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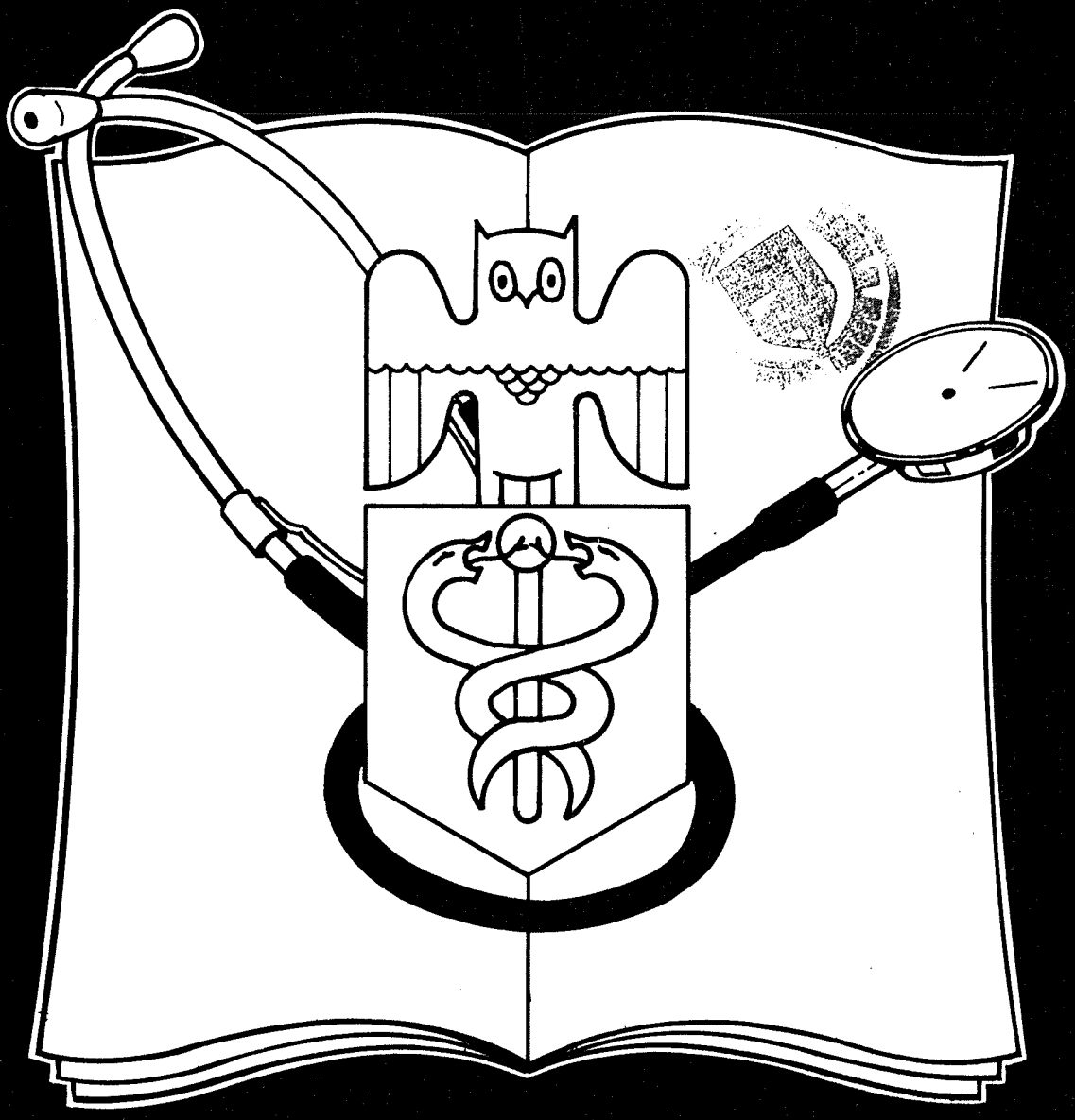

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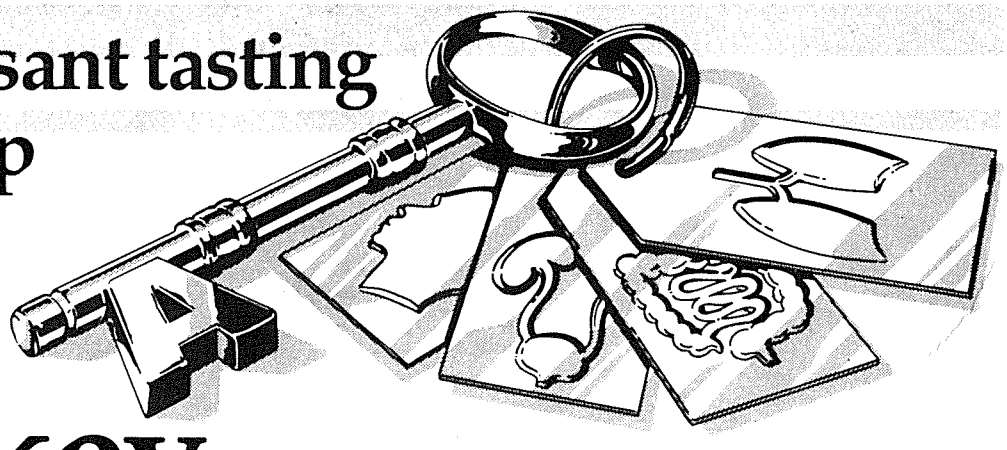

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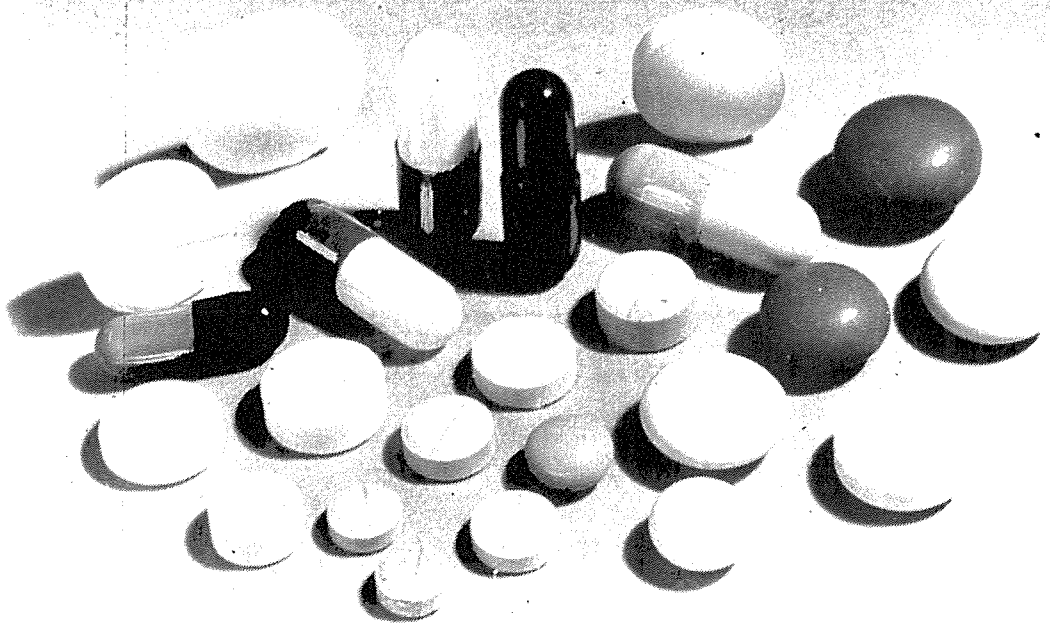

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Editor's Letter

Medi-Scope Issue No. 3 Jul-Sep 1983

Medical Students Journal
University of Malta Medical School
G'Mangia - Malta

The summer recession we were all longing for has come and also gone and everybody should go back to his/her books unless he/she has already done so. The fifth year students shall soon be sitting for their final examinations which will determine their future in the medical career; we all wish them every success and may they achieve what they have been working for during the past five years. The fourth years (and that includes most of the editorial board!) will be having their bit of tension with the Pathology final knocking at their door. Third year students will be doing the usual exams of Medicine, Surgery, Pathology and Obstetrics. Second years will be sitting for the finals in Anatomy and Physiology. Although everybody is up to his neck in work and study, ***Medi-Scope*** still makes its way to supply articles for those who can spare a few hours to read them!

In this issue's letter we must bring to the notice of all the readers that the Christmas number will be a special one in that proceeds will be in aid of a charitable institution. Hence we appeal to all who recieve or purchase this journal to kindly donate any additional contribution to make our donation sizeable enough to be given as a *Christmas Present* to those who need it badly e.g. the handicapped people. With this aim fulfilled we will have widened the scope of this magazine. We are most certain of your support and co-operation in this respect and on behalf of whoever will recieve our present we thank you sincerely.

It is very encouraging to see other students writing articles. Our request for more participation is being fulfilled as we see the great number of answers being passed on to us in response to the quiz we set up in the last issue. It is our wish to see such enthusiasm sustained for this is the only reward for the editorial board's hard work. We hope that we can maintain the high standard we accomplished so far.

Mr. Gauden Galea has resigned from the editorial board in view of his forthcoming exams. We thank him for the invaluable help he has given us. Good luck Gauden!

I am taking the opportunity here to congratulate, on behalf of the editorial board, our Honorary life president, Dr. V. Captur for the honour bestowed upon him by being elected member of the New York Academy of Sciences.

THE EDITOR

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Snakebites

CHARLES SAVONA VENTURA MD

"..... After we had escaped, we then learnt that the island was called Malta. And the natives showed us unusual kindness, for they kindled a fire and welcomed us all, because it had begun to rain and it was cold. Paul gathered a bundle of sticks and put them on the fire, when a viper came out because of the heat and fastened on to his hand. When the natives saw the creature hanging from his hand, they said to one another: "No doubt this man is a murderer. Though he has escaped from the sea, justice has not allowed him to live." He, however shook off the creature into the fire and suffered no harm. They waited, expecting him to swell up or suddenly fall down dead; but when they had waited a long time and saw no misfortune come to him, they changed their minds and said that he was a god....."

Acts of the Apostles 28;1-6

Introduction

All snakes currently present on the Maltese Islands belong to the family Colubridae, all members of which are non-poisonous to man. Some colubrids, exemplified by the local *Cat Snake*, have fangs at the back of the mouth. Though technically venomous, bites from these snakes are harmless to humans. The other Maltese snakes include the harmless *Black Whip Snake*, the *Leopard Snake* and the *Algerian Whip Snake*. Snakes are usually fairly timid creatures and they only bite humans in defence, when touched, trodden upon or cornered. When they strike, they do so with amazing speed, usually at the legs or hands of their attacker. In the majority of instances snakes prefer to avoid human contact.

History and Folkfore

Since the Maltese *Ophidea* species are harmless to man, the Maltese practitioner expects few problems from snakebite. However, the doctor may have to quench the effects of doubt and superstitions associated with apodal reptiles since time immemorial. The superstitious association of snakes on the Maltese Islands is seen in the snake image sculptured on an altar slab in the Neolithic Temple at Ġgantija in Gozo. This temple has been dated to 2800 - 2500 B.C.

The first written reference to the Ophidea of the Maltese Islands is in the Acts of the Apostles c.60 A.D. This refers to a 'viper' biting the apostle Paul, who came to no harm even though the natives expected him to swell up or suddenly fall down dead. Local swelling is an invaluable sign of viper envenoming, starting within minutes. Swelling is also a feature of poisoning by bites from the Asian cobra, though this usually appears one to two hours later. Collapse occurs with systemic poisoning after a variable period depending on the dose of venom recieved.

It would appear that Paul's snake was a viper or alternately a snake closely resembling a viper in its general appearance. The interpretations of this event in the light of the present resident snake species have been various. Folklore has it that from that moment, as a result of the apostle's intervention, the poison ceased to exist in the mouth of Maltese snakes. A further assertion which has been handed down through the generations is that the apostle banished all poisonous animals from the Islands, to an extent that vipers brought from abroad died on arrival (Lanfranco, 1956). In addition, it was held that fossil shark teeth known as tongues of St. Paul, fossil fish vertebrae known as serpent eyes, saliva of persons born on the feast of the Conversion of St. Paul, earth from St. Paul's Cave at Rabat, Malta, and powdered Maltese rock were efficacious in the cure of snakebites (Cassar, 1964).

More sensible authors have suggested that Paul's viper was an accidental imported visitor or that the poisonous species became extinct on the Islands with the growth of civilization. Others have maintained that the viper which bit the apostle was none other than the harmless *Leopard Snake*, which has very close superficial affinities to the *European Viper*. The symptoms and signs expected by the natives were a result of the influence of the Roman rulers of that time, who would have been familiar with the true viper (Lanfranco, 1956).

Whatever the explanation of the apostle's viper, the Maltese doctor will very likely be presented with harmless bites which, at the very most only need treatment as puncture wounds. However, there always remains the possibility that the bite is caused by species imported from abroad. Exotic species of snake have been occasionally recorded from Malta, these being imported with merchandise. The now naturalised species of *Algerian Whip Snake* and possibly the *Cat Snake* are thought to be species brought over from North Africa along with firewood consignments during the First World War (Borg, 1939). The introduction of an exotic poisonous species, probably a viper, has been recorded in the 16th century. In July 31, 1566 a ship arrived at Malta from Venice ladened with timber and iron. While

the pine planks were being unloaded, a poisonous serpent fixed its fangs into a seaman's hand. The bite was treated by a Maltese layman who adviced the seaman to make the sign of the cross over his hand and cover the hand and snake with powdered Malta rock. The serpent died forthwith, whereas the seaman remained hale and hearty..The author records this experience to prove the efficaciousness of ground Malta rock in the treatment of snakebite (Bosio, 1621).

This experience, if true, seems to prove one aspect of Maltese folklore. However, it may be argued that poisonous snakes have two types of bites; a business bite which the animal uses to kill its prey wherein it injects a deadly amount of poison, and a defence or warning bite which the animal uses to escape when threatened. Hence, poisonous-snake bite is not necessarily the same thing as snakebite poisoning (Reid, 1972). This would explain why not all cases of poisonous snakebite require the administration of an antivenom.

Poisonous Snakebite

The poisonous snakes of the world may be broadly classified, medically, into three major groups depending on the type of toxicity elicited by the venom (vide infra). The clinical features of systemic poisoning usually occur secondary to the primary emotional response which comes on rapidly within minutes of the injury. The commonest symptom following snakebite - whether the snake is poisonous or harmless - is fright. The frightened patient may appear semi-conscious, with a cold, clammy skin, feeble pulse, and rapid, shallow breathing. Local pain may also be a reaction to the emotional response. Fright reactions are those which may be encountered by the Maltese practitioner and are more likely to be present in adult patients, rather than young children who are rushed to the doctor by their parents.

The onset of systemic effects of snakebite poisoning occurs at a time dependant on the dose of venom injected, usually a half or one hour after the injury. The initial signs of systemic poisoning depend on the type of poison injected. Laboratory experiments show that snake venom contains a mixture of toxic factors including proteases, phosphatidases, neurotoxins, cardiotoxins, hyaluronidase and cholinesterase (Paton, 1977). Fortunately the main clinical patterns of snakebite poisoning in humans are distinctive and divisible into three main groups.

The **neurotoxic** group of venomous snakes include the *Elaphinae,* to which belong the cobras, mambas, kraits, coral snakes and all the poisonous Pacific-Australian land snakes. Elaphid bites usually result in few local effects, except in bites by the Asian Cobra which give local swelling one to two hours after the injury, and is followed later by blisters and local superficial necrosis. Early important diagnostic signs of systemic poisoning include ptosis and glossopharyngeal palsy. Severe poisoning is assumed if these signs occur within one hour, or less, of the bite. Rapid progression to respiratory failure and mental confusion occurs. Shock, possibly cardiotoxic, may also be a feature of severe poisoning. Death occurs as a result of the respiratory failure caused by the respiratory muscle weakness that occurs, complicated by the inhalation of secretions or vomitus. The neurotoxic features, if not fatal, resolve in about two or three days, but may exceptionally persist for as long as two weeks.

The **myotoxic** group of venomous snakes include the *Hydrophinae* or sea-snakes common in Asian-Pacific coastal waters. Sea-snakes bites lave no local effects. The first signs of systemic poisoning include general myalgia, followed three to five hours later by myoglobinuria. Severe poisoning is diagnosed if myoglobinuria occurs as early as one to two hours after the bite. It is followed within a few hours by the development of respiratory failure,which is often the cause of death 12 to 24 hours later. The electrocardiogram (ECG) is a useful investigative tool, giving early warning of impending death or acute renal failure. The muscle damage that occurs with these bites gives rise to a hyperkalaemia characterised by tall peaked T-waves on ECG. Full recovery after non-severe poisoning is prolonged to several months.

The **vasculotoxic** group of venomous snakes include the *Viperidae* or vipers. Viper bites are characteristically followed by local swelling and discolouration within a few minutes. If the dose of venom recieved is large, serous or sanguineous blisters may follow. The early diagnostic signs of systemic poisoning include haemoptysis due to a defect in the clotting mechanism. Severe poisoning is suspected if the local swelling reaches the knee or elbow within two hours of the bite. Shock and haemorrhagic signs, such as gum bleeding, ecchymosis and haemoglobinuria, develop. The severe shock and bleeding into vital organs result in a protracted death over two or three days. In less severe poisoning, the shock and haemorrhagic features resolve within a week. ECG changes in severe systemic elaphid and viper poisoning are similar with T-wave inversion and ST segment deviation.

Management of Snakebite

The management of snakebite in the Maltese Islands is in the large majority of cases very simple and aimed at the fright and emotional reactions. As noted earlier, these reactions are variable in severity, so that some patients will suffice with reassurance that the offending animal is harmless while others will require tranquillisers and placebo treatment. Local treatment as for small puncture wounds should be instituted. The mouths of all snakes are infested with bacteria and a wide-spectrum antibiotic should always be given as part of the treatment. A booster of toxoid is given with suitable precautions (Paton, 1977)

One must, however, keep in mind rare possibility of an exotic introduced species, particularly for bites from the Grand Harbour area. Poisonous species which are most likely to be introduced to the Maltese Islands include the vipers, which are the only poisonous forms of snake in Europe. Elaphine species may be introduced from the African coast. Patients

bitten by snakes often bring the dead offending animal along with them. A medical practitioner familiar with the local four snakes will very easily recognise the specimen as local or otherwise. If the species is unfamiliar, a careful search for the presence of fangs should be undertaken. Vipers have long erectile fangs, whereas elaphids and sea-snakes have short fixed fangs. It is important that the mouth and fangs of freshly killed snakes are examined with care since reflex action in a dead snake has been known to cause a severe bite. Should the offending snake not be captured, an examination of the bite-mark may be of help in distinguishing a non-poisonous form from a poisonous one. The bite of a non-poisonous snake is doubled U-shaped impression of small teeth marks. Characteristically a double fang mark with or without additional teeth marks suggests a poisonous species. The early diagnostic signs of systemic poisoning may be present.

The therapeutic measures for systemic poisoning include supportive measures to combat shock, respiratory failure and acute renal failure. Considerable controversy still exists over the best management of poisonous snakebite. This is probably because most bites are not fatal and the low mortality may mistakenly be ascribed to the treatment. If envenoming is minimal the chances of serious consequences are slight and therefore the most energetic therapeutic measures are unnecessary. Therapeutic measures include the administration of the specific antivenom made from horse serum. Antivenom is available for most species of poisonous snake throughout the world. Since all antivenoms are made from horse serum, their use carries a significant risk of allergic reactions including anaphylactic shock. Antivenom is indicated for bites causing local necrosis to prevent or minimize this unpleasant complication; and for potentially serious systemic poisoning as evidenced by hypotension, ECG changes, neutrophilia, and acidosis, along with abnormal bleeding after viper bites or, ptosis or glossopharynegal pslsy after elaphid bites.

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Osteoarthrosis

CECIL VELLA
MEDICAL STUDENT

Osteoarthrosis is one of the most common disorders effecting joints. It has a higher incidence in the older age group, but in a number of cases it can occur earlier in life. In the Maltese Islands this disorder is very common and this may be partly attributed to the higher incidence of obesity in the islands.

As the condition occurs mainly in the old, it is believed to be due to a degenerative process of the joints, or in other terms a wear and tear process within the joints. In fact, if a joint were never put under stress, osteoarthrosis would not develop. In conjunction with this it is evident that those joints which are more under stress, are more prone to develop osteoarthrosis. Thus in most cases it is the joints of the lower limbs which develop the disorder first.

Pathology of Osteoarthrosis.

Osteoarthrosis may be defined as being a *Degenerative Arthropathy*. It is the commonest form of chronic joint disease. Dispite its more common name — osteoarthritis, the diseases is *not* an inflammatory process but results from the destructive and degenerative processes which occur in the joint cartilage in old age and in a number of joint disorders. As already mentioned the disease may affect any of the joints but is more common in those joints under stress.

This form of osteoarthrosis in the elderly is called *primary* to distinguish it from the*secondary* type that occurs earlier in life.

Structural changes in the joints.

The changes which occur in the joint affects the various components of the joint differently. The following changes may be seen.

(a) *Articular cartilage:*

The major changes seen in the joint are found in the hyaline articular cartilage which is the primary site of the osteoarthritic changes. On microscopic examination one can see metachromasia of the surface layers probably resulting from rupture of the upper collagen network. Once this occurs the upper surface of the cartilage undergoes tangential flaking resulting in the characteristic fibrillation of the cartilage. As the changes progress the underlying subchondral bone is exposed, and the articular surface becomes irregular.

(b) *Bone changes:*

The exposed bone beneath the fibrillated cartilage eventually dies and becomes moulded to the shape of the joint. Cysts may also form in this part of the bone. The most characteristic changes which are found in the bones in the vicinity of the joint, are osteophyte formation. These osteophytes are formed by the cartilage at the edges of the joint and eventually undergo calcification. These outgrowths give rise to joint deformity and, at a later stage, they may detach and form loose bodies in the joint space.

(c) *Synovium changes*

In the early stages of osteoarthrosis the synovial membrane does not show any particulr changes. At an advanced stage however, the membrane may show fibrosis and eventually thickening. The joint may be swollen due to an effusion as a result of the synovitis that occurs as a reaction to debris within the joint.

Clinical Features of O.A.

As already mentioned most patients with osteorthrosis are elderly, that is they are suffering from the Primary form of the disease. In those cases where the disorder develops at an earlier stage, there is usually an underlying disorder, that is Secondary O.A.

The changes occuring in the joint do so slowly, thus the onset of the condition is generally insidious.

The main features are two:

(I) PAIN.

(II) GRADUAL LOSS OF FUNCTION.

The pain of osteoarthrosis develops very insidiously over a few years. At the beginning it is only mild and does not disturb the patient. As the condition progresses however, the intensity of the pain incxreases and eventually becomes disabling.

The gradual loss of function like the pain, also occurs very slowly. It usually starts as a slight restriction of movement but as the condition progresses the function of the joint is more and more impaired.

Other signs and symptoms in the joint include;

— effusion within the joint.
— thickening of the capsule.
— palpable or audible crepitations on movement.
— deformity of the joint, or inability of the joint to assume the anatomical position.

As it was pointed out previously the joints which are affected mostly are those which are under the most stress during life. For this reason it is not surprising to find that in most cases the first joints to be involved are the knees and ankles, followed by the hip joints. The joints of the vertebral column are also quite commonly involved. On the contrary those joints of the upper limbs such s the elbow, wrist and shoulder are not commonly involved, except when there is some underlying cause.

The diagnosis of osteoarthrosis is usually evident from the hisotry and examination of the joint, however it has to be confirmed by x-rays of the joint. Thus radiological examination of the joint should always be performed.

Radiological Changes in osteoarthrosis.

The changes which may be seen in a joint affected by O.A. are quite characteristic of the condition. In general the following changes amy be seen:

Narrowing of the joint space as a result of the destruction of the articular hyaline cartilage.

Subchondral sclerosis in the bone underlying the damaged part of the cartilage. This sclerosed bone appears denser than the surrounding bone.

Spurring or lipping of the joint margins. This, together with the narrowing of the joint space, is the most important change seen on the X-Rays. these characteristic "lips" of bone are due to the marginal osteophytes. Occasionally one of these marginal structures may dislodge and form a loose body within the joint.

Joint deformity which may develop after many years of the disease is also visible on the X-rays.

Aetiology of Secondary Osteoarthrosis

As we pointed out earlier when O.A. occures at an early stage in the life of the patient, there is generally some predisposing cause. This is, in the majority of cases, some form of injury to the joint either by trauma or disease.

On the whole there is mainly one form of trauma but there are many forms of disease which may damage the joint.

The following are the most common predisposing causes of secondary osteoarthrosis:

(1) TRAUMA. Injury to a joint or the bones forming the joint is a common cause of secondary O.A. Thus it is not uncommon to find patients that return, after a variable amount of time from their injury, complaining of progressive joint pain and restriction of movements. Deformity of bones follwoing fractures may also give rise to derangements in the adjacent joints leading to the formaltion of Secondary O.A.

(2) JOINT INFECTION. Suppurative arthritis although not very common nowerdays, frequently leads to Secondary O.A., especially in thsoe cases where complete resolution fails to occur.

(3) RHEUMATOID ARTHRITIS. This disorder of joints very frequently leads to damage of the articular cartilage in the joint, thus O.A. is quite common.

(4) JOINT and BONE DEFORMITIES . Deformities of joints or bone may be either congenital or aquired. Whatever the cause, unless corrected early, the deformity will lead to joint derangement and damage with consequent osteoarthrosis.

(5) BLEEDING DISORDERS. Haemophilia and other bleeding disorders in which there is repeated haemorrhages into the joint may lead to permanent damage with fibrous ankylosis and secondary asteoarthrosis.

(6) OBESITY. This may be considered to be one of the most important predisposing causes of O.A. The mechanism is well known. The increased weight leads to more stress on the joint thus, the wear and tear process is accelerated.This is the most common predisposing cause of O.A. on the Island, considering the very high incidence of this condition in the population.

(7) Other less common causes of secondary O.A. include Congenital Hip Dislocation not diagnosed in time, unresolved Perthe's disease, Osteochondritis, and other rare disorders of joints.

Management of Osteoarthrosis

Generally speaking the management of O.A. is difficult for the simple reason that the changes that occur in the joint are all irreversable.

There are mainly three forms of therapy for O.A. two of which are conservative and the other operative.

(a) Conservative Measures.

(I) No treatment except for reassurance is all that is required in a good number of cases.

(II) When the condition of the patient is more advanced some form of treatment will usually be required. There are mainly two forms of treatment for these cases. The first is by various forms of physiotherapy employing assisted exercises of the affected joints under the guidance of a physiotherapist. This method is beneficial in a good number of cases and apart from improving function it also helps to prevent rapid changes due to prolonged immobilization. The other form of treatment is by the use of the traditional NSAID (non steroidal anti-inflammatory drugs). There are various drugs in use including aspirin, indomethacin, butazolidine, naproxen, ketoprofen and others. The role of the NSAID in O.A. is rather limited and after prolonged use a number of complications may arise. On the whole the best drug for O.A. seems to be indomethacin.

(b) Surgical Measures in O.A.

There are a variety of surgical procedures which are in current use in the management of O.A.

The aims of surgery in this disease are the following:

(I) Abolition of pain which is of severe intensity and which is refractory to the conservative forms of treatment.

(II) To correct deformities both for functional as well as for cosmetic reasons.

(III) To improve the function in the deranged joint.

(IV) To improve stability in the affected joint.

Any one of these criteria may be an indication for reconstructive surgery. Another important point is the progression of the disease. In those cases where the condition is rapidly deteriorating even in the absence of symptoms, surgery is usually indicated.

The basic features of the various operations in

current use are summarised below:

(I) OSTEOTOMY
- — good pain relief.
- — joint left intact.
- — further procedure still possible.
- — no improvement in joint stability.
- — long term results uncertain.
- — slow post operative recovery.

Complications:
- — non union of osteotomy with instability.
- — makes later arthroplasty difficult.

(2) ARTHRODESIS.
- — pain relief guaranteed.
- — maximum joint stability.
- — long term results good.

Complications:
- — total loss of joint movement, and extra strain on other joints.
- — slow post-op recovery.
- — non fusion or fusion in bad position.

(3) TOTAL JOINT REPLACEMENT
- — pain relief is usually excellent especially the hip joint
- — mobility retained or improved
- — quick post operative recovery.
- — normal function restored in good number of cases.

Complications:
- — loosening of the components.
- — infection during joint replacement.

Although the total joint replacement is the ideal operation, there are other forms of arthroplasty which may be used and these include the Half-Joint replacements and the excision arthroplasty.

In the last few years there has been a great improvement in the techniques of joint prosthesis, however the only considerably good results are those for the Hip Joint. The use of prosthesis in other joints such as the knee, elbow and shoulder are still in the experimental stage and the evidence for this is the large number of prosthesis which appear on the market each year, many of which have nothing to offer.

In general one may say that the great advances in Anaesthesia and general surgical techniques the option of surgery is being offered to a large number of patients, many of which are elderly.

In conclusion one may say that the overall incidence of osteoarthrosis seems to be decreasing due to the better treatment of those conditions which may potentially cause osteoarthrosis in later life. Another reason for the decline is the fact that the greater part of the population is now-a-days aware of all the complications that may result from obesity amongst which is osteoarthrosis.

As regards the progress in the forms of treatment, these are bound to continue as many of these procedures are in current use for other forms of arthropathies.

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Visually Handicapped Children

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This is a numerically small but seriously disabled group. Visual disorders arise from a multitude of causes either within the eye itself or in those areas of the brain that deal with the interpretation of the visual phenomena.

Blind children have to learn from attentive listening, smelling, touching and manipulating whatever lies within arm's reach. They generally dislike soft toys; they need playthings which provide good *feelable* shapes and textures, and above all a meaningful language. It is enough for normal individuals to close their eyes inorder to appreciate the grevious deprivation of the totally blind. It is more difficult to understand the baffling, visual world of a partially-sighted child who may possess a few patches of the visual field; this may prove highly useful for near vision but the child has little notion of distances, perspectives and spatial relationships and therefore cannot readily appreciate our predominantly visually organized world.

Blind children are easily distracted by sound; they will be unable to focus attention on meaningful elements in the environment for long, and so, in self defence, tend to drift into a state of non-attention.

Young blind children unable to see how both hands function together may be unaware of how to bring this about. Even when bilateral function is explained and illustrated to them, they may still need to feel the position of one hand relative to the other. If in addition, they have a central disability for spatial recognition, their learning problems are indescribably complex.

People whose visual perception is very poor are still, however, able to lead full and happy lives, have a full time job and look after their own families. To do these things and to have a chance of complete independence, one must be very patient at training them to use the other senses to their full capacity in an attempt to make up for the other sense which they have not got.

What follows are some practical guidelines which may prove helpful at some time or another:

1. Blind children need more help than other normal children. They have to be taught to do things that normally would be easily learnt by copying. They must develop all the other senses to the maximum efficiency.
2. They must be cuddled and frequently talked to. They need to know that you are there!
3. Always tell them when you are going to touch them, or to do something startling. They cannot see what you are doing. They also appreciate your telling them what is happening around them. Hold their hands when something frightens them.
4. Always try to be honest and truthful. These children must be able to trust their helpers. Let them hear you laugh when you are playing with or teasing them. They cannot see your smile.
5. Give them things made out of solid, robust materials like wood and metal. These are better things to be manipulated than soft toys are. Tie the toys to the cot or play-pen so that the child can find them if they are dropped accidentally. One must ensure that very small objects are kept away from infants because they may end up swallowing them or even choking. Common items are preferable because these are the things they must learn to appreciate and identify!

6. Help them identify the various things around them. What is wet? What is gritty? What is sticky? What is soft? What is hard? What makes certain noises?
7. Let them help in the kitchen. This is a good place to use the senses of touch and particularly taste and smell. One may fit a guard to the top of the cooker and turn saucepan handles away from the sides and front to avoid any danger as much as possible.
8. To improve their sense of direction, you can play games such as by calling them and letting them find you whilst you keep calling.
9. To help them learn, let them do things with you. Let them dress and undress by themselves as well as wash and dry themselves. Help them get used to stairs but have a safety gate when they are exploring on their own.
10. Always inform a blind person when you have moved a piece of furniture.
11. Take them out and give them the opportunity to play outside in all weathers. Let them enjoy the company of other friends even at the cost of them getting a few bumps and bruises.
12. It is important for them to distinguish boys from girls. Let them help in bathing babies.
13. In case of partially sighted children ensure that they have proper glasses to wear at the times they should. If more than one member of the family wears glasses make sure that they are wearing their own and learn to recognise them. A spare set of glasses is advisable especially if the child cannot do without a pair. The glasses must be checked regularly to ensure they are clean, not scratched and properly maintained. Eyes have to be seen by the ophthalmologist and new glasses should replace old ones, if necessary.

Here are a few tips to help you guide a blind person:

The grip or linking position.

The blind individual is one step behind the helper, for better direction. The helper's arm is straight. The blind person's arm is bent holding the helper's just above the elbow.

Steps and staircases

Whether ascending or descending, the guide should be one step ahead, thus preventing the blind person from taking an extra step into thin air!

When going up, always inform the disabled by saying *steps up.* Walk in rhythm, one step infront with your partner's gripping arm being slightly stretched forwards and upwards until you reach the top. After having ascended the whole flight of steps, take a large stride forwards and stop, allowing the blind person to negotiate the last step. As he feels his arms resume its normal level of grip, he will know that he is on the landing. Likewise, when going down say *steps down.* Otherwise the procedure is very similar to going up.

Entering cars

The blind individual simply has to be helped by placing your gripping hand on the passenger door handle and tell him which way the car is facing. His hand then slides down your arm and locates the handle while the other find the roof.

Getting out of cars

You must check for the passenger whether the road is clear before opening the door. Then he can open the door with one hand and find the roof with the other.

Walking in single file

One often needs to do this in busy areas like shops and restaurants. Indicate the need for your partner to step in behind you by moving your gripping arm to the middle of your back, keeping your arm straight.

Doorways

The helper would have to go through the doorway first; the blind partner follows and closes the door. Take a look at the door, if the hinge is on the left, the partner should be on your left side with his left had free. Describe it as *door left.* As you turn the handle and move the door, the blind person can distinguish whether it is moving inwards or outwards. Inform him if the door is a swinging door.

When you are opening the door, do so with your gripping arm; otherwise your partner will be unaware of your actions.

Sitting

If possible approach the chair from in front, centrally, but whichever way you approach it, always place your grip hand on the back of the chair. Let your partner slide his hand down your arm to the chair back, then he can sort the rest out by himself. A blind person usually feels the side of the chair with the calf of his leg and checks the chair depth with his hand.

Conclusion

The day-to-day problems of parents of handicapped children have much more in common than in difference, while their advisers need to possess more virtues than compassion. After all their child is handicapped and for many years will continue to require the same affection, encouragement, patience and sensible discipline subject neither to over protection nor to intolerable strain.

Workers need to shed their ideas and to look, listen and record truthfully what is really happening and what additional measures could be undertaken to improve the situation. I pray to the Divine Lord to endow each and every one of us during our tasks in helping these beloved children so that through the medical and nursing staff we can help in reducing the handicap to a minimum and develop their capabilities to the full. Moreover, we must be endowed with intelligence, patience and sensitivity but above all with that important combination of insight, foresight and humour, it other words ordinary common sense!

Just because a child is handicapped, this does not imply that his life and that of his family are going to be devoid of happiness!

Epidural Medication for Relief of Low Back Pain

A Clinical Study

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On a sunny day in Africa 10 million years ago, give or take a few million, Mr. and Mrs. Ramapithecus and their children were out foraging for food. Like their primate cousins in the forest, they usually swung gracefully from limb to limb searching out nuts, fruits and berries. But this day was different. A fierce rainstorm had knocked all their favourites off the branches, and the Ramus, alas, were forced to descend from the trees to find something to eat.

Moving awkwardly on all fours, knuckles bent, they were ungainly creatures on the ground and also extremely vulnerable. Barely 3 ft. high, unable to see over the tall grass, Ramu suddenly found himself and his brood confronted by a snarling saber-toothed tiger. What to do?

The forest was too far off to dash to safety. So, in an inspired gesture, Ramapithecus reached for a rock with both forefeet, reared back on his hind legs and heaved the stone at the predator. Started to see this usually four-footed prey erect, the tiger cautiously retreated. But the apeman's triumph was costly. Unaccustomed to the abrupt, unright position, he was left doubled over in agony with a piercing pain in his lower back. (Time-Magazine, Cover Story, That Aching Back, p.30, 14 July, 1980).

Low back pain in a universal affliction. The cost of treatment, payments for litigation, compensation, quackery, and lost man hours render it also a significant burden to the economy and a major health concern worldwide. In terms of human suffering from chronic pain the cost is incalculable'. It has been shown that chronic pain and its intensity correlate well with high indices of psychological depression. A great number of treatment modalities such as transcutaneous nerve stimulation (TNS), acupuncture, biofeedback, psychotherapy, surgery, drugs, and epidural medication are currently used for the alleviation of low back pain. It has been demonstrated tht intrathecal administration of narcotics would suppress a variety of responses to pain in cats and monkeys[3].

Since then, the clincial application of both intrathecal[4] and epidural[5] morphine for treatment of different pain conditions in man has been reported. Also, epidural administration of different narcotics has been evaluated[6]. The Continuous epidural technique would seem to have particular advantages over intrathecal administration in the treatment of chronic pain in that it permits initial titration of the effective dose, incremental doses, and prolonged medication[7].

Experimental observations indicate that the epidural opiate analgesia is produced by a specific opiate receptor interaction rather than a non-specific axonal blockade[8]. This study was designed to evaluate the efficacy of 2mg morphine together with 5ml 0.25% bupivacaine administered epidurally in relieving low back pain.

Patients and Methods

A total of 34 male patients, physical status ASA I, gave informed consent after the procedure was explained in details. Table I summarizes the patients' data. All of our patients complained of low back pain and all have been on intermittent rehabilitation and drug therapy with mild analgesics. None of them had a past history of drug allergy. *Table 1* An epidural catheter was introduced at the L_2 - L_3 or L_3-L_4 level and advanced 2-3 cm cephalad. The catheter was fixed to the patient's back and a millipore filter was attached to the free and resting in the supraclavicular fossa. Treatment consisted of an epidural injection of 2mg morphine sulphate together with 5ml 0.25% bupivacaine followed by 3 ml of normal saline from a separate syringe. Patients were kept for observation 2-3 in the out-patient clinic and afterwards escorted home. Two days after the first dose patients were evaluated.

Those patients who had partial or no relief were reinjected with the same dose and if there was further but not complete relief, a third injection of the same dose was given. If no improvement was noted following the third dose, treatment was discontinued. In patients with complete or nearly complete relief after one week no further injections were administered. The out come of the treatment was assessed on the basis of patient's subjective pain ratings as having no relief, slight, moderate or complete improvement. More objective findings, such as improved straight leg-raising, increasing mobility, and mood improvement were noted. Treatment was considered successful if moderate or complete relief for a month or more following the course of treatment was obtained.

Results

The results are summarised in Table II. The overall treatment success of our study inversely correlated with the symptom duration. In none of our patients

TABLE 1. PATIENTS' DATA

No. of patients:	34
Sex	
Male:	34
Female:	0
Age (yr.)	
Range:	29-46
Mean:	37.4 (6.4)
Height (cm.)	
Range:	164-178
Mean:	169 (5.5)
Weight (kg.)	
Range:	66-92
Mean:	76 (6.9)
Duration of low back pain (months)	
3-12:	24
12-24:	6
24-32:	4

TABLE II. TREATMENT OUTCOME

No. of injections:	Moderate to complete improvement:	Partial improvement:	No improvement:
I	16	4	14
II	19	3	12
III	19	3	12
TOTAL:	19(55.9%)	3 (8.8%)	12 (35.3%)
Improved straight leg-raising:	14 (41.2%)		
Increased mobility:	18 (52.9%)		
Mood improvement:	13 (38.2%)		

were vomiting, urinary retention, constipation or respiratory depression noticed following epidural administration of morphine. Two patients suffered a transient attack of itching on the abdomen and thorax. *(Table II)*

Discussion

The human back is a fascinating intricate system of muscles, tendons and ligaments which keeps the vertebral column from collapsing. Yet, low back pain strikes almost everyone, the young and the old, both sexes, and the people of different occupations and social profiles.

Chronic pain is invariably accompanied by depression[9] and its treatment may be the most important aspect of pain management in some patients. With time, patients with chronic pain develop pain behaviour patterns that may become more harmful than the original nocioceptive substance[10]. Consequently, the treatment of low back pain is undertaken by many specialities and involves a number of interventions rather than a single therapy. Conservative treatment focuses mainly on adequate bed rest. It has been shown that both recumbency and decreased lumbar lordosis help to diminish intradiscal pressure [11,12]. In a recent study, however, it was demonstrated that both the *orthopaedic* hard bed and the waterbed were equally effective in alleviating low back pain, which appears to be in discordance with traditional teaching[13]. The effectiveness and advantages of the use of epidural opiates in different pain conditions in man have been repeatedly emphasized[14],[15]. Some reports, however, are casting doubts on the safety of epidural opiates [16] [17]. In our patients, none of the commonly reported side-effects like vomiting, constipation, urinary retention or respiratory depression were noticed. A transient attack of pruritus was the only side-effect reported by two patients. Patients whose pain had been present for less than a year prior to treatment were those who benefited most. Perhaps the pathological process in those patients has not reached the point of irreversibility as proposed by Murphy[18].

Very little is known concerning eventual neurotoxic effect of epidural opiates. It has been shown, in a postmortem histological investigation of some 30 patients that after long treatment with epidural morphine no neurotoxic effects on nerve roots or spinal cord were detected[19]. We have mixed morphine with bupivacaine because of the previous impression that a local anaesthetic agent may facilitate the binding of epidurally applied morphine to the apiate receptors alongside the scope of its action[20].

SUMMARY

The effectiveness of epidurally applied 2mg morphine sulphate together with 5ml 0.25% bupivacaine in the relief of low back pain was assessed in 34 male patients. Based on a patient's subjective pain ratings, moderate to complete improvement, lasting more than a month, was achieved in 19 (55.9%) patents. Treatment success inversely correlated to symptom duration. It is concluded that epidural medication with low dose morphine together with bupivacaine could be an effective tool in relieving low back pain either as a single or in addition to other forms of therapy.

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A Summary on Anxiety and Phobic Neuroses

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Although anxiety and the phobic neuroses are classified as separate entities, most patients with phobic anxiety also suffer from an elevation of their general (free floating) level of anxiety, and nearly all patients with generalized anxiety may experience an aggravation of their anxiety, often to panic intensity.

ANXIETY NEUROSIS.

Mild anxiety is an everyday experience which may be of use to the individual; if severe it is destructive. It is willingly sought by those who engage in dangerous sport and by those who watch them; the neurotic patient attempts to avoid it at the cost of severe handicap of his life style. The applications of this ambiguous word include:

1) An emotional state with subjectively experienced fear.
2) An unpleasant emotion which may be accompanied by a feeling of impending death.
3) Anxiety is directed to the future, implying that there is a threat.
4) There may be no recognizable threat, or one which, by reasonable standards is out of proportion to the emotion it provokes.
5) There may be subjective bodily discomfort and manifest bodily disturbance.

Anxiety must be considered under the two concepts of trait anxiety (an enduring aspect of the personality structure) and state anxiety (a temporal disorder). This must be considered because the management of the two forms is different, although they often occur together. Trait anxiety may be partly genetically determined and partly the outcome of early experience of the individual. State anxiety may result from stress or conflict (acute or prolonged) or it may arise in the absence of any sufficient stress -- an endogenous affective disorder. It may also be due to some processes such as premenstrual tension, disease of the limbic structures, thyrotoxicosis or tumours leading to overproduction of adrenaline. It may also result from ingesting excessive caffeine or other drugs like amphetamine. Anxiety may be experienced entirely as psychic discomfort characterized by apprehension, diffuse sweating, fear and panic. It may also be somatized: muscular pains, tension headaches, tremor, palpitations, diarrhoea, respiratory distress, feelings of dizziness, swaying and 'walking on cotton wool'. In mild degrees, anxiety may be no more than a slight awareness of discomfort. However, in clinical practice, it may present in an infinite number of variations.

Anxiety, Depression and Affective Disorders.

The feasibility of a true distinction between anxiety states and depressive illness has been considered in three broad areas: clinical features during the key illness, outcome studies and treatment studies.

Clincial Features

Various independent researches have led to the coinage of the word 'anxiety depression'. Although depreciated by some researchers, there does seem to be a considerable admixture of anxiety and depressive symptoms in most patients examined. One such important investigation used a self-report questionnaire divided up into five main areas of symptomatology: somatic symptoms, obsessions, interpersonal relationships, anxiety and depression. Apart from the somatic scale, it was found that the depressed patients recorded themselves as being severely disturbed on all the scales, including the anxiety scale.

Outcome Studies

An investigation of the outcome of 111 patients presenting with affective illness dominated by anxiety symptoms over a period of up to 2 years was carried out. All patients had received the same treatment; one large group in this sample had suffered from a sudden onset of anxiety without major precipitant stress and the prognosis for this group was much better than for the rest: this form of anxiety state was regarded as being basically depressive in nature. In another study of 112 patients diagnosed as anxiety neuroses, it was found that 44% subsequently suffered from depressive episodes.

Treatment Studies

There is yet the possibility that treatment response will serve to redefine clinical categories, at least in the area of affective disorder, but at present, the observation that some types of anxiety state respond well to several antidepressants, still causes confusion. In a study noting the response of phobic anxiety patients to phenelzine (on M.A.O.I. antidepressant), the following possibilities were considered:

a) that phenelzine is an antidepressant and phenelzine-responsive patients suffer from a depressive illness.
b) that phenelzine is an anxiolytic and responsive patients are primarily anxious.
c) that both depression and anxiety patients may be manifestations of the same core affective illness and that phenelzine is an anti-affective drug.

In other studies, further possibilities were considered:
1) Anxiety and depressive states have different

biological substrates, but that much of the symptomatology of the conditions is similar.
2) The two conditions represent the same reaction to stress but the characteristic symptomatology reflects the personality structure of the individual.
3) Chronically anxious patients become depressed and chronically depressed patients develop anxiety.

PHOBIC NEUROSES

Background to present concepts.

The term phobia is derived from the Greek word *phobos* meaning fear. Since antiquity, there have been descriptions of individuals who suffered from a morbid degree of fear of circumstances which would not be expected to call forth such perturbation. When it came to Freud, he divided the group of phobias in 2 classes, common (Exaggerated fears of things which are to some extent, feared by everyone, such as snakes and death) and contingent (fears of special conditions which inspire no fear in the normal man).

Concept of phobias as learned symptoms.

Many experiments have shown that fear responses can be produced by conditioning techniques. Miller (1948) showed that fear itself could act as a drive for further learning, and he demonstrated that escape from fear (drive reduction) served to reinforce learning. Symptomatic behaviour, especially phobias came to be viewed as a learned response to aversive stimuli. In 1958, Wolpe introduced the concept that, if a response incompatible with anxiety was introduced whilst the patient was exposed to the source of his anxiety, then on repeated performance, the fear would be gradually extinguished. On this principle he developed the therapeutic procedure known as 'systematic desensitization'. Neurosis was defined as 'any persistent habit of unadaptive behaviour acquired by learning in a physiologically normal organism'.

Definition, epidemiology and classification of phobias.

Marked fears of certain objects and situations are widespread in the population and there is no clear dividing line between a strong fear of a situation and a phobia. Therefore, a phobia can be defined on the following criteria:

1) Fear is out of proportion to the demands of the situation.
2) It cannot be explained or reasoned away.
3) It is beyond voluntary control.
4) The fear leads to an avoidance of the feared situation; or else if unavoidable, leads to extreme discomfort on the part of the individual.

A most informative survey on the prevalence of phobias showed it to be 76.9/1000, and of these 2.2/1000 were considered to be severely disabling. The most common phobias were of illness or injury, followed by storms, various animals and agoraphobia; most of the phobias were commoner in women than in men. A five-year follow-up of this survey showed that in children and adolescents, phobias improved without specific treatment, whereas more long-standing phobias in adults naturally have a poorer prognosis.

The Phobic neuroses were classified as follows by Marks (1969):

1) Phobias of external stimuli.
   a) Agoraphobia
   b) Social Phobia
   c) Animal phobias
   d) Miscellaneous.
2. Phobias of internal stimuli
   a) Illness phobias
   b) Obsessive phobias.

Some difficulties with such a scheme are now to be considered.

Agoraphobia

Means a fear of gatherings of people in the open; however, the term is usually applied when the patient has at least one or more fears: leaving home, wide open spaces, being alone, travelling, narrow confined spaces etc. In addition to the central fears, it is assumed that the patient shows a high level of general anxiety, and may experience panic attacks, somatic symptoms of anxiety and somatic depersonalization. In addition there may be a difuse collection of other phobias. It is therefore clear that patients may be classified under the label of agoraphobia who in fact suffer from a range of disorders.

Social phobia

Any large group of patients with phobias concerned with interpersonal situations, will be found to suffer from a degree of generalized anxiety intermediate to the groups classed as agoraphobia and those with specific phobias. Many patients with social phobias experience a marked degree of somatized anxiety in their feared situation (either developing the phobia in the setting of a traumatic situation, or else the phobia may originate in a state of depression or anxiety which may have resolved) such as blushing or hand tremors. Social phobia is therefore not a homogeneous clinical entity, but represents the prominent symptomatic manifestations of a wide variety of psychological disorders and psychiatric illnesses.

Specific phobias.

These are seldom so specific as the appellation may signify: there is often a generalization of fear to similar objects, so that the patient with a phobia of cats also dislikes the touch of all furry objects. Mood disturbance is clearly evident in many patients, who thus suffer from generalized states of anxiety or depression.

School phobia.

School phobia is not a homogeneous disorder, since children with this symptom show a varied clinical picture of diverse aetiology. Onset may be sudden or gradual and may or may not coincide with a particular upset at home or at school. There will normally be a marked somatization of anxiety, often abdominal pains and sickness or diarrhoea occurring before the hours of school attendance. When at

home, the child does not appear to be severely anxious. In many cases, a variety of personality disorders, relationship problems, neurosis in the parents and specific problems at school are found in varying proportions.

Aetiology of phobic neuroses.

Many factors contribute, among these being the constitutional liability of the individual to develop fear reactions, the personality structure, the selection and successful employment of coping mechanisms in the early stages of neuroses, the attitudes of family, friends and professional advisors, the potential threat, the presence of a mood disorder, and finally the presence and degree of secondary gain from the neurosis.

Personality structure

Some degree of abnormality of a specific personality trait underlies the liability to develop a phobic neurosis, though this is not universally present. With certainty, it can be said that patients suffering from phobic neurosis show some degree of dependency on others, and the more generalized the neurotic disturbance, the more likely is dependency to be pronounced. Many adults showing phobic symptoms in the setting of a general mood disturbance, are likely to have suffered from phobic problems in childhood, and it is probable that the majority of patients who develop social phobias, do so in the setting of some degree of personal sensitivity to the opinions of others.

Attitudes of the individual and the reactions of others.

The outcome of a fear and whether it hardens into a phobic neurosis will depend to a large extent on the attitudes of the individual and the reactions of others to his fear.

Mood disorder and affective illness.

A temporary disturbance of moods may provide the fertile soil in which the seed of a phobic neurosis may first germinate: the phobia may then subside as the mood returns to normal, or it may persist as a permanent aftermath of the mood disorder. Many phobic neuroses commence during times of unhappiness, insecurity or conflict which causes a reactive anxiety or depressive state, and in this setting, a trivial incident may be sufficient to cause a prolonged phobia. If a severe attack of panic occurs in some non-threatening situation, the patient comes to associate that situation with his panic and his apprehension leads to further panic and eventually to phobic avoidance.

Treatment of anxiety and phobias.

The treatment of anxiety based neuroses must rest upon a careful formulation of the condition of the patient. The correct therapy is selected after a careful assessment of the following factors:

1) The degree to which the manifest anxiety is a reflection of the basic trait anxiety of the individual.
2) The presence or absence of stress which is related to the occurrence of the anxiety. An attempt should be made to assess how the patient has coped with similar stress at earlier periods in his life.
3) The present and past life-styles of the patient, the range of his activities and the quality of the ralationships with others.
4) The duration of the neurosis and the past history of fluctuation in severity and recurrence of symptoms.
5) The presence of any particular strategies which the patient uses to cope with his symptoms.
6) The attitudes of others towards the patient's neurosis.
7) Apparent secondary gain from the neurosis.
8) The presence or absence of other psychopathological features like depressive symptoms.
9) Response to treatments that have been used for similar neurotic symptoms in the past.

After a formulation of the patient's case, the therapeutic approach must be considered, but sometimes a delay is advisable: it may be necessary to see a relative, special investigations may be required, and moreover, the process of a psychiatric examination may in itself have therapeutic effect.

Psychotherapy.

This should be carried out at weekly sessions each lasting about thirty minutes: time should be spent in considering what elicits anxiety, coping strategies which might be adapted.

Behavioural psychotherapy.

This involves behavioural techniques in a clinical setting. In the treatment of phobic neuroses, systematic desensitization is found to be effective in specific phobias only. The other major behavioural technique for the treatment of phobias has received the name 'flooding' and starts by encouraging the patient to experience anxiety and to confront his neurotic fears more abruptly (exactly the opposite of systematic desensitization).

Pharmacotherapy.

This can often be dramatically successful. In acute generalized anxiety, sedative drugs may be justified for a strictly limited period of time; they may also be effective in helping a patient to overcome a phobic state as long as he clearly understands that the drug is prescribed to help him face up to his anxiety, and should only be taken when he attempts to enter the feared situation. Sedative drugs are ineffective if there is an underlying affective disorder — this requires the prescription of an antidepressant drug. Drugs which are also established as having a role in the management of some anxiety - based neuroses are the β-adrenergic blockers.

Psychosurgery

Patients should only be considered for surgery if they have suffered from chronic anxiety states for many years and who have remained unresponsive to any therapeutic procedure.

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A General Practitioner's Emergency Bag

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When I was in recent years, doing emergency duties at the Polyclinic in Floriana, final year medical students who used to accompany us on emergencies, were, quite naturally, interested to know what a general practitioner should carry in his emergency bag. This is the sort of question I asked myself many times before qualifying some 24 years ago! (How time flies!)

I would like in this paper to give my personal views and choice of medical requisites a general practitioner should *wisely* carry in his bag.

First of all I would like to make it clear that when one is called on an emergency, one usually hears the voices of hysterical relatives yelling and shouting on the phone wanting a doctor immediately. Try to keep your calm, and above all try to calm down the caller! Tell him (or her) to give you exact details as to the address where you are needed; try to extract from him what the emergency is, and if you don't know the anonymous caller get from him the phone number he is ringing from. I usually encourage them to tell me what the problem is so that I would take along any medicines the patient is likely to need.

What I have said in the above introduction, shows that one simply cannot devise a *utopian* bag which can deal with all emergencies. Emergency calls are usually made late in the evenings, at night, on weekends and public holidays, in other words, when the pharmacies are closed.

What are the symptoms, that worry patients and relatives most to make them resort to emergency phoning? These, quite naturally are:- considerable and unusual bleeding, sudden onset of severe pain, coma, fits, syncopal attacks, breathlessness, very high temperatures (with rigors), marked vomiting and diarrhoea, generalized urticarial skin, rashes, etc. One's temptation would be to ring for an ambulance straight away, but in my experience a good 75% of such would-be emergencies can be adequately tackled at the patient's own home, if one has the will, skill and basic medical requisites with him.

The following illnesses are commoner at night:- pulmonary oedema, renal colics, status asthmaticus and painful physical conjunctivitis from welding flashes, plus, of course, crying restless babies!

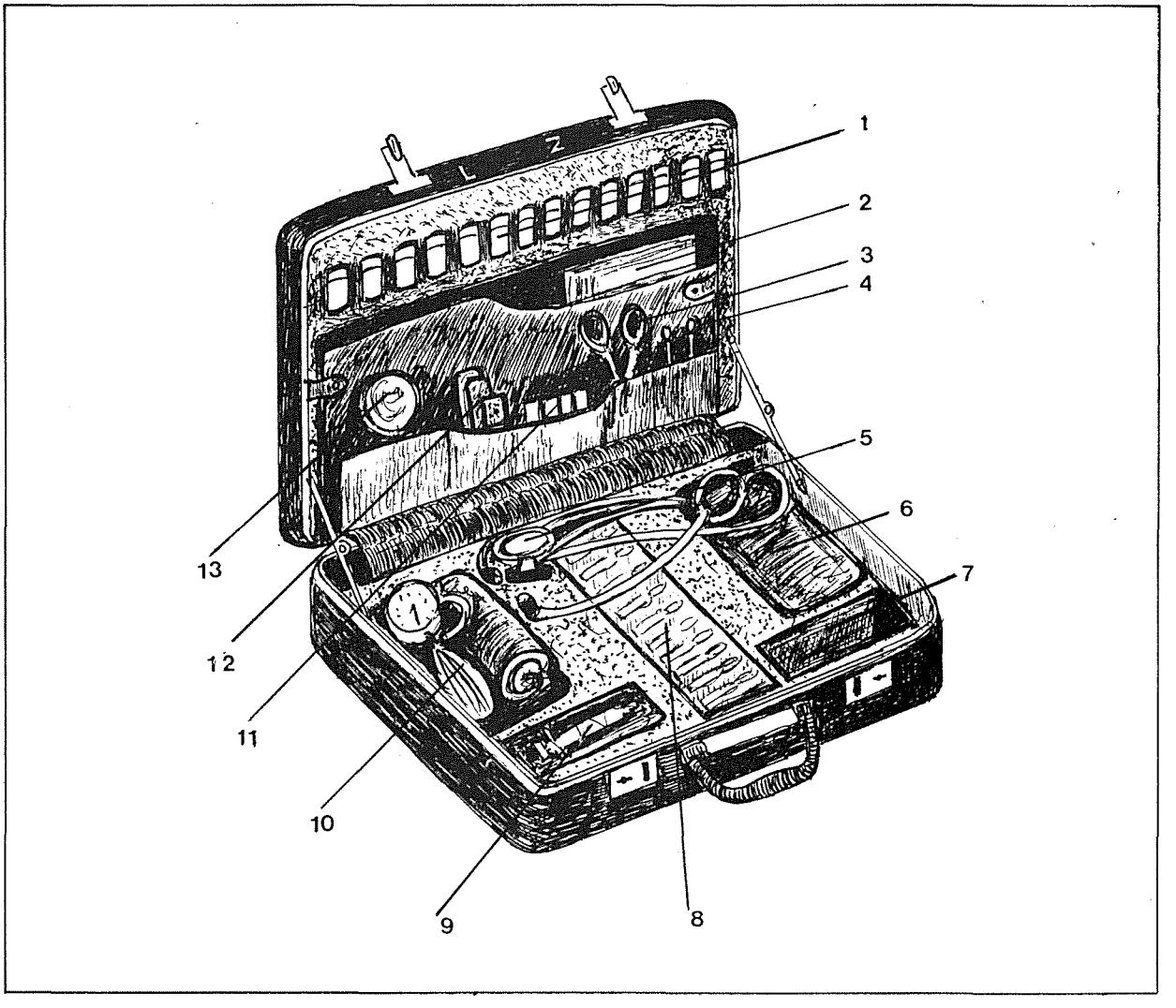
Having said this, I would like to catalogue the sort of requisites I carry in my case (see figure):-

1. Small labelled plastic bottles containing 'starter' tablets as follows: *Avomine,* Codeine, Penicillin, *Equivert, Largactil, Norgesic, Panadol, Negram, Avafortan, Saventrine, Valium,* pethidine, Erythromycin & Tetracycline.
2. Personal letter-headed prescription forms and other stationery including tickets of admission to hospital etc.
3. A small pair of scissors.
4. Two thermometers.
5. A lightweight stethoscope (bell and diaphragm).
6. A box containing a small Auriscope.
7. A spare compartment. This is useful to put in anything which one thinks might be needed e.g. children's suppositories, antibiotic injections, bottle mixtures, eye drops, etc.
8. A box with a transparent plastic lid top containing the following injections; Adrenaline, Dexamethasone, Digoxin, Ergometrine, *Largactil,* Frusemide, Morphine, Atropine, Pethidine, *Valium,* Theophylline or Aminophylline, Lignocaine Hydrochloride *(Xylocard), Avafortan* and *Sosegon.*
9. Disposable sterile plastic syringes plus some cotton wool swabs.
10. Anaeroid Sphygmomanometer.
11. Elastoplast dressings. Glucose and Albumin test strips.
12. A rubber band *velcro* tourniquet for i.v. injection purposes.
13. A small hand lens (remember that ageing G.P.'s are by no means immune to presbyopia!)

I will now suggest some DO's and DON'Ts

1. Do try to keep your bag (Attaché-case sounds to me more updated!) as light to carry as possible. Remember that most of our blocks of flats have long flights of stairs and no lifts, (or, as the Americans would say:- no elevators!)
2. Do remember to replenish your bag (case) with the items you would have used, immediately on returning to base (home).
3. Do mark your case with your initials and preferably your home address.
4. Do Spring clean your case very often and remove expired injections or tablets, remove dust and fluff plus pieces of cotton wool, that accumulate over the weeks.
5. Don't leave your case in your car to the mercy of the hot summer sun as most of your medicines will lose their potency and your thermometers will break on exceeding maximum temperatures.
6. Don't leave your case easily visible on your car seat when out in the evening, but leave it well tucked and hidden in the car booth. Many doctors had their car windows broken by drug addicts in the aim of acquiring morphine or pethidine on the *quick*. Remember that drug addicts know where it is most likely to find drugs and in desperation resort to anything to have an immediate supply.

I would like to end this article with this anecdote. An ex-Chief Consultant of mine once told me, "I can recognize the chronological academic age of general practitioners by the size of their bags. He explained that the newly qualified ones usually carry a very large one tucking into it whatever injections, tablets and medical paraphernalia thay might possess. The middle aged ones usually carry smaller lighter cases, because by now they have learnt the medical requisites one might need. Finally I can recognize the older ones, who discard their case completely, carry, just a stetescope in their pocket knowing that they are by now past doing emergencies and their solution is to simply ring a younger enthusiastic colleague to come to the rescue with his large bag of tricks!" This is a life cycle in a world of EXPERIENCE!



Diabetes:

Diagnosis and Classification

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SENIOR DIABETOLOGIST SLH.
LECTURER IN MEDICINE UNIVERSITY OF MALTA
PRESIDENT OF THE DIABETES ASSOCIATION

This article on Diagnostic Criteria and Classification has been based on the Second Report of the W.H.O. Expert Committee on Diabetic Mellitus, 1980.

General Considerations:

Diabetes may present with severe thirst, increased urine volume, rapid weight loss, and sometimes coma. Blood glucose concentration is grossly elevated. Glucose will be excreted in the urine, usually in large amounts. In this situation diagnosis is simply confirmed from blood glucose estimation without formal provocative tests. Random plasma glucose concentrations exceeding 2 g/l are diagnostic. The presence of specific microvascular disease, usually retinopathy, also establishes the diagnosis. When such symptoms and signs are absent and blood glucose levels less markedly elevated, measurements made under standard conditions, such as fasting or after a carbohydrate challenge, may be necessary to confirm or refute the diagnosis. Commonly the oral glucose tolerance test is performed. The importance of this testas a clinical diagnostic tool has been grossly overemphasized. It is useful only in clearly defined situations.

Table I shows diagnostic values for oral glucose tolerance test under standard conditions. Load 75g glucose in 250-350 ml of water for adults or 1.75 g/kg body weight (to a maximum of 75g) for children, using specific enzymatic glucose assay. Two classes of response are identified - diabetes mellitus and impaired glucose tolerance.

Proposed diagnostic procedure and criteria

The expert Committee recommended the procedure for diagnosis.

1. If symptoms of diabetes are present, perform random or fasting blood glucose measurement. In adults, random venous plasma values of 11 mmol/l (2.0 g/l) or more or fasting values of 8 mmol/l (1.4 g/l) or more are diagnostic. Random values below 8 mmol/l and fasting values below 6 mmol/l (1.0 g/l) exclude the diagnosis).
2. If results are equivocal, measure blood glucose concentration 2 hours after 75g of glucose taken orally after an overnight fast. Two hour venous plasma glucose values of 11 mmol/l (2.0 g/l) or more are diagnostic of diabetes. Values below 8 mmol/l (1.4 g/l) are normal and those in the range 8-11 mmol/l (1.4-2.0 g/l) are termed *impaired glucose tolerance.*

TABLE 1

	Glucose concentration		
	Venous Whole blood	Capillary Whole blood	Venous plasma
DIABETES MELLITUS			
Fasting	≥7.0 mmol/l (≥1.2 g/l)	≥7.0 mmol/l (≥1.2 g/l)	≥8.0 mmol/l (≥1.4 g/l)
and/or			
2 hours after glucose load	≥10.0 mmol/l (≥1.8 g/l)	≥11.0 mmol/l (≥2.0 g/l)	≥11.0 mmol/l (≥2.0 g/l)
IMPAIRED GLUCOSE TOLERANCE			
Fasting	<7.0 mmol/l (<1.2 g/l)	<7.0 mmol/l (<1.2 g/l)	<8.0 mmol/l (<1.4 g/l)
and			
2 hours after glucose load	>7.0-<10.0 mmol/l (>1.2-<1.8 g/l)	>8.0-<11.0 mmol/l (>1.4-<2.0 g/l)	>8.0-<11.0 mmol/l (>1.4-<2.0 g/l)

3. In the absence of symptoms of diabetes at least one additional abnormal blood glucose value is needed to confirm the clinical diagnosis (e.g. a 1-hour post glucose value of 11 mmol/l (2.0 g/l) or more during the first test or an elevated 2 hour or fasting glucose value on a subsequent occasion).

The Expert Committee recommended that the criteria outlined above be used as a guide to diagnosis until more detailed information becomes available on different populations, other diagnostic indices, and the development of complications. In the interim it is crucial that subjects with impaired glucose tolerance are not labelled "normal" and returned to the community by default. They have an increased risk of worsening to diabetes and of developing atherosclerosis. Different courses of action will be necessary depending on age, obesity, and the presence of other diseases. During pregnancy, the treatment for impaired glucose tolerance should be the same as for diabetes.

An Interim Classification

As an interim measure, the classification scheme prepared by the Diabetes Data Group of the National Institutes of Health, USA will meet many needs and is recommended for use. A simplified version is given in Table 2.

Measurement of Glucose in Blood

Whole blood or plasma can be used. Whole-blood values are approximately 15% lower than plasma values (except in anaemia). Note must also be taken of whether samples are capillary or venous. In normal subjects capillary values are an average 7% higher than venous values in specimens from fasting patients and 8% higher 2 hours after a glucose load.

The *bedside* estimation of blood glucose is now possible using various glucose oxidase methods, which may, however, give false values if the chemicals are not stored dry in airtight containers. The contact time of blood is critical with some versions of the glucose oxidase method. These methods are semi-quantitative. With all strip test and meters, careful attention must be paid to technique.

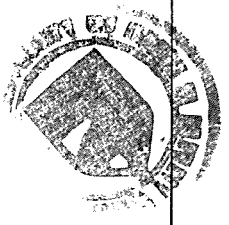
Oral Glucose Tolerance Tests

Formal dietary preparation is not recommended by most authorities unless the diet prescribes less than 125g of carbohydrate per day. In such subjects at least 3 days' preparation is advisable during which the intake of carbohydrate is limited to 150g per day.

The test should be performed after overnight fasting for 10-14 hours, although water is permitted. The first step is to take a fasting blood sample, after which the patient is given 75g of glucose in 250-350 ml water in 5-15 min. Special testing solutions are available for use instead of glucose. They consist of partial hydrolysates of corn starch and are less likely to cause nausea, but they are more expensive than glucose. Further blood samples are then taken 2 hours after administering the glucose, and some physicians also draw a one-hour sample. Smoking, eating and drinking must not be allowed during the test: It should be borne in mind that many factors (including drugs) can affect glucose tolerance.

TABLE 2

CLASSIFICATION OF DIABETES MELLITUS AND OTHER CATEGORIES OF GLUCOSE INTOLERANCE



A. CLINICAL CLASSES

Diabetes mellitus

Insulin-dependent type - Type 1
Non-insulin-dependent type - Type 2
(a) non-obese
(b) obese

Other types including diabetes mellitus associated with certain conditions and syndromes:
(1) pancreatic disease, (2) disease of hormonal etiology, (3) drug or chemical-induced condition, (4) insulin receptor abnormalities, (5) certain genetic syndromes, (6) miscellaneous.

Impaired glucose tolerance

(a) Non-obese
(b) Obese
(c) Impaired glucose tolerance associated with certain conditions and syndromes.

Gestational diabetes

B. STATISTICAL RISK CLASSES subjects with normal glucose tolerance but substantially increased risk of developing diabetes.

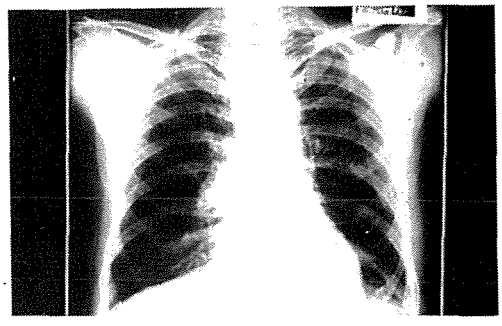
Previous abnormality of glucose tolerance
Potential abnormality of glucose tolerance

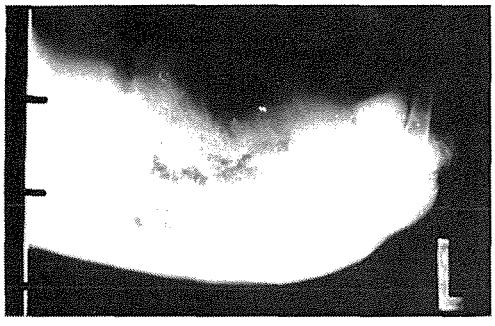
attach with answer

Clinical Diagnosis

This issue's *clinical tests* have been prepared by Mr. V.M. Sultana M.D. F.R.C.S., Senior Assistant in Surgery and lecturer in Surgery at the University of Malta. The photographs were kindly taken by Mr. Mario Sammut, a final year medical student.

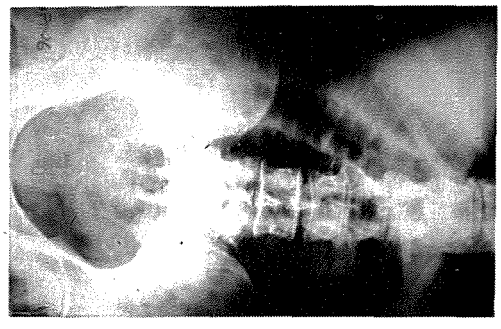
Case 1:





- What are your comments on these X-Rays?

Case 2: A 75-year old male with vague abdominal pains; no history of nausea or vomiting. Bowels were open in the morning. No other relevant symptoms. Past history revealed that patient was operated for left inguinal hernia 3 years prior to his presenting complaints. On examination, his general condition was good; **CVS:** pulse 88/min., regular and of good volume; Heart sounds normal; BP = 120/70; The JVP was not raised. **Resp. Sys:** Normal air entry; no adventitious sounds. **Abdo:** Slight tenderness in peri-umbilical region; slight rebound tenderness; bowel sounds present. **Investigations:** Hb: 14.8 g/dl, WBC = 14000/mm^3, urea = 40mg/dl, Na^+ = 132 mEq/l, K^+ = 4.5 mEq/l, Cl^- = 100 mEq/l, X-Rays vide infra:



- What are your findings from the X-Rays?
- How would you manage such a case?

The prize to be given to the first correct entry drawn by lot will receive an **Aneroid Sphygmomanometer**, kindly donate by **Aldox Ltd.**, agents for **Merck, Sharpe and Dohme.**

Result of "Clinical Diagnosis" in Issue No. 2:

Answer: The patient was receiving artificial Cardiac Pacing.
He (or she) is probably suffering from Complete Heart Block.

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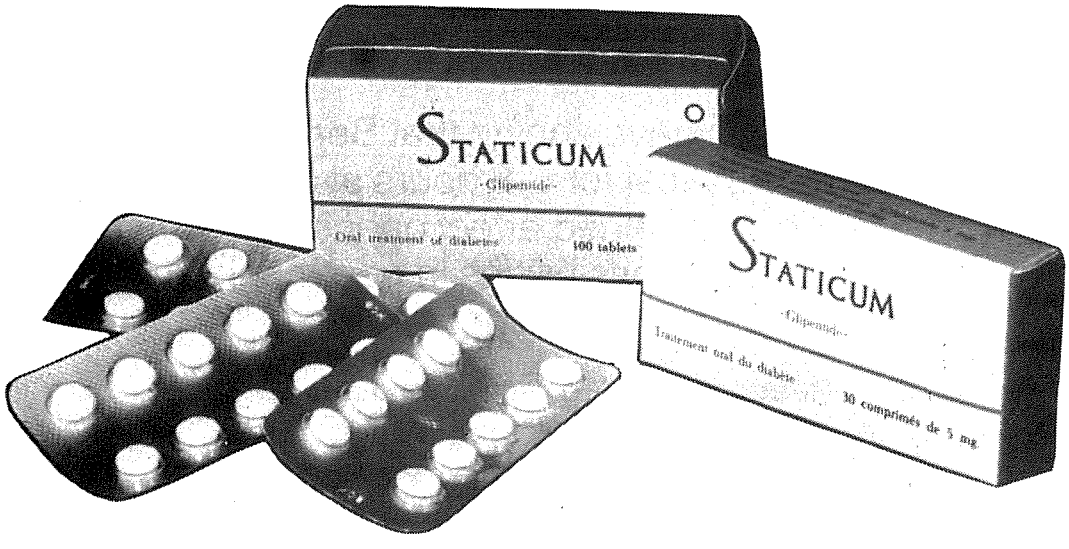

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