poser & Taveras also remarked on the relatively low percentage of positive angiographic findings (37%) in patients with characteristic intracranial calcification in comparison with the 65% positive findings in the group with no visible gyriform calcification.

TABLE 1

An analysis of the results obtained from radiological procedure performed on 3 different series of patients since 1957

	Number of patients in each series	Radiological Examinations					
		Typical "Weber type" calcification on plain skull X-rays		Pneumoencephalogram		Cerebral angiogram	
		No. of patients examined	No. of patients with positive findings	No. of patients examined	No. of patients with positive findings	No. of patients examined	No. of patients with positive findings
Poser and Taveras (1957)	15	15	3	5	5	15	12
Peterman et al (1958)	35	35	22	5	5	5	0
Falconer & Rushworth (1960)	5	5	4	5	5	2	0
Rimon & Katila (1966)	2	2	2	Not done	–	2	0

In both the series presented by Falconer & Rushworth (1960) and by Rimon & Katila (1966), the patients presented with extensive gyriform calcification at the time of cerebral angiography, whereas calcification was noted in only 2 of the 12 "positive" cases of Poser & Taveras (1957). The lack of positive angiographic findings could be attributed to the extensive intracranial calcification that these patients had at the time of examination. In the absence of convincing clinical evidence and characteristic intracranial calcification, however, cerebral angiography may be the procedure of choice to show intracranial anomalies.

This case is presented to illustrate some common radiological manifestations of encephalotrigeminal angiomatosis. The under-developed left hemicranium with compensatory vault thickening, combined with angiographic evidence of mid-line shift to the ipsilateral side and paucity of capillary filling in the left posterior parietal region, were indicative of underlying cerebral atrophy.

A few interesting features in the venous system were shown, amongst which was non-filling of the left internal cerebral vein. Two attempts were made to fill this vessel without success although in the series done with cross compression the contralateral internal cerebral vein was visualised in the Towne's projection. One may assume therefore that the left internal cere-

bral vein was absent in this patient. This could be part of the venous anomalies associated with this condition although so far no such reports have been recorded. The venous angioma was small and might have been missed if rapid serial films were not done. The small angioma appeared to drain into a large left basilar vein which in turn emptied directly into the vein of Galen. The latter appearance was noted by Taveras & Wood (1964) and again by Banna and Young (1970) who considered it an anatomical variant. Partial agenesis or thrombosis of the superior sagittal sinus with prominent draining veins is a well recognised appearance and was described in 3 of Poser & Taveras' 15 cases. Two of these 3 cases were associated with the complete form of encephalotrigeminal angiomatosis showing no visible intracranial calcification.

The present case can be regarded as a complete form of the disease with angiographic evidence of cerebral atrophy and venous anomaly. It illustrates the value of cerebral angiography when negative plain films of the skull cast doubt as to the exact form of the disease. If the use of this procedure can be limited to clinically complete, or incomplete forms without characteristic gyriform calcification, results could be more rewarding. It can then contribute more significantly in establishing diagnosis and in assessing those cases in whom hemispherectomy or surgical excision of the venous anomaly is being considered.

Acknowledgements

I am indebted to Dr. M. Somasundram and Dr. A.H. Ang for their advice and criticism. Grateful thanks are due to Dr. H.O. Wong for allowing me to publish this case and the Medical Illustrations Department for producing the photographs. I am especially thankful to Miss Janet Low for her help in typing the manuscript.

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Rupture of the Uterus:

Treatment by suturing the tear

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IN RECENT YEARS, interest has been focused on the conservative treatment of uterine rupture (Menon 1954; Swami and Patel 1960). Seth (1968) described his experiences of repair of the uterine rupture without carrying out sterilisation.

Materials

The study covers all cases admitted to the Department of Obstetrics and Gynaecology, Rumah Sakit Umum Kuantan for the period January 1968 to December 1969. All the cases of ruptured uterus were referrals from the outlying rural areas to the nearest District Hospital and from there to Rumah Sakit Umum Kuantan.

TABLE I
Methods of Treatment of cases of ruptured uterus, G.H. Kuantan

	Year 1968	Year 1969
Treatment	No. of cases	No. of cases
Hysterectomy	3	1
Suturing and sterilisation	1	3

Case Report

The first case in this report is described below so as to illustrate the problem and the method of treatment.

A 30-year-old Malay woman Gravida 7, Para 3, stillbirth 3, was referred to a district hospital from a remote village. She had been looked after by an untrained kampong bidan and since there was no progress in labour, the kampong bidan carried out 'urut' (Malay term for massage). Subsequent to this, the patient became restless and developed severe abdominal pain. The relatives then took her to the nearest district hospital, where she was diagnosed as a case of obstructed labour and immediately referred to Rumah Sakit Umum Kuantan on 22nd January 1968.

On admission to hospital, her condition was poor and clinical examination showed a shocked patient with a tense abdomen. Foetal parts were easily felt inspite of the tense abdomen. A diagnosis of rupture of the uterus was made, the patient was resuscitated, and an emergency laparatomy was carried out under general anaesthesia. The findings at laparatomy were, a dead foetus weighing 7lbs. 2 ozs. lying free in the peritoneal cavity. The uterus showed a large circular irregular tear in the lower segment extending laterally to the posterior surface. The uterus above the tear was only held on to the lower segment by a 2" broad posterior band. A decision was made to repair the tear. The ragged, traumatised edges of the tear were excised and the repair carried out in two layers.

Bilateral tubal ligation was carried out. Her post-operative period was uneventful apart from pyrexia which responded to antibiotics. She was discharged and sent home on the 13th post-operative day. She was followed up in the gynaecology clinic for over a

period of one year. She had no complaints. Her menstrual cycle was normal.

Discussion:

Rupture of the uterus is a rare condition but doctors in the rural areas would occasionally meet such cases. The patient is always an unbooked case with no previous obstetric care and the delivery managed by an untrained midwife or bomoh. A common aetiological factor in the causation of the rupture has been the practice of 'urut' or massaging the uterus in labour.

'Urut'

This is a common procedure carried out by the untrained midwife for a number of obstetrical and gynaecological problems. Sambhi (1968) described his experiences about the bomoh's abdomen. Urut or massaging the abdomen has been practiced to induce bleeding in cases of delayed period; to induce abortion; to induce labour and to hasten labour. It is a safe procedure in the gynaecological or postnatal patients where urut, in combination with the application of special oil, followed by abdominal binders with cloth, helps the patients to get their muscle tone back and reduce the incidence of genital prolapse. But urut carried out in late pregnancy or in labour in an attempt to deliver a patient is a dangerous procedure. The prolonged labour may be due to an obstructed labour and pressing the fundus with extreme violence would rupture the uterus (Ferguson & Peid 1958, Trivedi, Patel and Swami 1968).

In view of this widespread practice of urut in the rural areas, rupture of the uterus could occur in a young patient. Traditional obstetric practice has always stressed that the treatment of rupture of the uterus is hysterectomy. I feel that because of the special socio-economic-cultural factors, conservative surgery (i.e. resuturing the tear with or without sterilisation) has a special place in the management of these cases. The special factors which would influence the decision is as follows.

1. **Age** – The patients are usually young and reproductive and child-bearing is very important to these young rural mothers.
2. **Menstruation** – To the rural Malay women, menstruation is an important part of her life. With hysterectomy, this function is lost and the patient feels that she is not a complete woman. Post-hysterectomy patients always complain that they are not well and feel unhappy that the 'dirty' blood is not flowing out monthly. In spite of detailed explanation regarding the physiology of menstruation, these patients feel that to be healthy, menstruation must occur at monthly intervals.
3. **Poor general condition** – By the time the cases of ruptured uterus are referred to hospital, the condition of the patient is very poor. Inspite of active resuscitation, these patients present a grave risk for any major operative procedure like hysterectomy. In such cases, simple resuturing, followed by sterilisation, is the safest procedure.

Conclusion

In young rural women presenting with a ruptured uterus, simple resuturing of the rupture has a place in the treatment.

Acknowledgement

I am grateful to the staff of the Department of Obstetrics and Gynaecology, Rumah Sakit Umum Kuantan, for clinical and nursing care of the patients.

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Reduction of the Aspiration Hazard in Anaesthesia:
Use of Methohexitone-Suxamethonium Mixtures

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ONE OF THE CONSTANTLY recurring problems facing anaesthetists is the patient with a full stomach. The risk of anaesthesia in such a patient is considerably enhanced by the possibility of the patient vomiting or regurgitating the stomach contents and aspirating these into the lungs.

Aspiration continues to be a major cause of morbidity and mortality due to anaesthesia (Edwards et.al., 1956; Kinch, 1959; Clifton and Hotten, 1963; Gray, 1968), although it is a problem which is to a large extent preventable (Vandam, 1965). Aspiration is most common during the induction of anaesthesia (Lock and Griess, 1955) and is likely to produce its most deleterious effects during this period (Parker 1954). Consideration should, therefore, be given to the possible ways of preventing such aspiration during the period of anaesthesis induction prior to the introduction of a cuffed endotracheal tube (Mucklow and Larard, 1963).

Khawaja (1971) showed that a possible way of shortening this high-risk period and thus reducing the aspiration risk is by use of thiopentone-suxamethonium mixtures to induce anaesthesia. When a mixture containing thiopentone and suxamethonium in a dosage of 4 mg/kg body weight and 1 mg/kg body weight respectively was injected, the patient was unlikely to be aware of the suxamethonium fasciculations and the interval between the onset of unconsciousness and completion of endotracheal intubation was materially shortened.

Methodhexitone affects the cardiovascular system much less than does thiopentone (Weyl, Unal and Alper, 1958; Coleman and Green, 1960; Dundee and Moore, 1961) and provides more rapid recovery (Taylor and Stoelting, 1960; Elliott et.al., 1962; Swerdlow, 1964) and induction with the former drug may be preferable, at least in some cases. A study was, therefore, undertaken to assess the suitability of methohexitone-suxamethonium mixtures in the induction of anaesthesia. The present paper is a preliminary report of its use in 70 patients.

Material

Seventy adult patients, including 18 emergency cases, suitable for induction with methohexitone and suxamethonium as described below, were studied. The average age of the patients was 32.7 years (range 18 years to 61 years) and the average body weight was 50.7 kg (range 35.5 kg to 83.0 kg). The patients belonged in the first three grades, or the emergency counterparts, of the American Society of Anaesthesiologists classification of physical status.

Patients for elective surgery were premedicated with morphine sulphate 1 mg/stone (6.36 kg) body weight and atropine sulphate 0.6 mg injected intramuscularly about one hour before induction. In

patients for emergency surgery, where there was a risk of regurgitation and aspiration, atropine alone was given (Clark and Riddoch, 1962; Inkster, 1963; Wylie, 1963). Where necessary, a Levine tube or oesophageal tube was also used to empty the stomach as far as possible and to reduce the intragastric pressure. Narcotic premedication was also omitted in patients, whether elective or emergency, presenting for Caesarean section.

Mendelson (1946) and Dinnick (1957) suggested that obstetrical patients requiring a general anaesthetic be given oral antacids to reduce the acidity of the gastric contents. Most of such patients in this series received a 15 ml dose of magnesium trisillicate mixture (B.P.C.), which Taylor and Pryse-Davies (1963) showed was more effective than the aluminium hydroxide used by Crawford (1962).

Method

Anaesthesia was induced with methohexitone and suxamethonium. The dose of methohexitone, for the first 40 patients, was from 1.0 mg to 1.3 mg/kg body weight (average 1.21 mg/kg body weight) depending on the fitness of the patient. For the rest of the series a somewhat larger dose of methohexitone was used, the maximum being 1.5 mg/kg body weight and the average 1.42 mg/kg body weight. The reason for the increase is discussed below. The dose of suxamethonium was 1 mg/kg body weight to the nearest 5 mg and limited to a maximum of 50 mg. Just before injection, the appropriate dose of suxamethonium, previously measured out in a separate syringe, was drawn into the syringe containing methohexitone.

Endotracheal intubation was then performed in the usual manner after oxygenating the patient and spraying the larynx with 4% lignocaine. In obstetrical patients and patients for emergency surgery, the risk of regurgitation and aspiration was further reduced by preoxygenating the patients, using cricoid pressure (Sellick, 1961), omitting topical larynageal anaesthesia and intubating the patients as soon as the jaw was sufficiently relaxed. The time interval between the beginning of the injection of the mixture and completion of endotracheal intubation was recorded, using a stop-watch.

The next day, patients were questioned to determine whether they considered the induction of anaesthesia pleasant or not. They were also questioned to determine whether they had been aware of fasciculations or any tenseness in the facial or other muscles.

Results

None of the seventy patients regarded the induction as unpleasant. Two patients had had anaesthetics previously. One of these, who had had an inhalational induction, considered the present induction to be more pleasant. In the other case, where anaesthesia had previously been induced by the intravenous route, the patient did not consider the second induction to be any different.

One patient who presented for a Caesarean section mentioned that she had felt a tightness in the jaw muscles just before going off to sleep. When asked directly whether they had felt tightness or a fluttering sensation in the facial or other muscles, three patients remembered that they had indeed felt such a tightness – in the face in two instances and in the left thigh muscles in one case. These four patients had received doses of methohexitone of about 1.2 mg/kg body weight and two had not received any sedative pre-medication. None of them, however, considered the induction in any way unplesant. These patients belonged to the first group of forty patients. When the dose of methohexitone was increased in the second group of thirty patients, there was no case of awareness of fasciculations, and all the patients regarded the induction as pleasant.

In the emergency cases, where patients were preoxygenated, laryngoscopy could be started some 15 – 20 seconds after the onset of unconsciousness and intubation completed, in most instances, in well under 60 seconds. There was no instance of regurgitation or aspiration.

Discussion

When a barbiturate-suxamethonium sequence is used in the conventional manner in a patient with a full stomach, the high-risk period between the onset of unconsciousness and completion of endotracheal intubation, during which regurgitation is most likely and most harmful, is about 50 seconds. If the syringe containing suxamethonium is accidentally dropped or the needle in the vein gets displaced, this period will be much prolonged.

Khawaja (1971) showed that by using a thiopentone-suxamethonium mixture, the danger period is reduced to about 20 seconds. This shortening of the danger period, when the patient is unconscious but not relaxed, is likely to make induction safer, particularly in patients who are especially liable to regurgitate, such as obstetrical cases, patients with intestinal obstruction, etc. The use of methohexitone-suxamethonium mixtures shortens the danger period

to a similar extent and, hence, makes induction of anaesthesia safer. The risks inherent in the giving of two injections are avoided.

It appears from the results of the present study, the patient acceptability of the methohexitone-suxamethonium mixture is likely to be as high as that of the conventional method where the drugs are injected separately or of the thiopentone-suxamethonium mixture. When used in the lower dosage, four patients out of 40 (10%) were aware of suxamethonium fasciculations, although none of them considered the experience unpleasant. When a higher dose of methohexitone, about 1.5 mg/kg body weight, was used, none of 30 patients was aware of any fasciculations. It is, therefore, concluded that this higher dosage is preferable, except where the physical status of the patient makes a smaller dose desirable. Further evaluation of the technique is in progress.

The technique may be combined with other methods of reducing the chance of regurgitation, such as the use of a stomach tube, cricoid pressure and preoxygenation, and of rendering aspiration less harmful, such as the preanaesthetic use of alkalis in obstetrical patients.

Summary

Induction of anaesthesia with methohexitone-suxamethonium mixtures, as described, is a safe method for rapid intubation in patients who may have a full stomach. The period between the onset of unconsciousness and endotracheal intubation is considerably shorter than with the conventional technique. This technique may be combined with other methods of reducing the aspiration risk. When the smaller dose of methohexitone is used, there is a chance of awareness of fasciculations, but, in this series, no patient considered the induction to be unpleasant. When a dose of methohexitone of 1.5 mg/kg body weight is used awareness of fasciculations is unlikely.

Acknowledgements

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The Foix-Alajouanine Syndrome

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THE PURPOSE OF this paper is to report a patient with Foix-Alajouanine syndrome (cutaneous haemangioma with spinal malformations and subacute myelomalacia) not commonly found in this region. A brief review of the literature is also presented.

Case Report

A 13-year-old Chinese girl was admitted on March 1964 following sudden onset of headache and vomiting lasting for two days. Clinically, she had signs of subarachnoid haemorrhage and the diagnosis was confirmed by a bloody cerebrospinal fluid. No systemic lesions were observed at that time and radiological studies of the skull, chest and abdomen were reported as normal. On recovery bilateral carotid angiograms failed to demonstrate any cerebral pathology.

In March 1967, she was readmitted for the investigation of progressive paresis of the left leg without any other neurological symptoms. Examination then revealed a left spastic monoplegia without other neurological signs. Lumbar puncture showed a normal cerebrospinal fluid except for a high CSF protein of 140 mg%. No block of CSF flow was detected. A repeated carotid angiogram, as well as a lumbar air encephalogram, failed to demonstrate any pathological lesion. A month later, paralysis spread to the right leg, and she had difficulty in walking. The bladder and bowel functions remained normal. She had no headache, vomiting or visual changes.

On examination, she had spastic paraplegia, with the left leg more weak than the right. Tendon reflexes of both lower limbs were exaggerated and there were bilateral ankle clonus and Babinski signs. Muscle wasting was noted in the left leg but fasiculations were not observed in both limbs. Position sense was diminished from the level of the first lumbar dermatome (L1) downwards, but pain and temperature of the both legs were spared. Cerebellar signs were absent. Her mental functions and cranial nerves were not impaired. There was a thoracic scoliosis and a number of small cutaneous haemangiomata were observed on the scapular region. Bruits were not heard over the eyeballs, carotid vessels or the vertebral column.

Investigations showed normal blood and urine results. CSF was normal except for the elevated spinal fluid protein (100 mg%). Blood Kahn and L.E. tests were negative. X-rays of the thoracic and lumbar spines revealed thoracic scoliosis with small radiolucent areas in the bodies of the lower thoracic spines suggestive of spinal varices. A myelogram later confirmed the presence of a very extensive large haemangioma and varices of the whole spinal cord, extending from the cervical portion right down to the conus medullaris **(Figures 1 to 4)**.

In June 1969, she started to experience difficulty in controlling her bladder, but other neurological signs remained essentially the same. By September 1970, she was bedridden with paralysis of both legs and had bladder incontience; otherwise she was well, and had regular physiotherapy.

Comment

The syndrome of Foix-Alajouanine ("Subacute necrotizing myelitis") is characterised by myelomalacia due to the presence of spinal vascular malformations (angiomas) (Foix & Alajouanine, 1926). The vascular anomalies are large and abnormal arteriovenous angiomas found in the spinal meninges, intramedullary portion of the cord and sometimes, the nerve roots. The vessels, especially the veins, have peculiary thick walls ("arterialized veins"), and the histological picture shows marked hyperplasia and hylanosis without

Fig 1
Myelogram showing extensive spinal varices in the cervical region (A.P. view).

Fig 2
Same as Fig 1 (P.A. view).

any musculature although elastic tissues are found in the adventitia. (Cushing and Bailey, 1928; Laermittee et al., 1931; Greenfield and Turner, 1939).

The pathological changes in the veins indicate a pronounced, long-standing increase in the intravascular pressure found in conditions associated with arteriovenous shunts. The "myelitis" in this disease is due to the complications of these pre-existing angiomas which are sometimes found to bleed, thrombose and have other inflammatory changes. Thus, the patients may present as acute haematomyelia or sudden haemorrhage as in our case, or the onset of disease may be insidious from the gradual thrombophlebitis of the spinal vessels.

Clinically, the cases present with progressive amytrophic paraplegia, dissociated sensory loss, bladder disturbance and elevated spinal fluid proteins (e.g., our patient). Occasionally, cutaneous haemangiomas are observed and thus indicate a systemic involvement. The myelogram appearance is typical of the disease, showing massive spinal varices (Ransome and Mekie, 1942). The course is often progressive but long remissions, ranging from 9 to 22 years, have been reported. (Frey, 1928; Buckley, 1936; Meyer and Kohler, 1917). The syndrome has been the subject of many reviews (Jaffe & Freeman, 1943; Wyburn-Mason, 1943; Neubuerger et al., 1953; Antoni, 1962), but the pathogenesis remains obscure.

Although our patient had clinical and radiological evidence of this rare condition, the definite diagnosis can only be made at autopsy. Spinal vascular malformations seem to be extremely uncommon among Asians in Singapore. On the other hand, the intracranial form of arteriovenous malformations are not infrequently seen in this country (Tay et al., 1969; 1970).

The widespread vascular involvement in our patient precludes surgical intervention, but the smaller and more localised angiomas can be satisfactorily removed or ligated (Echols and Holcombe, 1941).

This case also illustrates the correct prediction of the spinal pathology purely by the close inspection of

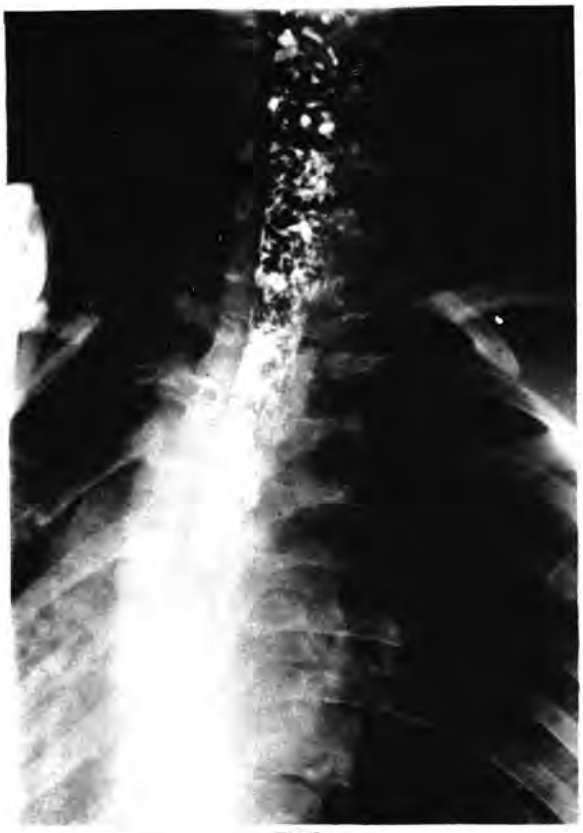

Fig 3
Varices extending into the cervo-thoracic level.

Fig 4
Varices in the thoracic and lumbar levels.

the cutaneous lesions, which in our case, was the multiple haemangiomata.

Summary

A case of Foix-Alajouanine syndrome is described and a short review of the literature presented.

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Multiple Drug Resistance and the Presence of R Factors in Enteric Pathogens

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THE TRANSFER OF drug resistance between **Shigella** and **Escherichia coli** was first demonstrated in Japan (Ochiai et al, 1959; Akiba et al, 1960). The term R factor has been used to denote the genetic determinant responsible for such transferable drug resistance.

It is now known that such factors may be present in many genera of gram negative bacilli (Mitsuhashi et al, 1967). Transfer can take place between different taxonomic groups, such as the **Enterobacteriaceae, Pseodomonas, Vibrio** and **Pasteurella** (Chabbert et al, 1969).

The use of antibiotics has resulted in the selection of bacteria carrying R factors, and drug-resistant gram-negative enteric bacilli is on the increase in many countries. This paper reports the incidence of multiple drug resistance and presence of R factors in enteric pathogens in Singapore.

Materials and Methods

The sensitivity patterns of 168 **salmonellae** and 44 **shigellae** isolated from clinical specimens have already been described (Sng and Lam, 1970; Sng and Lam, in press). Those resistant to ampicillin, streptomycin, tetracycline, chloramphenicol, kanamycin, neomycin and sulphonamide were selected for the present study. The R factor recipient strain used was **E.coli K12,** which was provided by Dr. H. Fukumi from Japan.

Media: Brain Heart Infusion (BHI, Difco) was used as propagating media for R factor transfer. For selection of resistant strains, DST (Oxoid) agar plates containing ampicillin (A; 25μg/ml), streptomycin (S; 12.5μg/ml), tetracycline (T; 25μg/ml) and sulphadiazine (Su; 100μg/ml) were prepared. To differentiate **E.coli** from non-lactose fermenters, eosin methylene blue (EMB) agar with 0.5% lactose was used.

Transfer of R factor from **salmonellae** and **shigellae:** 0.1 ml. of an overnight broth culture of each donor **(salmonellae** and **shigellae)** and recipient **(E.coli K12)** were mixed in 5 ml. of BHI broth. After incubation at 37°C overnight, serial ten-fold dilutions of the cultures were prepared in sterile normal saline. 0.1 ml. from each dilution was spread over the surface of DST agar plates containing the drugs. After overnight incubation at 37°C, resistant colonies were cultured on EMB plates for one day. Lactose fermenting colonies were then subcultured twice, after which drug resistance was determined on DST agar incorporated with the anti-microbial agents. A strain of **E.coli K12,** which had not been used as a recipient, was used as a control to check on the effectiveness of the anti-microbial drugs.

Transfer of R factor to S.typhi: The donor was **E.coli K12** which had previously been made resistant to ampicillin, tetracycline, chloramphenicol and sulphadiazine after conjugation with a strain of **Sh.flexneri** carrying the R factor. Six sensitive S.typhi strains were used as recipients. The procedure was the same as that mentioned above, except that non-lactose fermenting colonies were selected from EMB agar plates, subcultured and tested for drug resistance.

TABLE I

Distribution of Drug-Resistant Strains

Strains	Number	A	S	T	C	K	N	Su	No. resistant	% resistant
S.typhi	104	–	–	5	–	1	1	2	10	9.6
other salmonellae	64	1	9	6	–	1	1	14	23	35.7
Sh.flexneri	26	1	2	3	1	1	1	10	10	38.5
Sh.sonnei	18	–	1	1	–	–	–	6	6	33.3

TABLE II

Distribution of Multiple Resistant Strains

Species	No.	Resistance patterns	% resistant strains with multiple resistance
S.typhi	1	S K N	10
S.abony	1	K N	
S.derby	5	S Su	
S.paratyphi B	1	A S Su	34.8
S.weltevreden	1	S Su	
Sh.flexneri	1	A S T C Su	
	1	T Su	30
	1	S T K N Su	
Sh.sonnei	1	S Su	
	1	S T	33.3

Results

Among the strains studied, 9.6% of S.typhi, and about one-third of the other **salmonellae** and **shigellae** were resistant to either ampicillin, streptomycin, tetracycline, chloramphenicol, kanamycin, neomycin or sulphadiazine **(Table I).**

Multiple resistance was shown by one (10%) of the insensitive **S.typhi**, eight (34.8%) of the other resistant **salmonellae**, and three (30%) of the resistant **Sh.flexneri (Table II).** Two (33; 3%) of the resistant **Sh.sonnei** were multiple resistant. Resistance to kanamycin and neomycin always occurred together, and there was close association between resistance to streptomycin and sulphadiazine. Five **S.derby** strains had the same multiple resistance pattern.

One **S.paratyphi** B and three **Sh.flexneri** were found to carry R factors **(Table III).** All the strains were multiple resistant, but the resistance patterns which were transferred were different. All the multiple resistant **Sh.flexneri** carried R factors. In the case of **S.paratyphi B.** only resistance to ampicillin, but not streptomycin and sulphadiazine, was transferable.

Of the six **S.typhi** strains tested, one was found capable of receiving R factor.

TABLE III

Resistance Patterns of R Factors Transferred

Species	No.	Resistance patterns	% resistant strains carrying R factors
S.Paratyphi B	1	A	1.6 (non-S.typhi salmonellae)
Sh.flexneri	1	A S T C Su	30
	1	S T K N	
	1	T Su	

Discussion

R factors are extrachromosomal particles of DNA which confer on their cell hosts drug resistance, often multiple in nature, and may be transferred from cell to cell either by conjugation or transduction (Watanabe, 1969). As such transfer can occur between different genera of bacteria, it is possible for drug resistance to spread from non-pathogens to pathogens, thus giving rise to therapeutic problems (Chabbert et al, 1969). In Japan, the increased isolation frequency of multiple resistant **shigellae** strains, which reached 95% in 1965, has been ascribed to the appearance and distribution of strains carrying R factors (Mitsuhashi et al, 1967).

At the moment, drug resistance in enteric pathogens is not so common in Singapore (Sng and Lam, 1970; Sng and Lam, in press), but the percentage of multiple resistance amongst resistant strains is fairly high. Amongst resistant strains, 10% **S.typhi**, 34.8% of the other **salmonellae**, 30% **Sh.flexneri** and 33.3% **Sh.sonnei** showed multiple resistance.

From these strains, four (28.6%) were found to carry R factors. All the three multiple resistant **Sh.flexneri** carried R. factors. This constitutes 30% of resistant strains, and is less than what was found by Mitsuhashi et al (1967) in Japan, where 81% were found to carry R factors.

One **S.paratyphi B**, resistant to ampicillin, streptomycin and sulphadiazine, could only transfer resistance to ampicillin. Resistance transfer in **S.paratyphi B** is not so common, but has been reported before (Datta, 1968).

The R factor in **S.typhi** appears to be rare fortunately, Mare (1967) studied 506 strains of which 4 were found resistant. None of them transferred R factors, though they could receive them. However, Sompolinsky and Aboud (1967) have reported isolation of **S.typhi** carrying two R factors from the faeces of a patient. In our series, none of the **S.typhi** carried R factors, but one of the six strains tested received R factor from a donor. Thus the **S.typhi** are potential carriers of R factors.

R factors are present in enteric pathogens in Singapore, and could further increase through the frequent administration of antibiotics. The danger is that **S.typhi** strains might acquire the R factors. To prevent this, it is perhaps desirable that more thought should be given to the role of antibiotics in the treatment of uncomplicated enteritis of bacterial origin. Aserkoff and Bennett (1969) found that patients with **S.typhimurium** infection, when given antibiotics, took longer to clear the organism from their intestines than those not given the drugs. Furthermore, 97% of those given antibiotics excreted strains which had acquired antibiotic resistance, whereas none of those untreated excreted resistant strains.

Summary

The resistance patterns of 168 **salmonellae** and 44 **shigellae** were studied. Drug resistance was not common, but amongst the resistant strains, multiple resistance was found in 10% **S.typhi**, 34.8% of the non-**S.typhi salmonellae**, 30% **Sh.flexneri** and 33.3% **Sh.sonnei.** In four (28.6%) of these multiple resistant strains, the R factor was found. All three **Sh.flexneri** with multiple resistance carried R factors. The resistance patterns transferred were all different. One

S.paratyphi B also carried the R factor. Though **S.typhi** did not carry the R factor, one of six strains tested could receive it. In the discussion, attention is drawn to the potential danger that exists should **S.typhi** strains acquire R factors. It is suggested that there should be greater discrimination in the use of antibiotics for enteric infections.

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Congenital Duplication of the Gall Bladder:
Review of the literature and report of a case

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CONGENITAL DUPLICATION of the gall bladder is rare. The incidence of this anomaly is considerably less than that of the other segments of the extra-trepatic biliary system. Because of the rarity of gall bladder duplication, it has seemed desirable to report on this case.

Case Report

A 37-year-old Indian lady, gravida 14, para 13, gave birth to a baby. Several days later, the baby died of bronchopneumonia. At routine autopsy, the baby was noted to have complete duplication of the gall bladder, each gall bladder having a separate serosal covering. The cystic ducts joined at a Y junction and opened into the common bile duct through a single opening.

History

The diagnosis of complete duplication of the gall bladder is not new. Babylonian cuneiform characters record as an omen of victory a double gall bladder combined with gall stone in a sacrificial animal. Piling Elder mentioned the presence of a double gall bladder in an animal sacrificed at Angustus's victory at Actium in the year 31 B.C. The first case of duplication of gall bladder in man was reported by Blasivs (1674) and the first to be found at operation was reported by Sherren (1911).

Incidence

The incidence of this anomaly in animals is rather high. Because of this, Boyden attempted to determine the incidence of this abnormality in certain animals. He found that partial or complete gall bladder duplication occurred once in every eight cats examined and in the ratio of 1:28 in calves, 1:85 in sheep and 1:198 in pigs. Only 7 per cent of the duplications in cats were complete, whereas 52 per cent of those were in calves.

Boyden examined 9,221 autopsy cases and a series of 9,970 cases examined roentgeno-graphically; he found two cases of complete duplication in the former and 3 cases in the latter series. He gave the incidence in man as 1 in 4,000.

Millbouan (1941) reported the presence of 58 documented cases of complete duplication of the gall bladder.

Moore and Hurley (1954) could account for only 36 cases of total duplication of the gall bladder diagnosed at operation or autopsy. Of these, 27 were detected at operation, suggesting that the majority of double gall bladders will produce symptoms, usually in adulthood. Ryrberg (1960), in his review of the literature, noted the presence of 160 such documented cases.

Since 1960, several more have been added, mainly from non-English speaking countries and the purpose of this paper is to put on record yet another case of complete duplication of the gall bladder.

Aetiology

There are various theories as to the development

of the accessory gall bladder. The most probable explanation for the development of the accessory gall bladder in **Vesicae Fellea Dvplex** lies in the chance outgrowth from the embryonic bile passages (common bile duct, the cystic duct and hepatic ducts).

The development of a bilobed gall bladder is due to the failure of complete canalisation of the gall bladder bud.

Discussion

The incidence of complete duplication of the gall bladder is very low and the figure given by Boyden is probably correct and has been verified by others.

The number of operations performed on the biliary tract is increasing and it becomes imperative that surgeons are aware of the various developmental anomalies of the gall bladder. A thorough search for the accessory gall bladder may save a patient a repeat operation.

With improved methods of radiological examination of the gall bladder, the prospects of making a pre-operative diagnosis are high, provided the developmental anomalies are borne in mind. Further, one gall bladder may be diseased and the other normal in which case oral cholecystogram will show normal concentration of the dye in the normal gall bladder. This finding can be very misleading as in Moore and Harley's case. Sometimes, the oral cholecystogram may fail to show up the gall bladders but an intravenous cholangiogram will, as in the case of Antoine, Leclerc and Segal.

Gall bladder duplication may give rise to several problems:-

(1) If one shows pathology and the other is normal, oral cholecystogram will be misleading and give rise to diagnostic difficulties.

(2) When two gall bladders are present and both pathologically involved, and if only one is removed, the presence of symptoms after cholecystectomy may pose a diagnostic dilemma.

(3) Variation in sex, position and the number of the structures may pose technical difficulties and the chances of damage to the ducts are increased.

Summary

A case of complete duplication of the gall bladder in an infant is described and the relevant literature is reviewed and discussed. Reference is made to the problems of diagnosis and surgery in complete duplication of the gall bladder.

Acknowledgement

Thanks are due to Professor Dugdale for permission to publish this case and Mrs. Lee for typing this paper.

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A Foetus in the Abdomen of a Boy

by Mohan Lal

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I WOULD LIKE to record this rare case of "A Foetus in the Abdomen of a Boy", aged 2 months. This case is interesting because it is probably the youngest boy on record who has been successfully operated upon. Up to date, the boy is growing up normally. The last published case from this region was by Dr. Gopal Haridas in 1949.

Case Report

The patient, a Malay male infant, was first admitted to our paediatric ward on the 9th. June, 1969, when he was only 43 days old. The parents had noticed that there was a gradual distension of his abdomen since the age of 1 month. There were occasional episodes of vomiting, but his bowel habits were normal. The mother had been breast feeding him satisfactorily since birth.

Clinical examination on that day revealed an enlargement of the abdomen. On palpation, a cystic mass was felt in the right hypochondrium, about 8 cms. in diameter. A plain X-ray of the abdomen was done (**Fig. 1**) and it showed a large soft tissue mass shadow occupying the right upper part of the abdomen, displacing the gut downwards and to the left.

Before further investigations could be done, the mother took the child home at her own request and against medical advice. (This is common in this country). But the mother brought the child back for readmission on the 1st. July, 1969. The abdomen then had further increased in size. She then allowed the child to be fully investigated.

Previous History

There were no complications during the period of pregnancy. Neither was there any history of irradiation nor the ingestion of unusual drugs. The child was delivered normally at full term. Birth weight was 7 lbs. Post-natal examination report, on 18th. May 1969, recorded the child as healthy with a weight of 8¼ lbs.

Family History

The patient is the fourth son in a family of four. The elder two brothers are alive and well but the third died of pneumonia at the age of 2 months. There is no past history of twins in the family. The mother has no history of abortion.

Clinical Examination

Clinical examination on the 1st, July 1969, i.e. on the second admission, showed the general condition as fair and he appeared to be undernourished. The skin had little subcutanous fat, the limbs were thin, the cry feeble, and the child was easily exhausted during the course of the physical examination. The chest, with prominent ribs, was small in comparison to the grossly distended abdomen. The respiratory efforts were mainly thoracic as diaphragmatic movements were impaired. There was also a small umbilical hernia.

Palpation revealed a large cystic mass extending from the right hypochondrium to the right iliac fossa and encroaching medially up to the umbilicus. The mass could be felt per rectum.

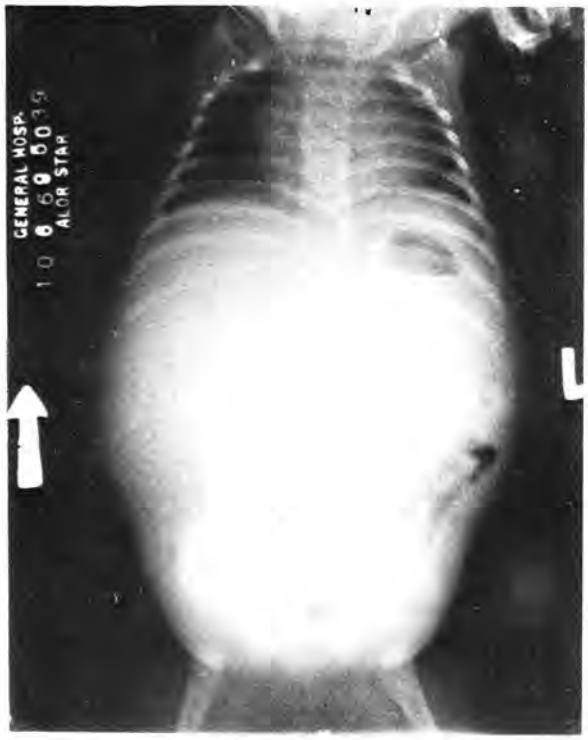

Fig 1
Plain X-ray of abdomen showing a large soft tissue mass shadow pushing the gut downwards and to the left.

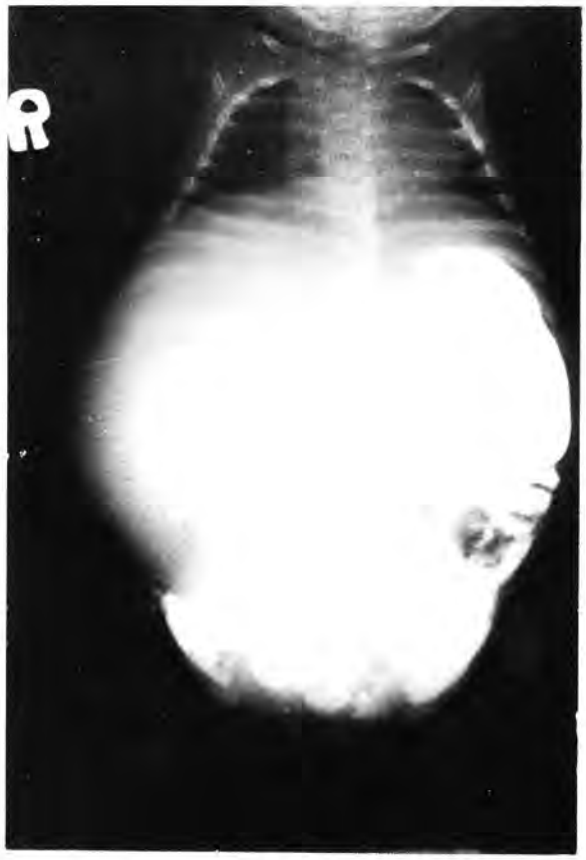

Fig 2
Barium meal and follow-through: shows stomach and duodenum displaced laterally to the left, with the small intestines also pushed downwards and to the left into the pelvis.

Investigations:

Plain X-ray done — mass larger than in previous film; no calcification detected; gut pushed to the left. The kidney shadows not seen. The appearance was suggestive of tumour of the liver or retroperitoneal tumour. Dermoid was thought of.

Barium meal **(Fig. 2)** showed stomach and duodenum displaced laterally, forwards and to the left. Further views showed that the stomach and duodenum were displaced anteriorly, i.e. the tumour was situated posteriorly.

I.V.P. **(Fig. 3)** showed both kidneys present and both excreted the dye satisfactorily. The right kidney lying in, or actually pushed, into the pelvis (over the right iliac bone). The left kidney was normal. The calyceal systems were normal.

The blood urea was normal (24mgm per 100 mls. blood).

Our investigations showed that the mass was a retroperitoneal cyst or tumour which was pushing the liver upwards, the right kidney downwards and the gut towards the left. The abdomen continued to get larger (25 ins. in circumference) and the patient became dyspnoeic. It was then decided to do a laparotomy. Laparotomy was done on the 20th. July 1969 under a general anaesthetic.

A transverse muscle-cutting incision, from below the 12th. rib to beyond the umbilicus, was done. A large cyst containing clear-coloured fluid was opened up. At the posterior wall of the cyst, a mass, 6 cms. X 2 cms., resembling a malformed foetus was seen. **(Fig. IV)** shows the mass. The cyst was adherent to the inferior surface of the liver but was dissectable. The lower end of the cyst was adherent to the right kidney, but was also dissectable. The cyst, together with

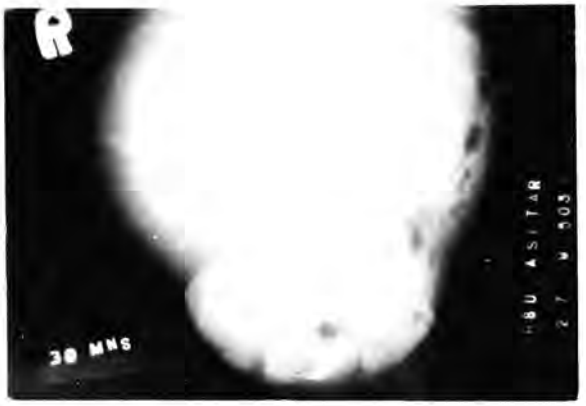

Fig 3
I.V.P. showing both kidneys present and excreting dye well: right kidney pushed into pelvis, left kidney in normal position.

Fig 4
The "foetus" with its limbs and its position in the abdomen as seen at laparotomy.

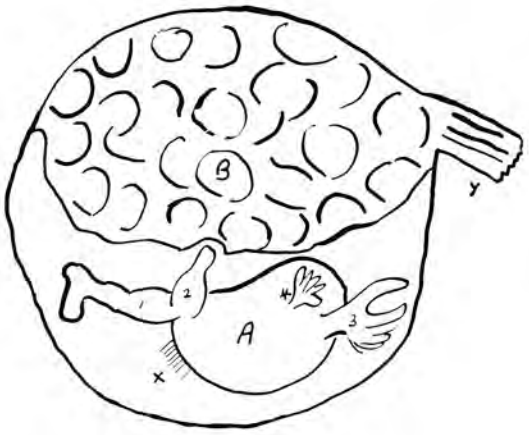

Fig 5
A – "foetus"; B – soft cystic mass; 1, 2, 3, 4 – limb buds; X – hairs; Y – pedicle, i.e. blood supply to mass from right renal vessels.

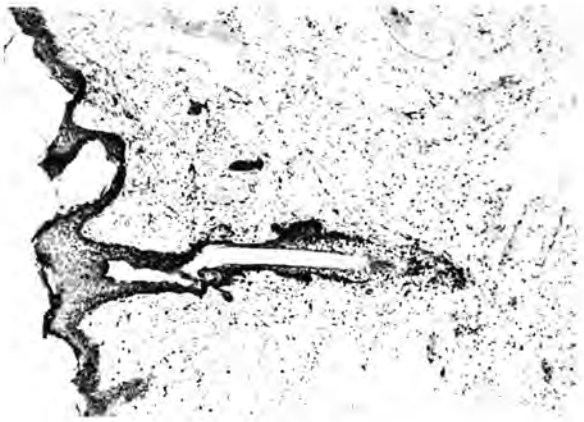

Fig 6
Microphotograph showing dermal elements.

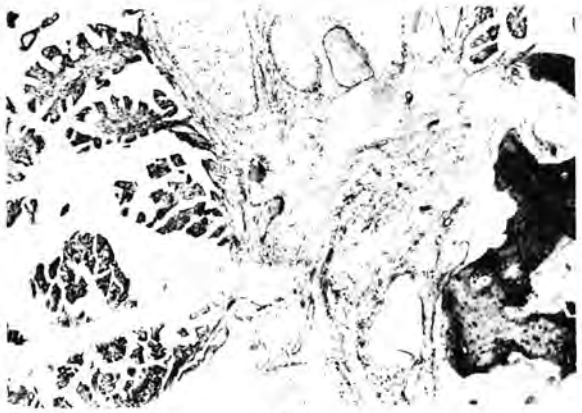

Fig 7
Microphotograph showing glandular elements, bone and cartilage.

the foetus-like mass, was completely taken out. As the blood supply came from the right renal vessels, the right kidney had to be sacrificed.

The patient made a slow but steady recovery and was discharged on the 21st. August 1969. Regular follow-ups showed that he was well and putting on weight.

The whole specimen was sent to the hospital pathologist, who reported as follows:

Naked eye appearance **(Fig. V)** The tumour is cystic with solid areas, almost rounded, measuring 10cms. in diameter. The cyst wall found torn in many parts (by operation). It consists of two areas – a solid

looking one (A in **Fig. V**), almost resembling a malformed foetus measuring 6cms. X 2cms. (in the widest area), with rudimentary looking limbs at either ends, irregularly placed and varying in size and shape (see 1,2,3,4 in **Fig. V**). No bony areas seen in the limbs, when the main mass was cut into. It was found to be composed of dense tissue with a cartilaginous feel in some areas. A few hairs were also seen (C in **Fig. V**). The other areas (B in **Fig. V**) were cystic all over – honeycombed in appearance. The cysts vary in size and shape.

Microscopic examination (**Figs. VI & VII**) section from 'A', i.e. the rudimentary foetus, showed dermal structures stratified squamous epithelial lining with sweat, sebaceous glands, hair follicles, areas of cartilage, bone and glandular structures mixed up with fibro-fatty areas and vascular mesenchymal tissue. Section from 'B' showed mostly fibro-fatty tissue with mesenchymal areas with few cysts lined by low cubical or atrophied epithelium.

Comments:

Is this a highly differentiated teratoma or an arrested development of the second twin in the body of the first twin? We cannot say.

Summary:

1. A rare case of "a foetus in the abdomen of a boy" is reported.

2. Diagnosis was made only after laparotomy as pre-operative X-rays showed no calcified parts. It is intersting that X-rays of the removed specimen showed areas of calcification.

3. This is probably the youngest boy to be successfully operated upon for such a condition.

4. It is the first reported case from Malaysia.

Acknowledgements:

In conclusion, I have to thank the Director General of the Malaysian Medical Services, Tan Sri Dr. Mohamed Din, for permission to publish this case; Dr. C. Subramanyam for the pathology report; Prof. Shanmugaratnam, University of Singapore, for his comments and microphotographs; Dr. T,G. Lim for the excellent general anaesthetic; Dr. Bhajan Singh for the photographs; and finally, Dr. Chin Yoke Hong and Dr. Y. Muthupalaniappan for help in the preparation of this paper.

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Book Reviews

AN EPIDEMIOLOGICAL SAMPLE SURVEY OF THE HIGHLAND, MAINLAND AND ISLAND REGIONS OF THE TERRITORY OF PAPUA AND NEW GUINEA, by A.P. Vines 1970, pp 621, Govt. Printer, Port Moresby.

THIS MOST COMPREHENSIVE SURVEY provides many basic medical statistics on the people of New Guinea. The text is divided into 24 chapters. The first seven provide background information on health services, attitudes to health and disease, and a quantitative analysis of housing, sanitation, etc. The remaining chapters deal with statistics on a wide variety of clinical data and laboratory measurements. Each chapter has a bibliography. There are several hundred tables, covering almost every aspect of health from alcoholic habits through various diseases, cardiovascular measurements, lactation and liver size, and left-handedness, to urine analysis. There is an appendix of more than 100 pages which describes the methods used.

It is both interesting and enlightening that this survey, which deals with health in one of the most under-developed countries in the world, is one of the most comprehensive surveys of its kind and used modern techniques of computer analysis.

This report is of great value in that it indicates the public health problems of the country and provides a great deal of basic data against which future changes can be measured. Twenty years ago, most of the people of New Guinea lived in a Stone Age culture; today, they are approaching independence. Great changes are occuring within a very short time. This survey will enable future workers to measure some of the changes in human health which occur as man changes from Stone Age living to a suburban materialistic existence. An understanding of some of these changes may be basic to the future health of mankind.

This report is essential reading for all those interested in epidemiology and tropical medicine.

W.H. Ewers

CONCISE ANTIBIOTIC TREATMENT by W.H. Hughes and H.C. Stewart 1970, pp 133 Butterworths, Lond. £1.00

THIS COMPACT PUBLICATION embodies the experience gained at St. Mary's Hospital, London, on this important subject and will serve as a ready reference book in an emergency for a busy practising doctor. It gives guidance on the drug and dosage most likely to be useful in the early stage of an infection before advice on the organism present and its sensitivity can be obtained. The relative toxicity of the drugs and the cost of treatment have been kept in mind. In addition to information on individual drugs and tables for quick reference to preparations, doses and routes of administration, there are short discussions on particular problems of dosage. This volume will also be useful for quick revision by the medical student preparing for his examinations.

EPIDEMIOLOGY OF NON-COMMUNICABLE DISEASES
Ed. by E.D. Acheson – British Medical Bull. Vol. 27. No. 1 January 1971. Published by Medical Dept. The British Med. Council, Lond. £2.00

THE FIFTEEN CONTRIBUTIONS, prepared by distinguished British specialists, cover a wide field in epidemiology, social, industrial and clinical medicine and medical statistics and will be of interest to all those engaged in community health programmes. Professor E.D. Acheson, the scientific editor, in his introduction calls attention to the changing meaning of the word "epidemiology" through the years, and shows how the implications of epidemiology techniques, assisted by modern statistical and computational methods and screening procedures, have become more pervasive.

ESSENTIALS OF MODERN MEDICAL TREATMENT by Vincent Norman. Revised Edn. 1970. pp. 168 H.K. Lewis & Co. Lond. £2 net.

THIS HANDY VOLUME is directed towards the general medical practitioner who is too busy to read through long detailed descriptions but requires an up-to-date account of the essentials of the medical treatment of common ailments. Prescriptions are included, giving doses and routes of administration of the drugs. There is an index.

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Ceporex (cephalexin)

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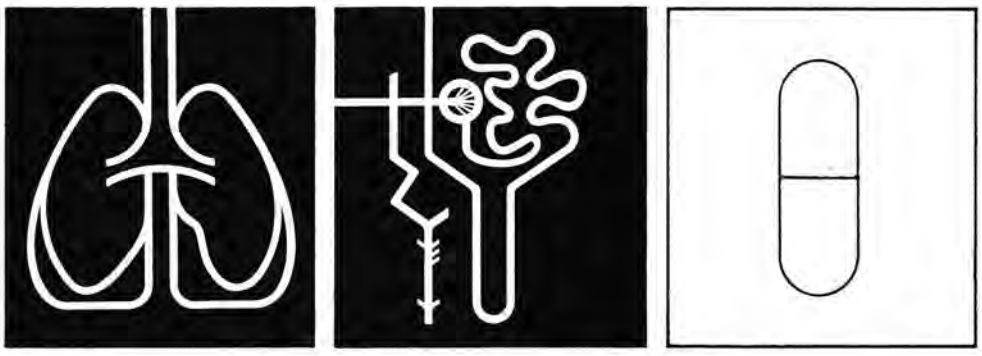

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