RES MEDICA
Journal of the Royal Medical Society

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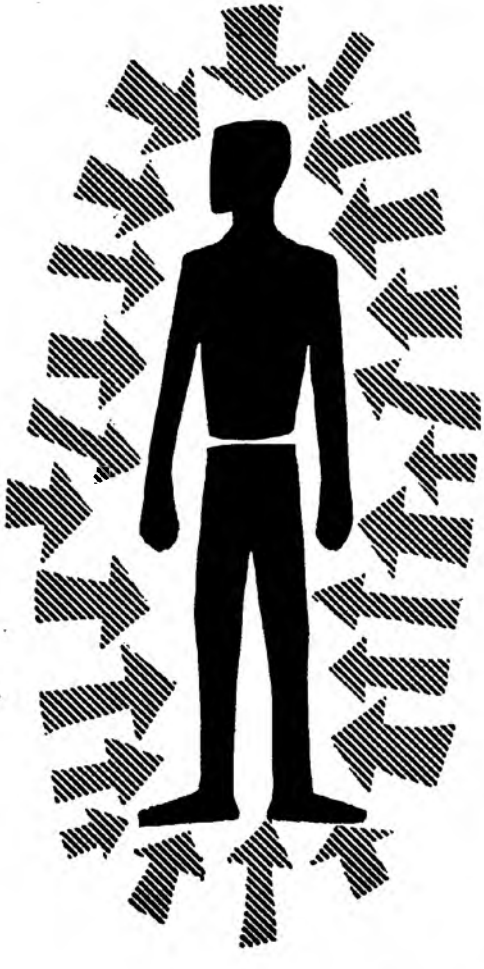

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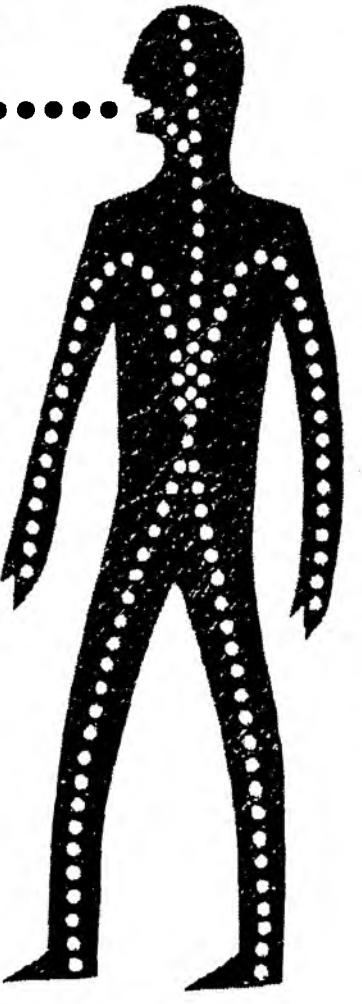

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ABDOMINAL CRISES

II.

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

The second of three articles written by the Author for Res Medica.

ABDOMINAL TRAUMA

With national concern critically focussed upon the increasing problem of accidents it is well to remember that although the vast majority of injuries affect the extremities and although these injuries are a cause of great morbidity it is injuries to the head, the chest and the abdomen which kill. The closed head injury of civilian life is more misleading and often more rapidly fatal than many open wounds where the brain is exposed. So with abdominal injuries the intact abdomen may make diagnosis difficult and encourage a false sense of security. If the abdominal contents are damaged, two disasters may befall. First—and most urgent—is internal haemorrhage from lacerations of the solid viscera, especially spleen or liver, or from tearing of vessels. Later, if hollow organs have been damaged, the escape of their contents will lead to peritonitis.

MULTIPLE INJURIES

Early assessment is most difficult in the patient with multiple injuries who is also unconscious. In the conscious patient a complaint of abdominal pain will usually focus attention on the possibility of intraperitoneal injury and where bleeding is severe, spasm or even rigidity of the abdominal muscles is the rule. In the unconscious there is no such complaint and muscle tone may not be demonstrably increased, but tenderness, the great localiser, often helps. Firm palpation of the abdomen evinces a response by cry or grimace or movement which suggests strongly that all is not well. But in the most urgent cases the problem is the diagnosis and treatment of a severely shocked patient. Management is simplified when we remember that head injuries do not in themselves cause shock and that the presence of a head injury in a shocked patient is never a contra-indication to transfusion. Indeed, the level of consciousness often improves when the circulation is restored.

If the rapid transfusion of blood or plasma expanders in the order of 1 litre in ten or fifteen minutes fails to restore the circulation then either uncontrollable internal bleeding is continuing or there is some other cause for the circulatory collapse. Central (cardiac) failure will be shown by distended neck veins ; cardiorespiratory failure is a likely explanation of continued shock

if there is a chest injury with paradoxical respiration and this must be controlled immediately by positive pressure respiration coupled with underwater drainage of the pleural cavity on the injured side or even bilaterally; the possibility of adrenocortical failure will be suggested by a knowledge of previous adrenalectomy or steriod therapy and confirmed by the dramatic response to intravenous steroids.

If shock persists despite these measures then internal bleeding is the presumptive cause and this is even more certain if initial reponse to rapid transfusion is followed by deterioration when the rate of transfusion is slowed down. Under these circumstances the abdomen must be opened forthwith, unless profuse bleeding from a pleural drain or cardiac tamponade in a case of chest injury dictates that thoracotomy be carried out first. When laparotomy is carried out for intraperitoneal haemorrhage it must never be forgotten that the induction of anaesthesia which abolishes vasoconstriction and relaxes the abdominal muscles may destroy circulatory compensation and allow further torrential bleeding. The surgeon must, therefore, be ready in the theatre and a full bottle of blood in place on the transfusion set before the anaesthetic begins.

Controllable major bleeding following trauma comes most often from upper abdominal injuries where liver or spleen are torn. In the case of the spleen the treatment is always splenectomy. The liver, on the other hand, must be sutured—a matter of considerable difficulty in many cases due to the consistence of the organ and the frequency with which lacerations involve the superior surface necessitating a thoraco-abdominal approach. Rarely, a week or two after apparently successful suture of the liver, the patient develops melaena or haematemesis due to the discharge of a deep-seated intrahepatic haematoma through the billiary passages. This form of internal secondary haemorrhage is always fatal unless further surgery with full exposure and drainage of the central hepatic cavity is undertaken.

When severe intraperitoneal bleeding follows major injuries of the lower abdomen or pelvis it very often arises from tears of major vessels such as the iliac veins and is nearly always fatal before effective treatment is available. Profound shock does not follow uncomplicated fractures of the pelvis and is an indication for immediate laparotomy.

INJURIES OF THE ABDOMINAL WALL

The diagnosis of underlying visceral damage is often confused by the presence of tenderness due to parietal injury. This is well recognised in relation to RUPTURES OF THE SPLEEN where there is often no more than tenderness and bruising over the lower rib for many days. The possibility of a subcapsular tear of the splenic pulp must be foremost in the doctor's mind in all such cases and the patient kept within easy each of medical aid for at least two weeks lest disastrous bleeding suddenly occurs. BOWEL, like the spleen, may be the site of a delayed rupture following an intramural haematoma and a blow sufficient to produce obvious bruising of the abdominal wall is always an indication for careful observation over the following two weeks.

When examining a patient who has sustained a blow on the abdomen it is important to note whether an EXTERNAL HERNIA is present. When the

intra-abdominal pressure suddenly rises, bowel within the intact peritoneal cavity is subjected to a uniform increase in pressure on all sides, but if a loop of bowel lies across or in a hernial sac the localised lack of support in relation to the defect in the abdominal wall greatly increases the chance of rupture at that point.

Of course, the injury may after all be confined to the abdominal wall. Contusion of the abdominal muscles is very common and rarely actual rupture may occur, as in the rupture of the lower part of the rectus abdominis which is classically associated with the spasms of tetanus or with sudden contraction of a muscle weakened by pregnancy or obesity. I have seen one case where the rupture occurred after trivial strain in an apparently fit young man. The diagnosis of leukaemia was confirmed from the blood aspirated from the haematoma which, as usual, tracked up behind the muscle to give a palpable hypogastric swelling. The diagnosis was suspected because of previous experience with leukaemia presenting as a partial rupture in other muscles.

THE DIAPHRAGM may suffer a peripheral detachment by coughing or vomiting or by crushing injuries which spring the chest wall. In minor cases the pain is similar to that of fractured ribs, but corresponds to the attachments of the diaphragm, is not associated with superficial tenderness and is not greatly relieved by strapping of the chest wall or infiltration with local anaesthetic. The symptoms subside spontaneously over two to three weeks. When the dome of the diaphragm is torn there may be immediate haemothorax and haemoperitoneum. The injury is usually a severe crush and laparotomy or thoracotomy is often indicated regardless of the suspicion of diaphragmatic damage. An additional complication is herniation of the stomach or, less often, the colon into the left hemithorax with acute gastric obstruction and severe cardiorespiratory embarrassment. Such herniation may be suspected from the history and clinical picture, especially when bowel sounds or gastric succussion are heard in the left side of the chest. Immediate operation is required.

Stab Wounds

Carrying the same diagnostic and therapeutic difficulties as the closed abdominal injury is the apparently trivial stab wound. A Wolf Cub was brought to hospital one ovening. On his abdomen was a tiny cut, 5 mm. long, just to the right of the umbilicus. He had been whittling wood the wrong way and when the knife slipped the blade—the small blade of a boys' penknife—pierced his clothes and entered the abdominal wall. There were neither symptoms nor signs to indicate intraperitoneal damage. Six hours later I opened his abdomen because of signs of spreading peritoneal irritation and found bleeding from an artery in his transverse mesocolon. The 3 cm. blade had not only penetrated his clothes and his abdominal wall, but had passed through the mesocolon to nick the anterior longitudinal ligament between aorta and vena cava! Any stab wound, however small, between clavicle and groin must be assumed to have penetrated the thoracic or abdominal cavities and demands admission to hospital.

PANCREATIC INJURIES

The pancreas is rarely injured alone although it may be contused—or even severed—by crushing injuries as it lies across the front of the vertebral column. Early diagnosis can be no more than conjecture and every surgeon knows to search the area of the pancreas and retroperitoneal duodenum when exploring the abdomen after crushing injuries. If found to be severed, the distal part

of the gland may be excised or an attempt made to repair the damage. The former course is probably the safer but whatever is done the area must be drained as fistula formation is common.

Pancreatic injury, like any other retroperitoneal injury, will cause a severe paralytic ileus. Acute pancreatitis is a rare complication of injury but not uncommonly a pseudo cyst of the pancreas forms slowly over two or three weeks. This may be a self-limiting collection of blood and exudate in the lesser peritoneal sac or its contents may be replenished by a tiny fistula from an injured pancreatic duct. The patient will complain of slowly increasing, continuous, boring upper abdominal pain often radiating to the back and sometimes accompanied by recurrent vomiting. There is a tender upper abdominal fullness and characteristic forward displacement of the stomach in the lateral x-ray. Simple external drainage will cure the peripancreatic haematoma but where a fistulous communication exists between the cyst and the pancreatic duct system an external fistula is avoided by draining the cyst internally into the stomach or small bowel.

Injuries to the Renal Tract

RENAL INJURIES seldom present as abdominal crises although the diseased kidney which is especially liable to rupture, may occasionally cause intraperitoneal bleeding simulating an acute abdominal emergency. Contusion of the kidney may accompany a ruptured spleen and the urine should always be carefully watched in such cases. The management of renal injuries is essentially conservative. Only if haematuria is excessive or an increasing retroperitoneal haematoma is present will surgery be undertaken. It is, of course, essential to confirm the presence of a functioning kidney on the uninjured side and this is most simply done by an intravenous pyelogram which is part of the emergency investigation of any case of renal injury. It must be remembered that a shadow will not be obtained on either side in the shocked patient—the investigation must be delayed until a reasonable blood pressure has restored glomerular filtration.

RUPTURE OF THE BLADDER may occur as an isolated injury from a blow on the full bladder but typically it accompanies a factured pelvis. Indeed, the immediate danger of pelvic fractures lies only in their association with damage to the bladder, rectum or great vessels. Ruptured bladder should always be suspected if the patient cannot pass urine, if he passes blood-stained urine, if he has strangury or if there is lower abdominal swelling or tenderness. Intraperitoneal rupture will quickly produce signs of general peritonitis but extraperitoneal rupture will give a slowly progressive pelvic cellulitis which spreads to the anterior abdominal wall and the inguinal regions. A catheter is passed into the bladder and, if the clinical picture is in doubt, an x-ray is taken after the injection of a small amount of contrast medium. The bladder should never be filled with fluid to test for suspected rupture as this merely increases the spillage. With either type of rupture immediate suture of the rent with continuous bladder drainage is required.

Injuries of Hollow Viscera

Apart from penetrating wounds rupture of the stomach is very rare. The colon, especially the caecum, may occasionally be ruptured in a closed injury, as was seen in underwater blast injuries during the war, and the possibility of delayed rupture of the caecum must be remembered after any blow in the right iliac fossa.

Wounds which involve the COLON are particularly dangerous. When the retroperitoneal portions of the bowel are involved a fulminating retroperitoneal clostridial infection is apt to follow. This type of injury is of some historical interest to Edinburgh University for it was in 1915 that Captain Fraser, later

Professor Sir John Fraser, first reported from France the use of Edinburgh University Solution (Eusol), developed a few months previously by Professor Lorraine Smith, in the treatment of gunshot wounds of the colon with retroperitoneal gas gangrene.

The SMALL BOWEL is most vulnerable at its fixed points where it cannot move away from a sudden impact. Ruptures from closed injuries are thus most common in the third part of the duodenum, near the duodenojejunal flexure, or where the gut is anchored by adhesions. The dangerous role of external hernias in this connection has already been mentioned. The ruptured bowel may show prolonged tonic spasm of its muscular wall so that peritoneal soiling is sometimes delayed for many hours.

Perforations from Within

Swallowed razor blades and other sharp objects may occasionally perforate the stomach while fish bones and, in some countries, toothpicks (as much part of the American way of life as the Martini and the Club Sandwich which they accompany) may penetrate the small intestine but perforations have been more commonly recorded in the large intestine after such diagnostic procedures as sigmoidoscopy or barium enema, or following the insertion of foreign bodies into the rectum.

On occasion, intraperitoneal performation of the BLADDER has followed the injudicious use of a catheter with a rigid or semi-rigid tip (such as a Tieman's catheter) for continuous bladder drainage. Only a soft, blunt tipped catheter should ever be left for long in an empty bladder.

ACUTE INTESTINAL OBSTRUCTION

PATHOLOGY OF ACUTE OBSTRUCTION

Obstruction to the passage of food through the alimentary canal may be primarily mechanical, vascular or neurogenic.

Intestinal obstruction of purely vascular origin is generally the result of thrombosis or embolism within the mesenteric vessels and is due to ischaemic paralysis of the intestinal muscle.

In mechanical obstruction the blood supply may be primarily compromised by the same mechanism which obstructs the bowel lumen, as in volvulus, or secondary strangulation may develop later due to increasing tension within the bowel. This latter mechanism is most commonly seen in the obstructed loop of small bowel within a hernial sac but occurs also in large bowel obstruction when a competent ileocaecal valve prevents decompression into the ileum. In this circumstance a "closed loop obstruction" is formed even in the absence of twisting or misplacement of the colon and, because the tension on the wall of a tube of varying diameter varies with the diameter, the risk of intramural strangulation of this kind is greatest in the caecum. In neurogenic ileus the muscular paralysis allows a much greater degree of generalised distension without the danger of intramural strangulation.

In the small intestine distension is mainly due to fluid. The greater part of the fluid is retained gastro-intestinal secretions. Although lymphatic absorption from distended bowel is increased, venous absorption is reduced by up to 80 per cent so that a vicious circle of fluid loss becomes established, and the importance of this fluid loss is obvious when one remembers that the

secretion of digestive juices amounts to some 8 litres in 24 hours, or rather more than twice the plasma volume. Generally speaking the jejunum decompresses itself proximally into the stomach and the colon often decompresses proximally into the ileum. Therefore it is in the ileum that retention of fluid is usually greatest and the reduction of venous absorption most serious.

The gas present is mostly derived from swallowed air and only a small part is due to diffusion into the bowel or bacterial fermentation. Because of differential diffusion rates the oxygen of swallowed air is absorbed so that the residue is chiefly nitrogen. Reduction in the proportion of nitrogen in the inspired air by the administration of oxygen will reduce the nitrogen tension in the blood and so facilitate the diffusion of nitrogen from the lumen of the bowel. This method of therapy may occasionally relieve distension but is usually disappointing owing to the preponderance of fluid in distended small bowel. Colonic distension is largely gaseous but most of this results from bacterial activity.

Distension at first increases tonic muscle activity in the bowel so that obstructed bowel becomes shortened by as much as one-third of its normal length and its wall becomes thick and oedematous. Apart from the local effects, gross intestinal distension may cause serious cardiorespiratory embarrassment especially in obese or emphysematous patients.

When secondary strangulation is present the vascular obstruction is initially venous and the rate and amount of exudate and distension are therefore increased, but in primary strangulation when arterial inflow alone is cut off—as in mesenteric arterial thrombosis or when both arterial and venous flow cease simultaneously, as a volvulus—there is little distension and no gas in the affected loop making diagnosis exceptionally difficult.

Once the circulation has stopped in a segment of bowel there is a marked tendency for venous thrombosis to extend proximally. Because of this, apparently normal bowel proximal to a strangulated segment may later necrose as a result of venous infarction and this sequence of events explains some distressing failures after resection of strangulated bowel.

The time taken for strangulation to produce necrosis of the bowel wall is so variable that a short history must never permit a sense of security. Such factors as local mechanical pressure will hasten the onset of necrosis—the constriction ring at the neck of a strangulated hernia is the first part of the bowel to become non-viable. The role of infection has been the subject of much experiment and there is no doubt that once viability is impaired infection can hasten the onset of necrosis and that, at least in dogs, the survival of strangulated bowel is greatly prolonged by prior administration of antibiotics. There is, however, little evidence that systemic absorption of bacterial toxins is important until necrosis of the bowel wall has taken place.

When the obstruction has been relieved it must not be assumed that all danger is past. The risk of retrograde venous thrombosis has already been mentioned. The released bowel elongates and relaxes providing a large cavity into which fluid passes at a dangerous rate through the walls of capillaries damaged by anoxia. This explains the more rapid death of animals after release of a long loop of strangulated ileum as compared to a control group in which the strangulation was not released.

DIAGNOSIS OF OBSTRUCTION

Three questions must be answered in every case where the diagnosis is considered. Is mechanical obstruction present? What is the site and degree of the obstruction? Is it strangulated?

The cardinal symptom of mechanical obstruction is INTESTINAL COLIC, felt usually as waves of non-radiating pain in the central abdomen. Auscultation will reveal that the waves of pain coincide with spasms of increased intestinal activity. Vomiting first of gastric and later of intestinal contents is characteristic but may be delayed in low obstructions while in high obstructions the vomitus will never become "faecal." Similarly, distension will be absent in the early stages of obstruction and will never appear at all in really high obstruction.

These facts are not sufficiently appreciated. After a jeep accident a young airman was admitted to my unit in the Middle East with a fractured 2nd lumbar vertebra. Despite a ravenous appetite he vomited all his food a few minutes after ingestion. For several days I foolishly insisted that a scaphoid abdomen, normal bowel sounds, a normal gas pattern on abdominal x-ray (see below) and the fact that the vomitus remained dark green all indicated a neurogenic obstruction. In fact, this clinical picture was pathognomonic of a very high mechanical block and at laparotomy the first loop of his jejunum lay trapped and necrotic between the 2nd and 3rd lumbar vertebrae. For once the anterior longitudinal ligament had given way and allowed the vertebral bodies to open and close like a vice, catching a loop of bowel in the process. That this bizarre mechanism of obstruction had not been previously recorded was no excuse for failure to recognise that mechanical obstruction of some kind was present and the confusion of thought which led to this young man's death has been on my conscience ever since.

Absolute constipation is a confirmatory symptom but, like "faecal" vomiting, comes too late to help in timely diagnosis. Undoubtedly, the most valuable aid to diagnosis is the plain x-ray of the abdomen. The presence of GAS SHADOWS in the small bowel is almost always pathological and this is the basis of radiological diagnosis. On this criterion it will be appreciated that x-rays will not give confirmatory evidence in cases of very high small bowel obstruction or in those primary strangulations of small bowel where no gas is trapped. Therefore, a negative x-ray in a patient with severe intestinal colic and vomiting is an indication for urgent laparotomy. In all other types of obstruction the combination of distended loops of bowel seen on the film taken with the patient supine and fluid levels seen in the film taken with the patient erect or in the lateral position will provide evidence not only of the presence of obstruction but of its site.

The presence of strangulation can be surmised from various signs—continuous pain is superimposed upon the original colic; local tenderness and rebound tenderness indicate spreading peritoneal inflammation; if a neoplastic or inflammatory mass can be excluded a palpable tender mass is highly suggestive of strangulation. What is much more important from the practical point of view is that the presence of strangulation can rarely be excluded with certainty.

MANAGEMENT OF INTESTINAL OBSTRUCTION

The introduction of a simple method of INTESTINAL DECOMPRESSION by continuous suction was a major advance in the management of intestinal

obstruction. Ideally a tube carrying an inflatable balloon near its tip should be passed through the stomach and carried by peristalsis progressively down the bowel until the point of obstruction has been reached. In this way the whole bowel above the obstruction will be effectively decompressed. Unfortunately, with the reversed peristaltic gradient found in acute obstructions it is rarely possible to induce the balloon to pass beyond the pylorus and in practice suction is applied to an indwelling gastric tube which must be of large bore—a Ryle's or similar fine tube cannot possibly cope with the large volumes of fluid which accumulate in the stomach and only creates a false sense of security. Suction should at first be continuous and applied with a negative pressure of about 75 cm. of water.

It must be appreciated that, except in obstructions due to neurogenic ileus or extensive adhesions (which make strangulation by herniation or volvulus virtually impossible) suction is not in itself a treatment for the obstruction. Successful suction will quickly relieve the patient of vomiting and colicky pain and the abdominal girth will diminish progressively as judged by repeated measurement. The intestine will recover its tone making surgery easier and safer—but operation remains necessary to deal with the mechanical problem. Wherever strangulation seems possible, and this includes the vast majority of cases, suction is to be regarded as no more than a necessary preoperative measure and operation will be carried out as soon as the patient's condition permits.

In those few cases where suction is continued as the definitive form of treatment success will be indicated by the appearance of gas in the colon on x-ray examination and later by the passage of flatus. The amount of fluid obtained by suction will diminish progressively and its colour will revert to the pale green or colourless fluid of normal gastric aspirate. Continuing dark green aspirate should suggest the possibility of an unrelieved high jejunal obstruction (as in the case described above). During the period of gastric or intestinal suction it is a common error to allow patients a limited intake of water by mouth in an attempt to assuage their thirst. Swallowed water is quickly aspirated with its added quota of salts. The patient's fluid and electrolyte imbalance is increased while thirst is not relieved. Careful mouth toilet and ice to suck will keep the patient tolerably comfortable until thirst is relieved by correct intravenous replacement.

The fluid aspirated by suction is fluid which has long since been lost from the tissues and INTRAVENOUS REPLACEMENT is urgently needed. It is impossible to correct at once the disturbances of electrolyte balance which may have developed over many hours or even days and to delay operation while such an attempt is made is unnecessary and unwise. In the acute obstruction of less than two days' duration electrolyte disturbances are not in themselves likely to add any risk to operation, therefore their correction will not make operation safer. The danger is that of acute peripheral circulatory failure and this is avoided by immediate transfusion of plasma or blood to restore the depleted blood volume. Operation is now safe and the long term restoration of the body's water and electrolyte pattern can proceed at leisure during and after surgery.

The use of enemata is confined to cases of large bowel obstruction especially in the left colon, where impaction of a scyballous mass in a malignant stricture is often the precipitating factor. If this is the case, an enema may tide over the emergency and allow later elective surgery under optimun conditions.

SPECIAL TYPES OF OBSTRUCTION

INTRALUMINAL OBSTRUCTION is relatively uncommon. In meconium ileus the infant's small intestine is blocked by inspissated meconium. The local

condition is due to an absence of normal pancreatic secretions and is part of a more general secretory defect known as mucoviscidosis. The defect, which is hereditary, is characterised by an excessively high salt content in sweat, saliva and sputum. This peculiarity is used in diagnosis and helps to explain the electrolyte difficulties into which these children may run in hot climates and the susceptibility to pulmonary infections which is such a common cause of death (staphylococci thrive in the high salt concentration of the bronchial secretions). Other anomalies such as bronchiectasis or cystic disease of the lungs are often present.

IMPACTION OF FAECES in the rectum may lead to a subacute obstruction in the elderly and bed ridden but more usually causes spurious diarrhoea. The wedging of a faecal mass within the stricture of a colonic carcinoma has already been mentioned. A more exotic type of obturation obstruction is GALL-STONE ILEUS due to ulceration of a gall-stone into the small bowel where it usually becomes impacted toward the lower end of the ileum. Occurring typically in elderly women with a long history of biliary dyspepsia the shadow of the stone may be seen on a straight x-ray which may also show gas from the intestine outlining the biliary tree. Less well known is obstruction due to a bolus of vegetable matter. This is found almost exclusively after gastrectomy or gastroenterostomy and is nearly always due to orange pith.

INTRAMURAL OBSTRUCTION may be due to congenital stenosis or atresias. Stenosis is usually at duodenal level and usually presents early with severe vomiting though an occasional case has first come to hospital in adult life. Atresias are most common in the ileum and are often multiple. They always present as acute obstruction in infancy. Imperforate anus might be included in this group.

Acquired strictures as sequelae of trauma, ischaemia or infection are unlikely to present as abdominal crises, but Crohn's Disease occasionally produces acute obstruction. In the large intestine diverticulitis and segmental colitis may both cause acute obstruction and in people who have been abroad an amoeboma may do likewise.

The majority of intramural obstructions are due to tumours. In the colon carcinoma is by far the commonest tumour but in the small bowel benign tumours causing obstruction by intussusception are relatively common whereas malignant tumours cause obstruction very late if at all. An unusual "tumour" which may obstruct the small bowel is a haematoma arising in patients with purpura or an anti-coagulant therapy.

EXTRINSIC OBSTRUCTIONS are most commonly due to hernias but obstruction by adhesions and bands comes a close second. The liability of every obstructed hernia to become strangulated makes early operation imperative. The diagnosis should be easy yet the absence of intestinal obstruction in a Richter's hernia, where only part of the circumference of the bowel is trapped, and the concealment of the femoral hernia in a fold of fat in obese female patients still give rise to disastrous errors in management. Conversely it must be remembered that a painful, tender inguinal hernia is strangulated only if irreducible. I have seen a perforated peptic ulcer diagnosed as a strangulated inguinal hernia because of the tenderness produced by an accumulation of peritoneal fluid within the wide-necked hernial sac.

Although bands and adhesions may be congenital, for example the band connecting a Meckel's diverticulum to the umbilicus or the adhesions between an undescended caecum and the right side of the abdomen which obstruct the duodenum in infants with malrotation of the gut, they are most commonly post-operative. Adhesive obstruction may develop very early after an operation and the distinction between post-operative neurogenic ileus and a mechanical

obstruction can be extremely difficult. The risk of strangulation is much greater with narrow bands than with broad adhesions. Such bands take time to form and it is therefore in the obstruction arising months or years after an abdominal operation that operation is most urgent, but even in the early post-operative period it is never wise to assume that ileus alone can explain distension and vomiting for more than a week especially after any operation in which loops of bowel have been anastomosed or exteriorised. Exploration usually reveals an internal hernia or a loop of bowel adherent to the operation wound.

VOLVULUS. Congenital errors in rotation and fixation of the gut are responsible for volvulus of the whole small intestine which is a common type of obstruction in the newborn and for volvulus of the caecum which may occur in adults. Abnormal fixation of the apex of an intestinal loop by a Meckel's diverticulum or an adhesion is responsible for volvulus of an isolated loop of small bowel. Volvulus of the pelvic colon, the commonest type encountered in adult practice, is much rarer in this country than in poorer communities due to the increased length and bulk of the pelvic colon which results from a vegetarian type of diet. The patient is usually elderly and the clinical picture is dominated by the rapid development of enormous gaseous distension. The radiological appearance is diagnostic. Except in the very early stages the twist is too tight to permit passage of a rectal tube into the obstructed segment and operation is urgently required if gangrene is to be forestalled.

INTUSSUSCEPTION in infancy nearly always arises in the ileocaecal region in a healthy baby without any underlying pathology in the bowel. The recurring spasms of severe colic and reflex vomiting with intervening periods of complete relaxation during which a mass is usually palpable and the presence of blood and mucus on the rectal finger are diagnostic of this type of strangulation. An opaque enema will not only confirm the diagnosis but will in many cases produce complete or partial reduction. When operative treatment is considered necessary, as it is in most cases, it is a matter of urgency but must always wait until shock has been corrected and lost fluid at least partially restored because infants tolerate dehydration very badly.

In adults intussusception is generally induced by some primary bowel pathology, and a simple tumour of the ileum is the commonest cause. Adult cases are therefore usually of the ileo-ileal type

VASCULAR OBSTRUCTIONS. Occlusions of small mesenteric vessels by embolus or thrombus are not uncommon in obliterative arterial disease. They may be silent or result in episodes of abdominal colic and distension. Tests for occult blood in the stool are positive. Such episodes usually resolve spontaneously although rarely a late result may be the development of a fibrous stricture in the involved segment of bowel. When major mesenteric occlusion occurs necrosis of the ischaemic bowel will usually follow. While resection of many feet of gangrenous small bowel is compatible with survival the surgeon will often find the entire small intestine to be involved. In a few such cases embolectomy or disobliteration has been undertaken with success and in one or two others immediate heparinization has resulted in survival of the involved bowel by preventing a spreading thrombosis. However, the mortality in this group of patients remains very high.

NEUROGENIC ILEUS. Autonomic dysfunction may result in increased tone in the gut. Thus in achalasia of the oesophagus or Hirschsprung's disease of the colon the aganglionic segment remains contracted and the colic of lead poisoning was referred to in the first article of this series. More commonly decreased muscle tone and diminished peristalsis are found and the condition is then known as paralytic ileus. This condition may follow trauma,

both intraperitoneal and retroperitoneal. It is a common complication of fractures of the spine or pelvis, and of operations on the abdominal aorta or lumbar sympathetic chains. Infection in the peritoneal cavity always results in decreased activity of the intestine in the inflamed area—Nature's splint, as it were, to prevent the spread of infection and allow rest to the inflamed part. Uraemia and severe cellular potassium deficiencies such as may develop in patients after long periods of gastric suction without potassium replacement, likewise cause paralytic ileus. Generally speaking surgery has nothing to offer in such cases and conservative treatment by intestinal suction and correction of fluid and metabolic disturbances is successful.

NERVE CELLS AND NEUROGLIA

By D. WHITTERIDGE, F.R.S.

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There are said to be 27,000,000,000 nerve cells in the human brain and there are about ten times as many neuroglial cells. On the whole, nerve cells are large and glial cells are small, so that their weights are about equal. The cytology of nerve cells does not provide any really startling information. There is a nucleus and a nucleolus complete with sex chromatin, and in the cytoplasm there is Nissl substance formed of RNA and endoplasmic reticulum, mitochondria carrying respiratory enzymes, and lipochromes. Fairly certainly the RNA is concerned with the synthesis of proteins which move out along the axon, and the rate of protein synthesis is increased during axon regeneration. The rate of oxygen uptake is very high, and weight for weight is said to be higher than that in any other cell. Its measurement presents great difficulties, as oxygen uptake of nervous tissue *in vivo* gives values 5 to 100 times those obtained in vitro. The metabolic activity of nerve cells is believed to increase about five times during and after nervous activity. At the same time, the amounts of cytochrome oxidase and RNA present increase, and there is an increased ammonia production suggesting proteolysis. Excessive activity is said to cause a decrease in cytoplasmic protein and a decrease in RNA. There is a vast literature on cytological changes in cells in overaction, exhaustion and after deprivation of sleep, but there cannot be said to be a coherent body of observed facts, let alone a satisfactory biochemical interpretation of the facts.

When we turn to the glial cells, we find they are divided into macroglial and microglial. The microglia are made up of astrocytes and oligodendroglia, and these cells may have a common origin. Astrocytes are large star-shaped cells with one or more processes which have end-feet closely applied to a capillary. The processes form fibres which provide mechanical support and perhaps exert tension. The oligodendroglia have fewer dendritic processes and are found especially in white matter where they seem, according to Peters of the Department of Anatomy in this University to be responsible for the myelin sheath, just as Schwann cells provide the myelin sheath of axons in the peripheral nervous system.

The microglia are phagocytic cells. They have been alleged to be mesodermal in origin, though others say they arise from the edge of the neural crest. Certainly they invade the foetal brain at a later stage than the macroglia. They pick up blood cells and lipoid material from disintegrating myelin and remove them from the site of a cerebral haemorrhage.

The glial cells have been studied in tissue culture, in which oligodendroglial cells can be seen contracting and relaxing in a cycle lasting 1-2 minutes. Whether this has any bearing on their function in the brain is not known. Astrocytes forming fibres can also be seen clearly in tissue culture. Pinocytosis, the ingestion of droplets of fluid at the free ends of dendrites, can also be seen in tissue culture.

The most startling fact about glial cells has been produced in the last few years by the electron microscopists, particularly Wyckoff & Young (1956) working on the brain, and recently by others on the retina. This is that in the brain there seem to be no cell spaces, that nerve cells are closely surrounded by glial cells everywhere except at synaptic junctions, where the cell membranes of two nerve cells are thickened or electron-dense, separated by about 200 Angstrom Units, and with an abundance of vesicles in the adjacent cytoplasm of one of the nerve cells. Exactly similar statements are made for the retina, which is, of course, embryologically of the same origin as brain. This is disconcerting to the neurophysiologist who is used to extracellular currents, which in peripheral nerve undoubtedly do flow in extracellular fluid and are responsible for part of the local current flow produced by the nerve impulse. The impedance of the cell wall of glial cells is not known, but it looks as if local circuits set up by impulses in nerve cells have to flow through glial cells.

The other implication of the denial of an extracellular space by electron microscopists is that the nerve cells must have glial cells as intermediaries in the supply of metabolic requirements from the blood stream and in the removal of waste products. This seems inescapable, and though diffusion would presumably slow up the carriage of glucose, etc., the existence of end-feet on the capillary would be satisfactorily accounted for. There are a few odd observations on the metabolism of glial cells, including the statement that their O_2 uptake decreases during cerebral activity.

The absence of an extracellular space in the brain would fit in well with the old observation of Ehrlich that trypan blue in the blood stream passes into the substances of all organs except the brain. This has led to the idea of a 'blood brain barrier' which excludes dyes, sodium thiocyanate and many other substances. The **behaviour** of substances which move slowly into the brain, and these include water labelled with deuterium, suggests that they undergo active transport probably across a cell membrane. It is an attractive suggestion that the cell membranes concerned are those of glial cells.

This view has been criticised on the ground that the tissues prepared for electron microscopy have been fixed and may have shrunk, at the expense of extracellular fluid only. Though this is just possible, it is true that frank shrinkage normally greatly increases extracellular spaces by removing intracellular water as well. If there is an extracellular space, some explanation of the failure of small and diffusible molecules to enter it must be provided, and so far there is no other plausible explanation. Various crude theories have been put forward in the past to suggest, as did Cajal, that swelling of neuroglia might produce sleep by forcing apart nerve cells and interrupting synaptic transmission. There is no evidence for this view, and the fact that glial processes do not intervene in the close proximity of synapses makes it unlikely. There is, however, one condition of cerebral disturbance which is very likely to be due to separation of synaptic surfaces, with or without separation of glial cells from nerve cells and consequent interruption of the metabolic supply lines. This is concussion.

It was shown during the 1939-45 war by Denny-Brown & Russell (1941) that blows delivered to the vertex of the fixed head were much less effective in producing concussion than blows to the occiput with the head free to move. This means that an acceleration is much more effective than compression. The mechanism was worked out later in the war by A. H. S. Holbourn (1943, 1945), who was an Edinburgh physicist attached to Sir Hugh Cairns and working in the Physiology Laboratory at Oxford. He made a model cross section of the brain using gelatin 'ripened' to make it stick adequately to an enclosing rigid layer representing the skull. This model could be made to oscillate about its centre, and polarised light was shone through it. This produced interference phenomena at the points of greatest stress. This is the photoelastic technique by which stresses in bridges, for example, can be predicted by loading models made of perspex. He found that rotatory acceleration produced some shearing stress all round the circumference, just as a tap to the handle of a cup will make the cup move, but the inertia of the coffee will make the bulk of it stay behind, with some shearing between layers of fluid near to the wall of the cup. However, at points where the 'bone' model projected into the gelatin, as the sphenoidal ridge does in a parasagittal section, there was severe stress.

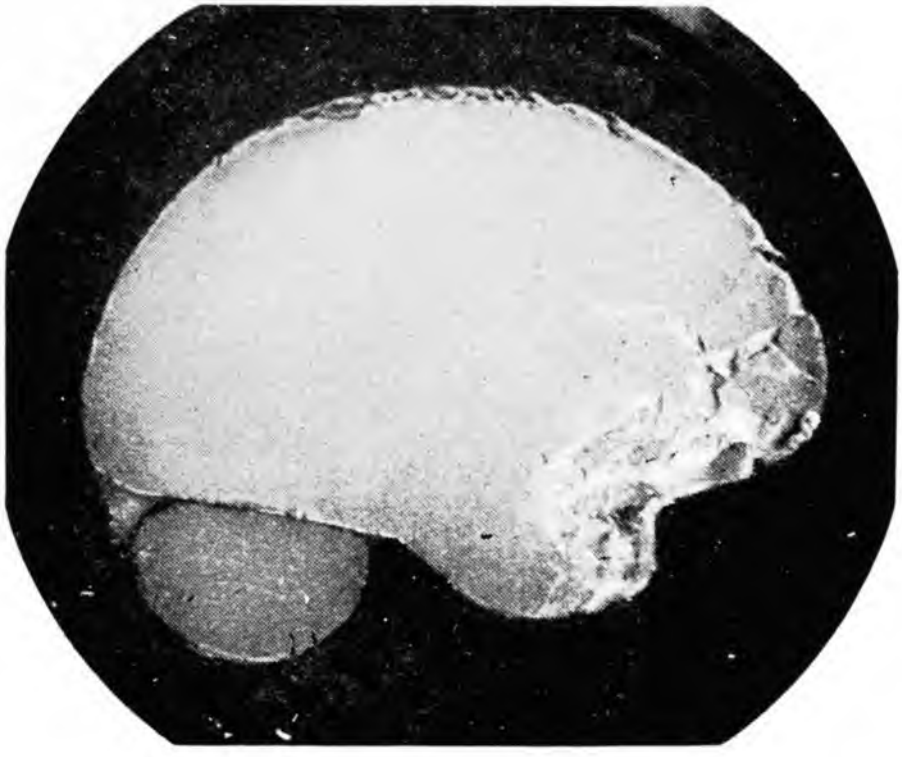

Fig 1.—Effect on a jelly of violent rotational jerking in its own plane.
(Holbourn, A.H.S. (1943). Lancet, 2, 438.)

When Holbourn compared his predictions of the sites of cortical damage with an actual brain of a head injury from a street accident, the fit was complete with the exception that theory predicted symmetrical lesions in both hemispheres, whereas the lesions were more marked in one hemisphere than the other. Subsequently the calvarium in a monkey was replaced by a perspex cap by Pudenz & Sheldon (1948) and the shearing movements of the brain relative to the skull were directly recorded by cine-photography. The gyri were

seen to move several mms relative to the skull during rotation. A blow on the back of the head may accelerate the brain sufficiently to cause severe damage around the temporal pole and the undersurface of the frontal lobe, and this used to be ascribed to contre-coup. Contre-coup injury is, in fact, a figment of the imagination. Naturally, deliberate attempts to produce a rotational acceleration of the skull, as in a blow on the jaw, will readily produce concussion. The ease with which brain damage can be produced in this way does suggest that the only sport in which head injury is deliberately produced, namely boxing, should be suppressed.

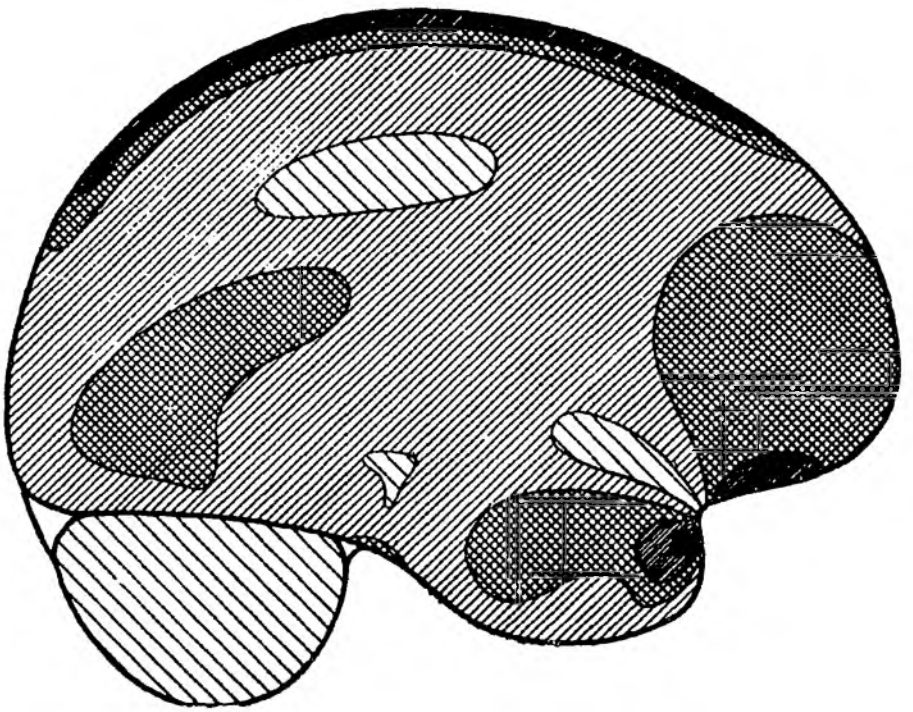

Fig. 2.—The shear-strains (=distortion) which arise when a gelatine model is rotated. The darker the shading, the greater the distortion. Note the comparative absence of distortion in the lateral cerebellar lobe and the high distortion at the tip of the temporal lobe.

(Holbourn, A.H.S. (1945). Brit. med. Bull., 3, 147.)

Recently some careful studies by S. Strich (1956, 1961) of the degeneration found in the white matter in patients who have survived for long periods in coma after severe head injuries have fully confirmed Holbourn's views on the sites of greatest damage in the brain. The whole question of these head injuries has been reviewed by Sir Charles Symonds, who has accepted Holbourn's views on its mechanism and Strich's evidence of its consequences.

The fact that paralysis of nervous activity is immediate, suggests shearing damage to synapses, but the death of nerve cells or their very slow recovery does suggest damage to their metabolic pathways.

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RESPIRATORY INADEQUACY

- *its study, diagnosis, and treatment*

By CHARLES HOPE

Extracts from a Dissertation read before the Royal Medical Society on Friday, 24th November, 1961.

How frequently in clinical practice do we have to treat the patient in respiratory distress? How frequently do we have to administer oxygen, and on what criteria do we do so? How frequently do such patients die, and how frequently do we claim their death as being **inevitable**?

Woolmer (1956) states that respiratory inadequacy exists "when the gas exchange between the lungs and blood falls below that between the tissues and the blood: when external respiration cannot keep place with internal respiration". [17] Melville Arnott (1960) is more direct, stating that respiratory inadequacy is the preliminary stage of respiratory failure, namely, "that condition in which the amount of oxygen and carbon dioxide in the blood stream is altered by an abnormality of the respiratory system".[23] Comroe *et al.* state specifically as the basic facts of respiratory inadequacy, hypoxaemia, CO_2 retention, and respiratory acidosis.[11]

Previously the concept of respiratory inadequacy has had comparatively little attention in the numerous conditions involving the lungs, other than in the case of acute laryngeal obstruction. To quote Melville Arnott, "*too much emphasis has perhaps been placed on the effect of these conditions on cardiac function, and the resulting varied symptoms have been regarded as cardiovascular rather than respiratory*".[23]

PULMONARY FUNCTION STUDIES

Pulmonary function studies will not tell where the lesion is, what the lesion is, or even that a lesion exists, if it does not interfere with the function of the lungs. They supplement but do not replace a good history and physical examination, and radiological, bacteriological, bronchoscopic, and pathological investigations. How can we use these studies to our advantage? The most useful method is to divide respiratory function into three zones—[11, 23]

1. VENTILATORY ZONE — comprising volume and distribution.
2. DIFFUSION ZONE — discussion of O_2 and CO_2 relationships and the effect of the alveolar capillary membrane.
3. PERFUSION ZONE — comprising the volume and the distribution of the pulmonary capillary blood flow.

Ventilatory Zone

Ventilation normally maintains the alveolar gas tensions at levels which ensure that the pulmonary capillary blood is saturated with oxygen before it leaves the respiratory part of the capillary and that it has rid itself of the necessary amount of CO_2 over the tension range of 46 - 40 mm. Hg. The requirements for this are therefore the appropriate function of the bellows mechanism and airway configuration to ensure a uniform distribution of the inspired air. Remembering that gas exchange occurs rapidly only in the alveoli, it is therefore obvious that the measurement of **alveolar ventilation** is the most important single measurement of this zone of function. It must be appreciated that measurements of lung volumes do not evaluate function. These are essentially anatomical and are unchanged by altered physiology.

Alveolar ventilation is the amount of gas which reaches the pulmonary alveoli, and will depend on the frequency of respiration, the tidal volume, and the respiratory dead space. Frequency normally lies between 11 - 14 breaths per minute in healthy basal conditions, but taken alone, cannot be very useful as an index of alveolar ventilation. The tidal volume is seldom measured clinically although this can be done easily even in a sick person by the use of various open and closed methods, by the use of a Douglas bag, or by the use of an ingenious plastic bag which can be carried easily in the pocket.[12] The normal average is from 500 - 600 mls. Again, this is not a useful value if considered alone, but if associated with the frequency, it may be useful in detecting gross hypoventilation.

The respiratory or physiological dead space is equivalent to the anatomical dead space, plus two additional volumes, namely, the volume of inspired gas ventilating alveoli which have no pulmonary capillary blood flow, and the volume of inspired gas ventilating some alveoli in excess of that required to arterialise the pulmonary capillary blood flowing around them. These two factors operate significantly only in patients with pulmonary disease, taking this in its broadest sense. In the normal person, the anatomical dead space is very nearly equal to the physiological dead space except in exercise. From this it is seen that the respiratory dead space is easily calculated from the basic equation—

$$\text{ALVEOLAR VENTILATION/MINUTE} = (\text{T.V.} - \text{R.D.S.}) \times \text{frequency.}$$

For patients in respiratory inadequacy it is easier to measure ventilation by their effectiveness in washing out alveolar CO_2.

$$\text{ALVEOLAR VENTILATION (mls.)} = \frac{\text{Vol. of exp. } CO_2 \text{(mls.)}}{\%\ CO_2 \text{ in alv. gas}} \times 100$$

This should demonstrate the importance of thinking along the lines of alveolar ventilation. It is not sufficient merely to count the respiratory frequency. Much more attention must also be paid to tidal volume.

Diffusion Zone

The rate of transfer of oxygen and carbon dioxide depends on the tension differences, length of the path, the specific solubility and diffusibility of the gases and the area of the membrane. CO_2 is much more diffusible that O_2 to the extent of 20 : 1 and the tension difference of 6mm. Hg. between the pCO_2 of the mixed venous blood and the alveolar air is sufficient to eliminate an adequate amount. Oxygen requires a greater diffusion gradient of the order of 64 mm. Hg., from 104 mm. Hg. in the alveoli to 40 mm. Hg. in the venous blood. The Hb is barely saturated when it leaves the alveoli, and in health a small tension deficit of 9 mm. Hg. exists, partly due to shunting and partly due to the admixture factor existing as a result of mechanisms in the ventilatory zone (previously discussed) whereby an increase in the dead space may occur.

Reduction in the diffusion capacity may be due to an increase in the diffusion pathway or a decrease in the area of membrane. Arterial unsaturation can be due to either these factors or to ventilatory insufficiency, when present in the absence of gross shunting. The arterial pCO_2 however, allows a differentiation. In ventilatory insufficiency there will be a decreased alveolar pO_2 and an increased alveolar pCO_2, and, as CO_2 has to accumulate in the plasma until it obtains the 6 mm. Hg. ascendancy over alveolar pCO_2 necessary for it to escape, there will be both hypoxia and hypercarbia.

In decreased diffusion capacity, or alveolar-capillary block, CO_2, being highly diffusible, still diffuses out at normal tensions. In fact, hyperventilation may actually wash-out CO_2 to produce an abnormally low arterial pCO_2. As arterial pCO_2 is 6 mm. Hg. less than venous pCO_2, it can be calculated from the value of the alveolar pCO_2 which is equivalent to that of mixed venous blood when a subject has rebreathed to equilibrium with a bag of air. Alveolar pCO_2 is calculated either by a rapid infra-red analyser, or by the Haldane method or one of its modifications. This promises to be of great value in clinical practice and will show unsuspected elements of hypercarbia in early stages.

Perfusion Zone

In respiratory inadequacy the usual defect shown by studies of this zone is pulmonary hypertension. The perfusion zone can be studied by cardiac and pulmonary catheterisation, and by the use of tracer isotopes and dyes.

Forced Expiratory Volume (F.E.V.) (21, 4)

This is a practical measure of the maximum ventilatory capacity (M.V.C.) and is easily measured in health and disease. The inspiratory and expiratory phases of the vital capacity as measured and recorded on a fast drum produce a record known as the vital spirogram. In the initial linear fraction of the expiratory phase, the volume and rate of air flow are related to those obtained in the expiratory phase of maximum voluntary ventilation (M.V.V.) The F.E.V. is half of the mean of six readings of the first 0.75 second periods of the expiratory curve. (0.75 is equivalent to the theoretical breathing rate of 40/minute.)

F.E.V. gives a better measure of the maximum ventilatory capacity than the M.V.V. It is easier and quicker to perform and has only a small standard error. F.E.V. is thus a measure of the vital capacity which is useful to an individual for hyperventilation (i.e. it is a valid measure of M.V.C. for expiration at a uniform rate proportional to the vital capacity).

AETIOLOGY OF RESPIRATORY INADEQUACY

Respiratory inadequacy occurs as a result of hypoventilation, that is, decreased alveolar ventilation, and is due to any process interfering with the normal function of the bellows mechanism, either in its moving parts or in its central and/or peripheral control mechanisms. These processes may be classified under several headings (Table 1.)

These aetiological factors may act either singly or in combination. At the time of commencement of therapy there may be little or no indication of the exact cause of the respiratory failure.(11, 23, 24)

CLINICAL PICTURE OF RESPIRATORY FAILURE

The clinical signs and symptoms in respiratory inadequacy can be many and varied. The distressed patient, dyspnoeic, cold, clammy, apprehensive, exhibiting purposeless movements; the paradoxical respiration, cyanosis, and perhaps coma of the major road casualty; the surgical risk, grossly obese, postoperatively in poor condition, with slightly lowered blood pressure and average, thready or full pulse: these and many others are the clinical syndromes which the house physician or surgeon, or the general practitioner may be required to assess and treat.

Dyspnoea can be the result of an obstructed airway caused by inflammation or secretion, the result of emphysema or of gross obesity with its concomitant mechanical embarrassment. It is the result of CO_2 retention and hypoxia. Cyanosis may be present in severe cases, provided there is enough circulating haemoglobin. This is due to arterial desaturation, and if this has been of long standing, there may be a compensatory polycythaemia. The symptoms of hypoxia are many and varied:— increased ventilation, increased pulse rate, incoordinated movements and decreased muscular efficiency; progressing to further increase in pulse and respiration, Cheyne-Stokes breathing, cyanosis,

TABLE I.

Aetiology of Respiratory Inadequacy

Depression of Respiratory Centre:	
General anaesthesia	Increased intracranial pressure
Excess morphine or barbiturates	Electrocution
High concentrations CO_2	Prolonged hypoxia or cerebral ischaemia
Cerebral trauma	
Interference with neural conduction or with neuromuscular transmission to the respiratory muscles:	
Traumatic spinal cord lesions	Neuromuscular block due to: curare; decamethonium; succinyl choline; nerve gases; myasthenia gravis; botulinus or nicotine poisoning.
Poliomyelitis	
Peripheral neuritis	
Diseases of the Respiratory Muscles	Acute myositis
Limitation of Thoracic Movement:	
Arthritis	Scleroderma
Kyphoscoliosis	Gross obesity
Emphysema	Chest wall injury (traumatic and surgical)
Ankylosing spondylitis	
Limitation of Pulmonary Movement:	
Pleural effusion	Thickened pleura
Pneumothorax	
Pulmonary Diseases:	
Decrease in functioning lung tissue:	
Atelectasis	Emphysema
Tumour	Pneumonia
Decreased distensibility of lung tissue:	Pneumoconiosis
Fibrosis	Congestion
Obstructive lesions:	
Tracheal compression	Asthma
Inhaled foreign material	Bronchospasm
Obstruction due to faulty endotracheal tube.	
Primary Hypoventilation Syndrome	

vomiting, asthenia, and fatigue. Further hypoxia leads to severe cyanosis, excitement, fall in blood pressure, syncope, and coma, with respirations initially deep, becoming shallow and frequent with inspiratory spasm, and ultimate death.[20]

Carbon dioxide retention is not separated clearly from hypoxia with regard to its clinical effects. There is mental confusion, drowsiness, coarse irregular muscular twitching, warm extremities, raised blood pressure and full pulse. There may be tachycardia, arrhythmias, accentuation of vagal stimulation, ventricular fibrillation, cardiac arrest and bronchoconstriction. Acute CO_2 excess produces a peripheral vasodilatation with a full bounding pulse. Central effects produce vasoconstriction with a raised blood pressure which is maintained until further excessive retention depresses the vasomotor centre to produce a sudden and dramatic fall in the blood pressure. The effects are similar to an excess of circulating adrenaline, and there is such a release in this condition.[17]

CO_2 retention produces a respiratory acidosis and decreased blood pH. The kidneys compensate by increasing the reabsorption of Na^+ and HCO_3^-, and increasing the excretion of H^+ and NH_4^+.[32] There may be oliguria with all its grave complications. Experiment in animals can produce oliguria in diffusion respiration, but it does not occur until CO_2 accumulates. Perhaps this is the cause—it has not been proven.[17]

Clinically, when the onset is relatively gradual and observation is possible,

there may be evidence of CO_2 retention in that there is a slow rise in the pulse and blood pressure, followed later by a fall.[24, 11, 23]

TREATMENT OF RESPIRATORY INADEQUACY

The general principles of therapy are early energetic treatment, with a clear airway, and an adequate *effective* ventilation. To some extent the exact method of treatment will be indicated by the factors predisposing to inadequacy, and in certain cases the method of treatment will be instrumental in treating the cause. Treatment will be discussed under the headings—oxygen therapy, artificial respiration, drug therapy and tracheostomy.

OXYGEN THERAPY

It is necessary to administer oxygen to patients in respiratory failure. It is traditional to do this by the use of intermittent oxygen, and various schedules have been recommended—[23]

(a) oxygen given for 20 minutes per half-hour.
(b) oxygen given for half an hour in every hour.
(c) oxygen given for 40 minutes in every hour.

By these means one is said to prevent progressive CO_2 retention as the removal of oxygen will produce a hypoxic stimulus to breathing and will allow "blowing-off" of retained CO_2. This is achieved however only at the risk of producing periods of extremely serious hypoxia, mainly because the oxygen and carbon dioxide stores in the body are very different. [Farhi & Rahn 1955[7]] Any change in the ventilation or in the inspired air will produce a change in the oxygen concentration within 1 - 2 minutes, whereas change in the concentration of carbon dioxide takes 10 - 20 minutes. Within 2 - 10 minutes after stopping the administration of oxygen the persistent high pCO_2 from the blood reduces the alveolar pO_2 to produce an arterial anoxaemia or rather hypoxaemia of a greater degree temporarily than if oxygen had never been given at all. It may be argued that patients overventilate to "blow-off" CO_2, but in many cases the mechanical function of the chests are much too poor to permit this. It is also argued that the poorly ventilated parts of the lungs retain sufficient oxygen to prevent hypoxaemia for several minutes. That this latter argument is completely spurious is seen if one recalls the previous pulmonary physiological evidence. It has also been found that within 5 - 6 minutes after stopping oxygen the arterial pO_2 has dropped to 15 mm. Hg. on the average. This is equivalent to a 20% saturation.[7]

It should be remembered that in some cases the only stimulus or mechanism maintaining respiration is the decreased pO_2 stimulus to the chemoreceptors, the respiratory centre being completely depressed by the high pCO_2. Oxygen therapy may then produce apnoea which may prove fatal, e.g. Cor Pulmonale.

By these and many other arguments the requirements for giving oxygen in respiratory failure can be deduced.[7] In summary they are that oxygen must be given continuously, that the oxygen concentration should be controlled to prevent it rising high enough to produce respiratory depression, or falling low and permitting recurrence or increase of the hypoxia. The results of various exhaustive trials suggest that these requirements demand control of the inspired oxygen concentration with an accuracy of $\pm$ 1% (7 mm. Hg. pO_2.) in the range 24 - 35%. The dead space must be reduced as much as possible to prevent CO_2 retention.[8]

To achieve these requirements, conventional methods have proved to be rather inadequate. In oxygen tents the concentration of oxygen cannot be controlled with sufficient accuracy and at the low flow rates required there is CO_2 retention. The nursing and physiotherapy problems are also intensified.

With nasal and pharyngeal catheters the dead space is greatly reduced, but the oxygen concentration produced is extremely variable as a result of the degree of patency of the nasal passages, whether or not the mouth is open or closed, and whether inspiration is deep or shallow. Masks are the best method to use when high concentrations are required, the high flow rate flushing the dead space and preventing reinspiration of expired air, but at low flow rates, the mean inspired oxygen concentration is extremely variable. The required control of inspired oxygen is thus difficult and the difficulty is increased with lack of knowledge of the minute volume and with leaks in the system. The dead space of masks is too large to be acceptable when the gas flow rate is below 5 - 6 litres per minute. One should also note that if respiratory depression occurs when a patient is given oxygen by conventional methods at low flow rates, the concentration of inspired oxygen is thus automatically increased. Hypoxia may therefore not return to *stimulate* ventilation.

As a result of these difficulties attempts were made to devise systems using air at high flow rate with controlled oxygen enrichment. This was obtained by a loose-fitting mask, with air supply from a cylinder or pump at a flow rate of 30 - 50 litres per minute, and with additional oxygen at a controlled rate to produce an accurate control of the inspired oxygen concentration. With this method, rebreathing is negligible because of the flushing ; there is no dead space, and the flow rate is very much greater than the inspiratory peak flow rate. The many considerable practical disadvantages were a contraindication to its use until the Venturi principle was adopted. The present design has a jet set to deliver 1.5 - 2 litres of oxygen per minute. This produces sufficient negative pressure to entrain 50 litres of air per minute, and give a basic flow rate of 50 litres of air containing 24% oxygen per minute. Additional oxygen is then added at low pressure through a second tube to produce any desired oxygen concentration. Air sampling has shown that the concentrations produced are $\pm$ 0.5% of the predicted concentration, and that the concentration of CO_2 is less than 0.5%. This is simple to use, economical of oxygen, and requires no humidification as atmospheric air is used. However additional further humidification is possible. Campbell (1960) suggests a very sensible routine for the treatment of respiratory inadequacy or failure.[8]

1. Decide if ventilatory failure is present or not by clinical or clinico-physiological methods and biochemistry. In the absence of failure one should give oxygen in the conventional way with high flow rates.

2. If ventilatory failure is present or suspected, 24% oxygen is given by the Venturi principle. The oxygen concentration is then increased by 2% steps (14 mm. Hg.) at 3 - 4 hourly intervals *provided there is no* CO_2 *retention.* When 35% oxygen is being tolerated, the conventional methods may be brought into use.

3. If there is CO_2 retention, continue at the highest rate which can be tolerated (approx. 25 - 26% O_2). This will relieve the hypoxia without further CO_2 retention.

4. Unsatisfactory progress will require Intermittent Positive Pressure Respiration with possible tracheostomy.

ARTIFICIAL RESPIRATION

Methods of artificial respiration have been in use for many years. There are three main groups.[17, 18]

1. Methods utilising the pressure changes on the chest wall, abdomen, or diaphragm.

(a) Manual methods.
(b) Eve rocking bed or stretcher.
(c) Cuirass or jacket respirator.
(d) Iron lung or cabinet respirator.

All of these may be of use in the treatment of inadequacy as an emergency measure, but they have several obvious limitations. They can be used with safety and effectiveness only in the normally healthy patient who has no thoracic cage fixity or osseous fragility, or any decrease in pulmonary movement or decreased distensibility of lung tissue. They require a clear airway.

2. Methods utilising pressure changes through the airway.[13]

(a) Mouth to mouth respiration.
(b) Manual compression of the reservoir bag or other device; i.e. the anaesthetic machine.
(c) Intermittent Positive Pressure Respiration.

(a) According to Cox, Woolmer, and Thomas[13], mouth to mouth, or mouth to airway EXPIRED AIR RESUSCITATION offers an effective method of respiration, and should replace the standard methods of emergency artificial respiration. In expired air resuscitation, tidal volumes in excess of one litre can be moved with each breath. Certain investigators have shown that standard manual methods without an artificial airway do not move a tidal volume greater than the victim's dead space in 25 - 80% of cases. Aesthetic objections are overcome by the use of an airway such as the Brook airway. Gastric distension is of apparently little importance except in the infant, and lung rupture is unlikely unless there is gross disease or if the victim is an infant. Infection is possible, but can be prevented by the use of a bacterial filter. Such risk is unimportant, however, in a case of emergency. CO_2 levels are found to be satisfactory in subjects and tolerable in donors, and the possible circulatory effects will occur in most other methods of artificial respiration also. However, this may be a difficult method for use with the conscious patient. A clear airway is also necessary and can be maintained by the usual manual methods.[15, 22, 29]

(b) THE ANAESTHETIC MACHINE may be used effectively in the treatment of respiratory inadequacy. The most effective combination is one including a soda lime cannister for the absorption of CO_2. The principles of the mechanism are similar to I.P.P.R. machines, with the exception that the pumping is done by hand.

(c) INTERMITTENT POSITIVE PRESSURE VENTILATORS are of three types—

(a) Pressure cycled ventilators.
(b) Volume cycled ventilators (pressure limited).
(c) Time cycled ventilators (pressure limited).

Pressure cycled ventilators have as a cycling mechanism a pressure sensitive valve which is actuated by changes in the patient's lungs at a pressure set by the anaesthetist. Volume cycled machines deliver a pre-set volume to the lungs irrespective of the pressure produced. They must be fitted with a pressure limiting device. Time cycled ventilators have the length of the inspiratory and expiratory phases determined by the anaesthetist. The valves open and close under electronic control, the absolute cycling times determining the number of cycles per minute. The pressure or air-flow is then varied to produce the required tidal volume.

It should be noted that triggers and humidifiers are available for employment in the ventilator circuit.[17, 18, 25]

Controlled respiration has a definite effect on the circulation. The low pressure pulmonary vascular system has an average pressure of 10 mm. Hg. with a range of 7 - 15 mm. Hg. I.P.P.R. transfers a similar pressure to the alveolar wall, and if the alveolar pressure is high, there is 'milking out' of blood from the lungs and a transient slight increase in cardiac output from the left heart, a damming back of blood to the right heart, and a decreased filling gradient of the right heart from the great veins thus leading to decreased cardiac output. A significant fall in cardiac output will not occur if the positive pressure is limited to a short portion of the respiratory cycle, if the expiratory phase at ambient air pressure is made as long as possible, and if no severe blood loss or other cause of hypovolaemia exists. Therefore a high instantaneous air flow, the positive pressure inspiratory phase, will produce an adequate tidal air. This is followed by a rapid passive deflation to zero pressure. Addition of a negative phase (not greater than -10 cms. H_2O) when the airflow has stopped at the end of the expiratory phase will reduce the mean pressure and improve the cardiac output.[17]

3. Methods using electrical stimulation.
   (a) Electrophrenic respirators.

These have practical disadvantages which limit their use considerably. In the past they have been used almost exclusively in the resuscitation of the newborn infant.

Drug Therapy

Some investigators and physicians advocate the use of respiratory stimulant drugs. Either 2.5% aminophylline or 25% nikethamide[23] may be given intermittently or continuously by a slow intravenous infusion. Close medical supervision is required as large doses are necessary, the aim being to increase the ventilation without producing the side effects of nausea and vomiting (aminophylline) or of convulsions (nikethamide). These investigators advocate the use of these drugs in conjunction with the conventional face mask methods. However, it is stressed that should these measures prove ineffective, due to the amount or stickiness of secretions, or due to dangerous ventilatory depression, tracheostomy and I.P.P.R. is indicated and should be resorted to without delay. Nevertheless because of the insecurity of these measures the patient may require such therapy for many days, and because the aetiological factors of the inadequacy may be contraindications to the effectiveness and desirability of drug therapy, I feel that although these measures may play an important part in the treatment of certain selected and mild cases, they have, as a whole, little indication for their use. Further it is not generally appreciated that the administration of intravenous respiratory stimulant preparations can produce an immediate respiratory and/or cardiac arrest in cases of inadequacy. This may possibly be due to the fact that these drugs are stimulating already exhausted and overworked vital centres.

TRACHEOSTOMY

Tracheostomy is indicated in all cases of respiratory inadequacy in which the dead space must be reduced either as a preventive or ancillary measure in the treatment. It is necessary for adequate bronchial toilet, in cases in which assisted respiration will be required for a considerable time, and in cases in which there is relatively acute or acute on chronic upper respiratory tract obstruction. With tracheostomy, strict aseptic technique is essential and humidification may be necessary to prevent tracheal crusting with its complications. Adequate toilet must be carried out to remove secretions and daily bronchoscopy may be necessary. Prevention of tracheal pressure necrosis must be ensured, and naso-gastric feeding will be essential as feeding difficulties arise with cuffed tubes and I.P.P.R.[17, 16]

GENERAL MANAGEMENT AND GENERAL NURSING CARE

It is possible to mention but the headings of the many duties which must be performed routinely while the patient is in the dangerous state of inadequacy or failure. The management is described for the patient on I.P.P.R. with tracheostomy.

1. PULSE and BLOOD PRESSURE must be taken and recorded every 10 - 15 minutes. This is essential to ensure satisfactory progress and must be enforced. It is preferable to have this done by a doctor initially.
2. There must be a special senior nurse and doctor (preferably an anaesthetist) in the presence of the patient 24 hours per day.
3. Proper TRACHEOSTOMY management must be carried out. This includes daily bronchoscopy.
4. DAILY CHEST X-RAY and clinical examination must be carried out to ensure against pneumothorax or some such complication. Equipment should be easily available to institute pleural drainage if necessary—e.g. water-seal drainage for spontaneous pneumothorax.
5. SEDATION must be adequate, and intravenous opiates or bariturates are indicated. Curarisation may be performed—e.g. in the tetanus case.
6. RENAL FUNCTION must be continually observed. 24-hour specimens of urine must be collected, if necessary by an indwelling Gibbon catheter. Fluid balance charts must be kept accurately.
7. BIOCHEMICAL INVESTIGATIONS must include B.U.N., serum electrolytes, Na^+, K^+, and Cl^-, and CO_2 combining power, as a minimum. Arterial pO_2 and pCO_2 readings would be of much greater value but present administrative difficulties make this impossible on a nation-wide scale.
8. NASO-GASTRIC TUBE FEEDING is essential to maintain the general nutrition of the patient. Overloading of the stomach should not be allowed to occur. This is a possible complication as a paralytic ileus occasionally follows tracheostomy.
9. PARENTERAL FLUIDS are required to maintain hydration, renal function, and correct the acidosis. Glucose, M/6 lactate, saline and added KCl are usually required.
10. HAEMATOLOGY should be done routinely as a precautionary measure. This should include a P.C.V. and white cell count. The haemoglobin level is of secondary importance to these above estimations.
11. ANTIBIOTICS must be given if indicated.
12. PHYSIOTHERAPY must be enlisted in attempt to prevent deep venous thrombosis, and to prevent joints from stiffening.
13. Aseptic dressing technique of the tracheostomy wound.
14. General nursing duties—prevention of pressure sores, bed-baths, change of clothes, etc. This is good for the patient's morale.

This formidable list is essential in every detail for the treatment of such cases. As the position improves, the intensity of management can be reduced, but *only under the direction of the specialist in charge*, and the patient can be weaned from the ventilator, and ultimately weaned from the tracheostomy, to commence a period of rehabilitation.

CONCLUSION

In conclusion I would emphasise that some knowledge of the condition of respiratory inadequacy should be essential for all medical attendants. I trust too that I have emphasised sufficiently what I consider the only permissible and rational approach to oxygen therapy. May I ask once more—How frequently do such patients die, and how frequently do we claim their death as being inevitable?

My very grateful thanks are due to Dr. D. T. McClements, Consultant Anaesthetist at Peel Hospital, Galashiels, for helpful advice and criticism during the writing of this paper and for introducing me to the diagnosis and management of this condition. Also, for advice and criticism, I must thank Dr. J. D. Robertson, Head of the Department of Anaesthetics, R.I.E., and Dr. C. T. Barry, Anaesthetic Consultant in Charge, Western General Hospital, Edinburgh.

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RES MEDICA

SPRING, 1962

PSYCHIATRIC SERVICES FOR CHILDREN

The lack of psychiatric services for children is acute. At the present time, the paediatrician, however greatly interested in the case, is often required to deal with frank emotional and behavioural problems in which he and the only available psychiatrist may have no special training.

Thus, in a recent report based on information obtained from paediatric units throughout the United Kingdom, the facilities available to adolescents who had left school were said to be in almost every case simply the general paediatric and adult mental services. For children of school age, both In- and Out-Patient psychiatric services were said to be very inadequate. Regarding in-patient treatment, psychiatrists are very often compelled to make use of beds in general paediatric departments. The waiting period for an out-patient appointment can be very long—in one extreme case, 2½ years. No information was available in the report regarding the facilities for children of pre-school age.

Such psychiatric services as do exist are established either by the Local Authority or as a unit within the National Health Service. Only about 2/3 of Local Authorities run any form of Child Guidance Service. Regarding England and Wales, and with a view to the treatment of maladjusted children within the educational system, it was recommended in 1955 by the Underwood Committee that the Local Authority should provide the premises, the educational psychologist and the psychiatric social worker, while the Regional Hospital Board supplied the psychiatrist. Whether or not child psychiatric services should be run along such lines is a question still not fully settled.

Both in the case of the Child Guidance Clinics run by the Local Authority and the school medical service run by the Education Department of the Local Authority, the psychiatrist is available almost invariably only on a part-time basis, or for consultation in the case of particular children. In either case, the psychiatrist is made available by the Regional Hospital Board. In Scotland, there are now seven full-time consultant child psychiatrists.

The position of the psychiatrist in relation to the juvenile offender is likewise barely satisfactory. Certainly in a large proportion of cases, repeated offences are essentially manifestations of reactions to psychologically disturbing situations, and must where possible be distinguished from true mental

illness. In the former type of case, the stable environment and the modified type of education available in an approved school may be adequate to restore the child. At any rate, information about the child's background is supplied to the juvenile court by the Education Authority and the probation service—often at short notice. A psychiatric opinion, should the court deem it necessary, is made available through the Child Guidance Service.

Out of every 5 approved schools in England and Wales, 3 have the services of a psychiatrist from the Regional Hospital Board, who may visit for regular sessions. Where that is not possible, a psychiatric opinion, if thought necessary, can usually be obtained at an outside clinic. A recent Home Office report speaks of the suggestions and the unobtrusive contribution of the visiting psychiatrist, the Headmaster retaining all executive authority. On the other hand, in a few cases psychiatric social workers have been appointed and work in close conjunction with the visiting psychiatrist and Headmaster. In Scotland at present, there is probably no instance of any contractual relationship between an approved school and the child psychiatrist. Instead, the frequency with which psychiatric opinion is requested depends upon the sympathy of the Headmaster to the psychiatric approach and the availability of a child psychiatrist. The arrangement, in fact, works quite well, but will probably become more formalised in the near future.

The juvenile court is also empowered to recommend a course of psychological treatment at a Child Guidance or psychiatric clinic, presumably where such is available. The Local Authority can send the child, on psychiatric advice, to a residential school for maladjusted children. Such schools are however, very few, as are the special Day Schools set up by a few Local Authorities, and unable to cope fully with the problem. At such schools, psychotherapy is sometimes available.

The situation shows slight improvement. For children requiring special medical investigation and treatment, there are now 8 children's psychiatric units in Scotland. These, understandably, should be no more concerned with investigation and diagnosis than with long-term treatment, but due to lack of accommodation in their premises, long-term treatment must very often be continued in less specialised departments or in schools for maladjusted children. Several children's psychiatric units have recently been opened—for instance, in 1959 the male adolescent unit with 16 beds at the St. Augustine's Hospital in Kent. But even regardless of facilities, the supply of staff is a major problem, for both nurses and psychiatrists require training in the handling of the specialised psychiatric problems of childhood and adolescence.

THE COMMON COLD

Over the last ten years or so there has been considerable interest in this ailment. Though the symptoms of this infection are not particularly severe, it is of considerable economic importance. It has been calculated that in one year over twenty million man working-hours were lost solely as a result of the common cold.

In 1946 it was suggested that the cause was a filterable multiplying body. Attempts at cultivating the organism and testing for its presence in vitro were unsuccessful and studies had to be carried out on human volunteers. In 1953 at Salisbury it was reported that an organism, retaining infectivity in human

volunteers, had survived ten passages through human embryonic lung tissue culture maintained in medium 199. Attempts to repeat this experiment failed. Since 1956 a number of organisms causing minor respiratory ailments have been isolated in the United States. These viruses were found to be related to the Cox-sackie and ECHO sub-groups of the entero-viruses and certain myxo-viruses that were finally classified under the names of para-influenza 1, 2 and 3. These viruses were studied at the Common Cold research unit at Salisbury and it was concluded that though they could cause the common cold in children they were not the principle actiological agent.

The Salisbury unit maintained that the commonest form of the common cold was typified by an increase in nasal discharge (measured by the number of paper handkerchiefs used by the infected volunteers), a sore throat, particularly at onset, and a cough. Usually the patients were afebrile. In 1960 Tyrrell reported that an organism producing typical symptoms of a common cold had survived a number of passages through human embryonic kidney tissue culture maintained in 199 medium supplemented by the addition of bovine plasma albumin and maintained at 33°C at an acid pH. The culture was well oxygenated using rollers. Since then three strains have been identified and cultured but there are probably many other, as yet uncultivated, causative strains. When the virus was cultivated cytopathic changes were observed in the cells of the culture medium. The cells became refractile and later dropped away from the glass of the container. These changes were not observed in control cultures or when the inoculating fluid had been heated to 56°C or maintained at a pH of 2. It has been found that cultures infected with common cold viruses gives rise to interference in the growth of influenza virus when subsequently inoculated onto the tissue culture. A quantitative estimate of interference, and hence of the growth of the common cold virus can be made by determining the amount of haemadsorption of human red cells onto the tissue culture cells infected with the challenging influenza virus.

The multiplicity of infecting organisms presents a poor picture as far as prophylactic immunisation is concerned. Claims of cross immunity between various strains have not been proved. However it is hoped that some non-specific antiviral substance such as interferon may be developed. Another possibility in prophylaxis is that study of the epidemiological characteristics of the viruses may enable methods to be developed that will limit spread of infection.

NORMALITY

its cause and cure

For many centuries mankind has sought to define normality objectively, and if all the men and women who have tried were laid lengthwise, heel to head, I should be found at the end of the line, flat out. All societies have their misfits, but the causes of normality in any one community are multiple. Societies have a code of behaviour to which its members are expected to conform but, just as this varies from one society to another, so do the permitted degrees of deviation from the code and the methods used to bring the misfits back into line. Divergencies are reflected in the marital status for, somewhere and at sometime, practically every imaginable pattern of marriage

has been regarded as proper. Toleration of deviation from the social norm seems to evolve with technical development. In officially Christian, monarchical, monogamous Britain, for example, you can profess any religion or none, drink to excess or remain "dry", refuse military service, practise successive (but not simultaneous) polygamy, speak in derogatory terms of the Government, the Church and the Monarchy, and, short of actual nudity or what is technically known as exposure, indulge in any eccentricity of dress. If you are a woman with homosexual desires the law will be little concerned with your behaviour because homosexual practices among women seem never to have been regarded as constituting a social danger and occur for the most part between consenting adults. If you are a man, however, your homosexual relationships with consenting adults in private may lead to imprisonment and social disgrace while promiscuous heterosexual intercourse will not receive legal distinction. Accepted behaviour changes in the same society as time passes, and it has been said that if you go into a room nowadays and see people smoking and wearing trousers they are more likely to be women.

I should admit, as *a priori* justifiable, the right of a community to interfere with its members in order to secure the biological necessities for all, but, I cannot admit its right to interfere in matters where one man's possessions are not obtained at the expense of another e.g. opinion, knowledge, and art. The fact that the majority of the community dislikes an opinion gives it no right to interfere with those who hold it. The same argument applies to interferences with personal morals. But, it seems, that tolerance only grows where fear declines. For centuries intolerance has been the result of man-made fear and has restricted the potential variety of human experience. Only when man has become master of himself as well as his **environment** can he afford the luxury and enjoyment of tolerance.

Contemporary society is coming to regard antisocial behaviour as a symptom of mental abnormality and appoints the doctor to administer not the treatment the patient "deserves" but the one he thinks most likely to effect a cure. But by what thread of dignity does normality hang? Should we not ask ourselves if our society is adapted to its individuals? Freedom is not the right to oppress others but the right to live as we choose and think as we choose when our actions do not prevent others from doing likewise. Dr. Eliot Slater has presented us with evidence which suggests that there is a small proportion of the population which is so constituted, perhaps in large part by genetical causes, as to be unable to form normal heterosexual relationships and to be strongly predisposed to homosexual ones. Professor Penrose suggests that variations in sexual polarity might be regarded as a perfectly normal trait, comparable with variations in stature, hair pigmentation, handedness, or visual refractive error. These traits are all probably dependent upon interaction between heredity and environment and the variation within all of them is probably of degree rather than kind.

"An important object of medical treatment of a homosexual person is to help him to reach a stage where he is able to exercise sustained restraint from overt acts which would bring him into conflict with the law. It will also help him to achieve self-discipline. This will lead to increasing *self respect* and enable him to feel he "belongs" to a society which does not condemn him outright." Hormone treatment undoubtedly diminishes libido and relieves patients from the anxiety by giving them the feeling that they can control their urges but should we prescribe a pill to bring the body into line with socially acceptable morality? Can we justify the use of a drug which eliminates an effect when you have not eliminated the desire that causes the effect? It depends upon what sort of society you want to live in.

Collectively Speaking

Do you talk of a *dosage*
Of Doctors, or *cure*?
Or is it a *consult*,
Or *temperature* . . . . ?
Whatever they're called
In the mass, they're agreed
That, after work, Guinness
Is just what they need.

after work you need a

GUINNESS

THE TREATMENT OF RENAL DISEASES

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Fellow of the Royal Medical Society.

Based on a Dissertation read before the Royal Medical Society on Friday, 8th December, 1961.

All the well-known processes of pathology—acute and chronic inflammation, immune reactions, degenerative changes, etc.—may be found in the kidney, and the forms of therapy required to reverse them are equally diverse. All these processes, however, lead to one thing, destruction of renal tissue. This destruction may be rapid and severe, followed by regeneration in many cases, or a slow but progressive destruction with fibrous overgrowth.

Broadly speaking, the kidneys are responsible for maintaining the stability of the body fluids. If acute destruction of renal tissue occurs the body fluids are acutely altered, whereas in chronic renal destruction the disturbance is slowly progressive. The acute upset of normal physiology accompanying rapid destruction is the syndrome known as acute renal failure, and the progressive physiological imbalance known as chronic renal failure is the result of chronic tissue destruction.

This leads me to the first and most important point I wish to make. Therapy in renal disease must be two-fold. Firstly, it must deal with the pathological process causing renal destruction. Secondly, it must correct the disordered physiology which the renal disease has created. Whereas the pathological processes are legion, they lead to only two types of physiological upsets—acute or chronic renal failure.

In renal failure of either type management consists of either augmenting or completely taking over the functions the kidney normally performs in homeostasis. These functions fall into four groups.

First, the EXCRETION of non-volatile end-products of metabolism. These pass into the glomerular filtrate and are either not reabsorbed or only partially reabsorbed by the tubules. Second, the maintenance of WATER BALANCE. The tubules allow only sufficient water to be reabsorbed from the filtered fluid to provide normal tonicity. Thirdly, ELECTROLYTE BALANCE. Normally the dietary intake provides an excess of Na, K and Cl. Na Cl is filtered and only the

right amount is reabsorbed. The appropriate amount of K is secreted by the tubular cells. These activities are under the control of the adrenal cortical hormones.

Lastly, ACID-BASE BALANCE. Bodily metabolism leads to the production of mainly acid end-products. These are buffered in the blood, mainly by the HCO_3 ion. Exhalation of the resulting CO_2 provides a rapid stabilisation of the pH. However, although the pH is corrected, the blood HCO_3—that is the alkali reserve—is depleted.

The function of the kidney in acid-base balance is, in fact, to maintain the normal plasma HCO_3 level, by producing one HCO_3 ion for every H^+ ion excreted.

CAUSES OF ACUTE RENAL FAILURE

HYPOVOLAEMIC SHOCK commonly arises from loss of blood, and renal complications are especially common after obstetrical haemorrhage. Loss of plasma after burning, and of fluid and electrolytes as in diabetic coma can also lead to renal damage. The renal failure is thought to be due to ischaemia of the kidneys, following intense vasoconstriction in an attempt to maintain the systemic blood pressure. The renal medulla has a poorer blood supply than the cortex, and hence prolonged shock leads first to acute tubular necrosis. If the state of shock persists for an extreme length of time, cortical damage ensues and irreversible renal cortical necrosis occurs. The treatment of a shocked patient found to be anuric therefore consists of immediate vigorous resuscitation with the appropriate fluid. In many cases recovery of the blood pressure will be followed by resumption of urine flow. If oliguria persists, acute tubular necrosis must be assumed to have taken place, and the resultant acute renal failure must be managed along the lines I shall indicate later. Spontaneous regeneration of the tubules leads to resumption of urine flow some ten to twenty days later. If anuria persists much beyond this time, renal cortical

TABLE 1

Causes of Acute Renal Failure

Mechanism	Condition	Treatment
Renal Ischaemia	HYPOVOLAEMIA	Volume Expansion
Acute tubular necrosis	SEPTICAEMIA	Chemotherapy
Renal cortical necrosis	HEPATO-RENAL SYNDROME	Chemotherapy
Direct tubular damage	CRUSH SYNDROME	None
	INCOMPATIBLE TRANSFUSION	None
Acute tubular necrosis	RENAL POISONS	None
	NECROTISING PAPILLITIS ..	Chemotherapy
Diminished filtration	GLOMERULONEPHRITIS	None
	DIFFUSE ANGIITIS	Steroids
Back pressure	OBSTRUCTION	Surgery
	INFECTION	

necrosis must be assumed to have taken place, and the prognosis is hopeless.

SEPTICAEMIA often leads to acute renal failure, and here again the mechanism is thought to involve ischaemia. Bacterial toxins have been shown experimentally to depress renal blood flow. The HEPATO-RENAL SYNDROME is an interesting rarity in which a severely jaundiced patient goes into super-imposed renal failure. This is now thought to be due to an E. coli septicaemia complicating the initial cholangitis. The treatment for both these conditions is clearly appropriate chemotherapy.

In the CRUSH SYNDROME renal failure occurs when the circulation is restored to a region which has had its blood supply occluded for many hours, as after a fall of rock in the mines. Renal damage is caused by the action of toxins liberated from the injured tissue. The same applies after INCOMPATIBLE BLOOD TRANSFUSION. These two conditions are therefore grouped with the RENAL POISONS, and really have no specific treatment.

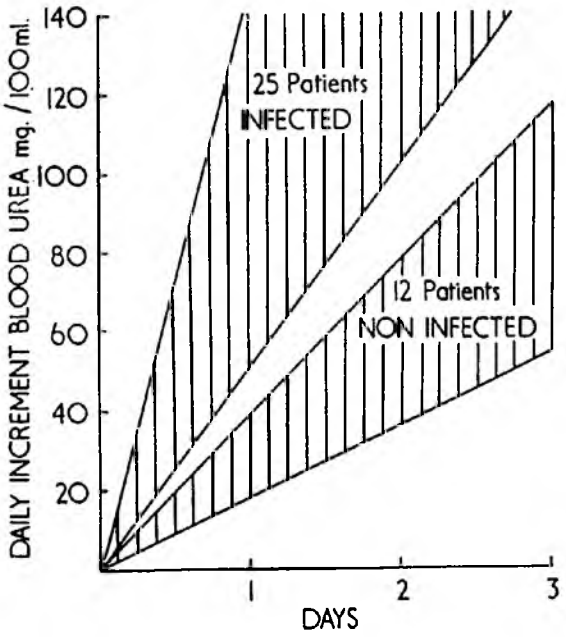

Fig. 1.—The influence of severe infection on the course of acute renal failure. The daily increment of blood urea was calculated for each patient over a period of 3 days.

(Robson, J. S. (1961). Symposium. Some Aspects of Renal Disease. Royal College of Physicians, Edinburgh.)

By NECROTIZING PAPILLITIS I mean very virulent forms of pyelonephritis which can cause acute renal failure by direct tubular damage. Treatment again is appropriate chemotherapy.

The next two conditions cause acute renal failure at the glomerulus by diminishing filtration. Acute GLOMERULONEPHRITIS has no specific treatment. By DIFFUSE ANGIITIS I refer to collagen diseases such as polyarteritis nodosa, which can at least be made to remit (in certain cases) by the exhibition of steroids.

Finally, OBSTRUCTION. In cases of total anuria, obstruction is nearly always the cause, whether from prostatism, stricture, pressure from a tumour, and so on. There should never be any hesitation in carrying out cystoscopy and retrograde pyelography in these cases, and surgery may be life-saving.

Across the bottom I have written INFECTION, in large letters. In an infection,

with accompanying pyrexia, tissue catabolism is greatly increased. The excretory load on the kidney is therefore increased, and a kidney which is well able to cope with normal conditions may be unable to excrete this additional load. Although output is normal the urine is of low specific gravity. Thus acute renal failure can exist even when quite large urine volumes are being excreted in an infected patient. Infection should always be remembered both as a primary cause of renal failure and as an exacerbating factor in failure from any other cause.

These, then, are some of the pathologies leading to acute destruction of renal tissue and the rapid development of oliguria or anuria. The renal destruction can rarely be altered by specific therapy, but in time spontaneous regeneration of the renal tubules frequently takes place. But during the anuric interval the well-known picture of acute renal failure appears. The absence of renal function leads to a rising blood urea and progressive derangement of the internal environment. This acute renal failure can rapidly kill the patient, but if the failure itself is treated, survival can frequently be prolonged until the new renal tissue becomes functional.

THE TREATMENT OF ACUTE RENAL FAILURE

As outlined, the kidney performs four functions, and each is impaired or abolished in renal failure.

The EXCRETION OF NITROGENOUS END-PRODUCTS virtually ceases, and therefore the production of these must be cut to a minimum. Protein is excluded from the diet. Secondly, a high caloric intake is given in the form of carbohydrate. This reduces endogenous production of urea to a minimum, as protein catabolism to provide energy is rendered unnecessary. The calories are supplied as dextrose, lactose or glucose.

The so-called anabolic steroids have been used to oppose protein catabolism. The initial promising results, however, were obtained in patients with renal

TABLE 2

Treatment of Acute Renal Failure

Failing function :	**Therapeutic measures:**
Excretion	No protein High caloric intake
Water balance	Fluid balance chart Clinical assessment Weighing
Electrolyte balance	Prevent NaCl depletion Prevent K+ intoxication
Acid-base balance	Administration of alkali

failure after post-partum haemorrhage, and the effect may be a specific one on the involuting uterus. It has also been found that while these drugs check the rise in blood urea, other end-products such as creatinine continue to accumulate and clinical deterioration occurs. There is probably no place for these drugs in the treatment of renal failure at present.

Secondly, WATER BALANCE. The anuric patient cannot excrete excess water, and unless fluid intake is restricted he rapidly becomes water intoxicated. The insensible water loss through the lungs and skin totals about 500 ml/day, and water administration must therefore be restricted to 500 ml plus the volume of fluid lost during the previous day, by vomiting or diarrhoea as

well as in the urine. If the patient is fevered, an extra ration must be given to replace the volume lost in the sweat.

Successful control of the state of hydration rests on three measures. Accurate fluid balance charts must be kept. The usual slap-dash affair is worse than useless. The exact quantity of all fluids given must be accurately recorded, and any loss by vomiting, diarrhoea or as urine carefully measured. The correct amount of water for a uraemic patient can only be prescribed on the basis of the previous day's fluid balance.

Secondly, the patient's state of hydration must be assessed clinically each day. A trace of sacral oedema or a few basal crepitations are warning signs of overhydration.

Thirdly, daily weighings are most helpful in indicating gain or loss of total body water.

The amount of water which can be allowed as a vehicle governs the number of calories which can be given. If 50% glucose is used an adequate caloric intake is ensured by any volume over 500 ml. At first this may be given by mouth or intragastric drip, but when the B.U.N. reaches about 100 mg % the incidence of vomiting makes intravenous administration essential. Venous thrombosis can be very troublesome, and some authorities use only 20% glucose intravenously. The passage of a plastic cannula into a large vein, frequent change of vein, and heparinisation of the infusion fluid all minimise this problem.

Thirdly, ELECTROLYTE BALANCE. The normal kidney controls this by excreting an appropriate amount of the dietary intake, which is normally excessive. As this excretion cannot occur in anuric patients, no sodium chloride should be given as a rule. Some salt is lost in the sweat and scanty urine, however, and if vomiting occurs salt loss may be serious. The salt lost in this way should be estimated, and a suitable amount of the water ration given as normal saline to correct this. The sodium depletion would otherwise lead to a fall in circulatory volume and further deterioration in renal function would ensue. Nevertheless, very slight overtransfusion with saline easily tips over these patients into pulmonary oedema, and salt replacement should be undertaken with caution.

With potassium the problem is a different one, for cellular catabolism leads to a steady release of the ion into the blood stream. The blood level therefore rises in the course of renal failure, and if there has been much cellular damage, this rise is very rapid. This is most marked in crush injuries and haemolysis, where cellular breakdown is extensive. Unlike sodium and chloride, high potassium levels are very dangerous, and ventricular fibrillation due to hyperkalaemia is a common terminal event in uraemia. It is important to prevent a high potassium level, as once this is established it is very difficult to lower. Potassium is rigorously excluded from the diet—fruit juice is quite a potent source. Cellular breakdown with potassium liberation is minimised by the high caloric diet already discussed. Depletion of sodium chloride must be corrected, as any diminution of circulating blood volume would allow liberated potassium to cause a correspondingly greater rise in blood potassium con-

centration. Finally, acidosis must be controlled, as it is thought that some of the excess hydrogen ions in acidotic states enter the cells in exchange for potassium ions, thus again causing a high blood potassium level.

If haemolysis or extensive soft tissue injury has occurred, however, the blood potassium will reach dangerous levels despite these precautions. Two emergency measures may be life-saving in these circumstances. A 50% glucose drip with 20 units of soluble insulin added to every 100 ml has been found to promote the passage of some potassium back into the cells. Unfortunately this requires the concomitant administration of quite large volumes of water, which may be dangerous. The alternative is the use of an ion exchange resin such as Resonium A, either by mouth or as a retention enema. These non-absorbable resins are saturated with sodium, and in the gut lose some of this sodium in exchange for an equivalent amount of potassium, which thus leaves the body per rectum. 30 g. of Resonium A may lower the blood potassium level by 1 m. eq/litre, and can be repeated thrice daily.

The final feature of acute renal failure is ACIDOSIS, as the anuric kidney is unable to excrete hydrogen ions, and the damaged tubules cannot manufacture bicarbonate. I have already mentioned the dangers of potassium intoxication which are intensified by acidosis. The bicarbonate level must be monitored, and if it falls seriously an appropriate amount of the daily water ration should be given as 1/6 molar lactate.

These measures constitute the basic regime in acute renal failure. The general medical care of these patients must maintain a very high standard, and extensive laboratory facilities are required. The blood chemistry must be analysed at least once every day. These patients rapidly become profoundly anaemic due to toxic depression of the bone marrow, and transfusion of packed cells may be required. The most important general measure, however, as I mentioned earlier, is the prevention of infection. This is important firstly because these patients are particularly susceptible, and secondly because if infection occurs the build-up of renal failure is much more rapid. Nursing in isolation is the ideal plan, but where this is not possible strict hygiene and the use of antiseptic creams, etc. can be rigorously enforced. If an infection does arise, antibiotics must be used with the greatest precision. No antibiotic must be used without the sensitivity of the organism being known. To make this possible routine daily cultures of blood, urine and sputum, should always be carried out. If this is done, by the time an infection becomes apparent clinically, the infecting organism is known and its sensitivity is being determined. The circumstances in which an antibiotic must be used "blind" therefore never arise.

Infection is the crucial factor in acute renal failure. If it is prevented, the patient can usually be tided over until urine flow is resumed by the conservative measures I have outlined. If infection occurs, renal failure progresses rapidly to a fatal stage despite these measures. Fig. 1 shows the rate of rise of BUN in infected patients compared with a non-infected group. It is in the treatment of this infected group that the so-called "artificial kidney" plays a principal part.

THE ARTIFICIAL KIDNEY

In the artificial kidney the patient's blood is led through a coil in which it is separated by a semi-permeable dialysis membrane from a bath containing

the plasma electrolytes in their normal concentrations. The patient's blood is thus allowed to equilibrate by simple diffusion with normal ionic concentrations. Blood urea and potassium levels are rapidly lowered, whilst bicarbonate ion passes from the bath into the blood to restore the alkali reserve. After some 4 to 6 hours of circulation through this coil, the blood chemistry will have been radically improved. The normal solution in the bath will of course have been altered, and must be changed at least once during dialysis.

Certain fixed biochemical criteria have been established as indications for dialysis. Thus if the blood urea level exceeds 350 mg % dialysis should be undertaken. This level is reached in a very few days in severely infected cases. A blood potassium level higher than 7-5 m. eq./litre (usually associated with soft tissue injury or haemolysis) or a bicarbonate level below 14 m. eq./l, calls for immediate dialysis.

Apart from these three fixed criteria, dialysis is undertaken if signs of undue clinical deterioration occur, such as mental confusion or the onset of twitching. Dialysis is also undertaken early in severely infected cases when it is obvious from the outset that conservative measures will be inadequate.

The only contra-indication to use of the kidney lies in the necessity for anticoagulants. In certain patients this is obviously a risk, but as dialysis is a life-saving procedure this risk must be taken. It must also be admitted that in using extra-corporeal dialysis we are interfering with physiological mechanisms which are far from being fully understood, and many unexplained sudden deaths occur shortly after the blood chemistry has been rendered apparently normal. These deaths occur, however, in patients who were extremely ill before dialysis, and thus they perhaps constitute an argument for using dialysis early rather than for not using it at all.

It should be remembered that the anuric phase of acute tubular necrosis is followed by a diuretic phase. During diuresis the patient can become dehydrated, and due to delay in the recovery of tubular reabsorptive powers serious urine loss of sodium, chloride and potassium can occur. Indeed, before treatment of renal failure became effective, over 25% of the deaths occurred not in the oliguric phase but during the subsequent diuresis. A plentiful water intake must therefore be ensured. Salt losses can be estimated by monitoring the serum electrolytes and also by measuring the quantities of the various ions lost in the urine. Supplements of these ions may be required until normal renal concentrating power is regained.

CAUSES OF CHRONIC RENAL FAILURE

In acute renal failure specific treatment is rarely able to influence the causative pathology. The important procedure is to treat the physiological dislocation and maintain the patient until spontaneous recovery of renal function takes place. Chronic renal failure however is caused by progressive fibrosis of the kidney, and no regeneration of renal tissue takes place. The most important step in chronic failure is therefore to determine the underlying cause of the condition and attempt to arrest the pathological process.

Table 3 shows a few of the more common causes of chronic renal failure. The order looks haphazard because I have placed the conditions which can be treated with most success at the top of the list. A chronic obstruction to the urinary tract as from prostatism can usually be eradicated. Chronic pyelonephritis is particularly liable to linger on, progressively destroying renal tissue, in the pre-

sence of obstruction. Removal of the obstruction followed by isolation of the organism and appropriate intensive chemotherapy may arrest this process. Tuberculous infection may also be eradicated. Hydronephrosis will regress after removal of the obstruction. The renal lesions in hyperparathyroidism, diabetes, and even amyloid disease may regress with treatment of the underlying condition. Control of the blood pressure will arrest the progressive renal sclerosis which accompanies hypertension. Treatment for polyarteritis, D.L.E., the

TABLE 3

Some Causes of Chronic Renal Failure	
Chronic urinary tract obstruction Chronic renal infection, inc. T. B. Hydronephrosis Hyperparathyroidism	Amenable to treatment
Diabetes Amyloid disease Hypertensive vascular disease	Partial recovery possible
Diffuse angiitis, D.L.E., etc. The leukaemias Chronic nephritis Polycystic disease	Palliative therapy only
Associated with the Nephrotic Syndrome	
Type II nephritis Chronic glomerulonephritis Diabetes	Amyloid disease Thrombosis of the renal veins

leukaemias and so on can only delay the inevitable fatal outcome. When these potentially treatable conditions have been ruled out, we are left with a hard core of cases due to chronic nephritis and polycystic disease which are not amenable to treatment.

Other treatable factors affect the course of C.R.F. I make no apology for returning once again to infection. Any systemic infection places an additional strain on the kidneys and intensifies the degree of renal failure. If the infection is eradicated, failure will become less marked, and the patient may continue tolerably well for a further indefinite period. All the causes of acute renal failure are particularly liable to occur in the course of chronic failure, and again if the acute phase is energetically treated the patient may regain reasonable health.

THE NEPHROTIC SYNDROME

I have grouped certain causes of chronic renal failure separately because they are often associated with the clinical picture known as the nephrotic syndrome. The hallmark of this syndrome is massive proteinuria, with marked lowering of the plasma proteins and consequent gross oedema. It can occur when the kidneys are involved by any of these processes, but conversely any of them may progress to chronic renal failure without ever presenting a nephrotic picture. Often the proteinuria will regress spontaneously, and the oedema clears. Whilst recovery may be permanent, most cases present again some years later with chronic failure. Indeed, some cases go on to extensive renal fibrosis with renal failure whilst proteinuria and oedema still persist.

In children the nephrotic syndrome is nearly always due to type II nephritis. That is, its onset is insidious, and no underlying cause such as diabetes exists.

The histology of the kidney presents a typical appearance. There is now good evidence that steroid therapy is of benefit in these cases. Arneil in Glasgow reviewed his patients at a date two years after the onset of the disease. Previous to the use of steroids only 62% of his patients survived this period. Using prednisolone the two year survival rate is 91%. Regression of proteinuria is quicker and more of the patients are symptom-free after two years. There is some evidence that under steroids the electron-microscopic appearances of the kidney sometimes return completely to normal. Although steroids have proved far from completely successful their use is thus a real therapeutic advance.

TABLE 4

Treatment of Chronic Renal Failure

Failing function :	**Therapeutic measures:**
Excretion	Low protein, high caloric intake Generous water intake
Water balance	Generous water intake
Electrolyte balance	Prevent NaCl depletion Prevent K+ intoxication (Prevent Ca ++ depletion)
Acid-base balance	Administration of alkali

In the adult presenting with the nephrotic syndrome one of the other causative factors, such as diabetic nephropathy is commonly found, and treatment is aimed at the basic disease. Even with diagnosis by renal biopsy, however, a certain number of so-called idiopathic cases do present, in which no specific abnormality is found, and which may be benefited by steroids.

THE TREATMENT OF CHRONIC RENAL FAILURE

As in acute failure, all the functions of the kidney are impaired. The ability to concentrate and dilute the urine is lost, and large volumes of urine isotonic with plasma are excreted. Excretion of urea is impaired and as in acute failure a low protein, high caloric diet is essential. Anorexia is common, but the disease may run on for several years and it is essential to insist on a good diet being taken. Vitamin supplements are commonly prescribed.

If the tonicity of the urine is constant it follows that the greater the volume of urine passed, the greater the excretion of waste products. A generous water intake should therefore be ensured to promote excretion. An intake of from 2 - 3 litres is also required to preserve water balance, since polyuria is a constant feature.

It is a common misapprehension that salt should be restricted in renal failure. There is no indication for salt restriction in the absence of oedema or cardiovascular disease. These patients become salt depelted, both through renal wasting and by vomiting and diarrhoea. Indeed a supplement of 5 G/day of sodium chloride should be given. Much of the malaise associated with chronic renal failure is in fact due to chronic dehydration from salt depletion and failure to replace the large urinary water loss.

Some rare types of chronic failure do lead to potassium wasting. Usually, however, levels tend to be high, and potassium intoxication is a danger. The

prevention of this is on the lines suggested in discussing acute failure. The calcium loss does not usually require treatment.

Lastly, pH balance. The mechanism through which the acidosis occurs in chronic failure is a complex one. The end result, however, is a depletion of the alkali reserve, and oral supplements of sodium bicarbonate are therefore the correct treatment.

The regime which I have merely outlined is suited to most cases of chronic renal failure, but the nephrotic group with gross oedema present a different problem. In the presence of massive proteinuria a high protein, high caloric diet is indicated. There is no call for water restriction, but sodium intake should be restricted to less than 1 gm/day. Where the use of steroids is not applicable, long term treatment with chlorothiazide and potassium chloride supplement should be instituted and continued for as long as the drug remains effective.

I have already stressed the dangers of infection, and the likelihood of acute episodes disrupting the normal chronic course. A third hazard is cardiovascular disease. The great majority of these patients develop hypertension, followed by cardiac failure which complicates the terminal stages of the disease. Anaemia is quite intractable, and in general the haemoglobin is kept at about 40% by periodic transfusions of packed cells. Diarrhoea, vomiting, muscular twitching, mental changes and convulsions all require symptomatic treatment in the terminal stages.

Chronic renal failure always progresses to a stage where despite treatment the blood urea is high and rising, the potassium level is dangerously high and the alkali reserve dangerously low. At this stage the artificial kidney can be used, but its benefits are short-lived. The grafting of permanent plastic cannulae into the forearm vessels is being developed, through which the patient can be periodically "plugged in" to the artificial kidney. One questions, however, whether it is justifiable to prolong a life, which is an increasing burden to the patient, in this way. The main function of the kidney in chronic failure is in the treatment of acute episodes when the patient still possesses sufficient renal tissue to continue a useful life afterwards. The future cure of chronic renal disease may perhaps come by a break-through in the field of kidney transplantation.

In all the renal diseases, the many pathologies interfere with a single physiological unit. My hope has been that the physiology of the kidney would provide a unifying backcloth against which it would be profitable to survey the whole range of its diseases.

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THE EDINBURGH INFLUENCE ON EARLY AMERICAN MEDICINE

By WALTER MERCER, Kt., F.R.C.S.

Extracts from the Inaugural Address to the Royal Medical Society on Friday, 13th October, 1961.

Mr. President and Gentlemen, thank you very much for the honour of your invitation, for the courtesy of your presence and for what I am afraid will have to be the patience of your listening.

I think it is not inappropriate to gather together some of the connections of our Medical School with the formative years of American medicine, and to present it in these rooms, for it seems that many of the American students to be mentioned were members of this ancient and honourable Society.

Important as have been the impulses derived from other sources, kinship, community of language and intercourse have combined to render the influence coming from Scotland the dominant one in the development of American medicine. This is particularly true of the Colonial period and the first half-century of independence of the United States.

It has been quite amazing to me to discover so many connections between this School of Medicine and the United States. There does not appear to be one state that has not had some connection in this way, and, indeed, I have had to make a selection and have inevitably omitted many.

America could boast of many educated and distinguished practitioners prior to the founding of the first Medical School in Philadelphia in 1765. The educated physicians were probably located on the Eastern seaboard in Pennsylvania, New York, Maryland and the Carolinas. The majority had gained their knowledge by apprenticeship, which often meant picking up fragmentary information on diseases during casual conversations with the doctor, and by medical reading. There were only sporadic attempts to impart instruction in groups—especially in anatomy.

NEW YORK

One of the most distinguished, as well as one of the earliest practitioners was Cadwalladen Colden (1688-1766). Colden, the son of a Presbyterian minister of Duns, Berwickshire, graduated M.D. in Edinburgh in 1705. He emigrated soon after, and practised in Philadelphia from 1708 to 1715. Finally he settled in New York. He had a wide scientific interest and published articles on botany and other subjects. He wrote an "Essay on Iliac Passion,"

an old term for what we call appendicitis, which was printed by Benjamin Franklin at Philadelphia in 1741.

Founded in 1754 by a Royal Grant from George II, Columbia College in New York began as King's College. After the revolutionary war it was re-opened as Columbia College and in 1912 the title was changed to Columbia University in the City of New York.

I should now like to consider a group of men centred about the medical department of this King's College who organised this, the second medical school in the American Colonies. There was John Band, then the most influential man in New York. However, he did not have any connections with Scotland except that his son, Samuel, came to Edinburgh. He sat under Cullen, Monro and Fergusson, carried off the Hope Medal, and gained his M.D. in 1765. Like a number of men from the Colonies he was a member of the Royal Medical Society. Band wrote home at this time saying that he hoped to start a Medical School at New York. His dream came true and he was made Professor of Medicine (1767 - 1776) at King's College. He addressed the first men to receive their medical degrees on the "Duties of a Physician." This was published and became a medical classic and is known as the first American publication on medical ethics. His plea for a hospital was not in vain and in 1767 a medical department within King's College was instituted. The medical school was opened with six professors, of whom four had been in Edinburgh. In 1780 Band operated on George Washington and opened a large carbuncle of the thigh.

John Jones belonged to the same King's College group. He studied in London under William Hunter and Percival Pott, and later studied in Edinburgh. He did the first lithotomy in New York and attended Benjamin Franklin in his last illness. When it was founded in 1767 Jones became the Professor of Surgery and Obstetrics at the Medical Department of King's College.

It was Peter Middleton and John Band who carried out dissections at New York (1752)—the first in the country, it is said, to be done before a class. Middleton was born in Scotland, and, after graduating in Edinburgh, served in a surgical capacity at the Battle of Culloden (1746). Later he received an M.D. at St. Andrews and was a member of the Royal Medical Society in 1749. He practised in New York and helped to organise the Medical School at King's College, and, in the Medical Faculty, was Professor of Pathology and Physiology (1767 - 1776) and of Materia Medica (1770 - 1776). He was the earliest historian of American medicine.

It would appear that these three men, Band, Jones and Middleton, all with Edinburgh connections, held between them many medical chairs in the new Medical Department of King's College.

There were, however, two other men with Edinburgh connections in New York at this early period. Samuel Lothian Mitchell (1764 - 1831) obtained his M.D. degree in Edinburgh in 1786 and became a life member of the Royal Medical Society. He did not keep strictly to the path of medicine but became Professor of Natural History, Chemistry and Botany at Columbia College. He founded the first American medical journal, the *Medical Repository*. John S. Bellings said of Mitchell that he had very properly been called a "choc of knowledge".

One of the best known medical practitioners in New York at the end of the 18th and beginning of the 19th Century was David Hossack. After taking his medical degree in the University of Pennsylvania he came to Edinburgh for two years and was a member of the Royal Medical Society. He practised

as partner of Samuel Band and was the first person in America to tie the femoral artery for aneurysm. He was also Professor of Botany at Columbia College and he founded the Elgin Botanical Gardens in 1801—now the site of the Rockefeller Center.

Nathan Smith, Professor of Physics in the Medical College at Yale was the son of a farmer in Massachusetts. He saw an operation and wanted to know more about the craft of surgery. In 1790 he graduated at Harvard and asked for approval to found a medical college at Dartmouth. Help was postponed, so, perhaps to perfect himself, he went to Edinburgh. From there he sent to America £30 worth of medical books and on his return established the Medical College of Dartmouth, being appointed Professor of Anatomy, Surgery and Chemistry. His lectures excited the greatest interest. President Wheelock, Smith's greatest helper, was so impressed that after returning from a lecture he led evening prayers in the Chapel by saying " Oh Lord, we thank thee for the oxygen gas, we thank thee for the hydrogen gas, and for all the gases. We thank thee for the cerebrum, we thank thee for the cerebellum and for the medulla oblongata." In some of his methods Smith was 50 years in advance of his time. His ingenuity was displayed in his manoeuvre in reducing congenital dislocations of the hip joint. He was the second person to perform ovarectomy and, perhaps, was entitled to the same honours as McDowell since the operation was the result of deliberate study and done in ignorance of McDowell's first operation 12 years previously.

CAROLINAS

The Carolinas owe much to Scottish settlers. Indeed there would seem to be a trace of Scottish accent even to-day in the speech of Charlestonians. In these early days much Gaelic was spoken. A Commonwealth Fund Fellow wrote : " One old man told me a tale of three young Highlanders who rowed up a river in the closing years of the eighteenth century and landed at the bluff not far from Crossereek. They saw a negro approaching and resolved to have a mild joke at his expense and asked him in Gaelic the way to the plantation for which they were bound. The slave belonged to Scottish settlers and spoke little else but Gaelic himself. He answered them in the dialect. It was the newcomers' turn to be surprised. 'Yes indeed,' said the negro, 'I am also Gaelic, and when you have been here as many years as I have, you will be just as black too.' The three jesters immediately jumped into their waiting boat and made haste to leave such an unhealthy atmosphere."

Dr. William Walshe says of the physicians of his time (1750 - 1775) that there was not a more attractive and cultivated group of medical men. Of these, Chalmers, Moultrie and Gardner were trained in Edinburgh.

Lionel Chalmers was born in 1715 in Argyllshire and studied in Edinburgh; he practised in South Carolina for 40 years. His papers on opisthotonos and tetanus were published by a society of physicians in London in 1767. He was succeeded by Tucker Harris, a Charlestonian who graduated in Edinburgh M.D. in 1771. Later, Harris became one of the original members of the Medical Society of South Carolina constituted in 1789.

The first native of Carolina to obtain an Edinburgh degree was John Moultrie in 1749. His son James was instrumental in organising the first Medical School in the Southern States which came into existence in 1824. This ever-persistent faculty carried on lectures and proceeded to open a hospital for the express purpose of bed-side teaching.

WEST INDIES

James Lind, known as the Father of Nautical Medicine, was born and educated in Edinburgh. After qualifying at 23 and obtaining his M.B. in Edinburgh in 1748 he entered the Navy and spent much of his time in the West Indies, carefully observing and recording everything he saw.

In 1753 he published his classic treatise on scurvy. This treatise contains the simple account of his classic experiment on scurvy which seems to have been the first deliberately planned controlled experiment ever undertaken on human beings. The results of his experiment are simply stated. Two patients with scurvy were given two oranges and a lemon or lime daily; at the end of six days they were fit for duty. At that time in the Navy as many men died from scurvy as from battle wounds. A consultant physician of today (F. M. R. Walshe) remarked that "Dr. James showed what scurvy is due to and advised its treatment with lemon juice as we do today." The Navy adopted Lind's recommendation after an interval of forty years. It was 150 years before physiology, having taken that time to come round to it, discovered the existence of Vitamin C.

PHILADELPHIA

About the middle of the 18th century, when American students were coming to Edinburgh in increasing numbers, without doubt the primary attractions were the eminence of its teachers and the practical training the school could offer. There were, however, other influences at work, above all the council and guidance of Benjamin Franklin, who availed himself of his friendship with Cullen and other professors to give letters of introduction which opened hospitable homes in Edinburgh.

Treated on terms of friendship and esteem by the most intellectual circles in Britain, Franklin used his opportunities to aid his countrymen in their efforts to better cultural affairs in America and the profession of medicine at least amply repaid him. He was responsible for Americans later notable in medicine coming to Edinburgh to study the subject—men such as Shippen, Morgan, Kuhn, Rush, and many others. He wrote much on medical matters, on diet, the heat of the blood, lead poisoning, and gout. In 1787 he was elected an Honorary Member of this Society for his service to medicine.

One who bore a letter of introduction from Franklin was William Shippen. After a period of study in London and Edinburgh, where he graduated M.B. in 1761 and was elected later an Honorary Member of the Royal Medical Society, he returned to Philadelphia and took up practice and teaching. The Pennsylvania Hospital had been founded in 1751 with Franklin as the first President, but medical education may be said to date from 1762 when William Shippen began anatomical demonstrations in his father's house. Shippen was learned, eloquent, equable and kind. He had a pleasing personality and a fine sick-room presence. He guided the medical department of the American Army during a considerable part of the revolutionary war. He was a co-founder of the first American medical school, a noted pioneer teacher, and a skilled surgeon and obstetrician.

Until the temporary abrogation of the charter in 1779 every member of this faculty of the first American medical school was a graduate in medicine of the University of Edinburgh.

Some time later Cullen received another letter from Franklin—"I thank you for your civilities you were so good as to show my friend, Mr. Shippen, last year. Give me leave to recommend one more friend for your advice and countenance. The bearer, Mr. John Morgan, is a young gentleman of Philadelphia whom I greatly esteem."

John Morgan was one of the most important men in American colonial medicine. He was born in 1735 and later came to Britain to work with William Hunter. He then spent two years in Edinburgh where he graduated M.D. in 1763, having become a member of the Royal Medical Society in 1762. He talked with his teachers of his plans to form a medical college. This he did on his return to Philadelphia in 1765. The Medical Department of the University of Pennsylvania, then known as the College of Philadelphia, thus became the first medical school in the American colonies. Morgan was the first Professor of Medicine and Shippen was Professor of Surgery and Anatomy. Morgan's inaugural address is a medical classic. In it he outlines a scheme for transferring medical sciences into the seminary and for the improvement of every branch of the healing art. Possibly he says, in a few years a plan may be adopted conformable to that which is followed in the so justly celebrated School of Physic in Edinburgh.

Shippen and Morgan were joined by Adam Kuhn in 1768 and Benjamin Rush in 1769—both graduates of Edinburgh University. Kuhn was a Professor of Botany and Materia Medica and was one of the founders. In 1808 he became President of the College of Physicians of Philadelphia. Little is known of his writings save a short letter on " Diseases succeeding transplantations of teeth " ! He entered the sick room at a given time, stayed a given time, and never suffered a deviation from his habits.

Now we come to the last of the quartet—Benjamin Rush—the most conspicuous medical character of the century in American history. He came to Edinburgh armed with letters from Franklin to friends here. He graduated M.D. in 1768 and became a member of this Society in 1767. Rush was admitted as a " Burgess and Guild brother of the City in its most ample form ". Unfortunately I do not know why this honour was conferred on a medical student of 22. His life in Scotland was noteworthy, quite apart from his medical associates, by his friendship with Wotherspoon whom he strongly and successfully urged not to refuse a second invitation to become President of Princetown.

Three subjects were close to his heart : the freeing of negroes ; the abolition of the death penalty ; and the restriction of the use of alcohol and tobacco. However, he wrote of the disease, thermic fever, describing it and its treatment in " An account of a disease occasioned by drinking cold water in warm weather." In regard to his statement " medicine is my wife and science is my mistress," Dr. Holmes has added the caustic comment—" I do not think that this breach of the seventh commandment can be shown to have been of any legitimate advantage to the owner of his affection."

He recognised but two kinds of remedies, stimulants and depressants, and held it to be the principal duty of the physician to decide as to which was the most advisable in a given case. He called calomel " the Samson " of materia medica, and his opponents contended that he was right since undoubtedly it had slain its thousands.

I should mention another more recent Philadelphian, Silas Weir Mitchell. He was of Scottish origin. Weir Mitchell's own scientific relations with both London and Edinburgh were close. He was an F.R.S., a member of the Medical Society of London and the Clinical Society of London, and in 1895 he received the Edinburgh LL.D. He was wont to refer with much pride to the close connections which existed between Edinburgh and the early medical institutions of Philadelphia. Weir Mitchell was the leading American neurologist of his time. He had many writings to his name and investigated arrow poison. He wrote " Injuries to Nerves," one of the earliest accounts of nerve lesions.

VIRGINIA

One of the most distinguished naturalists and medical men in Virginia in the early part of the 18th century was John Mitchell. He studied botany under Charles Alston at Edinburgh in the early part of the 18th century and settled in Virginia in 1721. He described many plants and also suggested full bleeding and purgation as treatment for yellow fever.

James Craik, who became a well-known physician in Virginia, was born in Arbigland, Dumfries, and studied medicine in Edinburgh. He treated the mortally wounded General Braddock at the battle of Mononghabela in July 1754. Later he became a close friend of George Washington and attended him in his last illness.

Arthur Lee was among the fourth generation of a family which had been in Virginia since 1641. He was sent to school in Eton, England, and later studied medicine in Edinburgh. He was a friend of Samuel Johnson and said "Johnson's outward appearance is very droll and uncouth. The too assiduous cultivation of his mind seems to have caused a very great neglect of his body."

From Johnson he learned that at Cambridge or Oxford students were never permitted to attempt Physic until seven years study had enabled them to take a degree of Master of Arts. "If you have time and a large fortune go to either of those. If you would choose immediately to enter upon physic and to attain knowledge therein to carry you through life at a small expense and a short time, by all means go to Edinburgh, for this Scottish education is like a house, built to last a man's lifetime." Boswell described him as "an old friend of mine" when he studied physic in Edinburgh. In 1755 the committee on *Secret Correspondence of the Continental Congress* asked Lee to become a confidential correspondent in London. So, as secret agent there, he was the first diplomatic agent of the American nation.

Apparently the Virginian students of medicine were the only ones from the American colonies who had their own organisation in Edinburgh. The second article relating to the Virginia Club states that "This institution is supposed to be solely for the improvement of the members in anatomy." The third article is curious—"That every member of the club shall make it his endeavour if possible for the honour of his profession not to degrade it by hereafter mingling the trade of apothecary (or surgeon) with it."

Ephraim McDowell, the father of ovariotomy, was born in Virginia in 1771 and started medicine as an apprentice in Stanton, Virginia. Three years later he came to Edinburgh to complete his studies under John Bell.

McDowell, through Bell's eloquent teaching, was early impressed with the sad and hopeless fate of women afflicted with ovarian disease. One, Mrs. Crawford, was suffering from an ovarian cyst. No surgeon had ever dared hazard an abdominal operation and, indeed, all were opposed to it. McDowell knew that if he operated and she died, no doctor would disagree with a coroner's jury that found him guilty of murder. He describes the operation, which was carried out while she sang hymns. The tumour was so large that he first had to cut it open and remove 15 lbs. of dirty gelatinous fluid and then the sack weighing 7½ lbs. It was only by threats that he induced her to remain an invalid for 22 days but at the end of that time she insisted on riding back to her neglected family.

MASSACHUSETTS

James Tyler attended medical lectures in Edinburgh and supported himself by acting as surgeon on a trip to Greenland. After a number of financial difficulties he sailed for Salem, Massachusetts, and became a prolific writer on a variety of subjects.

MARYLAND

The State of Maryland, too, bears the imprint of the Edinburgh School. Gustav Brown, who studied in Edinburgh, went ashore from an English vessel. A storm carried the vessel out to sea, leaving Dr. Brown to an enforced habitation. He later became a well known and much liked practitioner.

The name Upton Scott (1772-1814) is a famous one in the history of medicine in Maryland. He studied in Edinburgh and was a member of the Royal Medical Society. After a career in the army, during which he attended General Wolfe, he helped to found the Medical Faculty of Maryland at Annapolis and became its first president.

CONCLUSION

I have tried to present a picture of some of the men educated in part in Edinburgh who went, afterwards, to America and in the early formative years did something for American medicine. It was on the foundations laid by these pioneers in medical education that America built her medical schools. The Edinburgh School owes a debt of gratitude to these pioneers who carried across the Atlantic the reputation of their own school and added to it by their own endeavours.

That visitors from the Edinburgh School are so welcome to-day in America is due in no small way to those who carved the name of Edinburgh over two centuries ago in the history of American Medicine. It is, indeed, a goodly heritage that they have left us.

SIR CHARLES BELL

By ROBERT BUTLER

Fellow of the Royal Medical Society.

Extracts from a Dissertation read before the Royal Medical Society on 3rd November, 1961.

It is told that one restless night, being struck by a sudden idea, Sir Charles Bell awoke his wife exclaiming, "Eureka! I pray God not to have sent me an empty dream, or to suffer me in the morning to recover the thread of this nocturnal vision. If I see with my waking eyes what I have caught a glimpse of in slumber I shall assuredly leave behind me an immortal name."

This was the dream of a man of great vision and genius, who, retiring to sleep, awakes once more with his dominant thought, an idea matured by the coalescence of inspiration and protracted study.

There can be no doubt that Charles Bell had intellectual brilliance in great measure; during his lifetime he was surgeon, anatomist, physiologist, artist and philosopher. In fact his critics have said of him that his academic life was too highly faceted, and that had his work been steered along a more homogeneous pathway, his reputation might have been all the greater.

* * *

Charles Bell was born in the Fountainbridge suburb of Edinburgh in 1774. His father was an Episcopalian clergyman, the Reverend William Bell, who had four sons—Robert, John, George and Charles—Charles being the youngest. In spite of his meagre income the priest had managed to give his three eldest sons a good education; these three were destined to become men of great eminence; Robert became the Professor of Conveyancing to the Society of Writers to the Signet; John was to become one of Edinburgh's most celebrated surgeons and George was to occupy the Chair of Scots Law in this University. It was, however, the youngest son who would immortalise the family name. Charles was unfortunate in that when he was yet only five years old his father died, and the financial circumstances of the family became even more precarious than they had previously been.

At the age of ten, Charles did attend school—the High School in Edinburgh—where he remained for four years. Speaking about it later in life he said, somewhat derisively "The education at the High School was to me torture and humiliation. Adams, loved by all good scholars, was to me a stupid tyrant. Anything mechanical or tending to natural philosophy I comprehended better than my companions, but the memory of verse or Latin rules without intellectual comprehension of some principles, I was almost incapable of. Memory

should be cultivated, it bestows great advantages. Mine was ever deficient. I could not and cannot venture on a quotation either in conversation or in public discourse."

While still quite young, he began to assist in his brother John's anatomy school, and at the same time attended lectures in the University. During the following years he became very expert in anatomical drawings, and while still a student published a work called "*A System of Dissections*" which was illustrated by his own drawings.

Eventually Charles came to practise surgery but, mainly due to the fierce disagreements between the University and his brother John, he realised that such success as he might achieve would not be accomplished in Edinburgh. And so it was with mixed feelings that he decided to take the long road to London.

Fig. 1.—Charles Bell in 1804.

Courtesy Messrs. E. & S. Livingstone, Ltd.; from Gordon-Taylor and Walls, "Sir Charles Bell—His Life and Times," Edinburgh, 1958.

At the time Lord Jeffrey wrote to another Scot who had taken the inevitable path to the English Capital: "All the world migrates towards London. My good friend Charles Bell is about to follow your bad example; he has almost determined to fly and establish himself in the great asylum. I have a warm regard for him; I can also assure you that you will find him extremely unassuming, intelligent, honourable, grateful . . . . "

He went South not completely unarmed for he had achieved some fame in his published account of the "*Nervous System and Special Senses*" and in the "*Anatomy of the Human Body*," which he had written with his brother. Also with John he had stressed the importance of a sound basis of anatomy for the proper practice of surgery. In fact the two brothers had virtually created the subject of surgical anatomy.

In addition, he had to his credit two other works—the beautifully illustrated

"*System of Dissections*" and "*Engravings of the Arteries, of the Nerves and of the Brain*"; and to this period also belongs his first great work, the celebrated "*Anatomy and Philosophy of Expression*".

In the "*Anatomy of Expression*", tracing the causes of movements in the countenance and in the body frame under the influence of emotion and physical change, Bell concerned himself with the course, origin and destination of nerves. Even in his earliest edition he shows his great interest in the nervous system saying, "If we had a perfect knowledge of the function of the nerves, they might, on all occasions inform us of the causes of those actions which appear to us inexplicable". Similarly, his early discussions of the functions of the nerves of the face foreshadow much of what was later to flow from his prolific pen. Certainly this creditable contribution to the understanding of human and comparative anatomy did much to build up and enhance Bell's reputation in this country and on the Continent.

Fig. 2.—The Nerve of Bell, with writing referring to his concept of a Respiratory System of Nerves.

Courtesy Messrs. E. & S. Livingstone, Ltd.; from Gordon-Taylor and Walls, "Sir Charles Bell—His Life and Times," Edinburgh, 1958.

It was some time before Bell received any financial success in London; at first, his income was based on the number of pupils attending his anatomy courses. He did have a number of surgeons at his school, but it was not until a few years later—when he began his own private practice and became consultant surgeon to the Middlesex Hospital—that he reaped any substantial monetary reward.

Later, in 1812, and encouraged by his wife, he took over ownership and became principal teacher in the Windmill St. School of Anatomy, which the Hunter brothers had made so famous. Here Bell taught and dissected and continued to brood upon the functions of the nervous system.

What was Bell like as a surgeon? We are certainly told that he had great manual dexterity, and it is also known that many operations caused him much anxiety. As Bell has said—"When a surgeon first takes the knife in his hand, and is preparing with oppressive feelings to perform an operation, which may terminate the life of his patient, he is not always aware of what is the most difficult to be accomplished. His ideas are vague, his mind not settled to what he is to expect; the circumstances which ought chiefly to engage him are not distinctly before him; and no man has ever performed this painful duty without feelings that it is in the very course of the operation that he learns what is most necessary for him to know and practise."

The Napoleonic Wars which ensued at this time gave Bell much opportunity for the treatment of gunshot wounds. In 1815 the great Battle of Waterloo was raging, and it did not take Bell long to decide that he must go to help as best he could the wounded of that campaign. He arrived some eleven days after the fighting had finished and found around him a wretched picture of human misery. He worked for days together leaving himself little time for food or sleep. As he says he "operated until his clothes were stiff with blood, and his arms powerless with using the knife."

Some two and a half weeks after the battle Bell visited that historic theatre of war, and he described the scene in his diary— " . . . Already silence dwells here; for although it is mid-day, and the sun bright and all shining in gladness, yet there is a mournful silence contrasted with the scene which has been so recently acting. No living thing is here—no kites, no birds of any kind; nothing but a few wretched women and old men, scattered on heights and at a distance, and who are employed in gathering cannon-balls."

* * *

Of all Bell's contributions to the arts and sciences his discoveries and work on the nervous system will ensure for him everlasting fame. He had been preoccupied with the nervous system of the human body from a very early age. This is reflected in a passage from his "*Anatomy and Physiology of the Human Body*," which Bell wrote with his brother John. He said, "On the subject of nerves my reader will find an account of that system which I have delivered in my lectures for the ten years past. That I have been so long in placing my views in a systematic work will only prove my respect for the received opinion, but, the manner in which so many of my professional brethren have allowed themselves to be assailed by new and fantastic doctrines, shewing little respect for the old, makes me hesitate less in substituting opinions different from these hithero admitted."

The ideas on the function of the nervous system before Bell's time were essentially "Galenical" in outlook. Von Haller in the middle eighteenth century had considered the nerves to be hollow for the transmission of "nervous fluid" from the brain to the periphery and vice versa. He also suggested that there might be two varieties of nervous fluid—one for sensation and one for movement—both being present in any one nerve.

* * *

As early as 1807 he wrote to his brother George in **Edinburgh**, saying, "My new '*Anatomy of the Brain*' is a thing which occupies my head almost entirely. I hinted to you formerly that I was burning, or on the eve of a grand discovery. I consider the organs of the outward senses as forming a distinct class of nerves from the others. I trace them to corresponding parts of the

brain totally distinct from the others ; I take five tubercles within the brain as the internal senses. I trace the nerves of the nose, ear, eye and tongue to these. Here I see established connexions. Then the great mass of brain receives processes from these central tubercles. Again the greater mass of the cerebrum sends down processes or crura, which give off all the common nerves of voluntary motion, etc. I establish thus, a kind of circulation as it were. In this enquiry I describe many new connexions. The whole opens up in a new and simple light ; the nerves take a simple arrangement ; the parts have appropriate nerves and the whole accords with the phenomena of the pathology and is supported by interesting views."

Bell entertained the idea that the parts of the brain were distinct in function. It occurred to him that there were four divisions of the spinal marrow—first a lateral division, then a division into the back and fore-part. It also occurred to him that the spinal nerves had within the sheath of the spinal marrow two roots—one from the back part and another from before. These are the thoughts which led him to perform his classic experiments.

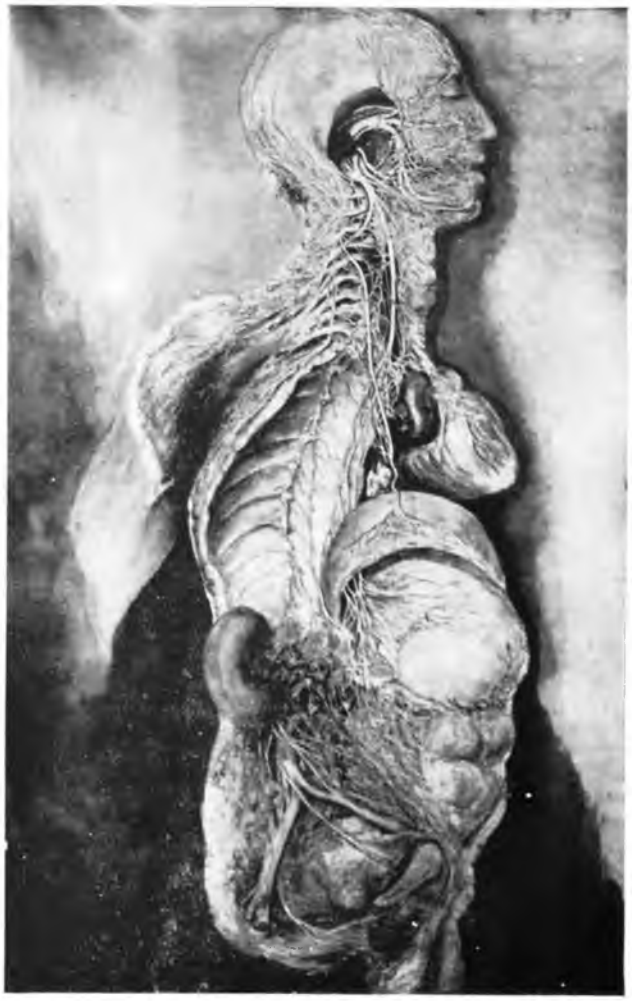

Fig. 3.—The autonomic nervous system, the cervical and brachial plexuses, the facial and trigeminal nerves.

Courtesy Messrs. E. & S. Livingstone, Ltd.; from Gordon-Taylor and Walls, "Sir Charles Bell—His Life and Times," Edinburgh, 1958.

In the first he opened the spine of an animal and injured the posterior filaments of the nerves—no motion of the muscles followed—then he touched the anterior division and immediately the parts were convulsed. In his second

experiment he destroyed the posterior part of the spinal marrow by the point of a needle—no convulsive movement followed ; when he injured the anterior part the animal was convulsed. Thus had Bell demonstrated a separation of function between the anterior and posterior nerve roots.

The following year, in 1811, Bell published his famous "*Idea of a New Anatomy of the Brain,*" which explained his views on the structure and functions of the nervous system. In it he states that the cerebrum and cerebellum have different functions and that diverse roles are incorporated in the varying parts of the cerebrum. "The nerves," he says, "are composed of filaments which, though distinct in office, are united for convenience of distribution." This would solve the question of how a nerve might lose one property and retain another. On the basis of gross anatomy, Bell wrongly believed that there were exclusive connections between the posterior roots and the posterior columns and the cerebellum ; and that in a like manner the cerebrum was connected with the anterior columns and the anterior spinal nerve roots. Throughout the entire paper there is no direct reference to the anterior roots being motor and the posterior roots being sensory in function. Whether he had intended to convey such a view is not altogether clear—but even in his letter to his brother George, a year earlier, he had talked of the part of the spinal marrow "having sensibility," and the "posterior and insensible part of the spinal marrow." Probably had Bell carried out more animal experiments, which in these pre-anaesthetic days he had an utter distaste for, he might have been able to give a clearer picture of the function of the nerves. But, his ideas and experiments on the spinal nerves and the trigeminal and facial nerves sowed such seeds of interest in the mind of the Frenchman, Magendie, that this scientist was able, after doing repeated experiments along essentially similar lines to those done earlier by Bell, to state clearly the functions of the nerve roots—namely that the posterior nerve roots were sensory and the anterior nerve roots were motor. (This is nowadays known as the Bell-Magendie Law). Bell always disputed the fact that Magendie had discovered the function of the nerves, and for many years a bitter controversy raged between these two great neurologists. Probably in the final analysis it would be fair to say that Bell had demonstrated the functions of the spinal nerves, but that he had left it to Magendie to confirm and interpret accurately his results. In spite of Bell's wranglings with this French scientist, he had a great reputation on the Continent—greater it is said than that of Harvey—and the story is told that when he visited Paris one time Professor Roux introduced Charles Bell to his class and in the same breath dismissed them, saying "*C'est assez messieurs, vous avez vu Charles Bell.*"

* * *

These are not the only important contributions which Bell made to neurology. He was, as has been pointed out, greatly interested in the nerves of the face. At this time it was customary to divide the facial nerve in the treatment of *tic douloureux*. It was Bell who pointed out the fallacy of performing this operation, which merely resulted in facial paralysis, showing that it was the facial nerve which supplied the motor nerves to the facial muscles of expression, and that the trigeminal nerve was both sensory and motor.

Bell's doctrines and his excellent clinical teachings won for him a large clinical practice in London, and in conditions involving the facial nerve he was certainly second to none. His medical classic "*The Nervous System of the Human Body,*" which was published in 1830, and which is surely the first text-book of modern neurology, contains, in addition to all his experimental and clinical observations, a number of case histories of his patients—many suffering from Facial Palsy—or as it was later to be known "Bell's Palsy."

In 1835 the Chair of Surgery in the University of Edinburgh became vacant, and Bell was offered the post. The position was certainly inviting, for Edinburgh was pre-eminent among the medical schools of Europe, and Bell would have a chance to see his family again, and as he remarked "London is a place to live in, not to die in." So, at the age of sixty-two, he returned north to his birthplace. But he was by now in the autumn of his existence and in the year of 1842 Bell died.

* * *

In the death of Charles Bell, the world lost not only a master anatomist and surgeon, but also one whose skill as a painter was quite superb. Together with his talented brush was coupled an eloquence of expression which added so much to his more philosophical works. Perhaps indeed his pursuits were too numerous to enable him to complete his "*New Idea*" of the nervous system, the great ambition of his life. Yet, without Bell's work, for how much longer would the nervous system have remained in such obscurity? Even if Charles Bell were denied his crowning glory, it was certainly he who shone the light of understanding on the hitherto inextricable confusion of the nervous system and pointed a path to the further elucidation of its hidden mysteries.

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RETROSPECT

225th SESSION

The Society has enjoyed another successful session. Of particular note were the Presidents' Annual Dinner and the launching of the Appeal Fund. At the Dinner, which was held in the Hall of the Royal College of Surgeons, and is said to have been quite a remarkable success, the Guest of Honour was Sir Derrick Dunlop.

THE APPEAL

At the Presidents' Annual Dinner, Sir Derrick took the opportunity in his speech to launch the Appeal on behalf of the Society. Since then, an attempt has been made to send copies of the Appeal to all male Edinburgh graduates in Medicine. Since we shall be obliged eventually to leave our present Hall—in the face of threatened compulsory purchase by the Midlothian County Council—an adequate response is imperative to enable the Society to build a New Hall on the site reserved for us by the University. In fact, the response to the Appeal is already encouraging.

THE SOCIETY

With regard to Public Business, the Addresses and Dissertations have been well received throughout the session. Private Business has been unusually well supported. The occasional introduction of guests, at Second Private Business, to lead discussion on particular topics has proved to be of particular value. Of the Inaugural Address, much is reproduced in this issue. The President's Valedictory Address, witty and informative, and likewise largely historical in content, dealt with the history of general practice.

Throughout the session, the Public Business Committee has been active in arranging film evenings, ward visits and other social functions. The Annual Ball, held in the Carlton Hotel was well-supported by members. The excellent cuisine and the warm *esprit* (to coin a phrase) both helped make a most pleasant and enjoyable evening.

ALIENATION OF THE INDEX MEDICUS

The question of alienating the Cumulative Index Medicus has again been raised. The annual subscription for this publication is high, the volumes are rarely consulted, and only a few of the periodicals referred to are available in the Society's rooms. The Society possesses the series complete except for the first two volumes. It is regrettable that such a need should arise, but, in accordance with the Obligation, the first step has been taken and the Librorum Custos was empowered to find, if possible, a worthwhile market.

THE JOURNAL

We are proud to publish in this issue the second in the series "Abdominal Crises," provided by Mr. I. S. R. Sinclair, Senior Lecturer, and Surgeon to the Royal Infirmary, and an article provided by Professor Whitteridge. The lecture, given to students in the Royal Infirmary and on which the article is based, has aroused the greatest interest.

To the coming session, the Society looks forward with confidence. The election of Mr. John Calvert to the office of Senior President gave the greatest pleasure, and the traditions of the Society will certainly be maintained.

BOOK REVIEWS

CLINICAL CHEMISTRY IN PRACTICAL MEDICINE, by C. P. Stewart and Sir Derrick Dunlop. Sixth Edition, published by E. & S. Livingstone, Ltd., Edinburgh, 1962. Pp. 359. Price 27s. 6d.

In this latest edition of a well proven and universally accepted textbook, there have been few major changes, reflecting the great popularity of previous editions. The revision has included recent advances insofar as they are significant in clinical medicine.

The student usually has some difficulty in coming to an understanding of the acid-base balance of the body. This perplexing subject is covered in an extremely lucid and unambiguous chapter. As is ideal, in a subject which is further complicated by the great variety of terminology, the writers make a point of defining all terms which are liable to cause confusion. Thus we are led from clear definitions of an acid and a base to all the irregularities which result from the failure of any one compensatory mechanism.

Used to advantage throughout the volume, this treatment, involving the application of first principles makes the book an excellent introduction to the use of laboratory techniques as an aid to diagnosis. When faced with a difficult case, there can be no better guide to further investigations.

The material added during revision includes a new section on magnesium and its relationship to electrolyte metabolism. The chapter on carbohydrate metabolism is substantially rewritten and contains much new information—in particular on the new hypoglycaemic drugs, the sulphonylureas. Interest in the baffling range of auto-immune disorders is reflected in a short section on the separation of plasma proteins by immuno-electrophoresis. Also, it is refreshing to see older terms such as the "carbon dioxide combining power" being replaced by the more convenient system of giving values in milliequivalents per litre. There is a short clear section on the relationship between cholesterol and coronary heart disease. As in previous editions the appendix gives clear instructions for the analyses, which, in the main, are performed in the clinical side-room.

Throughout, the printing and diagrams are of the same high standard as in the previous editions. There can be no doubt that this edition will enjoy the popularity of its predecessors, continuing to be essential reading for students and practitioners.

J.A.R.

THE PRINCIPLES AND PRACTICE OF MEDICINE. Edited by Sir Stanley Davidson, B.A., M.D., F.R.C.P., F.R.S.E. Sixth edition. Edinburgh, E. & S. Livingstone. 1962. Pp. xii + 1139. Price 35s.

As the sixth edition in ten years of this famous textbook becomes available, it becomes increasingly difficult to do anything other than enthuse over it, any minor criticisms that one might have being rather niggardly quibbles.

The physical dimensions of the work have once again been maintained at a reasonable level, there being an increase of merely 27 pages over the fifth edition. In spite of this, each chapter bears witness to a thorough review, and it is refreshing in a book of this size to see references to work as recent as 1961. Would that there were as little delay between the writing and publishing of other books!

Obviously, the therapeutic aspects

have benefited most from so recent a revision. However, in every aspect, there seems to be a freshness, when comparing this edition with the previous two. Perhaps this results simply from the reviewer's imagination, helped by the detergent whiteness of the improved quality paper.

As always from Messrs. Livingstone, the printing and binding are of a high standard; the number of slight misprints could be counted on one's fingers, putting to shame many more elaborate and expensive works. Finally, yet again the price remains unchanged, at a time when that of every other new edition is creeping up a few shillings.

If the above remarks have been largely subjective impressions of the new edition, rather than an objective review of the work as a whole, it is because, in this case, there is no question of whether one should buy the work, but simply of whether one should possess the 6th edition rather than the 5th. I hope the hesitant will have been convinced.

P.J.S.

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