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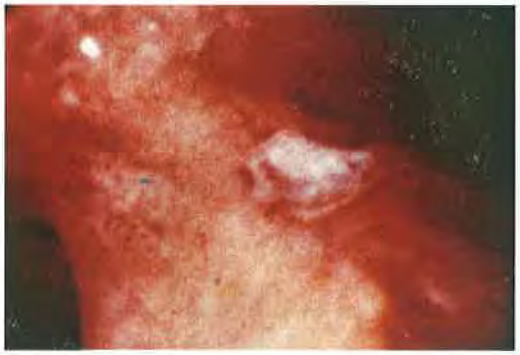

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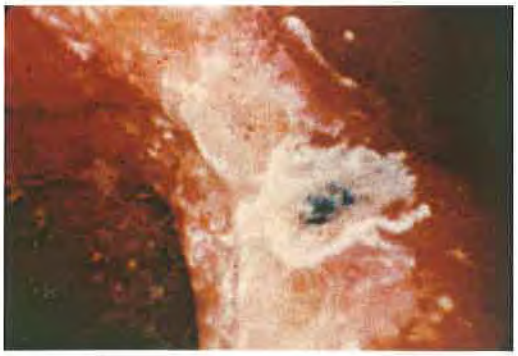

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Editorial
Snobbery — A medical approach

In our anxiety-laden haste to snub out snobbery in the schools, have we not overlooked certain fundamental facts of child-psychology and failed to assess and evaluate the negative effects of the anti-snobbery campaign?

Jean Piaget's Findings

The social climate in a primary school just cannot sustain snobbery in the sense that adults use the word. Innocent boasting, self-opinionated bragging and egoistic comparison there may be in children of this age-group — six to twelve years of age. They are part and parcel of the thinking processes inherent in the intellectual development of children. Jean Piaget who has been charting the intellectual development of children calls this phase of growth, the phase of "concrete operations". Conceptualization in the adult sense has not taken place. This phase of intellectual development is characterized by egocentricity. Egocentricity is definitely not snobbery. To impute snobbery in a primary school child is to overlook the psychology of his intellectual development. A child who does not brag, boast or speak in terms of "I" and "My" is probably suffering from some form of brain damage. We do not impute lust in a child because his sexual development has yet to develop. Why do we impute "snobbery" in him when his intellectual development has yet to acquire conceptualization?

The Role of Schools

A person who cannot feel comfortable in the presence of others who are more gifted than he, more intelligent than he or more wealthy than he, has the greatest need for corrective education. In our society, the rich co-exist with the less rich, the gifted with the less gifted and the more intelligent with the less intelligent. These are the facts of life. The sooner a person comes to terms with these realities of life the better it is for his mental health and stability. There is indeed no better place to learn these facts of life than in a school and there is perhaps no better period of life to learn them than school-life. A school worthy of its salt will help its students to recognise and cherish those values and attitudes which contribute to social harmony. Students need to learn tolerance — even to flamboyance and eccentricity in order to realise and create a cohesive nation free from petty bigotry.

Sensitization for Hurt

We cannot afford to have a situation where every one is preoccupied with snobbery — real or imaginary. We cannot have every person evaluating another person only in monetary terms and then accusing him of snobbery if perchance his nett worth exceeds his own. This form of sensitization for hurt is definitely unhealthy and an overzealous desire to snub out snobbery may result in snubbing out the psychological adjustment which everyone has to make if he is to live in harmony with all — the rich, the gifted, the intelligent and the fortunate. In a society like Singapore, harping on the differences between individuals, groups or sectors in monetary terms or in terms of human foibles or failings, does not contribute to harmony. It is the similarities and other binding factors that should be stressed. Sensitization for hurt and envy is a potent divisive factor which we cannot afford to have.

Little Sneaks?

Some of the rules and regulations formulated to snub out snobbery require that every student be the eyes and ears of the authorities. Are we not creating an environment similar to that of Red China during the period of the "Red Guards"? Little children with their red pocket books were indeed the eyes and ears of the authorities because this was the "respectable" thing to do and perhaps the most rewarding thing as well.

A Chinese Lesson

The etymological roots of the Chinese word

"stench" (臭) is interesting. It is derived from two words meaning "self" (自) and "cur" (犬). I suppose that when a person behaves like a cur – loud-mouthed and self-displaying, he is a veritable snob and this behaviour certainly stinks (臭). However, when such display is done virtuously (善), it takes on a different scent. Thus, (善 臭) means "sweet smelling". The moral of the lesson is obvious.

Crucify Him

Throughout the history and affairs of man, punitive measures are easier to pursue than any other known corrective measures. The fact that punitive measures always bring out the worst in man has never been a sufficient deterrence. The incessant cry of every age of mankind has been "Crucify Him!". Will we never learn that force is itself snobbery?

Utopian Dream

A society devoid of snobbery cannot be human. It could of course be an Utopian dream. In every society, at different times, in varying proportions and expressed in vulgar or subdued tone and intensity, different varieties of human snobbery are exhibited. These may be the snobbery of power, social snobbery, intellectual snobbery, moral snobbery and other less well defined but distinctively human snobbery.

Snobbery of Attire

Archenholtz had this to say of the attire of two professional classes, theology and medicine. "The enormous elongated wig, the stick with its great shapeless gilt knob, the satin coat and the haughty mien of the doctor are no more to be seen, as the new disciples of Aesculapius appear in the fashionable curled coiffure, dressed in the latest mode and carrying walking sticks, and by their gallant manners they try to charm their patients. The theologians used to wear a mountain of hair on their heads, had black robes, serious looks and above all a worthy appearance; now they have a double row of curled locks, well covered with pomade and powder, leather breeches, boots, laces at their breast, very fine handkerchiefs and diamond rings." These were the attire of two of the most noble professions in England in the 18th Century. Snobbery certainly has a long history.

Snobbery of Sex

Until 1869, the snobbery of the male sex prevented the admission of females into the medical profession. Sophia Jex-Blake was credited as the first member of the fair sex to agitate for the admission of women to the study of medicine. Finally seven girls were permitted to attend medical lectures at Edinburgh University. On their first day, they were received by the male medical students and their hired mob with the vilest abuse and pelted with mud. It was only in 1876 that the Russel Gurney Act recognised the right of women to study medicine.

Snobbery of Race

In his book Pax Brittanica, James Morris wrote:– "In the Gold Coast (Ghana), where a sizeable class of educated Africans existed, it had been decreed in 1893 that a third of the doctors should be Africans, but the system was abandoned – it was 'pretty clear to men of ordinary sense', Chamberlain himself commented, that British officers could not have confidence in native physicians." This was at the height of British imperialism and such snobbery had understandably the colonial secretary's endorsement. Can power be free from snobbery?

The Apple

Having taken the apple from the Garden of Eden, can man ever be all virtue and no vice? Commonsense dictates that if snobbery cannot be done away with, the least that can be done is to minimize its effects or much better, to turn it to advantage by desensitization. This then is the rational medical approach to the whole problem of human snobbery.

Santayana's Famous Dictum

Was it not snobbery on the part of the Royal College of Physicians and desensitization that led to the formation of the Royal College of Surgeons? Again was it not snobbery on the latter's part that created the Royal College of Obstetricians and Gynaecologists? Yet again, was it not snobbery of all these colleges that gave birth to the Royal College of General Practitioners? History repeats itself thus fulfilling Santayana's famous dictum that those who forget the past are doomed to repeat it.

History has taught us that snobbery need not be a disruptive force. The reaction to it need not be one of alarm. With appropriate response and desensitization, it can be turned to good advantage. It is undoubtedly the irritation that engenders the pearl. There are good reasons to believe that the College of General Practitioners, Singapore is a pearl of that nature.

L.V.C.

(Views expressed in the Editorial are not necessarily the official views of the College.)

Behavioural problems in childhood

Dr. E.K. Koh,
F.R.C.G.P., F.C.G.P.S.

"All children have behaviour problems. All parents have behaviour problems, and all teachers have behaviour problems."

R.S. Illingworth.

Behaviour problems in children are not at all uncommon. They result from a conflict between a child's developing personality and that of his parents, teachers, siblings or other children with whom he comes into contact. It forms part of the normal pattern of growing up.

Each child however learns to face up to his problems in his own fashion and in his own time. When he is able to cope with his problems in a manner acceptable to his parents and others around, no behavioural problems exist. When he fails to do so, behavioural problems result.

In dealing with children we have to remember that every child is an individual in his or her own right. Children behave and react differently according to their socio-economic backgrounds and experiences. Often what poses as a problem to one group of children may not constitute a problem to another. There are no simple text book answers that fit all cases.

The causes of destructive influences in childhood are too many to be fully discussed in this brief outline. Sula Wolff's excellent book 'Children under Stress" should be read by those who are interested and wish to know more.

In brief amongst the many and varied causes of destructive influences in childhood, the following could be regarded as being of some importance, rejection by parents, bereavement and other grief situations, illegitimacy, unsatisfactory or unwholesome peer group relationships, hostile home environment, low economic status of family with poor educational background, and difficulty in adjusting to work and life at school.

Middle Sibling Syndrome

There is one group of children whose problems are not so well known and who deserve some attention. It is not that this group is more important than the others, or that its problems are more difficult to cope with. It is only because this group is often unrecognised or forgotten. This is the group of children with problems I call the middle sibling syndrome. These are the children who are in the in-betweens in the family, they are neither the eldest nor the youngest of the children. Their problems often go unnoticed because they are unrecognised.

It is not what has been done to them that is so terrible, it is what has **not** been done for them that could be so destructive. The sad thing about the middle sibling syndrome is that mental trauma suffered in childhood frequently reaches out even into adult life.

Alfred Adler in 1928 was amongst the first to call the importance of birth order to the attention of medical psychologists. Since then much work has been done and published on the subject. It is a complex field of study and a controversial field of research better suited to medical statisticians and psychiatrists than perhaps the ordinary GP like myself. Price and Hare have shown well the pitfalls in studies which show statistical bias and ignore variables.

How do we recognise the middle sibling syndrome? The child brought in of course is neither the eldest nor the youngest child in the family. Frequently he is brought in because of some psychosomatic complaints like abdominal pain, vomiting, anorexia, enuresis, headache, rash or asthma, to which the G.P. could really find no cause. When one set of symptoms is relieved, this would be replaced by another set within a short space of time. This could go on until both the mother and the G.P. are at their wit's end. If we go a little into the behavioural history of the child, the mother would admit that the child is regarded "stubborn" or aggressive at home.

Alternatively instead of an aggressive child we may find a quieter withdrawn child who has been regarded as "unco-operative" by the other members of the family. The aggressive child is looked

upon as naughty and "impossible", the withdrawn child is often scolded for crying at the slightest excuse and with little provocation.

In both cases what is regarded by parents or teachers as naughtiness or anti-social behaviour of the child is nothing but a pathetic cry for love and attention.

It seems paradoxical that if these children are unable to get the love and attention they want, they often go on to make such a nuisance of themselves that they invite attention of a different kind — a whack in the pants from parents whose patience has been sorely tried.

Although this form of summary justice may seem a bit hard on a child needing love and attention, to withhold punishment would be to ignore the child's cry for attention. The parental-child bond may be even strengthened by punishment if it is fairly meted.

It is important for the child to understand that punishment does not mean rejection. Love without discipline may be bad, but discipline without love is equally disastrous.

The extent to which children in a family could suffer from the middle-sibling syndrome depends on many factors. Some of these are the size of the family, the extent of parental-child interaction, the economic status of the family, and the sex or age disparity between the siblings.

What makes the middle sibling syndrome different from other forms of sibling jealousy? Sibling jealousy shown by the first born on the arrival of a new baby does not last for ever. With the middle sibling syndrome the need for love and attention seems to be difficult to placate. There appears to be a deep-rooted sense of insecurity which needs a lot of attention and re-assurance.

Most parents may find that placating or spoiling these children do not always provide the answer to the problem. In fact strict discipline may be called for, and over-indulgence can be harmful. There must of course be love together with discipline and it is important that the child should get this message.

If the middle sibling syndrome is not recognised, the insecurity arising from this often persists even into adulthood. Many adults have emotional problems which they themselves do not relate to this. Some adults who have been denied things that went usually only to the oldest or youngest child when they were young, often become compulsive buyers when they become adults. Food fads is another way in which a middle sibling could use as a weapon to attract attention within the family, and these fads often persist into adult life.

The problem amongst Singapore children.

In Singapore amongst the conservative families Eastern cultural traditions are strong, and parental authority has never been challenged by the young. The generation gap exists, but this has been accepted by both sides. Discipline is strict and surprisingly enough there is little resentment by the younger generation. This has meant that our young people have grown up to respect authority and discipline and the numbers who pose problems to society by being delinquents and drug-takers are much less than in most Western cities of similar size.

Eastern families are usually extended families and with many aunts or grandparents acting as surrogate parents, middle siblings do get a fair measure of attention if not from their own parents, at least from some other adult around the place. The strict emphasis on discipline too also means no child is ever ignored. If a middle sibling is not given first class attention in the family, he can at least be assured that third class attention will speedily be administered if he becomes "impossible" in his behaviour.

With rapid urbanisation and economic progress the nuclear family has replaced the extended family in many of the urban housing estates in Singapore. The need for mothers to go out and work to help supplement family income in order to achieve a "higher" standard of living, has also meant that many parents are now unable to give the children all the attention they need. Where families are large and parents are unmindful or unheedful of the attention sought by their children, behaviour problems are beginning to set in.

The Report of the official Committee on Crime and Delinquency in Singapore states that, "studies show that delinquent youth comes from large nuclear families with more than five children per family." Over 70% of the sample studied belong to this group.

Singapore lies at the confluence of Eastern and Western cultures. In recent years influence of Western cultural patterns have brought on a slow erosion in Eastern cultural norms especially amongst the Western educated. In these families parental authority is nowadays not always unchallenged and the young are often keen to assert their own personalities. However even in these families, the Eastern cultural bond is still strong and absolute defiance of the senior generation is rare.

We realise we cannot ignore socio-cultural influences that will shape the coming generation. We owe it to our children and ourselves to heed their problems thereby preparing them to-day

for the adult world they will inherit to-morrow. The Chinese have a saying which goes,

"You can bend the young sapling,
but you cannot shape the old knotted pine."

Every child with a problem has a right to be heard. Every child that seeks love and attention seeks that which is only his due. Children should not only be seen, they should be heard as well.

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Hyperpyrexia in children and pitfalls in diagnosis

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DEFINITION

There is no magical figure which denotes a temperature above which it can be termed "hyperpyrexia". A better definition is the level of temperature above normal which an individual paediatric patient has, which is endangering his life. The doctor, therefore, must decide whether the temperature level is "hyperpyrexic" or not. In children, we take 102°F or 38°C as the level at which children may get complications, like convulsions. Of course some children with a low threshold may get fits below this figure.

Regulation of Body Temperature

The normal body temperature is 97°F to 98°F (36°-37° centigrade). In health, it is always kept fairly close to this level by maintaining a balance between heat gain and heat loss.

Heat gain

Heat gain is due to:

(a) heat produced in the body &
(b) heat taken under certain circumstances from the environment.

Heat loss

Heat is lost from the body in several ways:

(a) by **radiation** from the body to cooler objects at a distance.
(b) by **conduction** and **convention** to the surrounding atmosphere if the temperature is lower than that of the body. The air in immediate contact with the body is warmed.
(c) by **evaporation** of water.
The essential fact to remember is that when 1 gm of water is converted into water vapour, 0.58 calories of heat is needed and has to be taken up from the environment, and this heat is known as the latent heat of vaporisation.
Thus when 1 kg. of water evaporates 580 calories are taken from the immediate surroundings. Evaporation of water takes place from the lungs and from the skin. Evaporation of **sweat** is the principal means of heat loss when the body temperature tends to rise.

Syndrome of Heat Stroke

The syndrome of heat stroke develops in people working in hot environments. The symptoms are due to hyperpyrexia, salt loss and dehydration, in varying proportions. As the external temperature is high, heat cannot be lost by radiation and convection. If in addition the air is moist and still, evaporation cannot take place either. When the heat regulating mechanism breaks down, the body temperature begins to rise. A vicious circle is thus established with ever-rising body temperature and consequently increasing metabolism and increasing heat production. Circulatory failure develops and seizure and permanent injury to the central nervous system may occur.

Danger of Hyperpyrexia

It is stated that a febrile reaction is a good thing and that it serves as one of the defensive tools of the patient. Doubtless, it serves a useful purpose so long as one of the defensive thermostatic control of the intact temperature-regulating centre is intact. However, when it goes haywire and by itself causes pathology, the high temperature is inimical to the individual. We see in children innumerable cases of fits, with hyperpyrexia, fits which refuse to be controlled by large doses of anticonvulsants almost to the point of respiratory and circulatory failure, and the patients subsequently dying still with hyperpyrexia and uncontrolled fits. On autopsy, no specific lesion is found in the brain except for some oedema due to the fever and the fits. Quite a number of these are due to viral infections.

These "hyperpyrexia fits" are more common in Singapore than in Western countries mainly because we are living in a tropical zone, and "heat-waves" are common during the "hot months".

Thus, it seems logical that hyperpyrexia must be controlled in children.

I Methods adopted for cooling temperature of the body. Temperature of less than 104°F. Ice-cold sponging

(a) sponge the whole body with ice-cubes (use ice cubes in water)
(b) keep the body wet since evaporation of excess water helps to lower the body temperature
(c) direct electric fan on the body. If shivering occurs, use "lytic" cocktail
(d) take rectal temperature at 15 minutes intervals initially, than half-hourly if temperature is falling.
(e) If temperature is above 105°F take rectal temperature at 5 minute intervals.

Note

(a) Tepid sponging (using tap water) is ineffective in the tropics for high temperatures especially in children.
(b) There is no danger in lowering the body temperature more than 2 degrees F at a time. In fact the object is to lower the body temperature to normal or as near as normal as possible.

II Temperature of 104°F or more & rising

In addition to the above, place icebags as an emergency measure over:—

(a) great vessels, both sides of the neck, axillae and groins.
(b) heart

Remove the ice-bags one or two at a time for 1 minute (to allow for recirculation of blood in the skin under the ice bag) and replace them at the same site. Do not leave ice bags for longer than 5 minutes.

III "LYTIC" Cocktail

"Lytic cocktail is given intravenously in a drip to prevent shivering (which may cause a further rise in body temperature) when the patient is under Procedure 1 and II.

Composition (depends on Doctor's order)

Chlorpromazine	(Largactil)	50 mgms
Promethazine	(Phenergan)	50 mgms
Meperidine	(Pethidine)	100 mgms
Dextrose 5% to make up to		50 ml.

Dose

Initial 0.15ml per lb body weight to be repeated twice again at 20 minute intervals.

Maintenance

0.15ml per lb body weight to be given at intervals of 3 to 7 hours.

IV Oxygen as indicated.

V Antipyretics

Use syrup paracetamol one or two teaspoons t.d.s. depending on the age and body weight of the child. Aspirin is a gastrointestinal irritant and is to be avoided in children.

For severe hyperpyrexia use intramuscular metamizol*

Remember that antipyretics alone will not act unless simultaneous methods of cooling the body are applied, i.e. fan, sponging, ice etc.

Some Archaic Ideas about Body Cooling

Certain old-fashioned ideas about cooling the body have militated against saving such patients with hyperpyrexic fits (it must be understood that it is a certain type of paediatric patient we are focussing our attention on viz. those whose fits are due to hyperpyrexia and the fits were unable to be controlled by anticonvulsants). These archaic ideas have been summarised as follows:

(1) **That water used for cooling the body should not be too cold and that tap water should be used.**
The answer is that the tap water as advocated in Western textbooks has a lower temperature than the tap water here and that the body can be cooled and in fact has to be cooled by placing the patient in a tube of ice-chips and cold water to bring the temperature to low levels for hypothermia without any ill-effects.

(2) **That after sponging, the body should not be too wet**
In fact it has to be wet if the temperature is to be brought down by evaporation.

(3) **That directing a fan towards a hyperpyrexic patient is contraindicated**
Without a fan, evaporation cannot be effective and hence lowering of temperature by evaporation is defeated.

(4) **That vigorous methods of body cooling will cause Pneumonia**
This is due to wrong conclusion being drawn from a patient exposed to rain etc. without medical attention and food etc. A patient being cooled for hyperpyrexia cannot get infective pneumonia because he is always under

* This product has been banned in Singapore — Editor.

antibiotic cover and cooling has been and is being used for patients with pneumonia itself. In the latter instance, lowering the temperature conserves oxygen utilisation and aids more effectively in the fighting against the pneumonia.

(5) **That the temperature should be brought down only 2°F at a time**

In fact temperature should be brought down as early as possible from whatever high height it was before to even below normal temperature (hypothermia) without any ill effects so long as efforts are made to prevent shivering and vasoconstruction. Obviously if such a hyperpyrexic patient with a temperature of 106°F is brought down to 104°F, (i.e. 2°F down) then he will still die at 104°F because we have seen such patients dying at 100°F to 101°F. The temperature must be brought down at least to normal levels.

(6) **That cold compresses to the head seem to be the most effective procedure**

Cold compresses to the head are not effective enough, and the best method for bringing temperature down by conduction is applying cold compresses over the site of the great arteries, such as the neck, axillae and groin.

Summary of Treating Hyperpyrexia with Fits

(1) I/V drip is set up — source of fluids.
(2) Body cooling by cold ice sponging and ice over big vessels and direct fan for evaporation.
(3) Lytic cocktail if temperature above 104°F and if shivering occurs.

Nursing Requisites for Cold Sponging

(from nursing lectures to paediatric nurses).

Cold sponging is the sponging of body with water to bring down hyperpyrexia (39°C and above).

Requisites

Top Shelf

Trolley with basin, eight face towels, jug of water, bowl of ice, bath thermometer.

Bottom Shelf

Clean linen, long mackintosh, pail for soiled linen.

Nursing Action

Preparatory Phase	**Rationale**
1. Explain procedure to patient.	
2. Ensure privacy.	
3. Remove patient's blanket, drawsheet and mackintosh.	
4. Line mattress with long mackintosh, if there is no protective cover.	
5. Cover patient with drawsheet and remove pyjamas.	To prevent shivering.
Performance Phase	
1. Wash and dry face.	
2. Apply cold compresses to forehead.	Relieves headache.
3. Apply cold compresses also to the neck, axillae and groins. Renew compresses at frequent intervals.	These areas contain large blood vessels and this enhances heat loss through conduction.
4. Wet sponge arms, trunk and legs, leaving a film of water on the skin to evaporate.	Heat is lost through the process of evaporation.
5. Use long sweeping downward strokes for limbs and circular movements for trunk.	Friction increases heat production.
6. Add ice to water as often as possible when temperature above 40°C.	
7. Turn the patient and wet sponge his back.	
8. Watch for shivering throughout the procedure.	Shivering increases heat production and thus body temperature. This occurs when the rate of heat loss is greater than 20°C per hour.
9. Dab dry the patient.	

Note: The duration of sponging should not exceed 25 minutes. Allow 3 minutes for each limb and 5 minutes each for front and back of trunk.

Follow-Up Phase

1. Change bed linen, dress patient and make him comfortable.	
2. Switch on fan beside the patient.	To enhance heat loss through convection.
3. Clear away requisites.	
4. Using the same route check the temperature 30 minutes later.	To note the effectiveness of sponging.
5. Report and record temperature in clinical chart.	

CAUSES OF HYPERPYREXIA AND ITS PITFALLS

It is easy if a hyperpyrexia fits into a nice clinical diagnosis but often it is not easy to identify the cause immediately and, therefore, the diagnosis becomes a "hyperpyrexia of unknown origin". There are about ten causes of pyrexia of unknown origin in this country.

I Viral Infections

1. The commonest infection in children in the toddler age group is an **upper respiratory tract infection** but they seldom present a problem in diagnosis because of their short duration, usually 5 days or less. The commonest viral infections are the common cold, adenoviruses, rhinoviruses and during epidemics, Haemophilus influenzat infections. The following hyperpyrexia may be missed in the early stages:

a. **Haemorrhagic fever**
Occasionally fever may go beyond 5 days but the characteristic confluence of fine petechiae with thrombocytopenia and atypical monocytes with leucopenia would alert one to the correct diagnosis.

b. **Encephalitis**
Even viral encephalitis can come in without encephalopathic signs in the beginning. We have seen cases even with a normal CSF and after 2 days the CSF was typical of a viral encephalitis.

c. **Measles**
Measles always begins as a respiratory tract infection until the classical mobilliform rash and koplick spots appear. However, sometimes the steps of koplick spots may be missed because it is so transient.

d. **Infectious mononucleosis**
Here the enlarged tonsils with a white membrane on the surface together with hepatosplenomegaly gives the diagnosis away. The peripheral blood film will show the atypical mononuclear cells.

II Pyelonephritis

This is one of the most missed diagnosis for the simple reason that especially in infants, the only way to diagnose the condition is to find abnormal numbers of pus cells in the urine.

Dysuria, disturbances of frequency of micturition, etc. are not complained of in the younger age group. However, the clinical presentation may be of 3 types,

(a) abdominal type — fever with vomiting, diarrhoea and abdominal pain.
(b) pulmonary type — fever with breathlessness.
(c) C.N.S. type — fever with convulsions.

Pyelonephritis is often missed because the urine is not examined often enough in cases of gastroenteritis, febrile fits etc. It seems more practical to examine a fresh specimen of urine and to do a count in a counting chamber as for T.W. and it was found that greater than 50 pus cells mm^3 is significant. This method is rapid, reasonably accurate and can be done by any doctor.

It is stressed again that it is highly important to diagnose the condition and treatment must be adequate and prolonged (about six weeks) to prevent chronic nephritis with hypertension later on in life.

III Purulent Meningitis

A premature infant and sometimes even full term infants may present with fever and vomiting or only with fever and lethargy with an anorexia and yet have septic meningitis **without** the following signs:

(a) fits
(b) tense fontanelle
(c) neurological signs
(d) stiff neck and Kernig's sign

The absence of a bulging fontanelle, a most useful sign at this age is due to some degree of dehydration. Many cases may be missed and in premature babies, fever may be absent. They may present with lethargy and failure to feed. Thus, the only way to diagnose the condition is by lumbar puncture. Delay in diagnosis of this condition is very dangerous as early and adequate treatment of purulent meningitis in infancy is followed by full recovery, while delay in treatment will result in death or physical and mental crippling.

IV Collagen Disease

The commonest collagen disease presenting with fever and almost no other signs is rheumatoid arthritis in the 2 to 3 year age group. The temperature in such children is hectic. Sometimes there may be no enlarged glands or a palpable spleen but those cases presenting as a P.U.O. have no joint involvement at this stage. This pre-arthritic fever may last for as long as a month or two before joint swelling reveals the diagnosis finally. The E.S.R. will be high. Treatment with steroids brings the fever down in 24 hours.

V Tuberculosis

Tuberculosis giving rise to fever without much clinical signs to go by in almost confined to uncomplicated primary complex at the time of conversion and a Mantoux test will settle this. Occasionally, a miliary tuberculosis picture may be present radiologically without any clinical signs in the chest. Here, an awareness and an X-ray of the chest would reveal the diagnosis.

VI Subacute Bacterial Endocarditis

This is not common in children here but must always be considered especially if cardiac signs are present, and repeated blood cultures often settle this.

VII Neoplastic Conditions

The commonest neoplastic condition with presentation of fever without much clinical signs is acute leukaemia in the early stages. An awareness of the condition and a meticulous palpation of the spleen and the detection of petechiae with a careful examination of the peripheral blood would prevent misdiagnosis, and a bone marrow aspiration is necessary to confirm it.

VIII Typhoid

Often this is missed in the first week as the spleen is not palpable, but it is always thought of in the differential diagnosis of a pyrexia of unknown origin if the fever persists for over one week.

IX Hidden Pyogenic Infections

The following are often missed in the diagnosis of hyperpyrexia.

(a) appendicitis
(b) subdiaphragmatic hernia
(c) osteomyelitis
(d) otitis media
(e) brain abscess

X Psychological Causes

The last group of P.U.O.s have no known cause but the fever is mild and occurs daily. All investigations prove negative.

(a) **The periodic syndrome**
The patient gets fever in periods during which there may be headache, abdominal pain and vomiting. The temperature may be high but each bout lasts for about one week.

(b) **The thermometer syndrome**
Often the infant has a doctor or nurse for its parent and the fever is low grade, about 99°F. The patient is perfectly well in spite of the fever and the ESR is normal, appetite is normal and growth is normal. The temperature variation is normal, and depends on the efficiency of the thermoregulatory centre and in these children, the temperature centre may be immature. Such cases require reassurances.

Investigations for hyperpyrexia and P.U.O.s

(1) A thorough history and clinical examination
(2) P.B.F., T.W.C., Platelets
(3) Urine for pus cells
(4) M.T.T. for tuberculosis
(5) E.S.R. for collagen disease
(6) L.P. for intracranial lesions
(7) Blood culture and blood Widal and Weil-Felix
(8) Blood for virology – eg. adenoviruses, Japanese B encephalitis, haemorrhagic fever

Conclusion

Hyperpyrexia, a common symptom of illnesses in the tropics, must be treated efficiently by good nursing procedures and an elucidation into its cause is essential for proper therapy to be instituted.

Diabetes mellitus

Dr. Tan Bock Yam F.R.C.P.E.

A. INSULIN

Since the discovery of Insulin by Banting and Best in 1921, more knowledge about insulin has been acquired during the last 20 years and it is pertinent to make mention of this knowledge at the start of this evening's lecture. Briefly the important milestones are as follows:—

Sanger in 1956 mapped out its chemical structure and showed that insulin consists of 2 long chains of amino acids — the first chain with 21 and the second chain with 30 amino acids. The chains are linked by 2 bridges of sulphur atoms (disulphide bridges), and the first chain has in addition another 'sulphur bridge' across part of its length. There are slight structural differences between insulin from different species e.g. human, beef (bovine) and pig (porcine).

In 1963, two groups of research workers (in W. Germany and U.S.) reported almost simultaneously their ability to synthesise a material possessing insulin activity. This was closely followed by reports from workers in Shanghai of their success in the synthesis of insulin. However, over 200 separate and distinct chemical reactions are involved in the process and it will probably be some time yet before commercial production of synthetic insulin can become economically feasible.

Steiner in 1969 showed that insulin is synthesised in the beta cells of the islets as a single chain polypeptide precursor known as "PROINSULIN". This larger percursor substance also known as "BIG INSULIN" has little biologic activity. It is a single chain of amino-acids and during its storage in the beta cell, is split by a proteolytic enzyme into the double chain insulin molecule and a connecting peptide, the C peptide. Both insulin and C peptide are released in equivalent amounts into the blood and small amounts of proinsulin (no more than 5 — 15% of the total insulin in the blood) also circulate in the blood. Insulin after synthesis is stored in densely staining granules which are visible with the electron microscope. There are many theories as to how the granules discharge their stored hormones into the blood stream. The most likely is that the granules move through a system of microtubules to the periphery of the beta cell before discharging their insulin into the blood. This process requires energy, calcium and cyclic AMP. In a complicated chain of events of this kind, there are many points of possible breakdown, some of which might lead to diabetes.

A recent development has been the elucidation of the 3-dimensional structure of insulin by Dorothy Hodgkin. We now know that 6 molecules of insulin are joined together with 2 atoms of zinc to give a stable crystalline substance which is presumed to be present as the storage form of insulin in the pancreas.

The latest development is the break-through in human insulin biosynthesis using recombinant DNA and inducing the E. COLI bacteria to produce human insulin as described by H. Goodman (1979)

Mechanism of insulin secretion:

Under ordinary conditions a rise in blood glucose concentration perfusing the pancreas in the most important stimulus for insulin secretion. Stimulation of insulin secretion involves the release of the granules of preformed insulin as well as increased synthesis of new insulin. The response of the beta cell to a sudden increase in circulating glucose is biphasic.

(i) an immediate phase of insulin release within seconds from a rapidly mobilizable pool of pre-formed insulin and

(ii) a more gradual and sustained phase of insulin secretion in which pre-formed insulin in a second more slowly responsive pool, plus newly synthesised insulin and small amounts of proinsulin, are released.

The second phase depends on glucose metabolism within the beta cell. Amino acids such as arginine also stimulate insulin secretion and a pure

protein meal therefore raises the blood level of insulin. Other stimuli of insulin secretion include the GI hormones (gut glucagon and pancreozymin), pancreatic glucagon from the alpha cell, sulphonylureas (the oral hypoglycaemic drugs) and theophylline. In contrast, epinephrine is a potent inhibitor of insulin secretion by virtue of its alpha-adrenergic activity. So do drugs like acetazolamide, thiazide derivatives, phenytoin and diazoxide. Insulin release in response to glucose does not begin till the blood level has risen above 100 mgm%, thereafter the higher the blood level the greater the release of insulin.

Measurement of insulin in blood – insulin can now be accurately measured in the blood by the method of Radio-immuno-assay using the method of Berson and Yalow, and the normal range is from 10-20 microUnits per ml. In normal weight individuals, the insulin secretion matches the insulin requirements, in juvenile diabetics the insulin secretion is very low or absent and in maturity onset diabetics the insulin secretion is less than the requirement. In obese individuals, the insulin secretion and requirement are equal but at a much higher level in the obese non-diabetic than in the normal weight diabetic, while in the obese diabetic the insulin secretion is higher than even the normal weight non-diabetic but is insufficient to meet the insulin requirement. In obese individuals more insulin is required because of decreased tissue responsiveness to insulin.

The Newer Insulins:

There are 3 new additions to the insulin range of SI, PZI and IZS preparations.

1) ACTRAPID – Neutral Insulin Injection.
   This clear insulin is not acidic but neutral. It produces effects similar to SI, but its onset is slightly more rapid (hence its name) and its duration of action slightly more prolonged. It is prepared from pig pancreas and is less antigenic than the usual forms which are of the bovine type. It is useful in patients with insulin resistance.
2) RAPITARD – Biphasic Insulin Injection.
   This preparation is a mixture of 25% Actrapid (porcine) Insulin and 75% crystalline M C bovine insulin, the crystals of which fall between certain size limits to control its rate of release. Action commences from about 1 hour from the Actrapid and can continue up to 22 hours from the dissolution of the varying size crystals. The maximum effect of Biphasic insulin is produced from 4 – 12 hours.
3) MONOCOMPONENT (MC) INSULIN
   This form of insulin is produced by a method of isolation known as "Ion-exchange Chromatography" and is a purer insulin with little or no immunogenic activity. This form of insulin will be of particular benefit to those diabetics who have built up "insulin resistance". Such diabetics may require up to 1000 units or more of insulin daily to obtain control. By using MC Insulin the dosage may be reduced considerably. It is also useful in patients with Insulin Allergy and Lipoatrophy.

B. DIABETIC MICRO-ANGIOPATHY

One of the major problems of diabetes today is that of vascular complications – microvascular disease or microangiopathy. The morphological feature which best characterises diabetic microangiopathy is "thickening of the basement membranes of the capillaries" throughout the tissues of the body. The finding has been reported in such diverse tissues as the skin, skeletal muscle, adipose tissue, pancreas, peripheral nerves as well as in the eye and the kidney. The changes are responsible for such complications as retinopathy, nephropathy, neuropathy, dermopathy and peripheral vascular disease in diabetics. Both light and electron microscopy studies have shown these changes in the skin, muscle and kidneys. Muscle biopsy is more reliable than skin biopsy and certainly simpler and safer than renal biopsy. The quadriceps muscle is the best site for the biopsy.

The pathogenesis and mechanism of diabetic microangiopathy still remain controversial. Two conflicting views are presently held. One view headed by Siperstein et al. (1968) is that thickening of basement membrane precedes and may even cause CHO intolerance; is not the result of the CHO abnormalities of diabetes but rather represents an independent, and conceivably, even a primary lesion of the diabetic syndrome. They demonstrated these changes in biopsy material in at least 90% of diabetic patients and found that the changes are not related to the duration or severity of the hyperglycaemia. The other view held by Williamson et al. (1971) is that thickening of capillary basement membrane in muscle increases significantly with the duration of known CHO intolerance and increases appreciably with age. They found a definite correlation between duration of CHO intolerance and incidence of retinopathy and nephropathy.

The major vascular problems that confront the diabetic patient include:

1. Premature atherosclerosis — coronary and cerebro-vascular disease.
2. Retinopathy.
3. Nephropathy — intercapillary glomerulosclerosis.
4. Neuropathy.
5. Ulceration and gangrene of lower extremities.
6. Peripheral vascular disease.
7. Dermopathy.

C. THE ORAL HYPOCLYCAEMIC PREPARATIONS

It is now almost 20 years since the first oral hypoglycaemic preparation was used for the treatment of Diabetes Mellitus and the results published in Germany by FRANKE and FUCHS in 1955. This first preparation was CARBUTAMIDE and it was used successfully on the maturity onset stable non ketotic diabetics. It was the culmination of over 15 years of research work following the recognition of the hypoglycaemic effect of certain sulphonamide derivatives by JANBON in France in 1942. Much of the ground work was done by LOUBATIERES who showed that the hypoglycaemic action of the sulphonamide derivatives was dependant upon the presence of the pancreas. Carbutamide has now been withdrawn from clinical use locally, in England and America, because of bone marrow damage in a small number of cases, but is still being used in some parts of Europe.

The oral hypoglycaemic preparations fall into two main groups:

1. The SULPHONYL UREAS
2. The BIGUANIDES

These preparations have now been accepted as effective and safe methods of therapy, complementing the other two methods of treatment — insulin and diet. Notwithstanding the results of the prospective study by the University Group Diabetes Programme (1970) which reported that death from myocardial infarction is more common in patients treated with sulphonyl ureas than in control series receiving diet and placebo tablets or insulin, the use of these drugs have not been given up in UK or Europe nor even in the States. There are many objections to the group's interpretation of their findings. For those interested, further reading material can be obtained in BMJ (1970, 4,445), LANCET (1971, 1, 17), DIABETES (1972, 21,976 & 1035), Amer. J. of Medicine (Jan 1974).

Certain points regarding these preparations should be borne in mind:

1. The oral hypoglycaemic preparations are NOT ORAL forms of Insulin.
2. Like all drugs, these preparations are likely to cause toxic and side effects in large doses and in sensitive patients.
3. The absolute contraindications to the use of these drugs are:
   a) KETONURIA AND KETOACIDOSIS
   b) PREGNANCY
   c) MAJOR SURGERY AND SEVERE INFECTIONS
4. Use of these preparations is more successful in conjunction with dietary restrictions and after Diet alone has been tried and failed. It is not a substitute for a restricted carbohydrate diet.
5. Combined therapy using a preparation from each group or with insulin has achieved a greater degree of control than previously possible.

I. THE SULPHONYL UREAS

There are now a total of **NINE** preparations in this group:

1. CARBUTAMIDE (Invenol, Nadisan)	1955
2. TOLBUTAMIDE (Rastinon, Orinase, Artosin)	1956
3. CHLORPROPAMIDE (Diabinese)	1958
4. ACETOHEXAMIDE (Dimelor)	1962
5. TOLAZAMIDE (Tolinase)	1962
6. GLIBENCLAMIDE (Daonil, Euglucon)	1969
7. GLIBORNURIDE (Glutril)	1970
8. GLIPIZIDE (Glibenese)	1973
9. DIAMICRON (Gliclazide)	1974

The ones commonly used here are Tolbutamide, Chlorpropamide, Glibenclamide, Glibornuride and Gliclazide.

Action

The sulphonyl ureas act by:

1. Stimulating the release of insulin from the storage granules of the beta cells of the pancreas. It has been shown repeatedly that they markedly increase the plasma insulin level.
2. Reducing the output of glucose from the liver. This apparently results from a decrease in gluconeogenesis and a decrease in glycogenolysis.

Other observed effects of sulphonylureas include:

1. Potentiate the effect of insulin in depancreatised animals.

2. Decrease the degradation of insulin by insulinase, with large doses.
3. Decrease the plamsa free fatty acid level before any changes are observed in the level of blood sugar, after a single dose of sulphonylurea.
4. In the presence of actively functioning beta cells, sulphonylureas increase glucose oxidation and arterial-venous glucose differences.
5. Tolbutamide helps prevent the diabetogenic action of growth hormone.

Dose and Tablet Strength

Preparations	Tablet Strength (mgm)	Total Daily Dose Range (mgm)
Tolbutamide	500	500 – 3000
Chlorpropamide	100 & 250	100 – 500
Glibenclamide	5	2.5 – 15
Glibornuride	25	25 – 100
Gliclazide	80	80 – 240

The main difference between the various preparations is the duration of action – that of tolbutamide being relatively short and the drug is best given in divided doses, 3-4 times a day, while that of Chlorpropamide, Glibenclamide and Glibornuride are more prolonged and the drugs given once daily or at most twice a day.

As with insulin, the dose of the oral preparation is increased during an infection or illness; and, when maximum doses are used with little or poor response, then it is necessary to add metformin or insulin or to replace with insulin.

Therapeutic Use

The best response with Sulphonylureas is seen with patients in the following categories:

1. Maturity Onset Diabetes (over age of 40 years at diagnosis) who are generally non-insulin dependent.
2. Stable and non brittle Diabetes.
3. Non-ketotic Diabetes.
4. Low insulin requirements (generally below 40 units a day).
5. Uncomplicated Diabetes.

Young, juvenile diabetics who are usually insulin dependent respond poorly due to lack of functional islet tissue.

Obese diabetics are as a rule not given sulphonylureas as weight reduction is made difficult.

The sulphonylureas are best taken after meals but can be taken before meals if well tolerated.

Side Effects & Toxicity

The side effects and toxicity of sulphonylureas are low and the overall incidence reported in most large series is about 2%. When side effects develop it is important to stop therapy. Reported side effects include:

i) **Skin Rashes**

Being derivatives of sulphonamides, these preparations should not be used in patients sensitive to sulpha drugs. Pruritus, maculo-papular eruptions, urticaria and photosensitivity may occur. Exfoliative dermatitis has been reported but is uncommon.

ii) **Gastro-intestinal disturbances**

These include nausea, anorexia, vomiting, diarrhoea and epigastric discomfort. Taking the drugs after food or chewing/crushing them can reduce the occurence of these symptoms.

iii) **Blood Dyscrasias**

Leucopaenia, haemolytic reactions, thrombocytopaenia have been reported and in more severe cases pancytopaenia and agranulocytosis have occurred. Steroid therapy may be required.

iv) **Hepatic**

Toxic action on the liver may give rise to jaundice, but this usually rapidly clears up when the drug is discontinued. The jaundice is due to inflammation of bile ducts, leading to biliary obstruction – cholestatic jaundice. Chlorpropamide is the drug most likely to produce this complication, an action similar to chlorpromazine.

v) **Hypoglycaemia**

All the sulphonylureas can produce hypoglycaemia because of their pancreatic action. It can be very severe and has even led to death. Patients vary greatly in their tendency to develop hypoglycaemia, but generally the elderly diabetic is most prone, as are those who are debilitated, not eating well, alcoholics and those with impaired renal function. I have found them to be more common with the long acting and more potent preparations eg. chlorpropamide, glibenclamide and glibornuride, than with the shorter acting tolbutamide.

vi) **Intolerance to alcohol**

Alcohol can be more intoxicating than usual in those on sulphonylurea therapy. Facial flushing after taking alcohol may occur with chlorpropamide but is less common with the other preparations. This can be both embarrassing and discomforting. The reaction is rather similar to that produced by ANTABUSE – an exaggeration of the

normal response of small blood vessels to ethyl alcohol.

II. THE BIGUANIDES

These are unrelated to the sulphonylureas. The active chemical grouping is a condensed biguanide with which several side chains have been combined to form compounds with hypoglycaemic action. There are **three** main preparations belonging to this group.

1. PHENETHYL BIGUANIDE	PHENFORMIN	(D B I, Dibotin, Insoral)	1957
2. DIMETHYL BIGUANIDE	METFORMIN	(Glucophage, Diabex)	1960
3. BUTYL BIGUANIDE	SILUBIN	(D B V, W 37)	1960

Two long acting forms of Phenformin are commercially availabe:

1. DIPAR
2. GLUCOPOSTIN

The ones commonly used here are metformin, phenformin and dipar. (the lattar two are no longer used because of Lactic Acidosis complication).

Action

The way in which these drugs lower the blood sugar is not yet clearly known. In the diabetic the biguanides increase: -

1. Glucose uptake by the peripheral tissues and 2. Possibly insulin sensitivity.

There is some evidence that they impair glucose absorption, reduce hepatic gluconeogenesis and act on plasma insulin inhibitors and antagonists as well.

Whatever may be the mode of action by which the biguanides reduce the blood sugar level it is definitely known that they do not depend on the presence of functional islet tissue and do not stimulate the beta cells of the islets to release insulin as the sulphonylureas do. They do not produce hypoglycaemia in non-diabetics.

Because of their different mechanism of action, the sulphonylureas and biguanides complement one another and are used effectively when given together than when given separately. Such combined therapy are not only effective but also safer because smaller doses of each are used with a lower incidence of side effects and toxic reactions.

Dose and Tablet Strength

Preparation	Tablet Strength (mgm)	Total daily dose range (mgm)
Metformin	500	1000 – 3000
Phenformin	25	75 – 150
Dipar	50	50 – 150
Glucopostin	50	50 – 150

Both phenformin and metformin are given in divided doses three times a day while Dipar and Glucopostin are given as a single dose or twice daily. This is related to their duration of action.

Therapeutic Use

The hypoglycaemic action of the biguanides is less than that of the sulphonylureas.

Their chief uses are:

1. Given alone to the obese maturity onset diabetic in whom hypoglycaemia is achieved without further weight gain.
2. In combination with a sulphonylurea in the treatment of those diabetics whose blood sugar cannot be adequately controlled by a sulphonylurea alone.
3. In combination with insulin in some juvenile onset and brittle diabetics.

Side Effects and Toxicity

The biguanides are relatively free from toxic side effects. The main side effects are gastrointestinal in nature e.g.

1. Anorexia
2. Dyspepsia
3. Diarrhoea
4. Metallic taste in the mouth.

Other side effects include general malaise and Lactic Acidosis. The latter is more frequent with phenformin than with metformin. The production of anorexia by the biguanides may help in weight reduction in the obese diabetic. Recently, concern over the frequent occurrence of reported fatalities from Lactic Acidosis following Phenformin and D B I therapy, has led to the ban on Phenformin and D B I in many countries, including Singapore.

III. REGIME FOR ORAL HYPOGLYCAEMIC THERAPY

In the absence of the contra-indications mentioned earlier, oral therapy is offered to all diabetics attending the clinic.

All diabetics must first be treated by carbohydrate restriction. Non-overweight diabetics are advised not to take excessive CHO and fat and to omit sugar in the diet.

If no response to diet is achieved a sulphonylurea (or biguanide in the obese diabetic) is first used. Because of personal experience and easy availability. Tolbutamide is the preparation of choice. Initial dose is 500 mgm tds gradually increasing to 3000 mgm daily. If no improvement occurs a change may be made to a more potent second generation preparation like glibenclamide or glibornuride. Again a small dose is given initially, working gradually to the maximum dose. A biguanide, usually metformin, is added if no response is obtained. Dosage like before is begun from a lower dose stepping up to higher doses. If persistent symptoms, glycosuria and hyperglycaemia continue insulin is given and this may be given alone or combined with the oral preparation. Generally, combined insulin and oral therapy requires smaller doses of each than when either is given alone.

With oral therapy, a successful response is generally achieved within a month but sometimes it may not become evident till 8-10 weeks. During oral therapy, an immediate change to insulin is made as soon as ketonuria or ketoacidosis occurs. Secondary failures do occur with the oral preparation after initial successful response. When this occurs a change is made to another preparation of the same group.

D. FACTORS WHICH AFFECT THE DIABETIC CONDITION.

In the management of the diabetic patient it is important to remember that there are certain factors which may affect the control of the diabetes during therapy.

These include both medications and related conditions.

Medications:

The following drugs may raise the blood sugar level:

1. Acetazolamide
2. The thiazide diurectics — e.g. Chlorothiazide and to a lesser extent Frusemide.
3. Diazoxide (Hyperstat) used in the treatment of hypertensive emergencies.
4. Phenytoin for epilepsy
5. Corticosteroids
6. Thyroid hormones
7. Oestrogens — in oral contraceptives
8. Levo-dopa for parkinsonism.

The following drugs may potentiate the hypoglycaemic effect of the oral hypoglycaemic preparations especially the sulphonyl-ureas.

1. Salicylates
2. Sulphonamides
3. Barbiturates
4. Probenecid
5. Phenylbutazone
6. Monoamine oxidase inhibitors
7. Dicoumarol
8. Ethionamide
9. Propranolol
10. Alcohol

Certain related conditions may give rise to hypoglycaemia more easily during oral therapy and lower doses of oral preparations should be used.

1. Renal failure
2. Liver disease
3. Advanced age
4. Malnutrition
5. Deficiency of physiologic insulin antagonists e.g. Hypo-pituitarism.

Lecture given on 4th October 1974, Refresher Course, College of General Practitioners Singapore.

Management of ophthalmic emergencies

Dr Lim Kuang Hui, MD FRCS

"Trauma is one of the Captains of the Men of Death" (Duke-Elder). Truly, any traumatic condition of the eye has to be regarded as a surgical emergency for not only is the injured person alarmed by the associated cardinal symptoms of pain and sudden loss of sight but also because the loss of an eye is more grevious than loss of any other part of the body. Eye injuries may be seen in association with the following:—

Fracture of base of skull
Fracture of face bones
Laceration and open wound of eye and orbit
Enucleation of eye (Avulsion)
Superficial injury of eye and eyelid
Contusion of eye and orbit
Foreign body in eye and adnexiae
Burns (chemical and heat) confined to the eye
Injury to optic nerve(s)
Injury to other cranial nerves
Adverse effects of radiation: radiotherapy and ultraviolet light
Post operative complications: (Haemorrhage, disruption of operation wound, infection and persistent fistula)

Guidelines:—

The basic principles governing surgery apply to eye injuries. Determination of vision and examination of the globe can help to assess the extent of injury.

1) Traumatic conditions which involve a fracture may have to be managed as a head or face injury and referred to a surgical, ENT or dental unit.
2) Laceration and open wounds of the eye and orbit require immediate treatment according to established surgical principles, i.e. toilet and suture of the wounds in anatomical layers and exhibition of antibiotics to prevent infection.
3) In traumatic avulsion of the eyeball, it is never good practice to remove the eye at the time of injury. When the injured eye has to be removed at a later date consent will have to be obtained from the patient.
4) Foreign bodies in the eye and adnexae are managed according to general surgical principles. Special X-rays with a marker may be necessary to localise an intraocular foreign body before attempting its removal. In addition, the injured eye, especially when involving uvea, has to be watched for sympathetic ophthalmia.

Management of Penetrating Wounds:—

a) Prevent infection with antibiotics
b) Repair of wound in anatomical layers
c) Removal of intraocular foreign bodies
d) Watch for complications: Infection
Uveitis
Cataract
Glaucoma
Ophthalmia
Retinal Detachment.

5) Traumatic intraocular haemorrhage may occur in the vitreous or aqueous chamber. Aqueous haemorrhage (hyphaema) tends to settle with a level which can be observed in the anterior chamber. Rest and immobilization is necessary. The eye has to be padded, with atropinisation if indicated, until the haemorrhage subsides, in about 4 to 5 days.
6) Chemical burns require immediate first aid. The chemicals have to be washed away with water and irrigation must be kept up continuously for at least 15 minutes. Any subsequent treatment is generally hopeless once the offending chemicals have acted on the eye and immediate first aid must be emphasised. In the case of lime, as it will flake when water is added, it is important to remove all the lime particles using an eye ointment if necessary, before irrigation. If there is any forensic indication specimens may have to be saved for chemical analysis.

MEDICAL EMERGENCIES

Sudden Loss of Sight Associated with Pain:—

1) Migraine
2) Corneal ulceration
3) Inflammation of the optic nerve and retina
4) Orbital cellulitis
5) Glaucoma

Sudden Loss of Sight without Pain:—

1) Hysterical blindness
2) Iritis
3) Detachment of retina
4) Vascular catastrophies of the vitreous & retina
5) Amblyopia
6) Amaurosis

Immediate treatment:—

1) Sometimes migrainous neuralgia may be associated with transient defective vision: adequate diagnosis and reassurance suffice.
2) Corneal ulceration is always serious because of potential infection. Exhibition of potent antibiotics is imperative. In ophthalmia neonatorum instant application of penicillin eye-drops can save the eye from certain blindness.
3) Optic neuritis and retrobulbar neuritis present as a sudden loss of sight, often associated with pain. Although empirical, immediate steroid therapy can be effective.
4) Acute congestive glaucoma is the par excellent medical emergency. The disease is readily treatable with agents that lower intraocular pressure. Corrective surgery must follow. Prophylactic peripheral iridectomy for the fellow eye is also recommended.
5) Vitreous haemorrhage causes a dramatic loss of sight. The fundus cannot be seen and the red reflex is lost. Treatment of the primary cause is of value. Rest is essential. As absorption often occurs, "never despair" is advised. Vitrectomy may be considered if unresolved after a year. Laser for bleeding areas.
6) Retinal vein thrombosis or artery occulsion are often associated with artero-sclerosis, cardiovascular disease, and hypertension. Any associated systemic disease has to be treated. Anticoagulants may be useful. Full systemic steroids has been sight-saving when the condition is due to temporal arteritis. A quick acting vasodilator (amyl nitrite), intravenous nicotinic acid or retrobulbar injection of acetylcholine may be tried. Mechanical aids such as massage of the globe, paracentesis and inhalation of oxygen may be tried.
7) Retinal detachment occurs especially in a high myope. Bed rest, postural nursing and corrective surgery is indicated.

CASE DEMONSTRATION illustrating physical signs

R bin M, Male, 24
Road traffic accident on 21st April '74. Right optic nerve injury.
Note: Pallor of right optic nerve and Marcus-Gunn pupil.

A B H, Female, 66
Isolated Right VI Cranial Nerve Palsy. Diabetes Mellitus.
Note: Head turn to left to compensate for diplopia

L Y M, Female 29
Juvenile gaucoma. Left eye blind, divergent squint. Right eye has 6/6 vision, but tubular fields. Right optic disc pallor and cup. Intraocular pressure 16-20 mm Hg Right; 26-40 mm Hg Left.
Note: Small meiosed pupils, induced by strong meiotics. Will require medical treatment indefinitely.

M bin M, Male, 32
Right micropsia 3 days prior to admission. VR = 6/60, with central scotoma. Right idiopathic central retinopathy (I.C.R.); also called Central Serous Retinopathy (C.S.R.). Fluorescein injected intravenously shows leak in chorio-retinal vessels.

T S K, Male 65
1. Hypertension
2. Old P.T.B.
3. Liver cirrhosis (alcoholic)

Eye show: arterial attenuation; old central retinal artery occlusion. Temporal (giant cell) arteritis has to be excluded by ESR.

L S J, Female, 41
Left inferior temporal branch thrombosis with 3 months history of blurring of vision. Also been treated by physicians for hypertension (BP 170/90). On contraceptive pills since 1963. "Anovlar" containing Lynoestrenol 4 mg and ethenyl oestradiol 50 mg. Any relationship?

T S H, Female, 11
Right eye shows herpes simplex virus (H.S.V.) infection with dendritic ulceration on cornea and vesicles on eyelid. Was admitted to medical ward for meningitis. Herpes meningitis?

T T K, Male, 38
Bilateral corneal burns (acid) while unloading cargo off a ship. Note: Corneal scarring and conjunctival adhesions (symplepharon).

L S L, Male, 26
Bilateral corneal burns (ammonia) from assault. Also had head injury and previous admission to surgical ward. Cornea shows severe keratopathy, descement membrane folding, ulceration and vascularisation; hypopyon. Alkali burns are worse than acids or thermal burns because of penetration of these chemicals.

R binte U, Female, 14
Right eye injury (?sand entered). Corneal abscess, presumed fungal. Negative cultures but has improved with amphotericin B injections given subconjunctivally.
Ophthalmologists' Prayer: "Lord, if I must have an eye infection, let it not be herpetic or mycotic" (K.H. Lim).

H K M, Female, 34
Left neuritis. c/o sudden blindness in left eye. No physical signs except for Marcus-Gunn pupil; hence, "neither the patient nor the ophthalmologist can see anything". For physical investigations and prednisolone.

L K C, Male, 19
Right intraocular foreign body (I.O.F.B.) removed by magnet extraction. Hammer and chisel injury.
Note: residual vitreous haemorrhage.

C Y W, Male, 41
Left I.O.F.B. (copper). Unsuccessful attempt with extraction. Severe intraocular damage from the injury. Vitrectomy to be considered.
Note: hyphaema, lens opacity, vitreous haemorrhage, retained I.O.F.B.

Lecture and Demonstration given on 13th October 1974, Refresher Course, College of General Practitioners, Singapore.

Domestic accidents in a local community (Clementi estate)

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(Third Year Medical Students)

INTRODUCTION

Although deaths due to non-vehicular accidents have remained consistently at about 2% of all deaths over the last two decades, they refer only to the very tip of the iceberg. Many accidents, especially those that occur in the homes, do not result in fatalities. Being highly preventable causes of morbidity, domestic accidents will always be a subject of concern to families. The professions engaged in maintaining the health of a community through proper housing – the architects, engineers and doctors – are equally concerned.

As part of a student exercise in community health, a group of 20 students studied an old housing estate called Clementi Estate. Built in 1951 as quarters for married army personnel, it was released for civilian use in 1971. It is thus an old estate with many new dwellers, most of whom having been there for less than 5 years. They comprise mainly young adults in the low-income group, working as service or factory workers.

The first visit to Clementi Estate revealed many potential accident hazards like steep staircases, unstable kerosene cookers, exposed wires, and glass panes lying on open ground. Thus, it was considered worthwhile and interesting for the group to study the occurrence and pattern of domestic accidents in this estate. The specific objectives were as follows:

(1) To determine the incidence of domestic accidents in Clementi Estate over a 4-month period (January – April 1979);
(2) To determine personal factors associated with domestic accidents.

MATERIALS AND METHODS

The following definitions were used:

(a) an **accident** is the event that occurs at that point in time when the preceding factors or potentials interact to produce irreversible and recognisable results (U.S. Department of Health, Education and Welfare, 1958);
(b) a **domestic accident** is an accident that takes place in or around the home and for the purpose of study, the home must be understood to include the dwelling unit itself, the garden, yard, garage and all that is personal to the household (W.H.O., 1957; B.M.A., 1964). The relevant areas would also include the stairs and approaches to flats or rooms, so long as they are reserved for the use of tenants (U.S. Department of Health, Education and Welfare, 1958).

All persons in the survey sample were interviewed to determine the occurrence of domestic accidents during the 4-month period from January to April 1979. The information obtained included the following:

(1) Personal factors – age, sex, ethnic group, physical and mental disabilities;
(2) Description of accident – type, time, place and treatment.

The survey, conducted by 20 third-year medical students, took place in early May 1979. Standard questionnaires were used, and every attempt was made to standardise terminologies and language used.

RESULTS

I. Response

Out of 170 residential units sampled, 150 (88%) responded. "Non-response" was mainly due to the units being vacant permanently or temporarily. Altogether 515 persons of all ages responded to the survey.

II. Incidence

102 persons had at least one accident, giving an incidence rate (by person) of 19.8% for the 4-month period.

The number of episodes experienced by the 102 persons was 121.

III. **Personal Factors**

(a) **Sex**

Although females tended to have a slightly higher incidence rate, the difference is not statistically significant (Table 1).

Table 1
Incidence of domestic accidents, by sex

Sex	No. of persons with accidents	Incidence Rate (%)
Male	48	18.3
Female	54	21.2
Both	102	19.8

(b) **Ethnic Group**

Indians seem to have the highest rate while Malays the lowest, but again the difference is not significant (Table 2).

Table 2
Incidence of domestic accidents, by ethnic group

Ethnic Group	No. of persons with accidents	Incidence Rate (%)
Chinese	70	21.7
Malay	24	15.0
Indian	8	25.8

(c) **Age**

Children under the age of 10 years have a higher incidence rate than that found in other age groups ($p < 0.05$). There is the suggestion of a bi-polar distribution, with peaks at both ends of the age spectrum (Table 3).

Table 3
Incidence of domestic accidents, by age

Age-group (years)	No. of persons with accidents	Incidence Rate (%)
0–9	25	28.8
10–	20	20.6
20–	39	20.3
30–	10	16.1
40–	1	3.4
50–	5	17.9
60+	2	10.6
Total	102	19.8

(d) **Physical and Mental Disability**

21 persons in the survey population were found to have either a physical or mental disability or both. Of these, only 2 sustained domestic accidents and both were due to cigarette burns. The incidence rate for the disabled is much lower than that for the non-disabled (Table 4).

Table 4
Incidence of domestic accidents, by ability-status (mental and physical)

Status	No. of persons with accidents	Incidence Rate (%)
Disabled	2	9.5
Not disabled	100	20.2
Total	102	19.8

IV. **Description of Accidents**

(a) **Type**

From Figure 1, it can be seen that burns/scalds make up the largest proportion of accidents occurring in the home. Cigarette burns that occurred as a result of a smoker accidently burning himself or others are included. The most important group reporting burns/scalds other than cigarette burns were the females in the age-group 20-59 years.

Type of accident, by proportion of spells

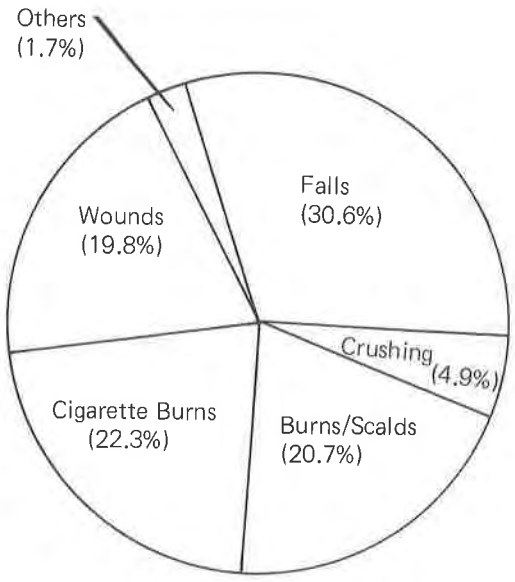


Figure 1

The next group include falls at ground level and from heights. Higher rates were reported for the younger age-group 0-19.

(b) **Place of occurrence**

For the place, time and treatment of accidents, only the most significant episode of each type of accident was considered.

The kitchen/dining room was the commonest place of accident (Figure 2).

Place of accident, by proportion of spells

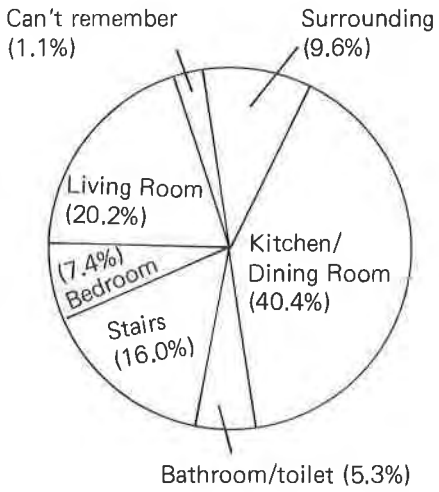


Figure 2

A high proportion of burns/scalds (excluding cigarette burns) occurred in the kitchen/dining room, unlike falls which tended to take place at the stairs or in the surroundings of the houses.

(c) **Time of occurrence**

There seems to be a fairly equal distribution among the three periods of the day, with a slight preponderance occurring in the night (Figure 3). This is especially so for falls, contrary to the occurrence of burns/scalds in the morning.

(d) **Treatment**

About 85% of all the accidents did not receive any professional attention — a third were not treated and the other two-thirds self-medicated. About 13% went to General Practitioners or Government Out-patient Dispensaries. None was serious enough to be hospitalised (Figure 4).

DISCUSSION

People vary in both their physical and personal characteristics. Some may be weaker in physical strength, others may be more "accident prone".

Time of accident, by proportion of spells

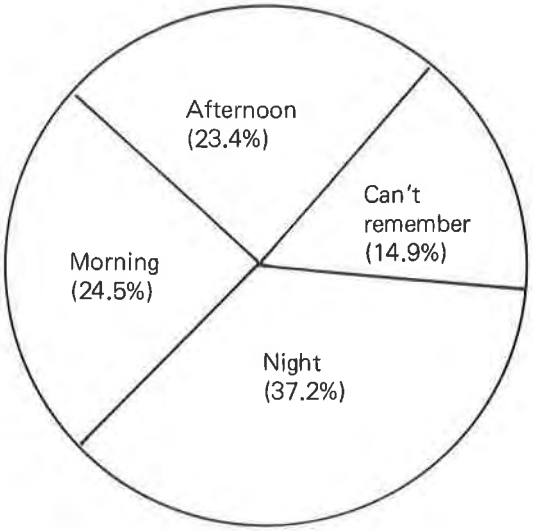


Figure 3

Treatment of accident, by proportion of spells

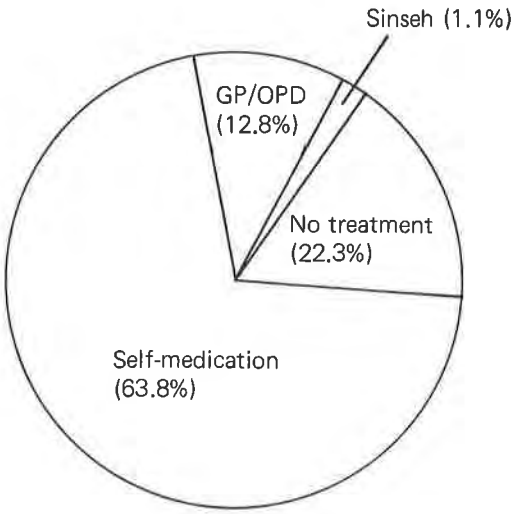


Figure 4

All these contribute to an accident in one way or another. However, it is difficult to quantify such characteristics. This study has merely concentrated on the more obvious and readily elucidated factors.

The main findings are summarised thus:

(a) Males and females, young and old have their

own risks for different types of accidents;
(b) Burns and scalds (excluding cigarette burns) are of major importance, especially occurring in females, aged 20-59, in the kitchen/dining room during the day;
(c) Falls are of concern among children and teenagers, occurring mainly at night and at the stairs;
(d) Majority (85%) of episodes are minor in severity, and thus need either no treatment or self-medication.

The only previous study on domestic accidents in Singapore was done in 1975, based on cases seen at the Accident and Emergency Department of the Singapore General Hospital (Chao, 1975). It would be erroneous to make any comparisons as these cases were selectively more serious (requiring emergency attention). There were more falls seen, and definitely much less of cigarette burns, minor burns/scalds and cuts.

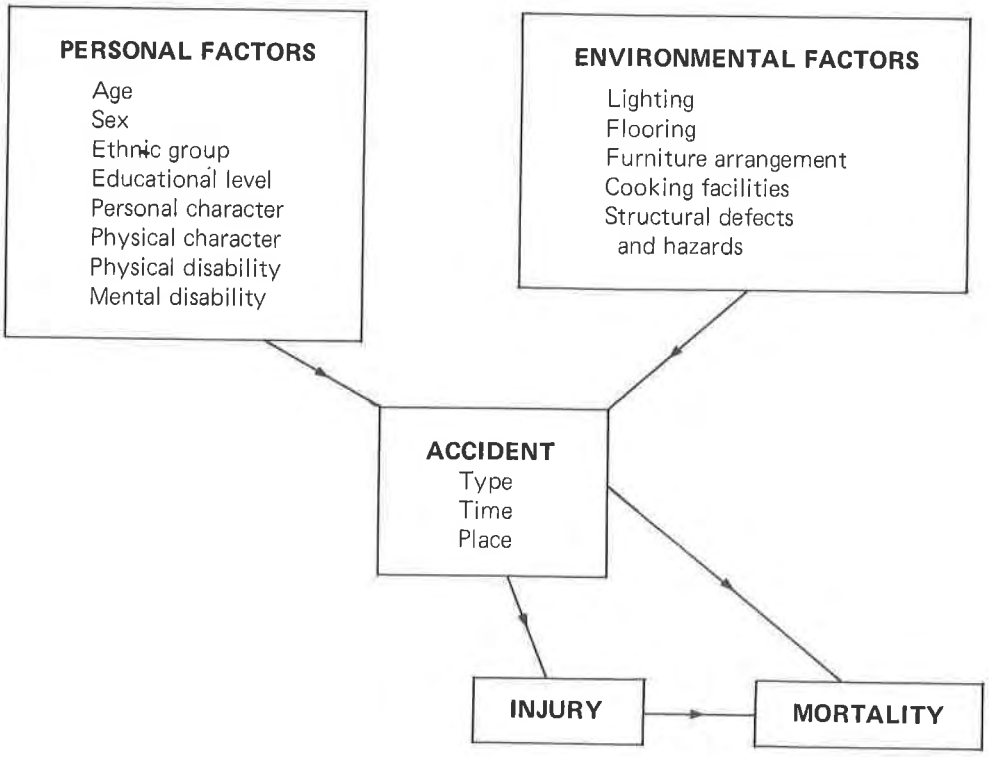
The many factors that predispose people to accidents can be classified under two headings — PERSONAL factors and ENVIRONMENTAL factors. The following diagram summarises the interaction of these factors in producing a domestic accident.

In line with the 'iceberg phenomenon', deaths and even hospital or clinic cases of domestic accidents pertain only to the very tip of the problem. The majority of incidents are minor, and do not require professional attention. Nevertheless, hazards abound, and the potential for more serious injury is always there. Thus, every effort should be made to control such preventable causes of morbidity, thereby improving the environment in which we live.

Some of the comparisons between factors should be studied with great caution in view of the small numbers involved. The only personal factor of importance was age, with children being most prone to accidents at home due to their inexperience, adventurous spirit and lack of physical strength.

No direct correlation between the presence of hazards and the occurrence of accidents could be made, due to small numbers and lack of information. But the list of accident hazards found include:

(a) slippery cement floors,
(b) inadequate lighting, especially at night,
(c) poorly arranged furniture,
(d) loose rugs on the floor,



(e) steep (45^{0} angulation) and poorly illuminated stairs,
(f) unstable kerosene stoves,
(g) potholes in the surroundings,
(h) broken bottles and glass in the surroundings,
(i) storage of water in large containers,
(j) exposed wires inside and outside the houses.

Some preventive measures that may be implemented are:

(1) For burns/scalds:

(i) putting up barricades at entrances to the kitchen, to prevent young children from having easy access;
(ii) keeping all inflammable material such as fuel and matches out of reach;
(iii) stabilising kerosene stoves with bricks;
(iv) discouraging smoking in bed;
(v) providing guards for all naked flames.

(2) For falls:

(i) providing barricades at the top and bottom ends of the stairs, preventing children from playing on them;
(ii) improving lighting of stairs;
(iii) filling up potholes in the surroundings;
(iv) ensuring that cement floors be kept dry;
(v) replacing slippery rugs with non-slip rubber-backed mats.

Although the total number of persons experiencing domestic accidents was 102, only 55 had taken precautions to prevent further accidents. Some claimed that they were ignorant of the type of preventive measures to be implemented while others were simply indifferent.

The need for safety education is very real. The National Safety First Council organises a biennial exhibition on home, road and industrial safety at a few public places. This should be made an annual event, at more venues than the present few. The Housing and Development Board can also increase their educational efforts through their own publication "Our Home" and notice boards of each block of flats.

As children form a good proportion of those having domestic accidents, schools should inculcate the need for safety awareness amongst the young. Mothers should also be educated, perhaps through the mothercraft sessions at the Maternal & Child Health Clinics.

CONCLUSION

An accident is generally regarded as an event without apparent cause. The real truth is, however, that unexpected accidents rarely occur. There are accident-prone situations and accident-prone people. If these situations can be eliminated and people taught to avoid such dangers then much of the accidents in the home can be avoided.

A word to the wise is sufficient but the foolish unfortunately have to learn the painful way.

Acknowledgement

Our thanks to Dr. T.C. Choo, Senior Government Pathologist, Dr. H.P. Lee, Senior Lecturer in Social Medicine & Public Health and all other members of the survey team for their help and who have made this study possible.

News from the council

THIRD SREENIVASAN ORATION

The Third Sreenivasan Oration, the Convocation and the Annual Dinner of our College will be held at the Shangri-La Hotel, Singapore on Sunday, 16 November 1980. The oration will be delivered by Dr Leong Vie Chung, a Fellow of our College.

EIGHTH COLLEGE EXAMINATION

The Eighth College Examination for the Diplomate Membership of the College will be held on :

Sunday, 26 October 1980 — Theory Papers
Thursday, 6 November 1980 — Clinicals

The theory papers will be held in the College Conference Room and the Clinicals will be conducted at the Alexandra Hospital, Alexandra Road, Singapore-0511.

CONTINUING EDUCATION UNIT

An In-Depth Course in Psychological Medicine comprising of 12 lectures, was run by the College from 27 June 1980 to 5 September 1980. Eighty-seven doctors registered for the course and it was well received.

The Chairman of the Continuing Education Unit, Dr Frederick Samuel, announced that the next in-depth course would be in Adult Medicine details of which will be released as soon as arrangements have been finalized.

TENTH WONCA WORLD CONFERENCE, 1983

Our College is making a very determined effort in the bidding to host the Tenth WONCA World Conference, 1983 in Singapore. A number of our members will be going to New Orleans to observe and study the organization and management of the scientific and social programmes of the World Conference.

MEDICAL NEWS

Are the results of safety tests on saccharin, using animals, valid in advising the use of this substance in man? Can saccharin be used safely in low carbohydrate diets?

This question raises several very difficult areas connected with toxicological testing of food additives. Screening with animals or some other type of biological assessment is a necessary safety measure before a food additive can be used. This testing, however, necessitates feeding the additive at several dose levels, some of which would be very much greater than those which would be consumed by man on a per kg body weight basis. The carcinogenetic activity of an additive is particularly difficult to assess at very low incidence rates because large numbers of test animals are required and turnours may arise spontaneously.

The concern with the safety of saccharin arose from studies where a very high level of intake of saccharin produced a low incidence of bladder tumours. The laws relating to the use of food additives in the USA are dogmatic in rejecting the use of an additive which at any level of consumption has been associated with the development of tumours. The evidence for the involvement of saccharin in producing tumours is not unequivocal – consumption of diets containing 50,000 ppm (5%) has reportedly not produced any excess of tumours in dosed rats compared with controls. On balance, the evidence at present suggests that the risks from ingesting saccharin at normal levels of consumption are very low indeed.

As there still seems to be some doubt whether the fourth or fifth Korotkoff sound is indicative of arterial diastolic pressure, would not a needle in the brachial artery inserted just below the sphygmomanometer cuff and connected to an oscillograph give the required information?

There is doubt whether muffling (Korotkoff, phase 4) or disappearance of sounds (phase 5) is a more accurate indicator of diastolic blood pressure.[1] The tendency in Britain and Ireland has been to use the fourth phase, whereas the USA has generally favoured the fifth phase. The controversy continues, but phase 5 is now more-generally accepted because the results are closer to those with direct intra-arterial measurement, and there is better agreement among observers. The disadvantage in choosing the fifth phase is that in high-output states the disappearance of sounds may be greatly below muffling, and sometimes the sounds do not disappear at all. When this occurs the fourth phase should be taken. The controversy might be best solved if both phases were recorded. Phase 5 correlates best with the diastolic pressure measured through an intra-arterial catheter, a technique that is not only feasible but is often used to obtain continuous blood pressure measurement in selected patients.[2]

1 O'Brien ET, O'Malley K. ABC of Blood Pressure Measurement, **Br Med J 1979; ii:982-4.**
2 O'Brien ET, O'Malley K. ABC of Blood Pressure Measurement, **Br Med J 1979; ii:1124-6.**

ACUTE RENAL FAILURE AFTER A BEER-DRINKING BINGE

It is not widely known that excessive ingestion of ethanol may precipitate acute renal failure in healthy individuals. Six patients who drank large amounts of vodka over several hours developed acute renal failure, occasionally with hepatocellular damage.[1] Oliguria persisted from 16 to 75 days. All patients required dialysis, and one patient died. We now report two similar cases of acute renal failure after heavy beer drinking with subsequent recovery after peritoneal dialysis.

Case 1

A 41-year-old motor fitter drank over two gallons (9l) of beer that he had made himself from a kit. He drank this beer between 4pm and midnight and complained of severe diffuse lower

abdominal pains throughout the night. At 8 am he awoke with headache, neck pains, abdominal muscle pain, and general muscle stiffness. From this time his urine output fell. He was referred to hospital after three days. Examination showed a drowsy but otherwise fit man with no evidence of dehydration.

Blood pressure with 130/80 mm Hg. Urine volume was 300 ml in 24 hours, and urine osmolality 376 mmol (mosmol)/kg. Blood urea concentration was 22.5 mmol/1 (135.5 mg/100 ml). Serum bilirubin concentration was 13 μmol/l (0.8 mg/100 ml), alkaline phosphatase activity 7 KA units, and aspartate transaminase activity 53 U/1. Cystoscopy showed no abnormalities, and ureteric catheterisation was unsuccessful. Plain radiography of the abdomen showed two kidneys of normal size. On conservative treatment the patient remained oliguric and his blood urea concentration rose to 59.6 mmol/l (359-0 mg/100 ml). Peritoneal dialysis was started, and diuresis occurred on his sixth day in hospital. Six months later his glomerular filtration rate was 97 ml/min/1.7 m^2 and effective renal plasma flow rate 361 ml/min/m^2.

The patient had been a weekend beer drinker from the age of 16 and was accustomed to drinking eight pints (4.51) of beer a day at weekends. He had never been ill before or lost time from work.

Case 2

A 28-year-old carpenter drank over 30 pints (17.1) of beer at a pop-music festival and developed diffuse upper abdominal pain and dull constant back pain throughout the night. His urine output fell. These symptoms persisted for three days, after which he was referred to hospital. On examination the patient was drowsy and jaundiced but with a strong physique. There was no sign of dehydration.

Blood pressure was 120/89 mm Hg. Bladder catheterisation produced 50 ml of dark brown urine. Urine volume was 400 ml in 24 hours, and urine osmolality 311 mmol (mosmol)/kg. Serum urea concentration was 22.9 mmol/1 (138.0 mg/100 ml), serum bilirubin concentration 133 μmol/1 (7.8 mg/100 ml), aspartate transaminase activity 100 U/1, and alkaline phosphatase activity 18-6 KA units. After five days serum urea concentration rose to 65 mmol/1 (391.6 mg/100 ml), but serum bilirubin concentration fell to 40 μmol (2.3 mg/100 ml), aspartate transminase activity to 146 U/1, and alkaline phosphatase activity to 11.7 KA units. Oliguria persisted for 10 days and peritoneal dialysis was started on his sixth day in hospital. A renogram taken during the oliguric phase showed severe tubular dysfunction. Obstruction of the lower urinary tract was excluded by ureteric catheterisation. In the oliguric phase glomerular filtration was 4 ml/min/1.7 m^2 and effective renal plasma flow 23 ml/min/1.7 m^2; three weeks later, after diuresis, these had risen respectively to 76 ml/min/1.7 m^2 and to 366 ml/min/1.7 m^2. The results of renal biopsy performed in the diuretic phase showed the normal histological appearance (Dr O G Williams). The patient made an uneventful recovery. He drank up 10 pints (5.7 1) of beer a day at weekends and smoked 30 cigarettes daily.

Comment

The sequence of events in our patients was similar to that described by Szepietowski *et al*[1]. Healthy individuals, after ingesting large quantities of ethanol over several hours, complained of severe abdominal pains with muscle stiffness, followed by suppression of urine. Hepatocellular damage occurred in some of these patients. There was some delay in admitting our patients to hospital since they had never been ill before and their symptoms were thought to be due to severe hangover. Admission to a surgical ward was eventually arranged because oliguria, combined with lower abdominal pain, suggested obstruction of the urinary tract.

Szepietowski *et al* suggested that acute renal failure may result from nephrotoxic damage by ethanol.[1] This is unlikely because the results of renal biopsy, performed on one of our patients, showed normal histological appearances. Acute renal failure has been observed after a bout of heavy drinking in chronic alcoholics in association with myoglobinuria and accompanying muscle tenderness and swelling.[2] [3] Furthermore, myoglobinuric acute renal failure has been caused by alcohol in six out of 20 patients[4]. The mechanisms by which rhabdomyolysis and myoglobinuria produce acute renal failure are not known.

STROKE

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M.R.C.P. (Edinburgh)

INTRODUCTION

Stroke accounts for the third highest number of deaths in Hong Kong. It is a common cause for admission into our hospital and heavily taxes our resources for health care. Stroke is a generic term for a wide variety of conditions causing neurological symptoms and signs and is to be preferred to the commonly used term Cerebral Vascular Accident (CVA). The latter is in fact a misnomer if we realise that stroke may be caused by disorder of the vasculature of the cerebellum or brain stem or even that of the spinal cord. A stroke of course does not occur by accident. Each episode of stroke has a definite pathogenesis and a set of predisposing factors. If we are ever to conquer the problem of stroke and to prevent its recurrence in a particular patient we have to understand the pathogenesis and predisposing factors. How sad will it be when a patient is diagnosed as a case of CVA and be left at that!

A very common practice in dealing with a patient suffering from a stroke resulting in some neurological disturbances is to label him as suffering from CVA, admit him into a hospital bed and do a lumbar puncture for him. If the cerebrospinal fluid contains blood the patient is diagnosed as a case of cerebral haemorrhage and if no blood is found then the diagnosis becomes cerebral thrombosis. This practice can be very dangerous because a patient harbouring a cerebral haemorrhage or a cerebellar haemorrhage may not show blood in the cerebrospinal fluid. If a lumbar puncture is done in a patient with an intracerebral haematoma the leakage of cerebrospinal fluid will cause the formation of pressure cones intracranially resulting in deterioration of the neurological state or ever death. Lumbar puncture in a case of spontaneous subarachnoid haemorrhage – a well-known clinical syndrome of headache of sudden onset with signs of meningeal irritation – can likewise be risky because in Hong Kong 50% of our cases of spontaneous subarachnoid haemorrhage are due to rupture of arteriovenous malformation (AVM) often associated with a sizable haematoma.

CLASSIFICATION

Stroke –	Haemorrhagic		Hypertensive cerebral haemorrhage
			Hypertensive cerebellar haemorrhage
			Spontaneous subarachnoid haemorrhage
			Ruptured aneurysm
			Ruptured AVM
			Haemorrhage from a neoplasm
		Vascular insufficiency	Carotid
			Vertebral
	Non-haemorrhagic	Thrombosis	due to atherosclerosis
			due to contraceptive
			due to neoplasm
		Embolism	

SURGICAL TREATMENT OF PROLAPSED LUMBAR DISCS

Most patients with the symptoms of lumbar disc protrusion respond to an adequate period of strict bed rest, and the main indication for surgical treatment is failure of such management to relieve pain. How long conservative treatment should be continued before operation depends on the severity of the pain, evidence of improvements and to some extent the socioeconomic circumstances of the patient. Repeated attacks may well be an indication for surgery, as may incomplete relief of pain preventing the patient's return to work. Significant neurological signs should be an indication for early operation, and the development of bilateral signs or symptoms, especially sphincter disturbance, makes operation urgent to avoid permanent damage to the cauda equina, which may occur from a large central disc protrusion.[1]

Though the main symptoms is pain, all patients have physical signs, and these are crucial to diagnosis. In O'Connell's classic paper[2] all 500 patients had tension signs – diminished straight leg raising – and spinal signs – deformity and reduced mobility from muscle spasm. Almost all, 96%, had neurological signs – muscle weakness or changes in tendon reflexes – and 80% some sensory change. The neurological signs will often indicate the level of the disc protrusion at L4/5 or L5/S1, where 90% occur. Such clinical localisation is not always easy, and myelography can be helpful in showing the level in at least 80% of patients. Myelography will also show up multiple lesions. Conditions such as a neurofibroma of the cauda equina, which can simulate a lumbar disc protrusion, will be excluded provided the contrast medium is viewed as high as the lower thoracic region. Radiculography with water-soluble contrast medium[3] identifies a higher proportion of lateral disc protrusions, which may

produce only slight blunting of the subarachnoid root pouch. Discography is not widely used in Britain. The absence of radiological confirmation of a disc protrusion should not be a bar to surgical exploration in a patient with the right clinical picture and poor response to conservative management.

In general, pain in the leg due to a disc protrusion compressing a lumbar nerve root responds well to operation, but pain in the back responds less predictably. Careful clinical assessment and selection of patients are essential if good operative results are to be obtained. Laminectomy is rarely performed nowadays except for large bilateral central disc protrusions, and the less destructive unilateral fenestration approach with the patient in the lateral position is usually preferred. The portion of disc protruded or extruded from the disc space is removed, together with all degenerate material from within the space. The nerve root needs to be carefully retracted to avoid injury, and care must be taken when clearing the disc space to avoid penetrating the anterior surface and damaging the iliac vessels or ureter. The volume of disc material removed bears little relation to the success of the operation, and bilateral removal of disc material is not required in patients with unilateral symptoms. [6]

Postoperative management varies with different surgeons. Some advise bed rest for three weeks with graduated increase in exercise, but others advocate mobilisation from the first postoperative day with active back exercises incuding forward flexion. This wide diversity of opinion suggests that fears that early flexion of the spine may result in damage are unfounded, and early activity has the advantage of rapidly restoring mobility. Since the aim is a pain-free spine, this seems more likely to be achieved with a mobile spine than one which is stiff and may depend on use of a lumbar corset for comfort. In these days of reduced resources the regimen of early active mobilisation has the major merit of allowing early discharge from hospital.

In a recent review of 323 patients operated on for disc lesions 86% of the men and 79% of the women were free of all symptoms, while 9% and 15% respectively had some persistence of back pain. Only 2% of men and 6% of women had more severe permanent symptoms, and only 5% developed recurrent protrusions; nevertheless, these figures may be underestimates, since less than half the patients had been followed for more than three years.

Failure of operation to relieve pain may be due to failure to find the disc at operation — a problem which myelography should prevent — but damage may be caused to the nerve root by the protrusion, by operative retraction, or by the later development of adhesions or arachnoiditis. These complications do not respond well to further operation, and for an unfortunate minority of patients persistent symptoms may result in inability to work and much suffering. The development of a lumbar disc protrusion, particularly in a man undertaking heavy manual work, should be regarded as a serious event: and even after successful treatment, whether conservative or surgical, patients should always be advised to avoid lifting with a bent back, and in heavy manual workers a change of occupation may be needed.

MEMORANDUM AS TO THE FOUNDATION OF A MEDICAL SCHOOL FOR THE STRAITS SETTLEMENTS IN SINGAPORE

1. A Medical School may be founded in one of two ways:

(a) As a school to supply merely local wants, and to produce men qualified to supply these wants and nothing more; or

(b) As a school of a higher order, which should commence, in a small way possibly at first, but on higher lines, to educate men in such a manner that the education they may receive shall be accepted as part, and subsequently it is to be hoped as the whole, of the curriculum required to admit them to examination by examining bodies in Ceylon, Madras or Great Britain.

2. As regards the first way, all that would be required is that Government determine what qualifications are necessary for men to fill subordinate medical positions, as high, say, as Apothecaries; to arrange for the education of students up to such qualifications, and to make such qualifications legal. These qualifications would not, of course, be by any means necessarily legal in places other than the Straits Settlements.

3. As regards the second way, Government would have to make arrangements for education of students according to the curriculum of some recog-

nised licensing body, commencing with compelling them to pass a recognised preliminary examination, and arranging for lectures and teaching generally, and for the payment of fees for such lectures and teaching, as shall bring these conditions on a par with similar conditions in recognised schools.

4. I think that, in starting a Medical School, it will be preferable to begin at once on the second or higher platform, firstly, because it is good to begin a thing properly at once; and, secondly, because though a beginning may be made in a small way, it would be in the right way and additions could be made afterwards without trouble; whereas if a beginning should be made in a purely local way, and change afterwards should become desirable, the whole thing would have to be begun ***de novo.***

5. I do not think it will be found practicable to commence operations before October 1st (the date on which Medical Schools open) 1890, therefore I would suggest that a total amount of $2,350 be placed on Estimates 1890, made up as follows:

General Expenses connected with opening Medical School	$1,000
Fees for Hospital Attendance and Lectures for six Students for two years, payable in advance at $150	900
Allowance to six Students for 3 months (October, November, December) at $15 a month each	270
Allowance to six Junior Students being educated for subordinate posts (Dressers, etc) at $10 a month each for 3 months	180
Total	$2,350

6. Of course, it is to be hoped that, with increasing prosperity and population of the Straits Settlements, students will hereafter present themselves who are able and willing to pay for their education. Such students of course, when qualified, will be at liberty to practise their profession in any way they may like, and will not be in any way under obligation to Government. Stipendiary students should, I think, enter into agreements to serve the Government for a term of years, as has been the custom with Straits Apothecaries hitherto educated entirely in Madras.

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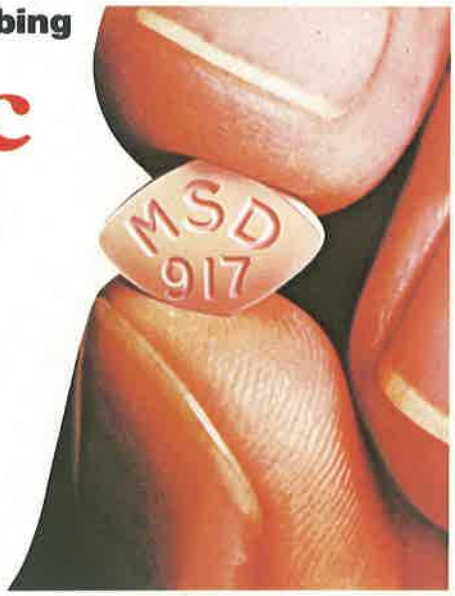

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