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Medical
Journal
of
Malaya

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THE SINGAPORE AND MALAYAN MEDICAL ASSOCIATIONS jointly hosted the delegates and observers to the fifth Council Meeting of the Commonwealth Medical Association held in August 1970. Those who attended the meetings, held both in Kuala Lumpur and in Singapore, were delegates from Australia, Britain, Canada, Ceylon, Ghana, India, Ireland, Jamaica, Malaysia, New Zealand, Pakistan, Sierra Leone and Tanzania, together with observers from the Australian Medical Association, the Medical and Dental Association of Botswana, the Barbados branch of the B.M.A., the Ceylon Medical Association, the Hongkong branch of the B.M.A., the Indian Medical Association, the Malayan Medical Association, the Mauritius branch of the B.M.A. and the Singapore Medical Association. Owing to difficulties in getting the necessary entry permit, the delegate from South Africa could attend only the second part of the meeting held in Singapore.

Among the officials of the Commonwealth Medical Association who attended were Dr. Gavin Johnson, the outgoing President and Dr. Derek Stevenson, the Honorary Secretary/Treasurer. Also in attendance was Dr. Alan Gilmour, the Medical Director of the Commonwealth Medical Advisory Bureau. One whose attendance was eagerly looked forward to by his many students in Singapore and Malaysia but who could not make it for private reasons was Professor D.E.C. Mekie, the executive Vice-President.

The meeting in Kuala Lumpur was chaired by Joint-President Professor A.A. Sandosham and declared open, owing to the unavoidable absence of Tunku Abdul Rahman, the Prime Minister, by Tun
Abdul Razak, the then Deputy Prime Minister of Malaysia. The meeting in Singapore was chaired by Joint-President Dr. Gwee Ah Leng and began with the formal installation of the Joint-Presidents by the outgoing President at a dinner at which Dr. Yeoh Ghim Seng, the Speaker of the Singapore Parliament, was the guest of honour.

Immediately after the approval of the applications for membership of the Tanzanian, Fijian, Jamaican and Sierra Leone Medical Associations came the proposal from Dr. A.M. Mamuiee (Tanzania) to expel the South African and Rhodesian Medical Associations from membership of C.M.A. as their governments' policies of apartheid and segregation were thoroughly inconsistent with its constitution. The aims and objectives of C.M.A. are:

1. To promote within the Commonwealth the interests of the medical and allied sciences, and to maintain the honour and traditions of the profession.
2. To effect the closest possible links between its members.
3. To disseminate news and information of interest.

It is well known that all is not well with the concept of the Commonwealth as an association of equal partners; there have been differences and divisions that have threatened seriously the continuance of this body on the political level. In fact, some countries like South Africa have severed connections with the Commonwealth in recent years. This council of nations understandably mirrors some of the problems faced by the C.M.A.

At the 18th Commonwealth conference held in Singapore in January this year, the racial issue came to the fore when the proposed sale of arms by Britain to South Africa was discussed. After many tedious hours of closed door meetings, the conference unanimously approved a modified version of the proposed Declaration of Commonwealth Principles enunciated by the Zambian President, Dr. Kenneth Kaunda, setting out guidelines for the future, and this saved itself and kept the Family of Nations together. It reads as follows: "No country will afford to regimes which practise racial discrimination assistance which in its own judgment directly contributes to the pursuit or consolidation of this evil policy." It was evident that the 31 nations within the Commonwealth regarded what unites them as more important than what appears to divide them. The same idea should hold good for the C.M.A.

In spite of the differences in culture, race, religion and political outlook, there is a strong bond of affinity among the medical fraternity for whom English has been the common medium of instruction and whose members are linked together by a bond of common tradition and the ideals of British medicine. The unifying effect has been such that the national medical associations, even of countries that have ceased to be members of the Commonwealth, seeing no sense in letting political differences break up the bond inherent in a common loyalty to medicine, have continued their membership of the C.M.A. However, with the presence at the fifth C.M.A. Council Meeting of more African and Carribean members, who are in close touch with the evil effects of the apartheid and segregation policies on the medical profession, it was inevitable that the continued membership of the South African and Rhodesian Medical Associations should be questioned.

To give the delegate of the South African Medical Association (Dr. P.D. Combrink) the opportunity to justify its continued membership, further discussions were postponed to the meeting held in Singapore, ably chaired by Dr. Gwee. In spite of Dr. Combrink's ready answers to the questions put, the fifth Council of C.M.A. resolved, by 13 votes for, none against, and four abstentions, to take the necessary steps to amend Clause 2 of the constitution so as to omit South Africa and Rhodesia from membership of the C.M.A. It was evident that several delegates were unhappy that a decision should have been taken on such an important issue without prior notice and the opportunity to obtain the considered views of their respective medical associations. An attempt to postpone decision was lost. Many realised that if a favourable decision was not taken then, the C.M.A. would probably break up, whereas there was the possibility of increasing membership to embrace all national medical associations of the Commonwealth who were probably holding back because of the continued membership of South Africa and Rhodesia. Incidentally, it was subsequently revealed (to avoid it playing a role in the deliberations on the South African and Rhodesian membership issue) that the Trustees of the Commonwealth Foundation had offered a grant of £30,000 over three years if the C.M.A. could find it possible to broaden its membership and expand the association's work and strengthen its headquarters staff and activities. This grant would not be payable if South Africa and Rhodesia continued as members of the C.M.A.

Dato (Dr.) Keshmahinder Singh's proposal, made on behalf of the M.M.A. that Professor Sandosham be
appointed C.M.A. Travelling Fellow for the coming session, was unanimously approved. This would provide him and the M.M.A. the opportunity to evaluate the health services in other countries and the ethical problems facing their medical profession. He could further the existing links among the medical profession in the Commonwealth countries he visited and encourage more national associations to join the C.M.A. Dr. Stevenson felt that the award of a fellowship for these purposes would be a development of significance to the C.M.A. in that it would mean bringing direct help to countries which obviously needed such assistance.

The meeting felt that the two-year interval between Council meetings of the C.M.A. was too long and in view of the cost of annual meetings, it was resolved that the executive officers of the association should meet at least once between full meetings of the Council. To deal with non-routine and non-urgent matters which could be circulated, it was felt desirable to set up regional secretariats with honorary secretaries to coordinate the activities of the national medical associations in their areas. It was tentatively agreed that the regions should be Southeast Asia, including Fiji, Australasia, the Caribbean and Canada, East Africa and West Africa.

It was also agreed that there was need for screening of candidates for bursaries from the Commonwealth Foundation and that the honorary secretary would refer individual applications to the National Medical Associations.

Four sub-committees were set up to work on problems before the next Council meeting, namely Canada and the Caribbean on the Constitution of the C.M.A., Malaya and Singapore on Ethics and Medical Ideology, B.M.A. on Finance, and East Africa on Medical Manpower.

Dr. A.G. Boohene of Ghana confirmed the offer of Accra as a site for the next meeting of the C.M.A. Council in 1972 and this was received with acclamation.

Several speakers eulogised the arrangements that had been made and the hospitality extended to the delegates, observers and their wives and the Council meeting terminated with a standing ovation accorded to the Joint-Presidents.

This is a brief account of the fifth Council meeting of the C.M.A. but naturally much else goes on at an international gathering than ever appears in its recorded proceedings. People meet their opposite numbers, delegates and observers exchange views privately and discuss their difficulties and everyone, including the ladies, have the opportunity of visiting new places and meeting socially the people of the host countries.
VALUE OF CEREBRAL ANGIOGRAPHY

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and A.H. Ang

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Introduction

CEREBRAL ANGIOGRAPHY was first described by Egas Moniz in 1927, when ventriculography was the method of choice in the localisation of brain tumours. His development of this new technique was probably motivated by the introduction of cholecystography at that time by Graham who successfully opacified the biliary system on intravenous injection of tetra-iodophenolphthalein, and noted few untoward side effects to the patient. Moniz realised that the first step was to look for an ideal contrast medium that would permit good opacification of the cerebral vasculature, with few side effects. After having experimented with various substances, he finally decided on 25% freshly prepared Na I, which appeared to satisfy his criteria to some extent. By 1931, he had performed over 300 examinations, but his technique of open dissection was not widely accepted and was generally regarded as rather dangerous and experimental. Loman and Myerson in 1936 introduced the percutaneous technique but it was not seriously considered nor practised and, as recently as 1945, some workers in the United States still dismissed it as "not a formidable procedure, but one that requires skill." However, Scandinavian workers helped by the use of Diodone, which had few side effects, persisted with this percutaneous method and by 1944, it had gained general acceptance. In England, Bull (1949) performed some 500 percutaneous carotid angiograms and although three deaths occurred in his series, there was no certain proof that any of them was directly due to the procedure. Engeset and his co-worker (1960), in reviewing 1,000 cerebral angiograms, found no complications attributable to the contrast medium or the arterial puncture. By this time, new and improved contrast media, the diatrizoate compounds, had been developed. Today, cerebral angiography is widely accepted as a safe and invaluable diagnostic procedure.

Technique

The procedure is performed under local anaesthesia on the sedated patient. In children under 16 years of age, and in apprehensive and unco-operative subjects, a general anaesthetic may be necessary.

With the patient supine and his head slightly extended on an A.O.T. changer, the common carotid artery is punctured as low down in the neck as possible, using an 18-G Cournand needle. Both walls of the artery are impaled and the needle is then withdrawn until its tip re-enters the arterial lumen, as evidenced by a vigorous flow of blood from the hub. The advantage of the Cournand needle lies in its having a blunt stylet which may be inserted after removal of the sharp stylet. It is then possible to advance the needle tip within the lumen of the artery without fear of perforating the posterior wall. A test injection of 4 — 5 mls. of 45% Hypaque shows the relationship of the tip of the needle to the arterial lumen and the presence or otherwise of an abnormality at the carotid bifurcation. If necessary, the tip of the needle in
CEREBRAL ANGIOGRAPHY

Figure 1. Left carotid angiogram showing frontal meningioma with (a) characteristic sunburst appearance in the arterial phase and (b) tumour blush in the venous phase.

relation to the arterial lumen can be altered. Filming is then carried out with an automatic serial changer. For routine purposes, five films are obtained covering a period of seven seconds which will include the arterial, capillary and venous phases. For the frontal and lateral projections, an injection of 10 mls. of Hypaque 45 given by hand is adequate to render good opacification of the cerebral vascular tree. Not more than three injections are given on one side except under special circumstances where oblique and per orbital views are required to display, for example, the exact locality of an aneurysm. An interval of 10 minutes is allowed to lapse between successive injections. This is done to reduce any adverse effects of the contrast on the brain. When the procedure is completed and the needle withdrawn, firm pressure should be applied to the puncture site for at least 10 minutes.

Complications
The diatrizoate compounds possess characteristics that match the ideal contrast medium in many respects. Used in small quantities and low concentrations, their toxicity is very low. Their relatively high viscosity gives good visualisation of very small vessels. Although with these modern contrast media and other refinements in technique complications are uncommon, they do occasionally occur. These manifest commonly as an aggravation of neurological signs, usually transient and not severe. Evaluation of deterioration of the neurological state of the patient is often made difficult by the concomittant effects of general anaesthesia and sedation. The common local complications include subintimal and peri-arterial injections of contrast material. Haematoma formation at the site of puncture occurs quite frequently and it is important to recognize it before it becomes large enough to compress the trachea. Recently, Ansell (1968) reported complications in 9.6% of patients requiring more than four attempts at puncture, with subintimal and peri-arterial injections accounting for 7.1%. Bull (1960) noted a 10% incidence of complications in 80 cases of clinically recognised strokes submitted to carotid angiography. It is obvious, therefore, that apart from careful use of contrast material, skill is required of the operator in reducing complications to a minimum.

Indications
(1) Tumour Demonstration and Diagnosis
It is now generally accepted that carotid angiography is the method of choice in investigating space occupying lesions of the cerebral hemisphere. The pathology of some tumours can be identified with a fair degree of certainty by their vascular pattern and "staining" characteristics. Many meningiomas show a typical "sunburst" appearance in the arterial phase, with the contrast material lagging behind in the venous phase in the form of a uniform, homogenous, well-defined "blush". These features are well illustrated in the following patient.

Case (1)
T.A.J., a 40-year-old school teacher, complained of headache of 4 years' duration, progressively getting worse during the past 6 months. For the past one
year, he had also noticed loss of libido and progressive blindness. At the same time, he was noticed to be forgetful and often unintelligible. The clinical findings pointed to a left frontal lobe tumour. Plain skull radiographs showed thinning of the floor of the sella turcica consistent with prolonged raised intra-cranial pressure. A left carotid angiogram (Fig. 1) showed a huge tumour in the frontal lobe extending backwards to the supra-sellar region. The tumour was supplied by a hypertrophied, tortuous anterior branch of the middle meningeal artery and had the characteristic “sunburst” appearance of a meningioma. A typical tumour blush was noted in the venous phase. The patient was successfully operated on in another hospital, where the angiographic diagnosis was confirmed.

Case (2)

O.S., aged 68, was a chronic depressive undergoing treatment in a psychiatric ward. On 14.4.69, he fell while in the toilet and lost consciousness for half an hour. Physical examination showed exaggerated reflexes, and plantar response was extensor on the left side. A bilateral carotid angiogram showed a suggestion of a thin subdural collection over the left convexity. No other abnormality was seen. The patient apparently recovered the next day and was discharged, later to be followed up. On 9.7.69, he was brought back to the hospital in a comatose state. Bi-
CEREBRAL ANGIOGRAPHY

Figure 3. (a) and (b) Left carotid angiogram showing aneurysm at origin of posterior communicating artery.

lateral carotid angiogram showed a huge lenticular avascular area on the left side (Fig. 2) and a similar lesion of smaller size on the right. Surgery confirmed the diagnosis of bilateral chronic subdural haematoma.

(III) Subarachnoid Haemorrhage

The excellent paper by Bull (1962) and the figures reported by Sutton (1962) leave little doubt as to the role of cerebral angiography in the investigation of subarachnoid haemorrhage. In Sutton’s series, a lesion was demonstrable in 75% of cases by bilateral carotid angiography and the percentage of positive findings rose to 96% when bilateral vertebral angiograms were done as well. The proportion of arteriovenous malformations in relation to cerebral aneurysms increased when the posterior fossa was thus investigated.

Case (3)

L.D.K., a 58-year-old female patient, was admitted on 9.8.69 with a history of loss of consciousness for 5 hours. There was marked neck rigidity and right-sided weakness; plantar responses were extensor. A lumbar puncture showed blood-stained C.S.F. and the diagnosis of subarachnoid haemorrhage was made. Her condition remained unchanged for 4 days and on 13.8.69 a bilateral carotid angiogram was done (Fig. 3). This showed an aneurysm, measuring 1.2 cm in diameter, at the origin of the posterior communicating artery on the left side. No vascular spasm nor evidence of a haematoma was seen. On the right side, two smaller berry aneurysms were noted, one arising from the trifurcation of the middle cerebral artery and the other from the bifurcation of the internal carotid artery.

Intracranial and Extracranial Occlusive Vascular Lesions

The segmental nature and multiplicity of these occlusive or stenotic lesions causing cerebral vascular insufficiency have been well established in the past three decades. Following on the work of Hutchinson and Yates (1957) who pointed out the frequency of involvement of the vertebro-basilar system by atherosclerosis, it is now accepted that these vessels should be investigated in patients presenting with manifestations of carotico-basilar insufficiency. The current accepted practice is to study the origins and course of the head vessels by arch aortography and to supplement this examination by carotid angiography where necessary. Recent advances in corrective vascular surgery and the limited success of anti-coagulant therapy have made it almost mandatory to investigate the carotid systems, particularly in young patients presenting with the stroke syndrome.
Case (4)

I.K., a 49-year-old truck driver, had been well except for mild hypertension for the past 2 years. For the past 3 months, he had noticed right-sided weakness, blurring of vision, and difficulty in speaking. Examination showed a blood pressure of 160/110. There was nominal aphasia. He also had right hemiparesis and homonymous hemianopia. The clinical impression was thrombosis of the middle cerebral artery, although an internal carotid artery occlusion had to be excluded. A left carotid angiogram was performed. A preliminary film of the neck following injection of 4 ccs. of 45% Hypaque ruled out a stenosis or occlusion at the common carotid bifurcation. The cerebral angiogram showed an occlusion of the main trunk of the middle cerebral artery, with non-filling of its distal branches in the arterial phase (Fig. 4a). There was retrograde filling of the distal middle cerebral arteries from branches of the anterior cerebral at 4.5 seconds (Fig. 4b). The diagnosis of middle cerebral artery thrombosis, with satisfactory collateral filling, was made.
CEREBRAL ANGIOGRAPHY

Angiomatous Malformations

These lesions are congenital in origin, although they usually present during adult life. The clinical presentation depends on the site of the lesion. Usually located superficially, these anomalies can shunt blood away from neighbouring areas resulting in underlying cerebral ischaemia and later atrophy. When situated over the motor cortex, the first manifestation may be that of Jacksonian epilepsy. Danger of rupture into the subarachnoid space or the brain substance makes it a neuro-surgical problem once discovered. Angiomatous malformations, once reported

Figure 4. Left carotid angiogram. (a) Early arterial phase showing occlusion of main trunk of middle cerebral artery with non-filling of its distal branches. (b) Late arterial phase showing retrograde filling from branches of the anterior cerebral artery.

Figure 5. (a) and (b) Late arterial phase of left carotid angiogram showing angiomatous malformation in the fronto-parietal and parieto-temporal region. Arrows indicate prematurely opacified hypertrophied draining veins.
as rare, are not that uncommon nowadays, with increasing use of cerebral angiography. They account for up to 15% of all cases of subarachnoid haemorrhage (Du Boulay 1967).

Case (5)
S.P.Y., a 37-year-old clerk, presented with Jacksonian epilepsy of 5 years' duration, associated with progressive weakness and hemi-anæsthesia of the right half of the body for the same period. Examination showed increased tone and reflexes of the right upper and lower limbs. The clinical impression was a focal lesion in the left parietal region. A left carotid angiogram showed a large angiomatous malformation in the left fronto-parietal and parieto-temporal region (Fig. 5). The vascular anomaly was supplied by branches of the anterior and middle cerebral arteries on the same side. The cerebral circulation was characteristically accelerated, with early venous filling. The presence of tortuous and enlarged draining veins is pathognomonic of this condition.

Discussion
Advances in technique and interpretation have made cerebral angiography a precise tool in the localisation and diagnosis of hemispheric lesions. Ventriculography is less frequently used on account of this but where a lesion is deeply situated near to the ventricular systems, air encephalography or ventriculography may still be necessary.

Where a tumour circulation is shown, its appearance is sometimes pathognomonic. This is illustrated in Case 1 where demonstration of a tortuous and hypertrophied middle meningeal artery and uniform, well-defined contrast staining up to 8 seconds put the issue beyond doubt. A diagnosis could have been made on plain films if attention had been directed to the prominent arterial impressions on the vault and an increase in density in the floor of the anterior and middle cranial fossae. In general, a little under 50% of meningiomas will show a tumour circulation and a meningeal arterial supply. This is particularly true of parasagittal and high convexity tumours. Where a tumour circulation is absent, a hypertrophied meningeal artery has still to be looked for as its presence may be the only clue to the diagnosis (Banna and Appleby 1969).

Cerebral angiography has an important place in the management of subdural haematoma, particularly of the chronic varieties. Here, the angiographic findings are able to show the number, location and extent of these lesions as illustrated in Case 2.

Case 3 illustrates the difficulties the radiologist may encounter in deciding which aneurysm has bled. There was no associated vascular spasm or vessel displacement on either side to give even a suggestion, and localisation in this instance was made on purely clinical grounds.

The carotid angiogram in Case 4 was done in the hope of finding a surgically correctable lesion in the internal carotid artery. Instead, a thrombosis of the middle cerebral artery was found. Serial films up to 8 seconds had not shown any stasis of the contrast in the distal middle cerebral branches and the presence of a propagating thrombosis could be safely ruled out.

Case 5 demonstrates the typical radiological appearance of an angiomatous malformation, although operative confirmation was not available because the patient refused surgery. It must be pointed out that investigations on this patient would have been incomplete if surgery was contemplated. Multiple feeding vessels to the lesion would have to be excluded and this would have at least required an ipsilateral vertebral angiogram with a contra lateral carotid study.

Summary
1. A technique of percutaneous carotid angiography is described.
2. The possible complications of the procedure are discussed.
3. Examples are presented to illustrate the main indications of cerebral angiography.

Acknowledgements
We wish to thank Dr. H.O. Wong for permission to publish the cases, the Department of Medical Illustration for the photographs, and Miss Janet Low for typing the manuscript.

References
The demand for abortion in an urban Malaysian population

by O.S. Ooi
MB B. Chir., DCH, MRCP (U.K.)

Introduction
A SURVEY OF THE WORKLOAD of an urban general practice in Penang was carried out from January to June 1970, the results of which will be reported elsewhere. The data presented in this paper are, however, derived from the same survey.

The extent of the demand for abortion was a surprise to the author. There seems to be a significant number of women who visit their general practitioners complaining of amenorrhoea of varying length and ask for injections to bring on their periods. A few of these are more brazen and experienced, and frankly ask for an abortion. From information volunteered by the patients and from conversations with other general practitioners, it appears that the practice has grown up for most general practitioners to give such patients an oestrogen progestogen injection, hoping that the patient is not pregnant and that withdrawal bleeding may be induced in this way. An MRCOG friend of the author has given his private opinion that such a procedure should not dislodge any existing pregnancy. There is, therefore, an ambivalence connected with this procedure with the patient believing that the general practitioner is trying to induce an abortion and the general practitioner really performing a parenteral pregnancy test.

It should, perhaps, be pointed out that the author was not previously greatly interested in the subject but as he proceeded with general practice, it soon became evident that this was a problem which he could not ignore and which should not be swept under the carpet. Abortion is a moral question that has to be decided by the whole community. It is the intention of this paper to provide some data on which moral decisions can be made and secondarily to make sure that the voice of Malaysia’s “silent majority”, the urban poor, be heard.

Material and Methods
The data in this paper is based on records made during consultations from January to June 1970. For details of the practice and of the way records were kept, e.g. the assignation of social class, the reader is referred to the main paper (Ooi, 1970). At the beginning of this period before the extent of the problem was realised, many details of interest were not specifically asked for: these appear under the category “not noted” in the various tables. From about March onwards, the particulars analysed in the following tables were routinely asked for and recorded.

Results
The total number of cases with amenorrhoea seen between January and June 1970, was 214.

Table I analyses the distribution of the cases seen with respect to age, Table II with respect to class, and Table III with respect to race.
### TABLE I
**Age Distribution**

<table>
<thead>
<tr>
<th>Age in Years</th>
<th>Number of Patients</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>14 – 17</td>
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<td>0.9</td>
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<tr>
<td>18 – 20</td>
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<tr>
<td>46 – 50</td>
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<tr>
<td><strong>Total</strong></td>
<td><strong>214</strong></td>
<td><strong>100</strong></td>
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</tbody>
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### TABLE II
**Distribution of Social Class**

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<th>Social Class</th>
<th>Number of Patients</th>
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<tr>
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<td><strong>Total</strong></td>
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<td><strong>100</strong></td>
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### TABLE III
**Racial Distribution**

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<tr>
<th>Race</th>
<th>Number of</th>
<th>Percentage</th>
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<tr>
<td>Chinese</td>
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<td>Malay</td>
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<td>Indian</td>
<td>2</td>
<td>0.9</td>
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<tr>
<td><strong>Total</strong></td>
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<td><strong>100</strong></td>
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### TABLE IV
**Table IV**

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<tr>
<th>Number of Children</th>
<th>Number of Patients</th>
<th>Percentage</th>
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</thead>
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<td>None: unmarried</td>
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<td>7.5</td>
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<td>None: Married</td>
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<tr>
<td>9 – 10</td>
<td>7</td>
<td>3.3</td>
</tr>
<tr>
<td>&gt; 10</td>
<td>2</td>
<td>0.9</td>
</tr>
<tr>
<td>Not Noted</td>
<td>3</td>
<td>1.4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>214</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

### TABLE V
**Age of Youngest Child**

<table>
<thead>
<tr>
<th>Age Youngest Child</th>
<th>Number of Patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1 yr.</td>
<td>59</td>
<td>31.4</td>
</tr>
<tr>
<td>1 yr.</td>
<td>22</td>
<td>11.7</td>
</tr>
<tr>
<td>2 yrs.</td>
<td>17</td>
<td>9.0</td>
</tr>
<tr>
<td>3 yrs.</td>
<td>12</td>
<td>6.4</td>
</tr>
<tr>
<td>4 yrs.</td>
<td>18</td>
<td>9.6</td>
</tr>
<tr>
<td>5 yrs. and over</td>
<td>42</td>
<td>22.3</td>
</tr>
<tr>
<td>Not Noted</td>
<td>18</td>
<td>9.6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>188</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

Note: Figures exclude the 26 childless women in the series.

### TABLE VI
**Place of Birth of Youngest Child**

<table>
<thead>
<tr>
<th>Place of Birth</th>
<th>Number of Patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penang Maternity Hosp.</td>
<td>86</td>
<td>46.2</td>
</tr>
<tr>
<td>Govt. Hosp. outside Pq.</td>
<td>16</td>
<td>8.5</td>
</tr>
<tr>
<td>Private Nursing Home</td>
<td>10</td>
<td>5.3</td>
</tr>
<tr>
<td>At Home</td>
<td>27</td>
<td>14.4</td>
</tr>
<tr>
<td>Not Noted</td>
<td>50</td>
<td>26.6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>188</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

Note: Figures exclude the 26 childless women in the series.

Table IV shows the marital status of the patients and the number of children in each family. Table V shows the age of the youngest child in each family and Table VI the place of birth of the youngest child.
DEMAND FOR ABORTION

Table VII shows the number of days at the time of consultation by which the period was overdue.

**TABLE VII**

<table>
<thead>
<tr>
<th>Length of Time</th>
<th>Number of Patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 - 3 days</td>
<td>10</td>
<td>4.7</td>
</tr>
<tr>
<td>4 - 7 days</td>
<td>56</td>
<td>26.2</td>
</tr>
<tr>
<td>1 wk. 1 day - 2 wks.</td>
<td>70</td>
<td>32.7</td>
</tr>
<tr>
<td>2 wks. 1 day - 4 wks.</td>
<td>38</td>
<td>17.8</td>
</tr>
<tr>
<td>4 wks. 1 day - 6 wks.</td>
<td>11</td>
<td>5.1</td>
</tr>
<tr>
<td>6 wks. 1 day - 8 wks.</td>
<td>16</td>
<td>7.5</td>
</tr>
<tr>
<td>&gt; 8 weeks</td>
<td>4</td>
<td>1.9</td>
</tr>
<tr>
<td>Total</td>
<td>214</td>
<td>100</td>
</tr>
</tbody>
</table>

Table VIII shows whether the patient had practised any method of contraception in the past. Table IX lists the reasons advanced by patients for stopping the oral contraceptive pill and Table X those advanced by patients for never having tried the pill.

**TABLE VIII**

<table>
<thead>
<tr>
<th>Previous Method of Contraception</th>
<th>Number of Patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral Contraceptive Pill</td>
<td>87</td>
<td>36.1</td>
</tr>
<tr>
<td>IUD*</td>
<td>6</td>
<td>2.5</td>
</tr>
<tr>
<td>Diaphragm, sheath, spermicides</td>
<td>17</td>
<td>7.1</td>
</tr>
<tr>
<td>Coitus Interruptus</td>
<td>5</td>
<td>2.1</td>
</tr>
<tr>
<td>Safe Period</td>
<td>3</td>
<td>1.2</td>
</tr>
<tr>
<td>Previous Abortion +</td>
<td>5</td>
<td>2.1</td>
</tr>
<tr>
<td>Previous Successful Injections</td>
<td>19</td>
<td>7.9</td>
</tr>
<tr>
<td>Chinese Herbs</td>
<td>3</td>
<td>1.2</td>
</tr>
<tr>
<td>None</td>
<td>44</td>
<td>18.3</td>
</tr>
<tr>
<td>Not Noted</td>
<td>52</td>
<td>21.6</td>
</tr>
<tr>
<td>Total</td>
<td>241</td>
<td>100.1</td>
</tr>
</tbody>
</table>

* Of the 6 patients who had used an IUD, 3 had had it removed because of menorrhagia; 2 because of fear that its long term presence might cause cancer; and 1 presented with amenorrhoea because the IUD had fallen out without her noticing it.

+ Some on repeated occasions.

Note: A patient may use more than 1 method of contraception. The total of this table is therefore greater than the total number of patients.

**TABLE IX**

**REASONS ADVANCED FOR STOPPING THE ORAL CONTRACEPTIVE PILL**

A. Medically Acceptable Reasons.

- Retrosternal discomfort
- Shortness of breath
- Palpitations
- Vomiting and/or Giddiness
- Headache
- Periods became irregular
- Periods became scanty
- Generalised pruritus
- Became thin
- Became fat

Total 54

B. Reasons Founded on Rumours and Fears.

- Was told that the pill is harmful if taken for too long (no ill effects specified)
- Frightened by adverse newspaper publicity
- Thought pill can cause cancer if taken for too long
- Friend had definite ill effects from pill
- "Constantly ill" while on pill
- Thought she was too weak to tolerate pill
- Thought it was necessary to stop pill during an acute illness

Total 3

C. Reasons due to Personality or Personal Circumstances.

- Forgot to buy pill or takes it irregularly
- Husband was away, then returned suddenly

Total 19

Note: Some women gave more than 1 reason for stopping the pill.

**TABLE X**

**REASONS ADVANCED FOR NOT STARTING ON THE ORAL CONTRACEPTIVE PILL**

- Never heard about the pill
- Was told that the pill is harmful (no ill effects specified)
- Thought the pill can cause cancer
- Thought she was too weak to tolerate the pill
- Friend had definite ill effects from the pill
- No time to buy the pill
- Too lazy to buy the pill
- Afraid she may not remember to take the pill
- Does not know where to buy the pill
- Thought she was too old to need the pill (patient was 38 years old)
- Husband objected to the pill

Total 36
The author neglected to note down in the records as a routine the reasons advanced by patients for not wanting the pregnancy and therefore no figures are available for this. However from memory and incomplete records, the reasons advanced are listed in Table XI.

**TABLE XI**  
REASONS FOR NOT WANTING PREGNANCY

<table>
<thead>
<tr>
<th>Reason</th>
<th>Number of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not married</td>
<td>16</td>
</tr>
<tr>
<td>Patient engaged in prostitution</td>
<td>6</td>
</tr>
<tr>
<td>Too soon in marriage to have baby</td>
<td></td>
</tr>
<tr>
<td>Wife is the sole wage earner</td>
<td></td>
</tr>
<tr>
<td>Too poor</td>
<td></td>
</tr>
<tr>
<td>Too many children</td>
<td>An overwhelming majority of patients</td>
</tr>
<tr>
<td>Too old</td>
<td></td>
</tr>
</tbody>
</table>

Fifty-nine patients volunteered the information that they had had treatment with another general practitioner before coming to see the author. It should be noted that this is an underestimate as the patients were not routinely asked if they had consulted another general practitioner before consulting the author. Only information offered or elicited during history taking are noted down. An unrecorded large number had also tried various Chinese herbs to induce the period before coming to see the author.

With regard to the author’s treatment of these cases, local custom was followed in some patients and an oestrogen progestogen injection prescribed. Others, especially those who had had treatment elsewhere or with a long period of amenorrhoea, were advised that an injection was unlikely to help, and the author tried his best to persuade them to accept the pregnancy, but it is unlikely that he succeeded with many. All were strongly urged to go on to recognised methods of contraception. With regard to results, 18 were known to have periods after the injection and 4 were known to become reconciled to the pregnancy. Results for the rest are not known.

It is unknown to the author why there should be such a great popular belief in the efficacy of injections by general practitioners as a means of inducing abortion. An unknown proportion of women subjected to such injections will have amenorrhoea due to causes other than pregnancy. In these, the injections will be successful. It is a bit surprising to the author that the numbers who had had successful injections should be sufficient to popularise the method.

**Discussion**

**The Desire for Family Planning.**

From the table of racial distribution (Table III), it can be seen that Malays and Indians form only 1.4% and 0.9% respectively of patients seeking a remedy for amenorrhoea compared with 6.1% and 4.0% respectively for the practice as a whole (Ooi, 1970). It is not known why this should be so but, if one wishes to speculate, the possible reasons may be that the idea of having abortions induced by injections given by general practitioners is not popular or widely known among them or that they resort to other methods of abortion, e.g., unqualified abortionists. It is also possible that Malays and Indians do not accept the advantages of small families, but for the Malays certainly, this is refuted by the figures of the Penang Family Planning Association (Annual Report 1969) which show a better follow-up rate with the pill for the Malays than for the other racial groups. It may well be that the Malays are more effective users of modern contraceptive methods and therefore have less need to seek abortions.

From this point onwards, the discussions and conclusions are concerned only with the urban Chinese as the number of Malays and Indians is too small for any further conclusions to be drawn.

There seems to be no doubt that the modern urban Chinese desire to limit the size of their families. They have thrown overboard the traditional preference for large families, many mothers stating with conviction that they regard a family size of two or three or four children as sufficient. This is supported by the figures of Table IV which show the large number of married women who seek abortions with only one to four children in the family. The modern urban Chinese also seek to avoid having children too close together. This is shown by the figures of Table V which show that the greatest number of women who seek an abortion has a child of less than one year old. It is interesting to note that the incidence falls to a minimum with the youngest child aged 3 years, then rises again, so that a considerable number of women whose children are aged five years and over seek an abortion — many of these are, of course, in the older age groups. An indication of the fact that the traditional Chinese way of life is changing is provided by the number of nulliparous married women who appear to be more strongly motivated by economic factors, e.g., the wish to go on working to maintain or increase the family income, than by the wish to start a family.
DEMAND FOR ABORTION

It is interesting to note that the demand for abortion is spread fairly evenly throughout the different age groups. It is interesting, too, that there were 7 women in the age group 46 to 50 years who demanded an injection to bring on their periods. All of these women came when their periods were only a few days late. Their fear of another pregnancy was so great that the author found it extremely difficult to convince these patients that they may be faced with the onset of menopause rather than with another pregnancy, and to wait 2 weeks for a definitive pregnancy test.

Table VII shows that the majority of patients came demanding their injections when their period was overdue by 2 weeks or less. This is perhaps a reflection of popular knowledge that the injection is unlikely to work after this time. It is also a reflection of the very strong fear of a further pregnancy that many women appear to have, causing them to rush to try any means to bring on their periods. This is pushed to its most ludicrous limits by the 10 women who demanded an injection when their period was delayed by as little as 1 to 3 days!

Failure of Modern Contraceptive Methods to Abolish the Demand for Abortion.

An argument often used by opponents of legalised abortion is that there should be no need for it with modern methods of contraception. But Table VIII shows that present methods of contraception still have a long way to go in stopping the demand for abortion. The author was somewhat surprised to find that as many as 93 (43.5% of the patient total) had practised a modern form of contraception (pill or IUD) in the past, and yet found the disadvantages and fear of these methods greater than the dangers of an abortion or the uncertainties of a hormone injection by a general practitioner. It should also be noted from the same table that, despite the availability of such methods, there are women who resort to repeated abortions as a method of contraception.

If modern contraceptive methods are to reduce the present level of demand for abortion, then ways must be found of increasing their effective use. Table IX shows that 40.7% (37/91) of the reasons advanced for stopping the pill were non-medical and unnecessary. If this figure is considered, together with Table X listing the reasons advanced by women for never having tried the pill at all, then it can be seen what a large proportion of unwanted pregnancies could have been prevented with the pill.

Considering that the desire of the modern urban Chinese to limit the size of the family is so strong, it is a matter of some surprise to the author that as many as 10 women in this series had never even heard about the pill! Most of these women belong to social class 4 and had large families. The existence of these women would seem to be an indication for further campaigns to be mounted to disseminate knowledge of the methods of contraception among the poorer social classes.

Table VI shows that 53.7% of the women in this series gave birth to their last child in a Government hospital. If the 50 women in the “not noted” category are excluded, the proportion rises to 73.2% (101/138). If this is taken in conjunction with the fact that 31% of the women have a child aged less than one year, then it would seem that the person most likely to demand an abortion is the woman who has delivered a baby in a Government hospital within the last year. It is surprising that this should occur despite the fact that all post partum women in the Penang Maternity Hospital are visited by a worker of the Family Planning Board for a chat on contraception plus one month’s free supply of the pill on request. This finding suggests that the family planning authorities should study and experiment with various ways of communicating the facts and methods of modern contraception to these post partum women. For example, will better results be obtained if these women are given a half hour detailed lecture in groups so that they can discuss the implications among themselves, or if pamphlets giving details of the various contraceptive methods and their advantages and disadvantages are distributed to them to take home for study, or if chats on contraception are given in the presence of the husband (in passing it should be noted that a significant number of the women demanding abortions are accompanied by their husbands who are just as keen that the pregnancy be terminated). It would also be interesting to see to how great an extent the personality, tact, and instructive ability of each family planning worker influences her patients’ decision to adopt a modern contraceptive method – for instance, is there any significant difference in the follow-up rates following chats by different family planning workers?

It is the experience of the author that whatever the reason the patients advanced for stopping the pill, the overwhelming majority did so without consulting any doctor whatsoever. It would seem a fair conclusion of this series that a great number of failures with the pill could be avoided if each and every woman given it, especially in family planning clinics staffed
by nurses only, could be told never to stop it when suffering either from definite side effects or anxiety engendered by rumours, without consulting a doctor first. The patient could be then either reassured or, if suffering from definite side effects, changed to another preparation or to another method.

Intolerance to Modern Contraceptive Methods.

However great a believer in family planning one may be, it is impossible to run away from the fact that there is a significant number of women who complain of definite side effects from the pill. It is a matter of speculation to the author what proportion of the women with medically acceptable reasons in Table IX did really experience these side effects and what proportion imagined they were suffering from these side effects after hearing rumours and stories from friends. However this may be, it is interesting to note that there is a surprisingly marked difference in the incidence of side effects between those reported in this series and those reported for European women on the pill. Headache is a very common side effect in European women (Grant, 1968) but was only complained of by one person in this series. On the other hand, retrosternal discomfort, shortness of breath, and palpitations figure very prominently in Malaysian women but are not mentioned as common side effects in European women (BMJ, 1968).

It is the impression of the author from this series that many women on the pill have little idea of what it is all about, and have had no warning of what side effects to expect and what to do about them should they occur. A proportion of these women buy their pills without prescription direct from pharmacies, and a proportion may be too stupid to grasp any explanations, but a proportion are women of average intelligence who get their pills from family planning clinics. It is suggested that a better follow-up rate may be obtained if women on the pill know what it is all about, and are warned that some may experience side effects, which may be intolerable enough in a few to necessitate changing to another method, but are reassured at the same time that such side effects are not permanent nor life threatening: it is suggested such women will not be alarmed when side effects occur, and therefore will not stop the pill precipitately. Although granted that warnings about side effects may cause the patient to imagine that she is experiencing them, yet rumours about side effects from the pill are so widespread that women given no official guidance fall an easy prey to such rumours.

It is possible that with growing acceptance of modern methods of contraception, unwanted pregnancies will cease. This might be an unachievable goal, as there appears to be women with large families who are unable to tolerate any method of contraception, however great their desire to limit the size of their families. The author has encountered a few patients in this series who had had the IUD removed for various reasons and had then been unable to continue with the pill because of side effects. Furthermore modern methods of contraception may fail: for instance, the author has great sympathy with the patient in the following case history.

Case No. 302421

A 46-year-old Chinese of social class 3. This woman had delivered 6 children, of whom 4 were living and 2 had died. The youngest child is 9 years old. She had had an IUD inserted 4 years ago and had her last check up 1 year ago when she was told everything was all right. She presented with 2 weeks amenorrhoea and on examination, the thread of the Lippes loop was no longer seen issuing from the os.

This woman had followed instructions and yet had become pregnant. She is elderly, already has many children and has a great aversion to having another child. There is a basic injustice in the situation which compels the doctor to say to the patient: “I am afraid I cannot help you. You have to take your problem elsewhere.” A humanitarian consideration of the problems of women in groups such as these, e.g. the woman who gets pregnant because she is unable to tolerate any method of contraception, the woman who gets pregnant while on a recognised method of contraception, and the woman who is unmarried, compel the author to urge that abortion should be legalised for these special groups. Clearly if abortion is legalised, safeguards will have to be incorporated, but it is not the author’s intention to discuss these here.

Conclusion and Summary

The twin objectives of this paper have been to set down facts and figures relating to the demand of women in an urban population for abortion and to publicise the fears and misconceptions of many women regarding modern methods of contraception so that steps may be taken to combat these fears. The desire of the modern urban Chinese for family planning is exceedingly great and yet innumerable women find themselves with an unwanted pregnancy. A total of 43.5% of those with an unwanted pregnancy had tried a modern method of contraception in the past.
DEMAND FOR ABORTION

and had then abandoned it, some for medically acceptable but too many for spurious and preventable reasons founded on ignorance and fear. Some suggestions for reducing the high dropout rate from the pill are discussed.

However, certain groups of women get pregnant despite sincere efforts to avoid it, e.g. those intolerant to all methods of contraception, and it is suggested that abortion should be legalised for these special groups.

Acknowledgements

My thanks are due to the members of my staff, Messrs. Khoo Boon Siew, Tan Cheng Loke and Teoh Kheng Bee, for keeping the records in good order and for helping in the analysis of the data. My thanks are also due to my father, Dr. Ooi Kee Wan, for advice.

References

Ooi, O.S. "An Analysis of the Work Load of an Urban General Practice in Malaysia with a Discussion of the Problems Encountered." Submitted to the Editor, Medical Journal of Malaysia, for publication.
The pattern of physiological changes in pregnancy in Malaysian women

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Introduction
STUDIES OF TOTAL WEIGHT gain in pregnant women in other parts of the world have revealed wide variations from about 6 kgm. (12 pounds) in Indian and Nigerian women (Hauck, 1960) to about 14 kgm. (30 pounds) in European and American women (Hyttten and Leitch, 1966). Thus far, there has been no planned study undertaken to map out the physiological pattern of weight changes in Malaysian women throughout their pregnancy and puerperium.

As from the middle of 1968, a prospective study was embarked to evaluate the pattern of weight changes in normal pregnant women at the Obstetric Unit, University Hospital, University of Malaya in Kuala Lumpur, Malaysia. The subjects that were incorporated into this study had to fulfill certain definite criteria. These were:

1. the subjects were healthy and normal
2. the exclusion of all abnormal pregnancies
3. the absence of manipulation of diet
4. the subjects were weighed at least throughout the last half of pregnancy
5. no medical treatment that might interfere with weight changes, such as diuretics and steroids, should have been given.

Method of Study
All pregnant women, who booked at the ante-natal clinic of the University Hospital before the 20th week of pregnancy, were incorporated into this study. Most of the women were weighed monthly until the 32nd week, thence every two weeks until the 36th week, and thereafter weekly till delivery. Accurate lever balance type of weighing was used, and patients were weighed with a fixed set of clothing, (weighing 0.2 kgm.).

The weighing machine is a SECA lever balance type, weighing correctly to one-tenth of a kilogram.
WEIGHT CHANGES IN PREGNANCY

The machine is periodically checked by one of the authors, usually at 2-weekly intervals.

Results of Study

The present report constitutes a preliminary communication of an extensive study project, still in progress. This study presents the pattern of weight changes in the first 300 subjects, who have been comprehensively documented.

Fig. 1 — Ethnic pattern

Ethnic Pattern

In all, there were 300 subjects studied, and of these, 186 were Chinese, 60 Malays and 54 Indians. Subjects of other ethnic groups were deliberately left out of this study.

Fig. 2 — Maternal age pattern

Maternal Age Pattern

There were 32 subjects in the age group 15–19 (Chinese 12, Malays 10, Indians 10), 181 in the 20–29 age group (Chinese 103, Malays 42, Indians 36), and 87 in the 30–39 age group (Chinese 71, Malays 8, Indians 8).

Fig. 3 — Parity pattern

Parity Pattern

There were 128 primigravida (Chinese 75, Malays 30, Indians 23) and 172 multiparae (Chinese 111, Malays 30, Indians 31).

Fig. 4 — Pregnancy weight gain pattern

Table I — Average Weights During Antenatal Period

<table>
<thead>
<tr>
<th>DURATION OF PREGNANCY</th>
<th>NUMBER OF READINGS</th>
<th>AVERAGE WEIGHT (Kilos.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>22</td>
<td>47.3</td>
</tr>
<tr>
<td>12</td>
<td>64</td>
<td>48.2</td>
</tr>
<tr>
<td>16</td>
<td>161</td>
<td>49.1</td>
</tr>
<tr>
<td>20</td>
<td>300</td>
<td>50.5</td>
</tr>
<tr>
<td>24</td>
<td>263</td>
<td>52.4</td>
</tr>
<tr>
<td>28</td>
<td>224</td>
<td>53.7</td>
</tr>
<tr>
<td>32</td>
<td>215</td>
<td>55.1</td>
</tr>
<tr>
<td>36</td>
<td>232</td>
<td>56.3</td>
</tr>
<tr>
<td>40</td>
<td>185</td>
<td>57.1</td>
</tr>
<tr>
<td>42</td>
<td>24</td>
<td>57.6</td>
</tr>
</tbody>
</table>

Average Antenatal Weight Pattern

Table I shows the results of the average weights of the 300 women incorporated into this study, during the various periods of gestation. Not all these subjects came up for ante-natal visits at all periods of gestation, and the average weights shown are the average weights for different numbers of subjects. For example, there were 300 readings at the 20th week of gestation, 263 readings at the 24th week of gestation, and 185 readings at the 40th week of gestation.
### Table II - Comparative Study of the Pattern of Weight Gain in Pregnancy

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Place</th>
<th>Subject</th>
<th>Parity</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kuo</td>
<td>1941</td>
<td>China</td>
<td>200 normal out of 4175 for complete records</td>
<td>PRIM.</td>
<td>101</td>
</tr>
<tr>
<td>Robinson et al</td>
<td>1943</td>
<td>U.S.A.</td>
<td>Normal Private &quot;Well Situated Economically&quot;</td>
<td>PRIM.</td>
<td>300</td>
</tr>
<tr>
<td>Scott &amp; Benjamin</td>
<td>1948</td>
<td>U.K.</td>
<td>Unselected (in London)</td>
<td>PRIM.</td>
<td>360</td>
</tr>
<tr>
<td>Thomson &amp; Billewicz</td>
<td>1957</td>
<td>U.K.</td>
<td>Hospital Patients with Normal B.P.</td>
<td>PRIM.</td>
<td>2868</td>
</tr>
<tr>
<td>Venkatachalam et al</td>
<td>1960</td>
<td>India</td>
<td>Poor Class Women of Tea Plantation</td>
<td>PRIM.</td>
<td>13</td>
</tr>
<tr>
<td>H.M. Hauck</td>
<td>1960</td>
<td>Nigeria</td>
<td>Normal</td>
<td>PRIM.</td>
<td>31</td>
</tr>
<tr>
<td>Sinnathuray &amp; Wong</td>
<td>1970</td>
<td>Malaysia</td>
<td>Normal</td>
<td>PRIM.</td>
<td>128</td>
</tr>
</tbody>
</table>

### Manipulation of Diet

<table>
<thead>
<tr>
<th>Weight Gain (Calculated from date:--)</th>
<th>First Weighed</th>
<th>Total Gain</th>
<th>Range</th>
<th>Net Gain Post-Partum</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>None</strong></td>
<td>&quot;Non-Pregnant Weight&quot;</td>
<td>FIRST 3 MONTHS</td>
<td>23.39</td>
<td>21.29</td>
</tr>
<tr>
<td><strong>Over Weight</strong></td>
<td>&quot;Usual Weight&quot;</td>
<td>3-4 mths.</td>
<td>24.3</td>
<td>6 weeks + 2.0 lb.</td>
</tr>
<tr>
<td><strong>Under Weight</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- restricted diet</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- encouraged to eat</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>War Time Rationing</strong></td>
<td>16-20 wks.</td>
<td>16-20 wks.</td>
<td>21.53</td>
<td></td>
</tr>
<tr>
<td><strong>None</strong></td>
<td>13 wks.</td>
<td>13 wks.</td>
<td>25.1</td>
<td>2 weeks + 6.05 lb.</td>
</tr>
<tr>
<td><strong>None but Poor Diet</strong></td>
<td>12 wks.</td>
<td>12 wks.</td>
<td>11.8</td>
<td>S.E. 1.67</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>13.6</td>
<td>S.E. 0.96</td>
</tr>
<tr>
<td><strong>None</strong></td>
<td>AVERAGE</td>
<td>From 3 months</td>
<td>6.6</td>
<td></td>
</tr>
<tr>
<td><strong>None</strong></td>
<td>20 wks.</td>
<td>8.20 wks.</td>
<td>17-14</td>
<td>4.4-30.8 + 10.8 lb.</td>
</tr>
</tbody>
</table>
Pregnancy Weight Gain Pattern

From the above results, it has been possible to plot a graph of the pattern of weight gain in pregnancy. The graph from the 20th to the 40th weeks of gestation are in bold lines, as they present the mean weights for a significant number of subjects. The rest of the graph is dotted, and present results from a lesser number of subjects. We have extrapolated the graph to evaluate the average pre-pregnant weight of the Malaysian women. The average pre-pregnant weight so assessed is 46.4 kgm. (102 lbs.). This is close to the average pre-pregnant weight of 202 young, nulliparous women, evaluated in a study by WONG and KUAH, which was 44.9 kgm. (98.7 lbs.).

From the above graph and the other results, the following conclusions can be drawn for the Malaysian pregnant women:

1. The weight gain from the 20th week to the 40th week of gestation was 7.33 kgm. (16.12 lbs.).
2. The weight gain from pre-pregnant weight to the 40th week of gestation was 11.32 kgm. (24.9 lbs.).
3. The weight gain from pre-pregnant weight to the 40th week of gestation for primigravida was 11.8 kgm. (25.9 lbs.).
   This is closely comparable to the average weight gain of 12.5 kgm. (27.5 lbs.) by 2,868 Scottish primigravida as shown in the study by Thomson and Billewicz (1957).
4. Primigravida gained about 0.65 kgm. (about 1½ lbs.) more than multiparous.
5. At end of puerperium, the Malaysian women had gained 4.91 kgm. (10.8 lbs.).

Comparative Studies

Table II shows the pattern of weight gain in pregnancy in various studies in other parts of the world. As one can expect, the greatest weight gain were seen in studies in the United Kingdom and United States, and the least in the studies from Nigeria. Our study has shown that the weight gain in pregnant Malaysian women is of the intermediate variety but closer to the patterns seen in the socio-economically advanced Western countries.

This is most probably a reflection of the general standard of living in this part of Malaysia. But firm conclusions on this point will have to await a more extensive survey, still in progress.

The weight gain at the end of puerperium is 4.91 kgm. (10.8 lbs.), and this figure is considerably more than those obtained in other studies, (Thomson and
Billiewicz 6.05 lbs. at the end of 2 weeks, and ROBINSON et al 2 lb. at the end of 6 weeks). Perhaps, this is because the Malaysian women are not as yet too concerned about their figures after delivery. This is also most probably tied up with the cultural patterns of the Malaysian women with reference to diet, pregnancy and puerperium.

Table III compares the distributional pattern of weight gain in the varied periods of gestation. It can be seen that the greatest gain in weight is in the 21-24 weeks of gestation, 0.45 kgm. per week (approx. 1 lb. per week). Thereafter, the weight gain decreases to about 0.35 kgm. (0.75 lbs.) per week at the 30th week, and 0.18 kgm. (0.4 lb.) per week at term.

![Graph of Mean Weight Gain in Pregnancy Pattern](image)

**Mean Weight Gain Pregnancy Pattern**

Figure 5 shows the distribution of the mean weight gain per week of the pregnant women in this study. Total weight gain by these women is not strictly comparable because duration of the pregnancies ranged from 38 - 42 weeks. In this graph (Figure 5) is shown the average weekly weight gains from the 20th week until delivery. The modal value is about 0.367 kgm. per week (0.81 lb. per week). This figure also shows that the range of total weight gain from the 20th to the 40th week of gestation is very wide, from 2 kgm. (4.4 lbs.) (i.e. 0.1 kgm. x 20 weeks) to 14 kgm. (30 lbs.) (i.e. 0.7 kgm. x 20 weeks). This indicates that it may be necessary to re-consider our present concept of only regarding a weight gain of 1 lb. per week in the later part of pregnancy as the criterion for recognising excessive weight gain of the pregnant mother in the ante-natal period.

**SUMMARY and CONCLUSIONS:**

1. The average maternal weight gain throughout pregnancy in Malaysian women is around 11.5 kgm. (25 lbs.).

2. The pattern of maternal weight gain in the Malaysian women shows a range of variation when the results of the study are comparatively reviewed with reference to the maternal age, gravida, and ethnic group patterns.

3. The average total weight gain in the Malaysian women in this study is about 1 kgm. to 2 kgm. (2 to 4 lbs.) less than the total weight gain, that has been reported in the European and American pregnant women. Whereas, when compared to the studies done in India and Nigeria, the average total weight gain in the Malaysian women is about 5 kgm. to 6 kgm. (11 to 13 lbs.) more than their counterparts in India and Nigeria.

**REFERENCES:**

Uncontrollable bleeding due to Hypofibrinogenemia in a case of acute Myelo-monocytic Leukaemia

by A.K. Banerjee

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MRCP (Edin), MRCP (Glas)
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Kuala Lumpur.

HYPOFIBRINOGENEMIA is a fairly well documented but rare complication of acute leukaemia (Rosenthal et al, 1955; Pisciotta and Schulz 1955; Didisheim et al, 1964). Although acute pro-myelocytic leukaemia has been the usual recognised variety (Hillestad, 1967; Rosenthal 1963; Pittman, 1966) to give rise to this complication, other cytologic varieties have also been found to cause this (Baker et al, 1944; Hirsh et al, 1967). There still exists considerable uncertainty as to the exact mechanism or mechanisms by which a fibrinopenic state may complicate acute leukaemia. Knowledge of such mechanism is very important from the point of view of management as the method of treatment may depend on its nature.

Recently, such a rare complication was encountered in the University Hospital, University of Malaya, and the details of the patient are reported below to illustrate the various diagnostic difficulties and problems in management. This happens to be the only case with such a complication among 35 adult acute leukaemic patients seen and treated in this hospital between May 1968 and April 1970.

Case Report

J.S., a 20-year-old Eurasian bachelor was admitted to the University Hospital on 30th July, 1969, with complaints of swollen, bleeding gums for ten days and a warm, tender, reddish swelling of the right forearms below his elbow for three days. He had never had any similar bleeding tendencies before; however, five days prior to admission, he had sustained a minor cut over his right great toe from which he had bled for nearly 24 hours. There was no associated fever, sore throat, bone pains or easy bruising of the skin. There was nothing significant in his past, family or personal history. He had not been exposed to any radiation, chemicals or drug therapy.

On examination, he was found to be of fairly satis-
factory general condition, although moderately pale and slightly icteric. There were no purpuric spots over his body nor had he any large lymph nodes. Both his upper and lower gums were swollen and haemorrhagic but his teeth were quite healthy. There was no evidence of pharyngitis or tonsillar involvement. He had a slightly warm, tender, fluctuant, oval, red swelling (measuring 8 cm. by 4 cm.) over the lateral aspects of his right upper forearm, and this was without doubt a haematoma. The right elbow joint was not affected. Examination of his cardiovascular, respiratory and nervous systems revealed no abnormality. His optic fundi were normal. On abdominal examination, however, his liver was just palpable but the spleen could not be felt. With such a short history and the physical findings, a provisional diagnosis of an active coagulation abnormality secondary to an acute leukaemic process was made and a number of investigations were carried out.

Investigations
The haematology results are tabulated in Table 1.

<table>
<thead>
<tr>
<th>Days after admission</th>
<th>1</th>
<th>3</th>
<th>4</th>
<th>6</th>
<th>8</th>
<th>10</th>
<th>13</th>
<th>17</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb. (G/100 ml)</td>
<td>7.9</td>
<td>8.6</td>
<td>9.9</td>
<td>8.6</td>
<td>6.3</td>
<td>4.6</td>
<td>7.3</td>
<td>3.3</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>23</td>
<td>26</td>
<td>31</td>
<td>23</td>
<td>20</td>
<td>15</td>
<td>21</td>
<td>9.5</td>
</tr>
<tr>
<td>MCHC (%)</td>
<td>34.4</td>
<td>33.2</td>
<td>31.9</td>
<td>37.4</td>
<td>31.4</td>
<td>30.6</td>
<td>34.8</td>
<td>34.7</td>
</tr>
<tr>
<td>Platelets (× 10³/µl)</td>
<td>12</td>
<td>22</td>
<td>12</td>
<td>6</td>
<td>11</td>
<td>16</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Leucocytes (µl)</td>
<td>1,600</td>
<td>900</td>
<td>1,100</td>
<td>1,500</td>
<td>2,100</td>
<td>1,600</td>
<td>3,800</td>
<td>1,100</td>
</tr>
<tr>
<td>Diff. Leuc. Count (%)</td>
<td>20</td>
<td>22</td>
<td>46</td>
<td>38</td>
<td>41</td>
<td>86</td>
<td>59</td>
<td>20</td>
</tr>
<tr>
<td>Blasts</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Metamyelocytes</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>16</td>
<td>16</td>
<td>40</td>
<td>18</td>
<td>12</td>
<td>6</td>
<td>9</td>
<td>30</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>62</td>
<td>50</td>
<td>14</td>
<td>44</td>
<td>47</td>
<td>8</td>
<td>32</td>
<td>50</td>
</tr>
<tr>
<td>Nucleated R.B.C.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Degenerate Cells</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Reticulocytes (%)</td>
<td>0.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total Bilirubin 2.3 mg/100 ml. Conjugated 0.2 mg/100 ml.
Unconjugated 2.1 mg/100 ml. S.G.O.T. 9 I.U./Litre.

Direct and Indirect Coombs Test negative. Bone Marrow Examination: Marrow was aspirated readily from the sternal manubrium. The films contained small grossly hypercellular particles and trails. Primitive cells predominated and were mostly blast cells with highly irregular nuclear and cytoplasmic structure. Although highly irregular and often having a monocytoid structure, the cells were almost all strongly peroxidase positive. They also showed weak, diffuse PAS reaction in the cytoplasm. Azurophil granules and Auer bodies occurred in many of the primitive cells. Myelocytes and metamyelocytes (including eosinophil) were present, but mature polymorphs were very scanty. The alkaline phosphatase score of the mature polymorphs was rather low (42), with predominance of low-scoring cells (score 0 = 65%, score 1 = 30%, score 2 = 3%). Erythropoiesis was relatively inconspicuous and showed macro-normoblastic and some megaloblastiform features.

Megakaryocytes were very rare. Occasional lymphocytes and plasma cells were seen as well as abnormal haemocto-blast-type reticulum cells. Iron was moderately abundant in the stores. This was an acute leukaemia of the myeloid series. There was very considerable structural variation, so that this most closely conformed to the Naegeli-type para-myeloblastic leukaemia or myelo-monocytic leukaemia. Serum Iron 224 µg/100 ml. Unsaturated Iron Binding
BLEEDING DUE TO HYPOFIBRINOGENAEMIA

Table 2. Results of Coagulation Tests

<table>
<thead>
<tr>
<th>Days After Hospital Admission</th>
<th>1</th>
<th>6</th>
<th>10</th>
<th>11</th>
<th>13</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Bleeding Time (mins)</td>
<td>&gt; 18 &lt; 30</td>
<td></td>
<td></td>
<td>0-10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Clotting Time (mins)</td>
<td>&gt; 21 &gt; 30</td>
<td></td>
<td></td>
<td>5-11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. One-Stage Prothrombin Time</td>
<td>29</td>
<td>26</td>
<td>18</td>
<td>14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Prothrombin Activity (%)</td>
<td>20</td>
<td>40</td>
<td>50</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Thrombotest (%)</td>
<td>6-10</td>
<td></td>
<td></td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Tests for overactive fibrinolysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) Clot Lysis Time (Hrs)</td>
<td>Strongly Positive</td>
<td>100</td>
<td>100</td>
<td>No Lysis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(b) Thrombin Titre Test</td>
<td>120</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Fibrinogen (mg/100 ml)</td>
<td>17</td>
<td>40</td>
<td>42</td>
<td>150-400</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Thromboplastin Generation Test</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Circulating Anticoagulants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Capacity 141 ng/100 ml. Total Iron Binding Capacity 365 ug/100 ml. Folic Acid 6.8 ug/ml. B12 3782 pg/ml. Unsaturated B12 Binding Capacity 4600 pg/ml. Bleeding Time more than 18 minutes. Clotting Time more than 21 minutes.


The results of other investigations are given in Table 2, which includes the results of the various coagulation studies. All these tests were carried out by standard methods as recommended by Dacie. Plasma fibrinogen levels were estimated by turbidimetric method.

The diagnosis of acute myelomonocytic leukaemia was therefore confirmed by these laboratory tests, and there was no doubt that this patient had a rather serious coagulation disorder arising from his leukaemia.

The patient was transfused with 1.5 litres of fresh whole blood (F.W.B.) on the second day of his admission (Fig. 1) and the gum bleeding seemed to be slightly less the day after although he started developing spontaneous bruises all over his body. He was started on Prednisolone (1 mg/Kg.) 15 mg, four times a day and 6 Mercaptopurine (2.5 mg/Kg) 50 mg, three times a day, both orally, on the third day. Two more units of fresh blood of 500 ml each were administered the same evening. His gum bleeding was slightly less. However, it continued to ooze for the next 48 to 72 hours, at the end of which, on the 6th day, he was given another unit (500 ml.) of fresh whole blood. At this stage, it was confirmed that his plasma fibrinogen was low and the next day he was given two units of fresh frozen plasma (F.F.P.) and six units of fibrinogen of 2 grams each intravenously over six hours. Although slightly less, the bleeding,
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Table 3. Thromboplastin Generation Test to Screen the Plasma Coagulation Factors Activity.

<table>
<thead>
<tr>
<th>Reagents</th>
<th>Incubation Time at 37°C (mins)</th>
<th>Clotting Time in Secs. of the Substrate Plasma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Normal Plasma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal Serum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lipoid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient’s Plasma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal Serum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lipoid</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The second T.G.T. also showed identical results.

However, continued and he was given two units of platelet-rich plasma (P.R.P.) the day after as his platelet count was extremely low. On further evidence to treat him very cautiously with a modest amount of E.A.C.A. and he received 12 grams of E.A.C.A. (Amicar) intravenously over a 12-hour period on the ninth day after admission. This was followed by the administration of three units of F.F.P. and one litre of fresh whole blood, and his bleeding improved to a considerable extent. On the eleventh day, he was given another eight grams of fibrinogen over four hours and this was followed by two further units of fresh whole blood. His clinical condition remained unchanged but unfortunately it started deteriorating again two days later. He continued to bleed from his gums and fresh crops of purpura appeared on his body. Two units of P.R.P. were administered on the same afternoon. There was no great change and he received two more units of F.F.P. the following day. Unfortunately, no more fibrinogen could be made available, and as he was not responding to any form of treatment at all, the outlook seemed absolutely hopeless. His fibrinogen level continued to drop and his peripheral platelet count was only 7000/ml. Despite almost continuous fresh blood transfusion over the 16th, and 17th post-admission days, he had a fairly large intra-cranial haemorrhage as evidenced by extensive fresh bilateral haemorrhages in his vitreous and fundi on the evening of the 18th day. He vomited out some blood and gradually sank into coma. Bouts of melaena and passage of blood-stained urine followed. Oozing from nose and gums continued and he ultimately expired in his sleep on the 17th of August, 19 days after admission to hospital. No post-mortem examination could be carried out.

Discussion

In 1955, Rosenthal and his colleagues reported the development of hypofibrinogenemia in seven patients with acute promyelocytic leukaemia (Rosenthal et al., 1955). Later in 1963, he published his observations on more detailed studies of 17 patients with similar illness whom he had seen over the previous nine years. Almost all his patients had a very fulminating short illness, poor response to therapy, and were dead within a few weeks due to massive intracranial or gastro-intestinal haemorrhage (Rosenthal, 1963). Since then, this complication has been noted by various other workers interested in this field, and cytologic types other than the “acute promyelocytic” variety have also been reported to have this complication (Hirsh et al, 1967).

Current opinion views the fibrinogenopenia in acute leukaemia as resulting from:


(b) Accelerated digestion destructive of fibrinogen as a result of: (i) primary overactive fibrinolytic activity (Cooperberg and Neiman, 1955; Piso-
BLEEDING DUE TO HYPOFIBRINOGENAEMIA

ciotta and Schulz, 1965; Fisher et al, 1960; Lee, 1962) or (ii) increased fibrinolysis secondary to coagulopathy (Fletcher, 1962), or
(c) Failure of fibrinogen production in the liver.
In many instances, it is not possible to determine which of these processes are responsible for the bleeding or whether they are all involved at the same time (Wintrobe, 1967; Fearnley, 1969).

There is no doubt that the severe, uncontrollable bleeding in this patient was mainly due to hypofibrinogenemia secondary to a fulminating acute leukaemia, although the associated thrombocytopenia must have contributed to it.

Fibrinogen deficiency due to hepatic involvement is a rare condition and it has been reported in cases of severe liver disease such as acute yellow atrophy (Conley, 1951). In those cases, the degree of parenchymal liver damage is usually quite gross. Although this patient was mildly jaundiced initially, his hepatic function and urinary findings were more suggestive of haemolytic rather than a hepatic jaundice. The transaminase and serum albumin were also normal and interestingly enough his jaundice improved later. Thus it seems unlikely that hepatic dysfunction contributed significantly to the deficiency of fibrinogen.

Thus one is left with the two other possible causes by which this complication might have arisen.

It has been suggested that the coarse granules in the leukaemia cells, probably produce "thromboplastin-like substance" (Pittman et al, 1966) and thus precipitate diffuse intravascular coagulation. A number of factors such as fibrinogen, prothrombin, factors V and VIII are 'consumed' fairly rapidly by the numerous micro-thrombi formed and a considerable number of circulating platelets is also absorbed to make the situation worse. This is the so-called consumptive-coagulopathy or defibrination syndrome.

On the other hand, a fibrinopenia may occur as a result of excessive activation of the plasminogen plasmin system and accelerated fibrinolysis. It is also possible that both are co-existent, the coagulative process giving rise to secondary fibrinolytic activity (Rodriguez - Erdman, 1965).

In the present case, it was extremely difficult to determine whether the fibrinogen deficiency was primarily coagulative or fibrinolytic. Since the fibrinolytic process was definitely over-active and there was no evidence of any gross reduction in plasma factors V and VIII (see the results of T.G.T. in Table 3), one would tend to think that the process was basically fibrinolytic. However, prothrombin and coagulation factors like V and VIII can also be reduced by excessive digestion by fibrinolysins, (Fearnley, 1969). A low plasma plasminogen level would have provided more concrete evidence in favour of a primary fibrinolysis (Flute, 1964), had this been estimated.

A prolonged one-stage prothrombin time, not necessarily reflecting a hypoprothrombinaemic state, is quite characteristic in any situation with fibrinogen deficiency. On the other hand, due to reasons given already, a true prothrombin lack may occur in these conditions.

The prognosis in these cases of acute leukaemia is extremely gloomy as response to any form of therapy is usually negligible (Rosenthal, 1963; McNicol and Douglas 1964). However, the use of anti-fibrinolytic agents, such as epsilon-amino caproic acid (E.A.C.A.), has been known to be of value in cases with excessive fibrinolysis (Nilsson et al, 1966) or, on the other hand, Heparin may be useful in the control of bleeding secondary to diffuse and intravascular thrombosis (Verstraete et al, 1963; von Francken et al, 1963).

In any case, these patients must receive ample quantities of fibrinogen, fresh frozen plasma, fresh whole blood and platelet concentrates in addition to their anti-leukaemia drugs. This patient was given a small amount of E.A.C.A. and that therapy was discontinued because the evidence in favour of his having a primary fibrinolytic process was not very convincing and there would also be a theoretical risk of precipitating further thrombosis with E.A.C.A. (Rachmilewitz, 1967). Unfortunately, all other measures taken did not help him very much and he expired even before his anti-leukaemia therapy could become adequately effective.

Summary

Fatal bleeding due to fibrinogen deficiency and overactive fibrinolysis as a complication in a case of acute myelo-monocytic leukaemia is described. The pathogenesis of this complication, especially in relation to this patient, is briefly discussed.

Acknowledgement

I am grateful to Dr. M. Somasundaram for his kind permission to publish this case. I am also deeply indebted to Dr. J.C. White for his excellent reports on the bone marrow and the peripheral blood films, to Mr. G.L. Chan for his kind help in various coagulation studies and to Mr. G. Rajendran for arranging the continuous supply of various transfusion materials and fibrinogen, etc.
References
Some aspects of refractive errors in West Malaysia

by S. Chandran
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University of Malaya.

Introduction and Method
THIS PAPER IS an analysis of a survey of 500 cases of refraction conducted by the authors at the Eye Clinic of the University Hospital, University of Malaya, during a 2½-year period (Sept. 1967 to Feb. 1970). The age distribution in relation to spherical error; astigmatism and anisometropia would be analysed and the role of contact lens in these errors discussed. The age varied from 5 to 65. The data for the very young and beyond 65 are less valid because of the small number and often may be associated with pathological condition and hence excluded from this study. The vision had to be improved to at least 6/9 in each eye to be included in the series. This is a study of hospital figures and does not claim to be representative of unselected population. With the problem of anisometropia, each eye is treated individually – hence 500 cases would give 1,000 eyes for the analysis.

Refractive errors were determined by retinoscopy. Cycloplegia was routinely used in children under the age of 10, in moderate hypermetropia and in difficult cases of refraction. Cross cylinder was used in subjective confirmation of astigmatism.

Results
Table I shows the age distribution in relation to refraction in the series as a whole – spherical refraction with half the astigmatic error added. The average mean error is calculated by algebraically adding the total error for each age group, divided by the number of eyes. Fig. A shows the distribution of cases in relation to age and Fig. B the average mean refraction in relation to age.

Table II shows the distribution of astigmatism – greater than 0.25D in relation to spherical error and Fig. C shows this graphically. Table III shows the distribution of astigmatism in percentage – 53% have no astigmatism or one less than 0.2D. Table IV shows the distribution of the axes of astigmatism for every 15 degrees and any stray was grouped with the nearest figure.

Table V shows the distribution of anisometropia – 152 cases ranging from 0.5 to 10.0D of which 0.5 to 1.0 makes up 93 cases.

Discussion
Age Distribution in relation to spherical errors. Table I and Fig. A show 11 – 25 age group predominates, with a peak between 16 – 20 (23.9%). This is not only because of schooling and employment that this group is refracted more often than others but because myopia has its greatest incidence and development during school years, it is also the period of growth.

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**TABLE I**

<table>
<thead>
<tr>
<th>Age Distribution in relation to Refraction (Series as a whole)</th>
</tr>
</thead>
<tbody>
<tr>
<td>---------------------</td>
</tr>
<tr>
<td>Greater than</td>
</tr>
<tr>
<td>-10.1</td>
</tr>
<tr>
<td>-8.1 to -9.0</td>
</tr>
<tr>
<td>-7.1 to -8.0</td>
</tr>
<tr>
<td>-6.1 to -7.0</td>
</tr>
<tr>
<td>-5.1 to -6.0</td>
</tr>
<tr>
<td>-4.1 to -5.0</td>
</tr>
<tr>
<td>-3.1 to -4.0</td>
</tr>
<tr>
<td>-2.1 to -3.0</td>
</tr>
<tr>
<td>-1.1 to -2.0</td>
</tr>
<tr>
<td>0.0 to +0.9</td>
</tr>
<tr>
<td>+1.0 to +1.9</td>
</tr>
<tr>
<td>+2.0 to +2.9</td>
</tr>
<tr>
<td>+3.0 to +3.3</td>
</tr>
<tr>
<td>+4.0 to +4.9</td>
</tr>
<tr>
<td><strong>Total eyes</strong></td>
</tr>
<tr>
<td><strong>Average Error</strong></td>
</tr>
</tbody>
</table>

The development of 1.0D of myopia would attract attention while a similar change in hypermetropia would pass unnoticed and this may partly contribute to the myopia excess in this series. European studies (Hirsh) show that the incidence of myopia increases markedly from 7 years to 18–20 and stabilises about 21 (Duke Elder). In this series (Fig. B) myopia commences at 5 (probably earlier) and continues to progress and reach a peak of -2.40D between 21–25 and then stabilises. This stabilisation is relatively delayed (by 5 years) when compared with the Caucasians. This is followed by a gradual shift towards emmetropia by the age of 45 and into low hypermetropia as the 50’s are approached (+0.86D).

In the Caucasian, there is an increase in the mean refractive state towards greater hypermetropia between 40–60 (Hirsh).

The ametropias of up to +4.0D show essentially the same range of values as the emmetropic eye, so that the error present is not mainly the result of any unusual values, but a failure of correlation, especially between axial length and corneal power. Sorsby et al (1957) state that the eye, with an ocular refraction in excess of ±4D, fall outside the range of eyes with component that are observed in the emmetropic eye.
REFRACTIVE ERRORS IN WEST MALAYSIA

TABLE II
Astigmatism of 0.25D or more in relation to Spherical Refraction

<table>
<thead>
<tr>
<th>Spherical Refraction in the least ametropic meridian</th>
<th>Degree of Astigmatism (d)</th>
<th>Total Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.25-0.5</td>
<td>0.6-1</td>
<td>1.1-2</td>
</tr>
<tr>
<td>9.1 to -10</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>8.1 to -9.0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>7.1 to -8.0</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>6.1 to -7.0</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>5.1 to -6.0</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>4.1 to -5.0</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>3.1 to 4.0</td>
<td>22</td>
<td>11</td>
</tr>
<tr>
<td>2.1 to 3.0</td>
<td>43</td>
<td>12</td>
</tr>
<tr>
<td>-0.1 to -1.0</td>
<td>85</td>
<td>48</td>
</tr>
<tr>
<td>0.0 to -0.9</td>
<td>48</td>
<td>26</td>
</tr>
<tr>
<td>+1.0 to 1.9</td>
<td>13</td>
<td>7</td>
</tr>
<tr>
<td>+2.0 to 2.9</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>+3.0 to 3.9</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>+4.0 to 4.9</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>TOTAL</td>
<td>254</td>
<td>130</td>
</tr>
</tbody>
</table>

TABLE III
Distribution of Astigmatism in Percentage

<table>
<thead>
<tr>
<th>Degree of Astigmatism (d)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0 or less than 0.25</td>
<td>53.8</td>
</tr>
<tr>
<td>0.3 to 0.5</td>
<td>25.4</td>
</tr>
<tr>
<td>0.6 to 1.0</td>
<td>13.0</td>
</tr>
<tr>
<td>1.1 to 2.0</td>
<td>6.3</td>
</tr>
<tr>
<td>2.1 to 3.0</td>
<td>1.7</td>
</tr>
<tr>
<td>3.1 to 4.0</td>
<td>0.4</td>
</tr>
<tr>
<td>TOTAL</td>
<td>100.0</td>
</tr>
</tbody>
</table>

and as such may perhaps be regarded as malformation of a pathological nature. In this series 17.8% of myopia (Table I) fall into the so-called pathological group. If the above criteria is to be used for the Malaysians, it would have to be modified with a myopia bias — probably —6.0D. Then the figure would be 7.9% which compares with 6.3% of Sorsby's 1960 series.

**Astigmatism**

**Relationship to Spherical Errors**

Table II, Fig. C show that most astigmatic errors are associated with spherical errors of a low order; +0.9 to -3.0 accounts for 77.0% with a peak between -0.1 to -1.0D (34.2%). Beyond this range, there is no correlation between these two errors. Table III shows 46.8% of cases have some degree of astigmatism. 0.25 - 0.5D accounts for 25.4% and errors greater than 0.6D for 21.4%. Most of the astigmatic errors of the low order (mainly less than 1D) unlike similar spherical errors often require correction — slight blurs are overcome by rapidly changing from one focus to another, thereby getting a composite picture utilising the clear component of each axes, this is fatiguing, it requires constant effort of accom-
moderation. This is so when engaged in work requiring precise vision.

Areas of Astigmatism (Table IV)
The direct (with the rule) astigmatism is corrected by axes 180 ± 20 degrees accounts for 230, the indirect (against the rule) by axes 90 ± 20 degrees accounts for 163 and the oblique for 75.

For the same degree of error, the direct produces less blurring than indirect but the former accepts a full correction poorly but the latter accepts well (Sloane). As the direct produces less blurring, patients may delay seeking visual care.

Anisometropia (Table V)
0.25D of difference causes a 0.5% difference in size of retinal image, a difference of 5% in the limit of tolerance in most cases (Whittington). Correction of 2.5D of anisometropia will render such patient monocular with glasses. There are 28 cases over 2.5D in this series. Since anisometropia is a predisposing factor towards amblyopia, strabismus may develop in the young (Sloane), hence an attempt is made to correct this. Corneal contact lens was advised for full correction to give best vision in each eye with binocular singular vision to meet the visual requirement of industry and different service fields. If the patient was unable to afford or tolerate contact lens, a compromise was resorted to by undercorrecting the more ametropic eye to achieve some degree of binocular vision.

Practical Aspects
In myopia, the concave lens is separated from the eye (+ ve) by air which creates an inverted Galilean telescope which minifies the image and this effect can be weakened by reducing the distance separating the -ve and +ve elements. A contact lens accomplishes this and gives the patient unaberrated peripheral vision, in addition to the larger retinal image. There is 8.7% of cases in this series over -6.0D who are ideal for contact lens. Contact lens for lesser errors are worn mainly for cosmetic purposes. A myope tolerates contact lens better than a hypermetrope as the images are minimised in the latter with contact lens.

Astigmatism can also be neutralised with contact lens with a better optical result. Errors greater than 3D causes the lens to 'rock back and forth' when fitted to the flatter corneal curve and produce blurring of vision (Gettes.) In this series, such error accounts for only 0.4%, therefore contact lens fitting poses no problem. From the myopic (over -6.0D),
astigmatic and anisometric point of view contact lens gives good optical results. Fitting techniques are constantly being improved and with more people being encouraged to use, the ultimate cost of fitting would be reduced.

SUMMARY
Some aspects of refractive errors on 500 cases (1,000 eyes) from the University Hospital are analysed. Age distribution in relation to the spherical error; astigmatism, anisometropia and the role of contact lens in these errors are discussed. The 11 – 25 age group predominate with a peak between 16 – 20; partly because myopia coincides with this period of growth and partly the development of myopia would attract more attention than a similar hypermetropia in the schooling age group. This series show a myopic excess. In the Malaysian eye, myopia commences as early as 5 years (Caucasian 7 years) and stabilises around 25 (21 for Caucasian), with a peak average mean error of −2.40D. Sorsby et. al. (1957) view that refraction in excess of +4.0D be regarded as malformation of a pathological nature would have to be modified with a myopic bias probably −6.0D for the Malaysian eye, then the figure would be 7.9% which compares with 6.3% of Sorsby’s 1960 series. Most of the astigmatic errors are associated with spherical errors of a low order +0.9 to −3.0D which accounts for 77.0%. The direct astigmatism accounts for 230, indirect for 163 and oblique for 75. Most of these low astigmatism, unlike similar spherical errors, require correction for constant precise vision. There were 152 cases of anisometropia of which 28 had a difference of over 2.5D. As this difference may render persons monocular and predispose to amblyopia, attempt is made to correct this with contact lens. The optical merits of contact lens for myopia over −6.0D (8.7%), astigmatism and anisometropia are discussed.

Acknowledgements
We wish to thank the Medical Records Department of this Hospital for helping to trace these case notes and Miss Yap for her assistance in the preparation of this paper.

References
1. Duke Elder, S. The Practice of Refraction, Pages 88 – 90.
2. Gettes B.C. Refraction, Page 51, Pages 56 to 57.
The role of Hypotonic Duodenography in the diagnosis of inflammatory and neoplastic lesions of the head of the Pancreas

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Introduction
THE PANCREAS is supposed to be, more or less, the tomb of radiologists. There are many direct and indirect roentgen methods for the diagnosis of pancreatic disease. These special techniques include the conventional barium meal examination, hypotonic duodenography, trans-hepatic percutaneous cholangiography, pancreatography through retro-peritoneal gas insufflation, transverse tomography, arteriography, splanchnography, venography, operative pancreatography and Magna Scan with seleno-methionine.

The large number and variety of techniques indicate that no single ideal method exists.

Among the various techniques carried out at present, hypotonic duodenography offers the unique feature of being a procedure which requires no special equipment.

Hypotonic duodenography is a method for studying the duodenum and other adjacent structures such as the region of the Ampulla of Vater, the lymph nodes draining the common bile duct and the pancreas, and most important, the head of the pancreas.

The information derived from this method is also of great help in the interpretation of pancreatic scans.

The value of hypotonic duodenography in the evaluation of diseases of the head of the pancreas is inherent to the close anatomical proximity of the two organs.

The pancreatic head, flattened from front to back, is lodged within the curve of the first three parts of the duodenum. In the region of the proximal portion of the second part of the duodenum, their anatomical relationship is extremely close but is less intimate further down. The neck of the pancreas can be explored to a certain extent by this method, but not the body and tail of the pancreas, unless the enlargement of these portions are remarkable.

Hypotonic duodenography produces a relaxed duodenum which is distended two to three times its normal size. The walls are pressed against adjacent organs, mirroring their contours. Its mucosal surface is stretched into a well-defined pattern and any small but important abnormality may be revealed.
Hypotonic duodenography was first introduced by Liotta in 1955 and refined by Mallet Guy and Jacquesmet in 1963.

Equipment and Technique
The procedure is first explained to the patient and his co-operation elicited. The posterior aspect of the oro-pharynx is anaesthesised by spraying with 2% xylocaine. The walls of a Salem sump tube, which is a double-lumen thin-walled polyethylene tube with an internal flexible guide wire, are well lubricated with xylocaine gel and passed through the mouth down to the stomach. Under fluoroscopic control and with the patient lying in the right lateral decubitus position, the tube is advanced into the duodenum and placed in the middle of the second part of the duodenum. In some cases, the patient has to be positioned in the right lateral decubitus, right anterior oblique or supine positions, when there is difficulty in passing the tip of the Salem tube into the duodenum.

After the injection, the metallic guide wire is withdrawn and, with the patient in the right lateral decubitus position, about 30 c.c. of viscous xylocaine 20% are injected into the duodenum through the Salem tube. Hypotonia of the duodenum is generally achieved after a few minutes. Then, with the patient in the same position, about 50 c.c. of barium (micropaque or barosperse) are slowly introduced into the duodenum under fluoroscopic control, and serigraphic studies of the loop are taken in various positions, i.e. supine, right anterior oblique, left anterior oblique and right lateral positions. The purpose of various positions is to allow the radiologist to explore all the duodenal walls from different angles, especially as far as the second part of the duodenum is concerned.

Finally, an air contrast study of the duodenum is performed under fluoroscopic control by insufflation of oxygen through the Salem tube. Serial studies are then taken again.

Discussion
In this paper, we shall consider the radiological signs which are found, on hypotonic duodenography, in inflammatory and neoplastic lesions of the head of the pancreas. The diagnosis of a pancreatic lesion should be based on not only the radiological picture but also on the clinical and laboratory findings. Only by a correlation of all the findings, can the clinician reach the final diagnosis after a comprehensive differential diagnosis.

The normal appearance of the hypotonic duodenal loop is that which shows:— (Fig. 1)
1. Smooth valvulae conniventes with uniform thickness of only a few millimeters each.
2. Good distensibility of the loop and no widening of the same.
3. Symmetry between the medial and lateral aspects of the loop.
Typical case of changes in pancreatitis. Note the signs of irritability of the mucosa, some levelling of the medial wall of the loop, thickening of the valvulae conniventes and swelling of the mucosa folds over the medial and lateral aspect of the loop.

4. No flattening or filling defects of the walls.
5. Possible visualization of the Ampulla of Vater as a small notch over the medial aspect of the second or third portion of the loop.

The radiological signs that point to a diagnosis of an inflammatory lesion of the pancreas are:— (Fig. 2a, 2b & 2c)

1. Thickening of the mucosa with swelling of the mucosal folds medially and often laterally.
HYPOTONIC DUODENOGRAPHY IN DIAGNOSIS

Case of carcinoma of head of the pancreas. The clinical symptoms and finds were in favour of an inflammatory lesion. Hypotonic duodenography instead showed a lobulated filling defect over a rigid medial wall of the loop with spicules formation only over the medial aspect of the duodenal wall and disorganisation of the mucosa relief at the same level. Some widening of the loop is also noted.

2. Signs of irritability of the mucosa.
3. Spicules formation over the medial and often over the lateral aspect of the duodenal wall.
4. Some leveling of the medial wall of the loop.
5. No destruction of the mucosa.
6. Small localised rigidity on the medial duodenal wall.

The radiological signs that are or are almost characteristic of a neoplastic lesion of the head of the pancreas are:— (Fig. 3a, 3b, 3c, 4a, 4b & 4c)
Case of Carcinoma of the Head of the Pancreas

Note flattening of the medial wall of the 2nd part of the duodenal loop with lack of mucosal folds at the same level. Some spicule formation over the medial aspect of the loop. Imprint over the duodenal cap.

A right lateral decubitus projection. Note irregular narrowing over the proximal aspect of the 2nd portion of the duodenal loop and filling defect over the anterior aspect of the loop.
HYPOTONIC DUODENOGRAPHY IN DIAGNOSIS

1. Constant rigidity of the duodenal wall medially.
2. No edema of the mucosa (unless the tumor is associated with inflammatory changes).
3. Destruction of the duodenal mucosa.
4. Irregular filling defect or defects of the loop, especially over the medial aspect.
5. Shortening of the folds.
6. Presence of ulcerations within the filling defect of the loop.
7. Rigid spiking of the medial aspect of the loop, especially when associated with adjacent irregular, lobulated filling defect.

It has been found that an examination with double contrast, after a moderate quantity of barium injection, constitutes the best technique since it permits a most accurate evaluation of the mucosa relief and makes the comparison of the medial and lateral wall of the duodenum much easier. Any discontinuity of the mucosa is best seen after the introduction of air or oxygen. It is best to study the duodenum in all possible projections, including the oblique and right lateral decubitus, in order to obtain complete visualisation.

Usually, numerous spot serigraphic films are taken, to help better in the evaluation of questionable rigidity of the duodenal wall. Not always is the differential radiological diagnosis between inflammatory and neoplastic lesions easy, especially when there is a combination of both. However, we are of the opinion that the presence of spicules on both the medial and lateral walls is characteristic of an inflammatory lesion. The evaluation of the minor mucosal changes of the duodenum is indeed not easy. It’s only with practice and careful examination of the films that a certain confidence of opinion can be obtained.

Summary

Hypotonic duodenography is a good and simple radiological tool for the diagnosis of inflammatory or neoplastic lesions of the head of the pancreas. The examination can also be carried out on out-patients and requires the minimum of instruments. It only causes a slight discomfort to the patient when the catheter is being introduced into the stomach and duodenum.

Some useful radiological signs for the differential diagnosis between inflammatory and neoplastic lesions of the head of the pancreas are described. The technique used is set forth, underlining the usefulness of the double contrast method for a better visualisa-
tion of minor changes which may lead to a better final diagnosis.

Acknowledgements

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REFERENCES

An appraisal of El Tor carrier state in Kelantan

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Introduction

SINCE THE CHOLERA OUTBREAK in which 50 confirmed El Tor cases and 20 contacts were recorded in Kelantan in 1964, not a single case of cholera occurred until 1969 when another outbreak was recognised during the period between April and August 1969, involving 80 confirmed El Tor cases and 55 contacts. From September, 1969 to November, 1970, there was no incidence of cholera in Kelantan.

A series of El Tor cholera outbreaks occurring in various parts of Malaysia from 1963 to 1970 are reported to be predominantly water-borne (Felsenfeld, 1967; Government of Malaysia, 1963; Leng, 1963 and Paul, 1970). We also observed that water played a predominant role in the transmission of El Tor infection during the outbreak in Kelantan in 1969. It is postulated by some school that outbreak and subsequent spread of cholera are caused by long-term carrier (Khan, 1967 and Wallac et al, 1967), but recently Tamyo et al (1966), Dizon (1965), Mackenzie (1965) and Chuttani et al (1967) have reported short duration of carrier state in their systemic studies in which not a single clinical case could be traced to have occurred due to a carrier. This report supports the current views of the latter school.

Materials and Methods

A total of 17,220 specimens of stool, water and shellfish were examined for V. cholerae. Each sample was first cultured in peptone water (pH 9.4) for six hours and then subcultured in cholera medium (Oxoid) for 24 hours. One litre of each water sample was filtered through milipore filter before culture. Bacterial colonies were examined morphologically and confirmed by serological test using Burrows Welcome sera. Polymyxin sensitivity and chicken red cell agglutination tests were carried out to identify the biotypes of V. cholerae. Stool of confirmed cases of cholera and contacts of both 1964 and 1969 epidemics were repeatedly cultured at variable intervals in order to assess the carrier state in the household and community.

Results

Stool of 55 suspected cases and contacts, 35 samples of water of wells and rivers, and 50 specimens of edible river shellfish collected during 1969 epidemic were found to be vibrio-positive (Table-1). Considerable number of cases and contacts appeared to be infected through drinking polluted water of wells and rivers; the latter seem to be contaminated through...
overhung latrines. An appreciable number of cases and contacts seem to be infected also through eating raw or partially-cooked shellfish containing El Tor vibrio. From 1966 to 1968, not a single case of cholera was seen in Kelantan. In the epidemic period between April and August 1969, ten short-term asymptomatic carriers were found; they excreted vibrios in their stool up to eight days. In the post-epidemic period between September, 1969 and November, 1970, neither a single case of cholera nor a carrier was demonstrated among 5,500 random cases of acute gastroenteritis and healthy persons (Table 2).

Discussion
Most of the confirmed cases and contacts of cholera of both 1964 and 1969 epidemics were seen in villages dotted along the banks of the tributaries of the Kelantan River near its mouth into the sea. They seemed to contact infection through drinking water from polluted wells and rivers which appeared to be contaminated from overhung latrines. Limited outbreaks of cholera have been found to be water-borne (Barua, Personal Communication). Water-borne epidemics are usually explosive in nature involving a large number of persons at a time drinking the water (Mukherjee, Personal Communication). A change from explosive water-borne to slower principally carrier-borne propagation has been reported by Tamyo et al (1966). Paul (1970), in his epidemiological survey of the 1964 cholera outbreak in Kedah, observed an initially minor carrier-borne followed by a major water-borne transmission of infection.

In this series, we observed few asymptomatic short-term carriers who excreted vibrios in their stool up to eight days. Chuttani et al (1967) found 6.7% asymptomatic household carrier and 4.2% community carrier in 1,186 specimens of stool of contacts of 163 confirmed cases of cholera; they were all short-term carriers who excreted vibrios in their stool up to seven days and all of them belonged to households with shallow tube well water supplies which were seen to be heavily contaminated by El Tor vibrio. They believed that the possibility of a spread of infection through short-term carriers seemed to be remote in the majority of households and communities because in their series 80.4% of the families had no carriers and there was only a single cholera case in 91% of the households. Secondly, in most of the cases, the drinking water was highly polluted and could equally be a possible source of infection.

Our observations in regard to the role of carrier and water in the transmission of El Tor infection

---

**Table 1**

<table>
<thead>
<tr>
<th>Nature of specimen</th>
<th>Vibrio-positive</th>
<th>Vibrio-negative</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stool of suspected cases (1969 outbreak)</td>
<td>80</td>
<td>4170</td>
<td>4250</td>
</tr>
<tr>
<td>Stool of contact (1969 outbreak)</td>
<td>55</td>
<td>7195</td>
<td>7250</td>
</tr>
<tr>
<td>Water of well/river</td>
<td>35</td>
<td>465</td>
<td>500</td>
</tr>
<tr>
<td>Shellfish</td>
<td>50</td>
<td>100</td>
<td>150</td>
</tr>
<tr>
<td>Stool of healthy person</td>
<td>0</td>
<td>5000</td>
<td>5000</td>
</tr>
<tr>
<td>Stool of confirmed cases/contacts (1964 outbreak)</td>
<td>0</td>
<td>70</td>
<td>70</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>220</td>
<td>17000</td>
<td>17220</td>
</tr>
</tbody>
</table>

**Table 2**

<table>
<thead>
<tr>
<th>Nature of specimen</th>
<th>Number of specimen</th>
<th>Short-term carrier</th>
<th>Long-term carrier</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stool of confirmed cases and contacts</td>
<td>205</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>Stool of healthy person</td>
<td>5000</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>5205</td>
<td>10</td>
<td>0</td>
</tr>
</tbody>
</table>
EL TOR CARRIER STATE IN KELANTAN

were closely similar to those of Chuttani et al (1967). It is clear on the basis of the study of this large series, from 1964 to 1970, that a long-term carrier most probably does not occur in Kelantan. If a carrier state would persist for a long time, it is reasonable to expect occurrence of sporadic cases or outbreaks of cholera in almost every year during the period between 1964 and 1970 when ecological and environmental conditions have apparently remained constant in Kelantan.

Summary
A total of 17,220 specimens were bacteriologically examined in July, 1969 to November, 1970. Ten short-term carriers, but not a single long-term carrier, were found. Their role in the transmission of El Tor infection in both the 1964 and 1969 epidemics of Kelantan is considered to be insignificant. In Malaysia, water-borne, rather than carrier-borne outbreaks of El Tor cholera seem to occur frequently. Water and edible river shellfish are regarded as the main vehicles in the spread of the 1969 cholera outbreak in Kelantan.

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References
Astrup studies in the newborn

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Introduction
BLOOD GAS ANALYSIS is important in the diagnosis and management of acid-base problems. The pH value indicates whether the patient is acidotic or alkalotic. The pCO2 value gives an idea of whether it is mainly a respiratory or metabolic type of acid-base imbalance. The base-excess value enables a fairly accurate calculation of the bicarbonate required to correct metabolic acidosis using the equation, viz: body weight in kg x 0.3 x base-excess = HCO3 mEq/L required. In the very young infants, the blood gas values can be estimated by the micro-Astrup technique using arterialised capillary blood from the heel. This study is an attempt to establish the normal values of pH, pCO2 and base excess of neonates using the existing facilities of the Clinical Diagnostic Laboratory of the University Hospital, Kuala Lumpur.

Materials and Method
Arterialised capillary blood was obtained from heel prick of ten normal, unselected babies, of age half to three days old, and of birth weights more than 2.3 kg.

The heel was warmed by immersion in warm water till it looked pink. It was then held with the investigator’s thumb pressing slightly anterior to the baby’s heel and the rest of the fingers of the same hand grasping the baby’s leg just above the ankle joint. After drying the heel with cotton wool, it was then stabbed with a lancet to a depth of about 5 mm. Blood flowed out freely and this was collected in a heparinised capillary tube, (Code NW/100/CT. Exogen Limited Glasgow). The tube was held in a slightly slanting position with the lower end dipped into the drop of blood, and the upper end closed with the index finger of the other hand of the investigator which was intermittently released to allow the tube to be completely filled by capillarity. In this way, five tubes of the blood were collected. Each tube was then sealed at one end with plasticine. A metal mixer was put into the capillary tube via the other end which was then sealed with plasticine. After this, the tube was held horizontally and the metal mixer moved to and fro with a magnet. The five tubes of blood collected were sent to the laboratory for estimation by the technician using the micro-Astrup technique.

At times, blood flowed from the heel slowly. This needed slight pressure on the heel with the investigator’s thumb to squeeze out the blood, or rubbing the heel repeatedly with dry cotton, or another stab wound on the heel concerned.

Results
The results obtained by the writer are shown in Table 1. The average values obtained for pH, pCO2 and base excess are 7.36, 34.9 mm Hg and -4.8 mEq/L respectively. A comparison with values obtained by other investigators is shown in Table 2.
Discussion

The acid base status of an individual is best assessed on arterial blood using the Astrup method. This, however, is not feasible, especially in paediatric patients. Fortunately, arterialized capillary blood provides a good approximation for practical purposes. Gandy et al (1964) obtained good correlation between arterial and arterialized capillary blood pH and pCO2 in healthy newborn over three hours of age. In newborn less than three hours old, correlation was not so good especially in regard to pCO2. The discrepancy was greater in infants with impaired cardiopulmonary function. The correlation for base excess, however, was good throughout. Malan et al (1966), using arterialized capillary blood, obtained results which compared very favourably with those of Reardon et al (1960) using arterial blood in normal full-term neonates of age even less than three hours. Ann Bannister (1966), in a study of 13 neonates with respiratory distress, found good correlation between arterial and capillary blood values for pH throughout; pCO2 correlation was poor. In the case of base excess values, correlation was good provided blood was obtained from an oedema-free site.

The practical aim in the technique of micro-Astrup study is to collect anaerobically blood which is as arterialized as possible, and send it without delay to the laboratory for estimation. Considerable errors, which will invalidate the results, can occur in the collection and final estimation of the blood. Since it is often the doctor who is responsible for the blood collection, this is the area where attention to details is of utmost importance in this discussion.

Arterialized capillary blood is obtained after the warming the heel. Gandy et al (1964) warmed the heels in warm water (42 degrees C) for ten minutes. Malan et al (1966) warmed for 15 minutes. The writer's warming technique is less standardised and is insufficient and this probably contributes to the more acidotic values compared with those of other workers. (Table 2). The correlation between arterial blood and capillary blood for unwarmed heels is very poor. Gandy et al (1964) found the difference to be as high as 0.15 for pH and 48 mm Hg for pCO2. Fischer and Toussaint (1963)'s results which were from unwarmed heels (Table 2) also showed a very acidotic picture. Adequate warming of the heels also promotes free flow of the blood, and thus prevents squeezing of the heel which adds tissue fluid to the blood and also causes venous congestion; furthermore, it enables sufficient blood to be obtained from just one stab of the heel and thus prevents the baby.
from crying which can cause a decrease in pCO2, (Heese et al, 1966).

The avoidance of air bubbles in the capillary tube is also very important because air bubbles interfere with the technical process of blood gas estimation on the micro-Astrup equipment. Air bubbles also promote loss of CO2 from the blood but this is a rather slow process (Siggard-Anderson 1965). In the writer’s limited experience, it is found that free-flowing blood from the heel, consequent upon adequate warming of the heel plus rubbing of the heel with a little vaseline before stabbing it, usually enable anaerobic collection of blood in the capillary tube quite easily.

High temperature promotes metabolism which can interfere with the results. Hence the blood collected must be sent to the laboratory as soon as possible. In this study, the estimation was done in less than half an hour. Gandy et al (1964) and Heese et al (1966) were amongst the many investigators who performed the estimation immediately or within one hour after collection during which time the blood specimens were kept in refrigeration. Heese et al (1966) emphasised the need to record the body temperature at the time of the Astrup study to allow for corrections to be made in determining the actual pH of the patient. This latter measurement is extremely important in the paediatric age-group, especially in premature and newborn infants in whom hypothermia occur both easily and rapidly. Temperature corrections for actual pH are made thus: add 0.015 for ever one degree C that the patient is below 36 degrees C, and subtracting 0.015 if it is above 36 degrees C.

In the premature newborn, the blood gas values are more variable compared with the full-term newborn in any sample studied. For premature babies of about 24 hours of age, Malan et al (1966) obtained values of pH, pCO2 and base excess ranging from 7.33 to 7.48, 27.6 to 43.0 mm Hg and -7.4 to 0 mEq/L respectively.

In conclusion, a proper technique of blood collection can influence significantly the results of neonatal Astrup studies, especially in regard to adequate warming of the heels immediately prior to blood collection.

Summary

A method of collecting blood for pH, pCO2 and base excess estimation of arterialized capillary blood of ten normal newborn is described. The factors affecting the results were discussed and the need to pay attention to details in blood collection was emphasised.

Acknowledgement

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2. Professor A.E. Dugdale of University Hospital for his advice and encouragement; this study was carried out during the writer’s internship in his unit.
3. Professor Lau Kam Seng of University Hospital for provision of laboratory facilities.
4. Dr. R. Menon of General Hospital, Kuala Trengganu, for advice in the preparation of this manuscript.

References

NEONATAL HYPERBILIRUBINAEMIA

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PAEDIATRICIANS WILL GENERALLY agree there is a high incidence of jaundice among newborn infants in this country. Although several extensive studies have been carried out in Singapore (Wong, 1964 & 1966), there have been no reports concerning the incidence of neonatal hyperbilirubinaemia and the underlying factors responsible for the high serum bilirubin levels in Malaya.

This study was undertaken in an effort to widen our present knowledge on the incidence and aetiology of neonatal hyperbilirubinaemia.

MATERIAL AND METHOD

The present study is based on observations made on 3,402 live-born infants who were delivered in the Maternity Unit, University Hospital, Kuala Lumpur, during the period January 1969 to June 1970. The infants (1,688 males and 1,714 females) comprised 1,851 Chinese, 759 Indians, 669 Malays and 123 of other races. They were the result of normal delivery in 2,710 cases, forceps delivery in 370, vacuum extraction in 59, breech delivery in 114 and Caesarean section in 149 cases respectively.

All newborn infants had estimation of cord erythrocytic glucose-6-phosphate dehydrogenase done routinely while ABO, Rhesus blood group typing and Direct Coomb's test were done in all mothers and infants.

All infants who developed jaundice whilst in hospital were closely observed and were only discharged when the jaundice showed signs of abating. Infants who developed moderate or more than moderate jaundice were kept under surveillance and had repeated estimations of serum bilirubin until such time when the peak bilirubin level was passed. Exchange transfusion was carried out in cases where the serum indirect bilirubin level exceeded 20 mgs/100 ml. Cases of Rhesus incompatibility had exchange transfusions much earlier for obvious reasons.

All well babies were generally discharged by the fifth day after birth. Those found to have G-6-P.D. deficiency were kept in hospital for at least ten days, and on discharge mothers were instructed to report back with the infant at the earliest suspicion of jaundice.

Serum bilirubin levels were estimated using the method described by Malloy and Evelyn, erythrocytic glucose-6-phosphate dehydrogenase was determined using the method described by Prankerd (1962). (Normal values for G-6-P.D. obtained by this method do not usually exceed 90 minutes, intermediate values fall in the range between 120-150 minutes; while values exceeding 150 minutes definitely indicate deficiency. Personal communication - Dr. J.C. White.)

All cases of neonatal hyperbilirubinaemia encountered in the present study were classified under the following aetiological groups:—
Table 1: Aetiology of Hyperbilirubinaemia in the Different Ethnic Groups in the Present Study and in Singapore.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Chinese (36)</th>
<th>Indian (9)</th>
<th>Malay (5)</th>
<th>% of total cases</th>
<th>% Singapore cases (Wong 1966)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic (&quot;Liver Immaturity&quot;)</td>
<td>15</td>
<td>1</td>
<td>3</td>
<td>38</td>
<td>25</td>
</tr>
<tr>
<td>ABO incompatibility</td>
<td>9</td>
<td>3</td>
<td>–</td>
<td>24</td>
<td>16</td>
</tr>
<tr>
<td>G-6-P.D. deficiency</td>
<td>8</td>
<td>–</td>
<td>–</td>
<td>16</td>
<td>43</td>
</tr>
<tr>
<td>Prematurity</td>
<td>3</td>
<td>–</td>
<td>–</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Rhesus incompatibility</td>
<td>–</td>
<td>4</td>
<td>–</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>Sepsis</td>
<td>–</td>
<td>–</td>
<td>2</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Respiratory distress syndrome</td>
<td>–</td>
<td>1</td>
<td>–</td>
<td>2</td>
<td>–</td>
</tr>
</tbody>
</table>

Rhesus incompatibility: The diagnosis was only made if the infant was Rhesus positive, the mother Rhesus negative and the Direct Coomb's test positive.

ABO incompatibility: This was limited to A or B infants of blood group O mothers. Specific tests to demonstrate antibody in infants and mothers' sera were not undertaken.

G-6-P.D. deficiency: This embraces infants with abnormal G-6-P. D. values in cord blood. (This includes deficiency and intermediate values.)

Prematurity: This includes patients whose birth weight was less than 5 lbs and in whom there was no other apparent cause to account for the jaundice.

Respiratory distress syndrome: This embraces infants with idiopathic respiratory distress syndrome who developed jaundice during the course of their illness and in whom no other cause was found to account for the jaundice.

Sepsis: This includes patients with septicaemia, umbilical, urinary tract or severe skin infections and no other cause to account for the high bilirubin levels.

Idiopathic: This embraces infants whose birth weight was over 5 lbs and in whom no other cause was found to account for the hyperbilirubinemia.

RESULTS

Incidence of Neonatal Hyperbilirubinemia

From observations made on 3,402 newborn infants during their stay in hospital, it was found that 50 infants developed jaundice severe enough to warrant exchange transfusion.

Type of Delivery

Of the 50 cases of neonatal hyperbilirubinaemia encountered in this study, 36 were the result of normal delivery. Chi-squared analysis was carried out to determine if there is a significant association between the type of delivery in these cases and the incidence of hyperbilirubinaemia. \( X^2 \) was found to be 1.8377, and at one degree of freedom 0.2 > \( P > 0.1 \). This is not significant. Thus it was concluded that there is no significant relationship between the incidence of hyperbilirubinaemia and the type of delivery.

Sex

There were 31 males and 19 females in the present series. Chi-squared analysis was carried out to determine if there was an association between the sex of the infants and the incidence of hyperbilirubinaemia. The value of \( X^2 = 3.1429 \) and at one degree of freedom is not significant as 0.10 > \( P > 0.05 \). Thus there is no significant association between the sex of the infants and the incidence of neonatal hyperbilirubinemia.

Ethnic group

The 50 cases in the present series comprised 36 Chinese, 9 Indians and 5 Malays. Chi-squared analysis was carried out to determine if there was an associa-
tion between the ethnic group of infants, and the incidence of hyperbilirubinaemia. X2 was found to be 7.6764 and at 3 degrees of freedom 0.10 P > 0.05. It is thus apparent that there is no significant relationship between the ethnic origin of infants born in this hospital and the incidence of hyperbilirubinaemia.

Aetiology of Neonatal Hyperbilirubinaemia

Table I summarises the incidence of the various causes of neonatal hyperbilirubinaemia in the different ethnic groups. ABO incompatible pregnancies accounted for 24 per cent of cases, G-6-P.D. deficiency for 16 per cent, Rhesus incompatibility for 8 per cent, prematurity for 6 per cent, sepsis for 6 per cent and respiratory distress syndrome for 2 per cent of cases respectively. The cause of jaundice was not established in 38 per cent of cases.

Exchange Transfusion

A total of 84 exchange transfusions were carried out in the 50 cases of hyperbilirubinaemia studied in 50 cases of hyperbilirubinaemia studied in the present series. No serious complications were encountered during these procedures apart from bradycardia and extra systoles which occurred in one case while another developed septicaemia a few days after exchange transfusion. There were no fatalities. Kernicterus did not occur in any of the cases observed in hospital except one who was taken home by the mother on the third day and was subsequently readmitted on the sixth day with severe jaundice and kernicterus. This infant had G-6-P.D. deficiency in addition to umbilical sepsis and prematurity. The fact that kernicterus did not occur in any of the babies observed in hospital is probably a reflection of the close vigil which was maintained and the timely use of exchange transfusions in preventing this complication.

DISCUSSION

There is a high incidence of hyperbilirubinaemia amongst newborn of Chinese, Indian and Malay origin. Approximately 1 in 70 babies delivered in this hospital required exchange transfusion. Although not statistically significant, the incidence appears to be higher in Chinese than in the other two ethnic groups. Approximately 1 in 50 Chinese, 1 in 80 Indian and 1 in 130 Malay newborn developed severe hyperbilirubinaemia.

The cause of hyperbilirubinaemia was established in 62 per cent of cases. The 2 commonest causes were ABO incompatibility and G-6-P.D. deficiency which together accounted for 40 per cent of cases. Sepsis, prematurity, Rhesus incompatibility and idiopathic respiratory distress syndrome accounted for 22 per cent of cases. Rhesus incompatibility is relatively infrequent compared with the high incidence in Western countries. In the present series, Rhesus incompatibility accounted for 8 per cent of cases and occurred exclusively among those of Indian origin. This is not surprising as the Rhesus negative rate, amongst the 10,798 patients who attended the ante-natal clinics in this hospital, was 0.1 per cent in Chinese, 0.2 per cent in Malays, and 0.8 per cent in Indians as opposed to 15 per cent in Europeans (personal communication - Mr. G. Rajendran). Amongst the 4 cases of Rhesus incompatibility studied in the present series was one case which was salvaged by intrauterine intra-peritoneal transfusion and multiple exchange transfusions following delivery. It is the first case of its kind to be salvaged by this method in Malaya.

G-6-P.D. deficiency is present in approximately 2 per cent of local Malays and Chinese, and in 0.2 per cent Indians (personal communication - Professor K.S. Lau). In the present series, hyperbilirubinaemia due to G-6-P.D. deficiency occurred only in Chinese infants. The reason for this is not clear.

On comparing the aetiology of hyperbilirubinaemia in babies born in University Hospital with that of babies born in Singapore (see Table I), it is seen that there is a higher incidence of hyperbilirubinaemia due to G-6-P.D. deficiency in Singapore. This can partly be accounted for by the marked predominance of Chinese in their population, as compared to that in Malaya. Approximately 22 per cent of babies born in this hospital are of Indian origin, and this has probably contributed towards the relatively lower overall incidence of hyperbilirubinaemia due to G-6-P.D. deficiency in the present series.

It is seen from Table I that the cause of jaundice was not found in 38 per cent of cases. Wong (1966) has attributed the cause of jaundice in such cases to “liver immaturity”, where there is a transient depression of hepatic glucuronyl transferase activity in the immediate neonatal period resulting in failure of conjugation. Brown and Boon (1965) found that jaundice occurred in approximately 90 per cent of Chinese infants, 30 per cent of European infants and 70 per cent of Malay infants who were born in Singapore. They studied a large number of environmental factors but were unable to conclude that any were responsible for the ethnic group differences. Genetic cause for liver immaturity was excluded because the 3 racial groups behaved similarly in contradistinction to British infants and it was considered unlikely that the
3 different ethnic groups possess the same genetic abnormality.

There have been some recent reports on pyruvate kinase deficiency in Hongkong Chinese. It is not unlikely that this deficiency is present in a proportion of our local Chinese population. It is quite obvious that more detailed research has to be undertaken to elucidate the aetiology of hyperbilirubinaemia in patients in whom the cause of the jaundice has been ascribed to "liver immaturity" or idiopathic hyperbilirubinaemia.

SUMMARY

Observations were carried out on 3,402 infants who were born in the Maternity Unit of the University Hospital, Kuala Lumpur, during the period January 1969 to June 1970. Of these, approximately 1 in 50 Chinese, 1 in 80 Indian and 1 in 130 Malay newborn developed jaundice severe enough to require exchange transfusion.

The common causes of hyperbilirubinaemia were ABO incompatible pregnancies and G-6-P-D. deficiency. The other less common causes were Rhesus incompatibility, prematurity and sepsis. The cause of jaundice was not established in 38 per cent of cases.

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Dr. Lau, K.S. — Department of Pathology, University of Malaya.
Dr. White, J.C. — Department of Pathology, University of Malaya.
Mr. G. Rajendran — Blood Bank, University Hospital, Kuala Lumpur.)
A triple-curve catheter for selective arteriography

THE TECHNIQUE of selective arterial catheterisation has been increasingly used in recent years, particularly since the advent of image-intensification with television monitoring and the availability of radio-opaque catheters have greatly simplified the procedure. Following the introduction of a suitably shaped catheter into the aorta, usually by the percutaneous trans-femoral route, the catheter tip may be manipulated under fluoroscopic control into almost any of the major branches of the abdominal or thoracic aorta. Subsequent injection of radio-opaque contrast material enables arteriograms to be obtained which are superior in quality and in diagnostic value to those achieved by aortography.

The factors responsible for the improved visualisation are:

1. The absence of overlying neighbouring branches of the aorta, particularly the abdominal aorta, which may obscure the area of interest; and
2. The absence of dilution which is inevitable with an aortic injection.

A further advantage is that with selective arteriography, much smaller volumes of contrast medium are required. The main setback of selective arteriography is that it is technically more difficult and time-consuming, with corresponding increase in fluoroscopic time, and consequently, radiation dose to both patient and operator. Any modification of technique that would simplify the procedure and shorten examination time is therefore most desirable.

Selective arteriography is often undertaken for the renal and coeliac axis arteries. The conventional catheter shape employed is one with a single curve centred about 1½ cm. from the tip, the angle of curve varying according to the patient, but usually in the region of 50 degrees. Our recent experience with a triple-curve catheter suggests that it is an improvement on the conventional model, both reducing examination time and increasing the rate of successful catheterisation.

Preparation and Description of Catheter

We use the radio-opaque catheters designed by Odman (1956) and manufactured by Kifa of Stockholm. These can be rendered malleable by immersion in hot but not boiling water, bent to any desired shape, and then made rigid again by dipping into cold water. For selective coeliac axis and renal arteriography, we prefer the green Kifa catheter, which has an internal diameter of 1.20 mm. and an external diameter of 2.40 mm.

The shape of the catheter is as shown in Figure 1. The first curve is centred about 1½ cm. from the catheter tip, the second curve about 5 cm. from the centre of the first curve, and the third curve about 5 cm. from the centre of the second curve but in the opposite direction. The curves are such that they become more gradual the further away from the catheter tip, and that adjoining straight portions of

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Fig. 1:  A: Conventional single-curve catheter.  B: Triple-curve catheter.

Fig. 2:  Coeliac axis arteriogram performed for suspected hepatoma in a patient with cirrhosis. Arrows indicate the three curves of catheter.

Fig. 3:  Selective renal arteriogram in a patient with non-functioning hydronephrotic left kidney. Arrows indicate the three curves of catheter.

the catheter would intersect at an angle of 90 degrees if projected.

We have found this catheter shape to be equally suitable for both coeliac axis and renal artery catheterisation. (Figures 2 & 3)

Advantages of Triple-Curve Catheter

The two additional curves of the catheter not only facilitate flexion but, by providing two pivotal points of contact between the catheter and opposite walls of the aorta (Figure 4), allow for improved control and stability. In particular, we have noted the following advantages:

(1) The curve at the catheter tip usually returns spontaneously upon withdrawal of the guide-wire. Should it fail to do so, the curve can be easily restored with a little manipulation.

(2) Rotational movement of the catheter is
CATHETER FOR SELECTIVE ARTERIOGRAPHY

Diagram showing tip of catheter in right renal artery. RRA: right renal artery. A: Aorta. AB: Aortic bifurcation. Arrows indicate the two pivotal points of contact between catheter and aortic wall which permit improved control and stability.

much easier to control.

(3) Once the desired artery has been entered, the catheter position is better maintained and the catheter is less liable to be dislodged by the recoil of injection. This applies particularly in the case of coeliac axis arteriography where higher injection pressures are used.

(4) The catheter can be advanced deep into an artery and readily manoeuvred into one of its branches. In this way, it is possible to inject selectively the main branches of the renal artery.

Summary

(1) A triple-curve catheter for selective coeliac axis and renal arteriography is described.
(2) Its advantages over the conventional single-curve catheter are discussed.

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References

Hepatocellular damage due to Methyldopa

by M.L. Wong

METHYLDOPA HAS BEEN widely used as an antihypertensive agent because postural hypotension is considerably less frequent and less severe than during treatment with guanethidine or a ganglion blocking agent. Toxic reactions associated with its use have not been common and these include granulocytopenia, drug fever and haemolytic jaundice. There have been frequent reports of the development of a positive Coomb's test without evidence of haemolysis in patients receiving the drug but this did not necessitate its withdrawal. The occurrence of jaundice due to liver damage in patients on methyldopa as a toxic manifestation is not widely recognised hence it is thought worthwhile documenting this case.

Case Report

The following is a case report of a patient who developed jaundice while receiving methyldopa. E.J. was a 24-year-old Indian woman, who was admitted on 24th December 1968, complaining of sudden onset of weakness of the right side of her face and inability to close her right eye. On examination, she was found to have a right lower motor neurone type of facial palsy and an elevated blood pressure of 170/120 mm.Hg. Her peripheral pulses were all felt and equal on both sides, and her heart was normal. Examination of other systems revealed no abnormalities. Investigations did not reveal any cause for her hypertension and as she had a strong family history, she was thought to have essential hypertension. Her blood pressure was satisfactorily controlled on reserpine 0.25 mg. t.d.s. and hydrochlorothiazide 25 mg. daily. About two months later, she was reviewed in the outpatient clinic and because of poor control of her blood pressure, methyldopa was substituted for reserpine. Six weeks later, she was readmitted because of anorexia, vomiting and darkening of her urine for three days. She was afebrile and jaundiced but the liver and spleen were not palpable. The serum bilirubin was 4.9 mg/100 ml of which 3.2 mg was conjugated; SGPT was 1240 I.U. and alkaline phosphatase was 18.5 K.A.U. Her serum proteins was 8.3 gm/100 ml with a normal albumin/globulin ratio. The urine contained a trace of bilirubin and urobilinogen. Methyldopa was stopped and she rapidly improved in that her symptoms subsided and a week after admission, her bilirubin fell to 3.7 mg/100 ml and the SGPT to 370 I.U. She was re-started on reserpine 0.25 mg b.d. and hydrochlorothiazide 25 mg daily. Her jaundice cleared completely after three weeks.

A week later, when seen in the outpatient clinic, she was again given methyldopa in addition to reserpine and hydrochlorothiazide to control the blood pressure at normotensive levels. After taking methyldopa for two weeks, she again developed symptoms of anorexia, vomiting, pruritus and jaundice. Two weeks after onset of jaundice, she returned to hospital. On examination, she was deeply jaundiced, the liver was tender and enlarged 3 cm below
the right costal margin, the spleen was just palpable and there were many scratch marks on the skin. The serum bilirubin was 10.6 mg/100 ml of which 4.1 mg was conjugated, SGPT 640 I.U. and the alkaline phosphatase level was 13.8 K.A.U. The urine contained bilirubin but no urobilinogen. The direct Coomb's test was negative and a plain X-ray film of the abdomen showed no opacities to suggest the pressure of gall stones. Twelve days after stopping methyldopa, the serum bilirubin fell to 4.5 mg/100 ml, the SGPT to 196 I.U. and the urine contained urobilinogen but no bilirubin. She made an excellent recovery and when seen in the clinic 10 weeks after discharge, she had no jaundice, the serum bilirubin was 0.6 mg/100 ml. SGPT was 31 I.U. and the alkaline phosphatase was 6.5 K.A.U.

Discussion
The Medical Letter (1) recently reviewed the adverse effects of drugs on the liver. The two principal types of adverse hepatic reactions recognised were cholestatic and viral hepatitis-like damage of the liver. It was thought that whereas cholestasis was not a hypersensitivity reaction, the viral hepatitis-like injury to the liver was the result of a hypersensitivity reaction. Methyldopa was one of the drugs incriminated to produce the latter type of effect.

Williams and Khan (2) in 1967 reported on the occurrence of non-haemolytic jaundice in two patients receiving methyldopa for the treatment of hypertension. One patient had symptoms of malaise, nausea, anorexia associated with progressive jaundice and dark urine, similar to that of viral hepatitis. The other complained of epigastric discomfort and jaundice. The patients had been exposed to the drug for six weeks and seven months respectively. In both patients, the serum glutamic pyruvic transaminases were markedly elevated and these returned to normal 8 weeks in one patient and 10 weeks in the other, after methyldopa was withdrawn. Coomb's test was negative in the first patient but positive in the second who also had an elevated alkaline phosphatase.

Another report was by Wyburn-Mason and Anastassiades (3) who noted the occurrence of jaundice in a patient who had been treated with methyldopa for about six weeks. Four-and-a-half weeks after stopping the drug, the jaundice disappeared and the elevated SGPT returned to normal. Morin et al (4), in a clinical study of 28 hypertensive patients on methyldopa therapy for an average of 8 weeks, found one who developed a raised SGPT. This fell to normal after the drug was stopped. Four months later, when therapy was restarted, the SGPT level rose again, falling as before on withdrawal of the drug. Irvine (5) reported a transient rise in serum glutamic oxalo-acetic transaminase in 4 out of 15 patients receiving methyldopa for a mean period of 18 weeks. The level was reported to fall to normal without stopping treatment over this period, but the exact time interval was not specified.

In the case reported above, the appearance of jaundice and viral hepatitis-like symptoms, together with abnormal liver function tests occurring on two occasions following methyldopa therapy and the rapid improvement clinically and biochemically when the drug was withdrawn, strongly suggest that the liver dysfunction was drug-induced.

If a patient receiving methyldopa develops jaundice, drug sensitivity must be thought of as a cause because the toxic effect on the liver appears to be reversible. All recorded cases and the one reported here have made a rapid complete recovery on withdrawal of the drug (7).

References:
Tetracycline-resistant Haemolytic Streptococci in Kuala Lumpur

by S.D. Ampalam and S.C. Cheng

Introduction
RESISTANCE OF PATHOGENIC BACTERIA to antibiotics is becoming an ever increasing problem. Over the past several years, there have been a number of reports which point to the existence of an appreciable proportion of tetracycline-resistant strains among any large group of beta-haemolytic streptococci.

Reports from Britain show an increasing incidence of tetracycline-resistant streptococci. Parker, Maxted and Fraser (1962) found that 12% of 921 streptococci of Lancefield’s group A, submitted to the Streplococcus and Staphylococcus Reference Laboratory at Colindale, were tetracycline-resistant. Mitchell and Baber (1965) found that 32% of 640 group A strains isolated in the Bristol area were tetracycline-resistant. Dadswell (1967) found an increase in the tetracycline-resistant group A streptococci from 1% in 1958 to 44% in 1965. Robertson (1968) found that the overall percentage of tetracycline-resistant streptococci (groups A, B, C, G and D) had remained almost stationary, varying from 28% in 1963 through 35% in 1965 to 27% in 1967.

In the United States of America, Kuharic, Roberts and Kirby (1960) found 20% of group A streptococci from clinical sources to be tetracycline-resistant.

In Australia, Lane (1962) found that 19.4% of 98 streptococci to be fully resistant to tetracycline.

Strains of naturally occurring group A haemolytic streptococci.

There is, at present, no information in Malaysia on the antibiotic sensitivity pattern of the haemolytic streptococci. The present study was undertaken to establish a base line of the in vitro antibiotic sensitivity pattern, and to observe if there is any significant proportion of resistant strains amongst the streptococci isolated from clinical material.

MATERIALS AND METHODS
Routine specimens were submitted from June 1967—March 1970 to the Bacteriology Department of the University Hospital which has busy out-patient departments and about 700 acute beds. Specimens were taken on sterile cotton wool swabs.

The table shows their sources and the numbers isolated from each site:

<table>
<thead>
<tr>
<th>Source</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Throat</td>
<td>356</td>
</tr>
<tr>
<td>Wounds and Abscesses</td>
<td>98</td>
</tr>
<tr>
<td>High Vaginal Swabs</td>
<td>10</td>
</tr>
<tr>
<td>Blood Culture</td>
<td>6</td>
</tr>
<tr>
<td>Ear</td>
<td>6</td>
</tr>
<tr>
<td>Sputum</td>
<td>4</td>
</tr>
<tr>
<td>Urine</td>
<td>3</td>
</tr>
<tr>
<td>Eye</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>485</strong></td>
</tr>
</tbody>
</table>
TETRACYCLINE RESISTANT HAEMOLYTIC STREPTOCOCCI

In the laboratory, the swabs were cultured on 10% layered ox-blood agar plates. All plates were inoculated on a 2 x 2 cm. area and streaked out with 3 successive series of streakings, flaming the loop between each. The plates were incubated overnight at 37°C., both aerobically and anaerobically.

All colonies showing beta-haemolysis were picked and subcultured to obtain pure growth. Sensitivity to antibiotics was tested by streaking blood agar plates with the strain, on which were then placed filter paper discs impregnated with antibiotics (MAST). The concentration of the antibiotic in each disc was penicillin 1 u.g, ampicillin 5 u.g, cephaloridine 5 u.g and erythromycin 5 u.g. A strain was considered resistant only if it grew right up to the edge of the disc.

The Lancefield grouping of the streptococci was determined by sensitivity to bacitracin (discs of 0.1 units – Mast), and by the precipitin reaction (Lancefield 1933) using sera of groups A, B, C and G (Burroughs Wellcome).

RESULTS
A total number of 485 strains of haemolytic streptococci of all groups were isolated, of which 126 (25.98%) were tetracycline-resistant.

Lancefield grouping on 315 strains showed 180 (57.1%) belonged to group A, 8(2.5%) to B, 40 (12.7%) to C, 51 (16.2%) to G and 36 (11.6%) to none of these groups. These figures compare closely with those of Robertson (1968).

Of a total of 180 group A strains, 29 were tetracycline-resistant giving the figure of 16.1% as tetracycline-resistant group A strains.

Erythromycin-resistance was noted in 5 strains (1.0%) of which only 2 belonged to group A.

All the strains were fully sensitive to all the other antibiotics i.e. penicillin G, ampicillin, orbenin and cephaloridine.

DISCUSSION
The overall tetracycline-resistance of 26% is lower than published figures for U.K. – (28-35% Robertson, 1968). Similarly, the figure of 16.1% for tetracycline-resistant group A streptococci is very much lower than the 32% of Mitchell and Baber (1965) or the 44% of Dadswell (1967). But the figure of 16% resistant group A strains is not very far from the 20% reported by Kuharic and Kirby (1960) in the U.S.A., and the 19% reported by Lane (1962) in Australia.

The concentration of the tetracycline in the disc used for the sensitivity testing was 25 u.g which is higher than that used by Robertson (10 u.g) and Dadswell (10 u.g), but Mitchell and Baber used 25 u.g per disc. It is possible, in fact very likely, that had we used a tetracycline disc of 10 u.g, the figures would have been higher than that obtained using the 25 u.g disc.

Resistance to erythromycin has been reported by Lowbury and Hurst (1959) of haemolytic streptococci isolated from 4 patients with burns. Lowbury and Kidson (1968) have also described erythromycin-resistant strains isolated from patients suffering from burns but they were also resistant to lincomycin. Dixon (1968) described a group A haemolytic streptococci isolated from a throat swab which was resistant to both erythromycin and lincomycin.

One of our erythromycin-resistant strains was isolated from a wound swab and was a group G, 2 were from throat swabs and were group A and the other 2 were also from throat swabs but not group A, B, C or G. It appears that our 1% erythromycin-resistant streptococci is fairly high.

The implication of the above findings is important. Firstly, we have established a base line for the tetracycline and erythromycin-resistant haemolytic streptococci in Kuala Lumpur. It would be interesting to see whether these figures increase or decrease in the future. This would depend on the policy of antibiotic treatment adopted in the case of streptococcal infections.

It is difficult to assess how widely tetracycline is used for the treatment of streptococcal infections, especially sore throats. Tetracycline resistance may be responsible for failure to cure the streptococcal carrier state or to halt the progress of established infection with streptococci. But clinical improvement with tetracycline treatment may be due to mixed infections and elimination of sensitive strains before the emergence of resistant strains.

McCormack et al (1962) considered that the hospital might provide an environment for the selection and dissemination of tetracycline-resistant streptococci very similar to the hospital staphylococci. This may be true. From our own experience, haemolytic streptococci isolated from the throats of normal schoolchildren aged 7 years, showed only about 5% tetracycline resistance (unpublished data).

The problem of the tetracycline-resistant haemolytic streptococci can be very largely circumvented, but, unless the present magnitude of this problem is appreciated, it may become even greater as tetracycline becomes cheaper.

It is, therefore, important that tetracycline should
not be used in the treatment of streptococcal infections unless the sensitivity of the infecting organism has been previously determined.

It is probable that by refraining from the use of tetracycline at present, that most of the haemolytic streptococci in the distant future would once more revert and become sensitive to tetracycline.

SUMMARY
Antibiotic sensitivity was done on 485 strains of haemolytic streptococci. Overall tetracycline resistance was found to be 26% and that of group A was 16.1%. The importance of this is discussed.

ACKNOWLEDGEMENTS
We would like to express our thanks to Dr. M.T. Parker of the Streptococcus Reference Laboratory, Public Health Laboratory Service, Colindale, London; Dr. D.J.H. Payne, Director, Public Health Laboratory, Portsmouth, for their advice and help, and Mrs. J. Daish for typing the manuscript.

REFERENCES
Fracture of the First Rib with Associated Fracture of the Clavicle

"If in another subject the outer end of the clavicle be forced backwards, so that it rests upon the first rib outside its tubercle, being separated from it by the scalenus medius, a heavy blow be struck horizontally backwards on its padded outer extremity, in some rare cases the inner end of the clavicle is dislocated forwards, but in the large majority of cases, the first rib is fractured at the point of impact. The facility with which this fracture of the first rib is produced is remarkable, and if one compares the relative thickness and strength of the first rib and clavicle and the mechanical advantages of the clavicle upon the first rib, one would not be surprised to find this fracture occur not uncommonly during lifetime."

(Lane W.A., Guy's Hospital report 43, 321, 1885).

SINCE THIS OBSERVATION was first made by Lane in 1885, little in the nature of publications appeared in the literature until about the end of World War II. A publication by Alderson in 1944 reporting a series of anomalies in the first rib, detected among routine skiagram of chest in naval personnel, stimulated considerable communications and debate (Alderson B.R., 1945; Hartley J.B. 1945). Close to 300 cases are known to be recorded in English language literature, the majority being retrieved in bulk from the radiological archives of the armed forces. It appears from reviewing the literature on the subject that papers hitherto have mainly limited itself to the hypothetical violent muscular action of the scalene (Jenkins S.A. 1952; Powell F.I., 1950) and serratus anterior muscles (Powell F.I. 1950) as the prime mechanism involved in the genesis of such fractures, and only a passing reference to a co-existing fractured clavicle with fracture of first rib have been made in Knoep's paper on fractures of ribs in 1945. This paper puts on record two cases of fracture of first rib with associated fracture of the clavicle and in the light of contemporary accident pattern, a hypothesis is offered regarding the mechanism of such fractures based on the sole pioneer experimental work of Lane (Lane W.A. 1884/1885).

Case 1

A 28-year-old labourer, while riding a motorcycle, collided with a lamp-post, hitting the left side of his
neck against the post violently. As a result of this, he sustained a closed fracture of the middle third of the left clavicle and fracture of the first rib in the region of the scalene tubercle. A coincident fracture of the left forearm was treated by closed manipulation and immobilisation in a cast. The clavicle fracture was treated in a sling. Subsequently, within two weeks, the patient noticed progressive weakness and wasting of muscles of the shoulder girdle which since has progressed to a complete flail arm. B.P. is equal on both sides but radial pulse disappears on 90 degree abduction of the arm.

Case II

A 25-year-old male, unable to negotiate a curve while driving a car, was hurled forward against the steering wheel and thrown out on to the road through the windshield. He sustained multiple lacerations with compound comminuted fracture of his face. Besides this, he had compound comminuted injury of the right shoulder with closed fracture of the clavicle on the same side together with fractures of the 2nd to
the 8th ribs and a flail right chest wall. On the opposite side, the clavicle and the first rib were found to be fractured. Dynamic stabilisation of the chest with intermittent positive pressure respiration and tracheostomy helped in the successful outcome of the patient.

Discussion
The normal anatomy affords considerable protection to the first rib from the effects of external violence. The immediate anatomical relations divide it into a fixed anterior segment to which is attached the costoclavicular ligament, a posterior mobile segment affording attachment to the scalene muscle, and a middle segment buttressed across by the subclavian artery. A sudden violent contraction of the scalene/serratus muscle is the commonly accepted cause of fracture. Nevertheless, solution of continuity through substance of the first rib have been variously attributed to

(i) a persistent synchondrosis between the two ossific centres of the bone (Gershon-Cohen, Delbridge, 1945)
(ii) a fatigue fracture (Alderson 1944) and
(iii) an actual developmental anomaly (Sycamore 1944).

All these papers are based on radiological appearances disregarding the role of trauma if any. Jones (Breslin F.J. 1937) in 1869 described a similar case following direct trauma on the neck. Later, Powell in 1950, in a series of 21 cases, described fractures following both direct and indirect trauma. Only in one paper (Knoep 1945) is there a passing remark on the association of fractures of the clavicle and of the first rib, with no explanation offered regarding the mechanism involved.

After considerable deliberation, Lane, in his presentation at the Pathological Society of London in 1884/1885, submitted that fractures of the first rib was the result of either direct trauma or indirect trauma transmitted through the clavicle or sternum. When transmitted through the clavicle or sternum. When transmitted through the clavicle, the point of impact in relation to the clavicle is its centre and should the clavicle fracture, it may seem to do so, “so to speak, across the first rib, the clavicle yielding instead of the other parts affected by the strain.” (Lane 1884/85). This was his casual remark.

In attempting to unravel the mechanism of fracture in the two cases, it is observed that in both cases, high speed vehicles have been brought to a rapid standstill after colliding with solid objects. This resulted in a rapid deceleration accompanied by deformation of the parts. When the force of deceleration generated is calculated from the formula \( b = \frac{v^2}{2s} \) (where \( b \) = deceleration, \( v \) = speed of vehicle and \( s \) = linear deformation), it becomes apparent that the resultant force was of an intensity, which Lane is unlikely to have been able to produce in the dissection hall on the cadavers, a force more horizontal than vertical, transmitted on to the first rib through the clavicle, resulted in fractures of both the bones at their classical sites before dissipating itself. No doubt a combination of forces has been active, as is evident from the other co-incident injuries.

Summary
Two cases of fracture of the first rib associated with fractured clavicles are described and an explanation on the mechanism of the lesion submitted.

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Disgerminomoma: A case report

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INTRODUCTION
Disgerminoma is an interesting though uncommon tumour of the ovary. The prefix 'dis' means two, and refers to the fact that it occurs in both sexes. The disgerminoma of the ovary is in fact the same tumour as the seminoma of the testes. Disgerminoma comprises about 3–5% of all malignant ovarian tumours. (Morris & Scully 1958.) It is characteristically a tumour of early life and used to be known previously as 'Carcinoma Puellarium'. A case of disgerminoma showing many characteristic features is described.

CASE REPORT
M.L.F., an unmarried Chinese girl, aged 16 years, was first seen in June 1969. She complained of an abdominal swelling of two months' duration and amenorrhoea for six months. Her menarche was at 14 years, and her periods were scanty, occurring every 3 to 4 months.
On examination, she was a healthy girl, with normal secondary sexominal examination, a smooth firm mass arising from the pelvis up to half way between the symphysis pubis and the umbilicus was present.
She was a virgo intacta and on rectal examination, a mobile mass about 4–6 inches in diameter was felt separate from the uterus. The uterus itself was normal in size and retroverted.
A pregnancy test (Gravindex) was negative and abdominal X-ray showed no evidence of calcification.
At laparotomy, a soft solid tumour, grey in colour, was found arising from the right ovary. The left ovary was small and had a smooth surface. A right salpingo-oopherectomy was performed, and recovery was uneventful.

Histology
The tumour was composed of pleomorphic cells in solid clumps and, in places, branching cords separated by fibrous strands. There were small follicles of lymphoid tissue separating these cells, features consistent with a diagnosis of disgerminoma.

Follow-up
Since operation, her menstrual period has been occurring monthly and the flow has also been normal.

DISCUSSION
Disgerminomas occur in youth generally before 20 years. It is said to be more common in intersexes and patients having the tumour exhibit signs of varying degrees of hypogonadism. This is similar to the male where imperfectly developed testes are much more
likely to develop seminoma. However, the tumour itself is not the cause of the sex deficiency which may persist after its removal.

This patient’s age — 16 years — corresponds to the classical picture and the scanty and irregular menstruation can be taken as a manifestation of hypogonadism. The surprising thing is that following the removal of the tumour, the menstrual periods have become normal. Although these tumours are most often ‘neuter’ i.e. not producing any sex hormones, some definitely have mild oestrogenic or androgenic influence. A mild androgenic effect was probably present in this patient, and the removal of this influence could be the reason for the restoration of regular menstruation.

As with many other types of ovarian tumours, the first evidence of its presence is the detection of a mass in the abdomen as in this patient. Although generally there is no characteristic effect on menstruation, as explained above, menstrual abnormalities may co-exist. When marked sex abnormalities are present and an ovarian tumour is detected, the strong possibility of disgerminoma must be considered.

Nevertheless, disgerminoma often occurs in apparently normal women, sometimes first presenting during pregnancy.

Aetiology

The tumour is believed to arise from mesenchymal cells which date back to the early undifferentiated phase of gonadal development. In this phase, the cells have not acquired either male or female characteristics, so that as might be expected the tumour has no effect on the sex characteristics of the patient. Such an origin, as postulated by Meyer, is supported by the fact that an identical tumour occurs in the testicle where it is called seminoma. This is as one would expect with tumours, which as it were lag behind the differentiating process in the gonads, which later develop into either testes or ovaries. Another point in favour of this hypothesis is the fact that in a considerable portion of the reported cases, the tumour occurred in individuals showing some degree of gonadal deficiency.

More recently it has been shown that the tissues of the disgerminoma are chromatin negative. There is also evidence that the growth itself or the tissue from which it arises has a sex chromosome complement of XY. (Jeffcoate). According to this view, the disgerminoma arises from ‘male’ tissue and even in an apparently normal woman, the ovary from which the tumour arises is likely to be the site of mosaicism XX/XY. This mosaicism has been attributed to dispermy, i.e. fertilisation of an ovum by two spermatozoa, one carrying an X and one a Y chromosome. Thus those who develop disgerminoma of the ovary have in their gonads an XY strain of tissue, even though their other tissues have an XX complement only.

Pathology

The disgerminoma tends to grow larger than other sex tumours. In this patient, the tumour was fairly large measuring six inches across. (Fig. 1.) They are solid tumours with a greyish yellow cut surface and they have a characteristic firm and rubbery feel. The growth is usually unilateral though bilateral tumours have been reported.

Microscopically, this is one of the most distinctive and easily recognisable of all ovarian tumours. The

Fig. 1. shows the cut section of the tumour.
tumour is composed of large cells having a spherical nucleus, which are arranged in bundles or alveoli separated by a network of connective tissue which contain lymphocytes.

Malignancy
According to the majority view, this tumour belongs to the malignant group. There is, however, much variation in the degree of malignancy, and it is extremely difficult to compute its exact incidence. The extreme view is that all are malignant but a figure of 25 – 30% malignancy rate seems reasonable. (Stabler 1963). In many cases, cure has followed simple removal of the tumour. In well encapsulated tumours, the prognosis is good but in the infiltrating variety, the outlook is unfavourable.

Treatment
Surgical excision is the treatment of choice. As these tumours occur in young girls, there is a natural tendency to be conservative and a simple salpingo-oopherectomy is all that is necessary. This surgical conservatism is fully justified by the reported results and by the fact that we are dealing with a tumour of low malignant potential, frequently involving young patients. However, radical surgery may sometimes be indicated if the tumour has infiltrated its capsule and involved adjacent pelvic organs.

Acknowledgement
I wish to thank the Director-General of Medical Services Malaysia for kind permission to publish this paper.

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Effects of Metabolic Acidosis — a review with case reports

by A.A. Khawaja

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METABOLIC ACIDOSIS occurring during anaesthesia has been shown to produce respiratory, circulatory and central nervous system depression. Brooks and Feldman (1962) described the clinical picture in such a situation: at the end of the operation, the patient remains unconscious or very confused. Respiration is absent or inadequate and is frequently gasping in nature, associated with tracheal and jaw tug. Cardio-vascular impairment is manifested by a progressive hypotension and cardiac arrhythmias may occur. Peripheral cyanosis is usually present. Death results from circulatory failure despite adequate artificial ventilation and the use of vasopressor drugs. These workers showed that this picture of "neostigmine-resistant curarisation" was, in fact, due to metabolic acidosis and could be successfully treated by the infusion of sodium bicarbonate.

There is also much other evidence that metabolic acidosis causes impairment of the cardiovascular and other systems. Price and Helrich (1955) found that a decrease in pH of 0.4 - 0.5 units is associated approximately with a 50% decrease in the mechanical ability of the heart. Wildenthal et al (1968) found that after an initial positive inotropic effect, acute lactic acidosis exerted a direct negative inotropic effect on the dog left ventricle and reduced the ventricular responsiveness to exogenous catecholamines. Significant depression in ventricular contractility was apparent at pH 7.10. Their data provide further evidence for the rationale behind the clinical use of alkalinising agents in acidosis.

Kittle and his co-workers (1965) reported that the mean arterial blood pressure declined slightly and gradually during metabolic acidosis. When pH values of less than 7.20 were reached, cardiac output declined and peripheral resistance increased. Clowes et al (1961), studying the effects of acidosis on cardiovascular function in surgical patients, were of the opinion that metabolic acidosis, with pH values above 7.2, may well cause serious circulatory disturbance. They noted that when arterial pH was reduced to a level between 7.25 and 7.20, it was usually associated with a serious reduction in the cardiac output and an increase of the total peripheral resistance. Stewart et al. (1965) observed that, clinically and in animals, extreme acidosis resulted in a sequence of arrhythmias which progressed through sinus tachycardia, electrical alternans, two - to - one heart block, and asystole. The sequence was reversed and cardiac function restored after administration of bicarbonate. In cardiac arrest, too, the heart is easier to restart and the rhythm is better when the metabolic acidosis present has been corrected (Ledingham and Norman, 1962; Brooks and Feldman, 1962; Lancet 1962; Stewart, Stewart and Gillies, 1962; Brooks, 1967).

Metabolic acidosis affects other body systems as
well. Lowering the blood pH raises the pulmonary arterial pressure (Silove et al., 1968), and pulmonary non-elastic resistance and work increase in metabolic acidosis (Peters and Hedgphet, 1966). Bersentes and Simmons (1967) found that while moderate acidosis resulted in renal vasodilation, more severe acidosis caused vasoconstriction. The vasodilation appeared to be a local effect of CO2 and in metabolic acidosis, there was a greater preponderance of constrictor effects over vasodilation. Nahas and Poyart (1967) reported that a decrease in arterial pH to 7·0 exerted an inhibiting effect on norepinephrine induced lipolysis and calorigensis. They concluded that the ability of the body to mobilise fuel stores and to increase metabolism above basal levels is inhibited by an acid pH. Metabolic acidosis also produces conditions least favourable to synthesis of liver glycogen (Geddes, 1967). Dinffenass (1965) postulated that metabolic acidosis, by increasing the internal rigidity of the red cell and hence increasing the local blood viscosity, may be a factor in the pathogenesis of thrombosis.

Recently, however, some evidence has been put forward to show that metabolic acidosis may not be responsible for the cardiovascular depression and other derangements that it has traditionally been thought to cause. Andersen et al (1967) found that metabolic acidosis produced no depression of cardiac output until the pH fell below 6·9, and that there was no significant change in arterial blood pressure or peripheral vascular resistance, though there was a progressive increase in pulmonary arterial pressure and resistance. The responsiveness to adrenaline was retained down to a pH of 6·8.

In an earlier paper, these workers (Andersen and Mouritzen, 1966) showed that when metabolic acidosis was produced by the injection of lactic acid in dogs, as the pH fell, the cardiac output increased and at pH 6·8, the output was 185% of control, and peripheral resistance was decreased. They suggested a re-evaluation of the practice of artificial correction of a moderately depressed pH for presumed cardiac benefits.

Anderson (1968), investigating the relation between metabolic acidosis and cardiac arrhythmias in acute myocardial infarction, concluded that the apparent predisposition of patients with metabolic acidosis to develop arrhythmias was probably related to the greater severity of their illness rather than a direct result of the acidosis, particularly since correction of the acidosis, although improving the general condition, did not correct the arrhythmia. Metabolic acidosis, hypotension and arrhythmias were closely associated. He was of the opinion that the metabolic acidosis was a result of the hypotension caused by circulatory insufficiency resulting from the arrhythmia rather than the cause of the arrhythmia.

And Rand et al (1968), investigating the effect of pH on blood viscosity, stressed that viscosity changes that accompany acidosis and alkalosis were negligible when compared with those found in other conditions.

It is difficult to reconcile these conflicting reports. Some of the variations may be due to the particular animal studied and whether the organism was intact or a heart-lung preparation. It may be that the metabolic acidosis produced in animals by the infusion of hydrochloric or lactic acid is in some way different from the clinical metabolic acidosis of hypoxia and anaerobic metabolism. Or a possible basis for reconciliation of the conflicting results may lie in the observations of Wildenthal et al (1968), who demonstrated that inotropic effects of acidosis on the heart may be interpreted as positive, negative or no change, depending on sympathetic-adrenal function and on the time of observation. Clinically, however, it is apparent that when a metabolic acidosis of greater than mild degree is present, not only is there no benefit in withholding alkalinising agents but that improvement usually follows correction of the acid-base balance. The following cases, where post-operative cardio-respiratory impairment was corrected by infusion of sodium bicarbonate, are reported as illustrating the point.

CASE REPORTS

Case 1.

A 31-year-old woman, who had had a previous Caesarean section and had a history of pre-eclamptic toxaemia during the present pregnancy, presented for a repeat section because she had made little progress during a 12-hour trial of labour. At this stage, she was having strong contractions and was rather distressed. Her pulse rate was 106 beats/minute and the arterial blood pressure, which previously was in the region of 130/90 mm of Hg, had risen 160/100 mm Hg. She had pitting ankle and sacral oedema, and her urine contained a trace of acetone.

Anaesthesia was induced with thiopentone and suxamethonium, preceded by 0·6 mg of atropine, and, after endotracheal intubation, was maintained with N2O:O2:0 and curare. The baby was delivered about 12 minutes after induction. At this stage, he placenta was found to be adherent and, during the separation, she lost about a litre of blood in a period of five minutes. Her systolic BP fell to 65 mm Hg.
EFFECTS OF METABOLIC ACIDOSIS

She was given a rapid infusion of 500 ml of lactated Ringer’s solution and 500 ml of whole blood. With this therapy, her colour improved and the systolic BP rose to 100 mm Hg. A further unit of blood was given somewhat more slowly. At the end of the operation, the systolic blood pressure was 120 mm Hg. The curare reversed satisfactorily with atropine and neostigmine and the patient was conscious within a few minutes.

About one quarter of an hour later, in the recovery ward, she was drowsy, blood pressure had fallen, pulse was weak and colour poor, she was sweating and obviously hyperventilating. Although the estimated blood loss had been replaced, she was given 500 ml of haemacel, a plasma expander. When this did not have an appreciable effect, she was suspected to be acidic. An arterial blood sample was, therefore, taken anaerobically into a heparinated syringe for blood gas analysis and she was given 90 mEq of sodium bicarbonate. Also, since she was hyperventilating, to cut down the work of respiration, she was re-intubated and respiration assisted using a Bird ventilator. This resulted in marked improvement of her blood pressure and pulse. Blood gas analysis showed: pH: 7.26; pCO₂: 23 mmHg; B.E.: -15.5 mEq/L; standard bicarbonate: 13.4 mEq/L, i.e. a severe metabolic acidosis partially compensated for by a respiratory alkalosis. She was given a further 135 mEq of sodium bicarbonate.

When seen about three-and-one-half hours later, her condition was much improved, with a good colour and pulse, an arterial blood pressure of 130–140/80 mm Hg, and a good urine output. Respiratory assistance was, however, continued overnight to allow her to be well-sedated and to get a good night’s rest. Her further post-operative course was uneventful except for a mild chest infection.

Case 2

This 55-year-old man presented initially with colicky pain in the right loin. Intravenous pyelography revealed bilateral renal calculi. Apart from a mild hypertension (BP 170/130), other systems were essentially normal and his blood urea varied between 37 mg and 47 mg%.

A right-sided nephrolithotomy and nephrostomy was done. Initially, there was good urine output from the nephrostomy and per urethra but then the nephrostomy started leaking into the tissues. To further complicate matters, a few days later he started bleeding from a duodenal ulcer, for which a Billroth II partial gastrectomy was done. When seen on the present occasion, he presented for closure of a duodenal fistula. His general condition was rather poor.

On the day before the operation, his serum electrolyte and blood urea results were: Na+: 119 mEq/L; K+: 6.2 mEq/L; Cl⁻: 93 mEq/L; blood urea: 78 mg%. The next day, however, the urea and serum potassium had come down to 65 mg% and 5.5 mEq/L and serum sodium and chloride had gone up and it was decided to proceed with the operation. Anaesthesia was induced with methohexitone and continued with nitrous oxide, oxygen, intermittent halothane, curare and moderate hyperventilation. His systolic blood pressure was 110 mm Hg before induction of anaesthesia and throughout the operation, which lasted a little less than two hours, remained between 110 and 130 mm Hg. Operative blood loss was estimated to be about 400 ml and he was given 500 ml of lactated Ringer’s solution and 450 ml of whole blood. Curare reversal at the end of the operation was satisfactory. About 45 minutes later, however, he was drowsy, blood pressure was 190/90 mm and pulse rate 120 beats per minute. Respiration was rather laboured and colour poor. Residual curariation was suspected to be present, but a dose of neostigmine of 0.5 mg had no effect. At this stage, the presence of acidosis was suspected. Blood was taken for analysis and he was given 90 mEq of sodium bicarbonate. Blood gas results showed: PO₂: 123 mmHg (breathing O₂ enriched air); pH: 7.13; pCO₂: 43 mmHg; BE: -15.5 mEq/L; standard bicarbonate: 13.2 mEq/L, i.e. a severe uncompensated metabolic acidosis. Calculation, according to the formula of Mellemgaard and Astrup (1960), showed the deficit of base in the extracellular compartment of body water to be 270 mEq. He was accordingly given a further 180 mEq of Na HCO₃. Since respiration was rather laboured, he was re-intubated and respiration assisted with a Bird ventilator. About two hours later, his condition was satisfactory and he was extubated and returned to the ward.

Case 3

A 44-year-old man presented with chronic gout, a right renal calculus and chronic renal failure. A radiotope renogram showed complete right ureteric obstruction, and a non-functioning left kidney. While awaiting surgery, his blood urea climbed from 90 mg to 500 mg per 100 ml of blood. This was treated by peritoneal dialysis. It was noted that he tended to develop metabolic acidosis which required oral supplements of sodium bicarbonate. He underwent several operations. On the present occasion, he was ope-
rated upon because of a ureteric stricture for which a Davies’ intubated ureterotomy was done. His preoperative condition was fair, with essentially normal serum electrolytes and a blood urea of 68 mg%. Anaesthesia was induced with thiopentone and maintained with curare, nitrous oxide, oxygen and moderate hyper-ventilation.

During the operation, which lasted a little over six hours, he lost an estimated three litres of blood and received 2000 ml of lactated Ringer’s solution, 2225 ml of whole blood, and, towards the end of the operation, 200 ml of 20% mannitol. A further two units of blood was transfused in the post-operative period.

Because of his tendency to develop metabolic acidosis and because of the long duration of the operation, he was expected to have similar post-operative problems as the first two cases. A few minutes before the end of the operation an arterial blood sample was, therefore, taken for blood gas analysis. This showed a marked metabolic acidosis with a B.E. of −13.5 mEq/L, which was being compensated for by the intermittent-positive-pressure-hyperventilation. He was given 180 mEq of sodium bicarbonate i.e. the calculated deficit of base in the extracellular fluid. When the operation ended a few minutes later, the curare was reversed satisfactorily and there were no problems in the immediate post-operative period.

Discussion

In the first case described, the severe metabolic acidosis was probably a result of the summation of the acidosis of labour (Derom, 1968), and that due to the period of hypotension and oligaemia with a contribution from the acidotic stored blood that was transfused (Lancet, 1962) and a minor contribution from the acidosis of anaesthesia and hyperventilation (Papadopoulos and Keats, 1959). In the second case, there was little blood loss and the acidosis was probably due to the chronic renal failure and loss of base through the duodenal fistula. Both these cases illustrate how severe metabolic acidosis may go unsuspected. And in both cases, correction of acidosis resulted in prompt clinical improvement.

The third patient, who was anaesthetised a few days after the second case, was expected, on the basis of his history of chronic renal failure and in the light of previous experience, to develop a metabolic acidosis. This, indeed, proved to be the case. Intraoperative correction of the acidosis prevented problems that might have been expected in the post-operative period.

These cases show that, notwithstanding experimental evidence to the contrary, metabolic acidosis may be expected to give rise to cardiorespiratory and central nervous depression in the post-operative period, though sometimes there may be a compensatory hyperventilation instead of the more usual respiratory depression described by Brooks and Feldman (1962).

In either case, sodium bicarbonate infusion can be expected to improve the patient’s general condition and cardiovascular respiratory function. If the presence of metabolic acidosis is suspected and diagnosed during the operation, as in the case of the third patient, treatment can be expected to prevent the post-operative upsets seen in the first two patients.

While disturbances of acid-base balance should always be diagnosed and treated on the basis of results of blood gas analysis, where facilities for such analysis are not available, a good case can be made out for a therapeutic trial of sodium bicarbonate infusion in patients suspected to have acidosis of metabolic origin. An infusion of NaHCO₃ of 1 – 1.5 mEq/kg body weight may be expected to produce some improvement in the patient’s condition in the presence of a metabolic acidosis and is not likely to do any harm even if such acidosis is not present.

Summary

Some of the recent literature on the effects of metabolic acidosis on the cardiovascular and other body systems is reviewed. Two cases are reported where metabolic acidosis produced cardio-respiratory and central nervous depression in the immediate post-operative period and sodium bicarbonate infusion reversed this depression. A third case is reported where the post-operative depression could have been expected but was prevented by the intra-operative correction of the acidosis.
EFFECTS OF METABOLIC ACIDOSIS

References


Algaphan in Obstetrics

by Johan A. M. Thambu

MBBS, MRCOG, AM
Pakar Perbidanan dan Sakitpuan,
Rumah Sakit Bersalin,
Kuala Lumpur,
Selangor.

Algaphan (D-propoxyphene) has been used in obstetrics to shorten the duration of the first stage of labour. This report describes the results obtained in clinical trials of 100 primigravidae in labour.

Materials and Method:—
The study was carried out in the Department of Obstetrics and Gynaecology, General Hospital, Kuantan, Pahang, during the period May 1968 to June 1969. The cases selected were all primigravidae in early labour and the control group were the alternate cases which did not receive any Algaphan. A special protocol was prepared and the patients received the injection Algaphan intramuscular route after the vaginal examination were carried out and the findings recorded. The dose of Algaphan given was 2 ml. which contained 75 mgm. D-propoxyphene. In the trial group, the patients were only given one dose of 75 mgm. Algaphan.

Results:—

Table 1 Ethnic Group/Age

<table>
<thead>
<tr>
<th>TRIAL GROUP</th>
<th>Ethnic Group</th>
<th>Age</th>
<th>15 - 19</th>
<th>20 - 24</th>
<th>25 - 29</th>
<th>30 - 34</th>
<th>35+</th>
<th>Total</th>
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<td></td>
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<td>30</td>
<td>24</td>
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<td>64</td>
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<td></td>
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<td>3</td>
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<td>73</td>
<td>10</td>
<td>6</td>
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<tr>
<td>CONTROL GROUP</td>
<td>Ethnic Group</td>
<td>Age</td>
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<td>20 - 24</td>
<td>25 - 29</td>
<td>30 - 34</td>
<td>35+</td>
<td>Total</td>
</tr>
<tr>
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<td>9</td>
<td>2</td>
<td>4</td>
<td>1</td>
<td>16</td>
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<td></td>
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<td>19</td>
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<td>69</td>
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<tr>
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<td>5</td>
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<td></td>
<td>Total</td>
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<td>18</td>
<td>55</td>
<td>23</td>
<td>4</td>
<td>0</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 1 shows the Ethnic group and age of the primigravidae under study. In the trial group, there were 23 Malays, 64 Chinese and 13 Indians and in the control group there were 16 Malays, 69 Chinese and 15 Indians. The majority of the patients were below 25 years (65% in the trial group and 72% in the control group).
ALGAPHAN IN OBSTETRICS

Table II Ethnic Group/Injection delivery time

<table>
<thead>
<tr>
<th>Ethnic Group</th>
<th>Time in hours</th>
<th></th>
<th></th>
<th></th>
<th></th>
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<tbody>
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<td>+</td>
<td>+</td>
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<td>5</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>Chinese</td>
<td>15</td>
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<td>17</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Indians</td>
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<td>4</td>
<td>1</td>
<td>1</td>
<td>1</td>
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<tr>
<td></td>
<td>30</td>
<td>34</td>
<td>23</td>
<td>8</td>
<td>2</td>
</tr>
</tbody>
</table>

Table II shows the Ethnic group and the Injection delivery time. The results show that 64% delivered between 0 to 4 hours, 31% between 4 to 8 hours and only 5% delivered after 8 hours.

Table III Cervical Dilation/Injection Delivery Time

<table>
<thead>
<tr>
<th>Cervical os dilation</th>
<th>Time in hours</th>
<th></th>
<th></th>
<th></th>
<th></th>
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<td></td>
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<td>+</td>
<td>+</td>
</tr>
<tr>
<td>os 1 tb.</td>
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<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
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<td>10</td>
<td>22</td>
<td>10</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>os 3 tb.</td>
<td>18</td>
<td>9</td>
<td>11</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>os 4 tb.</td>
<td>1</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Table III shows the relationship between the dilation of the cervical os and the injection delivery time. The data showed that the best results are obtained if the cervical os is more than 2 tbs. dilated, and the larger the cervical dilation, the shorter the delivery time.

Table IV Ethnic Group/Total Duration of labour

<table>
<thead>
<tr>
<th>TRIAL GROUP</th>
<th>Ethnic Group</th>
<th>Labour in hours</th>
<th></th>
<th></th>
<th></th>
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<tr>
<td></td>
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<td>+</td>
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<td>1</td>
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<td></td>
</tr>
<tr>
<td>Indians</td>
<td>1</td>
<td>7</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
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<td>19</td>
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<td>6</td>
<td>1</td>
<td>4</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>CONTROL GROUP</th>
<th>Ethnic Group</th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>24+</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0 - 4</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
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<td>5</td>
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<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Chinese</td>
<td>-</td>
<td>17</td>
<td>31</td>
<td>15</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indians</td>
<td>-</td>
<td>2</td>
<td>6</td>
<td>1</td>
<td>2</td>
<td>-</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-</td>
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<td>40</td>
<td>21</td>
<td>9</td>
<td>2</td>
<td>5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table IV shows the duration of labour in the control group and in the trial group. Although the injection Algaphan was given early in the first stage of labour (os 1 tb., 2 tbs., 3 tbs., or 4 tbs.) in the trial group, 17% had labour lasting 0 to 4 hours and the control group had not a single case with labour lasting less than 4 hours. Further in the trial group, 48% had labour lasting 4 to 8 hours compared to the control group which had only 24%. Table IV clearly shows that the total duration of labour was reduced by Algaphan given in the first stage of labour.

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Table V Blood loss in 3rd stage

<table>
<thead>
<tr>
<th>Blood loss in ozs.</th>
<th>0 – 5</th>
<th>6 – 10</th>
<th>11 – 15</th>
<th>16 – 19</th>
<th>20+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial Group</td>
<td>73</td>
<td>20</td>
<td>6</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Control Group</td>
<td>69</td>
<td>22</td>
<td>6</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

Table V shows the blood loss in the third stage in the trial and control groups. The third stage was managed in all cases by intramuscular syntometrine with controlled cord traction. From the table it can be noted that the blood loss was slightly reduced in the patients who had Algaphan in labour.

Table VI Apgar score of the baby at birth

<table>
<thead>
<tr>
<th>Apgar score</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
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<tbody>
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<td>Trial Group</td>
<td></td>
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<td></td>
<td></td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td>56</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>Control Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5</td>
<td>3</td>
<td>3</td>
<td></td>
<td>55</td>
<td>34</td>
<td></td>
</tr>
</tbody>
</table>

Table VI shows that injection of Algaphan in labour did not affect the baby as shown by the Apgar scores at birth.

Table VII Type of delivery

<table>
<thead>
<tr>
<th></th>
<th>Spontaneous vaginal delivery</th>
<th>Assisted Breech delivery</th>
<th>Forceps delivery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial Group</td>
<td>90</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Control Group</td>
<td>90</td>
<td>4</td>
<td>6</td>
</tr>
</tbody>
</table>

Table VII shows that 90% had spontaneous vaginal delivery.

Discussion:

Algaphan (generic name D-propoxyphene) has the following structure: -4-dimethylamino-1, 2-diphenyl 1-3 methyl 2 propronyl-oxbutane - hydrochloride. The dosage recommended is 2 ml. which contains 75 mgm D-propoxyphene.

The clinical trial at the General Hospital, Kuantan, had shown it to be a safe drug which is useful to shorten the duration of the first stage of labour. The best time to give it is when the cervical os is more than 2 tbs. dilated.

Algaphan given in the early stages of labour has the following beneficial effects.

(a) Reduces the duration of the first stage of labour.
(b) Alleviates the labour pain.
(c) Shortens the second stage of labour.
(d) Has no effect on the baby at birth.
(e) Has no effect on the third stage of labour.

Acknowledgement

I wish to thank the staff of the Department of Obstetrics and Gynaecology, General Hospital, Kuantan, especially Dr. S. Radakrishnan, Sisters Sharifah Ummi Khalsom, Alma Azizah Ishak and Rogayah Mohd. Nor for the clinical and nursing care of the above patients.
Book Reviews

LECTURES IN MEDICINE

THIS EXCELLENT BOOK conveniently fills in the gaps in the normal medical student's knowledge of medicine, usually based on textbooks which are generally not known for being up to date on the more recent aspects of medical advances.

Topics dealt with are of current interest and importance, lucidly written and clearly explained. I should imagine that all medical students would benefit from reading it and so would residents and doctors who are looking for concise explanations of some of the more important and recent medical topics that are not often found together in one book.

I agree with the selection of most of the subjects which, in my experience, are the ones that most physicians find are not properly understood by their residents and this handy little book definitely fills a long felt need.

Lim Kee Jin

PROCEEDINGS OF FOURTH ASIAN-PACIFIC CONGRESS OF CARDIOLOGY

THIS ISSUE (VOL. 5 NO. 4) of the Israel Journal of Medical Sciences contains the proceedings of the fourth Asian-Pacific Congress of Cardiology which was held from 1-7 September 1968 at Jerusalem and Tel Aviv, Israel.

There is a representative collection of papers from all over the world, and apart from the significant contributions of Japan and Israel, there is a sad paucity of Asian effort. Some of the papers are highly scientific and abstruse in their content but they convincingly demonstrate their importance in the elucidation of clinical phenomena. But there are also good epidemiological and clinical studies on preventive aspects of cardiac diseases. Most of the important modern trends in cardiology are mentioned, and the comprehensive nature of the congress is impressive and enlightening, apart perhaps for the cardiac surgical field which was rather inadequate. Generally, most of the papers are of a high standard and well edited and will be of interest to all clinicians, particularly those engaged in cardiology.

The various papers are neatly classified under different headings and the index reference makes it easy to look up any subject of special interest. The quality of the printing, diagrams and ECG reproductions are excellent. The volume is in a soft paperback binding and is priced at $2.50 (US).

This book is a recommended addition for all those interested in cardiology, particularly in Asia.

V. Thuraisingam


THIS BOOK, first published in 1941, provides a complete and comprehensive training course in first aid which can readily be understood by the beginner as well as by the more advanced student.

The sixth edition has been extensively revised by Surgeon Rear-Admiral Stanley Miles to include recent technical advances in cardiac and respiratory resuscitation and the treatment of shock and burns.

The growing publicity in recent years associated with accidents on the roads, in the factory, at home and at play is arousing the public conscience and increasing the demands for first aid training. The subject is of concern to a section of the population and many organisations within the community, such as, for example, the armed forces, the police, the fire services, the ambulance services, the St. John's Ambulance Brigade, Red Cross Societies, will greatly benefit from the use of this book. It should also be useful to all doctors called upon to help in giving first aid classes to members of the public.


THIS IS A COMPANION VOLUME to Bailliere's

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Handbook of First Aid reviewed earlier. In the Foreword, Norman Carpenter, chairman of the Medical Commission on Accident Prevention, points out that this book has been written to encourage and assist the doctor in the teaching of first aid. It fills an important gap between the first aid manuals and the larger books on medical and surgical emergency treatment. The lay public, even those highly qualified in first aid, look to the doctor for guidance in this essential subdivision of medicine and this book shows what should be taught and how it should be taught.

ORTHOPAEDIC SURGERY IN SOUTH AND EAST ASIA


THE PATTERN OF ORTHOPAEDIC diseases, as described in standard texts, is so different from that found in this part of the world that this is a welcome contribution for local surgeons and medical students by Professor R.D. Gunn who was for many years Professor of Orthopaedic Surgery in Singapore. The author presents the subject in a clear and concise manner and has copiously illustrated it with 255 figures of line drawings, photographs and X-rays.

All medical practitioners who have to deal with orthopaedic conditions and medical students will find it a useful tool.


THIS SMALL PAMPHLET by the Senior Nutrition Officer of the IMR serves as an introduction and a source of reference to food and nutrition with emphasis on foodstuffs and nutritional problems of Malaysia. It gives, in simple language, by reference to local articles of diet, the carbohydrate, protein, fat, vitamin and mineral requirements to maintain good health. There is also a recommended daily dietary giving the total calories, proteins, minerals and vitamins suitable for Malaysians of both sexes and all ages.

There is also available a Malay version of this bulletin. Medical practitioners will find it a convenient pamphlet to pass on to their more intelligent patients when advising them on their dietary habits.

INFORMATION FOR AUTHORS

Manuscripts

All papers should be submitted in duplicate and addressed to Dr. A. A. Sandosham, Honorary Editor, Medical Journal of Malaya, Malayan Medical Association, 26–18, Jalan Pekeliling, Kuala Lumpur. It is understood that papers submitted for publication have not been printed elsewhere. In exceptional cases, such papers may be considered if prior permission has been obtained by the author from the Editor of the journal in which it was first printed.

Manuscripts, written clearly and concisely, should be typed on one side of the paper with double spacing, giving wide margins. An indication should be given roughly as to where in the text the Tables and Figures are to be inserted.

Format

The format in general should be as follows: Title; Author’s name and degrees followed by his address; Introduction; Material and Methods; Results; Summary; Acknowledgements; References. Scientific names should be underlined. References should be given only when cited in the text, in alphabetical order, in the following form: Surname of author(s), initials; year of publication; title of paper; title of journal (abbreviated according to the World List of Scientific Periodicals and underlined); volume number double underlined; first and last page numbers of the work cited.

Illustrations

Illustrations and Tables should be in Indian ink or separate sheets of thick, smooth, white paper or Bristol board or in the form of photographs printed on glossy paper. Printing in colour may be undertaken at the author’s expense where black and white illustrations will be found inadequate. Legends to text figures or Plates should be typed on separate sheets.

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