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SOME PROBLEMS OF HEAD INJURIES

by PRITAM DAS, Lucknow.

A Survey of the Knowledge of Head Injuries

The very early period: The very early period say before Hippocrates is not characterised by any reasoned, Scientific or recorded knowledge on the subject. What ever little was done by the surgeon of those days was a traditional sort of surgery. However, there are a number of points worthy of note and of considerable interest.

It is a well established fact that surgical operations were done on the skull in the Neolithic period. It fell to the lot of Prunieres (1965) a general medical practitioner in France to accidentally discover one such skull, and it was for Broca to prove that the hole in the Neolithic skull was made by human hands in the living condition. Such skulls were found in the caves in western Europe, and were subsequently discovered in North Africa, South Sea Islands, and Peru in South America. None of the skulls found in Western Europe showed any signs of injury for which an operation might have been performed, and these Neolithic holings were in all probability done for Epilepsy, Giddiness as well as for religious rites. However, in the Inca Skulls of ancient Peru, the great majority of skulls show evidence that the surgical perforations were performed for traumatic lesions of the head. The neolithic holings were done by scraping with a sharp piece of flint or obsidian. The Inca method was different and consisted of completely sawing through a square or quadrilateral piece of bone. There seem to be four principal methods of these ancient holings performed four to six thousand years ago: (1) Scraping. (2) Boring a circle of holes and sawing the intervening space (how similar to the modern method). (3) By furrowing the bone by push and plough method around in a circular or oval fashion and lifting up the intervening piece of bone. (4) By sawing out a quadrilateral button.

About 50 years ago an interesting custom was discovered amongst the Indians of Peru. They used to fill the wound with a powder of Coca leaves, and then they found that after waiting a while they could do what they liked with the wound without causing pain to the patient. It may be that the Inca and the Pre Inca races employed the Coca leaf powder as a local anaesthetic for performing their trepanation. These leaves contain some 9% of cocaine. Besides cocaine probably two other anodynes were the earliest to be used and they were Mandragora and opium, as is mentioned in Papyrus Ebers 1500 B.C.

Information about the methods of trephiners of Blanche Bay is comparatively recent, but in all probability their custom for operating for head injuries is equally ancient. The skull is first thoroughly washed with fresh Milk of a young coconut, the surgeons hands are similarly washed before the operation. A triangular incision is then made in the region of the wound, further operation consists of scraping and picking out loose pieces of bone and constantly washing out of the wound with fresh coconut milk. Subsequent to the operation the skin was stitched with

needle and thread and dressed and bandaged in an elaborate fashion.

Perhaps the earliest record of injuries to the skull in a Papyrus written by a surgeon in 1600 B.C. It is significant and interesting to note that the surgeon of that time knew that pulse may be affected by injuries to the head. Further that a depressed fracture of the skull may occur without an obvious wound on the scalp and that paralysis of limbs and loss of speech may occur in some casse of head injuries.

Hippocrates and after: Leaving behind this very early period we come upon a phase when accurate observations were made and put in writing for the benefit of those who could not be near the masters and for the benefit of posterity. Here we come upon the genius of Hippocrates. Hippocrates chronicled his experiences on the wounds of the head some time in the fifth century B.C. He described five varieties of fractures which included fissures of the bone, simple contusion, depressed fractures, penetrating lesions, and above all, he knew that fractures could occur by contrecoup injuries. He also noted the occurrence of convulsions after head injuries and pointed out that if the injury was on the left side the convulsions occurred on the right side and vice versa. He was aware of the sepsis occurring and the bone also getting infected and even necrosed. He advised trepanation of the bone down to the duramater as soon as pyrexia developed and thereafter the patient be treated symptomatically. He described no signs for detecting fractures by contrecoup; rather he said that nothing could be done for them because it was not possible to know their existence or the site at which they were placed. However, the compound fractures received his greater attention. He advised trephining till the dura was reached and then attempt was to be made to lever up the depressed piece of bone. He also described the signs and symptoms of necrosis of bone occurring consequent upon injuries, and he always warned the surgeon not to injure or go deeper than the dura.

Galen also practiced the art of trepanation and so did the Arab Surgeons, but perhaps nothing new was added and the writing of Hippocrates were the last word for a long period of time.

Lanfranchi (1296) was the first to record the manifestation of concussion, although no clear explanation of its occurrence was offered until Petit (1750) suggested that it was due to vibrations set up in the brain by the trauma. Littre in the meantime (1705) had described negative autopsy findings in a fatal case of head injury and that lent much support to the idea that concussion was due to a shake up of the brain and no lesion was necessary to produce concussion and even death if the trauma was severe enough. Morgagani described the contrecoup injuries (1766) and suggested it to be due to the force transmitted through the brain, causing a thrust of it against the resistant bony confines of the skull. Quesnay (1787) was the first to describe the occurrence of abscess in the brain following trauma. Mareschal reported cases of fungus cerebri in the 18th century and Pott gave an account of the Pott's puffy tumour and focal osteomyelitis of the skull. Bright (1831) was the first to note the occurrence of peticheal haemorrhages distributed in the various parts of the brain and connected their occurrence with the causation of concussion. Various other authors subsequently described their occurrence and for a time they assumed quite an importance as being the cause of many a symptom of concussion and the clinical state that follows it. Dupuytren (1839) introduced the concept of concussion, contusion and compression of the brain, the less severe cases being of concussion those of greater severity and prolonged unconsciousness being of contusion, the last stage being that of compression of the brain and that perhaps occurred in most cases who died.

This period which extends into the last third of the nineteenth century was a time when fractures of the skull were given most of the attention and the cerebral injury was supposed to be secondary and

which the surgeon was probably not much worried about and even if he was he perhaps had the idea that nothing much could be done for it.

The period of experimental work: From this period we next pass to the phase which was perhaps the most important, the birth period of most of the important ideas which we stuck to till recently and still they are one of the foremost in our theory and practice. This was about the last third of the nineteenth century when great advances took place in medical sciences as in others. Here an attempt was made to understand the head injuries on the basis of Physiology and on the postmortem findings done in greater number and with greater accuracy. Much experimental work was done in this period and detailed study of the material from these experiments and the postmortem room was carried out.

Sir Astley Cooper (1824) carried out an interesting experiment. He trephined the skull of a large dog and then after seperating the dura from the overlying skull pressed upon the dura with his finger. When the finger was pressed deeply the animal endeavoured to avoid it. When the pressure was increased the animal became comatose and collapsed on the table. On removing the finger the animal became conscious again, stood up, turned round with giddiness and walked away. The pulse was observed to have become slower when the pressure was increased. In 1878 Duret performed an experiment with a similar object. He injected wax into the cranial cavity of a dog. The animal's respiration ceased. This led him to conclude that the increase of pressure inside the cranium was the cause of this condition, so to release this pressure he incised the atlantooccipital ligament and then opened the cisterna magna to let out the cerebrospinal fluid. The fluid gushed out in a stream and the animal became immediately conscious. Two years later von Bergmann confirmed Duret's wax experiments and further demonstrated the effects of increased intracranial pressure in a child suffering from a sacral meningocele. On pressing the meningocele the child first became restless and then passed into a deep sleep. On further compression the respiration slowed and subsequently became irregular. The pulse itself fell down to 40 minute from 149 minute. Thus proving that the increase of intracranial pressure leads to unconsciousness, irregularity and cessation of respiration and slowing of the pulse rate. A great mass of experimental work accumulated to which Sir Victor Horsley and Walter Spencer added a great deal and came to the conclusion that the paralysis of the medullary centres and loss of consciousness was due to increase of intracranial pressure. Stromeyer and Kocher based their theory of concussion on this very basis. Duret's theory is also a product of this very period.

In 1886 Jacobson published his paper on the extradural haemorrhage from observations on 76 cases. Cushing (1902) performed similar experiments by increasing the intracranial pressure by injecting fluids inside the cranium. He further observed the effects of pressure on the cerebral cortex by watching it through glass windows fixed in the openings in the skull, and came to the conclusion that rise of blood pressure inside the skull caused anaemia of the brain, thus providing sufficient proof to the theory of hyperacute anaemia as a basis of concussion.

It will be noted that in this period great attention was given to explain the features of concussion by repeating condition on the animals. It was characteristic of this period that the brain physiology was supposed to be disturbed as a result of increased intracranial pressure and this could explain most of the conditions met with clinically. As a natural corollary the treatment of head injuries consisted of reducing the intracranial pressure. The most reliable known method then was by operation, which was being already done for cases of extradural haemorrhage. Thus head injuries were considered as a matter of surgical emergencies, probably to be rushed to the operation theatre and a decompression performed. Sixty years or so ago Horsley

advised that when apparent death resulted due to raised intracranial pressure, artificial respiration should be performed and the skull be opened freely.

The modern period: It was natural enough that the mass of experimental evidence should be analysed again and the knowledge so gained be crystallised. This we find having been done in the first two decades of the present century. First the theories were simplified, through mostly the writings of Trotter. He built up his theory upon what Kocher and Stromeyer had already said, and we came to believe that when the head is struck it bends in due to its elasticity, this causes a generalised and marked compression of the brain, resulting in expulsion of blood from it and thus an anaemia which causes paralysis and clinical condition of concussion. It became more and more evident that a typical lesion was a contusion of the brain, followed by edema of the brain and this was pointed out to be responsible for the subsequent prolongation of unconsciousness and other symptoms. The problem of treatment was therefore natural enough diminution or prevention of the edema and subsequent rise of intracranial tension, and to bring back the contused brain to normal size.

Simpler means other than the operative were devised to reduce the intracranial tension. Lumbar puncture was already being performed since after its discovery in 1891 by Quincke. In 1919, Weed and McKiban showed that intravenous injection of hypertonic saline solutions in Cat markedly lowered the spinal fluid pressure. A year later Foley and Putmen showed that oral or rectal administration of hypertonic solutions produced exactly similar experimental results. The Hypertonic saline and Magsulph solutions were subsequently used clinically by Charles H. Frazier (1921) and were studied and put on sound statistical basis by Temple Fay in nineteen twenties and thirties. This form of treatment is being used in many clinics as a routine.

We also owe to Trotter the recognition of persistent cerebral contusion whose principal symptom is headache and that much of the post head injury symptoms can be avoided if care is taken to resolve the contusion completely in the early stages by thorough dehydration treatment.

The last decade or so: The last decade or so saw a number of new features and to my mind some remarkable advances are trying to shake off some of the misconceptions that had been gathered from the mass of experimental work carried out in the end nineteenth century. In these years Symond and Jefferson revolted against the prevalent theory of concussion, while D. Denny Brown and W. R. Russell of England showed by their remarkable experiments on animals that it is the movement of the brain inside the skull and not the rise of pressure which causes concussion. Evidence of movement of the brain was provided by the physicist Houlbourn of Oxford who showed that the brain is subjected to shearing and rotational strains. Greenfield has emphasized that concussion can be explained in terms of bending and stretching of the neurones, thus explaining it on the basis of morbid anatomy. Jefferson gave a remarkable idea that concussion is a sort of traumatic sleep and is perhaps due to injury to the sleep centres, which may be situated in the region of hypothalamus and upper part of the brain stem. He has further pointed out that once the forces of trauma are finished, a disease process starts which disturbs the enzyme systems and may be responsible for the death or some of the subsequent conditions of the patient.

On the side of treatment the dehydration treatment is being favoured but with some reserve and doubt is being cast if it is necessary in all the cases in view of the fact that main injuries are due to the shearing and rotational strains and not due to rise of intracranial pressure. Another new feature is the increasing use of inspection burr holes, whenever there is doubt whether a blood clot is responsible for the clinical condition of the patient, the axiom being that no body should die because an unsuspected clot has been left behind.

Lastly the electroencephalographic studies likely to throw more light on the nature of concussion and on the detection of collections of blood.

Thus in the evolution of the knowledge of the head injuries the first thing to attract the attention were perhaps the superficial wounds, and next the fractures of the skull had their day for a long time. All through this period less importance was attached to the injuries of the brain, and perhaps in the beginning no attention worth the name was given to the subject although the traumatic stupor must have been noted and the occurrence of paralysis after the head injuries had been recorded quite early. However, the injury to the underlying brain came to be regarded as more important and since then it is being attempted to solve the nature of the phenomenon of concussion and what repercussion is it going to have on its treatment. We have seen that our treatment is based mostly on the idea that the rise of intracranial pressure and the brain edema are the worst enemies and no pains are to be spared in removing them. Thus the two outstanding main problems of head injuries are firstly the nature of concussion and secondly the brain edema and rise of intracranial tension. I propose to examine both these questions in the subsequent pages.

The Nature of Concussion

Concussion has been defined by Trotter as an essentially transient state due to head injury, of instantaneous on set and showing widespread paralytic symptoms without any structural cerebral injury, and always followed by amnesia for the actual moment of injury. When we come to analyse the various symptoms of concussion, stress is laid on three points, firstly the loss of consciousness and its variations, secondly the disorders of the function of the medullary centres and lastly the disorders of the vegetative functions attributed to be residing in the hypothalamus. The nature of concussion has been baffling since perhaps its existence was recognised, and the scientific workers interested in the problem have been on it since Petit (1750) first said that it was perhaps due to shake up of the brain. The problem has been very actively studied since the last third of the last century and various proposals have been put forward and although it has not been completely solved, we seem to have reached considerably nearer the goal. The various suggestions put forward have been either those which base their theory on some vascular disturbances occurring in the brain at the time of impact, or those that the neurones are directly injured by the trauma, and lastly those who consider it not necessary to think in terms of anatomical injury, but rather that the neurones undergo a physiological derangement.

The vascular theory: The most popular explanation of concussion till recently has been the one associated with the names of Stromeyer (1864), Kocher (1901), and Trotter. Stromeyer considered the concussion was due to flattening of the vault of the skull at the point struck, which in turn pressed upon the underlying brain that led to the passive expulsion of blood from cerebral capillaries resulting in cerebral anaemia. Kramer and Kocher thought that concussion was due to momentary compression of the brain, which compression exerted by the cerebrospinal fluid collapsed the cerebral vessel in the same manner as does the rise of intracranial pressure produced artificially during an animal experiment. Whether such a deformity of the skull would occur when the head is unsupported while it is being struck was not very clear from Kocher's account. However, Kocher supposed that in such cases there was a mass movement of the brain which would have the same effect. This idea had already been postulated by Gussenbaur in 1880. Kocher also gave reason for prolonged unconsciousness by stating that the lasting effect was due to contusion, peticheal haemorrhages, and loss of blood supply to these areas for a longer period. Trotter enlarged upon the same theme and tried to explain in an accu-

rate and impressive way. He explained that the skull is by no means a rigid structure it appears to ordinary observation. When an external force is applied it is capable of yielding to a remarkable degree without breaking and of recovering to its original form with great elasticity. For example when a man falls on the ground on his head, the skull is compressed between the ground and the weight of the body, applied through the spine to the occipital condyles. Under these circumstances the skull as a whole undergoes deformation. It is flattened in the area at which the violence is applied and it bulges at the corresponding equator. The total effect of this, he argued, is a sudden "irresistable diminution of cranial capacity". The physical condition normally obtaining may be represented schemetically with sufficient accuracy if the skull is regarded as an elastic sphere containing a soft sponge like material. The meshes of the sponge are filled with fluid which also occupies spaces as are left in between the outer surface of the sponge and the elastic sphere which contains the sponge. In this way he said the intracranial space is occupied by the brain, the cerebrospinal fluid and the blood. And this filling of the space is complete. It becomes reasonable to understand that any diminution in the capacity of the container must be accompanied by a corresponding diminution of the contents, which would mean that some of the contents are displaced out. Of the contents the fluids being most mobile will be most likely to be displaced. The veins are more easily compressible and the tension in them is much less in comparison to that in the arteries. But he argued that this difference in the resistence to compression between the arteries and the veins is very small when it is compared with the force which is compressing them. The force of trauma being so great that the arteries and vein behave alike and the blood inside them is expelled alike. He pointed out that in an ordinary case "the brain is squeezed like a sponge." He further said that a very small amount of pressure will serve to cause obliteration of the capillaries and anaemia of the brain substance. The compressing force is applied to the capillaries from the very beginning, so that a wide spread capillary anaemia is instantaneously produced and leads to the corresponding paralytic symptoms. This compression lasts only momentarily because immediately after the blow the skull resumes its shape, the compression of the capillaries is removed and they become filled with blood again, and the circulation will get stabilised. When the vessels are being filled up again it will probably be more rapid in the veins than in the capillaries, therefore some relative anaemia of the brain substance would exist after the cause has ceased to act and this will be prolonged by the feebleness of the circulation. He further argued that it will take still more time before the circulation came to normal and secondary disturbances of circulation of the brain during this period are most probably responsible for the faintness, giddiness, and nausea which may persist well into the period of reaction. The sudden loss of consciousness and the generalised muscular collapse were regarded as clearly paralytic signs and the seat of paralysis was the cerebrum. The respiratory and circulatory signs pointed to a disturbance of the bulb. In the stage of reaction evidence of a variable amount of increased amount of excitability of the brain is noted as is shown by headache, full bounding pulse, raised temperature. Such general irritative symptoms Trotter explained by slight degree of increased intracranial pressure. This increase of intracranial pressure he said could be correlated with scattered small lesions in the brain and the edema with which such lesion get surrounded with. A slight degree of edema of the brain is therefore a constant sequel of head injury within some hours of the production of these minute lesions and may well be the cause of the common irritative symptoms.

In support of the theory we need evidence to show that inbending of the skull occurs, that a rise of intracranial pressure occurs, and that rise of pressure induces

anaemia of the brain. The Experiments of Felizet (1873) are quoted as proof enough to show that inbending of the skull occurs. He blackened the inside of the skull with charcoal and then showed that when it was struck it left a mark on the surface it was made to strike. For demonstration of rise of pressure inside the skull there have been a larger number of experiments. Krammer (1896) demonstrated a rise of pressure within a dried skull by distortion of skull when a heavy blow was struck on it. In the living animal he demonstrated transitory abolition of respiration in an animal struck with a heavy blow on the head. Scott (1940) has recently demonstrated by optical methods, a rise of intracranial pressure to about 300 mm. of mercury when a supported head was given a heavy blow. He showed that the rise is only brief and such a brief rise may cause loss of consciousness only for five minutes. In Denny Brown and Russell's experiments the rise of intracranial pressure was present although small, in these experiments the head was allowed to move. As to whether rise of pressure causes loss of consciousness there is a very large mass of evidence. The experiments of Cooper, of Duret and Horsley have already been referred to in the preceding pages. That blanching of the cerebral cortex occurs by the increase of intracranial tension was observed directly in animals by Beck (1865) and by Cushing (1901), through a glass window in the skull the surface of the brain was watched as the pressure inside the skull was raised. The surface of the brain was noted to become pale and without movement. As the pressure was released the animal became conscious, the cerebral surface vessels filled again and the respiration returned.

These were the grounds to hold that a blow caused an inbending of the skull that in turn raised the intracranial pressure which caused a cessation of cerebral circulation and momentary anaemia.

The theory of organic neuronal injury: or the theory of direct mechanical effect on the nervous tissue. The essential principle of this theory is that unconsciousness and the allied states can be explained by demonstrable changes in the neurones themselves. The various theories or direct mechanical effect have been examined in some detail by Greenfield recently. They may be divided into three: (a) the compression theory of Saerbruch and Breslaeur, (b) the vibration theory of Merinesco and lastly (c) the theory of deformation and stretching. The compression theory takes its basis from the fact that compression of nervous tissue causes a temporary loss of function. It does come to be that the cortex under the site of impact of the injury is compressed and bruised, and it is also a well known fact that blows on nerve trunks cause a loss of function for the time being. But will it be that compression of a localised area of cortex under the area of impact will cause a sudden loss of consciousness or a more remote effect such as the disturbance of the vegetative nervous system which are so common in head injuries. To explain these points as well Breslaeur postulated that the impact pushed the brain towards the foramen magnum and so effected the medulla indirectly.

The vibration theory seems to have no basis since there is no proof that the force is transmitted through waves of high and low pressure.

The theory of deformation and stretching is most ardently supported by Greenfield. This theory supposes that a blow on the head produces alterations in the shape of the brain and stretching of the nerve fibres and the blood vessels. These changes occur at the point of impact and also at various other places in the brain particularly at those sites where the movement of the brain is resisted by the dural septa such as the falx and the tentorium and at the bony ridges at the base of the skull. There is no doubt that there is widespread and apparently random effects of injury on the brain, the blood vessels are also torn and the vegetative system may be effected in the same manner. It also accounts for the edema and dilatation of blood vessels, local

or widespread. These effects may be considered to result from lesions of the walls of the arterioles and capillaries of such minor degrees as not to rupture them. Thus stretching of arteriole may cause damage to its endothelial lining or damage to its contractile mechanism. This will result in their remaining dilated whatever the pressure inside them. This seems to be more satisfactory explanation for accounting for the dilatation of the blood vessels found after head injuries. The dilatation of arterioles and capillaries associated with the damage to their endothelial lining would also explain the occurrence of edema. The other cause of edema could be that deformation and stretching of the nervous tissue result in the formation of metabolites which makes the walls of the capillaries more permeable. Greenfield points out that the evidence in support of it is the observation of Knauer and Enderlen that the brain tissue near the area of impact becomes acid to litmus.

Many authors have described alterations in the nerve cells in the damaged areas of the brain. According to most they may be swollen or shrunken and tend to stain darkly. The work of Rand and Courville is perhaps the most important. They found changes in all elements of the nervous tissue in the most damaged areas. The axis cylinders in the neighbourhood of injury were ruptured or injured in such a way that they underwent what Cajal called 'preservation necrosis'. Rand and Courvilles observations on the nerve fibers are new and of special importance as indicating that they are directly effected by the mechanical injury.

Besides the obvious haemorrhages, all authors describe the frequent presence of ring haemorrhages similar to those seen in coal gas poisoning. The explanation generally given for their occurrence is that they are caused by blockage of small vessels with haemorrhagic infarctions around a small necrotic area. The diapedesis of red cells is therefore an indirect result of ischaemic changes in the tissues.

This theory as Greenfield himself says is as difficult to prove as any other and there is no doubt that changes can always be demonstrated in any serious case of head injury in areas other those directly contused.

Duret's theory, which is also called the humoral theory may be considered here since the theory also supposes the direct injury of the neurones as responsible for the concussion. This theory supposes that a blow on the head causes a wave of cerebrospinal fluid being driven from the lateral ventricles towards the foramen magnum at such a pressure so as to deform the walls of the third and the fourth ventricles and the inter. Thus it comes to say that concussion could be caused by the impact of the cerebrospinal fluid on the thalamus, hypothalamus and the brain stem. It is held by many that the sleep centres are situated in the walls of the third ventricle or in the thalamus and it is the present day tendency to regard that lesions of the brain stem can cause loss of consciousness more easily than the lesions of the cortex of the cerebrum. It is also common belief that the changes in the respiration, pulse and the blood pressure is due to disturbance of the medullary centres, therefore Duret's idea that the impact of cerebrospinal fluid on the fourth ventricle causes these changes is tenable. As he demonstrated in his own experiment that when he let out the cerebrospinal fluid from the cisterna magna the animal became conscious and the respiration returned, in this case the pressure from the medullary centres is relieved. Thus we could assume that if a wave of cerebrospinal fluid could be demonstrated, this theory would form a reasonable basis for concussion. But its strongest modern supporter Berner has been unable to produce evidence that such a wave occurs. Greenfield points out that if it did we must expect it to tear the walls of the Iter and produce haemorrhages around them. He further says that no one will dispute the frequency of small pontine haemorrhages and dilatation of small vessels in the pons in cases of head injuries.

But it is very difficult to understand that those in the ventral half of the pons can be caused according to Duret's hypothesis. There is the possibility also that some of these do not occur at the time of the accident, but are related to the later alterations of the intracranial pressure. On the other hand in the lateral ventricles small tears and haemorrhages are quite frequent.

The theory of physiological neuronal injury is perhaps the oldest although it has been expressed in various different ways. Essentially it means the neurones undergo a purely nonstructural injury. Since a very long time it was believed that concussion could be caused by a shake up of the head and without obvious signs of violence to the head. Littre first described a fatal case of concussion in which he could not find evidence of violence to the brain. Hunter (1786) vaguely expressed that concussion was due to shake up of the brain. Brichsen (1866) came to the conclusion that spinal concussion was due to molecular disarrangement of the cells concerned. This probably was responsible for the origin of the term commotio cerebri of commotion of the brain cells. Filene (1874) demonstrated that the animals could concussed and killed by repeating small blows to the head. They claimed that the blows paralysed the centers of respiration and heart one by one and that concussion effected the various centres without there being any structural change to the brain cells. There is an extensive literature on the subject. Lately the report of Vance on postmortem room findings in 512 cases of head injuries points to the fact that there were sixteen cases in which no bruising or laceration of the brain could be found. The experimental studies of Denny Brown and Russell have put this so far neglected theory on a sound basis. These will now be considered in some detail.

The approach to the problem by these authors has been different from their predecessors. Firstly they introduced the idea that the head should be allowed to move after injury in experimental animals. Upto now most of the experiments on the animals were performed either by increasing the pressure inside the cranium by introducing wax or other fluids, or they were being performed by fixing the head of the animal and giving it a blow. This is different from what happened in the human head injuries because in the human injuries the head was never fixed, it either moved after it was struck or a moving head was brought to a stand by impact on the ground and moved even after that. This very idea was put by these authors into their experiments on animals. Another difficulty that confronts experimental studies is defining the states of unconsciousness, coma and stupor particularly because the animals are under anaesthesia. To overcome this difficulty they investigated the disturbances of general and reflex nervous reactions which are accessible in the lightly anaesthetised and the decerebrate animals. They thus demonstrated that accompanying the stilling or the stunning of the animals, there is a period of paralysis of the mechanism of the brain stem. The decerebrate animal also shows a similar paralysis. In lesser degrees the paralysis is for reflexes only, in severe degrees it is absolute. They have made their conclusions about the loss of consciousness as well and propose that conscious and intellectual function in man suffers from the same transient paralysis which has been demonstrated experimentally in the lower centres. They believe that these changes are applicable to the neurone in general and not to this centre or the other. They are in their opinion fundamental reactions to physical stress. They found no evidence of vascular spasm or stasis nor any evidence of fat embolism. And they conclude that "concussion is a generalised reversible 'Molecular reaction' induced by physical stress. This stress must be general and must reach a certain critical speed of application. In correspondence with the laws of Physiological stimulus it is the rate of change and not the absolute value which matters. The stress is clearly derived from the inertia of the brain when the head is accelerated, and from the momentum when it is decelerated".

In these experiments cats, monkeys and dogs were used. The head was struck on the parietooccipital region by means of pendulum, the animals head was allowed to move two centimeters after the blow, there after the head was brought to rest by a soft wool padding. Arrangements were made to regulate the speed at which the pendulum struck the head. They demonstrated a remarkable feature, that when the head was fixed and a blow of 28 ft. second was applied concussion could not be produced whereas even when a slight movement of the head was allowed concussion occurs with the same blow. It is obvious that it is the movement of the head that has made the difference. This they call acceleration concussion that is the one produced by the movement of the head. They found that acceleration concussion was a disturbance of nervous function which requires a certain intensity of injury, an adequate stimulus, as do other reactions. The threshold is limited to the movement of the head, and is expressed in sufficient rate of change, this change may be positive that is acceleration or negative that is of deceleration. Why should there be necessity of movement of the head or in other words why should movement of the head make the difference ? It necessarily requires that the brain also must move. It means that there must a flinging movement of the brain inside the skull due to inertia. The velocity of injury necessary to produce acceleration concussion they found in the case of monkeys and cats, was that struck by a heavy mass with a velocity of approximately 28 feet per second. They also found that the rate of changes of the velocity was the critical factor and this was the same for the brains of greatly different sizes.

Concussion thus produced effects the brain stem but there is no reason to believe that the effects are limited to this region. More severe injuries will produce wide spread effects. The sudden acceleration imparted to the brain in severe head injuries would be causing widespread distortions and strains throughout the various parts of the brain. These strains would be responsible for the occurrence of haemorrhagic lesions wherever they occur.

For the prolonged effect they propose that the same mechanism that brought about a transient paralysis can be extended to account for prolonged effects on the delicate mechanism such as is concerned in the full recovery of consciousness in man.

Discussion on the Nature of Concussion

The various theories have been considered in some detail in the preceding pages. Since the beginning of the present century the vascular theory as put forward by Trotter has been having its sway, and seemed to be the only convincing proposition. But in the last decade or so opinion appears to have changed completely against it and in all probability most of the surgeons are gradually ceasing to believe in it. But before we discard it we may consider the points for and against it.

The *case against vascular theory* has been strong, there are several onjections to it. In the first place a sudden blow on the Occiput or forehead might be supposed to increase rather than diminish the cranial capacity by making its shape approximate more closely to that of a sphere. As has already been written in the preceding pages Trotter himself said that the skull is flattened at the pole struck while it bulges at the corresponding equater. From this it cannot be assumed that there is such a diminution of cranial capacity that it squeezes the brain like a sponge. In the knock out blows of boxing as well as in the lateral blows on the head in which there is no diminution of cranial capacity the unconsciousness is as sudden. There is also no evidence that a very temporary emptying of capillaries as is envisaged in the vascular theory, will cause a loss of function of the nerve cells. Greenfield points out that injections of thorotrast into the internal carotid artery deprive a large area of capillary bed of blood for almost a second but do not usually cause a loss of function of the area involved. There is no doubt that continued anoxia may cause a sudden loss of consciousness but it seems unlikely that the nerve cells would suffer from anoxia from so momentary a stoppage of circulation as is postulated in Kocher's theory. In observations of Lennox and Gibbs consciousness was lost suddenly six to eight seconds after the heart had stopped beating (Greenfield). Witkowski (1877) reported a simple but important experiment which appears to dispose off the importance of vascular changes in concussion. He found that in a frog when the heart is removed the animal continues to show spontaneous movements and to right itself. Such a frog could still be stunned by a blow on the head and would then lie motionless for many seconds before the movements returned. Pollis (1894) published extensive investigations into the vascular changes in concussion. He held that the primary cause of death in concussion was a loss of equilibrium in the bulbar centres. Miller (1927) repeated the experiments or Witkowski and Pollis and he confirmed their findings. He showed that in the case of rabbits cerebral anaemia upto seven seconds does not cause loss of consciousness, as judged by the reactions of the animal to stimuli.

It would seem from the foregoing arguments that the vascular theory does not stand to reasoning. If it is so then what about such a lot of experimental evidence on which it was based. One thing suggests itself to me that is that we need discard only the second part of the theory and retain the first part. It appears the cerebral anaemia possibly does not occur and this we can cease to believe, we may also stop agreeing to a squeezing of the brain like a sponge. But we cannot say that concussion cannot be caused by a sudden increase of intracranial pressure. The experiments of Cooper, Duret, or Horsley, of Cushing and many others cannot be set aside. Only recently (1941) even in the experiments of Danny Brown and W. R. Russell who are quite critical of the vascular theory we find that a sudden rise of pressure causes a condition similar to the acceleration concussion. They point out that if a momentary large increase of intracranial pressure be made with great rapidity, phenomenon similar to experimental concussion occur: the corneal reflex is lost, respiration misses one or two beats, with inspiratory spasm and there is a small shallow rise of blood pressure. To get this result it was necessary for them to give a hard and rapid blow to the plunger of a syringe containing air and connected by a short piece of pressure tubing to an opening in the skull. They further remark that in this way the cerebrospinal fluid can be made to transmit a mechanical shock and induce an effect closely resembling that observed in acceleration concussion. They propose to apply the term compression concussion to this variety of abolition of reflexes. There are a few differences in the two types of concussion as pointed out by them. They are that respiration is more sensitive to compression, this agrees with most of the experimental data of those who previously produced unconsciousness in animals by injecting wax or other fluids. Further the corneal reflex recovers later than the pinna reflex, and the rise of blood pressure is much smaller and less steep. They obtained similar results on striking the bare dura, and further they state that the piston or plunger effect of a depressed fracture of the skull can cause compression concussion similar to that obtained in the experiments with forcing air by a syringe connected to the intracranial cavity.

It is thus evident that although we may discard the vascular theory, we cannot do away with the fact that a sudden rise of pressure inside the skull can give rise to a condition of concussion. We have also to admit that a blow on the bare dura is going to have a similar effect and in the same way a depressed fracture or as a matter of that an inbending of the skull sufficient to cause a sudden impact on the dura is likely to cause concussion. How does a sudden rise of intracranial pressure cause concussion if we are not to believe that it did so by causing anaemia of the brain ? For an answer we may turn to the physiological neuronal injury. It may be caused by a high rate of change as envi-

saged by Denny Brown and Russell to explain the compression concussion.

The theory of mechanical deformation supposes that stretching is severe enough to cause an anatomical injury to the neurone. Since we definitely know that concussion can be demonstrated to occur without the slightest evidence of lesion in the brain, we may safely assume that in less severe injuries there is no demonstrable lesion while in severe injuries the lesions are obvious. It is not that a more severe injury leading to visible stretching bending and tearing cannot cause concussion, but the point is that a much less severe trauma can cause concussion. Miller showed that a rabbit could be concussed and recover without damage to the nervous tissue as judged by the intra vitam staining by trepan blue in the subsequent five days.. The dye should stain any dead or necrosed tissue.

It appears quite reasonable to assume that the theory of physiological neuronal injury is quite satisfactory. It explains our clinical cases who get concussion from slight trauma such as a fall from a few feet or a patient while walking slips, falls down striking the head on the ground. In these cases it becomes difficult to assume that there was a great rise of intracranial pressure due to inbending and an anaemia of the brain; nor does it appear that in these cases the trauma is severe enough to cause a deformation and stretching. That the brain moves inside the skull and under goes shearing and rotational strains is amply proved by work of Holbourn, who has demonstrated that the behaviour of the skull and the brain during and immediately after an injuring blow is determined by the physical properties of the skull and brain and Newton's Law of Motion. The injurious effects could be either due to changes of velocity in a straight line or changes of velocity as the brain rotates around one of an infinite number of axes. Holbourn thinks that damage to the brain caused in the large majority of cases by rotation which causes severe shearing strain. The work of Holbourn has added coroborative evidence in favour of the Denny Brown and Russell's theory.

Jefferson has also commented favourably on the theory and states that the evidence of brain stem paralysis had been proved to be well founded with entirely a novel discovery that concussion could be more easily brought about if the head was allowed to move. He further points out that in these experiments a vital point was that concussion could be produced without deformation of the skull, and the essential factor was the suddenness of the acceleration imparted to the head. The active cause remains a sudden application of energy to the skull. He concludes that the fundamental of concussion except the unconsciousness can be left safely as resulting on a firm basis of experimental proof linked with clinical observation and we may say that the primary cause is a paralytic effect on the neurone of which the brain mass is composed.

He asks the question as to what part is played by the cerebral hemispheres, the cortex and the deeper nuclei in the concussional or contusional syndrome. He answers it by saying that the majority of writers have assumed that consciousness is mediated by the cortex. He himself does not deny that of full consciousness in the physiological sense this is certainly so. But believes that the unconsciousness in the head injuries is a sort of sleep due to injury of the sleep centre which may lie in the Hypothalamus, or the pons and the various parts of the brain stem. He thus names the unconsciousness of the concussion as the traumatic stupor or Parasomnia indicating that there is no response showing insight into or analysis of the problem set by the stimulus, at least no more than there is in a sleeping person. It may be that the injuries to hypothalamic areas or the brain stem may be responsible for the state of unconsciousness, but it will be difficult to prove it to be so in the present state of our knowledge. There is no doubt that the region of the hypothalamus and the floor of the third ventricle are liable to greater shearing strain as envisaged in the

work of Holbourn. He states that when the head moves from side to side on an anteroposterior axis, the pituitary fossa becomes an important projecting buttress and considerable forces are transmitted through it to the brain, this may account for the injuries inflicted on the region of the hypothalamus and the floor of the third ventricle. Jefferson points out in favour of his suggestion that the cortical lesions pure and simple do not cause it, nor do the massive excisions of the lobes, yet a small central or basal injury does so. In the loss of consciousness after lucid interval the mechanism may be similar, i.e. pressure on the brain stem by herniated uncus through the tentorial opening. The electroencephalographic pattern in sleep he points out is often very similar to that seen after concussion.

Greenfield also comments on the cause of unconsciousness in a somewhat similar tone. He says that the cause of loss of unconsciousness remains conjunctural, but in cases of lesion of brain stem unconsciousness seems more likely to occur, the nearer the lesion approaches the thalamus, so that both in clinical and experimental experience the state of consciousness seems to be centred in the thalamus and the region round about. It does not necessarily mean that there is a centre in the thalamus, although that may be true. He further says that the unconsciousness in head injuries may well be caused by a sudden deformation and stretching of important fibre tracts passing to or from the thalamus or the hypothalamus.

From the foregoing it appears reasonable to assume that the region of Hypothalamus down to the pons is quite likely to strains and this is the area also known to be concerned with sleep and it leaves one only to stretch the imagination a little to make conclusions.

The Cerebral Edema and rise of Intracranial Pressure

This subject is of prime importance in dealing with head injuries. As has already been mentioned our present treatment is based on the fact that after the initial injury a rise of intracranial tension is usual, and that edema of the brain is also present in most of the severe cases. The treatment is mostly directed towards the reduction of the brain edema and the reduction of the intracranial tension. The subject therefore is of utmost importance. It is a common belief that whenever there is prolongation of unconsciousness and the accompanying state of the patient some secondary complication such as contusion haemorrhage or cerebral edema has developed. It is the contention of certain authors that the prolongation of the state of concussion is the result of the same forces which caused the concussion, that is to say that if the trauma has been rather severe the damage to the neurones is more severe and thus its effects are more prolonged. That may be true, but a stage is reached when we will have to assume that either the initial trauma has caused a damage of a degree which is irreversible or that some secondary complications have taken place.

The increase of intracranial pressure could occur due to (i) edema of the brain as a response to trauma, (ii) to increase in the amount of the cerebrospinal fluid either by increased secretion or decrease in absorbtion, (iii) or due to congestion and haemorrhage of the cerebral blood vessels. No one would doubt about the last factor when it occurs and we will not discuss it. It has been believed for long time that a generalised edema of the brain invariably occurs after most of the head injury, doubt has however been cast recently that localised edema is more common around areas of contusion. Even this may not occur in less severe injuries.

Does a generalised cerebral edema occur? Several conflicting opinions have been expressed by various authorities on the subject. There are those who say that cerebral edema is a frequent occurrence. Apfelbach (1922) was of the opinion that a generalised edema of the brain occurs and he is probably responsible for this belief in America. He determined the ratio between the weight of the wet and the

dry brain of the patients dying of acute head injuries and he found an increase of water in some cases upto 3.7%. In many other cases he found no increase in the water content after trauma. He drew conclusions that the edema frequently occurred particularly when the cranial bones were fractured, and that it occurred a few hours after and within three days of the injury. Greenfield has objected to the validity of these findings on the basis that Aphfelbach used an undetermined mixture of grey and white matter and since these two components of the brain have considerable difference in their water contents, so the results obtained by him are not reliable. He argues that edema is rarely more than 10% and normally the difference in the water content of the white and the grey matter is 13%.

Trotter (1925) was of the opinion that the occurrence of the edema was probably the commonest complication after trauma to the brain. Although it is that the brain is not more liable to edema than other organs, but its situation in the rigid skull leaves no space for it to swell and hence the effects of a small edema are likely to be felt in the case of the brain. As soon as a part of the brain becomes edematous its circulation is threatened and according to the severity of the condition more or less impaired. This impeds the circulation in the veins which results in congestion. He also came to the conclusion that the symptoms of the edema are irritative and not paralytic. Edema he said could occur in consequence to any injury to the walls of the cerebral vessels. Of such injuries the most important being, contusion, closure from pressure and inflammatory process. The form of edema which occurs in cases of contusion takes about 48 hours to develope fully. This edema may extend to a greater or lesser extent into the surrounding brain. This swelling causes pressure on the surrounding veins, which in turn causes congestion and so further exudation. He further pointed out that if multiple foci of contusion are present as they are in a fairly severe case then the edema would be a widespread and a general one. In his opinion the symptoms of general edema are much more definite and they are the ones which have been known as those of cerebral irritation.

The most important work supporting the view for the occurrence of cerebral edema is that of Rand and Courville (1931, 1932). They state that the increase of intracranial pressure is the commonest occurrence after trauma. This is due to excess production of the cerebrospinal fluid. This is manifested they say by the recovery of increased amount of fluid on lumbar puncture and by the presence of excess of fluid in the subarachnoid space. This condition they referred to as wet brain. During life the wet brain is described as pale, with engorged cortical veins and the fluid seems to come directly from the vessels by transudation. The cerebral cortex was found to be doughy to palpation and soggy in consistence. They described droplets being seen on the surface of the arachnoid and call it weeping through perhaps because the fluid under the arachnoid is under pressure. In a fixed specimen they describe dilatation of the subarachnoid space, widening of the fissures and sulci and increased rounded convolutional outlines. The ventricular spaces were also found appreciably larger. Histologically they found the perivascular and pericellular spaces as definitely enlarged, the oligodendroglias swollen with vascuole formation inside their cytoplasm. The stroma specially in the white matter was found to be separated by droplets of fluid to form a visible meshwork. The epithelial cells of choroid plexus and the ependyma were swollen and showing increased number of vacuoles. All these features they pointed out suggest an excessive secretory activity of these structures.

This is they say the usual frequent picture in cases of severe injury of the brain produced with head in motion, they further state that this is not an invariable reaction to trauma. In a relatively uncommon number of cases a condition of dry brain is met with which means that there is an increased amount of fluid bound in the cytoplasm of the cells, the oligodendroglia and other elements. The generalised edema may thus occur in either form and both types may be found in the same specimen. They conclude that the increased production of fluid begins almost at once following the injury, and is accelerated rapidly in about four hours. In less severely injured cases they conclude the rise is more gradual and the peak is probably reached in eight to twelve hours and levels off in two to three days.

They also state that local edema occurs around the contusions and if they are situated near the vital centres may cause death by interference with vital centres in the medulla.

Pilcher (1937) performed experiments on dogs by making a weight fall on their heads. There was a sharp initial rise of blood pressure followed by a return to the normal within thirty minutes. Subsequently a more sustained rise began in some of the dogs in an hour or two after the injury. He then examined the water content of the brain at varying intervals after the injury. He found that there was a slight increase of water content in the grey matter and an increase of not more than two percent water in the white matter. There was one more interesting point in his experiments and that was that he gave intravenous dextrose to some of the dogs after in jury and subsequently measured the increase of water content. It was found that the brains of dogs treated by this dehydration showed no difference from those who were not put on this treatment. He concluded that there was a little increase of fluid in the grey matter. Vance (1927) reviewed 512 cases of death from head injury. He found that during the first few hours three was an increase of intracranial pressure with flattened convolutions. At this stage the blood vessels were engorged. Later 12 to 24 hours after the injury there was distinct swelling of the brain and the brain tissue was moist.

The latest experiments in the series proving the occurrence of edema are those of J. D. White (1943). White performed experiments similar to those of Denny Brown and Russell on cats and measured their brain volumes after examination. The measurement was done from the shrinkage of free space between the brain and the cranial cavity. Reduction of this space indicates the expansion of the brain. When cerebral concussion was produced in cats for even brief periods they found an increase in the brain volume upto a maximum of 5%. They found the degree of swelling to vary directly with the severity of concussion. It was found to make its appearance in fifteen minutes, and reach its maximum between five to twenty four hours.

On the other hand there are those who deny the existence of a generalised edema of the brain. Connor and Wright (1934) reviewed 1760 clinical cases of cranial and intracranial injuries with many autopsies. They could not find a single instance of general traumatic edema coming on 48 hours after injury. They do not think it occurs even after that since they have never seen it. Shapiro and Jackson (1939) reported some investigations on cerebral edema. They investigated the relationship of dry to the wet by weight in grey and white matter, separately correcting for the amount of blood present. They found that the injured brain contained less water than normal brains. Thus they found no evidence of true cerebral edema after trauma.

Greenfield (1942) has also found no evidence of a generalised edema of the brain. In the first place he points out that the observation of Apfelbrach are not reliable because he used an undetermined mixture of white and grey matter, and as for the observations of Pilcher, they have not been corrected for the increase of volume that might have occurred due to the presence of blood in the tissue. The increase reported by Vance is very slight. Greenfield reported on the histological examination of the 31 traumatised brains which he had examined. He found no evidence of generalised post-traumatic edema. But he did find some evidence of edema where the brain was bruised specially after a fracture of the skull or where it is penetrated by a missile. Around these there is often a zone in which the myelin stains poorly, and the astro-

cytes are swollen. This zone may be a few centimeters in width but rarely more than two centimeters. These investigations led him to conclude that the edema is limited to the area around the bruise and it was only great when there was overriding of the edges of the fractured skull. Even in such cases it usually effects only one pole. It is only slight and was only in the white matter underlying the bruised area of the cortex.

From these conflicting opinions one thing is definite and that is that all are agreed that local edema around the bruised area does occur. As a natural sequence if we were to consider a case in which there were widespread and numerous small bruises and peticheal haemorrhages we could conclude that there would be a general edema. It might be slight, but it will be there. However the results obtained by Vance and recently by White also contribute to the idea that a slight general increase in the brain volume does occur more frequently than not and it would be a mistake to conclude that a generalised edema did occur in all the cases of head injury, because even the strong supporters of the idea do not emphasize that edema occurs in every case. It may be that those who measure the increase in brain volume as a whole are measuring actually the increase that might have occurred locally only. Most authors agree that there is vascular congestion in the brain after head injury. It is reasonable to expect that there will be greater secretion or exudation of fluid and hence a wet brain.

From clinical stand point there seems to be no criterion for finding out whether a condition of brain edema exists or not. Out of the various clinical features suggested that may be due to the edema are that it causes symptoms of cerebral irritation, that it cause drowsiness or prolongation of unconsciousness, or bradycardia or an increase of pressure on the lumbar Manometery. None of these seem to be unfailing.

Is cerebral edema the cause of stupor : It appears from the work of White (1943) that the swelling of the brain is not related chronologically to the neurological changes accompany concussion. In his experiments the edema failed to appear until the cardiac, respiratory pupillary and corneal reflexes had returned to normal and the edema reached its maximum when the animal had completely recovered and had no sign of cerebral lesion. From the clinical stand point if the pressure measured at the lumbar puncture be considered in average cases as indicating intracranial tension, then we find that there seems to be no relationship between the depth of unconsciousness and the presence of the cerebrospinal fluid. The figures given by Russell (1932) show that a patient could be fully conscious while manometric reading was over 300 mm. of water, while a patient could be stuporose or comatose while his reading of the pressure was below 100 mm. or slightly above it. Similarly the figures provided by Turner (1941) indicate that a man could be fully conscious with a pressure over 180 mm. while he could be comatose at a pressure reading of below 90 mm. only. This shows that the cerebrospinal fluid pressure in the lumbar sac bears little relationship to the state of consciousness.

Is the edema cause of cerebral irritation : It has been stated that the cerebral edema causes venous congestion and not anaemia and perhaps due to this fact Trotter emphasized that the symptoms of edema are those of cerebral irritation. Many writers have agreed to it and in fact it is a general belief because the institution of dehydration treatment clears up the mind of the patient and the restlessness and irritation disappear in many cases in a short period. This is the general experience of most of the Surgeons. Symond and Jefferson pointed out very significant facts in this connection. They point out that traumatic stupor was a different state than the effects of increased intracranial pressure as in tumour, hydrocephalus etc. In the increased intracranial tension due to tumour etc. the mental state was that of drowsiness and apathy while in the traumatic stupor there was motor restlessness, confusion and delerium. The cases of

brain tumour respond to dehydration, while those of the traumatic stupor may not. The difference is due to the fact that in traumatic cases the damage to the neurones has occurred first while edema has followed later. Jefferson states his theme that the stupor in the average case is contusional and that even when edema occurs it is rarely alone the cause of protracted unconsciousness. He further points out that the restlessness, the irritability, the anger, and indeed the sham rage of some of the patients, recall the animal experiment of Bard, Fulton and Ingrahm, who produced it by lesions of the undersurface of the frontal lobes, which are more likely to be contused due to anatomical configuration of that part of the skull (Fig. 1).

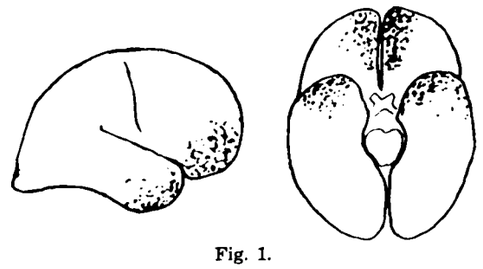

Fig. 1.

Showing areas where contusion is most commonly found. The under surface of the frontal lobes are usually bruised and may be the cause of cerebral irritation.

Is edema harmful ? and does it need to be relieved by dehydration, and does the dehydration treatment really reduce the edema ? These are some of the questions which are the subject of discussion and need in my opinion a clarification since they effect our line of treatment so much. It has been said above that there is a common agreement that local edema does occur around the contusion but there is difference of opinion as to whether a general edema occurs or not. After considering the various points of view. I concluded that a slight general edema occurs in most cases while in some it might be more marked and in others it might be absent. It is reasonable to appreciate that similar opinions will be held about the treatment which reduces the edema. It will be agreed that the dehydration treatment to reduce the edema will not be reasonable to be employed in every case since there are cases in which the intracranial tension instead of being high it is actually low, and in others it is not low but not raised either.

There are some who believe that even if the cerebral edema is present or the intracranial tension somewhat raised the dehydration treatment should not be given. They present two arguments in their favour : One thing the edema is not harmful and the other that the dehydration may be injurious. W. T. R. Russell (1934) has stated that cerebral edema is well known to be frequently present after severe cases of head injury. This edema produces rise of intracranial pressure, this is seen at the operation when we find that the brain bulges outwards if exposed at operation. Lumbar puncture pressure readings may also be high although the readings are not proportional to the depth of unconsciousness present. The pulse may be slow and it is reasonable to conclude that a slow pulse means a raised intracranial pressure. In his opinion cerebral edema is in itself rarely if ever a cause of dangerous increase of intracranial pressure. Because the mode of death of cases of head injuries does not in any way indicate that increase of pressure is an important factor. An increasing rapid respiration and pulse rate is the rule and this in itself gives sufficient indication that increase of pressure is not an important factor in causing the death of the patient. He examined a series of 200 cases 18 months after discharge from the hospital and found that only 3.5% had developed elilepsy although in none of these cases intense dehydration was carried out. Amongst these cases there were 24 such cases whose injury had been severe as to cause loss of consciousness for a period of over three days, of these 18 returned to full work within six months. Cerebral edema must have been present in many of them in the acute stage, yet the highly satisfactory recovery rate suggests that there was no damaging effect on the prognosis. That

suggests that the cerebral edema does not cause any harmful effect in itself. On the other hand dehydration treatment may be harmful because of the fact that by reducing the intracranial tension venous bleeding might start and oozing might occur where it might not have otherwise been. He further states that the bradycardia seen after head injuries for two or three weeks is due to edema of the brain or the brain stem, but the spinal fluid pressure may be quite normal, and he emphasizes that this postconcussional bradycardia is not necessarily associated with impairment of consciousness.

The same author in 1942 again commented on the cerebral edema and its effects. He points out that an abscess of frontal lobe causes hemiplegia which clears up after its aspiration and comparable improvement is observed when a decompression is done. Similarly the effects of obstruction of a cerebral vein are a transient with widespread loss of function. In head injuries a contusion or an haemorrhage is surrounded by edema which may contribute to transient hemiplegia. All these examples he says have one thing in common, that is that there is a local disturbance of cerebral function which is transient. It is not possible however to conclude that the edema in itself is the cause of loss of function because other associated circulatory abnormalities such as obstruction of a vein may be more important. It is also an interesting fact he points out that intravenous hypertonic solutions do not readily effect the edema around the tumours or abscesses and it is possible that the dehydrating measures effect the normal brain to a greater extent than the abnormal edematous brain. This hypothesis gives an explanation for disappointing results and the delayed relapses. He concludes that it is not possible in the present state of our knowledge to say that the loss of cerebral function is due to edema or due to the circulatory disorder which is the primary condition and which is responsible for the edema itself.

Paterson carried out a short study of the effects of intravenous injection of 100 cc. of 50% sucrose, in cases of brain tumours and other cases of raised intracranial pressure and came to the conclusion that the lumbar puncture cerebrospinal fluid pressure is reduced only to a small extent. The maximum reduction in his cases being 50 mm. of spinal fluid. This was maintained for less than one hour, at the end of this period the pressure rose again to 40 mm. above the pre-injection level. They also observed no clinical change in the respiration, pulse rate or the blood pressure. In some cases striking changes are noted in general condition after the dehydration, he also observed the same occasionally but he is inclined to the view that these changes are not due to reduction of the pressure as measured by the lumbar puncture.

Jefferson is also sceptical of the condition of the patient being due to compression of the brain. He says it is a fact that a number of our patients are not suffering from compression at all. This conclusion he supports from three observations. First that the pressure of the cerebrospinal fluid as measured by the lumbar puncture is often low or not notably increased. Secondly that intravenous hypertonic solution administration is not uniformly good or even satisfactory. Thirdly that during operations under local anaesthesia commonly reveles a low pressure brain when a high pressure bulging brain was expected. The difference between the results of hypertonic injections in cases of trauma and that of brain tumour is also there. In brain tumour cases the patients drowsiness becomes less while in the head injury it usually shows no change. He points out that the difference is not due to the fact that there is no edema but because the patient's state is not entirely due to it. In head injuries wide spread damage has occurred first and edema has only followed later.

Thus the treatment by dehydration has not been advised by some because the edema and increased pressure are not a

marked feature, and even if the edema is present it does not seem to have harmful effects or at any rate it is not responsible for the postconcussional state. In some cases the edema and the raised intracranial tension are not present, rather a reverse condition that is a lowered intracranial pressure exists. It has also been given as a reason that a slight rise of intracranial tension might be useful because it prevents venous bleeding and if the tension were to be reduced this might cause venous haemorrhage. It has also been shown by Russell that his serious cases were no worse when they were not treated by intensive dehydration, in fact the dehydration treatment did not seem to effect the ultimate prognosis in his opinion.

On the other hand there have been others, notably Temple Fay and Lambert Rogers who have strongly advocated intensive dehydration treatment. Temple Fay bases his argument mainly on the fact that there is swelling of the brain and since the skull is rigid, space has to be provided for the swollen brain by reduction of the amount of cerebrospinal fluid, and the amount of the tissue fluid in the brain. He advises intensive dehydration treatment because in his opinion besides cerebral edema, a state of medullary edema also exists which compresses upon the medullary centres and is responsible for the slowing of pulse. He points out that the effectiveness of the use of hypertonic solution by the bowel or intravenously depends first on the adequate substraction of fluid from blood volume, this in turn leads to withdrawal of fluids from the body tissues. These methods will fail if the patient is given liberal amounts of fluid to drink and restriction of fluid intake is not done. It is a prime importance that production of cerebrospinal fluid should be curtailed by lessened intake of water. His experience of several years has shown that adult patients placed on total daily intake of twenty ounces of liquid and a dry diet containing not more than twenty ounces of water weight will not produce sufficient spinal fluid to increase intracranial pressure. On this routine he showed that only a few c.c. of spinal fluid can be obtained on lumbar puncture. He further showed that if the patient was put on a liquid intake of thirty two ounces increased pressure phases will occur and the lumbar puncture will yield 30 to 60 c.c. of fluid with each tap. He emphasises that the clinician has to recognise that he is dealing with volume relationship and not with pressure alone. On the eighth to tenth day following the injury these patients are gradually allowed more fluids until the intake of 32 ounces is reached. They are required to follow the restricted fluid and dry diet for three months after which their habits get changed and they come to appreciate that symptoms of headache, dullness loss of attention and memory and concentration arise from excess intake of fluids. He has given figures of the results of his treatment in which he has indicated that the mortality at the Temple University Hospital was 18.2% after the institution of this rigid dehydration treatment, while it was 25.6% in the control series of three years before the dehydration treatment. He claims that in his series the patients put on this regime of diet and intestinal dehydration regain consciousness promptly, are free from headaches, loss of initiative, memory disturbances, and mental fatigue so common in previous group. They returned to full activity in many cases in three months of the injury whereas in the previous series they took nine months. The follow up results of their cases are most impressive. They described 92% as free from post traumatic symptom complexes usually designated as neuroses, mentally retarded, or deteriorated types.

Lambert Rogers (1943) has again vehemently spoken of the dehydration treatment. The argument in its favour he says is that the bruised tissue whether comprising of brain or any other structure must be permitted to swell if it is to recover completely. If the swelling is not permitted there will be a reduction in its blood supply owing to the compression of the blood vessels supplying the damaged tissue. This

is detrimental. The most practical method of providing the space for the swelling is to reduce the amount of the spinal fluid by means of dehydration. He gives the details of the routine which he follows as propping the patient up in bed, giving retention enema of six ounces of 30% magsulph every six hours and restricting the fluid intake by mouth, a certain degree of thirst being the criterian of effective dehydration. Giving the results, he states that 550 cases were treated between 1929 and 1940. Of these only 25 patients died, the majority of them soon after admission. An absence of immediate and late complications was noted by him. Traumatic delirium that is cerebral irritation was infrequent and when it did occur was only of a mild character, and the absence of noisy, shouting restless type of patient was not to be seen. After discharge not one of these patients required readmission for the so called post concussional state. He attributes these improvements to the recognition of the importance of brain swelling and the provision thus made for it.

Trotter (1929) pointed out that if the swelling which is the normal reaction to injury is not allowed, the blood supply of the part is diminished and the ensuing anaemia may well result in cortical atrophy, or symptoms develop which are known as a post-concussion syndrome. This post-concussional state may concievably represent incompletely resolved cerebral contusion or subsequent atrophy due to failure to provide space for swelling of the brain

Thus the main arguments upon which the dehydration treatment is based is that it reduces the general edema of the brain, and also allowing space to swell it provides for better resolution of the contusion of the brain. They have shown that dehydration treatment lowers the mortality reduces morbidity. The figures of Lambert Rogers being even more impressive than those of Temple Fay.

It sometimes is a problem to arrive at a conclusion in the face of convincing arguments on both sides. There is no doubt that cerebral edema of a localised or general nature does occur in most of the serious cases of head injuries. It cannot be denied also that in some cases the intracranial tension is actually low and there is no evidence of cerebral edema, although the patient is seriously ill and shows clinical signs of cerebral compression and there is no blood clot either. It also is a fact that in cases of cerebral irritation the patient usually does become quieter after dehydration treatment, so does he by administration of sedatives alone. All seem to be agreed that dehydration is positively harmful in the initial stages of injury when the patient is in a state of shock. Few will disagree that the dehydration should be done even if spinal pressure is found to be low persistently or when on making inspection burr holes the brain is found to be not of a bulging type. There are cases of definitely cerebral hypotension. Leriche has described a syndrome caused by a diminished tension in the cerebrospinal system with symptoms of torpor, mental stupor or sometimes coma. There is no paralysis or contraction of pupils and the reflexes are positive. Such cases recover by injecting intravenously 30—40 c.c. of distilled water. There is almost an immediate increase of cerebrospinal pressure and the relief of symptoms. It is the usual impression that the post head injury headaches are due to rise of intracranial pressure. But the fact remains that these cases can be divided into two. Those with hypertension and those with hypotension headaches. The former are relieved by propping up in bed and dehydration treatment and the later by giving fluids freely. There is no doubt that hypotension is many times present both in acute cases and after the acute phase has passed off and dehydration treatment in them will be disastrous.

There do remain a few cases in whom there is marked intracranial tension as shown by lumbar puncture. It has been said that lumbar puncture readings are not reliable, and this is forwarded as an argument by the protagonists of dehydration treatment that these readings need not be taken to indicate a state of pressure inside

the skull. It is a fact that at times the lumbar pressure does not reflect the true state of pressure in the cranium. But what reliable criteria have we got at our disposal to know the rise of intracranial tension. The depth of unconsciousness, the bradycardia, the headaches, the restlessness and vomitting have all been ruled out as being unreliable at one time or another. I feel that of all the criteria perhaps the most important is the lumbar puncture readings. To my mind it sounds reasonable that if the clinical picture along with the lumbar pressure readings indicate a rise in the intracranial tension then dehydration treatment be carried out provided the above mentioned contraindications are absent.

High pressure and low pressure states: In the foregoing pages the cerebral edema has been mainly discussed. Needless to point out that if edema is present a rise of intracranial tension will be a natural accompaniment. Besides the edema, the pressure could be higher with increased secretion or decreased absorption of the cerebrospinal fluid. The reverse could happen that is there could be decrease in the amount of the cerebrospinal fluid due to decreased secretion or increased absorbtion. Both the conditions are present in cases of head injuries, and both conditions may be present in the same patient at different times. The excess of fluid will obviously lead to an increased pressure and a diminution of amount will lead to a state of hypotension.

That an excess of fluid is present with an increase of pressure is the general belief and it appears to be that it is more commonly present. The studies of Rand and Courville as detailed in the preceding pages leave little doubt that there is an increased production of spinal fluid in cases of head injuries. This is also confirmed by the work of all those who regard that a general cerebral edema occurs. Clinically also most cases of head injuries show an increased spinal fluid pressure on lumbar puncture; inspection barr holes under local anaesthesia also show many times the spinal fluid spurting out as though under pressure and a bulging brain. This is a common belief and there seems to be no doubt about it that in many cases a condition of increased pressure exists.

The reverse is however less well known, perhaps because it was recognised later at a time when the ideas about an increased pressure had been fully ingrained in most of the minds. The existence of hypotension was first recognised in 1915 by Leriche and by 1920 he described quite a well marked syndrome of hypotension occurring in closed head injuries. He observed that in cases of fracture of the petrous bone or the cribriform plate of the frontal bone in which there was a loss of cerebrospinal fluid, though the ear or the nose developed symptoms similar to those due to hypertension and that these disappeared immediately after intravenous injection of 40 c.c. of distilled water. This led to the conclusion that the symptoms were due to hypotension. What was remarkable in these observations was that the symptoms disappeared at the same time as the fluid escaped in a jet from the ear or the nose, indicating that a rise in the spinal pressure had brought the relief of symptoms. Similar condition was observed by him in cases in which a foreign body was removed from the brain and that led to a communication with the ventricle so that there was loss of cerebrospinal fluid to the exterior. In them as well the injection of distilled water had the same effect. Similar observations were later made in closed head injuries as well.

The symptoms of hypotension are the same as those of hypertension but are less marked. There is headache, loss of consciousness, may be coma, a slow pulse, and hyperthermia.

Every one is familiar that there are cases which show a low pressure on spinal manomentery and some of them have been put on record as well. Sydney Smith described one such case in 1926, and Malloch and Clark have recently (1946) described a case in which there was no fluid at all as

proved by lumbar puncture and cisternal puncture. In this case when the cisternal puncture was done the air rushed into the subarachnoid ventricular system with a noise and this led to his recovery as well. It is obvious that in this case there was a negative pressure.

Russell also agrees that during the last war the British neurosurgeons have come to recognise what they call a low pressure state. He further states that there is no doubt that in many cases of prolonged post traumatic stupor a condition of low or even negative pressure is present. In these cases the lumbar pressure is low and on inspection burr holes, the brain falls away from the dura. Obviously a state of dehydration exists. Is this dehydration the result of inadequate intake of water, or of biochemical disorder due to hypothalamic disorder ? is the question posed by him and he answers it by suggesting that the pituitary and hypothalamus mechanism control water metabolism to a very great extent and it seems probable to him that this mechanism gets disordered by trauma. Sydney Smith (1926) also suggested that the pituitary may be responsible for the hypotension in his case.

How often and in which case hypotension is going to occur is difficult to say but the clinician has to be alive to its possibility because the dehydration treatment is going to have disastrous results in these cases.

The need of lumbar puncture: The lumbar puncture in head injuries is not advised by some on the ground that it may lead to cerebellar herniation into the foramen magnum. By others it is advised as a routine stating that if it is performed in the lateral position and if only a small amount of fluid is allowed to escape then there is not much danger of herniation. The lumbar puncture on the other hand offers a number of advantages :— (i) it does give some idea of the cerebrospinal pressure, (ii) it tells whether blood is present in the spinal fluid or not. Rowbotham advises the lumbar puncture to be performed after 18 hours of injury.

Perhaps enough attention has not been given to the withdrawal of blood from the cerebrospinal fluid. The presence of blood is highly irritating and as shown by Weed the presence of serum in the spinal fluid retards its absorption because of its increased osmotic pressure, rather it draws more fluid from the circulation into the subarachnoid space.

One more usefulness of the lumbar puncture drainage is in those cases of head injury suffering from shock as well. In them one would like to administer fluids yet the fear of cerebral edema by such an action is always at the back of our mind. To save the situation, the fluids may be administered and the lumbar puncture be done to remove any excess of cerebrospinal fluid produced.

Summary

1. A brief review of the evolution of the knowledge of head injuries is made. In the pre-historic period the interesting fact emerges that some of the cranial perforations were performed in the Neolithic period for head injuries under local anaesthesia. One of the methods was very similar to the modern one. Also in 1500 B.C. the Surgeon knew that pulse may be effected by head injuries and paralysis may occur. In the period of Hippocrates and after upto the last third of 19th century attention was mostly directed to fractures. Thereafter more attention was given to cerebral injury and the experimental physiologists came in to solve the problem of post-traumatic stupor and allied states. The result of all these studies was that is in a rise of intracranial pressure which was responsible for the most of the ills. Attempts to reduce the intracranial pressure was made first by a decompression operation later by lumbar puncture and lastly by dehydrating the patient.

2. The nature of concussion is dealt with in some detail and it is concluded that the theory of Physiological neuronal injury as put forward by Denny Brown and Russell is most reasonable. The vascular

of Stomeyer, Kocher and Trotter which was popular upto now is being discarded. But it is possible to reject only a part of it and not the whole of it. We may say that a momentary anaemia of the brain does not occur, but we cannot say that a sudden thrust on the dura by a depressed fracture of the skull or its inbending cannot cause concussion. This phenomenon may be explained on the same basis 'compression concussion' of Denny Brown and Russell, but it needs to be studied further.

3. The occurrence of cerebral edema is discussed and all are agreed that a localised cerebral edema around a contusion occurs. There is a sharp difference of opinion as to the occurrence of generalised cerebral edema. It is also found that a state of increased intracranial tension does not occur in all cases of head injury, rather a condition of low pressure may be present in some cases with similar clinical features.. Naturally a marked difference of opinion is found on the subject of dehydration treatment. The state of hypotension is discussed and the reasons advanced for its possible cause are given. Hypotension also causes stupor, bradycardia, and hyperthermia. That cerebral irritation is caused by contusion of the under surface of the frontal lobe and not by the edema seems reasonable.

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VOLVULUS

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Introduction:

The practical importance of a disease varies with its incidence and its severity. On either of these grounds, volvulus deserves our close attention in India.

In a series now presented treated in a medium size district hospital it was observed that intestinal obstruction from all causes was the commonest form of acute abdomen. This agrees with a published observation of Bhargava[2] and contrasts with the incidence in Western countries where the commonest emergency by far is acute appendicitis.

Out of a total of 160 cases of intestinal obstruction treated during the last five years, 33 have been due to volvulus, namely 20%. This contrasts with the much lower percentage given by Wangensteen[14] of 4% in a large series in the U.S.A., but agrees with the observation of Ojha[10] of 19% in a series of 110 cases. It is considerably less than the 51% found by Bhargava in the series of 398 cases he reports. This latter is an extremely high figure reminiscent of the reported incidence in Russia and the Baltic where it commonly accounts for 50% of cases of intestinal obstructions (Perlman quoted by Wangensteen[14]).

Enough has been said to show the importance of volvulus, therefore, in this country, where it appears that intestinal obstruction is the commonest abdominal emergency, and where volvulus accounts for 20% to 50% of these cases. The relative percentage of volvulus compared with that due to other causes of intestinal obstruction in this present series is shown diagramatically (diagram no. I-A) and this is compared with a similar diagram from Wangensteen's series (diagram no. I-B).

A point to which particular attention will be drawn is the considerable differences which exists between volvulus of the

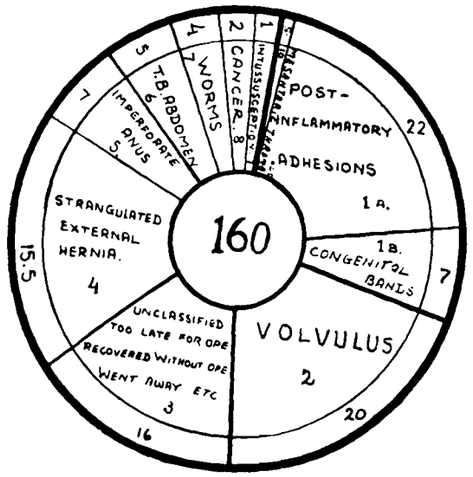


Diagram I-A.

Chart indicating percentage of all causes of intestinal obstruction during years 1948—1953 at the Evangeline Booth Hospital, Ahmednagar. Total number admitted with intestinal obstruction = 160. Approximate percentage of surgical admissions = 3%.

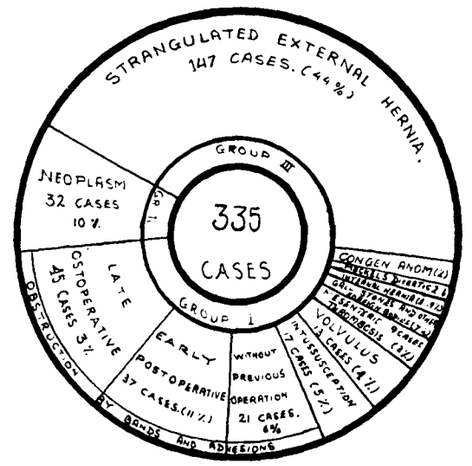


Diagram I-B.

Similar chart for comparison from Massachusetts General Hospital — Wangensteen — 10 year period. Total number admitted with intestinal obstruction = 335. Approximate percentage of surgical admissions = 1%.

small intestine and caecum on the one hand, and volvulus of the pelvic colon on the other, in pathogenesis, in diagnosis, in treatment, and in prognosis.

Pathogenesis

Definition :

Volvulus is the term applied to the twisting of any part of the bowel on its mesentery so that the ends are closed and there is a greater or lesser degree of occlusion of the blood vessels. Further, the bowel above the twist is also obstructed. For a torsion to occur there must be a moveable loop (which implies the presence of a mesentery) and some degree of fixation of the two ends of the loop. Also the nearer the two ends are together and the more fixed they are, the likelier a torsion becomes. The likelihood is again increased if there is any adhesion at the apex of the loop.

Pathogenesis of Volvulus of the Pelvic Colon

These conditions are most frequently fulfilled in the *Pelvic Colon,* of which there are 20 instances (60%) in the present series, compared with 12 (34%) of the small intestine, and 2 (6%) of the caecum. Yet the average age of the cases of volvulus of the pelvic colon is 50, which contrasts with the average of 30 in the small intestine and caecal cases. This suggests some long acting and acquired causes in additional to any congenital predisposition. The congenital predisposition may consist of a long pelvic loop. Cunningham's Text book of Anatomy gives the length of the pelvic colon as varying between 5" and 35" with an average length of 16" or 17". Edgren quoted by Brusgaard[5] reported that the average height of the mesocolon was 5" while in cases of volvulus it was 10" in height. This elongation of the mesentery is well illustrated in an x-ray (No. 1) of a case after reduction.

The attachment of the base of the pelvic mesocolon varies from an obtuse to an acute angle and the latter which brings the ends close together is clearly a predisposition also. But these facts do not explain

X-ray No. 1.

Barium enema of elongated and distended Pelvic Colon after reduction.

the late age incidence. It appears likely that (a) diet (b) habits of eating are important. It is apparently the case that volvulus is more frequent in the countries with vegetarian diet than in those with non-vegetarian diet, i.e. in India and Africa (Kerr and Willis[8]) and Eastern Europe. It is especially significant that the incidence rose sharply in Norway (Bruusgaard[3]) during the war years when the diet temporarily became more vegetarian and contained more roughage.

An inquiry into the diet of the Maratha farmer group, in which volvulus mostly occurs in this area, showed that their consumption of cereal is about 1 seer a day or 7 seers a week, which can be compared with a ration scale of 3 seers a week which is fairly adequate for sedentary workers. The farmers also are in a habit of eating one main meal at midday (and one or two subsidiary meals) which consists almost

entirely of cereals and a little vegetable or dhal. The residue of such a heavy meal will tend to accumulate in the pelvic colon and weigh down a congenitally long colon, and may well, over a period of years, elongate it further and so pull on the mesentery tending to shorten the base of the mesocolic attachment. The weight of solid excreta in the pelvic colon reservoir which might exert this pull can be judged from the fact that Kirk[6] states that in calculating for the disposal of solid excreta 16 oz. daily must be allowed for persons on vegetarian diet compared with 6 oz. for those on a non-vegetarian diet. It may be that there is a neurogenic factor also as is suggested in one case in which a barium meal was given to a patient three weeks after reduction of his volvulus and it was found that the barium did not reach the pelvic colon for 48 hours and was still not evacuated, after which the patient tired of coming for more x-rays (2). I do not think

X-ray No. 2.

Delay in emptying colon in a patient who had repeated attacks of Volvulus. The head of the Barium meal has reached the pelvic colon only after 48 hours.

the habits of evacuation as such can be blamed as these are usually very regular in a village population.

As indicated above, volvulus occurs almost exclusively in the farmer who is himself a workman, or in the farm labourers. The predominence of males is very marked (18 males to 2 females) and this is somewhat puzzling, although the wider pelvis and the more lax abdomen of the female have been given as reasons. I will not consider here the actual mechanism of the primary attack except to state that a loaded colon and peristalsis must play a part, but once an attack has occurred there is a considerable tendency to recurrence and the pathological basis for this was evident in several cases of the series where there was thickening of the mesocolon with a "shrinking mesocolitis" evidenced by white matted fibres criss-crossing the mesentery and bringing the ends of the loop closer together; and in one case there was a band attaching the apex to the parietal peritoneum. In this series the 20 attacks recorded were in 14 patients, 3 patients being admitted three times each and one twice. Apart from the admission to hospital for recurrence, the history is often given of repeated minor attacks which are relieved spontaneously. One man actually complained of repeated attacks of inability to pass urine but he proved on investigation to have no urinary obstruction but recurrent attacks of volvulus with an enormous dilated, stomach-like, pelvic colon revealed at operation during a remission. The ends of the bowel were also observed to be narrowed markedly in this case and somewhat in others.

The explanation of the minor attacks probably lies in the fact that the first twist of the mesentery is of 180 degrees only. The symptoms arising from this depend on whether the twist along the bowel is distributed evenly or whether there is an acute torsion at one or both ends. In the latter case the clinical picture of volvulus may occur. Frimann Dhal[6] states that 35% of cases of established volvulus have only 180 degrees rotation, about 50% go on to a rotation of 360 degrees and the

remaining 15% proceed to 540 degrees. This rotation is generally but not always in a clockwise direction. In the case of a rotation of a 180 degrees the next massive peristalic movement may result in untwisting, with relief of symptoms or alternatively the twist may be increased by another 180 degrees causing an irreversible condition because of progressive distention, resulting in what has been aptly compared to a balloon tyre clamped between the anterior and posterior walls of the abdomen.

In general, then, it can be said that in volvulus of the pelvic colon there is a congenital predisposition *plus* slowly acting acquired causes of a mechanical nature which are of major importance. There has been no evidence of diverticulitis in the series nor has there been any history of recurrent dysentery.

Pathogenesis of volvulus of the caecum and small intestine

The Pathogenesis of *volvulus of the caecum* and adjoining ascending colon *and of the small intestine* is much more directly dependent on congenital abnormalities. The average age incidence of this group is 30 only compared with 50 in the pelvic colon group. The male sex again predominates in the proportion of 12 to 1 which is surprising in view of the strong congenital factor in aetiology. Possibly village women with acute abdominal conditions attend hospital less frequently and die unrelieved.

Volvulus of the caecum :

As regards the mechanism of *volvulus of the caecum* and the part of the ascending colon adjacent, it is clear that this cannot occur without an abnormal congenital meso caecum. This is well illustrated in the single patient of the series (who had two attacks) and is shown in diagram No. II. There are here two obvious fixed points, the proximal one being related to a firm peritoneal band which passed from the mesentery of the terminal ileum near the ileo-caecal valve to the right iliac fossa, and the distal one being at the attachment of the ascending colon to the posterior wall.

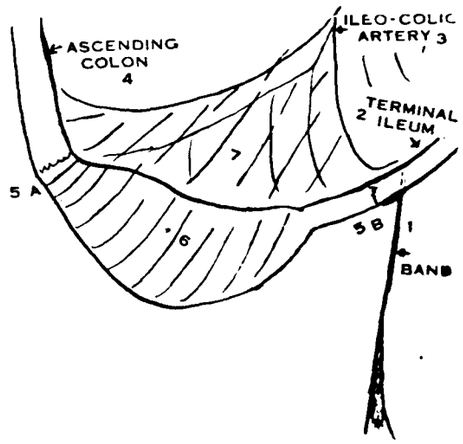


Diagram II.

DIAGRAM OF CAECUM AFTER UNTWISTING

1. Band from ileum to iliac fossa.
2. Terminal ileum.
3. Ileo-colic artery.
4. Ascending colon.
5. (A) Pressure lines.
   (B) At points of torsion.
6. Congested elongated caecum.
7. Congested thickened meso-caecum.

The occurrence of this mesocaecum has been described often in this connection and is due to the failure of fusion of the meso-caecum which exists in a stage of foetal development and which normally fuses with the peritoneum of the posterior abdominal wall in the third stage of rotation of the bowel (Dott[5]). However, the band from the posterior leaf of the mesentery of the terminal ileum and about 2" from the ileo-caecal valve does not appear to have been described clearly before. Besides its relation to volvulus of the caecum described above, it appears significant also in relation to the more difficult problem of the pathogenesis of small intestine volvulus.

Volvulus of the small intestine :

It is said that this does not occur unless predisposed to by previous inflammation or a congenital abnormality, but this latter is not clearly defined (Rodney Smith[11]). Normally the fixed points of the small intestine viz. the duodeno-jejunal junction and the

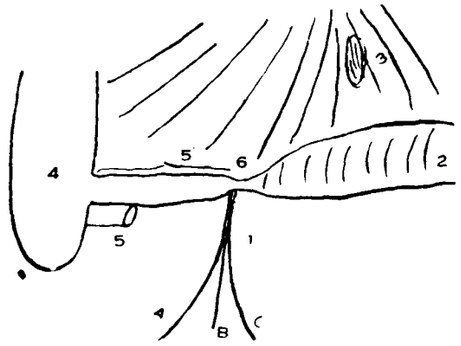


Diagram III.

DIAGRAM OF SIMPLE OBSTRUCTION BY BAND

1. Band from terminal ileum dividing into 3 parts
   (A) to Right iliac fossa
   (B) to Brim
   (C) to Pelvis.
2. Congested distended terminal ileum.
3. Enlarged gland in congested mesentery.
4. Mobile caecum.
5. Ileo-caecal fossa.
6. Constriction at attachment of band.

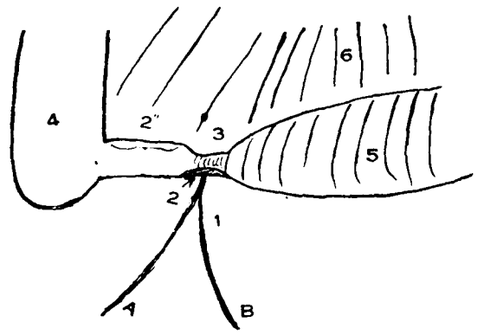


Diagram IV.

DIAGRAM OF BAND IN SMALL INTESTINE VOLVULUS

1. Band from terminal ileum to —
   (A) Right iliac fossa.
   (B) Pelvis.
2. Fressure necrosis at site of torsion.
3. Constriction of bowel at band attachment.
4. Caecum.
5. Congested dilated bowel.
6. Congested thickened mesentery.

ileocaecal junction are very far apart. A true rotation of the whole of the small intestine on the axis of the superior mesenteric artery only occurs when the normal development stops at the end of the second stage of rotation and the caecum lies in the subpyloric region, so approximating the two usual fixed points and resulting in a volvulus neonatorum (Jolleys[7]).

In the adult the ileo-caecal junction is on consideration normally hardly a fixed *point* because the terminal ileum is inserted on the medial side of the loose caecal bag with a free mesentery on that side which allows a considerable range of movement in all directions to the terminal ileum at the junction. This is clearly an ideal arrangement to avoid the likelihood of a volvulus. If this normal arrangement does not hold because of a precaecal fixation of the mesentery, then a condition arises which predisposes to a volvulus. In this series of eleven proved cases of small intestine volvulus and one probable (relieved spontaneously during or immediately before operation and recognised by congestion of the lower ileum and signs of lines of pressure) two cases showed a well defined band similar to that observed in the caecal case, fixing the mesentery at its point of attachment, and in one case a band which extended on the left (inferior) leaf of the mesentery up towards the duodeno-jejunal junction. I have made a series of sketches immediately following operation in some cases where I have observed the ileocaecal band (diagrams III, IV & V). Its relation to Lane's ileal kink (which was attributed by Lane to adhesive or reactive peritonitis, following the adoption of the upright posture in man) is uncertain as judged by the diagram appearing in Rose and Carless's Text Book of Surgery[13]. Moreover Sir Arthur Keith is there quoted as attributing such bands to irregularity in the normal process of the descent and attachment of the caecum to the posterior abdominal wall, and the observations in this series tend to support such a congenital origin. It is of great interest that Ojha mentions 3 cases of a band in the ileocaecal region out of 13 in his series (excluding cases due to tuber-

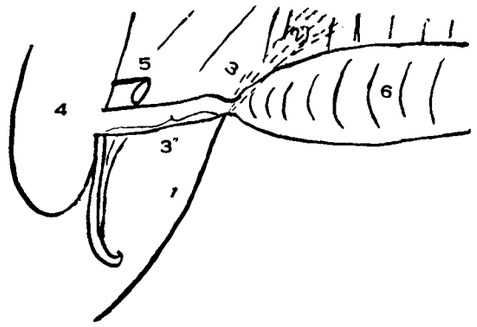


Diagram V.

DIAGRAM OF BAND IN SMALL INTESTINE OBSTRUCTION

1. Band from terminal ileum to right iliac fossa.
2. Fibrosis extending behind ileum to inferior leaf of mesentery.
3. Constriction of small intestine at site of band.
4. Caecum.
5. Ileo-colic fossa.
6. Distended small bowel above band.

culosis). My colleague Dr. A. B. Cook of Anand has also observed cases of these bands (personal communication).

In this connection I would like to draw attention to a short series of 4 cases of *simple obstruction* by kinking of the small intestine at exactly the same point but without volvulus each associated with a definite raised peritoneal band which constantly arose from the lower leaf of the mesentery and passed to the peritoneum of the right iliac fossa. In one case it extended as a fibrotic inflammation into the adjacent mesentery and caused a narrowing of the ileum. In another of these cases the caecum was only partly descended and the band extended up to the region of the ascending colon out of sight. These cases suggest that the kinking of the lower ileum at a fixed point followed by distension of the lower ideal coils may in some cases develop as a simple obturation type of obstruction, but in others the heavy lower coils during hyper-peristalsis may become twisted 180 degrees on the mesentery (usually in a clockwise direction) and this gives rise to the added hazards of volvulus and strangulation. The upper point of the twisted loop appears to be quite variable, the cases in this series showing from half the small intestine to about 2½' to be involved. It is suggested, therefore, that the upper point is, so to speak, fortuitous, depending on the accidents of peristalic movement, the essential factor in aetiology being the congenital precaecal fixation at the lower point.

To revert to the cases of volvulus, apart from the 3 cases with bands, another case showed a mobile caecum and mesocaecum which had been drawn over almost to the mid line, making four cases of evident congenital abnormality. There were also two cases of inflammatory adhesion, one of the apex of the twisted loop to the parietal peritoneum at the site of an old bullock-gore, and the other of an adhesion to the site of an inguinal operation for undescended testicle a year previously. One other case was associated with pregnancy. In the remaining five other cases there was no evident congenital abnormality but the distal point of torsion was in each case from 1" to 5" from the ileocaecal valve, the caecum being in a normal position. It has occurred to me, although I have not yet had the opportunity of testing it on further cases at operation, that these cases may have been due to a *congenital precaecal attachment* of the mesentery about the point where torsion later occurs, with a resulting slight degree of mobile caecum, which could easily be overlooked if not specially examined. This condition would be more liable to lend itself to volvulus than the normal attachment of the mesentery at the ileocaecal junction. The important point would then be shown to be a short mesentery and a precaecal fixation during the fusion of the mesentery, the presence of a band being an added factor in some cases either primary, or secondary to the resulting pull.

Clinical Aspects

In view of the considerable clinical and other differences between volvulus of the small intestine (and caecum) and of the pelvic colon they will be considered separately.

Volvulus of the small intestine and caecum:

The patient, usually an adult male aged about 30 (limits between 25—68) presents himself with a history of a severe abdominal pain for 24—48 hours. The more severe the pain the earlier he tends to come and conversely those that come later have a sub-acute type of volvulus, the longest history noted being twelve days. Two main types of volvulus, are therefore observed:

(a) The more acute type where there is strangulation and threatening or actual gangrene already on admission. The patient in this group is shocked and has a rapid pulse, 110—150, of poor volume and history of vomiting. The temperature is not of much significance and may be sub-normal. There is a moderate, not great, distension of the abdomen and diffuse tenderness. Peristalsis is not seen usually and on auscultation the signs of hyper-peristalsis are occasional or absent. There *may* be a localised swelling due to the actual twisted small intestine coils or caecum but this has only been observed once. The white cell count in *one* case was found to be 17,000 but this is not constant. Five of the cases belonged to this acute type and such a picture immediately suggests threatening or actual gangrene. The subacute type, however, may also include cases of gangrene and one patient came on the fourth day with marked distention and visible peristalsis and proved to have extensive gangrene.

(b) The subacute type complains of inability to pass faeces or flatus, colicky pain and distention and the general condition may be fairly good. In this type peristalsis can easily be seen and heard and there is not generalised tenderness to the same extent as in the acute variety.

Every case of volvulus of the small intestine has some degree of strangulation of the blood supply and therefore urgent diagnosis and treatment is essential. It is especially the acute group where the classical signs of obstruction are not marked that is dangerous and the temptation to temporize and use a duodenal tube with Wangensteen's method of suction or a Miller-Abbott tube must be resisted.

The only indication in this series of 160 cases including all types or obstruction accepted for using these (in view of the high incidence of volvulus found) was a clear history of a recent septic inflammatory process such as an appendicitis, where experience showed that they did better with suction and no fluids by mouth and adequate anti-biotic therapy, than with laparotomy, as this adhesive type of obstruction does not commonly develop gangrene. Even when there is no volvulus and obstruction is due to a band, either congenital or late inflammatory, temporizing is considered dangerous because of the liability to pressure necrosis of the bowel by the band.

Plain x-rays in the standing position have been used in the series regularly for

X-ray No. 3.

Typical low small intestine obstruction without volvulus.

X-ray No. 4.

Irregular fluid levels in strangulated volvulus of small intestine with broad hoops in small intestine above due to inhibition of peristalsis above the volvulus.

X-ray No. 5.

Gangrenous small intestine loops in volvulus pointing to the ileo-caecal region.

all cases of intestinal obstruction and found to be of great value. However, here also the plain x-rays of an acute small intestine volvulus with strangulation does not give the very obvious picture which is presented by an acute simple (obturation) obstruction or a volvulus of the pelvic colon. There is a blurring due to free fluid and the fluid levels may be few and the inverted hoops broad due to reflex inhibition of peristalsis above the volvulus (x-ray no. 4) which contrasts with the well marked narrow loops mounting one above another up to the left hypogastrium with differential fluid levels in the limbs of each loop, (indicating active peristalsis) in a case of simple obstruction without strangulation, as illustrated in x-ray no. 3. Sometimes only a few small gas bubbles are seen in the blurred background, probably in the actual strangulated loop, and sometimes these may be seen as almost circular loops with the open base pointing to the ileocaecal region (x-ray no. 5).

On opening the abdomen there is an excess of free fluid which is blood stained if gangrene is threatening or present and the coils are deeply congested always and of course blue-black if actual gangrene exists. The caecum is pale and collapsed. Reduction must be extremely gentle. The points of danger are not only near the apex of the loop but also where there is local pressure necrosis of the wall of the terminal ileum at the position of twist. In late cases the site of the twist may show adhesion of the tissues and haemorrhages into the thickened mesentery and grossly enlarged mesenteric glands. The first movement should be to determine whether there is any adhesion of the twisted coils to the parietal peritoneum at the point of

any previous operation or injury, and if present this must be divided first. Subsequently untwisting is tried gently in the anti-clockwise direction first, and if this fails in the opposite direction.

Resection of lengths of small intestine, varying from 2½ feet to 11 feet, was carried out in four cases with one survival as regards the small intestine resection but she later died (as mentioned subsequently) due to a concomitant volvulus of the pelvic colon. Three other cases had local pressure necrosis only at the site of constriction. In such cases, when possible, the line of threatening necrosis was simply buried with a few interrupted catgut sutures and two such cases survived, but in the third one the bowel was opened during reduction along the line of necrosis and an open anastomosis of the two ends had to carried out and the patient died. Of the remaining seven cases without gangrene or pressure necrosis three survived and four died, a rather disappointing result, making a total mortality of 57% in fourteen treatments. On examining the causes of this high mortality compared with a much lower one in the pelvic colon series the first factor is the undoubted exudation of toxic fluid through the damaged small intestinal wall into the free peritoneal cavity, (Aird 1) the nature of the toxic agent being probably a proteose or peptone of B. coli origin.

The mortality in this group may be contrasted with that of external strangulation in inguinal herniae of which there were twenty-five in this series with a mortality of two only, viz. 8%. The explanation of this gross difference in the same series probably depends upon (1) the external strangulation comes earlier to hospital, (2) the toxic fluid exuded through the damaged wall is outside the main peritoneal cavity—and must not be allowed to enter it, (3) there is less handling of the bowel even if a resection is necessary. In the cases without gangrene where a good result was anticipated after reduction one (the only old man in the series) died due to poor general condition and hypo-proteinaemia, and another, a sub-acute, after reduction had uncontrolled diarrhoea and slowly deteriorated in eight days. There are yet two cases where there was no obvious mechanical or general reason for their death and here the explanation may be that advanced by Devine[15] and others that the highly toxic contents of the closed loop which are not absorbed to any extent into the portal system, because of defective circulation, after reduction, pass into healthy bowel where they are rapidly absorbed and cause a grave toxaemia. This would account for the gradual deterioration and rise in pulse rate in these two cases who died within twenty-four hours.

The mortality in this series as regards small intestine obstruction is the same as that reported by Ojha (67%) but much more than that reported by Bhargava (27%). It could probably be improved by:—

(a) *Spinal anaesthesia* which makes the operation mechanically easier and would also tend to a more rapid restoration in the bowel motility so that, as reported by Ojha, the patient may have an evacuation even before he leaves the theatre. This has not been used in this series because the use of a high spinal anaesthesia in a poor risk patient requires a specially qualified anaesthetist which was not available. Therefore general anaesthesia was employed and this is a practice approved by Wangensteen, provided always that the stomach has been emptied before the operation and is kept empty by constant suction during the operation. In order to aid closure, use was made of intravenous injection of Myanesin 10 cc., and also in a recent case which survived prostigmin was given on the table and a flatus tube passed and this was repeated four hourly. This procedure is regarded as safe only if the bowel is not damaged.

(b) A greater attention to blood chemistry, especially blood protein, blood chloride and alkali reserve would certainly pay large dividends. In this series adequate and controlled intravenous saline was used

but usually blood or plasma was not available at short notice.

Volvulus of the pelvic colon :

The large bowel series consists of twenty clinical attacks occurring in fourteen patients which at once draws our attention to the high rate of recurrence. As regards age, the average is fifty, which males predominating in the proportion of 12 to 2, one of the women being pregnant and having an interlocking small intestinal obstruction also. There is often a history of previous attacks. There were three deaths, a case mortality of 16%, or if a patient who ran away and probably died is included, 20%. As compared with small intestine volvulus where an acute origin is frequent, here a sub-acute type is more common. The patient presents himself usually after 48 to 72 hours or later with a history of an acute abdominal pain with vomiting at onset. There was subsequently been absolute constipation and increasing distention. The distention may be enormous and the patient complains of discomfort with acute exacerbations of colicky pain. On examination the maximum swelling is in the upper abdomen more on the left then on the right, and sometimes the distended coil can be visualised through the abdominal wall. Hyperperistalsis is not usual, in fact it has only once been clearly observed, when it was strikingly obvious. The pulse is of good volume and fairly slow 80—90. There is not real tenderness although the patient will complain of pain on firm pressure. The temperature is usually normal and the patient not in shock. The clinical picture, once appreciated, is fairly easy to recognise and diagnosis can be established with certainty by a plain x-ray. In an early case the pelvic loop is shown distended by gas (x-ray no. 6) but a little later there is an extensive outpouring of fluid which gives a characteristic picture, aptly likened to a pair of scales, which is pathognomonic (x-ray no. 7). The remainder of the

X-ray No. 6.

Early volvulus of pelvic colon with distension by gas. Fluid not marked. (Note distension of caecum).

X-ray No. 7.

Typical x-ray appearance of volvulus in the pelvic colon with resemblance to a pair of scales.

X-ray No. 8.

Barium enema in case of volvulus of pelvic colon with typical appearance of 'beak of a bird or prey' at the torsion site.

colon may show some gas, especially the caecum, but there is usually little evidence of small intestine obstruction (hence little vomiting and good general condition). Further confirmation is hardly necessary but a barium enema introducing the tube into the rectum only, shows a characteristic picture at the recto-pelvic junction where the twist gives the barium an appearance of "the beak of a bird of prey" (x-ray no. 8).

The usual treatment has in the past been laparotomy, although Bhargava has already recommended the careful use of a flatus tube "even in volvulus up to four days duration". The good results obtained in this series can be largely attributed to a further extension of the use of the flatus tube but the introduction should always be under direct vision with a signodoscope. This method is recommended in a paper by Brusgaard[3] reporting a series of 168 cases at the Ulevaal Hospital in Oslo. There were only four deaths out of 136 reductions by this *method*, the total number of deaths being 13 in a series of 168 treatments by all methods. This good result is strongly supported by this present series in which the method was used twelve times without a single mortality. In two of these cases the patient had previously had a reduction by laparotomy and in one case after reduction by flatus tube, doubt was felt about the viability of the bowel and the position of the tube, which was resolved by laparotomy which showed the tube in satisfactory position and no perforation. One man however became frightened by the procedure which failed and refused a laparotomy and ran away with a likely fatal result.

The technique is not entirely simple and cannot be carried out without adequate precautions. The following rules are suggested.

(1) That there must be no evidence, or even suspicion of strangulation.

(2) The diagnosis must be confirmed radiologically.

(3) Sigmoidoscopy should be carried out by the surgeon in a theatre ready to proceed at once to laparotomy if needed.

(4) If the bowel wall at the site of torsion is grossly congested, suggesting the possibility of local pressure necrosis, the tube should not be passed.

(5) There must be no satisfaction, unless the distension and pain are completely and rapidly relieved and the patient's general condition shows no evidence of peritonitis in the post-operative period.

As regards the technique, the normal pelvi-rectal junction is about 15 cms. from the anus. However, in a volvulus, it may be pulled up much higher and even a normal 30 cm. long sigmoidoscope may just fail to reach the twist although it is usually in sight. Only when the twist of the bowel can be seen by the sigmoidoscope is the rectal tube (50 cms. long of 7 cms. diameter) introduced under vision, and it usually passed easily through the twist resulting in a not unwelcome bath of liquified faeces for the surgeon. Sometimes the

end of the sigmoidoscope slips past the twist itself and the barrel fills with liquid faeces and reduction takes place. In fact reduction has occurred by a too zealous radiographer pushing a rectal catheter for a barium enema up beyond the twist without any ill-effect, and a case is recorded of an old gentleman relieving his own volvulus by the rectal tube once a week for 20 years (quoted by Brusgaard[3]). The procedure is justified because the pelvic volvulus is generally a *loose* twist. It failed in one case — in the case mentioned who ran away because the twist appeared to be carried over to left so far that the barrel of the sigmoidoscope could not be manipulated to see it because of the obstruction of the right tuberosity of the ischium. It would, of course, be fatal simply to push blindly, and if manipulation does not demonstrate the twist the method must be abandoned. Another practical point is that the flatus tube must have another tube of equal length attached to it so that as the sigmoidoscope is withdrawn the tip of the flatus tube can remain in its place above the site of torsion. After removal of the sigmoidoscope the lower end of the flatus tube is stitched or strapped to the anal region for 24 hours.

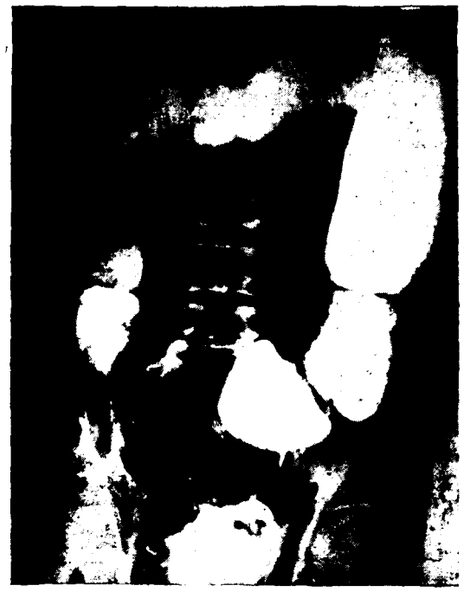
The next method of reduction is that of laparotomy and the passage of a rectal tube from below after laparotomy has been carried out. It is again emphasised that this must always be carried out, if there is any evidence or suspicion of gangrene or peritonitis. The clinical condition in the smaller series of cases where such gangrene or peritonitis is present shows a rapid pulse, diffuse tenderness and continued vomiting. In two of the four cases which were judged to be suspicious cases of gangrene, vomiting had actually stopped on the day of admission in two and both of these proved at laparotomy not to have actual gangrene. One of these survived operation and reduction but the other developed peritonitis. In the remaining two cases there was frank massive gangrene. Time seems to have little relation to the onset of gangrene because the worst case of all had symptoms for 24 hours only. This patient had frequent vomiting and an almost imperceptible pulse and marked generalised tenderness. The first case of gangrene has already been mentioned in the small intestine series as having interlocked coils of both the small intestine and the pelvic colon with gangrene of both in a late pregnancy. After doing a classical Caesarian section the affected small gut loop was resected and the pelvic loop was exteriorised by a Paul Mickulicz procedure. She promised to make a recovery for seven days but then the upper stump of the loop became gangrenous probably because of vascular mesenteric damage and a spreading septic thrombosis of the mesocolon, and she developed general peritonitis. To avoid this in the second case both ends of the pelvic loop were closed after resection of the gangrenous part and then a caecostomy was done to relieve pressure, hoping at a later date to re-anastamose the ends of the pelvic colon. Unfortunately the patient died as the operation was being completed (x-ray no. 9).

In choosing which method of treatment should be adopted it is felt that laparotomy has no advantage over the simpler reduction by a sigmoidoscope except where there are clinical grounds for suspicion of threatening gangrene or peritonitis. No more is achieved and there is the added risk of a laparotomy in an often elderly and rather weak person. Some persons have advocated fixing the pelvic loop after reduction but experience indicates that the recently distended bowel is not fit for any sutures on which there may be tension.

The question of the radical cure of the condition still remains. Clearly the cure is a resection of the pelvic loop and an anastomosis, without any slack to allow torsion at an interval after the attack. Although four patients of the series had two or three recurrences in hospital, only two of them gave permission to proceed with the radical cure, and none of those with only one attack. One resection was carried out seven days after the admission of the patient with acute volvulus for the

X-ray No. 9.

X-ray of great distension of a gangrenous pelvic loop with fluid level (to left) and distended ascending colon to right.



X-ray No. 10.

Barium enema. X-ray of colon taken 14 days after resection of pelvic colon. (There is relative stenosis at the point of resection but no symptoms of obstruction).

third time (which was relieved by sigmoidoscope and flatus tube). There was adequate preparation and a planned resection was carried out uneventfully as shown by x-ray no. 10, taken 14 days after resection of 35 cms. of the pelvic colon. The other case was found to have sub-acute congestion and induration of the meso-colon and had not had adequate preparation so a Paul Mickulicz type of resection was performed. It was satisfactory except that the patient had to be in hospital for seven weeks after the operation instead of two, as for the internal one-stage abdominal resection.

Discussion

The main question which arises is whether a conservative method of treatment is justifiable in either the small gut or the large gut varieties of volvulus. This again depends on the viability of the gut under the conditions of torsion which arise in volvulus of the small and large intestine respectively. Anatomical studies of the blood supply have been made. Ross[12] states that the small intestine stands distention better than the large because the communicating vessels at the bases of the vasa recti and across the bowel are much more developed in the small than in the large intestine. This study was especially made in relation to the question of safe levels of resection for cancer of the large bowel. It does not seem to give due credit to the ability of the pelvic colon to distend hugely in volvulus without rupture or gangrene, unless the twist of the *main* vessels seriously damages the blood sup-

ply. On the other hand Elsberg, quoted by Wangensteen[14], states that the blood vessels run mainly outside the muscles or between the muscle coats in the large intestine and more submucously in the small intestine where they cannot stand so well the pressure of distention as in the large bowel. This may offer some explanation of the greater liability of the small intestine to gangrene; but observations at operation suggest that the main question is the degree of pressure on the main vessel, that is the *tightness* of the twist at the base of the mesentery rather than the relative blood supplies of the bowel walls.

In general this series seems to show that many cases of volvulus of the small intestine are liable to early gangrene and therefore a conservative method such as by the Miller Abbott tube is strongly contraindicated; while most cases of volvulus of the large intestine do not proceed to early gangrene and therefore the conservative method of reduction is justified and gives, in fact, better results than have been obtained by the routine use of laparotomy. The decision as to which method to adopt in a particular case depends on the clinical evidence, aided by x-ray and laboratory investigations, and if the conservative method is chosen it must be subject to reversal immediately if full reduction is not rapidly obtained.

TABLE I

SMALL INTESTINE AND CAECAL VOLVULUS

14 Treatments in 13 Patients

Gangrene	No. of cases	Died	Living	No Gangrene	No. of cases	Died	Living
Resection ...	4	3	1	Surgical Reduction ...	5	4	1
Oversewing Pressure necrosis ...	2		2	Spontaneous Reduction (a) One on table ...	1		1
Open Anastomosis at Pressure Necrosis ...	1	1		(b) One Recurrence (Caecum) ...	1		1
	7	4	3		7	4	3
Total mortality		57%					
Combined Total ...		57%					57%

Mortality Rate for Small Intestine only excluding Caecum: 67%

TABLE II

PELVIC COLON VOLVULUS

20 Treatments in 14 Patients

Gangrene	No. of cases	Died	Living	No Gangrene	No. of cases	Died	Living
Resection (Internal) ..	1	1		Spontaneous Reduction	1		1
Exteriorisation and Resection ...	1	1		Reduction by Sigmoidoscope and Tube ...	11		11
				Reduction by Laparotomy and tube	5	1	4
				Ran away after failed Sigmoidoscopy ...	1	(1)	
	2	2			18	1 (1)	16
Total mortality ...		100%				6%	(11%)
Total combined (treatment) mortality ...				16% (20%)			
Total combined (patient) mortality ...				21% (28%)			

TABLE III

Total Results in 33 Treatments in 26 Patients*

Small Intestine & Caecum	No. of cases	Died	Living	Large Intestine	No. of cases	Died	Living
Treatments ...	14	8 57%	6	Treatments ...	20	3 (1) 16% (20%)	16
Patients ...	13	8 61·5%	5	Patients ...	14	3 (1) 21% (28%)	10

Total Mortality:—			
Treatments ...	32·3%	(35·3%)	
Patients ...	42·3%	(46·1%)	

* One patient had both small and large intestine volvulus; therefore is included on both other tables separately.

Note:—The figures in brackets refer to a patient who went away without treatment.

SUMMARY

1. A series of 160 cases of intestinal obstruction is presented of which the over all mortality was 31% (45 deaths and 4 patients who went away without treatment, presumed dead).

2. Intestinal obstruction forms the commonest abdominal emergency in the period under study. Comparable incidence of appendicitis during this period was 58% and of gynaecological emergencies 17%.

3. Volvulus forms the second largest group of cases of intestinal obstruction accounting for 20% of total cases, being only exceeded by post inflammatory obstruction with 22%.

4. Volvulus of the pelvic colon accounts for 60% of the treatments for volvulus, the small intestine for 34%, caecum for 6%.

5. The mortality rate for volvulus of the small intestine and the caecum in the series is 57%, while that of the pelvic colon is 20%.

6. The reasons for this difference in mortality in the two groups are discussed.

7. A conservative method of reduction for volvulus of the pelvic colon is recommended, subject to adequate precautions.

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POTT'S PARAPLEGIA

(A Study of 41 cases)

by B. MUKOPADHAYA, Patna.

Pott's paraplegia or paraplegia caused by tuberculous disease of the spine is one of the worst complications of skeletal tuberculosis. Pott was not the first person to recognize the association between the two conditions. There are references to the co-existence of paraplegia with angular deformity of the spine in the Hippocratic corpus. Dales Champs (1570) co-related the two conditions as cause and effect but Pott's vivid and masterly description of the condition remain unsurpassed upto-date and the condition is named after him almost universally.

This paper is based on a careful follow-up study of 41 cases of Pott's paraplegia seen among 315 cases of Pott's disease of the spine since April 1949. This gives an incidence of nearly 13.3% — a figure slightly higher than that of other authorities. It is quite possible that because of the relative gravity of the condition and helplessness of such patients, these cases tend to come up for hospitalization and the figure of 13.3% is not a true index of the incidence of this complication in an unselected group. The fact that such patients are being treated at a particular centre tend to attract these cases towards the centre. This is proved by the fact that of late we are getting more cases of Pott's paraplegia than we did a year or two ago.

Of these 41 cases, 14 were in children below the age of 12 years out of a total number of 160 patients of spinal disease below that age — an overall incidence of just less than 9%. The rest of 27 cases were among 155 patients between the ages of 12 and 50 years — an incidence of 17.4%. In our series, this complication is strikingly higher in adult patients and relatively less common in children — a fact which is contrary to the experience of many other authorities.

The distribution of the site of the disease in the paraplagic cases was as follows :—

	Cases
Atlanto — axial tuberculosis	— 1
Upper cervical	— 1
Upper dorsal	— 12
Mid dorsal — 5th to 9th seg.	— 20
Lower dorsal — below 9th	— 7

Thus it is obvious that this complication is predominantly a hazard of dorsal spinal tuberculosis. It is rare in disease of other segments of the spine. The following factors very likely influence this incidence (a) relative immobility of the dorsal spine making compensation for a deformity difficult, (b) the relatively extensive body destruction met with in dorsal disease. This may be largely due to spread from body to body caused by the paravertebral abscess, and (c) the mediastinal space tending to limit the collection in the paravertebral area without adequate drainage and thus giving rise to tension inside the abscess cavity.

Of these 41 cases, only one was the result of tuberculous disease of the laminae. The rest were cases of vertebral body disease. The case of laminar disease involved the 2nd and 3rd dorsal vertebrae. It was the only case of tuberculous disease of the laminae met with in our total series of 315 patients.

Clinical features :

Our experience confirms the usual variability of these features. It varies in onset and in 5 patients, 3 children and 2 adults, was the first symptom noticed by the patient and his relations. In these cases, the patient had no knowledge of disease of the spine although when investigated, the disease was found to be of more than 3 or 4 months' standing. The paraplegia varies in degree being sometimes complete below a certain level, sometimes only partial and

sometimes asymmetrical on the two sides. No single type of paraplegia could be classed as characteristic of this condition. The course and progress of the condition is also variable. Where the paraplegia starts early in the course of the spinal disease, the progress is usually rapid and is accelerated by lack of treatment but treatment cannot altogether prevent its occurrence. In 3 patients of this series, the paraplegia started and progressed while the patient was under strict conservative treatment. Muscular weakness, incoordination and spasticity usually appear first and progress to a paraplegia in extension, with increasingly frequent attacks of general flexor spasm. These spasms can be extremely painful and exhausting to the patient and may seriously interfere with the patient's sleep and general nourishment and render conservative care almost impossible. As the paralysis deepens, it may become a paraplegia in flexion. In the worst cases of all, the paralysis may become almost completely flaccid. Although motor changes are often the first to appear and predominate the clinical picture, they are sometimes preceeded by changes in sensation. In distribution, the sensory changes also are variable ; generally the extremities of the limbs are affected first and most completely. Interference with the sense of position and passive movements is often an early feature and the last to disappear. There is no constant rule as to the order of the loss of the various types of sensation. Disturbance of sphincter control is often a late feature. Occasionally it is an early sign. Retention of urine and faeces occur at first but later and in the most severe cases, sphincter control is lost with incontinence of urine and faeces. Once sensation and visceral reflexes are lost, the patient is threatened with bedsores and ascending urinary infection.

Inspite of the extreme variability of the clinical course and progress of this condition, 3 distinct clinical types can be recognized — types that conform to the description of Butler and Seddon (1935).

Type A — Paraplegia with early active disease arising within 6 to 18 months of the onset of the tuberculous focus in the spine. This is the commonest type and explains 39 of our cases. Of these 38 were cases of vertebral body disease and one of laminar disease. Broadly speaking this type of paraplegia persists and diminishes in direct relationship to the tuberculous process in the spine. These cases can be further subdivided into —

(a) Slow onset type passing gradually but progressively to complete loss of power — 35 cases.

(b) Sudden onset type or sudden exacerbation of an initially mild paraplegia leading to complete loss of power — 4 cases.

Although this type of paraplegia broadly corresponds to the progress of the spinal disease, there is no exact parallelism between the two conditions and we have met with cases where the vertebral disease begins to improve while the paraplegia continues to persist and threaten the life or the limbs of the patient.

Type B — Late onset paraplegia appearing years after the attack when the lesion appears clinically and radiologically either under control or healed. We have come across only two cases of this type and thus its incidence in our series is much lower than other published series. One case came over a year ago but failed to turn up for further treatment. The other case is comparatively fresh and is still under observation and treatment and thus we have very little experience about the course and outcome and also ultimate management of this type of paraplegia.

Type C — Transient paraplegia — Some disturbance of cord function as shown by weakness or absence of the abdominal reflexes or by an increase in the tendon reflexes in the legs, occurs in about 50% of children with cervical or thoracic caries at some stage in their illness, but cannot be classed as true paraplegia. It is always incomplete and disappears rapidly on the institution of correct therapy. Such cases have not been included in this review.

Pathology :

The well recognized variability of the onset, progress and end results of this condition suggests that more than one single factor must be at play in bringing about this complication of caries spine. The disease in the spine is often more extensive than the x-ray and clinical features suggest. All the soft parts on the front and sides of the spine and even the superficial tissues of the back show oedema and congestion. The tissues are vascular and bleed very freely at operation. The degree of vascularity varies with the stage of the spinal lesion — active spreading, stationary or in the healing stage. A paravertebral abscess is not an invariable feature of paraplegia although it is present in the vast majority of the cases. In 1 case of vertebral body disease on whom we operated, no fluid abscess cavity was met with. There was extensive tuberculous granulation tissue both inside and outside the spinal canal, around the vertebral body and also in the mediastinal space. There was also soft tuberculous bony debris inside the spinal canal. In the case of laminar disease, nearly four inches of the spinal theca was surrounded almost on all sides with tuberculous granulation tissue. There was no fluid abscess. In all the other cases operated upon, active tension paravertebral abscesses were met with. On opening into the spinal canal, other causes of pressure on the cord were met with — (a) sequestrated portions of bone and intervertebral fibro-cartilage, (b) small isolated intraspinal abscess which has no free communication with the paravertebral abscess outside.

From the appearances seen at the operation, which is limited ot type A cases, we are satisfied that 3 factors are at play in causing this type of paraplegia —

(i) The spread of active tuberculous granulation tissue which always remains extradural but lies in intimate contact with the duramater. There is no evidence of a true tuberculous inflammation of the dura. On stripping the granulation tissue the dura looks normal in appearance and texture. The cord lying in the centre of an area of intense tissue reaction and separated from it only by the duramater, undoubtedly shares both in the oedema and the vascular changes. Butler has shown from post-mortem material that the most striking change in the involved part of the cord is seen in the white matter and consists of numerous clear round areas particularly in the distribution of the crossed and uncrossed pyramidal tract areas. This factor is present in almost every case and explains the onset of paraplegia in the absence of truly mechanical causes of compression.

(ii) In many cases, there is a mechanical factor also present. This mechanical pressure can be caused by fluid pus under tension inside the bony spinal canal, by pressure of accumulated tuberculous debris inside the canal which cannot escape outside, by pressure from sequestrated pieces of bone or intervertebral fibro cartilage. All these factors we have come across in our cases.

It is possible that tension in a paravertebral abscess which communicates with the intraspinal space may also cause mechanical pressure. This may be aggravated by respiratory movements which causes the abscess cavity to produce a water-hammer effect on the spinal cord. That such a factor plays a part is suggested by the following observations —

(a) A clinically palpable cold abscess is rarely associated with paraplegia.

(b) In two of our cases, the paraplegia rapidly improved as soon as the paravertebral abscess tracked to form a superficial palpable abscess.

(c) Simple drainage of the paravertebral abscess in two cases of paraplegia who had extensive bilateral pulmonary lesions and was not therefore fit for major surgery, produced a partial but dramatic improvement in the paraplegia. After this partial recovery, the paraplegia remained stationary.

We have not met with any cases of true narrowing of the bony canal consequent on the subluxation or dislocation of one vertebral segment over another. As a matter of fact we have been impressed by the way the posterior segments of the vertebrae adjust themselves and maintain the normal potency of the canal. This we have seen in cases who have come up for posterior spinal fusion in the presence of very severe degrees of deformity.

Thus it is our experience that pressure is always a factor in the production and maintenance of a paralysis which shows any signs of persistence and the pressure is almost always anterior to the theca and always extradural.

(iii) Vascular thrombosis in the segmental blood supply of the cord may occasionally be the cause of irrecoverable paraplegia.

Treatment :

In order to assess the trend in treatment, it is necessary to review the past, assess the problem facing us to-day and then consider the manner in which it is being faced and can be improved in the future. It appears that in the past there has been a tendency to regard this paraplegia as a relatively benign disorder. Jones and Lovett (1923) refer to " the rare cases when recovery does not take place " and add that " in children, paralysis is often the precursor of recovery (from the vertebral disease) as it ensures rest in a manner that nothing else can." Hugh Owen Thomas used to say when paralysis occurred in the case of an unruly child, " now we shall have a chance to get him well ". No special treatment was indicated for the paralysis which was regarded as an episode in the evolution of that particular patient's spinal disease.

This attitude continued to dominate this complication of spinal caries till the researches of Sorrel and Madame Sorrel — De'jerine (1932) and also of Butler and Seddon (1935) threw fresh light on the etiology and mechanism of paraplegia in Pott's disease and drew pointed attention to the presence of mechanical factors in the causation and continuation of paraplegia. Since then interest in paraplegia has quickened and the almost fatalistic attitude has given place to a more rational assessment of the problem of treatment in individual cases. From the therapeutic standpoint, the problem is to know what degree and duration of compression the cord can tolerate before serious and permanent changes occur and also to appreciate to what extent mechanical compression as distinct from the effect of extradural tuberculous infiltration is causing interference with cord function. Lack of response or further deterioration in the paraplegia inspite of efficient conservative care is an unfavourable sign and calls for a reconsideration of the therapeutic regime. The conservative treatment, once paraplegia is progressive, should not be persued too long. Conservative care includes attempts at slow, gentle hyperextension of the affected portion of the spine and also skull traction, in disease of the uppermost part of the dorsal spine. Other features that call for reassessment of the case are —

(i) Complete loss of voluntary power persisting for more than 3 months.

(ii) Presence of complete sensory loss.

(iii) Sphincteric involvement especially when there is loss of sphincter control.

(iv) Urinary infection, irregular fever and bedsores, which are progressive.

(v) Paraplegia in extension which is passing on to paraplegia in flexion especially when there are severe painful flexor spasms which render conservative care almost impossible.

(vi) Flaccid paralysis.

If any of these signs are present or appear while under treatment, it is to be assumed that early release of pressure from the cord is indicated. If this is delayed too

long, then complete recovery of cord function is very unlikely, even if the disease in the bony spine gradually heals. The methods that can be used and has been tried to decompress the theca are :—

(i) Aspiration — this is a very unsatisfactory method and technically risky and difficult. We tried it in one case when the patient had a very large upper dorsal paravertebral abscess which was causing respiratory distress. She had complete motor, sensory and visceral paraplegia and also had urinary infection, bedsore, severe anaemia, albuminuria and bilateral active disease in the lung. She was quite unsuitable for any major surgery. The abscess was aspirated on 3 occasions at weekly intervals and each time about 100 c.c. of tuberculous pus was removed but this had no effect on the paraplegia.

(ii) Costo-transversectomy — this as we have already indicated, is not a reliable operation. It has its use in the bad risk case and it is reported, that occasionally it may lead to a brilliant success.

(iii) Laminectomy — In our opinion laminectomy is never justified in Pott's paraplegia except in laminar disease or in the rare "spinal tumour syndrome" type of Pott's paraplegia. In vertebral body disease, the cause of pressure always lies anteriorly, the integrity of the bony spine depend on the spinous processes and the laminae, there is a tendency for the theca to herniate backwards following the operation whereby pressure is caused at the upper and lower limits of the laminectomy and a future fusion operation, when necessary, is rendered grotesquely difficult. We have used this method with the single example of laminar disease of our series and the result was most satisfying.

(iv) Rachotomy — This operation was designed by Capener of England. It is sound in principle as an attempt is made to attack the actual cause of the compression anterior to the theca. However, the operation has never gained any popularity and has not been given a fair trial.

(v) Antero-lateral decompression — this operation was first reported by Alexander and Dott (1946). They reported 18 operations with 12 complete recoveries, 15 survivors and 3 operative deaths but the recoveries were sufficiently satisfactory to encourage other workers to try out the method. The operation is performed with the patient lying either on an anterior plaster shell or in the semi-prone condition on the side. The position naturally makes anaesthesia difficult and maintenance of full oxygenation a tricky business. It needs the services of an efficient and experienced anaesthetist who understands the needs and difficulties of the surgeon. A slightly curved para-vertebral incision is made centred on the gibbus and the superficial muscles of the back are detached from the region of the spinous processes. The flat muscles of the back are stripped from the posterior 3 or 4 inches of two or three ribs in the region of the gibbus and also from the posterior aspects of the transverse processes and laminae. During this part of the operation bleeding can be profuse and difficult to stop and can easily frighten a beginner but the operation must continue with the use of pressure pads over the areas of bleeding, retained by self-retaining retractors. Now the posterior part of the ribs, the transverse processes and the outer ends of the laminae are exposed. At this stage the costo-transverse ligaments are snipped with a pair of sharp fine scissors and the posterior ends of the ribs are exposed subperiostially. The ribs are cut 3 to 4" from the transverse processes and pulled out from their vertebral articulations by gentle twisting movements. This leaves the transverse processes bare which are nibbled away upto their bases. The removal of the ribs usually leads straight into the paravertebral abscess when present, which drains away at this stage and can be further emptied by suction. Because of the severely altered anatomy of the diseased parts, the only certain guides to the theca are the exposed intercostal nerves. One or two of these are now traced backwards to the corresponding inter-vertebral foramen and the bone round the foramen is gradually nibbled away in all directions, a procedure which removes the pedicles, lateral part of the laminae and antero-lateral portions of the bodies of the vertebrae. This permits entrance into the spinal canal and the theca can be visualised. The surface of the exposed theca is cleared of all compressing structures, if necessary, by wide removal of bone from the back of the diseased bodies. It is at this stage that mechanical causes of compression of the theca are revealed. The operation is continued until the theca lies freely inside the spinal canal with no suggestion of pressure and the colour of the vessels on its surface looks normal. Occasionally one is rewarded with the sudden restoration of pulsation of the cord — an incident that occurred in 3 of our cases. However, its demonstration is not essential to the recovery of function in the cord. As far as possible, tuberculous granulation tissue surrounding the theca and the vertebral bodies should be removed. Every effort should be made to prevent damage to the dura mater as such an accident may lead to tuberculous meningitis.

The operation is hazardous and sometimes frightening and calls for the greatest anaesthetic skill to maintain the patient fully oxygenated. It is performed on ill-patients and can give rise to fairly severe shock. All methods of combating surgical shock should be ready before embarking on this operation. The post-operative management requires arduous care from the nursing staff. The operation, temporarily at least, weakens the mechanical stability of the spine. The patient therefore must be maintained in complete immobilization on the back in a suitable plaster shell for 8 to 10 weeks following the operation and should not be turned even for the removal of stitches. Any movement at this stage can be really hazardous. This happened in one of our cases. In upper dorsal disease, skull traction must be maintained in the post-operative phase.

Inspite of all these drawbacks, the operation can be most satisfying both to the surgeon as well as to the patient when performed carefully and adequately in the properly selected case. No experience can be more exciting than to find completely paralysed patients able to move their feet and know that they were doing so within 12 hours of the operation. We had such experience in 2 of our cases. In other cases the recovery is more slow but the speed and quality of the recovery is much better than that obtained after years of fixation.

So far we have not been fair to the operation. We have tended to perform it in the very severe and desperate cases or even in hopeless ones and only when we felt helpless with conservative care and that we are encouraged to attempt it and to persist with it inspite of such adverse conditions, speaks volumes in favour of the procedure. We are of opinion that when paraplegia due to tuberculous disease of the vertebral body calls for an operation, it is the procedure of choice. The exact indication will depend on the experience of the surgeon. Gradually as the operation gets standardized, more early cases will be submitted to surgery in order to reduce the suffering of the patient. In our opinion, the operation has very little effect on the actual tuberculous process in the bone. The treatment for the bony lesion must continue on accepted lines inspite of the operation. It only removes one hazard from the patient's life and thus gives him a better chance to fight the bony focus of disease. It is also our opinion that anti-tuberculous drugs has made such surgery safe and possible and in their absence, the risks and dangers would be severe. It is our opinion that in dorsal diseases with para-vertebral abscess, these drugs should be withheld initially unless the diagnosis is made at a very early stage and early arrest of the disease appear possible. When late destruction and avascularity has already set in, the drugs may be withheld to avoid development of drug resistance, so that in case paraplegia appeared at a later date, suitable surgery can be performed under the protective umbrella of these drugs. In our opinion there is room for early drainage of para-vertebral mediastinal abscess under protection of anti-

tuberculous drugs. This may help to reduce the incidence of paraplegia in dorsal disease and may also help to limit the extent of bone involved.

Some of these cases will ultimately require spinal fusion. So far we have done so in 3 of our cases. It appears to us that some form of dependable fusion operation performed at the time of the antero-lateral decompression may be the future line of growth in this type of surgery.

An analysis of the 41 cases followed up so far, with complete details of the cases submitted to antero-lateral decompression brings out strikingly the value of this operation in suitable cases.

Of the 14 cases of paraplegia below the age of 12 years, the case of atlanto-axial disease died suddenly, while still under treatment, from a pathological dislocation of the atlas. Twelve of the rest recovered under prolonge d conservative care, the time required for recovery from paraplegia varying from 6 months to 12 months. In two cases of extensive upper dorsal disease, the paraplegia relapsed — in one case 12 months and in the other case 18 months after the cessation of conservative care. The relapse in each case was cured under conservative treatment. These were cases of very extensive disease. It is our present opinion that early drainage of the paravertebral abscess may have reduced the extent of destruction in these cases and thus cut short materially the progress of the spinal lesion. Operation was performed on only one case of upper dorsal disease. This patient's condition progressively deteriorated inspite of conservative care and finally he developed complete flaccid paralysis, urinary infection and bedsores. This was our first case of antero-lateral decompression and we performed the operation as a desperate measure. The child recovered, though slowly. Thus in our experience antero-lateral decompression is called for rather infrequently in children below the age of 12 years.

Of the 27 cases of paraplegia in patients over the age of 12 years, 11 were operated upon. 8 patients had antero-lateral decompression, one had laminectomy and 2 patients had costo-transversectomy. Of the remaining 16 patients, 6 died a painful lingering death — the result of complications consequent on paraplegia. Three of them came at a stage when they were totally unfit for any surgical procedures although they had been under medical care previously. The other 3 patients refused to be operated upon. This gives a mortality of 37.5%. Three patients, two of whom had costo-transversectomy and the other repeated aspirations, are still living but their condition is such that they are very unlikely to survive. Thus in our opinion Pott's paraplegia in adults, once it persists and progresses, is a very serious complication and carries a very high mortality. It is our opinion that the paraplegia quickly undermines the general health of the patient and lowers the resistence of the patient to tuberculous infection. The majority of the fatal cases had secondary tuberculous infection of other organs especially involvement of the lungs. On the other hand, the results of antero-lateral decompression have been most gratifying. We have had no operative deaths so far.

Conclusion

Experience with the treatment of Pott's paraplegia has convinced us that surgical attack on the paraplegia as well as certain other aspects of Pott's disease holds out better hope for these patients than the time-honoured regime of persistant conservative care. Surgery performed judiciously will save the life of a fair proportion of otherwise fatal cases, will reduce the morbidity in others and may help to cut short the period of treatment. It will certainly reduce the sufferings of the patient materially. Its scope and limitations has not been established fully as yet, but the results so far obtained fully justify extensive trial with these methods. Such surgery was hazardous before the days of anti-tuberculosis drugs but the protection given

Pott's Paraplegia

Serial No.	NAME	Durat site of le:	Result of operation	End result
1.	Sheo Nandan 11 yrs. M	16 m D3	Slow recovery	Complete recovery from paraplegia. Recovery from spinal disease.
2.	Ram Bahadur 18 yrs. M	10 m D.	Return of power on day of operation	Complete recovery from paraplegia.
3	Mahendra Prasad 43 yrs. M	13 m D.	Rapid recovery of power & relief of spasm	Complete recovery. Beiug considered for spinal fusion.
4	Dhanesh Singh 43 yrs. M	15 m D.	Rapid recovery	Complete recovery. Spinal fusion done.
5	Basudeo Modi 31 yrs. M	2 y D.	Slow recovery	Complete; spinal fusion performed.
6.	Mrs. D. Bagchi 44 yrs. F	15 n D.	Slow recovery	Complete. Still in plaster.
7	Vijay Pratap Singh, 19 yrs. M	18 m D.	Rapid improvement in power & sensation	Suddenly relapsed during change of plaster 3 weeks after operation.
8	Khakia 24 yrs. F	3 y D.. I	Rapid recovery	Complete. Under treatment.
9.	Sabitri Debi 17 yrs. F	12 m D2 D3	Rapid recovery	Complete. Under treatment.
10.	Lukhu Gope 35 yrs. M	12 m D. I	Slow recovery	Only sensations returned.
11.	Bundela Ray	13 m D. I	Slow recovery	Complete recovery of sensation. Partial recovery of power. Spasms persist.

THE CAPUT SUCCEDANEUM

*by PONNU ISAIAH and JACOB CHANDY, Vellore.**

Very little has been done to elucidate the anatomy, physiology and pathology of the caput succedaneum. It is generally believed and given in textbooks that it is the swelling in the area encircled by the girdle of contact and that it is produced due to the pressure of the maternal passages against the scalp, interfering with the free venous return in the vessels of the scalp, the oedematous infiltration occurring over the area which is free of pressure; thus the position varies according to the presentation.

But the work done in new born babies who have died due to various causes have shown conclusively that it really is a mechanism for adjustment of intracranial tension at birth during the passage of the head through the rigid bony canal.

Fig. 1.

X-ray of the skull after injecting radio-opaque material through one internal jugular vein of a baby delivered by Caesarean section and died soon after delivery.

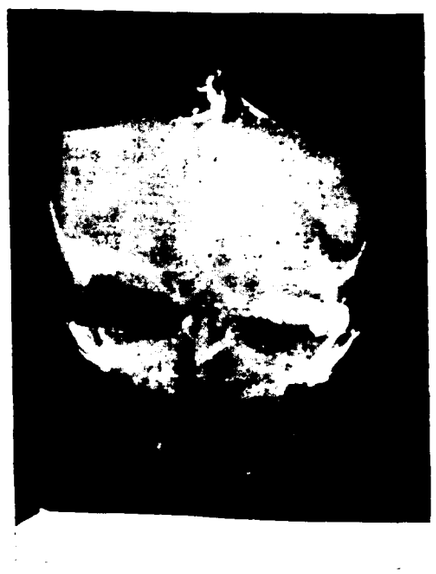

Fig. 2.

X-ray of skull of a baby who died immediately after a difficult breech delivery.

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Materials of investigation :

New born babies dead either during labour or within 24 hours after birth; in this are included various presentations. Some cases where the baby had not undergone any stress as the child was removed by caesarean : one case with caput at birth and the baby died after three days.

Technique:

The internal jugular vein on one side is exposed at the neck and a 18 G Cannula is introduced. The venous system of the head was then washed out with water using at least 50 cc. and then a thin aqueous suspension of Barium Sulphate is injected through the Cannula; at least 50 cc. of the suspension is used. X-ray of the skull A.P. & Lateral views was taken

Fig. 3.

X-ray of a normally delivered baby who died 5 hours after delivery. A.P. view.

in stereo positions. Some of the cases where the X-ray showed abnormalities of the venous sinuses were dissected and studied.

Observations :

(1) Emissary veins are not open ordinarily if the head has not undergone stress enough to produce caput.

(2) In the case of the Caesarean baby which did not have any stress at all, the emissary veins were not open.

(3) In one case of breech presentation where there was delay in the extraction of the head there was an attempt for one branch of the emissary vein to open up. But where the head was easily extracted the emissary veins remain closed.

(4) When a caput was present, the emissary vein of the region of the caput opened up widely and the fully dilated venous pattern at the caput was filled with the radio-opaque material.

Fig. 4.

X-ray of a normally delivered baby who died 5 hours after delivery. Lateral view.

(5) The emissary veins in the regions other than at the caput did not open.

(6) When the child lived long enough for the disappearance of the caput, the emissary vein closed and did not open up when the radio-opaque material was injected.

Conclusion :

The caput succedaneum is not just oedema of the soft parts due to continued pressure at that region during labour, but it is formed as a result of the adjustment of intracranial tension during the passage through the bony canal by opening up of the emissary vein at the area where there is no pressure, that being the opened cervical canal. The venous blood in the sinuses escape into the scalp with every contraction, thereby reducing the intracranial content when the capacity of the head is reduced due to increased pressure acting on the head by the contraction of the uterus. The venous dilatation at the caput leads to venous stasis and oedema results due to transudation. Thus the caput succedaneum is a safety mechanism for the adjustment of intracranial pressure during birth.

The emissary veins are those that pass through the foramina in the wall of the cranial cavity connecting the superior

sagittal, the sigmoid, and the cavernous sinuses with the veins outside the skull.

The superior sagittal sinus is connected through the unpaired foramen caecum which lies between the ethmoidal and frontal bones to the veins of the nose and the paired parietal foramina to the veins of the scalp. The former is present in the child but seldom persists in the adult. This is one of the veins that opens up in cases of face presentation.

The sigmoid sinus communicates via the mastoid foramen with the posterior auricular vein and via the condylar foramen with the sub-occipital veins.

Ophthalmic veins have no valves and so blood in them pass in and out of the cavernous sinus to the infra-orbital, facial and other veins; this being the other vein that opens up in cases of face presentation.

The cavernous sinus is also connected via the foramen ovale and foramen vasalii (phen present) to the ptyerygoid plexes of veins and via the carotid canal to the deep veins of the neck. Similarly, the basilar and occipital sinuses communicate with the spinal veins.

Even in adult life the blood in them flow in either direction and the same function of safety valve mechanism is carried on. It is not unusual to find the emissary veins and foramina enlarged in cases of gradually increased intracranial tension. This leads to enlarged scalp veins seen clearly in cases of Hydrocephalus in children. Similar procedure of injecting radio-opaque material was carried on in a few cases which died with increased intracranial pressure. This showed few of the emissary veins opened out.

It is believed that in cases of sudden increase of intracranial pressure as in cases of injury or haemorrhage, a certain amount of C.S.F. is displaced into the spinal canal. Whether simultaneously blood is pushed out through the emissary veins remains to be studied.

Roughly about 15 to 20% of still births and Neonatal deaths are due to intracranial haemorrhage. The haemorrhage is mostly due to rupture of venous sinuses which are within the layers of the dura and are fixed to the skull. Even in spontaneous difficult labours intracranial haemorrhage occurs due to ruptured venous sinus.. The possibility is more when the head is compressed antero-posteriorly in cases of deep transverse arrest and in instrumental deliveries.

The causes of intracranial catastrophes may be:—

(1) Tonic contraction of the uterus where the safety-valve mechanism is not able to function properly.

(2) Faulty safety-valve mechanism due to abnormalities in the sinuses or emissary veins. The venous sinuses show a great deal of deviation from the normal. Different varieties are described with illustrations in reference no. 2.

Some of our cases which showed lack of filling of the superior sagittal sinuses or abnormal pattern were dissected out. Fig. 5 shows a case in which the superior sagittal sinus was closed where normally it joins the transverse sinus and Fig. 6 shows bifid sagittal sinus at its posterior end where each joins the transverse sinus separately.

The abnormalities of the venous sinuses and the damages occurring to the brain

Fig. 5.
The superior sagittal sinus closed at its posterior end.

Fig. 6.
Bifid posterior end of the superior sagittal sinus.

during labour produce various immediate and remote consequences.

Neilson and Courville, Penfield and Keith, and Penfield and Humphreys have emphasized the importance of birth injury and asphysia in epilepsy. Earl Baldwin and Penfield in their recent paper on Temporal lobe seizure state the following: By Electroencephalography a large proportion of epileptics were found to suffer from temporal lobe seizures. This type of seizure may begin or continue in adult life although the cause is often to be found in the mechanism of birth compression. Out of an analysis of 157 cases of temporal lobe seizures, 100 of these (63%) showed evidence of anoxia at birth by pathological study which was variable in extent and appearance. They have called this pathological entity as incisural sclerosis. It is concluded that this sclerosis is produced by temporary herniation of the temporal lobe through the incisura of the tentorium at the time of birth and that the lesion ripens so slowly that the temporal lobe seizure manifests itself many years after birth.

Experimentally they have shown that compression of the heads of still born babies does produce temporal lobe herniation and that unless the head is frozen the evidence of this herniation disappears and that may be the reason why it is missed during routine autopsy examination.

Their experiment supports the previously described causes of intracranial catastrophes. In a normally contracting uterus the safety-valve mechanism comes into play and there is less chance for herniation. But in a tonically contracted uterus as in the experiment where continued pressure was applied to the head, the safety-valve mechanism fails and there is more chance of temporal lobe herniation. What is called by the above mentioned authors as incisural sclerosis is called by Keji Sano and Nathan Malamid as Sclerosis of the cornu Ammonis and it is said that Spielmeyer referred to it first in 1927. Thus the recognition of this late morbidity gives the obstetrician additional responsibility, and it is he with whom the prevention of these latent maladies is left. Although experiments have not been done in human, a great deal of work has been done on young animals. Fazekas, Alexandar and Himwich and Glass et al observed higher resistance to anoxia in new born rats, rabbits, dogs and guinea pigs than at any subsequent time. If the arrest of circulation was not longer than six minutes, there was complete recovery of function but interruption of blood flow for more than eight minutes or longer produced permanent damage to the brain. It is a well known fact that administration of Carbohydrates increases the resistance to anoxia. Snyder observed that sodium Pentobarbital increased the resistance of the respiratory centre to asphyxia in new born rabbits.

Other drugs as Thiourea; Thiouracil; Vasopressine; Sulphanilamide and cooling are also found, experimentally, to increase the resistance to anoxia.

As the use of sodium pentobarbital and glucose is practicable they can be given to all mothers coming for delivery so that it is carried to the foetus through the placental circulation. It is worth the trial when we realize the number of cases attending the neurological clinic whose malady is attributable to their birth.

Summary

The older concept of the formation of caput succedaneum is given.

Details of investigations carried out in still born and babies who died in the neonatal period with the observation and conclusion is discussed.

Brief anatomy of the emissary veins is mentioned.

Immediate and late results of intracranial catastrophes as observed by various neurosurgeons is given. Probable treatment for preventing this is outlined.

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CONNECTIVE TISSUE REACTION TO IMPLANTED POLYETHYLENE CELLOPHANE AND ITS POSSIBLE USE IN THE TREATMENT OF HERNIA

(An experimental study)

by P. K. SEN and S. N. KOTHARE, Bombay.

The tissue response to implanted cellophane first attracted attention in 1939 when Irvine Page used it to induce hypertension in dogs by wrapping kidneys with it to produce peri-renal fibrosis. Since then this substance has been used in experimental animals in various regions with the object of producing various changes, but in the main to produce occlusive changes around vessels. The use of cellophane in the treatment of aneurysms is now widely known (Abbott, Croot, De Takats, Harrison) and the extent of its benefit and limitation is being appreciated.

The use of cellophane induced fibrosis has also been employed in the surgical treatment of patent ductus arteriosus.

Ingraham and Clagett have, on the other hand, used polythene sheets for repairing dural and pleural defects, respectively, and have shown a total absence of tissue response to implanted cellophane. McKeever has similarly used polythene sheets as an interposition membrane after synovectomy and Wheeldon has used the material for reconstructing tendon sheaths. The effects of various cellophane plastics used as tissue implants in surgery therefore appear to be

Fig. 1.

Photomicrograph showing a strip of cellophane surrounded by young collagenous tissue with sparse lymphocytic infiltration at the end of six weeks. (H & E x 100).

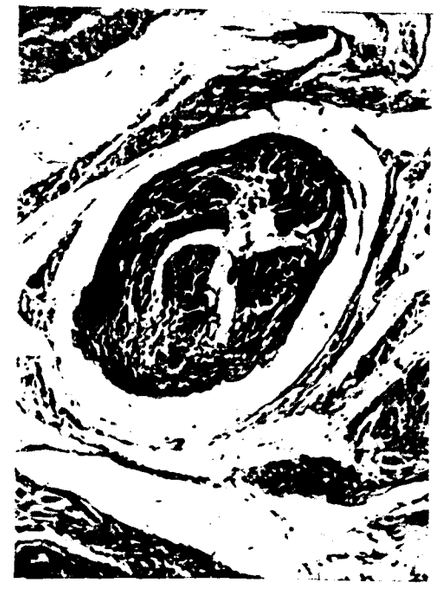

Fig. 2.

Photomicrograph showing a silk suture surrounded by fibrous tissue infiltrated with chronic inflammatory cells. Thin strands of fibrous tissue infiltrating the surrounding tissue can be made out. (H & E x 100).

protean and confusing. Reactions from absolutely none (Ingraham) to severe and progressive fibrosis (Poppe) have been described and therefore it is very necessary to learn the exact chemical structure and the presence of any impurities (Yeager and Cowley) before using a plastic implant for any particular purpose, reactive or protective. Between the two extremes of cellophane reaction there are plastics which excite only localised and non-infiltrating reactions.

Yeager and Cowley in their study of cellophane as fibrous tissue stimulant suggested that it may be possibly useful in the treatment of large herniae. It is only with this facet of the problem that we have concerned ourselves.

Method :

Sixteen adult male rabbits were chosen for this experimental study, mainly because the inguinal canal of the animal contains a patent funicular process and therefore mimics the indirect inguinal hernial sac in man.

Under ether anaesthesia and aseptic conditions the left inguinal canal was opened, the funicular process (sac) ligated, and strips of polyethylene red cellophane (Dobecknum & Co., Sleveland, Ohio) used to appose the free edges of the inguinal ligament and the conjoint tendon after the manner of a Bassini operation. On the right side a similar procedure was carried out, using black silk as suture material to serve as a control. This was done in ten animals. In six the cellophane was implanted in the anterior abdominal wall on one side and a silk suture control on the other.

The animals were sacrificed at periodic intervals ranging from one week to six months. The entire inguinal area was

Fig. 3.
Photomicrograph showing the two cellophane strips with condensed bands of fibrous tissue on either side 14 weeks after implantation. (Mallory's Aniline Blue x 100).

Fig. 4.
Photomicrograph showing four strips of cellophane surrounded by bands of fibrous tissue, the amount of fibrous tissue seems proportionate to the amount of implanted cellophane. (H & E x 100).

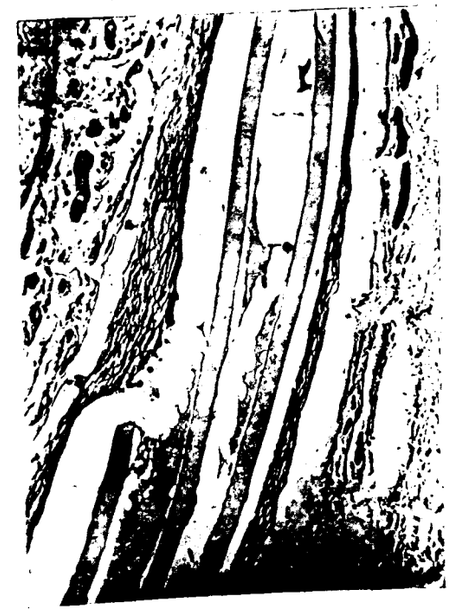

Fig. 5.
Photomicrograph showing cellophane strips after 20 weeks interval ; the fibrous tissue has ceased to increase in amount in spite of such a long interval. (H & E x 100).

removed, fixed, blocked in paraffin and serial sections made. They were studied using a standard haemotoxylin-eosin stain as well as special stains for fibrous tissue.

Results :

During the earlier phases (one week) an inflammatory reaction developed, which was much milder around the implanted cellophane compared to the reaction around the black silk suture, but fibroblastic proliferation commenced even at this stage, and was definitely more marked around the cellophane strip.

At six weeks inflammatory exudate was much less, and around cellophane a strip of localised young collagenous tissue was seen, while around the silk suture fibrosis was diffuse and infiltrating. Inflammatory reaction seems to have subsided completely at twelve weeks and fibrosis was denser. Studies at sixteen, twenty and twenty-seven weeks showed increasing fibrosis, around cellophane with no inflammatory reaction while mild inflammatory reaction was present around the implanted silk. By about twenty-one weeks the increasing fibrosis around cellophane seemed to have become stable. This fibrosis, was strictly localised as a band around the cellophane. Increased fibrosis was also seen around silk sutures, but it was less and of a diffuse nature, having an infiltrating quality. No bursa like space were seen around the cellophane as described by Wilson et al.

Conclusions :

It appears that with the materials used localised and controlled reactive fibrosis could be produced around the cellophane, while this fibrosis began early, even during the first week after implantation it was not well established until the third month. The fibrosis stopped increasing before the sixth month and was never invasive and diffuse as it was around silk which seemed to produce much more inflammation and less fibrosis than cellophane.

Comment :

The use of cellophane in hernia surgery certainly offers some interesting possibilities as it has been well said that controlled sepsis (and thus fibrosis) is the hernia surgeon's best friend. However, from this study it would seem that effective amount of fibrous tissue formation does not occur till about the twelfth week and most statistical studies on the recurrence of inguinal hernias after operation place their highest incidence within that period (Rains). That would suggest that the use of cellophane strips alone in hernia repair may not be surgically wise. Strips of cellophane however, could be wrapped around the routine cotton or silk sutures used in hernia repair with advantage. Such strips would promote dense and localised fibrosis in six month's time and be as effective as fascial sutures, while in the earlier postoperative period the strength of the silk or cotton suture would serve to maintain the repair.

A similar idea has been used successfully in the ligation operation of patent ductus arteriosus (Harper et al) and though it is now nearly outmoded it still has a place in the treatment of difficult and recurrent patent ducts. Perhaps in the hernia problem too, cellophane may be well utilised in certain difficult situations. We have not as yet tried the clinical use of cellophane in operation for hernia for want of proper follow-up in our hospital type of patients. But we believe that there is a definite case for the judicious use of polyethylene cellophane in hernia repair.

Summary

Studies have been carried out in 16 rabbits on the tissue reaction of cellophane implanted in the inguinal canal and anterior abdominal wall of rabbits. Progressive and localised fibrous around the cellophane has been noticed without much inflammation. Controls of implanted silk sutures showed less fibrosis and much infiltration and inflammation. The use of cellophane strips is suggested in hernia repair as *reinforcement* around the silk or linen sutures which are used routinely, to promote dense localised fibrosis, mimicking the effect of fascial sutures.

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PLASMA CELL MASTITIS

(Report of a case with simultaneous involvement of both breasts)

by J. B. SHRIVASTAV and K. D. SHARMA, Nagpur.

Ewing (1925) was the first to suggest the name "Plasma cell mastitis", to this condition. His associate, Cutler working in the laboratory of the Memorial Hospital found only 10 cases on record. A detailed study of the clinical data and pathologic findings, established plasma cell mastitis as a specific clinical and pathological entity and was described as such by Cheatle and Cutler (1931). Cases have been described from time to time but the number reported in the literature has not been large. Cutler (1949) has reported 10 cases, Manglik and Mathur (1952) have reported one case. Gordon F. Cassie (1953) who reviewed the literature in great details, has reported one case of "Plasma Cell Mastitis". We are reporting this case because of the rarity of this condition. It is also of interest because of some unusual features showing a departure from the classical description given.

CASE REPORT

A Hindu, female, aged 30 years was admitted on 19-6-1950 in Irwin Hospital, Amravati (M.P.) for chronic mastitis of the right breast of five months' duration. The complaints started soon after a delivery, with pain in both the breasts. The child died 5 days after delivery. Five months after delivery a lump could be felt in the right breast. The lump was hard, tender and of the size of a cricket ball. It was aspirated on 24-6-1950 and was found to contain thick blood tinged fluid with white flecks which were thought to be pus. Microscopic examination of the aspirated material showed plenty of R.B.C., a few pus cells and staphylococci.

As the patient was running a temperature, the breast was incised on 5-7-1950 but no pus came out. Biopsy pieces were sent from both the

Fig. 1.

Note the periductal distribution of cells. The duct epithelium does not show any evidence of proliferation. (L.P.)

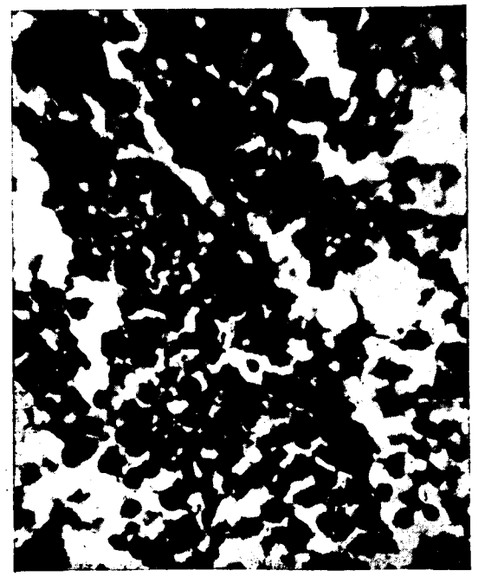

Fig. 2.

Showing sheet of plasma cell. (H.P.)

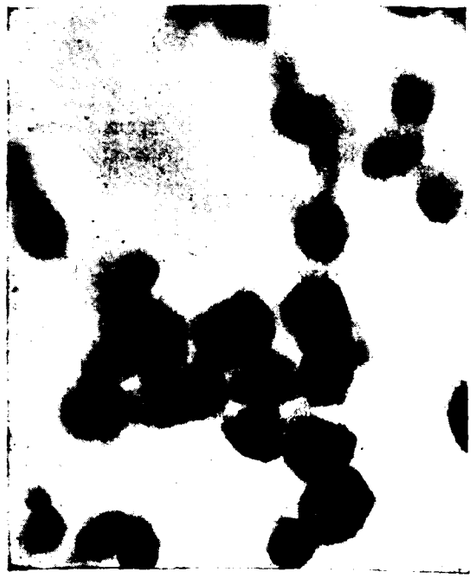

Fig. 3.

Plasma cells with eccentric nuclii and cart-wheel arrangement of chromatin. (Oil Immersion)

breasts and we reported on the pieces as "Plasma Cell Mastitis". The wound in the right breast did not show signs of healing and the breast was amputated. Unfortunately the amputated breast could not be sent to us for histological examination.

Gross appearance of biopsy pieces: Biopsy pieces were very small and were sent in two bottles labelled right and left breast. Paraffin blocks were prepared from each piece.

Microscopic appearance: Biopsy from both the breasts show similar features. The tissue is composed almost exclusively of sheets of plasma cells surrounding the ductules (Fig. 1). These cells have eccentric nuclei with peripheral arrangements of chromatin giving a cart-wheel appearance (Fig. 2 & 3). Other type of cells are lymphocytes. Some of the ductules are dilated and are lined by single layers of epithelium. There is no proliferation of the lining epithelium, thus not justifying the name — "Mastitis Obliterans". The lumina of the ductules are patent and do not show any inspissated milk. Crystals of fatty acids are not seen in the periductal tissue.

Discussion

Plasma cell mastitis is a disease of parous woman. There is general agreement on its basic etiology that the retained secretions of the duct which have extravasated in the periductal tissue excite plasma cell reaction. The extravasation of retained secretion can occur either as a result of changes in the chemical composition of the secretion (Cutler 1949) or due to focal infection of the duct wall, liberating retained products in the periductal tissue to produce inflammatory reaction. Another feature which is in favour of retained secretions as the eitology of plasma cell mastitis, is the usual history of difficult nursing in these women (Cromar & Dockerty, 1941). Some cases give history of trauma over the breast which can be directly responsible for extravasation of retained contents as well as for production of traumatic fat necrosis in the breast.

Usually it is said that plasma cell mastitis starts in a non-lactating phase of the breast of a parous woman. The case of plasma cell mastitis occurring in a lactating breast has been reported by Manglik & Mathur (1952). This occurred in a woman aged 30 years who had her last child one and a half years back and had not stopped feeding the child till the onset of her illness. The present case occurred in a woman who had borne children, but the symptoms appeared immediately after delivery. The sudden onset of symptoms could have been an aggravation of the dormant stage of the disease, which was primarily initiated by the retained milk products from a previous pregnancy. The symptoms became worse as the child died five days after delivery and the breast could not be emptied of its secretions. The consequent over-distension of the ducts lead to extravasation of the contents and thus produced plasma cell reaction in periductal tissue. Probably focal infection of the duct wall was also responsible for the extravasation, as the aspirated contents showed some pathogenic organisms like staphylococci.

Bilateral involvement of both breasts in plasma cell mastitis is very rare. The only

case recorded is of Cutler (1943). This occurred in a woman aged 37 years. The involvement of the second breast was about 7 years after the first one. In the case presented symptoms started in both the breasts simultaneously. In the right breast inflammation showed signs of localization. Biopsy taken at this stage from both the breasts showed typical plasma cell mastitis. At the end of five months, the woman was left with a lump of the size of a cricket ball in the right breast. In the left breast symptoms subsided without a residuum. Such a case of plasma cell mastitis has been observed by Geschickter (1945), where the area of induration and redness appeared suddenly in the region of the nipple and disappeared within several days without a residuum.

The case is interesting for two definite reasons —

(1) Bilateral and simultaneous involvement of both the breasts in a lactating phase;

(2) The inflammation subsided in one breast without a residuum while in the second breast a residual lump was left.

Summary

1. Bilateral involvement of breast.

2. Disease involved both the breasts simultaneously.

3. The symptoms started with lactation.

4. Inflammatory changes subsided in one breast without leaving a residuum.

We wish to express our thanks to the Superintendent, Irwin Hospital, Amravati, for the clinical data and to Shri Joshirao for the microphotographs and Shri S. Shah for technical help.

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MEDIASTINAL TERATOMA

*by O. P. MITAL, R. H. BETTS, and T. THOMAS, Vellore.**

Mediastinal tumours present an interesting diagnostic problem. There is a wide variety of neoplasms occurring in the mediastinum and an accurate preoperative diagnosis in every case is not possible, but a thorough consideration of all clinical and radiological investigations does go a long way in reaching the near probabilities. A consciousness of the fact that mediastinal tumours are not really so rare as were considered about a decade ago, and that they call for surgical interference to which quite a large number of them are amenable is essential to recognize them early and to treat them. An important group of these, the teratoma, is illustrated by the following cases, the first of which was discovered when totally unsuspected.

The first case recorded and described was in 1823 by J. A. Gordon. Since then 251 cases have been reported in the literature up to 1951.

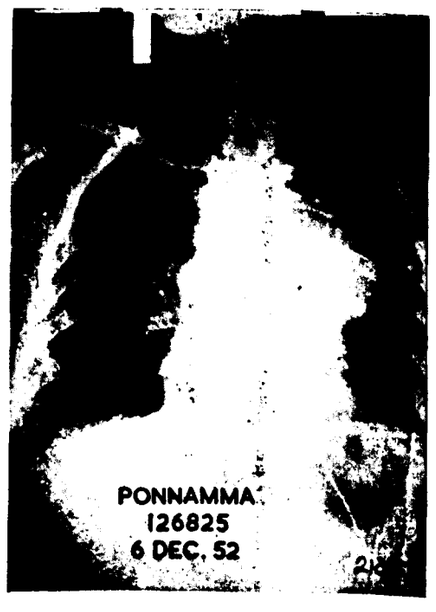

Fig. 1.
Case 1. Roentgenogram on admission.

CASE I.

P. This 45-year-old housewife entered the Hospital on December 6, 1952. She had a history of pain in the left arm extending from the neck to the elbow for six months. This was followed in a few days by pain and tenderness in the left upper chest. At about this time she developed cough and expectoration, the sputum being purulent and measuring about 2 oz. in 24 hours. She has also had blood-stained sputum now and then. First X-Ray chest was taken on August 13, 1952 and it showed an opacity in the left upper chest extending from the mediastinum. Re-X-ray after one month, showed an increase in the size of the opacity. It was after this that the case was referred to us for treatment.

Examination revealed clubbing of the fingers and toes. There was decreased fremitus and dullness to percussion over the left upper chest anteriorly, specially over the first, second and third

Fig. 2.
Case 1. Left lateral projection. Lesion in anterior mediastinum.

*From the Dept. of Thoracic Surgery, Christian Medical College Hospital, Vellore (S. India).

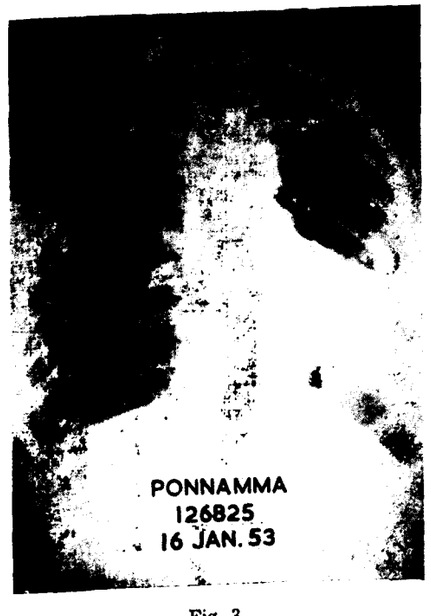

Fig. 3.
Case 1. After removal of cyst.

ribs, and included intercostal spaces, which were also tender on palpation. The breath sounds were impaired over this area and rales and rhonchi were present. The right lung was clear.

Laboratory investigation revealed a high E.S.R. The Papanicolaou report on sputum was negative. Roentgenograms revealed a rounded opacity extending into the left chest from the anterior mediastinum.

Bronchoscopy was essentially negative and Bronchography revealed a very slight displacement of the trachea posteriorly and that the bronchi of the anterior segment of the left upper lobe did not fill well.

On December 23, 1952, left thoracotomy was performed (T.T.). A large cyst situated in the anterior mediastinum and extending into the anterior segment of the upper lobe was found. It had extended medially so that quite a large segment was immediately under the arch of the aorta. During separation from the apex the cyst punctured in one place, thus confirming the diagnosis of dermoid cyst. Examination of the fluid which escaped revealed Cholesterol content of 125 mgm.%. After freeing up the cyst from the lung it was found that quite a good part was arising from the pericardium and could not be dissected off it. It was also involving the phrenic nerve. The pericardium was opened into and the involved part situated postero-laterally was excised and the cyst removed.

Pathological examination revealed a specimen weighing 700 grams, an oval mass measuring 7.5 cm. x 6 cm. x 4 cm. which had been ruptured at one point. On section, the wall showed irregular nodules some of which showed fine black and white hair. The wall showed yellow areas and sebaceous material in places.

Microscopic inspection revealed that the cyst was lined by stratified squamous epithelium. The solid areas on the wall contained ciliated pseudo-stratified epithelium. There were other glands lined by goblet cells, glandular tissue resembling pancreas, lymphoid tissue, cartilage and sebaceous glands.

Diagnosed as a case of Teratoma.

The post-operative course was disturbed by atelectasis of the left lung on the second post-operative day. Bronchoscopic aspiration was done and frequent intra-tracheal suction resorted to. Due to delayed expansion of the lung, there were a few pockets of fluid which, however, cleared up after repeated aspiration and patient was discharged on January 16, 1953, 24 days after operation.

CASE II.

M.S.L. This 34-year-old Engineer was admitted to the Christian Medical College Hospital, Vellore, on February 11, 1953. He stated that he had been suffering from recurrent attacks of asthma since 1949. In September 1949, he had coryza which persisted and culminated in an attack of asthma. Was all right with symptomatic treatment in about one week. Since then, he had about two attacks of cold a year lasting approximately a week and ending up in what he called asthma. The one in March, 1952 was rather severe. Roentgenogram taken in July 1952 revealed a suspected dermoid cyst. The only other complaint was an occasional pain in the left upper anterior chest. Examination of the chest revealed a few rhonchi over the left chest.

On February 24, 1953 left thoracotomy was performed (R.H.B.). A tumour about 3 cm. x 6 cm. was found in the anterior mediastinum sitting just above and anterior to the hilum. It was connected to the lung by a few filmy adhesions. It was removed.

Pathological examination revealed a specimen consisting of a large, oval, encapsulated, firm, cystic mass, with a yellowish colour. On section the wall was hard in places. It was found to be filled with greasy yellowish thick material. The sebaceous material contained white hair. At one place in the wall were found three teeth firmly attached to it. On microscopic examination, the cyst wall showed fibrous tissue, fat, collections of

Fig. 4.

Case 2. Roentgenogram on admission showing a dense rounded opacity just above the left hilum.

Fig. 5.

Case 2. Left lateral projection showing the growth situated in the anterior mediastinum and containing teeth.

Fig. 6.

Case 2. After thoracotomy and removal of teratoma.

Fig. 7.

Case 2. Photograph of the specimen of teratoma removed at operation. The teeth are seen attached to the wall.

foreign body giant cells and groups of lymphocytes. The cyst was lined by stratified squamous epithelium with keratinization. Underneath this were seen numerous sebaceous glands.

A diagnosis of Cystic Teratoma was made.

Patient made an uneventful recovery and was discharged 17 days following operation.

CASE III.

V.R. This 18-year-old boy who looked no more than one of 12 years of age was admitted to the Christian Medical College Hospital, Vellore, on March 14, 1953. He stated that he had cough and expectoration with pain in the left chest anteriorly for the last six years. The pain was intermittent. He complained that his growth had been stunted and that he was not able to carry on with his studies at school. He had also noticed that he got breathless, more than normal, on running and was very easily fatigued. First x-ray was taken on April 12, 1952 and a simple tumour was diagnosed. Re-X-rayed on September 12, 1952 and advised to go over to us for treatment; but patient could not come to us before March 14, 1953. Examination revealed dullness to percussion on left side from second to fourth intercostal space anteriorly and in the axilla. There was some tenderness in the second and third intercostal spaces. Breath sounds were audible only at the apex on the back. The right side was normal. There were rales present all over the left chest. Patient was raising about 100 c.c. of purulent non-odorous sputum per day.

Roentgenograms brought by the patient with him showed a sharply defined rounded opacity occupying most of the left chest and containing teeth in it.

Laboratory investigations revealed a very low Haemoglobin (7 gms.), and a moderate leucocytosis 12,000/cmm. The sputum contained Gram positive cocci in chains and diplococci and Gram negative bacilli.

Bronchoscopy revealed a stenosis of the lower lobe bronchus to almost half the normal size. The upper lobe orifice could just hardly be visualized.

On March 30, 1953 left thoracotomy was performed (T.T.). A very large, hard, rounded mass occupying almost all of the left chest was encountered. The anterior segment of the upper lobe was incorporated in the wall of the tumour. The lower lobe was atelectatic and small, lying between the tumour and the mediastinum. It was planned to remove the tumour and resect a part or all of

Fig. 8.

Case 3. Roentgenogram on admission. Shows a big mass in the left chest, with multiple irregular areas of bony density.

Fig. 9.

Case 3. Left lateral projection again shows the growth occupying the whole chest in the anterio-posterior plane and containing teeth in its upper and anterior portion.

Fig. 10.

Case 3. After left pneumonectomy and removal of teratoma.

the upper lobe. But during the procedure, the main pulmonary artery as well as the inferior pulmonary trunk were compromised. In view of this, as also due to the fact that the lower lobe had been atelectatic for the last few years, Left Pneumonectomy was completed. The tumour mass was also very firmly bound down to the pericardium. After considerable and patient dissection a plane of cleavage could be established and the mass dissected free off the pericardium.

Pathological examination revealed a specimen consisting of left lung with a large rounded tumour connected to the upper lobe measuring 9.5 cms. x 10 cms. x 6 cms. On section, the mass was bony hard in places and had to be sawn through. The section showed a variegated appearance with fat, cartilage and bone, and cystic spaces filled with sebaceous material and black hair. The lung was compressed and formed a narrow rim around the tumour. On microscopic inspection, various bits taken from the mass showed cartilage with calcification and ossification, fat, fibrous tissue, smooth muscle, thickened vessels, stratified squamous epithelium, mucous glands, hair follicles, numerous sebaceous glands, well-formed bone trabeculae with marrow, lymphoid tissue, brain tissue, nerves, choroid plexus and cholesterol spaces in laminated tissue.

Fig. 11.

Photograph of specimen of teratoma removed at operation with the lung adherent to it along most of its periphery. Multiple irregular areas of bony deposits and teeth are seen scattered in it.

Examination of the lung revealed that the upper lobe showed oedema fluid, with patchy atelectasis. There were collections of macrophages and eosinophils. The lower lobe bronchus showed desquammation of the lining epithelium and a few chronic inflammatory cells.

Diagnosed as a case of Teratoma Mediastinum.

The post-operative course was uneventful and the patient was discharged 21 days following operation.

Etiology

This has been a field for such speculation and the various hypotheses are an indication of the lack of our knowledge in this direction.

According to Nicholson, " they are pathological manifestations of physiological growth." Ewing believes that extragenital teratoma may be derived from " Isolated totipotent blastomeres or from early budding of the blastoderm or multipotent material of distinct regional stamp." Teratoma of the mediastinum has been considered by some to originate in the third and fourth branchial clefts. It almost always occupies the region of the thymus. Some think that they are a result of faulty embryological development of the thymus since this is the only branchiogenic structure to descend into the mediastinum, while Harrington and others have reported cases in regions other than in the mediastinum.

Lloyd Rusby has concluded after a long discussion that the problem remains far from solved.

Pathology

Teratomata are some of the most complex tumours. They truly depict the immeasurable potentialities of living cells.

Ewing in his classical work has classified dermoids into two groups : The simple dermoids and the teratoid tumours. Willis, however, is inclined to regard them as having a common aetiology. He emphasizes that even in the simple dermoid cysts, searching examinations will reveal tissues of mesodermal and endodermal origin in addition to those of epidermal origin.

Incidence

The majority are said to occur in young adults. These tumours are, it would appear, the result of a faulty embryogenesis and would show up at puberty or afterwards due to some environmental factors. They are usually situated in the anterior mediastinum. The majority are in the mediastinum, projecting into one side or the other of the thoracic cavity.

Clinical and Radiological features

The clinical manifestations depend upon chiefly the size and location of the tumour. Recent reports from the West have proved that an appreciable number of these growths have been discovered accidentally on mass radiographic survey of large numbers of persons.

Once symptoms have become manifest, cough and pain are the two commonest initial symptoms. Even a small tumour in close proximity to the trachea or a bronchus may produce dyspnoea, wheezing and symptoms of pulmonary infection. Except when a mediastinal growth is of a very large size the respiratory symptoms are usually due to pressure on the trachea or a bronchus rather than compression of the paramediastinal pulmonary tissue. Haemoptysis may occur with both benign and malignant growths. Although these tumours may markedly displace vascular structures, the flexibility of the vessels usually prevents any obstructive symptoms. Occasionally, a cyst may rupture into the lung and becomes secondarily infected. The expectoration of sebaceous material, cholesterol granules or hair is diagnostic of a dermoid cyst or teratoma.

Physical signs except in cases of very large growths are rarely of much practical value. Dullness on percussion is usually found with some alteration in the character of the respiratory murmur. There may be some bulging of the chest and other signs due to pressure if the growth is large. Signs attendant on some complications like infection, bronchiectasis, rupture of the cyst into a bronchus or pleural cavity, atelectasis of part or all the lung may be elicited.

The Radiological appearances are more or less typical. A sharply defined, round or oval shadow is seen in the anterior mediastinum projecting into one of the pleural cavities. Although the commonest situation of these tumours is in the anterior mediastinum, it is by no means invariable. The growth may be of a homogeneous density more or less but it is not infrequent to find irregular areas of increased density or even teeth seen clearly inside the homogeneous shadow. These appearances are strongly in favour of a teratoma. In our cases 2 & 3, these were clearly seen, and made us suspect the diagnosis of teratoma.

Inquiry regarding earlier roentgenograms should always be made. Comparison with previous films may aid in determining the rate of growth and the duration of the mass. The lateral roentgenogram is far more informative than the P.A. one, but naturally both must be studied together. Since a wide variety of tumours occur in the mediastinum and although the various investigations, clinical, pathological and radiological, give us a fair idea of the growth being one of the several possibilities, an accurate diagnosis before operation may not be possible. This is emphatically true when one has to decide whether it is benign or malignant.

Treatment

The only treatment that will have any lasting benefit for teratoma of the mediastinum is surgical intervention. The assumption that a sharply defined well encapsulated mass is benign is neither wise nor safe. Usually, therefore, all mediastinal growths should be removed unless it is proved that they are only one manifestation of a generalized process in the body. Further the teratomata are potentially malignant and such a change may occur even after a long time. Even if they remain benign, they have a progressive course and are liable to produce complications at any time.

Other Tumours of the Anterior Mediastinum

The most common tumours in this region are the dermoid cysts and teratomata, the lympho-sarcomata, Hodgkin's disease, thymic tumours and pleuro-pericardial cysts. Bronchogenic cysts may also occur in the anterior mediastinum, but they are more commonly seen in the posterior mediastinum. Near the diaphragm and in the cardiophrenic angle the pleuro-pericardial cyst is much the commoner tumour, but it has to be differentiated from diaphragmatic hernia, pericardial fat pads and liver herniation. Parasitic cysts such as hydatid cysts simulate radiologically almost any mediastinal tumour; skin tests help in differential diagnosis. The basal metabolic rate determination will help in differentiating a substernal thyroid. Both Thymoma and Lymphosarcoma are radiosensitive. Saccular aneurysm of the ascending aorta or arch of the aorta may be distinguished by angiocardiography with other findings.

Summary

Three cases of mediastinal teratoma treated surgically in the Department of Thoracic Surgery of the Christian Medical College Hospital, Vellore (South India) from December, 1952 through the year 1953 are reported. Of these, the first was not diagnosed before operation. All the three cases had the growth in the Left Anterior Mediastinum. A rather protracted history with typical radiological findings is to be noted. A consciousness of the fact that these tumours are not extremely rare and that they demand surgical intervention is essential for early diagnosis and treatment. A few important clinical and radiological features are described. A very brief review of some other anterior mediastinal tumours concludes the report.

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SOME OBSERVATIONS ON THE MANAGEMENT OF URETEROCOLIC ANASTOMOSIS

by K. N. UDUPA, Mandi.

In a brief case report[1] sometime back the author had discussed about the method that he had adopted in doing the Nesbit's *Uretero-Colic Anastomosis.* Since then he had gained additional experience of performing this procedure in 12 more cases and this paper is mainly based on his observations on the management of these cases together with a review of the recent literature on the subject.

As was known to all Coffey did the pioneer work to standardise this procedure as a safe and simple one in the properly indicated patients. However some hazards inherent with the procedure were still met with in a certain percentage of cases. In order to overcome these difficulties Nesbit[2] and Cordinnier[3] devised newer methods of direct mucosa to mucosa and end to side *anastomosis.* With the adoption of these methods together with the use of antibiotics it was expected that there would not be any difficulties in the management of these patients. *Although this might be* true with regard to the immediate post-operative complications, it has now been felt that all the newer advancements had not materially changed the occurrence of *late complications in a fair percentage of* patients. Therefore, recently, the whole question of this operation was under the re-examination of many investigators interested in this field, and it is hoped that all the existing difficulties will be overcome in the near future. Before going to discuss about these complications, I will discuss briefly the method I have adopted in performing this procedure.

Technique: I have selected only one method of anastomosis namely the Nesbit's method, which I believe is the operation of choice and which gave very satisfactory results in my patients. Further I am also in agreement with the opinion expressed by Jacobs and Sterling[4] "because it is impossible to recommend any single mode of operation as an optimum one and because even slight deviation from minute technique may be followed by impaired results, those whose opportunities of performing ureterocolic anastomosis are limited will best serve their patients by perfecting a technique in one method of their choice." Hence I adopted only Nesbit's method in all my cases.

The detailed technique of this operation has already been described by me in my previous paper[1]. It consists of the following: For three days before the operation Sulphasuxdine three tablets 5 times a day or Terramycin two capsules three times a day are given. On the day before and also on the day of operation a simple Enema followed by colon wash is also given. The operation is usually done under spinal or general anaesthesia and the abdomen is opened by a right lower paramedian incision. After putting the patient in a steep

Fig. 1.

Shows the incision on the posterior peritoneum at the brim of the pelvis. This figure also shows the technique of isolation and cutting of the ureters.

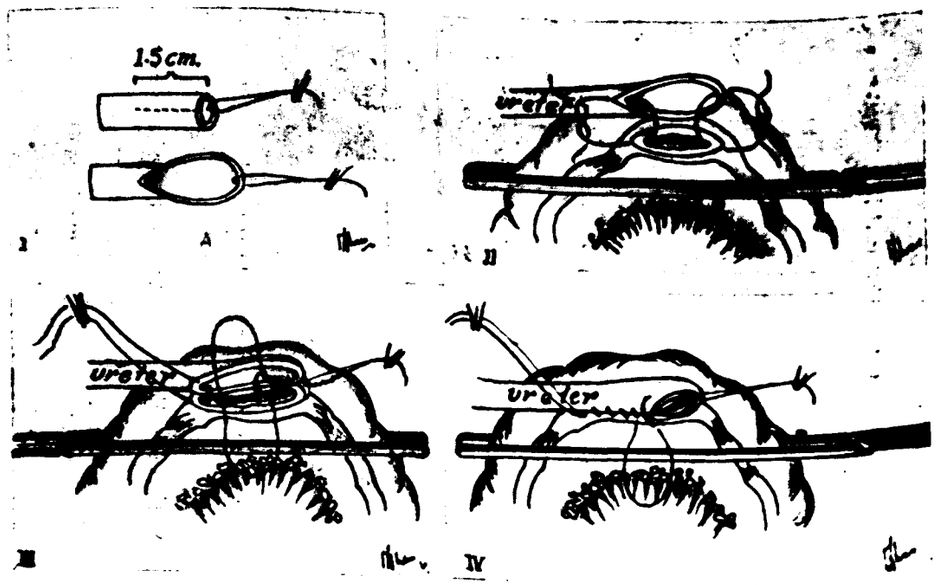

Fig. 2.
Shows the method of anastomosis of the ureter into the Sigmoid colon as per Nesbit's technique

Trendelenburg's position, the posterior peritonium on the right side is incised and the ureter is isolated and cut as close to the bladder as possible (Fig. 1). While isolating the ureter care should be taken not to mobilise too much of the ureter, that it may not become devoid of its blood supply.

Then the ureter is made to spatulate by making a longitudinal incision on its cut end for a distance of about 1.5 c.m (Fig. 2). The end of the ureter is then anastomosed with the sigmoid colon by a single running suture of 000 catgut taking all the coats of the ureter to all the coats of the colon (Fig. 2). This is further strengthened by a few loose interrupted sutures from the serous coat of the colon to the ureter all around the anastomosis. The lateral margin of the incised peritonium is then loosely stitched to the colon covering the site of anastomosis (Fig. 3). The same procedure is carried out on the opposite side and the abdomen is closed as usual. A flatus tube is kept in the rectum for the following 7 days to drain urine and Sulphasuxidine or Terramycin is given as before for 7 days in the post-operative period. Plenty of fluid, a low residue diet and alkaline diuretic mixture are given throughout the post-operative period. This was the method we followed in all of our cases with minor modifications as individual cases needed and except a few complications as given in Table 1, we did not find much difficulty in the managements of these cases.

Indications for performing this operation :

(1) Congenital anomalies like ectopia vesica or Severe degree of hypospidia or Epispidia with incontinence of urine. We had only three such cases in our series.

(2) All traumatic lesions causing injuries to the male or female urethra with incontinence of urine or injuries to the genitourinary system of females, like vesicovaginal fistula or any other traumatic condition causing constant dribbling of urine. We had in all 8 such cases in our series.

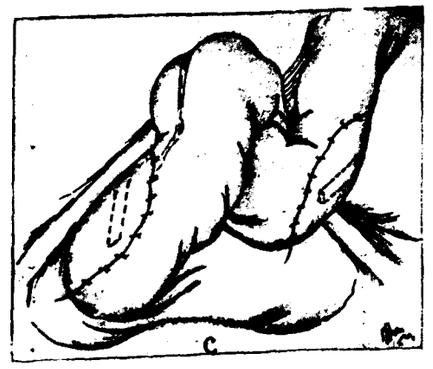

Fig. 3.
Shows the method by which the colon is attached to the brim of the pelvis by suturing the cut end of the posterior peritoneum around the anastomotic area.

(3) Intractable vesical tuberculosis causing severe degree of discomfort. In this series we had only one such case.

(4) A long standing non-tubercular contracture of the bladder. We had no such case.

(5) Any malignant diseases of the bladder necessitating total cystectomy. We have not so far come across any such cases.

These are the usual indications for doing ureterocolic anastomosis. In addition to this we had also undertaken this operation in a female patient who had complete destruction of her urethra due to Lymphogranuloma inguinale followed by incontinence of urine.

Inspite of all these indications discussed above one should not take this operation lightly and perform it on patients who could be easily treated otherwise or the condition itself is not dangerous to life. When it is decided to do this procedure, it should be noted that it can be successfully performed only at a proper time. For example in congenital anomalies it is better to perform this operation when the children become 4 or 5 years old since before this period they tolerate this operation very poorly. Similarly in traumatic lesions like vesicovaginal fistula, with superimposed pelvic infections and inflammations, one should not undertake this procedure unless one is absolutely sure that there exists no inflammatory process in the pelvic region. Otherwise there is every chance that the ureterocolic anastomosis may not unite properly and there may arise severe complications. After having a bad experience in one of his own cases Jacobs[4] had rightly emphasized that the transplantation of ureters should be delayed until complete subsidence of pelvic infections. We too had such a sad experience in our own case, and a brief history of this patient will amplify these points.

CASE REPORT

Mrs. P. aged 35 years was admitted to the hospital with the complaint of constant dribbling of urine per vaginum since the last four months. In all she had delivered six children and in her last delivery about four months back she had a very protracted labour for three days. After the delivery she started dribbling urine per vaginum and also she ran a very high temperature for a few days, accompanied with pain in the lower abdomen and foul smelling discharge from the vagina. She could overcome most of these troubles by medical treatment within a month or so, but her main complaint, namely, the dribling of urine still persisted.

On examination, almost the whole of the anterior vaginal wall was absent and the bladder mucosa could be seen clearly protruding into the vagina. Since the defect was very big with a diameter of about more than two inches, we did not feel justified in attempting local closure. Hence an ureterocolic anastomosis was decided upon and was performed by the usual method. The right ureter with a normal size and calibre was readily isolated and anastomosed to the sigmoid colon. But, for a long time we failed to locate the left ureter, because the retroperitoneal tissues on that side were still involved in a chromic inflammatory process. However, after a prolonged search and dissection we located it surrounded by much inflammatory tissue. The wall of the ureter itself had chronic inflammatory changes and therefore it had lost its elasticity. In the beginning we hesitated to anastomose such an unhealthy ureter, but ultimately decided to go ahead with the anastomosis which would naturally avoid a second operation. While doing the anastomosis, because of chronic inflammation in the ureteric wall, it used to tear very easily. However, with some difficulty, we could complete a fairly satisfactory anastomosis on this side also.

As expected she started to have a very stormy post operative course. She passed very little urine per rectum, and inspite of all our efforts the urine quantity did not increase. She also developed signs and symptoms of acute peritonitis indicating that there was leakage of urine through the anastomosis on the left side. Unfortunately before we could decide to reopen the abdomen and put a drainage tube she died on the 6th post operative day. Permission to do a post mortem could not be obtained.

From the above it is clear that whenever there is any recent history of pelvic inflammation, one should never attempt doing ureterocolic anastomosis in view of the difficulty in locating the ureter especially on the left side and also in view of the unsatisfactory result of such an anastomosis. Therefore, this operation should be performed not only when there is an absolute indication for it, but also at the proper time.

Complications :

It is well known that Ureterocolic anastomosis is followed by certain complications in a fair number of cases in the later stages. However, the majority of cases do overcome such initial complications and lead their life quite normally for a long time. Out of 13 cases, we did not notice any major complications in 10, and as far as our information goes they have been leading normal lives till now from two months to three years after the operation. In the remaining 3, one died of peritonitis in the immediate post operative period, giving a mortality rate of 7.6% in our series. In Jacob's series[4] collected from all the British Urologists the immediate mortality rate was 21%. On the other hand Cordonnier and Lage[3] reported a series of 56 cases without any immediate mortality. From this it is clear that the mortality rate varies from series to series depending upon the original disease for which it has been performed, the age of the patient and also on the experience of the Surgeon in performing a particular type of operation. Two of our patients developed repeated attacks of Pyelonephritis. One of them had very mild attack, and was overcome by antibiotic and other treatment ; the other one is still getting repeated attacks inspite of all possible treatments. Details of this case will be given later.

Immediate complications :

Immediate complications are almost the same as those seen following any major abdominal operation. Paralytic ileus which occurs due to excessive purgation, too much bowel preparation by ways of Enema or colon washes, and also due to prolonged exposure or rough handling of the bowels during this operation. All these should be avoided for prevention of ileus, and when it becomes very distressing injection of Carbochol or acetyle choline usually overcomes the trouble. We had two such cases and both of them recovered by the injection of Carbochol. Peritonitis is more commonly due to faecal contamination at the time of operation and also due to subsequent leakage at the site of anastomosis. However since the introduction of broad spectrum antibiotics like Terramycin and Aureomycin in the pre and post operative periods such incidences are rare. Leakage at the site of anastomosis may occur, if the technic of the anastomosis is not correctly followed, or if the ureter is unhealthy. It may also occur if the ureter is stripped too much from its blood supply which leads to necrosis of the ureter. All these causes could be prevented if proper precautions are taken at the time of the operation. We had only one patient who developed this complication and expired on the 6th post operative day as already discussed in detail previously. Urinary and Facecal fistulae are rare in the anastomosis cases, although it used to be the common complications following transplantation of ureters by Coffey's technique. The urinary infections in the immediate post operative period are rare. These are some of the common complications and they could be prevented if all possible precautions are taken from the very beginning.

Late complications :

It has been noticed commonly that not all the patients who had smooth post operative course in the immediate post operative period do well in their later life and a fair number of cases develop some late complications which have now been mainly attributed to electrolytic disturbance. Quite a

large number of investigations have recently been carried out by various investigators to find out about the nature and causes of these complications[6, 7, 8, 10]. Although there seems to be some unanimity over the nature of these disturbances there has been so far no agreement about the causes of these complications. A brief review of the literature will throw some light on the subject.

Ferris and Odel[6] reported findings in 141 patients who had undergone bilateral ureterosigmoidostomy in the Mayo Clinic. Out of these 79% of the cases had increased chlorides and 80% had lowered alkalies in the blood indicating that most of these patients were suffering from hyperchloraemic acidosis. In addition to these they also found slight raising of blood urea level in the majority of their patients, from their investigations they came to the conclusion that this type of acidosis occurred in their patients as a result of absorption of chlorides across the rectal mucosa from the urine stored in the rectum and sigmoid colon. They did not believe that such a disturbance could be caused by derangement of the kidneys.

Wilkinson[7] investigated this problem in six of his cases and concluded that due to accumulation of urine in the colon and also due to hurried emptying of the ileum, there is increased colonic secretion with consequent base loss and dehydration, a state of chronic water, potassium and bicarbonate loss with chloride and sodium retention.

On the other hand Berglin and Mathieson[8] made observations in 31 of their patients one month to three years after ureterosigmoidostomy and found varied electrolytic changes not only in different patients, but also in the same patients on different occasions. However, in all these cases hyperchloraemic acidosis was the constant finding. They attributed this disturbance to the increased colonic pressure transmitted to the pelvis of the kidneys through the ureter causing renal parenchymal damages particularly to the tubules. The increased intratubular pressure decreases the glomerular filtration rate which in its turn produces diminution of excretion of chloride and sodium. An infection of the kidneys will greatly aggravate these chemical imbalance. They did not believe that there is any substantive absorption of urinary salts across the colonic mucosa. Almost the same findings and explanations were given by Kekwick et al[9].

The most important investigation amongst all was carried out by Lapides[10] in 22 patients with ureterosigmoidostomy, and in six without any such operation. He reached the conclusion that reabsorption of urinary constituents did occur from the colon constantly following ureterocolic anastomosis and if the renal function was normal, the kidneys tried to excrete all the excess of salts found in the blood thereby keeping the body electrolytic balance at its normal level. But as soon as renal impairment occurred, as by an attack of Pyelonephritis, the signs and symptoms of hyperchloraemic acidosis also appeared because of salt retention. In all, 23% of his patients maintained normal health throughout whereas the remaining 77% gave histories of recurrent attacks of Pyelonephritis, associated with Pyelographic abnormalities like dilatation or non-visualisation of the pelvis of the kidneys and also with electrolytic imbalance. All these abnormalities appeared months or years after the operation. These observations were further supported from the results of a series of experiments in which he produced the condition artificially. In this he instilled urine by drip method into the colon of three patients with normal kidneys and into three others with impaired kidneys for a number of days ranging from 3 to 12. None of these six patients had any type of ureterocolic anastomosis. Results of this experiment showed that no electrolytic imbalance occurred in patients with normal kidneys while the patients with poor renal function showed electrolytic disturbance of the pattern seen after ureterocolic anastomosis. From the above observations and experiments there remained very little doubt that renal damage was the most important factor in the production of the electrolytic and

other disturbance found in the post operative period of ureterocolic anastomosis.

A number of later investigators also supported these views[4, 11]. Important amongst them were Jacobs and Sterling[4] who made the following observations in their collective series.

There was no doubt that the chloride of the urine was absorbed from the colon but this was not the only factor as was supposed by Ferris and Odel. Their study revealed that renal tubular damage was an important factor for the development of biochemical changes in these cases. Ascending infection and back pressure on the ureter might be the actual reason for the damage of the renal tubules.

There are thus two schools of thought regarding the causative factors for the development of delayed complications—one believed in the reabsorption theory and the other in the renal damage theory. We believe in the latter theory since our only patient who is still having this complication amply proves that renal damage is the main factor for the development of these complications. Case report of this patient will greatly clarify the problem not only with regard to the causative factors, but also regarding symptoms and treatment.

CASE REPORT

Mr. R. B. aged about 20 years was admitted to the Civil Hospital, Mandi, with the complaint of constant dribling of urine over the abdominal wall since birth. Excepting this he had no other trouble and was keeping fairly good health throughout. On examination an Ectopia vesica with a severe degree of Epispadia was found. An excretory Pyelography was done and both the kidneys were found normal.

An ureterocolic anastomosis was decided upon and was carried out by the usual Nesbit's method after a thorough preoperative preparation. He had a very smooth post operative course and he used to pass a large amount of urine per rectum every day. He gradually gained control over his anal spinctures and at the end of the second week he passed urine only 6 times in 24 hours. He was discharged from the Hospital at the end of the third week and he continued to remain well at his home. At the end of three weeks after the discharge from the Hospital he suddenly started to have acute pain in the left lumbar region followed by high rigor. At that time he also passed very small quantities of turbid urine per rectum. It was thought that he had an attack of Pyelonephritis and therefore he was put on a course of Terramycin, two capsules three times a day for a week. By this treatment the temperature and pain subsided within three days of starting the treatment. Since then unfortunately he started to have repeated attacks of Pyelonephritis almost every fortnight inspite of all the treatments and his condition became very precarious.

He was readmitted to the Hospital for a thorough investigation and treatment. Although we had no arrangements to carry out the detailed biochemical tests, we closely observed his clinical and other conditions during the attack and afterwards. Every time just before the attack he used to feel excessive weakness and fatigue, anorexia, nausea, some salty taste in the mouth and excessive thirst. By the appearance of all or some of these symptoms he could predict that an attack is imminent shortly. A day or two afterwards he used to pass turbid urine per rectum followed by high fever and pain in the loins. During this period, he also used to vomit frequently and to pass diarrhoeic stools several times. He used to feel very thirsty but could not drink water, because of vomiting. During this period we took the intravenous pyelographic pictures several times but every time there was non-visualisation of the pelvis of the kidney. From all these observations, we came to the conclusion that he was having repeated attacks of Pyelonephritis as a complication of ureterocolic anastomosis accompanied by all the typical symptoms of biochemical disturbances. Therefore, we gave the following treatment with fairly good success :—

(i) In order to overcome the renal infection streptomycin 1 gm. was given every day for the next two months.

(ii) Alkaline mixture containing Soda Bicarb and Pot. Citras was given every four hours. Similarly salt intake was also restricted to overcome hyperchloraemic acidosis.

(iii) To diminish the absorption of chlorides from the colonic mucosa a daily colon wash was given.

(iv) Further to decrease the chances of transmission of back pressure into the pelvis of the kidneys from the colon, a rectal tube was introduced and kept in the rectum constantly. This not only decreased the pressure in the colon and renal tubules, but also prevented the urine accumulating in the colon.

Under this treatment he completely overcame his troubles gradually and regained his lost health to a considerable extent. He was still continuing the treatment except the streptomycin till the writing of this paper and we believe he will have to continue this treatment for a long time before we could expect a permanent cure.

From this case report, it is clear that renal damage is the main factor for the development of complications and we are in complete agreement with the views expressed by Lepides[10] and Jacobs and Sterling[4].

Cause of renal damage :

Here we should also briefly discuss the possible causes for the development of renal complications so that we may be able to prevent them in our future patients. As discussed by Creevy[5] briefly there are four possible causes for the development of kidney complications :—

(a) Direct ascent of bacteria from the colon into the kidneys through the vessels of the implanted ureters. However such a possibility is comparatively rare.

(b) Secondly, as Berglin and Mathieson[8] emphasised, there is fairly good evidence to show that intracolonic pressure can force intestinal contents into the ureters and renal pelvis. Such a reflux of gas and possibly faeces into the renal pelvis can readily occur in the direct anastomosis and may give rise to pyelonephritis at any time.

(c) The third possible cause for renal damage is stenosis at the site of anastomosis. This was the common complication after the Coffey's indirect method of transplantation and could be eliminated by the present direct mucosa to mucosa anastomosis. Such a stricture at the site of anastomosis may give rise to urinary obstruction leading to hydronephrosis, pyonephrosis or pyelonephritis.

(d) Usually the urine that has been diverted into the sigmoid colon goes upto the caecum (observed Radiologically by Lapides[10]). From there a sufficient amount of salt and water of the urine is reabsorbed into the circulation which is again excreted by the kidneys. Such a vicious circle naturally increases the work load of the kidneys which if continued for a long time may fail completely. Probably this is one of the important factors and that is why some persons after this operation show early disturbances where as others live comfortably for a long time depending upon the maintenance of good kidney function.

Summarising, amongst all these four causes the first and the last namely the direct spread of infections and excessive renal load cannot be avoided with any type of operation. However, one should do everything for the prevention of regurgitation of the colon contents into the ureters and stricture formation. So far none of the methods discussed above guarantee prevention of these complications. Recently several methods have been described to overcome these troubles. Leadbetter[12] has modified the method of Nesbit, in which the ureter is anastomosed to the mucosa of the colon and then the ureter is passed through a submucous tunnel for a distance as was done in Coffey's method. Creevy and Reiser[11] have suggested that all these patients should have a terminal colostomy so that the distal loop of sigmoid colon with ureters will have no faecal contamination and therefore will not have any renal complication. Very recently Methieson[13] has described an entirely new method of ureterocolic anastomosis, in which the ureter is made to protrude like a nipple into the colon surrounded by colonic mucosa by which both reflux and stricture formation are prevented. All these various methods are very recent ones and one should await further reports before one could adopt them for general use.

From all these various discussions on the subject, it will be very clear that the problem is not as simple as it was once thought to be, and many questions are yet to be answered. Till then, one should attempt to do this operation only when no other procedure will cure the condition and once it has been decided upon, the best procedure that will cure the condition and that one can do with ease should be adopted as the procedure of choice, since so far none of the methods described are without drawbacks. Only after taking into consideration all these factors should one perform this operation if he is to get good results.

TABLE I

Giving detailed informations about the patients who underwent Nesbit's Ureterocolic Anastomosis.

S. No.	Name	Age	Sex	Disease	Post Operative complication		Result
					Immediate	Delayed	
1.	A. C.	45 years	Female	Vesicovaginal fistula (U.V.F.)	Nil	Nil	cured
2.	J. C.	50 ,,	do	Tuberculosis of the bladder	Nil	Nil	do
3.	K. M.	35 ,,	do	V. V. F.	Nil	Nil	do
4.	M. T.	32 ,,	do	V. V. F.	Nil	attack of Peylonephritis	do
5.	S. T.	4 ,,	do	Ectopia vasica	Nil	Nil	do
6.	P. K.	35 ,,	do	V. V. F.	Peritonitis	Nil	Died on the 6th post operative day
7.	B. T.	12 ,,	do	Ectopia Vasica	Nil	Nil	cured
8.	M. R.	22 ,,	do	V. V. F.	Nil	Nil	cured
9.	R. K.	20 ,,	Male	Ectopia Vasica	Nil	Repeated attacks of Peylonephritis	Under treatment
10.	S. K.	25 ,,	Female	V. V. F.	Paralytic ilius	Nil	cured
11.	R. M.	35 ,,	do	Stricture urethra with Incontenance of urine due to Leymphogranuloma inguinale	Nil	Nil	cured
12.	N. D.	30 ,,	do	V. V. F.	Paralytic ilius	Nil	cured
13.	D. R.	60 ,,	Male	Traumatic stricture with Incontenance of urine			

SUMMARY

1. Although ureterocolic anastomosis is a fairly safe procedure in properly indicated cases, it should not be taken lightly in view of late complications like Pyelonephritis and biochemical disturbances.

2. Recently a number of new procedures like Nesbit's or Cordinier's methods have been described after the original description of Coffey. But so far it has been found that none of them are without drawbacks.

3. By adopting the Nesbit's technique, the author had the experience of performing this operation in 13 patients with one post operative death due to peritonitis following leakage at the site of anastomosis.

4. The late complication of this operation namely, Pyelonephritis accompanied by biochemical disturbances was seen in only one patient of ours who is getting relief by the use of antibiotics alkalies, and the constant use of the rectal tube.

5. There seems to be some dispute over the causative factors for the development of late complications. But there is a fair amount of agreement that the occurrence of renal infection is the main cause for the development of this complication, the reabsorption of urinary salts being the secondary one.

6. In order to overcome these late complications quite recently some new procedures, and also some modifications of the old procedures have been tried. But still they have not yet been standardised. Till then one should perform this operation with caution.

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CASES & COMMENTS

PRIMARY LYMPHOSARCOMA OF APPENDIX

(A Case Report)

*by SANTOKH SINGH ANAND, MANMOHAN SINGH and SUKH DEV KHANNA, Amritsar.**

Lymphosarcoma — originating in the appendix is rare enough to warrant report of a single case.

CASE REPORT

A Police Constable, 39 years of age, was admitted to the V. J. Hospital, Amritsar on 1-11-1951 with the complaint of repeated attacks of colicky pain starting from the umbilical region and radiating to the right hypochondrium and epigastrium of eighteen days' duration. Each attack of pain came after every ten to fifteen minutes and lasted for two to three minutes. He also complained of gradually increasing distension of the abdomen. The patient had been passing two to three loose motions a day. Elsewhere before admission, he had been tapped and what was believed to be pus was drawn out by a trocar and canula passed in the usual site for tapping ascites. On the day before admission, he had developed hiccough also.

Physical Examination: Hypersthenic build, slight anaemia. No emaciation or cyanosis and no jaundice. No glands palpable in any part of the body. Tongue moist and clean. Pulse 88 per minute, temperature 98°F, Blood pressure 110/75 mm. Hg.

The abdomen of the patient was markedly distended with fulness in the flanks. Superficial veins were found prominent in the abdominal wall, which moved sluggishly with respiration. On percussion, there was dullness in the flanks and the hypochondrium. Stiffness, dullness and fluid thrill was positive. Spleen and liver were not palpable. On auscultation, marked peristaltic sounds were audible. Rectal examination revealed no abnormality.

Investigations: The blood count was as follows:— Total red blood cell count 3,600,000 per c.mm., total leucocyte count 9,030 per c.mm., differential:— polymorphonuclears 69%, lymphocytes 29%, monocytes 1% and eosinophils 1%, haemoglobin 12 gms.%.

The preoperative diagnosis of peritonitis was made and the patient was operated upon on 4-12-1951, under spinal anaesthesia. A midline suprapubic incision, revealed blood stained fluid in the peritoneal cavity. The omentum presented a remarkable appearance being markedly oedematous, granular and studded with hundreds of small nodules varying in size from a pin head to a small pea. The appendix was found to stand out turgid with a markedly thickened wall and its meso-appendix thickened and oedematous. Innumerable enlarged glands were seen in the substance of the mesentery specially near the mesenteric border of the intestines. Specimens of free fluid in the peritoneum and gland biopsy were taken from the mesentery and the abdomen was closed in layers. Six hundred ccs. of blood were given to the patient. The patient died the same evening.

Autopsy findings: Before handing over the body to the relatives, the abdominal organs were removed from the body. The whole of the greater omentum and the lesser one were found to be thickened, pinkish yellow in colour, friable and studded with numerous light yellow nodules varying in size from that of a millet seed to that of a berry. The whole of the small gut was covered with tiny plaques, greyish pink in colour, scattered all over the gut wall and more marked as one proceeded from the duodeno-jejunal flexure downwards. The mesentery was shortened, thickened and its folds contained radially arranged nodules varying in size from a pin head to an almond, all along the mesenteric border of the small gut. These swellings were also found in the proximal portion of the mesentery but were not so well marked there. Section of the nodules gave uniform resistence to the knife and exposed a pale homogeneous surface. The Peyer's patches were normal.

At the ileocaecal region the appendix was found to be 8 cm. in length and 1.5 cm. in circumference at the proximal end. It was turgid, firm and fairly erect. Its surface was smooth. The meso-appendix was also markedly thickened like the mesentery of the small gut. On section the appendix wall was found to be thickened, with the lumen narrowed to a pin point.

The caecum and the large gut showed a few surface plaques. The appendices epiploicae were all thickened and looked like tiny grapes varying in size from a quarter to half an inch in diameter. Their feel was firm and their section similar to that of the glands in the mesentery of the appendix. The pelvic mesocolon showed thickening to a lesser extent and its appendices epiploicae were grossly enlarged up to diameters of 1"-2". The mucous surface of the colon showed no abnormality.

Microscopic examination: Smears from the peritoneal exudate showed numerous malignant cells. The lumen of the appendix was encroached upon by proliferating masses of lymphoid tissue which had invaded all the coats but chiefly the

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Fig. 1.

Photograph showing thickened and erect appendix and lymphoid nodules in the mesentery.

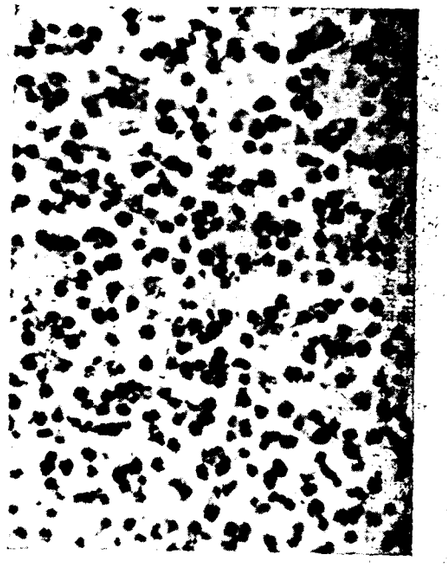

Fig. 2.

Photomicrograph of the inner coats of the appendix revealing extensive infiltration by lymphoid tissue. (x 400)

inner ones. The inner coats were replaced by an extensive and diffuse growth of lymphoid tissue. The cells closely resembled lymphocytes and showed a few mitotic figures and at places they had infiltrated in between the muscle fibres and had destroyed them.

Infiltration by similar cells was found in the mesenteric glands, meso-appendix and appendices epiploacae. The walls of the stomach, small and large intestines showed scanty infiltration by the lymphoid tissue in the serous coat only.

DISCUSSION

In this case, the uniform homogeneous appearance of the markedly thickened and turgid appendix with almost complete replacement of the inner layers of its walls by lymphosarcomatous tissue suggests that the disease originated there.

A review of the literature reveals 23 undoubted cases of sarcoma, primary in appendix and out of these 17 were lymphosarcoma. The cases reported cover all ages from 4 to 51 years, but there seems to be a tendency for a higher frequency among the younger age groups (Knox, 1945). In most of the cases the disease was not suspected till the time of operation. The cases where the growth at the time of operation was confined to the appendix are reported to have been doing well on follow up after operation.

SUMMARY

The clinical features and postmortem findings in a case of generalized lymphosarcoma of the abdomen presumably originating from the appendix are given.

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A CASE OF OSTEOGENIC SARCOMA WITH UNUSUAL FEATURES

by V. BALASUBRAMANIAN, Madras.

Secondary skeletal involvement from a primary osseous tumour is always uncommon. In this connection we have to consider only the true osseous tumours like osteogenic sarcomata and others. Pulmonary metastases are the earliest, commonest and in most cases the only evidence of spread of the disease. Osseous secondaries come next. Soft tissue metastases are yet rarer.

In the case of diffuse osseous secondaries it may be asked whether all these are the result of spread from one focus or whether they are the manifestations of the multi centric origin of the disease. This can be decided only by a study of individual cases.

Such secondaries must of course be distinguished from local recurrences. The latter are even rarer than secondaries, as far as osteogenic sarcomata are concerned. This is because of the fact that pulmonary metastases and malignant cachexia often terminate life before recurrences have time to develop.

The prevention of stump recurrences is a factor governing the treatment of the disease, whether by radiation or surgery — particularly the latter. The incidence of stump recurrences following amputations has been estimated variously. Coley (1949) records a stump recurrence of 5% in a series of forty cases.

The rarity of stump recurrence has led to a certain amount of conservative surgery in osteogenic sarcoma of some bones. This is particularly useful in tumours involving the proximal bones of the extremities. Thus one can amputate through the affected bone in sarcoma of the lower of third of the Femur, or Humerus instead of disarticulation or removal of the whole girdle. Even in the cases treated conservatively stump recurrences are rare.

CASE REPORT

The patient, a male, aged 25 years was admitted in August 1951, for a rapidly growing swelling of six months' duration, in the region of his right knee. Clinically and radiologically it was a typical case of an osteogenic sarcoma arising from the upper end of the Tibia (Fig 1 & 2). The regional glands were not enlarged. A skiagram of the chest did not reveal any secondaries.

So, an amputation was done through the lower third of the thigh. The pathology report confirmed the diagnosis.

In December 1951 — three months after the amputation the patient came for check-up. The stump was healthy ; a skiagram of the stump was, however, not taken. A skiagram of the chest revealed mutiple secondaries.

In April 1952 — seven months later the patient returned with a fusiform swelling in the amputation stump. A skiagram of the stump revealed malignant infiltration of the stump with a typical sun-ray appearance (Fig. 3).

Fig. 1.
Clinical photograph showing Sarcoma of the upper end of the tibia.

Fig. 2.
Radiograph showing sarcoma of the upper end of the tibia.

Fig. 3.
Shows the involvement of the stump by the neoplasm. Note focus in trochanter.

Fig. 4.
Shows the gross involvement of the stump. Note the soft tissue metastasis in the pectoral region.

Fig. 5.
X-Ray of chest.

During his stay in the wards he developed a soft tissue swelling in the pectoral region (Fig. 4). Biopsy of this also showed infiltration with spindle cells. After sometime a focus appeared in the greater trochanter, of the same side. Chest skiagram revealed multiple secondaries (Fig. 5).

The patient died on 22-5-1952.

Discussion

The chief points of interest in this case are the occurrence of the disease in the stump and the nature of the same.

The various possibilities are stump recurrence, metastasis and a manifestation due to multicentric origin of the disease.

Stump recurrence is rare even in amputation through the affected bone i.e., amputation through the Femur in growths of the lower end of the Femur. This rarity has led to conservative resection of the Fibula in some cases. This is a case where amputation has been done through a bone higher up. Again the stump recurrence, if it is so, has occurred after nearly eight months.

Could the disease in the stump be a metastasis ? If it were so, it is decidedly unique.

Of course the long period of delay is against a multicentric origin.

The focus in the trochanter should be considered as a secondary deposit. The deposit in the pectoral region is also of interest as soft tissue metastasis in osteogenic sarcomata are very rare.

Summary

A case of osteogenic sarcoma of the Tibia treated by amputation is described because of the subsequent appearance of the disease in the stump and soft tissues.

My thanks are due to the Dean of Government General Hospital, Madras for permission to report this case.

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INDURATIO PENIS PLASTICA (WITH CALCIFICATION)

by R. V. RAJAM, Madras.

The presence of a bone in the penis called the os penis or baculam has been stated to be a universal mammalian feature. The shape and size of the os penis assumes fantastic and bizarre forms. In the whale, it is stated it measures two meters in length and 40 cm. in circumference whereas in the anthropoid apes, it has degenerated into a small insignificant structure. In the upward evolutionary process, the bone has progressively diminished in size, until in the highest of the primates, i.e. man, it has completely disappeared. In mammals, with poorly developed erectile tissue, the os penis serves the purpose of effective copulation. It is stated that squirrels have a razor like os penis designed to excavate into the almost occluded vaginal lumen, brought on by the tremendous desquamation of the vaginal epithelium during the follicular phase of the ovarian cycle.

The condition to be described in this paper, " one of the more common of the rare diseases of the penis " in man, is said to have no phylogenetic connection with the os penis of the lower mammals, although some claim that it may be an atavistic tendency manifested in some human beings. The fact that the condition is observed only infrequently in middle aged individuals, and actually interferes with proper erection causing difficulty in copulation, is against the theory of an atavistic throw-back. Induratio Penis Plastica has been described under several names — Fibrous Cavernitis, Chronic Cavernitis, Chronic indurative Cavernitis, Fibrous Sclerosis, Fibrous Plaque. The disease was first described in 1743 by La Peyronie, Physician to Louis XIV of France. It consists of an induration of the tunica albuginea of the corpora cavernosa or the inter cavernous septum or both, forming a plaque, cord, nodule or band readily palpable and freely movable under the skin. The corpus spongiosum and the urethra are never involved. The development of similar nodules in the substance of the erectile tissue of the corpora cavernosa has been very rare. It is said the plaque tends to extend longitudinally rather than transversely. The progress of the condition is slow and insidious. The calcification or ossification of the plaque has been reported to be very rare and rarely diagnosed during life.

Incidence :

The disease was formerly considered a rarity. But from the many reports which have been appearing in medical literature it would appear that the disease is by no means un-common. In 1928 Polkey found only 541 cases reported in the literature. Burford in 1940 with a description of 40 cases of his own brought the total reported cases to 630. During a practice of 30 years, the author has observed a dozen cases of the disease, mostly in middle aged persons between the 4th and 5th decades.

Aetiology :

The cause of the disease remains unknown in the majority of cases although a number of factors, inflammatory, traumatic, metabolic and degenerative has been put forward, from time to time, by different observers. Ricord in 1840 described fibrous induration of the corpora cavernosa in syphilis. Sacher analysing the causative factors in a series of cases reported the incidence as follows : —

Diabetes and Gout	— 23.9%
Syphilis	— 11%
Gonorrhoea	— 9%
Trauma	— 8.2%
Rheumatism	— 2.1%

Calloman in a critical review of the aetiology and pathogenesis of induratio penis plastica, has ruled out the factors of inflammation, disordered metabolism, or degenerative arterial changes as possible causes for the occurrence of the condition. The indurative tissue changes occurring in

the course of syphilis, gonorrhoea and lymphogranuloma venereum, are distinct both in their location and histopathology and have nothing in common with plastic induration of the penis. The occurrence of the condition in patients with Diabetes or Gout is more incidental than causal. Zur Verth and Scheele found among 200 cases of Induratio Penis Plastica only 13 associated with gout and 15 with rheumatism. The same authors reported only 12 cases of Diabetes in 100 patients with Induratio Penis Plastica. The close histological resemblance between Induratio Penis Plastica and Dupuytren's palmar contracture, described by several investigators seems to point that they are related conditions and probably have a common aetiology. The coexistence of the two conditions in the same patient reported by several observers, lends some support to this hypothesis. Further observation is indicated on a larger number of cases. The essential histological features of surgically removed indurations are compact bundles of connective tissue with a paucity of cellular elements and the complete absence of inflammatory cells. Between the bundles of collagen fibres, spindle-shaped cells with rather poorly stained nuclei are found in small numbers. An analogous histological structure has been found in Dupuytren's contracture. Sachs and Rothschild as quoted by Calloman, have called special attention to the occurrence of characteristic cell accumulations round the smallest blood vessels. These consist of spindle-shaped cells with large intensely stained nuclei resembling embryonic mesenchymal cells. Calloman poses the hypothesis of pre-existing embryonal mesenchymal tissue, as the primary essential pathogenic factor in the development of penile induration. Individual predisposition and trauma are suggested as complementary factors which finally incite the embryonal tissue to active proliferation. Other benign fibromatous formations such as naso-pharyngeal fibroid tumours, osteitis fibrosa, leontiasis osseum, arthritis deformans, are listed by Von Gasa along with plastic induration of the penis and dupuytren's palmar contracture as a group of closely related conditions with a common primary pathogenic factor of embryonal mesenchymal rests which are stimulated to activity and proliferation by either an endogenous genetic factor of fibroplastic diathesis or exogenous factor of trauma. Calloman suggests that further investigation by critical analysis of the clinical and microscopic material may yield more conclusive results than exist at the present time.

Symptoms :

The patient discovers the condition accidentally or may notice a deformation or angulation of penis during erection. In sensitive patients, pain may be complained of on erection, and fear of pain may render a patient impotent. Rupture of the diseased penis during erection or coitus has been recorded. The act of micturition is not affected.

Diagnosis :

The diagnosis is made usually from the history of deformity on erection and by palpating the shaft of the penis between finger and thumb. A fibrotic plaque cannot be visualized by x-rays unless it is calcified.

Treatment :

Many therapeutic measures have been tried — treatment by excision, injection of fibrolysin, electrolysis, diathermy and x-rays, but without any beneficial result. In the earliest stage when the nodule is quite small, a certain measure of success has been claimed for x-rays. As the condition may be a collagen disease, it may be worthwhile to try the effect of Cortisone or ACTH on the disease.

With this short description of the disease the following case of calcified or ossified corpora cavernosa is presented.

A male aged 50 years was referred to the writer by a private physician for examination and advice.

The patient has been a diabetic for nearly a decade with blood pressure of 160 100. He has had no special treatment for diabetes. He is otherwise healthy, of active habits and never denies himself the material pleasures of life. Two years ago during a casual palpation of the penis while bathing, he felt a painless, cord like thickening in the substance of the shaft of the penis and according to his statement has since been growing slowly in size and extent. There has been no pain or discomfort. The organ looks normal and the patient has not noticed any deformity or angulation of the penis during erection. No pain or difficulty is felt during sexual intercourse. But the patient has been noticing a slight decrease in the circumferential girth of the turgid shaft. On further questioning about his sexual habits and technique he came out with the statement that some time prior to his noticing the lump in the penis he felt a sudden exquisite twinge of pain during a vigorous act of coitus with a virgin. The pain was as evanescent as it was sudden and was not followed by any outward swelling of the organ. Examination revealed a circumcised penis with penile hypospadias. Palpation of the shaft showed a linear, slightly irregular, stony hard plaque extending from the base of the glans penis to the root and situated between the corpora cavernosa dorsally, with similar but smaller less easily palpable plaques extending transversely on either side from the longitudinal one. No tenderness was felt on palpation. Radiographic examination of the penis showed an extensive calcified or ossified plaque involving the length of the pectinate inter-cavernous septum with transverse and lateral prolongations giving a superficial resemblance to the vertebral column and ribs of a sardine from which the flesh has been removed vide (x-ray photographs). The fact that with such extensive calcification the power of erection has not in any way suffered, is evidence that the disease is limited to the tunica albuginea and the pectinate septum, and the capacity

Fig. 1.

Fig. 2.

Figs. 1 & 2. X-ray photographs of the penis showing the progressive calcified induration (taken on 30-4-52 and 14-11-52).

Fig. 3.

X?ray photograph of the penis showing the progressive calcified induration (taken on 28-1-53).

of the erectile tissue to swell is not seriously affected for the present. What the future holds out for the patient cannot be visualised now. There is no evidence of fibromatous formation in any other part of the body.

Comment :

Although the fibrous tvpe of Induratio Penis Plastica has been described and reported often in medical literature, the report of the calcified or ossified variety has been rare. It is stated that the discovery of the ossified condition has been made in a few patients usually at autopsy, sometimes after excision and rarely by x-ray examination. In the case under report the calcification was suspected clinically because of the stony hard, inelastic feel of the plaque, and confirmed by x-ray examination. It is true that the x-ray examination revealed a greater extent of the ramification of the calcified plaque than was suspected on clinical examination. Inspite of such an extensive calcified involvement of the sheath of the corpora cavernosa, pain or discomfort or angulation of the penis during erection or inability to perform coitus, seems absent, according to the statement of the patient. In this case trauma to the sheath of the corpora cavernosa in a chronic diabetic might have initiated the process of a productive chronic fibrous tissue reaction which later underwent a progressive calcification.

Summary

A rare case of calcification or ossification of corpora cavernosa is presented with a review of the more common fibrous type of induration which is a precursor of the ossified type. The writer has great pleasure in acknowledging the help of Dr. Mohamed Mumtaz Ali, a leading private physician for referring the case and for supplying the relevant information on the history of the patient, and the x-ray photographs of the condition.

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HYDATID DISEASE OF THE SPINE

*by R. NIGAM and V. G. GHARPURE, Nagpur.**

Hydatid disease is less common in this country as compared to Australia, Syria, South Africa and New Zealand[3]. Indian Statistics are yet lacking as regards the frequency of sites of the disease. According to Australian Hydatid Registry Buckley[2] reported 1802 cases. The frequency of the sites of disease in 1802 cases was Liver and Peritoneum 63.3%, Lungs 24.5%, Muscles and Fascia 4%, Bones 2.5%, Kidneys 2.1%, Spleen 1.4%, Brain 1%, the other viscera such as Heart, Breast, Thyroid, Parotid, Prostate and Pancreas were responsible for 0.8% of the cases.

This clearly shows that with the exception of the liver and lungs other sites are rarely affected. In India hydatid disease has been reported in the liver, peritoneum, heart, lungs[7], parotid glands[1], innominate bones[4], breast and the kidneys. There has been no report of a case of Hydatid disease of the spine.

CASE HISTORY

Bajirao 32 yrs. Hindu male farmer by profession of Amraoti District (M.P.) was admitted to the Medical College Hospital, Nagpur with the following complaints :—

Pain in back radiating to the front of the chest. On left side ..	8 months.
Swelling in the back ..	7 months.
Paraplegia and Loss of sensation below the level of xiphisternum	2 months.

Eight months prior to admission the patient had pain in the back and chest more marked on the left side. He also used to get girdle pains at the level of the xiphisternum. There was no past history of any venereal disease or trauma or tuberculosis. On examination he was well built. There was a diffuse cystic swelling 4" x 3" situated on the back at the level of 5th to 8th dorsal vertebrae extending from the midline to the lateral border of the left sacrospinalis muscle. Slight scoliosis present to the right side. Skin over the swelling was free. The swelling was fixed to the spine and the ribs. Tenderness was present over dorsal 5th to the 8th vertebrae. Neurological examination showed complete loss of motor power in the lower limbs with increased tone. Knee and ankle jerks exaggerated on both sides. Plantar response was extensor on both sides. There was complete loss of sensation below the level of the xiphisternum. The blood picture was normal and there was no eosinophilia. Cerebrospinal fluid was clear, under normal pressure, Queckenstedt test showed partial obstruction with a plateau like curve. Protein, chlorides and sugar contents of C.S.F. were normal. Skiagram of spine showed ill-defined homogeneous opacity at the level of 5th to the 8th dorsal vertebrae, obliterating the structure of the vertebral bodies and destroying the bodies and transverse processes of the vertebrae and the vertebral ends of the ribs (Fig. 1 & 2).

With the above history and findings the provisional diagnosis was that the case may be one of tuberculosis of the spine with a paravertebral cold abscess or a case of spinal tumour. With these findings in view the case was operated upon under intratracheal gas and oxygen anaesthesia. The swelling was explored by a median vertical incision. The swelling contained pus and daughter cysts. There was a large cavity encroaching into the left pleural space and extending in the vertebral canal and pressing on the spinal cord. Laminectomy was done from D5 to D8. Daughter cysts were found between the dura and laminae of the vertebrae. The cavity was evacuated and then cauterised with formaline. Wound closed without drainage. Postoperative course was uneventful. Patient made a complete recovery of his paraplegia and now he is able to stand and walk with slight support.

Discussion

This case is reported because of its rarity, possibly the first case of its kind in the Indian medical literature. Hydatid disease of the other parts of body like liver, lungs and peritoneum is not rare in India. The diagnosis is difficult and this is usually established at operation only. It does not cause prominent symptoms until very late. Attention of the patient is drawn towards it either due to its size or due to its pressure effects and sometimes due to anaphylactic reaction as a result of rupture of cysts. History is not clear, neither is the association with dogs proved in most of the cases. In India most probably it is not the close

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Fig. 1.
A.P. View of Spine. Dorsal Region.

Fig. 2.
Lateral View. Thoracic Spine.

association with dogs that is responsible for the disease but it is the infection through contaminated water, food or vegetables with the excreta of dogs or through flies. Bone is one of the rarest sites. It is a slow and progressive disease. When bones are affected, bone formation is inhibited resulting in solid masses of hyaline material. The bone expands in a globular or fusiform manner.

The X-ray picture may simulate a giant cell tumour or osteitis fibrosa cystica from which it may be difficult to distinguish[5]. Medical treatment is of no help. The cyst requires exposure and isolation from the surrounding parts. It is evacuated and cauterised with formaline and then allowed to heal by granulation tissue. As other parasitic diseases it is a preventable disease.

Summary

A case of Hydatid Disease of the Spine is reported. According to statistics Hydatid disease of bone is very rare and probably the spine reported in the Indian medical spine reported in the Indian medical literature.

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HIDRADENOMA OF THE VULVA*

(A Clinico-Pathological and histogenetic study)

by D. BHASKARA REDDY and FATIMA, Madras.

Tumours of sweat glands are rare. Peterson[6] is said to have reported the first case in 1893. They may arise in any part of the body although the breast is the frequent site. Reports of these neoplasms arising from the vulva are infrequent. Pick[7] in 1904 while reviewing reported cases, gave detailed description of three vulval sweat gland tumours. A study of the literature reveals 80 cases of sweat gland tumours of the vulva reported upto date. In recent years Novak and Stevenson[5] recorded fifteen cases and discussed their histogenesis and grade of malignancy. According to them uncontestable clinical and histological evidence of malignancy in these tumours was recorded by Schiffmann in 1920 and Eichenberg[2] in 1934; they view with suspicion the large number of cases of malignant sweat gland tumours reported by McDonald et al[3]. According to Novak[5] only two showed adeno-carcinomatous changes of undefinable histogenesis in thirty reported cases.

The benign sweat gland tumours of the vulva are rarely clinically recognised and occasionally reach a size over 1 c.m. in diameter. Their indolence in growth and

Fig. 1.
Photomicrograph illustrates elongated processes arising from basal cells, many of which show lumina. (H & E x 60)

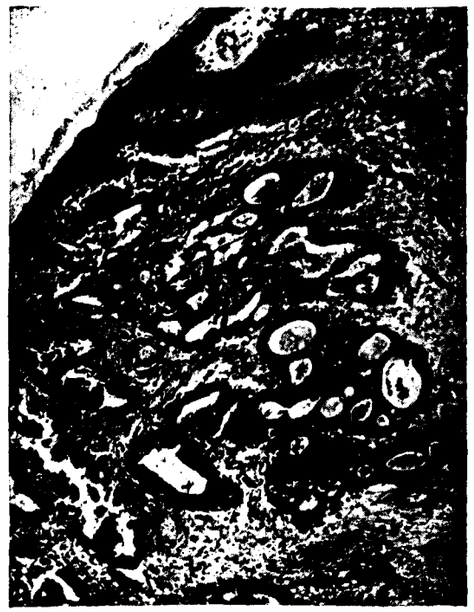

Fig. 2.
Fhotomicrograph shows adenoid cystic epitheliomatous pattern of hidro-adenoma, arising from the basal cells. (H & E x 60)

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diminutive size contribute to clinical latency while breach in the skin covering the tumour and subsequent bleeding force the patient to seek medical aid. The exact age incidence of these tumours naturally is difficult to estimate. They are often recognised for the first time during the age period 40—50 years ; the lowest age recorded is 27 years. Owing to its asymptomatic and benign nature unless the condition is borne in mind by the Surgeon, it is likely to be missed and this accounts for the small number of reported cases.

CASE REPORT

A nulliparous female, M., aged 50, sought medical aid on 12-6-'52 in the Gynaecological clinic of the Women and Children Hospital, Egmore for a nodular swelling in the region of the vulva of four months' duration. She had observed blood stained discharge for sometime. Menstrual and obstetrical history noncontributory. Since four months the nodule has been increasing in size and at no time had it been painful.

Patient's general condition fair. Heart and lungs nil remarkable. Inguinal glands not palpable.

Local examination : A nodule 1.25 c.m. diameter was located adjacent to the urethra on the medial aspect of the labia minora. It was ulcerated and was found to bleed to touch but not tender. Preoperative biopsy done on 13-6-'53, showed normal covering squamous epithelium. Arising from the basal cells were elongated processes interrupted at regular intervals by tubular-like structures lined by a single layer of cells (occasionally double) with a central lumen containing light bluish staining material (Fig. 1). Deeper down was seen a cluster of these structures with a definite adenomatous pattern.

Biopsy diagnosis : "Hidradenoma".

On 26-6-'53 she was readmitted in the hospital. On 1-7-'53, under gas and O_2, the nodule was excised by wide incision covering the vaginal wall. The growth was seen to extend into the fornices. Bleeding was controlled by multiple sutures. Pencillin therapy controlled post operative febrile reaction. The wound was healed on 10-7-'53 when she was discharged. Ever since she has been visiting the hospital for clinical check purposes and so far no recurrence or metastases have been observed.

Fig. 3.
Photomicrograph illustrates papillary cyst adenomatous pattern of hidro-adenoma.
(H & E x 60)

Fig. 4.
Photomicrograph illustrates intra-luminal massive haemorrhages, a feature of hidro-adenoma.
(H & E x 60)

Biopsy study of the operated material : Multiple sections of bits of reddish friable material was subjected to histological study. The surface epithelium was stretched and thinned out in some places and in other places showed evidence of proliferation. The basal cells stained deeply. These cells were seen in a pattern similar to that of adenoid cystic epithelioma (Fig. 2). The lumen of the cystic spaces contained light stained eosinophilic material. The lining epithelium of these glands was single or multilayered and bore a close resemblance to the basal cells. They were hyperchromatic and of variable size. Deeper still was seen wild papilliferous proliferation with cystadenomatous pattern (Fig. 3). Necrosis and haemorrhage were significant features of the tumour mass (Fig. 4). Hyalinisation of the haemorrhage covered with a mantle of proliferative cells often mimic the appearance of products of conception. The cells were hyperchromatic and presented marked variation in size and shape. Intraluminal haemorrhages of the glands presenting a peritheliomatous pattern was a significant histological feature (Fig. 5). Sections from the deeper parts of the tumour revealed infiltration of the stroma by papillary cystadenomatous processes (Fig. 6). Innumerable tumour emboli in the venules were observed (Figs. 7 and 8).

Revised Biopsy Diagnosis : "Hidradenoma — Malignant".

Fig. 5.
Photomicrograph shows peritheliomatous pattern of the tumour. Collections of red cells bordered by multilayered epithelial cells, (H & E x 100)

Fig. 6.
Photomicrograph illustrates papilliferous cystoadenomata infiltrating interstitial tissue.
(H & E x 60)

Discussion

Meyer, Pick and Novak have conclusively established the histogenetic relationship of hidradenoma of the vulva to the apocrine type of sweat glands normally found in this region. Figs. 1 and 2 illustrate the undoubted origin of the neoplasm from the basal cells of the squamous epithelium covering the region. The twin layered (some times more than two layered) adenomatous and often the papilliferous cystadenomatous nature of these tumours is well exemplified by the case reported. The polymorphic histological patterns of hidradenoma illustrated by Figs. 1, 2, 3 and 4 bear close similarity to six cases recorded by Danforth[1] and Evanston in 1949.

Novak and others are emphatic that the majority of the sweat gland tumours are both clinically and histologically benign and any reported case of malignancy in these is more likely to be adeno-carcinoma of non-definable origin and is often the reason for infrequent clinical recognition of the condition by the Gynaecologist. Preoperative biopsy in the case recorded showed no evidence of malignancy. Infiltration of the interstitial tissue by papillary cystadenomatous processes, deep staining nuclei, variation in size and shape of the cells and innumerable tumour emboli revealed by histological study of multiple sections of operated material are indisputable microscopic evidences of malignancy. The histological findings portend future possible metastases and point to the necessity for radical surgical treatment by wide excision and for keeping the patient under observation for an indefinite period as is being done in the case recorded.

Fig. 7.
Photomicrograph illustrates multiple tumour emboli. (H & E x 60)

Fig 8
Photomicrograph illustrates details of the tumour emboli. (H & E x 120)

Summary

1. Literature pertinent to Hidradenoma is briefly reviewed.

2. The polymorphic histologic patterns observed by others are confirmed.

3. The necessity for a detailed histological study of the operated material to disclose any evidence of malignancy that might not have been observed from a surface (in-adequate) biopsy of the tumour is emphasised.

4. The origin of hidradenoma from apocrine glands of the vulva is illustrated by the case report.

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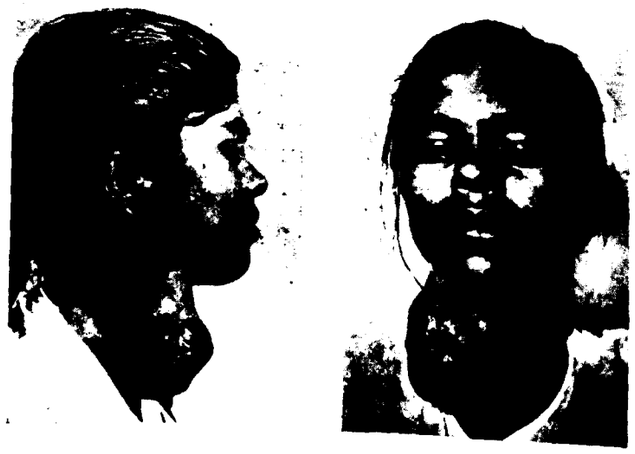
TERATOMATA OF THE THYROID GLAND

by H. S. BHAT, Vellore.

Reports on teratomata of the thyroid gland are infrequently met with in the literature. The previous reports of such teratomata were not made on detailed microscopic study. These tumours are of great interest to the pathologist, but to the clinician, teratoma of the thyroid gland is yet another congenital swelling in the neck of the new-born. According to Willis[1] teratomata are true tumours composed of multiple tissues foreign to the part in which they arise. During the early development of the embryo before the oral membrane perforates, the neck is absent and the rudimentary thoracic organs are in close proximity to the oral region. This relationship changes later on when the head extends and the neck forms. Disturbances originating in the anterior end of the notochord and the adjacent tissues may be transmitted to the prospective mediastinal tissues and the anterior cervical tissues via the oral membrane during the early development. It is significant that most cervical teratomata involve the thyroid gland which arises as an outgrowth from the oral entoderm close to the oral membrane. Since these tumours apparently arise from totipotent cells, no single tissue can be mentioned to denote their individuality as was suggested by Saphir[2] with regard to thyroid tissue. Hence in teratomata of the neck, one has often to rely on anatomical situation rather than the presence of thyroid tissue. In identifying teratomata of the thyroid the relations of the superior and inferior thyroid arteries may be considered. Since only a few cases in the literature have shown this feature, its significance is not great. But it does indicate that the primitive tissue which ought to have formed the thyroid with all its functions, changed into a teratoma with its multiplicity of tissue structure. The presence of true adult thyroid tissue in these tumours recorded in the literature does not seem to be of much importance since very few records stated it to be of much importance since very few records stated it to be present. Brain tissue seems to be a predominant characteristic of teratomata in this region. Most cases reported since 1895 show glial tissue or



Figs. 1 & 2.
Clinical photographs showing the tumour.

Fig. 3
Clinical photograph showing the incision

Fig. 4.
Photograph of the tumour removed.

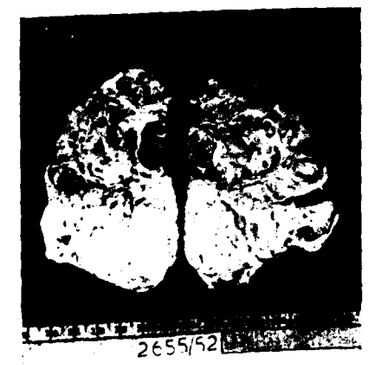


Fig. 5.
Cross section of the tumour.

ganglion cells as a feature in such tumours. Encapsulation has been described quite frequently. More recent records describing these features are those of Wilson[3], Potter[4], Bale[5] and Perkins et al[6]. Teratomata of the neck were associated in many cases with dystocia, and still births. Only 3 cases in Bale's series were found malignant. The low incidence of malignancy which is about 5% in teratomata of the neck is significant when compared to the higher incidence in teratomata described in other regions. This probably is due to the fact that these infants die early of respiratory obstruction or they have been treated early (McGoon[7]).

CASE REPORT

K.B. a healthy female, 14 years of age, was admitted into hospital on 3-9-'52 with a mass in the right anterior triangle of the neck, measuring 9 x 5 x 4 cms. The tumour was present at birth and it was increasing in size gradually from its original size of about 1" diameter. Birth was normal. Ulceration started about 5 years before admission and the sinuses were discharging straw coloured fluid but no blood or pus was seen at any time. Pain and difficulty in breathing or swallowing were absent all along. The presence of the large mass in the neck was the only cause for seeking admission.

Family history: This did not reveal any significant features.

Clinical findings: A nodular mass with irregular superficial ulcers on the summit and a few sinuses at the lower end, was seen in the right anterior triangle of the neck. The skin over the tumour showed thinning and stretching in some areas. The tumour was nodular to feel and cystic in one area but it was not tender anywhere. No pulsations were felt and no bruit was heard over the tumour. Slight vertical movement with deglutition could be demonstrated. Right sternomastoid was stretched over the posterior aspect of the "tumour". A few firm lymph nodes were palpable at the upper end of the deep cervical group, on the right side. The edges of the tumour were clearly palpable and well demarcated. No stridor was elicited.

Examination of the throat and nose showed no abnormalities. Cranial nerves were normal in function. Other systems were found normal. Transillumination showed no areas of increased translucency.

Investigations: Blood pressure 115/70 mm. of mercury. Pulse 80 per minute. Respiration 20 per minute. Total white blood cell count 11,600/cmm. Differential count: Polymorphs 52%; Lymphocytes 36%; Eosinophils 12%. Blood Kahn — negative. Smears and cultures from the discharge showed only bacillus proteus and staphylococci.

Diagnosis: The presence of the tumour from birth and the location suggested a branchial origin. But the vertical movement of the tumour with deglutition was in favour of a thyroid tumour. The tumour was hard and cystic in places. So a pre-operative clinical diagnosis of a malignant adenoma of the thyroid gland was made.

Radiological examination of the neck and chest showed no evidence of pressure on the trachea. Heart and lungs showed no pathology.

Operation on 15-9-'52: Under endotracheal ether, the tumour was exposed with a vertical incision bifurcating over the mass to include the ulcerating areas. A well developed capsule was seen. The carotid sheath was identified and isolated. It was found to be pushed well out to the

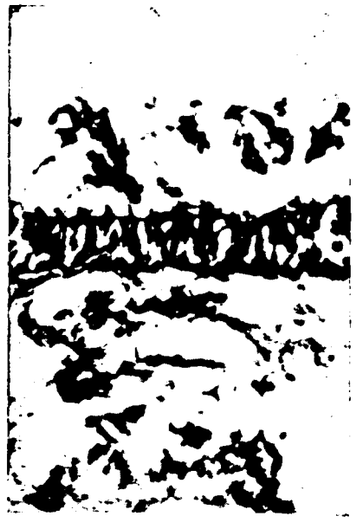


Fig. 6.

Microscopic section showing ciliated pseudo-stratified epithelium. (x 460)

Fig. 7.

Section showing stratified squamous epithelium. (x 460)

Fig. 8.

Section showing cartilagenous tissue. (x 460)

Fig. 9.

Section showing brain tissue. (x 460)

lateral side. The mass was separated first at the upper pole, then at the lateral edge and finally at the lower end. The superior thyroid artery of a fairly large size was seen disappearing into the upper part of the right lateral edge of the tumour. Dissection in the lower part of the tumour was easy and no evidence of any other vessels entering the tumour was present. The tumour was dissected out from the trachea and the thyroid cartilage on the right side from which it was separated by a well developed capsule. There was no normal thyroid tissue seen on this side and exploration carried to beyond the middle line revealed the normal looking thyroid gland. After haemostasis the wound was closed in layers.

Pathology: Biopsy No. 2655|52.

Macroscopic: Tumour was measured 9 x 3 x 3 cms. and it was partly cystic and partly solid, covered over by fibrofatty tissue. The cut section showed a variegated appearance. In places it was cartilagenous and multiloculated with some spaces containing fat and others sebaceous material.

Microscopic: Sections taken from different parts of the tumour showed fibrous tissue, cartilage, smooth muscle, mucous glands and cystic spaces, some lined by ciliated pseudostratified epithelium and some by squamous epithelium. One cyst showed marked proliferation of epithelium in its wall. Some sections showed brain tissue with ependyma lined spaces.

Impression: Teratoma.

Summary and Comments

One case of teratoma of the thyroid gland is described and the essential features described in previous records are mentioned. The case record given above presents many similarities to cases reported in the literature. The low incidence of malignancy in teratomata of the neck as compared to such tumours elsewhere is significant. The absence of the right thyroid lobe and the apparent supply of the tumour by the superior thyroid artery is suggestive of the origin of the tumour. The presence of a well developed capsule at operation and brain tissue in microscopic sections seems to be predominant features in most of the recorded cases.

I express my gratitude to Dr. J. S. Carman, Professor of Surgery and Dr. M. Balasubrahmanyan, Associate Professor of Pathology for their valuable guidance.

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OSTEOID OSTEOMA*

(A report of three cases)

by C. VYAGHRESWARUDU, Madras.

Introduction

An osteoid osteoma is defined as a small oval or roundish tumour-like nidus composed of osteoid tissue deposited with a substance of highly vascularised osteogenic connective tissue. The lesion even when it is fully evolved does not exceed a centimetre in its maximum dimension. It develops either within the spongiosa or in the cortex of the bone. When it develops in the spongy bone, only a thin rim of reactive sclerotic reaction is present, but when in relation to the cortex a dense beyond the osteoid tissue is seen. It is most common between the ages of ten and twenty years. The favourite sites are the long bones of the body (Femur, Tibia and small bones of the foot and the humerus and fore-arm in the order of its incidence). It is not seen either in the skull or in the rib (Lichtenstein).

Jaffee was the first to report five cases in the year 1935 all occurring in the spongy portion of the bone. Later Jaffee and Lichenstein reported that the lesion developed in relation to the cortices of long bones. The total number of cases so far recorded are about one hundred.

The duration of symptoms varies from six months to two years. Lichtenstein quotes a case where the symptoms persisted for seven years and were completely relieved after operation. In all cases the main symptom is pain increasing in persistence and severity and often keeping the patient awake. Locally, swelling becomes apparent at a later date.

Radiograms of these cases show a nidus, small, oval or roundish in form which is relatively radioluscent and surrounded by a sclerotised zone with a dense shadow reflecting the reactive sclerosis of the lesion. The perifocal dense area extends several inches above and below the lesion. The typical nidus will be evident only by either overexposing or toning down the plates.

*From the Dept. of Orthopaedics, Govt. General Hospital, Madras.

Pathology

When an osteoid osteoma of the spongy bone is removed intact without crumbling, it stands out as a circumscribed reddish bony focus. When a thickened cortical boneblock containing a nidus in its interior is excised and serial slices made, preferably with a band saw, it will reveal the focus unconfined in one or two of the blocks. When the nidus is broken up into small fragments the latter may be mistaken for bits of granulation tissue (Lichtenstein).

Microscopically section shows a circumscribed focus of Osteoid tissue, more or less calcified, atypical bone, developing within a background of richly vascular osteoblastic connective tissue. Within the reactive new cortical bone one may observe small focal collections of lymphocytes in the immediate vicinity of the nidus, but this feature apparently reflects nothing more than an expression of chronic irritation.

Jaffee and Lichtenstein have argued out the possibility of the lesion being inflammatory in their article and conclude that it is best interpreted as a neoplasm and specially as a peculiar benign tumour of the bone of osteoblastic connective tissue derivative.

Treatment

Surgical removal will effect immediate and lasting cure, if removed completely. No recurrence is seen. Pain disappears dramatically. It is suggested that it should be removed by block resection to include the nidus for complete removal as well as for purposes of study. The fate of untreated lesions is not known and no relevant information is available in regard to the possible effectiveness of irradiation.". "Lichtenstein".

Summary

Of the three cases two are girls and the third is a male adult. The girls are of the same age, i.e. 14 years, the boy is about 20 years. Duration of the symptoms in all the three cases ranged between nine months to one year. All of them tried, for relief of pain which was the main symptom, various treatments none of which were of any use. One of the girls had been taking "Cibalgin" tablets totalling about 300. This used to give temporary relief from the pain. In all the three cases the lesion was in the femur, just about its middle. The lesion was on the outer aspect of the femur in one case, but in the other two cases it was in the inner aspect of the femur. In all cases the nidus was seen in the cortex and was demonstrated by taking X-rays with different intensity.

At operation the lesion i.e., the thickened bone has been chiselled out by a gouge osteotome. The bone was very hard and felt like marble bone. The bone did not look like that of inflammatory origin. The pathology reports of the nidus, from our pathology department was "chronic inflammatory tissue". In spite of the pathology report we believe that it is a case of osteoid osteoma. Since, as has already been pointed out, bits of tissue examined separately may look like chronic inflammatory tissue. (Lichtenstein).

All the cases are relieved of their symptoms. The first two cases were followed up for two years nearly, and there was no recurrence of the symptoms. The latest X-ray showed filling up of bone which has been chiselled off.

CASE REPORTS

CASE 1.

Name — Miss X. Age — 14 years, Female. Occupation — Student. Date of admission — 14-5-1951. Date of discharge — 20-6-1951. Complaint — Pain in the right thigh. Duration — One year.

History: Since one year patient was having pain in the right leg, sometimes referred to the knee. The pain was continuous and was boring in character. She was taking treatment under various doctors. She used to take "Cibalgin" regularly daily for relief of pain since the beginning of the pain and she had tried all sorts of analgesics for the relief of pain.

Family history: Five brothers and sisters. None of them suffered from this disease.

General condition: A fairly well nourished girl of about fourteen years. All other systems are normal.

Local condition: There was no visible swelling in the thigh on the right side. No visible veins

Fig. 1.

Case 1. Pre and post operative x-ray photos of femur.

Fig. 2.

Case 2. Pre and post operative photos of femur.

were present. Skin normal. On passing the hand slight swelling was felt in the postero-medical aspect of the thigh. On deep pressure pain was felt. It was neither warm nor tender. Movements of knee and hip normal. There was no wasting of muscles.

Investigations :—

X-ray of the right femur showed thickening of the cortex on the postero medial aspect seen both in A.P. and Lat. views. In one picture there was a small decalcified area in the cortex over which new bone was seen. Medulla normal.

Blood calcium 10 mgms. Blood W.R. Negative. R.B.C. 4.2 millions per cm. Blood pressure 110 70. Urine : Albumin—nil, Sugar—nil. W.B.C. 7000 per cm. Differential count :—Poly. 75%. Lymph. 20%. Eosin. 5%.

Treatment :—

She was operated upon on 30-5-51 and had an uneventful post-operative period. Pain disappeared after the operation. On 20-10-51 patient reported no recurrence of pain. On 25-653 patient replied the follow up card that she is doing well.

CASE 2.

Name — Mr. X. Age — 24 years, Male. Occupation — Student. Date of admission — 15-8-1951. Date of discharge — 16-9-1951. Complaint — Pain in the right thigh. Duration — Nine months.

History : Patient complained of pain in the right thigh for over nine months. He had tried various methods of treatment but had no relief. Five months ago he was examined in the orthopaedic department and nothing was made out ; the symptoms were suspected to be functional. He was advised some exercise. Patient had no relief and reappeared in the month of August 1951. This time a practitioner had noticed a small palpable swelling in the thigh and he was referred to the orthopaedic department.

Family history : Nil particular.

General condition : A well nourished muscular individual of about 24 years. All other systems were normal.

Local condition : There was no visible swelling in the right thigh. Active movements of the hip and knee were quite normal. On passing the hand over the thigh a fullness was felt over the lateral aspect about its 1 3rd. Skin over the swelling was normal. No warmth. No tenderness. Pain present only on deep pressure.

Investigations : —

X-ray shows a fusiform thickening of the cortex in the upper 1 3rd of the right femur affecting only one side. Medulla is normal. In the cortex was seen a small decalcified area.

Blood W.R. Negative. Blood calcium 10 mgms.%. Blood pressure 125 80. W.B.C. 6800 per c.m. R.B.C. 4.5 million per c.m. Urine : Albumin—nil. Sugar —nil.

Treatment :—

On 20-8-51, patient was operated on and had an uneventful recovery. He had relief from pain

Fig. 3.
Case 3. Pre operative x-ray of femur.

Fig. 4
Case 3. Post operative picture.

after operation. On 1-6-53, patient reported for re-X-ray. He has no recurrence of pain. X-ray showed complete filling up of the original cavity made at operation.

CASE 3.

Name — Miss X. Age — 15 years, Female. Occupation — Student. Date of admission — 9-2-1953. Date of discharge — 10-4-1953. Complaint — Pain in the right thigh. Duration — Eight months.

History : Since eight months she was having constant pain in the middle of the left thigh, shooting down sometimes to the knee joint. A few days ago she noticed a little lump on the inner aspect of the thigh. She had treatment of various kinds for the relief of the pain, but without relief.

Family history : Nil particular.

General condition : A fairly well nourished girl of about 15 years. No other disease in the body made out on examination.

Local condition : There was a visible swelling over the inner aspect of the left thigh about its middle. The skin over the swelling was healthy and normal, no visible veins present. On palpation the swelling was hard and appeared to be arising from the bone underneath. The edges could be defined easily. Tenderness present only on deep pressure. Movements of the hip and knee are normal. No wasting of muscles.

Investigations :—

X-ray of the left femur shows a thickened area in the middle of the femur on its inner aspect and a small nidus is seen in the cortex of the femur.

Blood calcium 9.7 mgms.%. Blood W.R. Negative. R.B.C. 3.5 million per cm. W.B.C. 7000 per cm. Blood pressure 100|70. Urine—Nil particular.

Treatment :—

On 11-2-53 patient was operated on and had normal recovery. Reported on 30-6-53. Patient is free from pain.

My thanks are due to Dr. V. R. Thayumanaswamy, M.S., F.R.C.S., M.Ch. (Ortho.), Professor of Orthopaedics and Orthopaedic Surgeon, Govt. General Hospital, Madras and to the Dean, Govt. General Hospital, Madras for permitting me to publish these cases.

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OSTEOCLASTOMA FROM THE TEMPORAL BONE

(A Case Report)

by J. B. SHRIVASTAV and K. D. SHARMA, Nagpur.

Osteoclastoma arises usually from the epiphyseal ends of the long bones. Osteoclastoma arising from the skull, apart from the jaw, is rare (Lord, Stewart, 1943). Records of the John Hopkins Hospital for 35 years show only 22 cases of osteoclastoma in the skull, 14 in the mandible, 6 in the maxilla and 2 in the rest of the skull — both of them in the sphenoid. In a recent article D. Govinda Reddy (1953) has reported 96 cases of osteoclastoma, examined in the Madras Medical College over a period of 10 years, of which 13 occurred in the maxilla and mandible. The other bones of the skull were not involved. In our series out of 15 cases over a period of four years, 9 cases were from the long bones, 5 from jaws and one from the skull bone.

Very few cases of osteoclastoma from the temporal bone have been reported in the literature. Lutz (1900) Nicolai (1912), Dodertem (1913) and Ramadier and Tomaray (1937) have reported cases of osteoclastoma from the temporal bone. Lord and Steward (1943) have reported a case from a female aged 50 years who was complaining of tinnitus and vertigo. A polyp was found to be occupying the right auditory meatus. It was removed but not subjected to any histological examination. The polyp recurred and it appeared to be coming from the middle ear. Microscopic examination this time revealed it to be typical osteoclastoma. This case was studied in great detail during life as well as at autopsy. In spite of its wide extension in the temporal bone, the tumour remained benign.

Fig. 1.

Photograph of the Tumour occupying the external auditory meatus.

Fig. 2

Note the giant cells typical of osteoclastoma. The stroma shows evidence of Fibrosis (H.P.).

We are presenting this case due to the extreme rarity of osteoclastoma from the temporal bone.

CASE REPORT

A female patient aged 28 years was admitted in the Surgical Ward on 20-3-1952. She gave the history of a small pimple over her right ear on the tragus of 3 months' duration, which went on growing and was accompanied by pain in the ear and headache. It was excised in the District Hospital but recurred. This time she developed some discharge from the ear and was running a low temperature.

On examination, a hard growth of the size of a small lemon was found in the right ear in front of the tragus (Fig. 1). It was movable and bled on touching. Regional lymph nodes were enlarged.

A clinical diagnosis of either a Dermoid or an Osteoma was made. Biopsy was performed on 28-3-1952 and the histology showed features of infected fibroma. On 16-5-1952 diathermy removal of the growth down to the pedicle was done. The pedicle was coagulated by diathermy needle; middle ear was not explored due to sepsis. Post-operative period was uneventful.

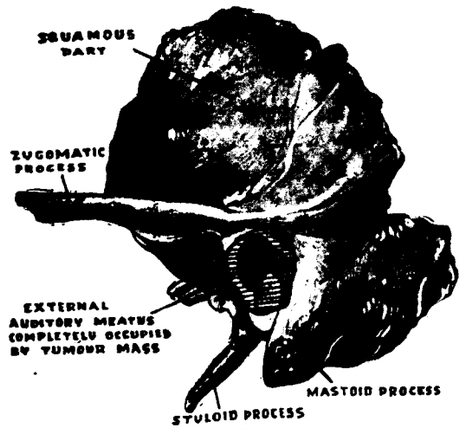

Fig. 3.

Diagram showing the site of the Tumour in the Temporal bone.

Gross and Microscopic appearance of Tumour Mass: Tissue consisted of an irregular hard and dark brown piece measuring 3 x 2.5 x 3 cms. Cut section showed greyish areas with dark brown periphery. The centre of the tumour was soft whereas the remaining parts were bony hard.

Microscopy: The tissue shows admixture of spindle cells and multinucleated giant cells, distributed uniformly throughout the field. The giant cells possess 10—40 nuclei, scattered irregularly in a blue stained cytoplasm. The tumour shows a large number of thin walled vascular channels. There are plenty of collagean fibres as a result of sepsis and secondary fibrosis. The biopsy material was taken from one of such areas and, therefore, could only show the structure of a fibroma. Histology is typical of benign giant cell tumour or Osteoclastoma (Fig. 2).

COMMENT

A case of Osteoclastoma of the temporal bone is reported. The tumour remained confined to the external auditory meatus and apparently was not coming out of the middle ear. The tumour recurred very rapidly within three months of its removal in the District Hospital and on its subsequent excision and histologic study proved to be benign giant cell tumour.

We express our thanks to Dr. G. L. Sharma, Dean, Medical College & Hospital, Nagpur for allowing us to publish the case report and Dr. R. S. Seth for the photograph of the case and Shri S. Shah for the technical help.

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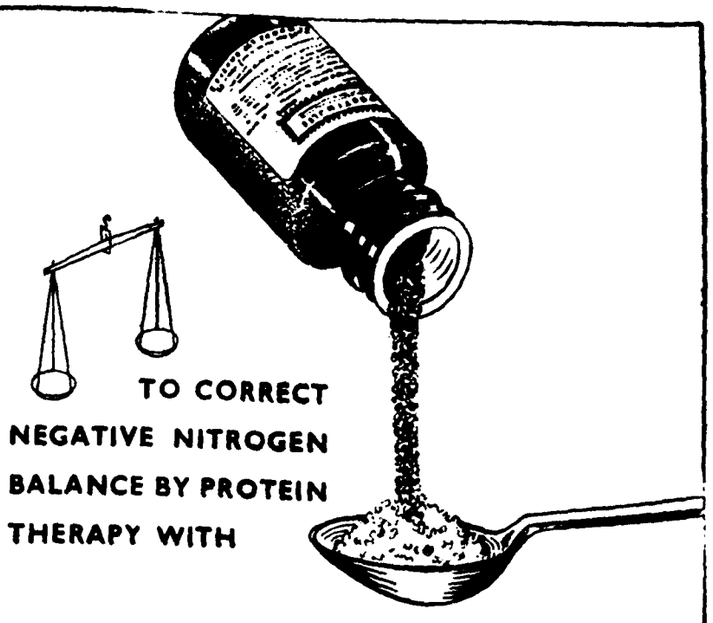

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