RES MEDICA
Journal of the Royal Medical Society

Contents

ISSN: 2051-7580 (Online) ISSN: 0482-3206 (Print)
Res Medica is published by the Royal Medical Society, 5/5 Bristo Square, Edinburgh, EH8 9AL

Res Medica, Spring 1963, 3(3)
doi: 10.2218/resmedica.v3i3.393

Volume III. No. 3. Spring 1963

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Published Quarterly: October, January, April, July

Annual Subscription: 21s. post free

Editor: JOHN BRUCE

C.B.E., T.D., P.R.C.S. Ed., Hon. F.A.C.S., Hon. F.R.C.S. Eng.

RES MEDICA

THE JOURNAL
OF
THE ROYAL MEDICAL SOCIETY

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ABDOMINAL CRISES

By I. R. S. SINCLAIR, F.R.C.S.

The last of three articles written for Res Medica

ACUTE INFLAMMATORY CONDITIONS

With the exception of the pancreas it is the hollow viscera which are involved in acute inflammatory disease. Always beginning as a localized process in the wall of the viscus the rapidity with which the inflammation spreads to the peritoneal space to produce peritonitis depends largely on the time taken for necrosis and perforation of the inflamed area to occur. If the reaction is slow, the inflamed area has often become adherent to adjacent structures or wrapped in omentum before perforation occurs so that even after this disaster the peritonitis remains localized for a further period and an abscess forms.

Faced with the diagnostic problem of a patient who shows the features of general peritonitis it is sometimes impossible for the clinician, on physical signs alone, to decide in which part of the abdomen the trouble has arisen though a careful history will often give a strong lead. However, under the conditions prevailing in this country the patient is generally seen at a sufficiently early stage for some localizing signs to be present. It is convenient in practice to divide patients into those suspected of upper abdominal pathology and those whose clinical picture points to a lower abdominal or pelvic origin of the inflammation.

UPPER ABDOMINAL CONDITIONS.

Perforated Peptic Ulcer is the most dramatic of the common abdominal catastrophes. Though the well-known symptoms and signs of peptic inflammation may have been present for many months or years this is not invariably so. Because the commonest sites of peptic ulceration are separated from the anterior abdominal wall only by the liver edge and lie high above the great omental apron the possibility of sealing off the inflamed area by omental or peritoneal adhesions is limited: perforation is usually free and involvement of the anterior parietal peritoneum immediate. Therefore, though occasionally a small leak of gastric juice may produce a localized reaction and a clinical picture suggestive of an acute exacerbation of ulcer dyspepsia, much more commonly the period of localization to the upper abdomen is short or even non-existent. The dramatic onset, the unremitting severity of the pain which freezes the patient into immobility, afraid even to breathe, and the intense reflex contraction of the anterior abdominal muscles usually makes diagnosis easy in a patient with a history of ulcer dyspepsia. Apart from the very small leak, confusion arises in those rare cases of perforation of a gastric ulcer into the lesser peritoneal sac which confines the reaction to that deep cavity remote from the anterior parietes and the examiner's hand. In cases of doubt a plain x-ray of the abdomen taken with the patient in the upright position will

often confirm the presence of a perforation by demonstrating free gas beneath the diaphragm. This preliminary film is also of value in revealing inflammatory changes in the lungs in a high proportion of cases of perforation. Already present on admission due to the restricted ventilation caused by pain, these changes have in the past been erroneously labelled as post-operative complications.

With few exceptions the safest method of treatment of a perforated peptic ulcer is by immediate operation. When there is only a small localized leak, in the aged or where other complications exist, expectant treatment by continuous gastric suction and intravenous fluid therapy may be preferred but is a method requiring constant vigilance and carrying a significant risk of failure. Whether the surgeon decides to carry out simple suture of the perforation or to perform some type of gastrectomy with removal of the ulcer, depends partly on technical considerations (it may be impossible to suture or plug a large gastric perforation) partly on the condition of the patient and not least on the experience of the operator. In the fit subject the risks of gastric resection in the first few hours after perforation are no greater than those of simple closure of the perforation, given expert surgery and anaesthesia, and in patients with a long history of dyspepsia or with previous complications a second operation may thus be avoided.

Acute Cholecystitis and Cholangitis. Acute inflammation in the gall bladder produces great tension at an early stage, because the infective process is nearly always due to stagnation as a result of calculous obstruction of the neck of the gall bladder. Acute symptoms arise long before perforation occurs and localizing symptoms and signs are correspondingly prolonged. For this reason diagnosis is often relatively easy. A tensely distended inflamed gall bladder may project beyond the liver margin if previous chronic inflammation has not produced fibrous thickening and contracture of its wall—so-called empyema of the gall bladder— but muscular guarding and rigidity often prevent its recognition on examination. General peritonitis is rare but some degree of peritoneal reaction is usual and may result in a trickle of inflammatory exudate down the right side of the abdomen. In this event tenderness may be quite marked in the right iliac fossa.

Although the management of acute cholecystitis is usually conservative, urgent reappraisal is called for should the pain and local signs persist or increase after forty-eight hours. This may mean that the gall bladder wall has become gangrenous or, especially in elderly patients with a palpable mass, that the gall bladder has undergone torsion. Where pain in the right iliac fossa makes the exclusion of acute appendicitis impossible, it is much better to do an immediate cholecystectomy than to risk leaving a gangrenous gall bladder.

Acute cholecystitis is rarely accompanied by a significant degree of jaundice but from time to time one encounters a patient whose symptoms and signs, otherwise suggestive of cholecystitis, are accompanied by jaundice and rigors. The triad of pain, jaundice and rigor, known for the past century as Charcot's biliary fever, indicate a diagnosis of infective cholangitis secondary to a partial obstruction of the common bile duct. Untreated, or wrongly treated by antibiotic therapy without surgical drainage, such patients may progress to develop multiple liver abscesses or to die from what has loosely been called the "hepato-renal syndrome." Deaths from acute renal failure in patients with biliary disease have been recorded intermittently over many years and much has been written on the possible association between the two systems. Recently somewhat discredited, I believe this syndrome to be a real and important entity and have encountered it on a number of occasions during

the past three years in the artificial kidney unit at the Royal Infirmary. In each case acute renal failure developed in a patient with infective cholangitis and in every one the renal failure persisted and blood cultures continued to yield coliform bacilli until surgical drainage of the biliary tree was carried out. Recent experimental work on the effect of coliform endotoxins on renal blood flow may shed light on the fundamental nature of the syndrome.

Acute Pancreatitis. This condition is not so rare as many believe. If it is sought, it will be found nearly as commonly in this country as in North America or Scandinavia despite the virtual absence of the alcoholics who comprise a large proportion of cases of pancreatitis in these two areas. Many theories and much experimental research has left the causation of the condition still uncertain. The finding of biliary calculi in 50 per cent. of cases of acute pancreatitis does not necessarily imply a causative relationship and Opie's (1902) much quoted finding of a gallstone impacted at the ampulla of Vater in a fatal case of acute pancreatitis is exceptional. On the other hand anatomical studies in fatal cases have revealed a potential common channel between the biliary and pancreatic ducts at their lower ends in almost all cases and this, together with much experimental evidence, suggests that biliary reflux, perhaps due to spasm or oedema at the ampulla during a phase of secretory activity following a meal or the consumption of alcohol, is a likely explanation in many cases. Obstruction of the actively secreting gland, even in the absence of biliary reflux causes pancreatitis in dogs and the definite hazard of injury to the pancreas or to its major ducts during gastroduodenal surgery explains why post-operative pancreatitis is today one of the principal early complications of gastric resection. There is no evidence that primary bacterial infection is ever responsible for pancreatitis but oedematous or subsacute pancreatitis is a well-known complication of mumps and it may be that the oft-maligned diagnosis of "abdominal influenza" is sometimes justifiable and describes a similar entity. During the past few years experimental evidence has been put forward to support the view that either a sensitivity reaction to foreign (bacterial) protein or a vascular occlusion may explain most cases of acute pancreatitis but the evidence for these views is not yet sufficient to command general acceptance.

Certain diagnosis of acute pancreatitis is often impossible prior to laparotomy. In its most acute form the sudden onset and signs of peritonitis may simulate the clinical picture of a perforation but more often the deep retroperitoneal situation of the pancreas prevents early involvement of the anterior parietal peritoneum so that there is tenderness rather than rigidity and back pain is very common. Proximity to the pylorus and to the sympathetic plexuses causes pylorospasm with reflex retching and vomiting and early paralytic ileus. The amount of fluid exudate which can be lost into the loose retro-peritoneal and mesenteric tissue planes is much greater than is often realized and this cause of hypovolaemia rather than any esoteric form of trypsin shock probably accounts for the profound hypotension which is occasionally found in very severe cases.

Estimation of the serum amylase level is sometimes of great help in clinching the diagnosis but too much reliance must not be placed on this test. Abnormally high levels may last for only a few hours and comparable levels may also be found in other acute upper abdominal conditions, notably perforation of a peptic ulcer. Radiological examination to exclude the presence of free gas under the diaphragm is always a wise precaution before the diagnosis of acute pancreatitis is accepted. At laparotomy the diagnosis is usually apparent from the oedematous and often bile-stained tissues around the duodenum and pancreas and from the flecks of white saponified fat in the omentum and mesenteries even before the pancreas itself is examined but even the operative

findings may be misinterpreted. A few weeks ago I encountered a patient in whom the dramatic onset of excruciating pain with intermittent exacerbations accompanied by reflex retching and a tender but not rigid abdomen led to a tentative diagnosis of internal strangulation or torsion. At laparotomy the whole small bowel was cyanosed and the mesenteric vessels non-pulsatile. The diagnosis of mesenteric vascular thrombosis was disproved at autopsy two days later when it was found that the oedematous and necrotic pancreas had obstructed the superior mesenteric vascular pedicle as it traversed the groove between the body of the gland and the uncinate process.

So long as the causation remains in doubt treatment must be based on the general principles of combating hypovolaemic shock and of resting the inflamed gland by continuous gastric suction to prevent the access of gastric acid into the duodenum and by autonomic blocking drugs to inhibit the neurogenic component of pancreatic secretion. Provided the diagnosis can be made with certainty immediate operation is not desirable but if, as is usually the case, the diagnosis is only substantiated at laparotomy surgical decompression of the biliary tree is rational if there is evidence of biliary obstruction. Drainage of the peritoneal cavity has nothing to offer in the early case in contrast to the later stages when drainage of a pancreatic abscess or of a pseudo-cyst may be life saving. The vogue for administration of steroids has little factual evidence to support it and the use of the trypsin and kallikrein inhibitor "Trasylol", much vaunted in Europe during the past two years has so far been unimpressive in my hands. Because secondary infection may develop in the devitalized pancreas it has been customary to give a broad spectrum antibiotic from the time of admission to hospital and this practice certainly reduces the mortality of experimental pancreatitis in dogs. However, the liability of dogs to develop serious infective complications of diverse abdominal catastrophes is incomparably greater than that of man and having regard to our present hospital bacterial environment and the grave risks of injudicious and uncontrolled antibiotic therapy it may be wiser to with-hold such treatment until definite evidence of bacterial infection appears. On the theory that the rapid disintegration of the pancreatic tissue is due to auto-digestion by non-specific intracellular enzymes rather than by the specific enzymes which these cells secrete and in view of the very high metabolic rate of pancreatic tissue, attempts have been made in recent years to arrest the progress of acute pancreatitis by the administration of thiouracil and have been reported to yield dramatic results. I have no personal experience of this method.

LOWER ABDOMINAL CONDITIONS

Acute Appendicitis. Turning to the lower abdomen we come to the commonest of all acute abdominal emergencies. It was Sir David Wilkie who first stressed the different natural history of the primarily catarrhal appendicitis and the primarily obstructive type of disease. The former, which is much less common, may often subside spontaneously whereas the latter almost invariably proceeds to gangrene and perforation unless the appendix is removed surgically.

Pain from an inflamed or obstructed appendix is usually felt for some hours only in the general area of reference of mid gut derivatives, namely the central abdomen, and, especially in obstructive appendicitis, the pain is colicky so that simple gastro-enteritis or indigestion is suspected. At this time, during which one or two episodes of reflex vomiting are common, tenderness will be present over the appendix itself, but it is only when the parietal peritoneum is involved by a spread of the inflammatory exudate that the patient experiences spontaneous pain over the appendix. This classical sequence of events makes the diagnosis very easy in the typical case but unfortunately few cases

are typical. In general, there is only slight pyrexia and the pulse rate, which is normal in the early stages, soon rises to a disproportionate extent, whereas in acute pyelonephritis, which may be suspected if urinary symptoms result from proximity of the appendix to the ureter or bladder, the temperature is usually much higher in relation to the pulse rate, and rigors are more common. In pyelonephritis loin pain is usual and the absence of rebound tenderness and crossed tenderness (Rovsing's Sign) help to exclude intraperitoneal inflammation. Certain other manoeuvres such as stretching the psoas muscle (by hyper-extension of the hip) or the obturator internus (by medially rotating the hip) sometimes help to confirm a diagnosis of appendicitis in doubtful cases, the production of pain indicating an inflammatory reaction in proximity to these muscles. The importance of a rectal examination, especially when the appendix lies in the pelvis, remote from a hand palpating the abdomen above it, was stressed in the first article of this series, where other pitfalls in diagnosis were also discussed. The high retrocaecal or paracolic appendix may simulate gall bladder disease, and, especially in the elderly, a retro-ileal appendix may present minimal signs in the early stages, later simulating a subacute intestinal obstruction with distension and fluid levels on x-ray examination. Early diagnosis is always important but it is particularly vital in the child, whose omentum has not yet grown sufficiently to reach and wrap around an inflamed appendix, and in the elderly whose reaction and resistance are low. In these two groups the progress to generalized peritonitis is rapid and it is in these that most of the fatalities are still found.

In general, the correct management of acute appendicitis is immediate appendicectomy, whether the condition is diagnosed after two hours or several days. Only if a palpable mass indicates that nature has already succeeded in localizing the infection is conservatism justified. With rest and antibiotics such cases usually subside. If not, simple drainage of the abscess is all that is advisable. Whether or not drainage is needed at this stage, interval appendicectomy should always be carried out some two or three months later when the reaction has settled. In every conservatively treated patient over the age of forty a barium enema should be carried out as soon as the acute symptoms have settled. The precipitation of acute obstructive appendicitis by a tumor of the caecum is well recognized and it is tragic deliberately to defer operation on a caecal carcinoma for three months just because it has presented as a case of right iliac fossa pain with a palpable mass.

Acute Diverticulitis. Diverticula of the colon are so common that diverticulitis and its complications are one of the most frequent causes of lower abdominal peritonitis. Occurring most often in the pelvic colon, the clinical picture of diverticulitis has been described as left sided appendicitis. This is not a very accurate analogy as the type of patient is different—usually obese, constipated and elderly—the central pain of mid gut disease is absent and the progress is generally slower, a pericolic abscess being much commoner than general peritonitis in the untreated case. Treatment is at first conservative, with dietary restrictions and antibiotics. Only if symptoms fail to subside or if colonic obstruction or, occasionally, melaena, complicates the situation is operation carried out. In the presence of marked pericolic inflammation and adhesions the diseased segment of bowel is left untouched and a proximal (transverse) colostomy is fashioned to divert the faecal stream and allow the infection to subside. Resection of the diseased segment can then be carried out as an elective procedure before the colostomy is closed.

GYNAECOLOGICAL EMERGENCIES

Acute Salpingitis must always be considered in cases of lower abdominal peritonitis in women. The frequent onset at the time of a menstrual period,

the presence of vaginal discharge, the bilatral tenderness and, if a tubo-ovarian abscess has formed, the presence of a palpable pelvic mass all suggest the correct diagnosis. The infection is usually of low virulence and responds to conservative treatment. Operation is therefore avoided unless acute appendicitis cannot be excluded.

Lower abdominal inflammatory conditions are sometimes simulated by *rupture of a Graafian follicle*. The patient is usually a young woman and the symptoms tend to occur around the middle of the menstrual cycle. The pain starts in the lower abdomen and as a rule is a constant dull ache unaccompanied by vomiting. Laparotomy is usually undertaken on the presumptive diagnosis of acute appendicitis.

Rupture of an ovarian cyst may also give rise to a picture of pelvic peritonitis or of intraperitoneal bleeding. *Haemorrhage into an ovarian cyst* causes severe, continuous, increasingly lower, abdominal pain accompanied by tenderness and rigidity but, despite the muscle spasm, it is usually possible to feel a large, tense spherical swelling. *Torsion of an ovarian cyst* on its pedicle results in a more dramatic onset of severe pain, often varying in intensity and accompanied by reflex vomiting, suggesting some form of internal strangulation. All these conditions require operative treatment.

Ectopic Gestation. Tubal pregnancy may present as an abdominal emergency in several ways of which the most important are either a slow intraperitoneal leak of blood simulating a pelvic peritonitis or a massive intraperitoneal haemorrhage in which the signs of general peritonitis rapidly appear accompanied by pallor and hypotension. There is a history of recently absent or abnormal menstrual bleeding in almost every case but there is no characteristic type of irregularity. Complete amenorrhoea followed by bleeding is the most common sequence of events. Even with the slow intraperitoneal leak it is remarkable how often pain is experienced in the left shoulder and the patient should always be specifically asked about pain in any sites other than the abdomen. A history of fainting is also very suggestive of intraperitoneal haemorrhage. Blood in the peritoneal cavity acts as an irritant and usually causes fever and leucocytosis as well as stimulating bowel contractions which, by giving rise to intestinal colic, may confuse the diagnosis. Colicky pain may also result from uterine contractions.

Although it may sometimes be possible to palpate a mass in either iliac fossa it is the vaginal examination which is most important. The softened cervix, the palpation of a tender, boggy, mass in the Pouch of Douglas and often the trace of blood on the examining finger will confirm the diagnosis.

In cases with massive bleeding there is no time for close interrogation or prolonged examination. A glance at the pallid, clammy, restless woman is enough to suggest haemorrhage as the cause of the abdominal pain and during the child-bearing years the most likely source for this lies in the pelvis. Such patients are the most urgent of all abdominal emergencies. Infusion of dextran or plasma is followed by whole blood transfusion as soon as possible but in desperate cases laparotomy to control the bleeding may be necessary before resuscitation is possible.

VASCULAR EMERGENCIES

INTRAPERITONEAL HAEMORRHAGE

Spontaneous intraperitoneal bleeding may arise from many different sources. In young women a ruptured ectopic gestation is the most important cause (see above). *Rupture of an aneurysm of the splenic artery* is another vascular disaster which is not only much commoner in females than in males but is encountered very often in the later months of pregnancy. The most common pathological basis is atheromatous degeneration with mycotic

aneurysm in association with bacterial endocarditis coming second. Although sudden massive haemorrhage may be the first sign of trouble the initial rupture is often to some extent controlled by the formation of a false aneurysm in the retroperitoneal tissues around the hilum of the spleen or in the lesser sac and rupture into the general peritoneal cavity occurs after a delay of hours or even days. In such cases the early clinical picture may simulate some upper abdominal inflammatory crisis such as perforation of a peptic ulcer into the lesser sac or acute pancreatitis. Though rare, the diagnosis of this emergency is of great importance because delay in operative treatment is almost certain to be fatal and, if full advantage is to be taken of the period of grace given by a preliminary leak the condition must always be kept in mind in a woman who develops acute upper abdominal symptoms during the last trimester of pregnancy. Operative treatment is itself a matter of considerable hazard. The gravid uterus make proper exploration of the posterior abdominal structures impossible and, as the foetus is always dead, rapid caesarean section may be necessary despite the added risk in a critically ill patient. Even then, the infiltration of the tissues with blood makes identification of the source of bleeding extremely difficult.

Spontaneous rupture of the spleen occurs usually in the presence of splenic disease though it has occasionally occurred in a normal spleen in the last month of pregnancy or during parturition. In the tropics the most frequent cause is malarial enlargement but in this country glandular fever and leukaemia are the usual diseases.

Spontaneous rupture of minute congenital aneurysms probably accounts for those occasional cases of "abdominal apoplexy" in which a bleeding point is not detected at laparotomy or autopsy. Over the past fifteen years I have operated on three patients who presented as cases of general peritonitis only to find the peritoneal cavity full of blood which proved to be arising from a renal carcinoma. In each case the assumption that the haemorrhage indicated an advanced and inoperable malignancy was disproved at the post-mortem examination which showed the blood to be coming from distended normal veins and not from the tumour. The kidney was technically removable and in none of the three cases were there evident metastases. It would therefore seem reasonable to carry out a trans-abdominal nephrectomy in these circumstances as being the only way to arrest the bleeding and at the same time offer a reasonable prospect of cure. Why these distended veins should have ruptured is not clear. The obvious explanation that the tumour, as its wont, had grown into and obstructed the renal vein did not hold good in any of my three cases.

RETROPERITONEAL HAEMORRHAGE

In an elderly patient the most likely cause of severe intra-abdominal haemorrhage is *rupture of an atherosclerotic aortic aneurysm*. Rupture into the general peritoneal cavity is rare, most cases showing a very extensive retroperitoneal extravasation. As with other types of bleeding it is surprising how often the haemorrhage takes the form of a small preliminary leak which is temporarily controlled by the tension in the surrounding tissues and by clotting. The acute onset of sudden severe constant abdominal pain, often radiating to the back or legs may not be accompanied by obvious signs of blood loss until the final catastrophic rupture takes place. Generalized tenderness may prevent adequate palpation so that the presence of a pulsatile mass is easily missed especially in an obese patient. A bruit is rarely recognisable on auscultation after an aneurysm has ruptured—especially if hypotension is marked. Progressive distension due to ileus caused by the retroperitoneal haematoma coupled with reflex vomiting may simulate the picture of acute

intestinal obstruction with internal strangulation. The distended bowel may also obscure the radiographic shadows of calcification in the wall of the aneurysmal sac but x-ray examination should never be omitted. Although a past history of recurrent back or abdominal pain (the latter sometimes in the form of colicky pains during digestive activity—that so-called "abdominal claudication") is a useful pointer, such a history is obtained from less than half of the patients. Conversely, when abdominal or back pain is caused by an aortic aneurysm the situation is one of some urgency for the onset of symptoms rarely precedes rupture of the aneurysm by many months. The only treatment carrying any chance of success is resection of the aorta and its replacement by a homograft or woven prosthesis—a long and dangerous procedure under difficult conditions when rupture has occurred.

Retroperitoneal haemorrhage is not uncommon in haemophilia and should always be kept in mind when a known haemophilic presents with symptoms and signs of an abdominal emergency, especially if a tender mass is palpable. This is the only circumstance in which laparotomy is to be avoided in cases of suspected intra-abdominal haemorrhage.

ACUTE GASTRO-INTESTINAL HAEMORRHAGE

By far the commonest condition causing acute gastro-intestinal bleeding is peptic ulceration (about 85 per cent.) with gastric erosions and portal hypertension tying for a very good second (about 5 per cent). The remaining 10 per cent. of cases is made up of a variety of conditions such as hiatus hernia, gastric tumours, peptic ulcer in a Meckel's Diverticulum, angiomata, ulcerative colitis, diverticulitis, blood dyscrasias and aneurysmal rupture into the alimentary tract.

The role of surgery in cases of *bleeding peptic ulcer* has undergone a revolutionary change during the past fifteen years. It is now recognised that early operation is essential if safety is to be achieved and the accumulation of data from large series of cases has taught us that the patient with a known ulcer, who is over 40 years of age and who has either a previous episode of bleeding (or perforation) or who suffers a second haemorrhage after admission to hospital is best treated by immediate operation. Indeed, the older the patient the less well is blood loss tolerated and the sooner should operation be carried out. There is little evidence that adequate blood replacement adds to the risk of further bleeding and it is still too common to leave a patient hypovolaemic or even hypotensive for many hours, with the risk of vascular thrombosis or acute renal failure.

The importance of recognizing acute haemorrhage in *portal hypertension* lies more in the need to avoid than in the necessity to carry out urgent operation. Haemorrhage from oesophageal varices tends to be self-limiting and major surgery is ill-tolerated in patients with severe liver damage. The haematemesis is usually severe, with vomiting of red blood and a history of ulcer dyspepsia may be absent.

To assist in accurate pre-operative diagnosis of doubtful cases emergency contrast radiography using the water-soluble medium "gastro-grafin" has proved of great value and as a last court of appeal before surgery oesophagoscopy may reveal blood welling up from below the cardia and thus exclude oesophageal varices as a cause. The recent advent of the flexible fiberscope may increase the value of emergency endoscopy in the differential diagnosis of gastro-duodenal bleeding. The use of a Sengs-taken* tube to produce traction tamponade on the lower oesophagus will, of course, show whether the bleeding is coming from oesophageal varices or from below and this method of pressure control of bleeding varices may be continued for periods of up to forty-eight hours.

Intravenous infusion of pituitrin will significantly lower the portal pressure by causing splanchnic vasoconstriction and this simple procedure is well worth trying in the acute phase. The possibility of a prothrombin deficiency due to the underlying liver disease must be remembered and intravenous Vitamin K. should be given. Once immediate control of bleeding has been achieved the question of elective porto-caval anastomosis can be considered. In the intervening days the risk of hepatic coma from absorption of protein from the blood-filled gut is very real in cases of hepatic disease and purgation to eliminate the blood coupled with antibiotic therapy restrict bacterial activity are an integral part of the treatment.

In this necessarily sketchy and incomplete review of abdominal emergencies the emphasis has been laid on diagnosis and I have written of the rarities as as well as of the common place.

If we see a bird on a tree the chances are, of course, that it is a sparrow, not a canary. Yet it is just for this reason that we remember the solitary canary when we have forgotten the multitude of sparrows. This is no bad thing. Recognition of the sparrows among the abdominal crises is our daily task and, well done, saves many lives, but to recognize the canaries adds that spice which sustains our enthusiasm and stimulates our senses.

When dealing with abdominal emergencies time is not on the side of our patients, though it may make thing more obvious for us. Delay can be fatal, and, in the words of Francis Bacon "There is surely no greater wisdom than well to time the beginning and onset of things."

*(A triple lumen tube carrying two balloons. The distal spherical balloon is inflated in the stomach and traction applied to pull the balloon back against the cardia. The proximal elongated balloon is then inflated within the lower oesophagus.)

THE BIOLOGY OF MONGOLISM

By R. A. BAILEY

Fellow of the Royal Medical Society.

Based on a Dissertation read before
the Royal Medical Society on Friday, 2nd March, 1962.

Mongolism is a neuro-endocrine disorder, based on a molecular disturbance, which is manifested by a chromosome abnormality. The mental defect is probably due not specifically to a gene or gene complexes on the chromosomes but to a generalised imbalance of the chromosome set as a result of aneuploidy.

There are three types of chromosome abnormality which have so far been described in mongols

1. Non-disjunction.
2. Translocation.
3. Mosaics.

NON-DISJUNCTION

Mongols in this group have 47 chromosomes with chromosome 21 trisomy. This was first described by Lejeune in 1959[1] and then by Court Brown and his colleagues[2] at the Western General Hospital in Edinburgh in 1960.

An American family described by Miller et alia[3] throws an interesting light on non-disjunction. The father died of chronic lymphatic leukaemia (a condition in which chromosome abnormalities have been demonstrated). His sister and his niece by another sister were both mongols. His wife and another son were both normal, genetically and mentally.

His son was a mental defective (IQ 21) with strabismus, cleft palate, eunuchoidism and multiple skeletal abnormalities. This boy's sex chromosomes were found to be XXXY and for this to be possible no fewer than three non-disjunctional events must have occurred. It is probable that all three of these events occurred on the father's side because :

(a) The father was 32 and the mother 23 when the child was born and non-disjunctional effects are more common with increasing age.

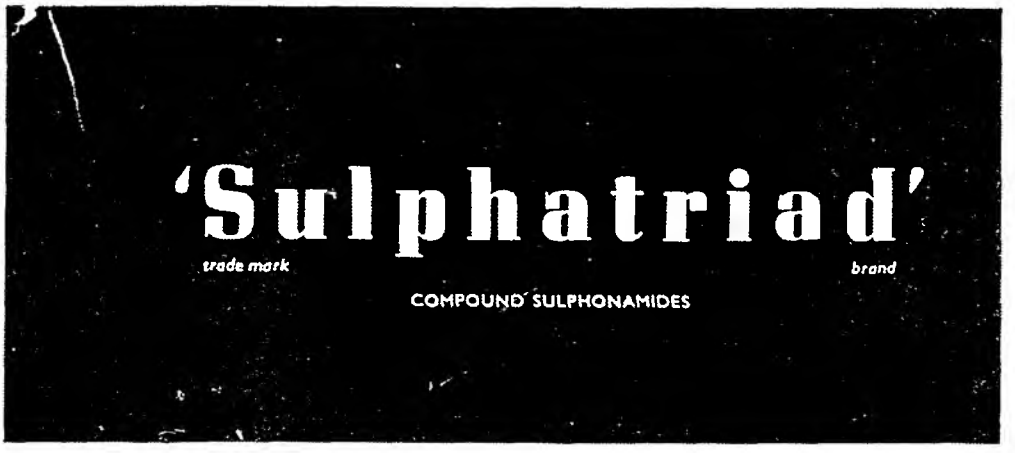
(b) The father's sister and niece were both mongols and probably had meiotic non-disjunctions.

TRANSLOCATION

This was the next type of chromosome abnormality to be described in mongols. Fruccaro, Kaysir and Lindsten[4] were the first workers to described such a case (1960).

Mongolism can be inherited by translocations involving chromosome 21 and one of the other acrocentric chromosomes—numbers 13, 14, 15 and 22. A chromosome count would reveal only 46 chromosomes in such cases but Karyotype analysis shows that extra material was present on one of the acrocentric chromosomes.

Penrose[5] points out that in cases of 21 : 22 translocation paternal age is a highly significant actiological factor. In these cases the average age of the fathers were 10 years above that of fathers in the general population and in only one of the 8 cases so far described was the father under the age of 40.



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This was not found in cases of 21 : 13, 14 or 15 translocation.

It has been shown[6] that the increased risk of young mothers with one mongol child producing a second child similarly affected is mainly due to families in which translocation has occurred. This is borne out by a Swedish family[7] in which the parents were healthy. The wife's first pregnancy ended in abortion. Her second pregnancy resulted in a live born mongol as did her third. These children unfortunately died before chromosome analysis could be carried out. The fourth pregnancy resulted in a live born mongol of translocation resulting in a family of mongols.

If a mother is under 25 and her first child is a mongol the chances of her producing another mongol are fifty times that of a random group of women of the same age.

If the mother is between 25 and 34 her chances are only five times greater than that of a random group of the same age.

If the mother is over 35 her chances are the same as that of a random group of the same age.

Young mothers of mongol children will run a high risk of producing a second affected child because either they or their husbands have a chromosome abnormality. In the older mother this is a rare event so that the risk is the same as the random risk allowing, of course, for age.

MOSAICS

The last type of chromosome abnormality described in mongols are the Mosaics. Mosaicism in cytogenic usage describes a condition in which a substantial minority of cells differ from the majority in their chromosomal content.

Six cases of mosaicism have been described either in mongols or in people with mongoloid features. Four cases[9-12] have had two stemlines (i.e. some cells have had 46 chromosomes and others 47 chromosomes) and two cases[13-14] have had three stemlines (46, 47 and 48 chromosomes).

This mosiacism may be explained by mitotic non-disjunctions in a normal diploid resulting in one cell with 48 chromosomes and one non-viable cell with 44 chromosomes. The 47 chromosome cells may arise by chromosome loss through anaphase lagging, a phenomena which has been described in plants.[15]

SUMMARY

Three types of genetic abnormalities have been described in mongols. These have been briefly reviewed here.

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RES MEDICA

AN EXERPT FROM THE PRESIDENT'S VALEDICTORY ADDRESS, 1962

Those of us who saw Dylan Thomas's version of the Burke and Hare story at the festival, "The doctor and the devils" would not only have been delighted at the play itself, which was outstanding, but might also have noticed something of interest to the Society. For there, to the left of the stage, was an eight foot tall reproduction of that print which the Society has used in its Christmas cards, showing side by side, Knox's school of anatomy, and the Royal Medical Society's first hall. This stimulated in us a curiosity as to the part our members may have played in the finding of anatomical material. It is to be hoped that they were not involved in murder; but certainly they knew of the "resurrectionists"; for in 1828 John Reid, a member of our Society wrote in a letter "I am busy dissecting, now the subjects are pretty plentyiful. People may watch us as they may, but we will have them in spite of them. There are rascals here who will do anything for money, and these are fit hands for such jobs."

At times, indeed, the students themselves lent a hand; an earlier account from the diary of John Knyveton, which was written in 1751 while he was a student in Dr. Urquhart's School of Anatomy, is so dramatic as to be worthy of quotation in full.

"Nov. 7th.—Vastly tired this morning as a result of an Escapade from which I count myself lucky to escape without Grievous harm to life and limb. Mr Bloomfield did yester-eve put to me that we should disinter the body of the hanged woman for the advancement of our anatomy and the glory of medicine; and so after some talk I agreed and we approached our worthy teacher, who warning us of the dangers—for hanging is not the least penalty, one is likely to be torn to pieces by the mob should they learn of it—did then commend our diligence and whilst saying that he would have no hand in it and would know nothing of it should it come to light, did call his huge manservant to him and give instruction that he was to help us . . . and so to Dr. Urquhart's to enter it by the small gate to find that the Doctor had gone out but his man and Mr. Bloomfield and Messrs. Pope and Sinclair gathered in the Anatomy room very comfortable before a fire, smoking and discussing a glass of wine. So with them to pass the evening in pleasant discourse. I growing drunk on the wine, very potent, and when the clocks had struck the half after twelve to collect spades and grapples and to muffle ourselves in thick cloaks

. . . And so into the lane and to the graveyard where Mr. Pope did Belch so loud causing Dr. Urquhart's man to swear vilely vowing that he would rather have a school of apes to help him than such turnip heads. The grave not easy to find, there being very many in a small place, and the moon did come out from behind the clouds which I did not care for as we were more likely to be seen but with its aid to find where the mould had been newly turned. George Bloomfield very vehement to dig up the coffin only to find this being opened did prove to contain an old woman very foul. The Mr. Sinclair on sitting down did find the ground give way under him, and so we found the hanged wench and dragged her out and put in the sack which Mr. Pope and I did carry between us and with great haste to the lane and so to the Doctor's again, all mired and sweaty. George Bloomfield did brew us a bowl of punch and we in need of such a specific.

Lord, what a business this be, this quickening of the Aweful dead, at night when the powers of evil be abroad, amongst the tombs and the earth and the dreadful worms! Fit work only for men of Brutish Minds! Did resolve then to have no more of it, but on reflection did realise that nothing is gained without labour and so as Medicine be the most noble of the arts so the gateway to it is correspondingly difficult and arduous to pass. Slept on a couch at the Doctors, and to home this morning at Mr. Hunt's and with him and Mrs Hunt to church, where I heard a tolerable sermon aptly enough on the resurrection, and wonder what his Reverence would say of my night's activities. Shall to bed early this night."

ON THE BANDWAGGON?

Each year a great deal is written, and far more is spoken, about the failings of our system of medical education. While this is a most healthy sign, let us realise that in exaggerating faults there is a most unfortunate tendency to forget the virtues. Let us not bury our talents beneath the soil of conflicting criticism.

We all recognise the cynic who says that our lecturers must make us think medicine, not swallow it. Surely, on these occasions, it is the speaker who is most able to remedy the situation, in one particular case at least. Moreover, there is much to be gained by highlighting the features of our curriculum which have been found most valuable. We all readily admit that a system cannot be uniformly good, but it is just as unlikely that it is uniformly bad.

Throughout the medical course, a variety of teaching methods are experienced, and a number have been well received. What springs to mind immediately, is the series of clinico-pathological conferences organised by the pathology department at the end of the systematic course of lectures. While a number of the class felt that, as examinations were drawing near, their time could be more wisely spent, those who attended found them extremely valu-

able. Those who were chosen to prepare and present the clinical and pathological data, found this a good way of assessing their knowledge of the particular subject. This type of teaching is useful because it demonstrates the value of relating all aspects of disease, medical, biochemical and pathological.

There are few who deny the value of learning medicine, 'at the bedside'. Naturally there are those who are unfortunate on their choice of hospital or ward for this purpose, but generally the medical staff welcome the opportunity to discuss the cases on their wards. Those who are able to see in full the emergency treatment of status asthmaticus, for example, will then be far more certain of the best procedures to employ when they themselves must diagnose and treat such a condition. It is important, also, to see patients immediately after admission, before a sure diagnosis has been established. In reasoning out the most likely diagnosis and in justifying it to others, one soon learns to pinpoint the most relevant findings of the history and physical examination.

During clinical clerkships there is still more that can be learnt. There is much more time to take histories from each patient than there is later on, as a harassed houseman. Talking to each patient is a good way of broadening ones own experience of people, quite apart from the practice obtained in enquiring after the relevant symptoms. The future doctor ought to have experience in dealing with all types of patient, as well as with all types of disease. It was wisely said that he who knows only medicine, does not even know medicine.

The centre of a great deal of adverse criticism, is the 'formal' lecture. Before we make any further attack on this form of teaching, well established at Edinburgh, we should realise that there are two very distinct types of lectures; the one makes us think around the subject, whatever it may be, and the other supplies the basic principles in an easily classified form. To each there is a very useful purpose. The first is probably of most value later on in the course, after a suitable introduction. It is about the second that most criticism is heard. At this stage of our academic careers, it is argued, we should not require to be spoonfed with facts which are to be found in any textbook. This is a perfectly valid opinion, but a great number will still wish to be told the essentials, so that in their reading, they are not confused by so much unnecessary detail. A system based on one or other type of lecture will never please everyone. What must be realised is that both types of lecture are of value and consequently they can be complementary. The introduction to a subject, in terms of the 'basic facts' type of lecture, could be followed by more general lectures on the 'growing points'.

We realise that in most organised systems there is much to be commended and much to be criticised. Let us mention the advantages of the present system along with the disadvantages, so that those who are in a position to make the necessary changes can do so with a clear idea of the merits. It is on these merits that new schemes can be based. It is too easy for us to jump on the band-waggon of destructive criticism instead of putting forward serious suggestions for improvement.

" Oh, wad some powr the giftie gie us,
To see ourselves as ithers see us,
It wad frae mony a blunder free us . . . "

(Burns)

Limerick from a G.P.

(Yes, even Doctors write rhymes about Guinness)

Said a lively young fellow named Innes,
"I could never get by without Guinness.
From glass or from wood
It tastes just as good
And, my goodness, what goodness therein is!"

M.R.C.S., L.R.C.P.

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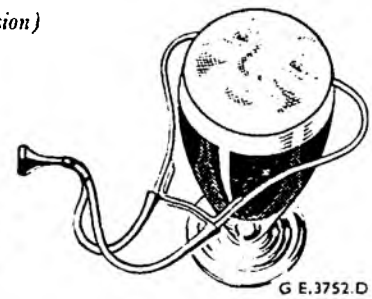

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"SOME ASPECTS OF FUNCTIONAL ORGANISATION IN THE CEREBRAL CORTEX"

DAVID G. HARDY, B.Sc.

Stimulation of a sensory receptor initiates a nervous message, which, on arrival in the brain, undergoes modification; and may produce, in the mind, a related sensory experience. Some recent investigations on the vertebrate visual system have shed some light on the peripheral coding and central analysis of this kind of sensory message.

Some of the earliest investigations on the nature of the retinal message were made by Adrian and Matthews in 1927 and 1928, using eyes excised from conger eels. On recording action potentials from the whole nerve, they found that, in complete darkness, the nerve was almost wholly inactive; but illumination produced a discharge of high frequency impulses. These declined in frequency, on continued illumination, to a steady low level of activity, which lasted for the whole period of the illumination. On turning off the light, they noted a slight diminution of frequency (lasting for about 1 10 second), followed by a short-lived high frequency burst, and then complete cessation of activity. The latency of these responses, and their maximum spike frequency, were related to the quantity of light falling on the receptors in unit time; but this only held for retinal areas less than 1 mm in diameter, and was not effective with areas greater than this. Movement of a light, or shadow, was a more effective stimulus than a steady object. This then, was an "on" and "off" activity.

The activity of *individual* optic nerve fibres, and, therefore, of invidual ganglion cells, was first analysed by Hartline in 1938. Using the bullfrog's eye he exposed the fundus by removing cornea, lens and choroid, and dissected out single optic nerve fibres from the small bundles on the retinal surface. By this extremely difficult technique, he found that not all the fibres responded to the same stimulus. They seemed to fall into 3 main categories, which he named "on", "off", and "on-off". This diversity of the response was independent of the local physical conditions of stimulation (i.e. Temperature, pH, anoxia, etc.) and was probably, therefore, an inherent property of the ganglion cells.

The "on" type of cell (comprising 20% of the total investigated) responded when a light was on, and maintained its discharge as long as the illumination continued. Those that responded with a burst of impulses when the light was switched on, and again when it was switched off, but fell silent during continued illumination, were called "On-Off" cells (50% of total); while those that gave a short burst only on the "Off" of illumination, were called "Off" cells (30% of total). These categories were not rigid, however, and occasional

intermediate types were seen. The receptive field of each ganglion cell was about 0.5 — 1.0 mm. in diameter.

Some "On-Off" cells had a central plateau of greater sensitivity (its diameter varying between 0.2 and 0.6 mm). Light outside this area had to be about 1,000 - 10,000 times brighter to achieve the same response, and could *inhibit* the response which *would* have occurred if that part had not also been illuminated. This seemed to indicate an inhibitory surround to the receptive fields of the cells.

Although the essential character of the responses remained unchanged, the duration of exposure to "illumination" did affect the strength of the response: i.e. the strength of the "Off" response depended on the time of exposure to the proceeding light—the strength of the "On" response on the time of exposure to the preceding *dark*; while the "on-off" cells showed a combination of both. Re-illumination of the retina could suppress the "off" discharge and a strong response to small movements could be elicited in the "on-off" cells. Barlow, in 1953, using a Micro-electrode technique of recording from single fibres on the intra-optic (retinal) surface of the exised frog's eye preparation, confirmed most of these findings.

Also in 1953, Kuffler investigated the organisation of the receptive fields of single fibres in the mamalian optic nerve. By inserting micro-electrodes through the intact eyeball of a cat he could record from single units on the retinal surface. He found, contrary to Adrian and Matthews in the eel, that, in the dark adapted cat there was spontaneous activity in the absence of illumination. (A micro electrode placed in the centre of the optic disc recorded a mass spontaneous discharge, which could be suppressed by dim illumination of the whole retina.) With a small exploring spot of light and background illumination, he confirmed, for cats, that all units had a central area of greater sensitivity, with either "off" activity, or "on", predominating. For example, in the "on-centre" type, when the spot was shifted towards the periphery of the receptive field, the "on" activity decreased and "off" activity appeared. With the spot even further in its periphery, a zone of pure "off" activity could be detected. This arrangement was reversed in units with an "off-centre". Illumination of both areas together produced little or no activity. Thus each receptive field had a mutually antogistic centre-surround arrangement.

The role that this type of receptive field organisation played in the analysis of visual patterns in the frog, was investigated in 1959 and 1960 by an American team. This group, Maturana, Lettvin, McCulloch, and Pitts, used a series of stimuli which they thought might be significant to the frog in catching prey—small dark discs a few degrees in diameter, broad and narrow dark strips, small squares, etc. These could be used, moving or stationary, against a series of different backgrounds. Using these visual stimuli, and recording single units by Micro-electrode penetration of the optic nerve, they found that the ganglion cells had five different functional classes—one recording light intensity, and the other four each giving a maximal response to one quality, or configuration of qualities, in the visual stimulus. Each ganglion cell responded only to one type of configuration and any departure from this gave a reduction in, or disappearance of, the response. With the exception of the class that indicated light intensity, they seemed to be independent of the general illumination, the nature of the backgroumd, and the state of dark adaptation of the eye. The receptive fields for each of these 5 classes appeared to be uniformly distributed over the retina.

The first class called "sustained-edge detectors", responded to a sharp edge, either darker or lighter than the background, moved through the receptive

field of the cell. The response was largly independent of the shape of the object, or its curvature; but there seemed to be an optimal size, speed and position, for a maximal response. The activity could be suppressed by a switch to complete darkness, but was restored on restoration of the illumination. This group of receptors were equated with Hartline's "on" group. The second group were called "convex-edge detectors", and gave a maximal response to movement of a very small object 1-3° in diameter. They gave no response to straight edges, but were sensitive to corners, and tended to respond only to movements towards the centre of the receptive field. A light object on a dark background gave a poor response—unless it had a dark shadow. These two classes of ganglion cell were not entirely separable, and a small number were found to give an intermediate response. Thus the two classes may have represented the two peaks of a bimodal distribution, so that different cells might respond maximally to slightly different stimulus parameters (i.e. each may have a different optimal object size etc.).

The third class were sensitive to the "on" and "off" of light, and to movements in any direction in the receptive field. These "changing-contrast detectors", equivalent to Hartline's "on-off" grouping, gave no activity with a stationary contrast, but *did* respond—although poorly—to movements of a banded or complex background. The fourth class "dimming detectors", responded to the "off" of light, or to a moving object (irrespective of its size or shape) in proportion to the dimming it produced. These were thus equivalent to Hartline's "off" receptors; but changing conditions were essential for the responses, continued darkness soon ended the activity. The last class were the "dark detectors". These were found to be continuously active even in light, but their activity was inversely proportional to the light intensity, giving a maximal response in the dark. The retinal distribution seemed to be uniform for each type of ganglion cell receptive field, although the total numbers of each class differed considerably.

Similar results were obtained by recording from single units in the optic tectum of the frog (units thought to be the terminals of the ganglion cell axons).

Hubel and Wiesel, in a paper published in January, 1962, demonstrated the existence, in the cats visual cortex, of a system of organisation not unlike that in the frog. However, in the cat, these cells, which likewise responded to one invariable in the visual stimulus, seem to be of true cortical origin. In a previous paper (1959), they had shown by using a small light-spot stimulus, and recording by micro-electrodes from single units in the cat's striate cortex, that the receptive fields of these cells were divided into mutually antagonistic excitatory and inhibitory regions. These were not necessarily circular, or even symmetrical, and their arrangement determined the required stimulus size, shape and orientation for maximal response. In 1962, they again recorded from single units in the cat's striate cortex, but this time used a series of different stimulus shapes on a tangent screen background. With these, they found that the receptive fields of these cortical cells could be divided into two functional classes, "simple" and "complex" according to the type of visual stimulus needed to activate them.

The responses of the "simple" cells could be predicted by exploring the receptive field with a small light-spot. These had receptive fields divided into antagonistic, excitatory and inhibitory zones; and thus movement of an object out of an inhibitory, and into an excitatory zone would be a very effective stimulus. However the orientation of the stimulus was critical, as not all the

receptive fields had similarly arranged excitatory and inhibitory zones, for example—a narrow central zone with two antagonistic flanks, symmetrical or asymmetrical; large central zones and small antagonistic flanks; and those with one excitatory and one inhibitory region. (Here the most effective stimulus would be an "edge" moving one way only—from the inhibitory to the excitatory zone).

The "complex" cells had responses not predictable by mapping the receptive fields with small light-spots, which, if effective at all, gave just a mixture of "on-off" regions. They were of four main types, each activated by a different combination of stimulus parameters. The first type required a horizontal slit of highly specific size (1 8° wide 3° long), but the exact position of this slit in the receptive field was not critical, as long as it remained horizontal. An upward movement of the stimulus produced a discharge, a downward movement suppressed it, and if the slit was stopped, there was no discharge. The second type was also activated by a slit of similar dimensions, but oriented 10 o'clock - 4 o'clock. Movements either up or down stimulated it, but stopping it stopped the discharge, as did changing the orientation. The third type responded to an edge, vertically oriented, either with excitation or inhibition depending on whether the area of brightness was to the left or to the right, but again its position in the receptive feld was immaterial. The fourth type only responded to a dark bar on a light screen, not the converse. In the example studied, the orientation was critical (horizontal), and it was sensitive to downward movements only—giving a weak, inconsistent response to upward movement, and none at all to movements left and right. Except for the injured ones, all the simple and complex units that they recorded, showed some sort of response to a visual stimulus; but their receptive fields showed great variation in size (although the complex ones tended to be larger).

By recording from cells in sequence during one micro-electrode penetration, they found that successive cells tended to have identical axis orientation. But changes of angle did occur, and these were unpredictable. The cell types tended to be layered according, roughly, to the cortical layers, i.e. "simple" cells were confined mainly to layers 3, 4 and 6, while "complex" cells were extremely rare in layer 4. Although the receptive fields were of different size, there was at least a gross topographical point-to-point- representation of the retina preserved down through the columns, with, as usual in the visual system, a correspondingly large area devoted to the area centralis of the retina. This topographical representation seems to be an essential prerequisite of functional organisation in the vertebrate visual system, and has been seen at most levels (e.g. tectum or superior colliculus, lateral geniculate nucleus, and cortex) in most species (refs. 7 - 11). The magnification of the central area is likewise a common feature and may be related to the number of ganglion cells devoted to the central area (Jacobson, 1962).

It can, therefore, be seen from this type of experiment, that the mosiac of active and inactive receptors (rods and cones) must be of a rather specific pattern to activate a retinal ganglion cell in the frog. But, in the cat, the ganglion cells are activated by a less complex pattern of receptors (Kuffler's centre-surround arrangement), while the cortical cells require a *more* complex receptor organisation. Hubel and Wiesel have suggested a tentative scheme for the possible interconnections of just such a system. In this several centre surround ganglion cells connected in a specific way will determine the stimulus requirements of a "simple" cortical cell, and several of the "simple" type will determine those of a "complex" cell. Although only a few

preliminary experiments have been conducted on the primate visual cortex (monkey), these would seem to indicate that a similar arrangement—of cells with simple and complex receptive fields, grouped into discrete columns and preserving the point-to-point topography—may well exist, but large differences are likely.

The process whereby the geometrical arrangement of inter-connections between groups of neurones can serve to abstract, for a particular animal, the significant parameters from a mass of sensory data, and the central vertico-topographical segregation of these parameters, may well be a general principle of functional organisation in the vertebrate nervous system. Some work done by Mountcastle in 1957, on the somatic sensory cortex in monkeys, indicates a similar columnar organisation of incoming sensory data. But here, the sensory modality (e.g. touch, pain, pressure, etc.) seems to be the functional grouping used. Topographic representation of the body surface is likewise preserved. And Penfield, in his 1958 Sherrington Lectures, cites the case of a patient E.C., who had experiential hallucinations just before a major epileptic convulsion. In these, he (E.C.), always relived a childhood experience where he had snatched a stick from the mouth of a dog. Any subsequent experience where he saw "someone snatching something from someone' served to trigger off another attack; and electrical stimulation of the temporal cortical lobes did the same. Penfield suggests that this "snatching" no matter in what aspect, may result in a neurone impluse reaching the neurone pattern that records previous "snatching" experience, and that this might be thought of as a "*biological cross-indexing of abstract characteristics of experiences* so that each fresh example has its immediate connection with (similar) previous experiences".

I should like to thank Dr. Gaze for his interest and advice in the preparation of this article.

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31st October, 1962.

CONGENITAL DISLOCATION OF THE HIP

Diagnosis and Treatment

PETER J. SWARBRICK M.B., Ch.B.

Based on a Dissertation read before the Society on January 19th, 1962.

The subject of Congenital Dislocation of the hip (C.D.H.) is so vast and complicated that anyone who attempts to correlate the known facts and numerous hypotheses into a neat, compact monograph, is doomed to failure before he starts. One of the main difficulties is the almost universal disagreement about the various aspects of the disease, whether of actiology, pathology, diagnosis or treatment. In these pages, I shall confine myself to discussing some of the active steps one can take in the prevention and cure of the condition.

In very few orthopaedic conditions can early diagnosis have such a profound effect on the eventual result. No longer should we see the type of case so often described in the older textbooks—the waddling gait, hyperlordosis, and pelvic tilt of the fully developed bilateral dislocation. In this respect I find it rather disappointing to say the least that in the latest edition of one Paediatrics textbook, published in 1960, the author is forced to admit that "It is often not until the child attempts to walk that the diagnosis is made." By this stage much damage has already been done, and even if a good eventual result is obtained, treatment is considerably longer, and more complicated.

For this reason, it should be a routine step, during the early examination of the newborn infant, to carry out the few simple tests for instability of the hips, which will show up even at this early stage. Examination as follows takes very little time, and may save much unnecessary hospitalisation.

I. *Loosening of the Hip*—related to the flaxidity of the capsule due to the susceptibility to relaxin—is shown by a diminished active and exaggerated passive mobility.

II. *Assymetry of Buttock Skin Folds*—are very often seen in early cases, although not everyone agrees as to their reliability.

III. *Simple abduction* of the hips is probably the most important individual test—most cases show some limitation of abduction.

IV. The value of *Ortolani's "Click" Test* in the new born is disputed. In this procedure, during rectangular abduction, the femoral head can be felt to "click" over the edge of the acetabulum. Obviously, this requires some subluxation to be already present, and though some people regard it as a suitable test in the young infant, it is probable that it is of little value until later.

Should any of these clinical tests reveal suspicion of an abnormal hip, radiological examination should be made. X-rays should also be taken in any child born into a family with a history of the disease. There can be no excuse for ignoring the influence of heredity.

The chance of any abnormal hip being missed, and proceeding to subluxation can be minimised if, apart from routine examination in hospital-born babies, district Midwives are instructed in these simple clinical tests, and are shown the importance of early diagnosis. The truth of this statement has been seen in those parts of the Country such as Fife, where Midwives are given this instruction.

V. *Radiographic features at this stage.*

Straight X-ray shows a markedly sloping acetabular bony roof. The osseous nucleus of the femoral head is normally not present until 6 months, but in C.D.H. its development is retarded. Anteversion of the femoral head can be seen—the normally medial oblique direction of the femoral neck being changed to a fore-shortened, vertical position. On medial rotation of the limb the femoral neck once again assumes its correct relationship to the acetabulum.

Subluxation and Dislocation

Although all cases should be diagnosed at the first stage, there are still many instances in which the child is not seen until a year or more of age.

The mother often notices that the child is late in starting to walk, and is resistant to any encouragement. The signs of limited abduction, and assymetrical buttock skin creases, are, if anything, more pronounced, shortening of the leg is usually seen, and the phenomenon of "telescoping". The Trendelenberg test becomes positive as subluxation proceeds. The femoral artery, which in the normal lies immediately anteriorly to the femoral head, and can be felt pulsating, becomes implapable as the head rides up.

Eventually, in neglected cases, the features of gross dislocation are seen—widening of the hips, a marked limp in unilateral cases, and a waddling gait in bilateral cases, with hyperlordosis and a tilted pelvis.

It is when the hip has reached the stage of subluxation, that normal radiological control gives way to arthrographic control, and the latter is essential in order that adequate treatment can be carried out. The vital part that arthrograms play will be demonstrated when I consider treatment, but the essential thing that such a procedure shows, is whether the limbus is being everted by the femoral head, or whether the head has escaped the confines of of the limbus, which has become inverted. The value of arthography has by no means been universally accepted, partly because not everyone holds the view that the limbus plays such an important role in the treatment, and also partly probably because the technique of arthrography is not too easy, and needs constant practice to become familiar, both with technique, and with the evaluation of results.

Treatment

When considering treatment, I must of necessity, give mainly only a narrow selection of those methods that seem, in the light of present day knowledge and experience, to offer the best chance of success. However, I would like to mention briefly, some of the methods used at earlier times for the aleviation of the disease.

In 1701 Verdue reported a successful reduction of the dislocated femoral head of a cadavre by traction on the thigh. As soon as traction was released

however, redislocation occurred. Dupuytren denied any possibility of cure and deplored any attempt at treatment. In the late 1830's Hymbert and Jacquier used forced instrumental extension and attempted reduction in one sitting on a girl aged eleven—a pretty traumatic procedure. Pravaz attempted a much more gentle manoevre—maintaining traction on the thigh for 8-10 months. As soon as the head came down to the level of the acetabulum, he applied exaggerated extension, with abduction, and inward pressure against the greater trochanter. This method, which was a considerable advance, failed mainly because late cases were treated, and maintenance of the reduced position was unsatisfactory.

In 1888, Poggi carried out the first open operation, reducing the head, and fixing it in a surgically deepened socket. Hoffa and Lorenz improved this technique but this treatment, although its advocates claimed successful results, was not generally accepted. The hazards of operation were still too great, and some of the opinions of the results may be gathered from the comment of the contemporary surgeon—"Before operation, Hoffa's patients walk like ducks; after operation, they walk like operated ducks".

It was left to Lorenz to introduce a new concept. At the end of the last century, he suggested conservative treatment, and divided the two objects of treatment into separate, equally important parts—Reduction and Retention. His reduction was achieved at one sitting under general anaesthesia. Retention of the reduced position was held for a specific and prolonged period, exerting a stimulus on the acetabular cartilage during the stage of most extensive growth.

This met with considerable opposition at the time, from the then leading Surgeons, but has since been vindicated, and only recently has any method evolved which is likely to replace it.

In considering present day treatment, I should like to divide the topic into two parts—The Classical procedure as propounded by Lorenz and Hass, together with associated operative procedures, and the modern methods advocated by Mitchell, Scott and Somerville.

Firstly the *Classical Procedures*:

At an early stage, that is in predislocation and early subluxation, Hass recommends simple abduction on an abduction bar, until ossification of the acetabulum is satisfactory.

Late subluxation and dislocation are treated by manipulation under general anaesthesia. Preliminary longitudinal traction is applied to the limb, to attempt to bring the femoral head down, and to stretch the contracted soft tissues. After a fortnight's traction the following manoevre is carried out—While an assistant fixes the pelvis, the operator, maintaining longitudinal pull, flexes the thigh to 90°, thereby bringing the femoral head from a posterior and superior position to a position posterior to the acetabulum. The flexed thigh is now swung out into 90° of abduction, so that the femoral head now lies at the posterior rim of the acetabulum. Pressure on the greater trochanter should now push the head over the rim, and into the socket. The hip is now held in this "Frog" position by a hip spica. Hass's variation of Lorenz' original manipulation is to increase flexion to 100-125°, by which means he claims the head fits more deeply in the socket. Retention in this position is necessary for at least a year, after which there is gradual mobilisation. The degree of success of this method over a short period can be quite considerable, but long term results are much poorer, estimates of long term success varying from 60% to

as little as 10%. There are several possible causes for this—(1) Redislocation—due sometimes to the excessive anteversion, which may or may not correct itself spontaneously. Probably more often, redislocation is due to the interposition of the limbus between socket and joint. No doubt in some cases, the limbus atrophies, or the head accommodates itself to the reduced size of socket, but in many instances, redislocation occurs on removal from the spica, or on weight bearing. (2) Osteochondritis of the femoral epiphysis—according to Scott this occurs in about 25% of all cases reduced by this method. This is possibly due either to the force employed in the reduction, or to torsion of the capsular vessels in the "Frog" position. This is, to many people, a serious drawback to the procedure. Other disadvantages are the long time of plaster retention, and apart from the resulting detriment to the child, this imposes problems of nursing, toilet, etc.

There have been several operations devised, mainly for use in the later stages, when secondary changes have taken place, or closed reduction has failed. Lorenz used to gouge out the fat and connective tissue of the socket and, after traction, replace the head. There is obviously no chance of anything like a normal joint developing here.

Various forms of shelf operations are still carried out in many centres to keep the hip stable. Either an extra-articular buttress is constructed, extending beyond the joint margin, or else the slope of the acetabulum is altered by means of an incomplete osteotomy above the joint. This has the disadvantage of altering the contour of the socket, and so altering the internal congruity of the joint, predisposing to degenerative changes. A recent modification of this method has been introduced by Salter. He carries out an innominate osteotomy of the affected side, so that the whole acetabulum is rotated. Thus the joint surfaces are maintained in complete congruity and, by means of wide incision, he is able to reduce the head deep to the limbus. Early, short-term results appear very satisfactory.

Finally, there is the Colonna operation, in which the capsule forms an arthroplasty.

In recent years, there have been attempts to improve on the foregoing classical methods, both as regards final successful results, and also in the length of time necessary for treatment. Out of these endeavours has come a system of treatment that has developed largely due to the efforts of Mitchell in Edinburgh, and Scott and Somerville in Oxford. In most respects, the methods are the same in both places, and it is the Edinburgh procedure that I shall discuss.

At the stage of Primary Instability which will usually be detected soon after birth, treatment is simple. Abduction is maintained by means of about four napkins and, with satisfactory home conditions, maternal supervision with frequent radiological control is satisfactory. A stable hip, with satisfactory acetabular development, has usually come about by the normal age of walking. This prophylaxis is a safer method of treatment than that of "Controlled Observation", advocated by Leffmann. Even though he claims that the majority of radiologically unstable hips are normal by the age of eight months, there was the small number (5 out of a series of 80) who did not and whose active treatment was thus postponed several months.

Where more abduction is needed in early subluxation the Forrester-Brown Splint is highly satisfactory, and adjustment to it can be carried out on an out-patient basis.

We now come to the point where hospitalisation is needed, the subluxated

and dislocated stages. As I said before, arthrographic control is essential here.

Summary of the Procedure.

An arthrogram is carried out first to discover the position of the limbus. The immediate treatment is similar whether the limbus is everted (i.e. Subluxation) or inverted (i.e. Dislocation). The child is put in the Jones double abduction frame, and longitudinal fixed traction is applied to both lower limbs in the neutral position. The bottom of the bed is elevated to quite a considerble degree, to apply counter-traction. The child is left thus for 10 days —if the head of the femur is pulled down, abduction is started. The preliminary traction is most important, and probably many of the criticisms of the method stem from failure to carry this out efficiently. Abduction proceeds gradually—by one hole, alternate sides, each day. Sometimes, the abductors of the thigh are very tight, and require preliminary tenotomy. In most cases, however, gradual abduction is sufficient to stretch them. When 70° of abduction is obtained, the hip is re-X-rayed, and if cross-pull is required to bring the femoral head down to the socket, a weight varying from ½ - 2 lbs. is applied. This maximum is never exceeded in young children. The frame is then abducted to 90°, and pelvis again X-rayed. Longitudinal traction is loosened, the cot levelled completely, and the head allowed to ride into the socket.

At this stage, the procedure for subluxation and dislocation diverge. If the limbus was only flattened and everted in the original arthrogram, the hip is held in medial rotation to compensate for the anteversion and in slight abduction. Until recently retention in a Batchelor Plaster was practised, in which the abduction was increased to 90°, but this has shown some tendency to cause osteochondritis .

Should the limbus be inverted open removal of it is undertaken and the hip put in a plaster spica. If the degree of anteversion necessitates it, a derotational osteotomy is carried out a month later, and the child again immobilised in plaster spica for a further six weeks. The spica is then removed, hips are X-rayed out of plaster, and below knee traction is applied for a fortnight; The child then starts exercises in the swimming pool and physiotherapy, and is generally mobilised.

It will be seen that the length of treatment is considerably cut down by this method. Redislocation due to persistence of an inverted limbus, or to femoral anteversion, are eliminated. In Scott's series of subluxations treated on the frame up to 1953, he estimated the incidence of osteochondritis as 8%; more recently it has been shown that it is possible to reduce this complication to virtually nil by avoiding the use of the Batchelor Plaster.

One criticism of the method is that if the limbus is removed the child is subjected to several operations—the excision, the derotational osteotomy, and the later removal of the osteotomy plate. In this connection, it is interesting to note that Wilkinson has recently shown that maintenance of normal young rabbits' hips in medial rotation cause increased anteversion, whereas the frog position corrects this. One might suggest, therefore, that maintenance of the frog position after excision of the limbus might save the further osteotomy. However, with the possibility that this position might cause osteochondritis, it would be a rather risky procedure to carry out.

The above procedures have been found sucessful up to the age of six; after this secondary contractures make some such operation as the Salter innominate osteotomy the procedure of choice.

Summary.

In this short paper, I have only touched on a few aspects of the diagnosis and treatment of C.D.H. The methods are many, their protagonists and antagonists legion. Perhaps I may finish by quoting a remark by Oschner at the beginning of this century: "There's still quite a little that remains to be discovered regarding this disease." That has some claim to be the understatement of the century.