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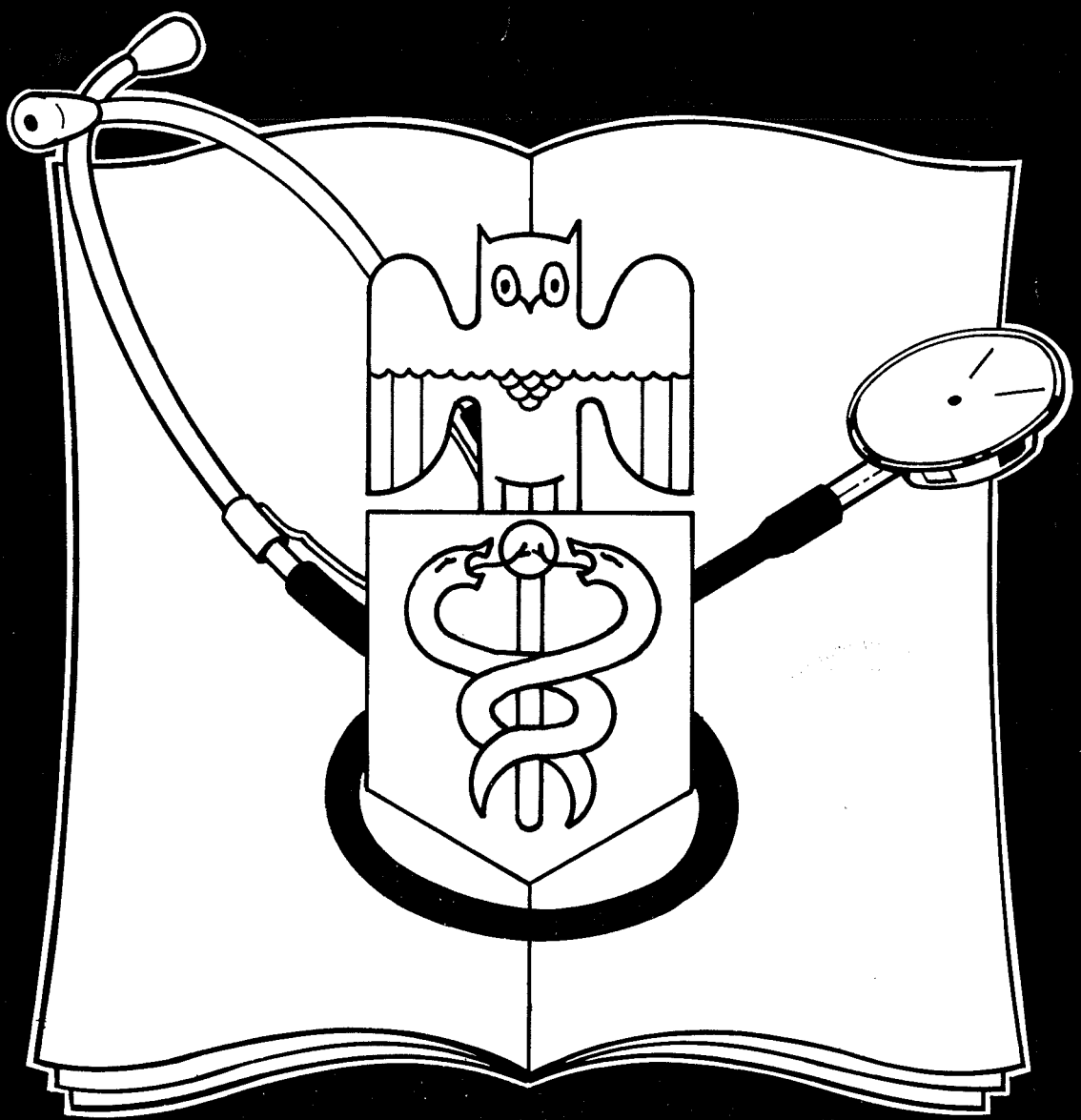

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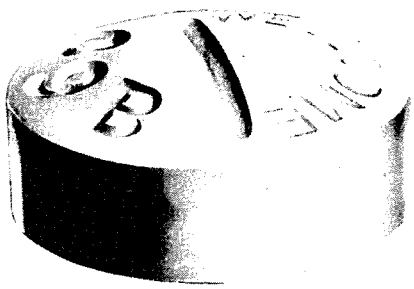

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Editor's Letter

Mediscope Issue No. 5 1984
Medical Students Journal
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This issue celebrates the first birthday of this publication which has been replacing the previous medical students' journal *Chestpiece*. We are having encouraging support both financially, through advertisements and sales, and with respect to articles from doctors. Though such articles are piling in our office, we still lack contributions from students. Many students have come up with some quite unusual and promising titles, however, the articles have never been written and even if written they were never submitted to us. This is a great pity! Any work (essays, case presentations etc.), can be left at the medical school secretary's office.

We would like to congratulate our ex-fifth-year colleagues who have successfully completed their M.D. course this February, and we would like to wish the best of luck to those who through some mis-fortune, failed to pass all the exams in this session.

This is the last issue which I shall be editing as it is about time to hand over to a new member who will be responsible for the next few issues. The new editor shall be Mr. Rupert Calleja whom, I am certain, will work with the same spirit and enthusiasm as I have done through the first five issues. We hope to see more third and fourth year students showing interest in this magazine which has an established popularity not only with students but more so with doctors and specialists. I have enjoyed each moment as I worked with the editorial board to establish and maintain *Medi-Scope* on firm ground. The end-product of our efforts is most gratifying. I am only too sorry to dismember myself from this journal. I do hope that the magazine will live on as a teaching medium and a further opportunity to learn more about the ever expanding branches of medicine and surgery.

The editorial board has managed to collect a generous sum of money in aid of the handicapped people at *"Id-Dar tal-Providenza"* in Siggiewi. On their behalf, the board thanks all those who have assisted us in fulfilling this aim. We hope that in future other sectors of our society will receive funds raised through the mediation of *Medi-Scope*. Before our donation is made, anyone wishing to contribute his/her share can still do so upto to end of April. The amount collected will be announced later.

THE EDITOR

Contents

Hydranencephaly

SAFRAZ ALI, MBBS, D Path
DEPARTMENT OF PATHOLOGY, ST LUKE'S HOSPITAL

A case report of hydranencephaly is presented with discussion upon morphology and etiology. The diagnosis was made by autopsy examination upon the corpse of a six day old male infant.

Introduction

The term hydranencephaly was introduced for the first time by Spielmeyer in 1904.[17] However, Breschet was the first to describe the condition in 1823.[10] Later in 1929 Cruveilheir described it as anencephalie hydrocephalique.[5] The term hydranencephaly designates a congenital malformation of the central nervous system in which cerebral hemispheres are reduced to fluid (C.S.F) filled membranous sac with a relatively normal sized cranium.

Case report

Six days old male baby was admitted to St Luke's Hospital with inertia, bradycardia and hypothermia. Suction and muscle reflexes were absent. Pupils reacted to light. The baby developed arrhythmia and died on the same day.

The baby was born to a twenty-year-old primigravida after 38 weeks of gestation. Mode of delivery was through forceps application. Post mortem examination revealed the corpse of a baby weighing 2 kg, the head circumference being 30 cm. Cracial cavity contained a fluid filled sac weighing 15 grams. The fluid was clear. No cerebral hemispheres were seen. Cerebellum was present. Spinal cord was developed normally. Carotid and basilar arteries were present. There were no other developmental abnormalities. Microscopic examination of the membranous sac revealed gliosis and no neurones were seen. Sections of cerebellum and spinal cord revealed normal structure. The lungs showed oedema, congestion and pneumonia.

Etiology

The condition may develop any time between third month to full term utero.[7] There have been only two cases reported wherein the condition developed post natally. Benda, in 1945, described a case in which the child developed hydranencephaly at the age of two years. The other case is described by Smith in 1967 were hydranencephaly developed subsequent to meningococcal meningitis in a child at the age of two years.[18]

Several etiological factors have been proposed in causation of hydrancephaly. Prenatal occlusion or agenesis of carotid artery and jugular vein was considered as essential pathogenetic mechanism in hydranencephaly by Norman in 1958.[15] Myer in 1968 and 1969 produced hydranencephaly in monkeys by bilateral carotid and jugular ligation an by incomplete placental abruption.[13,14] Wildi,[20] Haymaker[9] and Booth[3] considered Herpes Simplex and other viral encephalitides to be responsible for production of hydranencephaly in utero.

Halsey[7] in 1971 considered infective process to be responsible for liquefactive necrosis of the brain and thus production by hydranencephaly. Despite the fact that Miller in 1963[12] and DeMyer in 1963[6] found chromosomal abnormalities and suggested a causative contribution in production of cerebral malformation, there have been only three recorded familial instances where hydranencephaly developed in twins.[8,16,17]

After perusal of the literature all these suggestions seem to be valid and well documented. Therefore one may conclude that hydranencephaly results from a variety of etiological factors and that pathogenetic mechanism may not be the same in all cases destructive process following vascular occlusion or agenesis, and infective process being the most likely pathways leading to production of hydranencephaly.

Morphology

Morphologic changes occur in the brain and eyes mainly. Cerebral atrophy, thalamic atrophy and absence of medullary pyramids are common to all cases. Cerebellar abnormalities, though not a constant feature, are a common occurence.

The histologic structure of the hydranencephalic sac is usually predominantly astrocytic. It is usual for the ependymal lining to be lost in encephaloclastic process, though occasionally some fragments may be preserved. Neurones are absent. Absence of neurones is an important distinguishing feature, albeit posthumously, from severe hydrocephalus.

Changes in the eye consist mainly of gliosis of the inner retinal layer, complete absence of the ganglion cells, rods and cone cells and absence of retinal vessels.

There are only two instances where hydranencephaly was associated with changes in organs other than brain and eyes. Halsey et al[7] reported a case in 1971 in which hydranencephaly was associated with polycystic kidneys. The other instance was reported by Bauer et al[1] in 1977 when they reported a case of hydranencephaly in association with gyrated scalp.

Diagnosis

Clinical features include hypothermia, bradycardia, physical inertia, feeding difficulties, incoordinated eye movements, light reflex may be absent, strabismus and nystagmus. Occasionally, jaundice is present. Positive transillumination of the cranium is always obtained. Diagnosis can be made antenatally by ultrasound.[19]

Prognosis

Prognosis in hydranencephaly is extremely poor and dependent upon the integrity of hypothalamus, corpus striatum and brain stem tegmentum. Most of the patients die within first month of life. Longest survival has been reported by Crome and Sylvester[4] when the child lived up to $4^1/_2$ years. However, the child had spasticity, convulsions and blindness.

An interesting case has been reported by Lorder[11] where a child with hydranencephaly had a relative normal development. The author fails to furnish any valid explanation for this phenomenon and admits this case to be an enigma.

Summary

Hydranencephaly is a congenital condition in which cerebral hemispheres are replaced by a thin sac containing C.S.F. The sac wall consists of a pia and arachnoid overlying the glial layer. The condition may result from a variety of developmental or destructive abnormalities any time in utero, e.g. vascular occlusion or agenesis of carotid artery and jugular vein and infections. Atrophy of cerebral hemispheres, atrophy of thalamus and absence of medullary pyramids are characteristics of all cases.

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Intraventricular and Periventricular Haemorrhage in the Newborn —

A Frequent Early Complication in the Low Birth Weight Infant

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Intraventricular Haemorrhage (IVH) is the most important single event in the early neonatal period which imparts such a catastrophic effect on the prognosis of the ill preterm, both as regards mortality as well as morbidity. A number of aetiological factors are concerned in the causation of this complication; a few, at least, are preventable to a certain extent. Various workers in the field have over the past few years thrown new light on the pathogenesis and diagnosis of this problem, to a degree which begs a reappraisal of the concept.

The increase incidence of subependymal and intraventricular haemorrhage in preterm infants has been related by pathologists to the structure of the immature periventricular subependymal area and its vascular system, particularly the distinctive anatomy of the subependymal germinal matrix, the maturation-dependent vascular supply to the region, the structure of the periventricular capillaries, the regulation of cerebral blood flow, systemic haemodynamic events and fibrinolytic factors within periventricular cerebral tissue. (Volpe, 1982). The contribution of parturition events were studied by Bejar et al (1981) who utilised serial realtime ultrasound scanning of the brain through the anterior fontanelle; uterine contractions were shown to cause sufficient deformations of the preterm pliable skull to cause an increase in intravascular pressure resulting in peri/intraventricular haemorrhage. Further support has been rendered by Horbar et al (1981) who reported on the decrease in incidence of haemorrhage in infants of mothers who received tocolytic agents.

Dykes et al (1980) analysed 151 preterms under 35 weeks of gestation and who required intensive care for more than 24 hours; diagnosis of haemorrhage was confirmed by CT scan, ventricular tap or autopsy. Intracerebral haemorrhage was noted to be positively correlated with a number of factors. 42% of the 151 infants developed an IVH, and the associated factors of importance have been tabulated below.

Alveolar rupture associated with a	74%
incidence of IVH PaO_2 under 30 Torr	69%
PaO_2 under 50 Torr	48%
$PaCO_2$ over 50 Torr	49%
Intermittent mandatory ventilation	52%
Peak Insp. Pressure over 25 cm H_2O	70%
I:E ratio more than 1:1	66%
Severe Hyaline membrane disease	59%
Patent ductus arteriosus	62%
$NaHCO_3$: after 24 hours of age	60%
Volume expansion: under 24 hrs of age	54%
Mean arterial pressure under 25 mmHg.	59%
Light for gestational age	50%

(I Inspiratory E Expiratory)

The *single* most important associated factor was alveolar rupture which occurred in 26% of the infants studied; Hyaline Membrane Disease (HMD) occurred in 85% of the infants with alveolar rupture, and the incidence of bleeding in this group rose to 82%, so that the onset of alveolar rupture in HMD would contribute heavily towards the likelihood of the development of an intracerebral and a much poorer prognosis.

Among the factors which Bejar et al could not positively correlate with an increase in incidence of IVH were actual birthweight and gestatioal age, male sex, low Apgar score (below 5), osmolality exceeding 300 mmol/l, sodium exceeding 150 mEq/l, CVP over 6 cm H_2O, and PEEP over 5 cm of water, unattended delivery, hypothermia, obstetric trauma and coagulopathy; the converse is true, however, with the last-mentioned factor, in so fas as IVH/PVH is an important aetiological factor in triggering off the coagulation cascade in Disseminated Intravascular

Coagulopathy (Cockburn & Drillien, 1974).

Although other workers suchs as Lipscomb et al (1981) and Hill et al (1982) have also correlated pneumothorax with PVH/IVH Cooke et al (1981) were not able to confirm this association, and have alternatively suggested that endotracheal tube blockage is the important aetiological factor by causing hypoventilation and consequent hypoxia and hypercapnoea with resulting cerebral vasodilation this with a relief of the tube obstruction and consequent improved ventilation, the increased cardiac output would rupture the dilated cerebral vascular bed and cause an IVH/PVH. Recent work has demonstrated the pathogenic importance of impaired vascular autoregulation in the human premature. (Volpe, 1979).

Present data in fact suggest that sharp increases in blood flow are dangerous because of the pressure passivity of cerebral blood flow regulation in the sick preterm.

Lou et al, 1979; Goddard et al, 1980; Fujimara et al, 1979). Goldberg (1980) had also demonstrated the importance of rapid volume expansion in the pathogenisis of IVH, whilst Milligan (1980), using jugular venous occlusion plethysmography, has shown that transfusion-induced elevations of blood pressure resulted in striking increases in cerebral blood flow causing an IVH with 12 hours. Severe pulse waves to the premature brain may also be produced by a patent ductus arteriosus (Periman, 1981; Lipman, 1982); such changes would play a major role in the pathogenesis of IVH.

The Doppler technique has recently been adopted in IVH/PVH to confirm the increase in cerebral blood flow velocity associated with a rise of blood pressure soon after alveolar rupture and pneumothorax (Hill, 1982). Other conditions associated with temporary elevation of blood pressure in the preterm include simple motor activity, handling, seizures and apnoeic attacks; prophylactic measures to offset these occurrences may present some difficulties, but the importance of a minimum of handling, blood sampling and other investigations and interventions, needs to be particularly stressed to enthusiastic medical staff. *Primum non nocere* is particularly applicable in this case.

Diagnosis of IVH/PVH is based first of all on the identification of the clinical setting, i.e. essentially any preterm infant in a special care baby unit; lumbar puncture initially reveals the presence of large numbers of RBCs and elevated protein, and later this is followed by xanthochromia and diminished glucose. Confirmation is made by CT scan or ultrasonography. (Volpe, 1979).

The study of Tsiantos (1974) on autopsied infants had timed the occurrence of PVH/IVH to the second day of life. The more wide-spread use of realtime ultrasound scanning of preterms in Special Care Baby Units has recently disclosed the very high percentage of bleeds which occur in preterms in the first day of life, with an average age of 6 hours; whereas CT scan had yielded a 45% incidence of this complication in preterms, ultrasound scan has detected a bleed on 90% of infants under 34 weeks of gestation. (Bejar et al, 1980). 49% of these haemorrhages are large, and some bleeds are detected as early as 15 minutes of age; resolution may take as long as three months. CT scanning, however, still remains the choice investigation for parenchymatous lesions, subdural haemorrhages or collection and lesions in the posterior fossa.

Since most haemorrhages originate at the level of the head of the caudate nucleus, which is nearly directly below the anterior fontanelle, a coronal view with the linear array ultrasound transducer is particularly useful for the detection of haemorrhages within the the germinal matrix at this site. Sagittal views are useful for the demonstration of the extent of the bleed; whilst both views complement each other in the estimation of ventricular size.

A recent study by de Crespigny (1982) has yielded results similar to those of Bejar et al (1980); 65% of infants weighing less than 1500g who developed an IVH/PVH did so in the first 5 hours of life, thereby enhancing the probable aetiological role of labour events in pathogenesis. Parturition events themselves are the topics of current research in the investigation of the sequence of events leading to these haemorrhages in low birth weight infants, and, in particular, the hypothesis is being studied that a bleed may already be present at or very soon after birth, whilst postnatal events such as alveolar rupture would actually cause an extension rather than the initiation of such a bleed (Hill, 1982). Ultrasound scanning assumes great importance in these instances by detecting small bleeds very early on and alerting the neonatalogist to the risk of extension. With an established haemorrhage, serial scans and OFC (Occipito-frontal circumference) measurements are invaluable in diagnosing and following the progress of complicating hydrocephalus and the effectiveness of therapeutic lumbar punctures and ventricular taps through the assessment of the cerebral mantel and ventricular size. Volpe (1979) shown that the initial presentation of posthaemorrhagic hydrocephalus is progressive ventricular dilatation with normal intracranial pressure. Fifty per cent of these resolve, without treatment, withing a month, on average. With the remaining cases of normal pressure hydrocephalus who continue to manifest ventricular dilation the treatment of choice at present is serial lumbar punctures or drugs which decrease CSF production, i.e. acetazolamide, frusemide or glycerol. If there exists no adequate patent communication between

the lateral ventricles and the lumbar suberachnoid space, or if there is raised intracranial pressure, ventricular drainage is required if irreversible brain damage is to be prevented. A temporary external ventriculostomy can be left for a week, but may later be repeated, and allows the infant to grow and attain better health to withstand a more permanent ventriculo-peritoneal shunt.

Donn et al (1981) have utilised US scanning to monitor the effectiveness of phenobaritone in the prophylactic measurement of IVH/PVH, and a beneficial effect has been demonstrated on a dose of 5mg/kg/day for seven days, after a 10mg/kg bolus on admission. An incidence of 13% of haemorrhages in the treated group contrasted remarkably with the 47% incidence in the untreated infants. This effect has been confirmed clinically in our SCBU where phenobarbitone is routinely administered to preterm infants in the first week of life, with the added advantage of enhancing hepatic glucuronation which is immature in these infants. We have also observed a decreased incidence or attenuation of apnoeic attacks in these infants on phenobarbitone and a study is under way to establish a possible relationship between intracerebral haemorrhage and apnoeic attacks, particularly in preterms under 34 weeks of gestation. Morgan et al (1981) have failed to confirm Donn's findings when they used intramuscular phenobarbitone as a bolus (20mg/kg) in the first few hours of life; neither have they been able to obtain significant prophylactic benefit from Etamsylate which is thought to reduce capillary bleeding by polymerising hyaluronic acid, thereby reinforcing the basement membrane of the capillary; it is also thought to increase platelet adhesiveness.

The field still provides very fertile soil for further research, but these recent developments in the pathogenesis and diagnosis of IVH/PVH must of necessity modify the lines of its management, and the preventive aspect in particular needs to be stressed. Protection of the perterm's pliable skull against excessive uterine contraction, prevention of premature delivery, the acquisition of a good L:S ratio prior to delivery and the careful prophylactic use of betamethasone are primarily the concern of the obstetrician, but further postnatal responsibility needs to be extended to the neonatologist who should avoid therapies which can cause sudden increases in cerebral perfusion; he should also utilise minimal mean airway pressure to achieve normal tidal volumes and optimal lung compliance in ventilated infants, and ensure a correct I:E ratio. Volume expanders and sodium bicarbonate are to be used with great caution and within the desired recommendations, particularly in the first two days of life. Serial blood gas analysis is required to monitor oxygen and carbon dioxide tension in order to avoid hypoxia and hypercapnia. The maintenance of an adequate mean arterial pressure and the use of prostaglandin synthetase inhibitors in the management of PDA (Patent Ductus Arteriosus) present special problems to the neonatal team, who should not at the same time forget to keep handling to a minimum. The prophylactic use of phenobarbitone is recommended.

The gravity of this complication places the affected infant at great risk of developing psychomotor retardation, so that follow up of survivors extends well beyond the neonatal period.

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The First Open Heart Surgery in Malta

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Introduction.

Although in 1947 Professor P.P. Debono courageously closed a patent ductus arteriosus in a young patient, with quite good results, the operation itself was really an extended thoracotomy. The first ever open heart surgery to be performed in Malta, on the 8th November 1983, is the one about to be described. Throughout the time the heart was stopped; for over an hour, its work was taken over by a heart lung machine operated by local and foreign technicians, the latter having come over with the surgeon Mr. A. Yates of Guy's Hospital, London, U.K. Besides the aortic valve replacement, the surgeon performed another cardiac operation, a mitral valvotomy on a young woman.

The Patient

A.D. was a 39 year old clerk who wieghed 67kg, was 1.5m tall and had a surface area of 1.8m^2. He was a moderate social drinker and used to smoke over 30 cigarettes daily. Until 20 years of age he felt quite fit but noticed increasing dyspnoea and palpitations expecially after exercise, as after a game of football. His gener practitioner noted the hear murmur but advised only rest and avoidance of heavy exertion. For 19 years the patient carried on his normal life activities but when the palpitations increased and were really troubling him he sought specialist advice, first in Malta and then abroad. A diagnosis of aortic valve stenosis and incompetence with left ventricular dilatation was established by ultrasonography. Before pulmonary hypertension could occur he was strongly advised to undergo operation. Although the patient consented at once, the decision to have the operation locally presented some difficulty as the patient was afraid to be the first one to inaugurate cardiac surgery in Malta. When the visiting surgical team assured the patient that all was prepared he gave his full consent.

The Machines

As open heart surgery was the first ever to be performed locally the Health Authorities had to provide a number of machines at a considerable cost.

A *SARNS Four Pump Heart Lung machine* along with its accompanying heating-cooling unit was procured just in time for the operation. This machine has four roller type external pumps and during the procedure one pump was used to drive blood into the aorta at a pressure of 300 mmHg while the other three were used as suction pumps. A *Spiraflow Oxygenator* was attached to the machine and oxygen at 5 l/min was passed over the blood during the bypass procedure.

Supporting machinery included a Fibrillator and Defibrillator with both Internal and External paddles and a Three Channel Monitor for ECG, and two pressure lines.

The Procedure

The night before the operation the patient received Nitrazepam but he slept poorly and was sedated in the morning with Papaveretum and Scopolamine. In the anaesthetic room he received a sleeping dose of Thiopentone, a medium acting relaxant Pancuronium and a full dose of Fentanyl. He was intubated and at once put on a Manley Ventilator.

An arterial line using a *Hewlitt Packard transducer* was set up in the left radial artery and a reading of 160/80 mmHg registered. Two big venous cannulae were inserted in both internal jugular veins. A temperature probe alongside an oesophageal tube and a Foley urinary catheter were introduced.

The heart lung machine was filled with 3 litres of Ringer Lactate Solution and was set running with the tubes clamped and the temperature adjusted to 38 C. After preparing the patient, a midline sternotomy incision was performed from the suprasternal notch down to the xiphisternum, and the sternum sawn through with a pneumatic oscillating saw. After a routine inspection the pericardium was opened, in such a manner that the operative field was seperated from the lungs.

Heparin in a dose of 18,000 units was now administered. Through a probe individual chamber pressures were taken and gave the following readings: right ventricle pressure 27/0, left ventricle pressure 160/65 and aortic pressure 110/60. An arterial blood sample was sent to the laboratory for blood gases estimation: pH 7.4, pCO_2 33mmHg and pO_2 139 mmHg. Silastic tubing was introduced through the

right atrium and through the aorta, care being taken to void out any remaining air in the tubes before clamping them. A purse string secured both tubes in place.

The paddles of the fibrillator were now applied and the heart was fibrillated. The silastic tubing was connected to the machine and pumping started. The aorta was clamped just below the tube site and slowly slit open to reveal the grossly diseased valve. One of the cusps was totally replaced by vegetations and hard calcified material giving a peculiar scraping sensation. The coronaries were flushed by a cardioplegic solution containing Magnesium, Potassium and Lignocaine (St. Thomas Formula) diluted in 1 Litre Ringer Lactate and this cold solution was poured slowly over the heart to lower the temperature and prevent any ischaemic damage during bypass time. Half a litre of blood was now withdrawn from the aortic cannula and stored for administration at the end of the procedure. This is done for postoperative administration of platelets and clotting factors.

After dissecting out the diseased valve and measuring the space so opened, a Carpentier Edwards aortic biosynthetic valve size 27 was chosen and carefully sutured in place employing interrupted and continuous sutures. When the valve was well and evenly anchored in place the sutures were tightened and the stem of the prosthesis was broken off. The aortic incision was hermetically closed. Throughout this procedure the heart-lung machine had been pumping blood at 4.4 litres per minute calculated at 2.5 l/m^2 surface area/min at a pressure of 300mmHg and a temperature of 38 C.Gradually the cardioplegic solution was shut off, rewarming started, and the aortic clamp was released slowly. Suction from the left ventricle was gradually stopped, and the 500 ml blood removed before opening the heart was run in again.

Gradually a return to normal of the cardiac pressures was achieved by increasing the preload and at the same time decreasing the shunting of blood to the machine by slow clamping of the venous cannula. When the right atrial pressure had risen to 10mm Hg the tube was clamped and then removed, and the atrial defect oversown carefully. The aortic tube was now removed and the aortic slit carefully sutured. A second half litre of packed cells blood was now given to make up for the blood volume remaining in the machine.

Despit rewarming, massage, adrenaline (1:10000 solution) cardiac injections and repeated defibrillations at 15 Joules and even 35 Joules, the heart failed to establish sinus rhythm and the measured aortic pressure remained unsatisfactory. Verapamil 40 mg dose I.V. and a drip of Isoprenaline I.V. were without effect on the unstable heart and only Amiodarone 50 mg slowly I.V. achieved a satisfactory output pressure of 80 mmHg and a regular heart rate at 120 per minute.

Two wide bore drainage tubes were put in place one at the bottom of the pericardial sac and the other lying across the heart up to the level of the base of the aorta. The pericardium had to be left open in its upper 2/3 as any attempt at suturing and so tightening the heart was immediately accompanied by arrythmias. Continuous under water suction was applied through these tubes, utilising a *Roberts pump*. The left atrium was cannulated anew and the tube lead out of the chest wall and connected to a monitor for continuous recording. A sample of arterial blood withdrawn at this stage gave the following results: pH 7.4, pCO_2 36.7mmHg, pO_2 97 mmHg, Na+ 130 mEq/l and K+4.8 mEq/l.

Protamine sulphate was now given slowly I.V., the dose calculated at 200 mg/m^2 surface area- a total dose of 400 mg. The exact dose was, however, determined by withdrawing blood samples and estimating the activated clotting time using a Haemochron device till the previously doubled time returned to a control level of 140 seconds.

The sternum was closed using six wire sutures and subsequently both the subcutaneous tissues and the skin were closed seperately. The patient was constantly ventilated using 3 litres Oxygen flow and 6 litres Nitrous Oxide flow per minute. Ventilation was stopped and Oxygen reduced to a minimal flow of 2 litres per minute during the bypass procedure. During the transfer to the ITU the patient was aided to breathe by an Ambu-bag using only Oxygen at 5l/min but once in bed an Air/Oxygen mixture was used for ventilation until *weaning off* was started some hours later, the E.T. tube being removed after six hours in the ITU. No drugs were used for reversal of muscle relaxation.

The total urine output during the bypass was 150ml of dark urine but this increased during the stay in the ITU as thiazide diuretics were administered, cardiac output improved. The antibiotic used was Cephaloridine 1g, given at the end of the procedure. Pain was allayed by analgesics and as the prosthesis used was a biological one no anticoagulant therapy was used but antiagglutination drugs — Persantin and aspirin were administered. The patient was transfered to a general ward in 24 hours and was out of hospital a week later.

Conclusion

It is the authors' sincere hope that this operation and its successful outcome will encourage more Maltese cardiac patients to come forward and have their operation done at St. Luke's Hospital, at first by a visiting caridothoracic team but eventually by a Maltese cardiothoracic surgical team. This will not only justify the enormous expense incurred in procuring this costly apparatus but will instill a certain pride of achievement by the medical profession similar to that experienced by Professor P.P. Debono 36 years ago.

Diabetic Ketoacidosis

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Introduction

The diabetic-related comas listed in order of their probable frequency are;

1) Hypoglycaemic coma.
2) Diabetic Ketoacidosis
3) Hyperglycaemic Hyperosmolar non-ketotic coma.
4) Lactic acidosis.

Whilst in general, diabetic ketoacidosis occurs in patients with the insulin-dependent form of the disease, and the syndrome of hyperosmolar non-ketotic coma is commoner in the elderly diabetic, it is not uncommon to meet a combination of these disorders in a variable degree of severity in the same patient.

This discussion, taken from studies and accounts by leading investigators including Foster and McGarry in the U.S.A. and Alberti, Johnston and Owens in England (see chart) wll concentrate on diabetic ketoacidosis. Although as stated above a combination of diabetic comas can occur, in general, Insulin Dependent Diabetes Mellitus (I.D.D.M) patients ordinarily do not progress to marked hyperosmolar coma, but hyperosmolar non-ketotic coma can develop in a patient with a Type I diabetes.

Diabetic Ketoacidosis is a common illness among diabetics, especially Type I patients, in whom it still has a mortality rate as seriously high as 6-10%. Death may be due to:

(a) Derangements that are directly attributable to ketoacidosis.
(b) Complications associated with the illness itself.
(c) Abnormalities induced by the treatment.

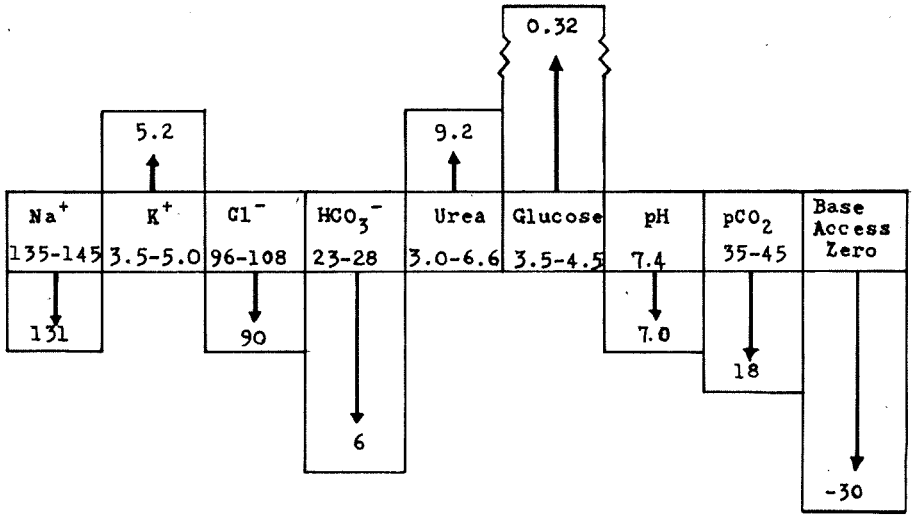
Since a substantial amount of deaths is probably preventable if appropriate treatment is applied, sound education on this disorder is crucial.

Metabolic Derangements

Among the many effects of uncontrolled diabetes, two interdependent processes are of primary importance:

(i) Alterations in glucose production and disposal, which cause osmotic diuresis, volume depletion and dehydration;

(ii) Accellerated ketogenesis which causes metabolic acidosis.



Plasma (Serum) osmolality averages 320 (normal range 280 - 300).

Potassium

Total body stores of potassium are depleted in D.K.A. patients on admission. Potassium concentrations in plasma however tend to be high initially, decreasing once therapy is started because of extended losses and potassium returns to cells as acidosis is reversed and normal glucose metabolism is restored. The degree and rate of replacement should be guided by direct measurements of the cation in plasma and E.C.G. monitoring.

Bicarbonate

Administration of bicarbonate is the controversial practice. It seems wise to reserve bicarbonate only for very severe acidosis i.e. when the pH is less than 7- and stop administration when the pH reaches 7.2, in order to avoid rebound metabolic alkalosis as ketone bodies are metabolised.

Phosphate

Phosphate depletion in D.K.A. is often severe. As is the case with potassium, initial plasma values may be low or high because of trans-membrane shifts in distribution of the ion, and hypophosphataemia can easily occur once treatment has begun. Although some studies seem to indicate that phosphate infusion has no effect on the course of diabetic ketoacidosis, nevertheless if pretreatment phosphate levels are low, it may appear reasonable to consider administering potassium phosphate (when potassium is also needed) to avoid extreme hypophosphataemia.

Insulin

Insulin is required for the treatment of D.K.A.. It lowers the plasma glucagon level, counteracts the effect of glucagon on the liver, inhibits the flow of fatty and amino acids from the periphery, and enhances glucose use in target tissues. In recent years, investigators have been concerned primarily about the amount of insulin that should be given, with a general trend moving from the high dose to the low dose regimens.

Although most patients are found to respond adequately to low-dose regimens, some patients with D.K.A. require large amounts of insulin to reverse the illness-this being thought to be due to insulin resistance operating in D.K.A.

-i- The rarer type, which could be called "prereceptor-receptor resistance" this being caused by antibodies to insulin, high concentrations of stress hormones, intrinsic abnormalities of the insulin receptor, or a combination of these factors. It can only be overcome by achieving high concentrations of insulin in plasma (and interstitial fluid).

-ii- The commoner type of insulin resistance in D.K.A. is probably caused by defects in intracellular metabolism that are beyond the receptor level-"post-receptor-binding resistance" - a deficiency in glucose transport units being suggested as the cause. Low doses usually suffice in these cases, where the resistance is not generally overcome by increasing the concentration of insulin in plasma.

Regarding insulin therapy some salient points should be made:

(I) Volume depletion and vascular collapse may impair the absorption of insulin injected intramuscularly, and therby delay the response in some patients-in such cases i.v. insulin is recommended.

(2) If the low-dose schedule is to be followed, at least 6-10 units per hour (for adults) should be given; and if there is no fall in the level of ketones or increase in pH within 3-4 hours after start of therapy, large doses of insulin should be given without delay.

(3) Because plasma glucose levels often fall before the reversal of ketogenesis, glucose should be infused as necessary during treatment to avoid hypoglycaemia, but insulin should **not** be slowed or stopped when the acidosis or ketosis are still present simply because the glucose level is falling.

Complications

Shock

Vascular collapse in D.K.A. is ordinarily due to a combination of profound volume depletion and acidosis. It usually responds to treatment with fluids and bicarbonate. If the response is not satisfactory, gram-negative sepsis or silent myocardial infarction may be present. If shock persists, blood should be given.

Infection

Infection is a common accompaniment of decompensated diabetes hence a careful search for this should be made in every patient particularly if fever is present. Pneumonia, pyelonephritis and septicaemia are the commonest problems. A rare infection, peculiarly associated with D.K.A. is mucormycosis, and death my follow in 10 days if treatment with amphotericin B is delayed.l.

Vascular Thrombosis

Many features of D.K.A. (including dehydration, contracted intravascular volume, low cardiac output, increased blood viscosity, underlying atherosclerosis and other haemostatic changes) favour thrombosis. Although this may occur in any muscular artery, cerebral vessels seem particularly susceptible especially when plasma osmolality is high.

Cerebral Oedema.

This is a rare but generally fatal complication of D.K.A. occuring usually in children, and developing several hours after the start of therapy. The cause is unknown, and various factors including osmotic disequilibrium, insulin, hypoxia and a rapid decrease in plasma oncotic pressure have been incriminated. Mannitol appears to be the only effective treatment, although dexamethasone is usually administered simultaneously. Passive hyperventilation seems a reasonable adjunct to therapy. Even in the uncommon cases where the patient survives, permanent brain damage may result.

Diabetic ketoacidosis is generally initiated by decreased insulin therapy or by 'stress' that renders the normal insulin inadequate. In either situation glucagon concentrations rise, and the ratio of glucagon to insulin increases, usually with concomitant increases in the levels of adrenalin, noradrenaline, cortisol, and growth hormone. In the periphery a catabolic state is produced, with mobilization of substrates that are used by the liver for synthesis of glucose and ketone bodies.

In the liver the enzymes that carry out these processes are simultaneously activated. Cyclic AMP activates glycogen phosporylase, which stimulates the breakdown of glycogen; at the same time glycogen synthesis is inhibited. Most other hepatic events are related to the glycogen-induced fall in the level of fructose 2,6-diphosphate which increases the liver capacity for gluconeogenesis through disinhibition of fructose biphosphate and which blocks glycolysis with the subsequent impairment in the formation of Malonyl-CoA. The fall in the level of Malonyl-CoA in turn activates the oxidation of fatty acids which leads to increased production of acetoacetic and beta-hydroxybutyric acids. The end result is hyperglycaemia and ketoacidosis.

Clinical Manifestations

Presentation

Classically, patients in Diabetic Ketoacidosis (D.K.A.) present with vomiting, thirst, polyuria, weakness, altered sensorium and air hunger. Abdominal pain and other symptoms are less frequent.

The commoner precipitating factors are cessation of insulin intake, infection and/or emotional distress, but in many cases no evident cause can be detected.

On physical examination, patients tend to have tachycardia, but blood pressure is usually normal. Although an elevated temperature strongly suggests infection, the latter may still be present even if the temperature is normal. Kussmaul's respiration is frequently present, however the ventilatory rate may fall with seven acidosis, and thus respiration may paradoxically increase after treatment is initiated.

Regarding the term 'Diabetic coma' only about 10% of the cases are actually unconscious and as many as 20% have no discernable clouding of consciousness beteen these extremes, the spectrum ranges from drowsiness to precoma.

Although there are no specific findings in D.K.A., concurrent illnesses may leave their characteristic signs.

Laboratory Findings.

Although the average concentration of plasma glucose in D.K.A. is about 500 mg/dl., levels range from near normal to the extreme concentrations that are characteristic of hyperosmolar coma.

Volume depletion (the severity of which probably determines the degree of hyperglycaemia) is usually moderate, with plasma levels of urea nitrogen in the range of 25 to 30 mg/dl.

Metabolic acidosis is due primarily to the accumulation of acetoacetic and beta-hydroxybutyric acids in the plasma, and plasma acetone (derived from non-enzymatic deoxycarboxylation of acetoacetate). Levels are very frequently high on admission-in the range of 5mMol.

The plasma level of sodium tends to be low despite a modest increase in osmolar concentration, because glucose, in the absence of insulin, is osmotically active across the cell membrane. Avery low sodium level (Less than 120 mMol./L) is usually due to severe hypertriglyceridaemia, which can be recognized from visual inspection of the plasma (presence of fat) and for ophthalmoscopic examination (lipaemia Rentinalis).

The initial potassium concentration in plasma tends to get high because of metabolic anicdoisis, but may be normal or even low depending on the duration of illness and previous nutrition.

The only important abnormality in the rountine blood could and in the picture is leukocytosis.

Treatment

The management of this disorder consists basically of:

(a) Establishing the diagnosis.
(b) Assessing fluids and electrolytes.
(c) Administering Insulin.
(d) Keeping a fluid chart.

Fluids

Both depletion of extracellular fluid volume and dehydration occur because of the osmotic diruesis induced by glucose, and both electrolytes and free water are needed in the therapy. Most investigators favour 0.9% saline, reserving half-strength (0.45%) saline if hypernatraemia supervenes; some authorities consider lactated Ringer's solution in cases of hyperchloraemic acidosis. The rates of administration of replacement fluids depends on the degree of dehyration, body weight, urine output and clinical response.

The fluids are meant for the repair of the circulation and renal function; they also lower the plasma glucose level (even in the absence of insulin), by enchancing glucose excretion via increased urine flow and by decreasing levels of counter regulatory hormones that stimulate hyperglycaemia.

In contrast to glucose levels, concentrations of acetoacetate and beta-hydroxybutyrate do not fall when fluids are given without insulin.

The free water deficit needs to be reversed only after extracellular fluid volume is restored; solutions containing glucose (e.g. 5% glucose) or 0.45% saline being appropriate vehicles.

A Study of Uterine Fibromyomas

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ABSTRACT

This study consists of 210 cases of fibromyomas confirmed histologically, collected over a period of two years. An attempt has been made to study the symptomatology and effects of these tumors in order to see how far the findings in this series agree with accepted norms. Some important correlations and variations from reported findings in the literature have been highlighted.

Introduction

Uterine Fibromyomas (Fibroids) are the commonest of all pelvic tumours. It has been stated that 20 per cent of all women have one or more fibroids at death (1). These tumours are slow growing and often symptomless. When symptoms such as menorrhagia occur, they do so in women between the ages of 35-45 years. Uterine fibroids are supposed to be commoner in nulliparous or relatively infertile women. They have been shown to bind oestradiol in greater amounts than does the normal endometrium (2). It is often said that excessive oestrogen stimulation promotes the growth of fibroids. As such, fibroids have been associated with conditions such as follicular cysts of ovaries, endometriosis, and endometrial carcinoma (3).

Material and Methods

This study consists of 210 cases seen at St. Luke's Teaching Hospital in Malta over a period of two years. All the patients underwent surgery, and the diagnoses were confirmed histologically. An attempt has been made to study the prevalence, symptomatology and the effects of fibroids on these cases. The authors have also tried to find out how far commonly accepted features and associates of uterine fibroids correlate with this series. The degenerative changes of the tumours, within the limits of histological examination of specimens, were also studied. The retrospective studies were carried out by referring to old case records, and follow-up of operated cases.

Findings

Prevalence: Fibroids at St Luke's Hospital were found to be the most common genital tract neoplasm. In fact, from 250 cases of genital tract neoplasms picked at random and confirmed histologically, 52% were fibromyomas (Table 1). This high frequency conforms to the expected pattern in other parts of the world.

Age: The age distribution followed the expected pattern (Table 2), the commonest age group between 40 - 44 years. The categories comprising the lowest distribution were from the two extremes of ages.

This may reflect incidental diagnosis, attempts at management without surgical intervention, or the patients being symptom-free, especially in the younger group. An arrest or slowing of activity is very likely after the menopause through at least 10 per cent continue to grow (3). This may be another reason for the low prevalence in this age group.

Parity: In this study 73.8 percent of the cases were parous, while only 26.2 percent were nulliparous (Table 3). Of the former group, 66.4 percent had less than three children, while 33.6 percent had four or more. In the nulliparous group 59.6 percent of the women were unmarried. The married nulliparous groups comprised only 11.4 percent (21 cases) of the total number of cases. In this group only three women gave a history of miscarrages, some of which were never confirmed. The incidence of nulliparity in relation to fibroids was significantly low in our series. This is similar to the study done by Kurman and Norris (4).

Haemoglobin levels: A very important aspect of this study was the haemoglobin levels of patients, which should have reflected problems of bleeding. A borderline value for anaemia was taken at a haemoglobin level of 10g/dl. conventional trends were inapparent in this study. For instance only 20.4 percent of cases were anaemic. These had a hypochromic microcytic blood picture. Fifty percent of these had haemoglobin levels less than 9g/dl. Although the majority of women had problems with menstruation such as prolonged bleeding, only a minority were really anaemic. Of this small group, an interesting feature was that 81.3 per cent were over the age of 40 years. Perhaps this reflected higher parity, or delay in coming to hospital for treatment. Although it is said that manifestations of anaemia commonly result from menorrhagia (3), this was not apparent in this series.

Situation of tumours: The situation of the tumour mass did appear to influence the presenting symptoms (Table 5). Intramural fibromyomas were commonest in the group presenting with menorrhagia. Multiple fibromyomas were common, and sometimes distributed in all layers of the same uterus. While only 9.3 per cent had submucous fibroids. As it was difficult to determine the size of some tumours in the retrospective studies, it was not possible to obtain an accurate correlation of size to symptoms or anaemia. Some tumours are said to grow to an incredible size, without producing any appreciable symptoms, unless there is compression of adjacent organs or structures (6). The potential of

these tumours to grow without producing symptoms was exemplified by one case in this series. A fibroid uterus weighing 9 kg was removed at operation. The 52 year old patient presented to the hospital complaining of abdominal swelling. A history of irregular menstruation with periods of amenorrhoea was only elicited on direct questioning.

In the prospective group, symptoms such as backache and lower abdominal pain were found to be commoner in cases who had large subserous fibroids.

Associated conditions: Associated malignant and non-malignant condition in relation to fibroids were also studied. While malignant conditions were insignificant in our series, certainly there were non-malignant associations. Of these, cystic changes in ovaries and adenomyosis were predominant. In this series all the ovarian cysts were of the simple non-malignant type, and were seen in 19.9 percent of our cases. Adenomyosis and endometriosis considered together, were found on histological examination in 20 percent of cases. Both associations were significant, and have been reported in other series. However, when two conditions with a high incidence are correlated, their co-existance may be fortuitous[3].

Degenerative changes in fibroids: Degenerative changes in fibroids are a result of interference with the capsular circulation. In this series, the fibromyomas were found to have undergone degenerative changes in a little less than half of the cases (Table 6). The commonest was hyaline change which occured in 28 per cent of cases, a frequency which is in keeping with the normal trend[3]. Sarcomatous change was noted in 2.3 per cent, a figure which was higher than the reported normal of 0.2 per cent (5).

Operative procedures: The majority (87.3 percent) of patients in this series underwent a total abdominal hysterectomy with or without bilateral salpingo-cophorectomy (Table 7). A vaginal hysterectomy and pelvic floor repair was performed in 6.1 percent of the cases. These latter cases had fibroids which were equivalent to less than 12 week uterine size, and required pelvic floor repairs. In a few of these, the fibromyoma was an incidental finding. In 14 patients (6.6 percent)1, a myomectomy was carried out. Half of these patients were 35 years of age or less and included 2.5 percent who were single. These figures reflect the general management of fibroid cases at St. Luke's Hospital, where myomectomy is attempted in the younger patient still wishing to have children. It has been shown that recurrance after myomectomy is a distinct possibility occuring in 2.3 percent of cases. this occurs because the stimulus to regrowth is maintained, or because seedlings are overlooked at the time of operations. The tendency to the formation of fibroids in the uterine tissues is a pssing phase and not a continuing defect, for otherwise recurrence after myomectomy would occur more frequently than reported. It has been suggested[7] that myomectomy performed before the age of 30 years is more likely to be followed by a recurrence.

Discussion

Fibromyoma is the commonest tumour in the human body. The aetiology is quite unknown, though oestrogens have been incriminated. In view of their frequency, the symptomatology and effects of fibroids are well documented. This study of 210 cases highlights some important correlations and variations from reported findings in the literature.

Fibroids have been said to be the rewards of virtue, and babies the fruit of sin[3]. This tumour was not commoner in the nulliparous women. In fact only 26.2 percent belong to this category. The tumour did no seem to affect the abortion rate to a significant degree in either group. In the parous group however 30 percent had1 one or more miscarriages, but the majority of them had two or more children. This variation compares with women of Negro origin who develop myomata when young and despite having had children[1]. Of the 14 patients who underwent myomectomy with an aim of preserving their reproductive function, none became pregnant. The follow-up period has however not been long enough, being only of two years in some cases. The pregnancy rate after myomectomy has been reported as 38 percent[7]..

The majority of patients presented after the age of 40 years with a variable symptomatology. The majority complained of menorrhagia. In some cases, we were unable to elicit the duration of this symptom from the case notes alone. However 62.7 percent of patients in the anaemic group presented with menorrhagia. Perhaps this reflected adelay in referral to hospital. Manifestations of anaemia have been said to commonly result from menorrhagia[3]. This study however indicates that the majority of patients who presented with menorrhagia had a haemoglobin level above 10g/dl. Some cases who were subjected to attempts at correction of the anaemia by haematenics and blood transfusions were found to be resistant to therapy. These cases benefitted from timely surgical intervention, which broke down a vicious cycle. In fact some of our cases which were operated on with haemoglobin levels below 10g/dl improved tremendously after surgery. Too long a delay may have brought on adverse consequences.

One expects submucous fibroids to be commoner in patients presenting with menorrhagia and anaemia. However, this series showed submucous fibroids to be just as common in the anaemic as in the non-anaemic group. In fact there appeared to be no significant correlation between the site of tumour and the haemoglobin levels in our series of cases. Unless the uterine cavity is enlarged by submucous fibroids, the heavy periods may be due to the generally increased vascularity of the uterus. In cases where heavy periods are associated with only

small fibroids, the conclusion drawn is that the condition is one of coincidental or associated dysfunctional uterine bleeding[1].

Histological study of the fibromyomas showed that hyaline change occured in 28 percent of the cases. This change caused no added symptoms, but may have contributed to problems of clinical diagnosis. Ultrasound scans together with clinical signs, were sometimes insufficient to distinguish between fibroids and ovarian cysts. Broad ligament fibroids too, posed problems of diagnosis, but were rare in our series. From the four cases that had undergone sarcomatous change, only one had symptoms of abdominal pain in addition to menorrhagia.

Whether or not uterine leiomyomas ever undergo malignant transformation to become leiomyosarcomas is a controversial point [8]. If they do occur, such transformation is rare accounting for 0.2 percent of cases[5]. This series of cases had a higher incidence. Sarcomatous change in a fibroid may be suspected when there is evidence of rapid growth in the tumour in any age. When there is unquestionable postmenopausal growth in a fibroid, sarcomatous change is almost certain. However at least 10 per cent of myomas continue to grow after the menopause[3]. If the sarcomatous change is near the uterine cavity, postmenopausal bleeding may occur. The absence of sacromatous tissue in material obtained at curettage by no means rules out this possibility[9].

The associated incidence of endometrial carcinoma was extremely low in this series (1.4 per cent). This reflected a younger sample group which was investigated and treated for fibroids. A delay in diagnosis and treatment might have resulted in the development of this condition, and hence a greater correlation.

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TABLE 1: **Frequency of genital tract neoplasms** (Random Sample n § 250)

SITE OF NEOPLASM	PATHOLOGY	Number	Percentage
Body of uterus	Carcinoma	31	12.4
	Sarcoma	2	0.8
Non-malignant fibroids	Non-malignant fibroids	130	52.0
	Other non-malignant neoplasms	2	0.8
Cervix	Carcinoma	9	3.6
Ovary	Malignant neoplasms	8	3.2
	non-malignant neoplasms	65	26.0
Vulva and vagina	Malignant and non-malignant	3	1.2
	neoplasms	3	1.2

TABLE 2: **Age Distribution**

Age (years)	Number	Percentage
20 - 29	4	1.9
30 - 39	42	20.0
40 - 49	125	59.5
50 - 59	34	16.1
over 60	5	2.5
Total	210	100.0

TABLE 3: **PARITY IN RELATION TO FIBROIDS** (includes 3 cases of miscarrages)

PARITY	Number	Percentage
Nulliparous (single)	34	16
Nulliparous (married)*	21	10
P 1-3	103	49
P 4-5	31	15
P 6 and above	21	10
Total	210	100

TABLE 4: **SYMPTOMATOLOGY OF FIBROIDS**

PRESENTING COMPLAINT	ALL CASES		NON-ANAEMIC		ANAEMIC	
	No.	%	NO.	%	No.	%
Symptomless	32	12.2	32	14.6	0	-
Post-menopausal bleeding	37	13.9	36	16.5	1	2.1
Menorrhagia	78	29.3	51	23.3	27	57.5
Irregular bleeding	42	15.8	32	14.6	10	21.2
Epimenorrhagia	6	2.2	3	1.4	3	6.4
Intermenstrual bleeding	6	2.2	6	2.7	0	-
Other symptoms	65	24.4	59	26.9	6	12.8
Total	266	100.0	219	100.0	47	100.0

TABLE 5: Situation of Fibroids

Site	Number	Percentage	Anaemic	Percentage	Non-anaemic	Percentage
Intramural	146	65.0	26	63.4	120	65.5
Subserous	38	16.9	5	12.2	33	18.6
Submucous	23	10.2	4	9.7	19	10.3
Others	17	7.5	6	14.6	11	6.0
Total	224	99.6	41	99.9	183	100.4

TABLE 6: DEGENERATION IN FIBROIDS

Pathological Change ANGE	Number	P Percentage E
Hyaline change	59	28.0
Necrosis	16	7.6
Calcification	11	5.2
Haemorrhage	6	3.8
Malignant change	5	2.3
Cystic degeneration	3	1.4
No change	108	51.7
Total	210	100.0

TABLE 7: Operative Procedures

Operation N	Number	P|Percentage
Total Abdominal Hysterectomy : BSO	183	87.3
Vaginal Hysterectomy and repair	13	6.1
Myomectomy	14	6.6
Total	210	100.0

Alcoholism and Related Problems

DR. ABRAHAM GALEA MD
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SENIOR LECTURER IN PSYCHIATRY
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Alcoholism is a condition in which the individual has lost control over his drinking or is unable to abstain.

Problem drinking is a term used to describe abnormal drinking which has not yet progressed to the stage of dependence. It is important from the clinical point of view because heavy bouts of drinking on weekends may be dangerous to the family, to the self and to others like driving, work etc. The Characteristics of Alcoholism are (Jellinek 1946) as follows:

Pre-Alcoholic Phase: when alcohol is more taken as a relief and this becomes more regular while tolerance is increased;

Prodromal Phase: marked by *blackouts* in which the drinker, after a moderate amount of alcohol by his own standards, may show no signs of intoxication and be able to carry out acts requiring a high degree of coordination of which he subsequently has no recollection - an important warning. At this point the patient has more *blackouts* alternating between a superficial attitude to his problem and short periods of insight and guilt.

The Crucial Phase is ushered in by a loss of control in which the ingestion of even a very small quantity of alcohol sets up a compulsive demand for more which ceases only when his stomach or nervous system calls a halt.

The Chronic Phase is marked by prolonged periods of intoxication with absence from work, serious deterioration in ethical attitudes, covered by a variety of shallow realisations of his drinking behaviour; difficulty in obtaining the usual spirits may now lead to drinking of cheap spirits like methylated spirit.

The more important points that the individual is an alcoholic are:

i) Subjective awareness that a few drinks are not enough and one drink leads to another till his *promised upper level* is exceeded:
ii) Withdrawal phenomenon - morning withdrawal symptoms such as shakes sweating and *butterflies* in the stomach. These are relieved by more of the drug, and hence the morning drink. Withdrawal fits may occur.
iii) Tolerance Phenomena. There is first a raised tolerance, and then in the later stages of the illness tolerance declines and the alcoholic *begins to get drunk on less.*
iv) Amnesias. Alcohol amnesias or blackouts are a frequent phenomena. There is no actual loss of consciousness but in the morning the patient cannot remember the night before.
90% of Alcoholics are not diagnosed.

Blocks to Diagnosis (Griffit Edwards)[2]

i) Expecting the alcoholic to conform in appearance to the skidrow stereotype;
ii) Expecting the alcoholic to be someone with gross physical signs. An enlarged liver will seldom be found, but **Palpation under the Right Costal Margin** will often elicit tenderness. Gross alcoholic neuritis is a rarity, but absent or diminished ankle jerks are a common clue. An obvious tremor is unlikely but a slight tremor of the outstreched hands or the tongue may be suggestive evidence.
iii) Forgetting that women too suffer from alcoholism.
iv) The alcoholic often comes into the surgery, asking for a 'certificate' on nebulous grounds; or asking for something for *bad nerves*; asking for something *for his stomach*; asking for a certificate on nebulous grounds; or carrying a letter from the casualty department for a minor accident.
v) Occupation hazards like publicans, travelling salesmen, journalists, entertainers, hard pressed executives.

Assessment of an Alcoholic:

i) The amount he drinks:

Patients hide the amount they consume so that in asking always assume twice the amount the patient volunteers. A heavy drinker is usually drinking half a bottle of whisky a day or eight pints of beer and one must definitely prevent escalation beyond this point. He should be asked whether he drinks in the morning; whether he worries that supply of spirits is not enough for his demands; whether he hides empty bottles; whether he shows signs and symptoms of physical and psychological sequelae (See Chart).

ii) Assess damage to mental health:

Bad nerves; irritability; explosive espisodes; blackouts; irresponsible behaviour, pathological jealousy; transient 'confusional state' with fears hallucinations and tremors (Delirium Termens); hallucinosis; memory defects with confabulation (Korsakoff's or Wernicke's): depression.

iii) Assess damage to physical health

"not feeling at all fit"; peripheral neuropathy; liver damage; brain organic syndrome; trauma; peptic ulcer; avitaminosis; Tuberculosis; impotence.

iv) Assess damage to social health:

Motor vehicle accidents; absenteeism from work; firing from duties; family friction and breakdown; debt; wife battering or other cruelties; loss of sexual relations.

Investigations:

Blood Count and Picture might reveal hypochromic anaemia from malnutrition as well as thrombocytopaenia (Folic Acid deficiency). Increased cholesterol is a usual finding. Abnormal Liver Function may be detected as well as unsatisfactory Renal functioning.

Treatment:

Dealing with alcoholics may be very difficult but rewarding. One decides first whether to handle the patient at home or refer him for consultation.

Hospital treatment is *Never* the answer as alcoholism is a life-long problem. Hospitalisation is indicated in the withdrawal stage of alcoholism where the patient is nursed in a quiet room; given enough sweetened drinks, sedated with Heminevrin 200 mg three times daily (or chlorpromanzine 50 mg t.d.s.) and given infusion of large doses of vitamin preparation. He may be admitted to hospital in Korsakoff/ Wernicke's syndrome as well in advanced liver damage.

Consultation with a specialist is called for when the patient is on the stage of becoming an alcoholic, social factors are threatening and psychological functions are disrupting the family's and the patient's well being. The Consultant/Specialist may order routine management and may also order psychotropic drugs like Diazepam 5mg tds, Chlordiazepoxide 10 mg tds or Thioridazine 25mg tds. Sometimes the patient is initiated under specialist supervision with Disulfiram (Antabuse) or more recently Citrated Calcium Carbimide (Abstem) which though producing less severe reactions and toxic effects with alcohol, is still dangerous, as alcohol and disulfiram produce acetaldehyde with consequent rapid pulse, vasodilatation, dyspnoea and a feeling of distress which may be fatal unless i/v Ascorbic Acid is given promptly. The Specialist may refer the patient to psychotherapy, hypnosis, behaviour therapy, group therapy (Alcoholic Anonymous - P.O. Box 300 B'Kara) or even spiritual therapy.

Management at home

i) Acceptance of the patient:

The doctor and the patient must reach the conclusion that the primary problem is alcohol. In this respect the patient must be *Motivated* to do his best to overcome the problem. He must realise that he is weak in the face of alcohol and he must be ready to function at a lower level without alcohol. He must also accept that he may not be completely true in matters related to it and may be questioned. He must keep below the "break off" point of 150 mg% in the blood (1/2 bottle of spirits or 8 pints of beer). He must avoid going to parties though he may host one. Being a host adds the responsibility of behaving in front of guests. The doctor immediately starts *building the patient's health* — with good wholesome food; sugared drinks, exercise, rest and vitamins. He makes himself available as *a friend* for consultations and ready to bear with him in *changing a life style.*

ii) Setting Realistic Treatment Goals:

The ultimate goal should be total abstention of alcohol. This is easier said than done and some patients are overmotivated and grin and bear it. Others relapse and relapse. In each case the doctor takes him on. The patient is told to avoid one party after another and also one starter after another for a limited period till the patient realised that the can do it at some time, perhaps most of the time and exceptionally all the time. The patient is shown clearly the advantages of being free without hang-overs, without making a fool of himself; without hurting people and with the serenity of clear judgement. Sometimes he is praised for being *off* at particularly difficult periods and often to smile at his superiority over his former colleagues.

iii) Treat the whole situation.

The family must activate treatment. The debts are dealt with; apologies spread to the casualties of his loose tongue when drunk; the bar may excuse him serving two instead of "don't know how much." At times he must see the responsibility of his action like leaving his wife as a wall flower while he teases other women with dirty jokes; of driving home alone when he is under the influence of alcohol; of apologising himself when he offends; of paying for the damages he incurred. Hard words are usually avoided and threats are no use.

Prognosis

The success rate is not high but encouraging 20% get well and dry; 20% never respond and 60% are not cured but saved.

GUIDELINES FOR EARLY DETECTION OF ALCOHOLISM

PHYSICAL CLUES:

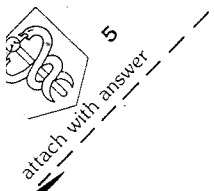
Gastrointestinal: Vomiting often before breakfast, nausea, dysphagia, vague abdominal pain.
Neurological: black outs, insomnia, headaches.
Cardiovascular: palpitations, bradycardia.
Traumatic: frequent accidents, falls or injuries, cigarette burns on hands and chest.

PSYCHOLOGICAL CLUES:

Behavioural:
- Frequent financial difficulties:
- Excessive abstenteeism and poor performance at work
- Marital discord and multiple separations
- Immoderate use of coffee, tea, tobacco, drugs.

Emotional:
- Anxiety
- Panic attacks
- Hallucinations
- Depression
- Suicidal ideas

Laboratory findings
- Hiperlipidaemia
- Abnormal Liver/Kidney Function
- Anaemia



Clinical Diagnosis

Mr. V.M. Sultana MD FRCS, Senior Assistant in Surgery and lecturer at the University of Malta is presenting the following cases in this issue's quiz:

Case 1:

A 68 year old male is being treated for a recent myocaridal infarction. He suddenly complained of crampy perimbilical pain. He looked obviously ill and sweaty. On examination he had tenderness in the right iliac fossa with early distension of the abdomen. Pulse, 130/min. regular; B.P. 100/60. Air entry into the lungs, good and equal on both sides. WBCC 14000/mm^3; Hb 15g/dl; urea and electrolytes, normal.

- What is your differential diagnosis?
- In your opinion what is the true diagnosis?
- Outline the management of such a case.

Case 2:

A 33 year old female presented with joint pain and fever. The fever started two days previously. Later that day, she suffered pains in the wrists, hands and right knee. The next day she noted a rash on her neck. On examination her temperature was 100 F. She had a faint maculopapular rash on her neck and face. There were prominent, tender posterior cervical lymph nodes. The wrists, hands and right knee were tender and warm but no effusions were present.

- List the differential diagnosis.
- What is the actual diagnosis?
- Which test is most likely to provide the diagnosis?
- What precautions should this young lady take?

Results of "Clinical Diagnosis" of issue number 4.

Case 1:

(a) Gallstones in the gall bladder.
(b) Slight hydronephrosis of the right pelvicalyceal system due to partial obstruction at the pelviureteric junction.

Case 2:

(a) Cystitis secondary to a hypertrophied prostate.
(b) (i) Diverticulum of the urinary bladder
(ii) Obstruction of the R pelviureteric junction residual contrast medium in the right pelvis.
(iii) Some osteoarthrotic changes.

Winner: Oscar Aquiling (V yr med. stud.)
Prize: A **G.P.'s Attache' Case** kindly donated by **Messrs. Michele Peresso Ltd.**

The Winner of Clinical Diagnosis 5 shall receive a **Doctor's Elastoplast Set** donated by **V.J. Salomone Ltd., 10 South Street, Valletta,** together with a **Papermate Set** donated by **Messrs. George Borg Ltd., Merchants Street, Valletta.**

ESSAY WRITING COMPETITION

All Doctors and Medical Students registered in Malta are invited to typewrite an essay of 2000 words maximum on the title:

"PROGRESS IN ANAESTHESIA SINCE WORLD WAR II"

The closing date for submission of the essay is

15th OCTOBER, 1984,

and the successful one will be published in the journal of the Association, the Acta Anaestesiologica Melitensis. Each essay is to have a nom de plum and in a separate sealed envelope, the competitor should write his name, address and nom de plume.

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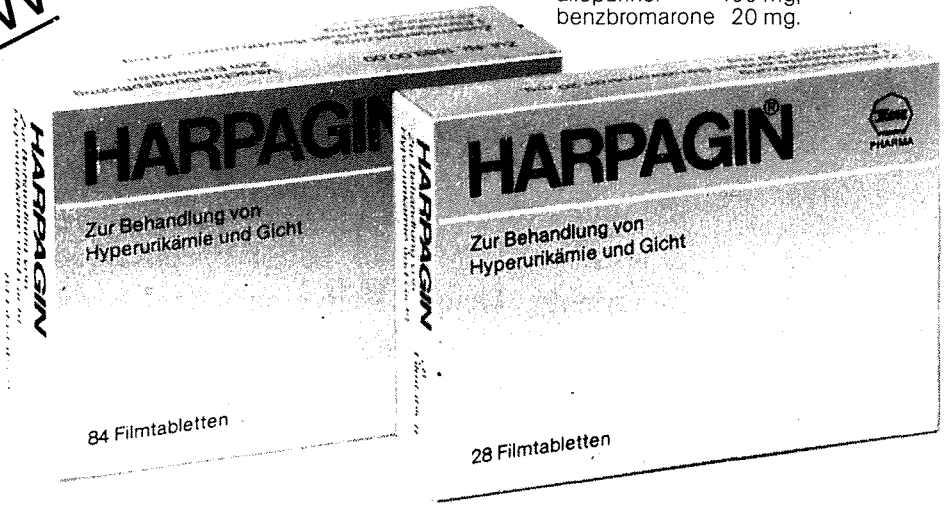

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