RES MEDICA
Journal of the Royal Medical Society

Abdominal Crises 1

I. S. R. Sinclair
F.R.C.S.

Abstract
Despite the mechanical contrivances which clutter our modern world solutions to the crises which constantly beset us in every sphere still depend upon the wisdom of individuals. This is nowhere more true than in the management of abdominal crises which at some stage falls to the lot of every medical man. If disaster is to be averted, a correct diagnosis has to be made and a correct line of action determined within a space of time so short that the opportunity to call upon other opinions or to invoke the corroborative aid of laboratory tests may be severely curtailed. For the vast majority of doctors who are not practising surgeons the overriding responsibility is to decide whether an abdominal crisis is such that operative treatment may be needed. If the medical student, nurtured in the hospital environment gains the impression that this decision is one of no great difficulty he is forgetting that the wheat has already been separated from the chaff before the patient ever reaches hospital. It is a tribute to the general practitioners that so few patients are needlessly referred to hospital and that even fewer are referred with serious complications already established.

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

Res Medica, Autumn 1961, 3(1): 16-24
doi: 10.2218/resmedica.v3i1.372

ABDOMINAL CRISES
I.

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

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

PROBLEMS IN DIAGNOSIS

Despite the mechanical contrivances which clutter our modern world solutions to the crises which constantly beset us in every sphere still depend upon the wisdom of individuals. This is nowhere more true than in the management of abdominal crises which at some stage falls to the lot of every medical man. If disaster is to be averted, a correct diagnosis has to be made and a correct line of action determined within a space of time so short that the opportunity to call upon other opinions or to invoke the corroborative aid of laboratory tests may be severely curtailed. For the vast majority of doctors who are not practising surgeons the overriding responsibility is to decide whether an abdominal crisis is such that operative treatment may be needed. If the medical student, nurtured in the hospital environment, gains the impression that this decision is one of no great difficulty he is forgetting that the wheat has already been separated from the chaff before the patient ever reaches hospital. It is a tribute to the general practitioners that so few patients are needlessly referred to hospital and that even fewer are referred with serious complications already established.

THE HISTORY

The key witness in the doctor's efforts to reconstruct the pattern of events is, of course, the patient himself. The general practitioner has usually a considerable advantage over his hospital colleagues through personal knowledge of the patient which helps him to assess the reliability of the witness, but on occasions this knowledge may be dangerous and the memory that a particular patient or his relatives have often "called wolf" in the past must not be allowed to prejudice the family doctor in his assessment of each new episode. Even the most reliable patient may be reduced to incoherence by the distraction of pain or the exhaustion of vomiting while the senile, the psychotic, the unconscious and the very young by their inability to communicate deprive the doctor of his first vital diagnostic weapon.

The Interpretation of Pain

Except in the case of internal haemorrhage by far the most important single symptom of intra-abdominal pathology is pain. The threshold for and reaction to pain undoubtedly vary greatly from one individual to another. The new-born

infant is virtually insensitive to pain: the thick-set labourer accustomed to blows and tolerant of the pangs from a mouthful of carious teeth may conceal a gangrenous appendix without admitting to more than minimal abdominal discomfort. Again, pain appreciation may be disturbed under certain abnormal conditions, most notoriously after some major operation or injury. Under these circumstances the pain of the primary condition and the effects of analgesic drugs may obscure the true diagnosis. Quite as important under these circumstances is the inability of the attending doctors to maintain their alertness. Patients are as likely to develop abdominal crises in hospital as elsewhere yet once a patient has come under our care with an established diagnosis it goes against our nature to think that some other catastrophe may befall him and against our training to explain a fresh symptom on the basis of some new and unrelated condition. There is some truth in the charge that there is no more dangerous place in which to develop an abdominal crisis than a surgical ward.

Pain sensation may be impaired or absent in certain disease processes affecting the conducting tracts such as tabes dorsalis or syringomyelia, or following mechanical division of the tracts. A young soldier was admitted to hospital with a complete paraplegia due to a bullet wound of the upper dorsal spine. He deteriorated suddenly the following day with vomiting and a silent distended abdomen. At laparotomy there was a perforation of the jejunum with gross generalised peritonitis. Similar silent perforations have been reported in patients who have had a bilateral splanchnicectomy to control malignant hypertension or to relieve the pain of chronic relapsing pancreatitis. The splanchnic nerves carry only those afferent fibres whose peripheral endings lie in the visceral walls and the visceral peritoneum. The sensory supply of the parietal peritoneum passes along the segmental somatic nerves so that after splanchnicectomy only true "visceral" pain is abolished and pain will be felt as soon as the parietal peritoneum becomes involved. Thus the early central abdominal pain of acute appendicitis will be absent but local pain and tenderness in the right iliac fossa will appear as usual.

Suppression of the inflammatory reaction by gluco-corticoids provides another mechanism whereby the pain response may be modified and silent gastro-intestinal perforations have occurred in patients under treatment with these drugs.

Because of the median origin and scanty cortical representation of the intestinal tract the truly visceral component of abdominal pain is felt in or near the midline and is poorly localised. It shows some degree of segmental distribution but the limits of segmental innervation vary from patient to patient. Moreover, with the continuance of pain central summation results in a diffusion of the ascending impulses over an increasing number of segments. In other words the pain will become more diffuse even in the absence of a spreading pathological process within the abdomen. This explains the great importance of ascertaining the location of pain at the onset of the illness. At this time visceral pain from "foregut" organs—stomach, proximal duodenum, biliary tree and pancreas—will be located in the epigastrium; from the "midgut" organs—small intestine, appendix, ascending and transverse colon—in the umbilical region; from the hind gut, in the hypogastrium.

Back Pain is a frequent feature in disease of certain abdominal organs. Where the disease process involves the posterior parietes, as in pancreatitis or extensive carcinoma, this is deep somatic pain. Sometimes, however, back pain is present without any such involvement of the parietes due partly to misinterpretation by the sensory cortex of the origin of the painful stimuli; partly to spread of subthreshold visceral impulses on the posterior column

synaptic areas to nearby neurones carrying stimuli from the body surface; and partly to the anatomical limitation of the number of ascending fibres available, so that the same central fibre may perforce be used to convey messages brought to the cord by both visceral and somatic peripheral fibres. The posterior body wall is less liberally endowed with sensory fibres than the anterior wall and this may explain why a patient will rarely complain primarily of back pain but will often admit to its presence on being questioned. When, however, some disease is present in the components of the posterior body wall, for example osteoarthritis of the spine, the summation of subthreshold stimuli from the viscera with similar mild stimuli from the deep somatic nerve endings of the vertebral column may together produce back pain as a primary complaint. Although visceral pain may be misinterpreted as coming from both the posterior and anterior areas of the body surface it is remarkable that it is never described by the patient as following the distribution of the intercostal nerves: girdle pain does not arise from the hollow viscera. In most cases this so-called referred pain is experienced in areas which correspond fairly closely to the site of the viscus concerned and so blends with the true visceral pain but sometimes, through a developmental rearrangement of segments, referred pain may be felt in remote areas, for example the shoulder-tip pain of diaphragmatic irritation.

The most potent stimuli of visceral sensory endings are crushing, stretching and ischaemia. In contrast to the hollow viscera, solid organs are rarely subject to distension or strangulation and rarely give rise to acute pain. Renal pain arises from the renal pelvis and not from the parenchyma: the pain of splenic infarction is accurately localised somatic pain due to irritation of the parietal peritoneum. However the peritoneal capsule of a solid organ like the liver is sensitive to the tension produced by vascular engorgement so that acute congestive heart failure may mimic an upper abdominal crisis.

The mode of onset of abdominal pain is a valuable diagnostic pointer. Catastrophic abdominal pain of sudden onset is always of peritoneal origin and is due either to perforation of a hollow viscus or to torsion or strangulation. Inflammation, on the other hand, gives rise to an aching pain of gradually increasing intensity without the throbbing so common in inflammatory lesions of parietal structures. This pain is due to stretching of the visceral peritoneum over the swollen part and is similar to the pain of an engorged liver or of a tense pancreatic cyst. The same aching pain is felt in obstruction of a hollow viscus but in this case the spasmodic pain of excessive smooth muscle contraction is superimposed. Such "tension pain" is immediately relieved when the distension passes off. Thus relief may follow the drainage of a pancreatic cyst, the release of an obstructed loop of bowel or the removal of an obstructed appendix but it may equally well follow the rupture of the cyst or bowel or appendix into the peritoneal cavity. Sudden spontaneous relief of abdominal pain which has been constantly present for some hours must be viewed with the utmost suspicion. In the same way the sudden disappearance of pain from an active peptic ulcer is more likely to be due to the sudden decompression of the hyperaemic tissues by haemorrhage than to any medicaments and calls for a very close watch for signs of internal haemorrhage. In the absence of haematemesis or melaena the earliest indication of bleeding in the recumbent patient is very often the appearance of beads of sweat on the forehead. I have noticed this on a number of occasions some time before there was any rise in pulse rate.

Rarely, a patient will describe abdominal pain as cutting or stabbing in character. This type of pain is found where inflamed serous surfaces rub

together and so may be located in the upper abdomen in diaphragmatic pleurisy or the left hypochondrium and flank in perisplenitis. In the absence of such pathology this type of pain is suggestive of functional illness or of the rare but never to be forgotten tabetic crisis.

Vomiting and Bowel Dysfunction

When vomiting is present information as to its nature is second in importance only to analysis of the pain picture, although because of the great variety of diseases which may provoke vomiting, especially in children, the presence of vomiting *per se* is not of great help as a localising sympton. As with pain, the longer vomiting lasts the more likely is there to be a serious cause. The stage of reflex disturbance in acute appendicitis or in perforation of a peptic ulcer is short, and it is unusual for vomiting to be repeated more than once or twice. Torsion or internal strangulation on the other hand is productive of early and repeated reflex vomiting, the vomitus remaining small in quantity and watery or bilious in appearance.

In one typical case of "acute appendicitis" vomiting had occurred no less than seven times: the operative finding was torsion of a long Meckel's diverticulum. On another occasion repeated retching in a supposed case of perforated duodenal ulcer was due to torsion of a pedunculated mass of fat on the lesser omentum.

Obstructive vomiting classically progresses from clear gastric juice to green bile, then straw-coloured jejunal contents and finally to "faecal" fluid. But this progress is not always seen, being possible only in low intestinal obstructions. Besides, to await faecal vomiting before diagnosing obstruction is no less heinous than to await clinical metastases before diagnosing cancer.

The presence of constipation is of relatively little diagnostic value in the acute abdominal crisis and conversely the occurrence of a bowel movement shortly after the onset of symptoms does not exclude intestinal obstruction. However, the absence of a bowel movement for even one day after vague abdominal pains have developed remains one of the greatest hazards to the patient in his own home, and especially to children for the administration of a purgative remains common practice in cases of abdominal pain. In the presence of an obstructive lesion the augmented intestinal activity produced by purgation greatly increases the risk of perforation of the obstructed part. Should the clinical picture suggest to the doctor that his patient might have appendicitis, a history of recent purgation is a positive indication for hospitalisation.

Diarrhoea is often present in cases of food poisoning and is helpful in the differentiation of gastro-enteritis from appendicitis but it is otherwise rare in temperate climates.

THE EXAMINATION

Inspection of the abdomen, so often neglected, is directed first to observe movements—respiratory, pulsatile or peristaltic; secondly to observe the presence of abnormal fullness due to distension or masses; thirdly to look for less common findings such as the urticarial rash of food poisoning, the vesicular segmental rash of herpes, the "caput medusae" of engorged periumbilical veins of portal hypertension or hepatic metastases or signs of bruising in cases of injury.

PALPATION is always performed—after a fashion. Sir James Mackenzie wrote "There is not to be found yet any person who can intelligently palpate an

abdomen." The secret lies in the warm hand, the gentle pressure with the pads of the fingers and the recognition that just as one equation with two unknowns is insoluble, so it is pointless to expect results if the examining hand moves about while the patient is asked to breathe deeply! In particular, palpation should at first be light and cover all quadrants of the abdomen. Only in this way will relaxation be achieved enabling the presence of local rigidity to be detected. Deep palpation follows and will elicit tenderness of the parietal peritoneum, and the presence and characteristics of masses. There is no doubt that inflamed viscera are themselves tender to deep palpation. Thus the inflamed gall bladder, duodenum or appendix is acutely sensitive even when the anterior parietal peritoneum is not involved. Palpation must always include the external hernial orifices: in the obese patient the fold of the groin can easily hide the small bulk of a strangulated femoral hernia. When a peritoneal surface is inflamed any sudden change in position or tension gives rise to pain. Because of the unpleasant sensation produced by sudden pressure on the abdominal viscera, the sudden application of pressure is unhelpful in recognising peritoneal pathology but the sudden withdrawal of the palpating hand will produce pain only if the peritoneal surface is unduly sensitive. This phenomenon of "rebound tenderness" is quite non-specific but I believe it to be a useful index of some degree of peritoneal irritation and hence of primary intra-abdominal pathology. Certain more subtle tests such as the presence of hyperalgesia of the abdominal skin due to viscero-cutaneous segmental summation are used by some surgeons but have proved of little practical value in my hands.

Just as the sudden relief of pain may give rise to a false sense of security so the absence of acute signs in abdominal examination is liable to misinterpretation. The lack of signs of intraperitoneal pathology in the heavily muscled male has already been mentioned and the same is true in the obese and the debilitated. In the patient severely ill with typhoid fever or ulcerative colitis the only sign that perforation of the bowel has occurred may be the sudden onset of peripheral circulatory failure. The absence of pain in the early stage of peritonitis in the patient on cortisone, with a spinal cord lesion, or after splanchnicectomy is paralleled by an equivalent lack of physical signs. But the commonest pitfall occurs in the patient with an acutely inflamed pelvic appendix. In the early stages such a patient may show no abnormality whatever on careful abdominal examination, though rectal examination will disclose acute tenderness in the pelvic peritoneal pouch. When tenderness does spread into the abdomen it may first appear in the left iliac fossa to which the inflammatory exudate is diverted by the pelvic meso-colon. The inflamed retrocaecal or retro-ileal appendix may likewise produce only minimal signs of peritonitis, and the same is true of acute pancreatitis and of perforation of a gastric ulcer into the lesser peritoneal sac, in all of which circumstances the anterior parietal peritoneum is protected from the pathological process.

ABDOMINAL PERCUSSION is of limited value except in the detection of and distinction between a pelvic mass, such as a distended bladder or ovarian cyst, and gross ascites. Attempts to elicit shifting dullness are seldom justified in acutely ill patients while the reduction in liver dullness in cases of perforation of a hollow viscus is so inconstant as to be of little diagnostic value.

AUSCULTATION is directed principally to the detection of an increase or decrease in peristaltic activity, or more rarely to the elicitation of a friction rub as in splenic infarction or of a systolic arterial murmur in arterial aneurysm. It is often forgotten that the normal abdomen may remain silent for periods

of over one minute. To be of value, therefore, auscultation must be prolonged. Furthermore, active peristalsis may continue in the presence of localised intraperitoneal pathology while it will cease altogether in severe shock due to extra-abdominal injury.

ANCILLARY DIAGNOSTIC AIDS

Despite what has been said about the need to establish a diagnosis quickly in the absence of ancillary techniques the value of laboratory and radiological techniques must never be forgotten. The leucocyte count, microscopic examination of the urine and determinations of serum amylase and blood urea are all extremely useful tests in the abdominal crisis. The value of the plain abdominal X-ray has long been recognised, especially to demonstrate the presence of free peritoneal gas (indicative of perforation of bowel) or of gas and fluid levels in the small intestine (indicative of intestinal obstruction). More recently the emergency use of contrast films has become established. With water soluble opaque media, for example, the presence of gastroduodenal or bladder perforations can be safely and reliably confirmed.

EXTRA-ABDOMINAL CAUSES OF ACUTE ABDOMINAL SYMPTOMS

Acute infections

Apart from the pancreatitis of mumps it is not uncommon for acute infections in children to give rise to abdominal manifestations. Thus an acute pharyngitis is often associated with abdominal pain and vomiting, due perhaps to a reactive mesenteric adenitis. In such cases the history of a sore throat, the faucial congestion, the flushed face with circum-oral pallor, the localisation of abdominal tenderness along the line of the mesenteric attachment above the usual location of the appendix and the absence of signs of progressive peritonitis may enable a confident diagnosis to be made and expectant treatment adopted.

Thoracic disease

In older patients confusion between CORONARY THROMBOSIS and an upper abdominal emergency may lead to serious errors in management. When pain of thoracic origin is referred to the abdomen the muscles may exhibit reflex spasm which closely mimics rigidity, but tenderness is absent under the rigid muscles and the muscle spasm often relaxes during inspiration, a phenomenon never seen in rigidity due to intra-abdominal pathology. The presence of pleural or pericardial friction rubs and of other cardiac or respiratory symptoms and signs will usually resolve the dilemma. If not, the electro-cardiogram has reached a remarkable degree of accuracy in the detection of either myocardial infarcts or major pulmonary emboli and in the distinction between these, while estimation of the serum transaminase reveals an elevation in most cases of myocardial infarction within twelve hours. However the transaminase may also be elevated in liver disease or acute pancreatitis.

DISSECTING ANEURYSM of the aorta may occasionally present as an abdominal crisis but as the dissection almost always starts in the ascending aorta the history is of pain starting in the neck, chest and back and travelling downwards through the abdomen to the legs. This history associated with diminution of pulses in some or all of the major branches of the aorta is virtually pathognomonic. The basis of contemporary treatment is the attempt to ensure by surgery what nature rarely achieves unaided—the re-entry of the dissecting current of blood in the lumen of the aorta.

Spontaneous rupture of the oesophagus characteristically occurs during a bout of post-prandial vomiting. The sudden retrosternal pain is more likely to be mistaken for a coronary thrombosis than a perforated peptic ulcer in the absence of a dyspeptic history. The tear, always at the left side of the lower thoracic oesophagus, ruptures early into the left pleural space where air and fluid containing food or gastric juice accumulate and upward extension of air

through the mediastinum may produce surgical emphysema in the root of the neck. The only chance of survival lies in immediate suture of the rent.

Spinal disease

OSTEOMYELITIS of the vertebral bodies is an uncommon cause of abdominal crisis. To add to the diagnostic difficulty this condition sometimes arises in the wake of an acute abdominal upset, perhaps due to direct spread of infection by way of the vertebral veins or the lymphatic trunks which course close to the vertebral bodies. Clinical examination always reveals localised tenderness over the spine of the involved vertebra while in some cases a tender, boggy mass is palpable per rectum, not anteriorly, as in a pelvic abscess, but posteriorly between rectum and sacrum. As with early osteomyelitis in other sites X-ray examination is negative but direct aspiration culture of the vertebral body may prove the diagnosis. The greatest hazard of a delay in effective treatment is thrombosis of the spinal arteries. If this occurs there will be a sudden onset of irreversible paraplegia due to infarction of the cord.

TABES DORSALIS is now rare but by no means extinct. Although tabetic crises usually involve the lower limbs most acutely, paroxysms of stabbing pain in the abdomen with severe vomiting constitute the well-known gastric crises while in yet other cases the clinical picture resembles acute intestinal obstruction. Interrogation generally reveals that leg pains are present and these are always of a stabbing or "transverse" character quite unlike the shooting pains of sciatica or dissecting aneurysm. The combination of such pains with "pins and needles" in the limbs is almost pathognomonic of tabes but even in their absence the routine of examining papillary and tendon reflexes in every abdominal case will avoid needless surgery.

In HERPES ZOSTER the development of cutaneous erythema and vesiculation is often delayed for 3 or 4 days after the onset of posterior root irritation. Vesicles may develop on the visceral surfaces as well as on the skin so that haematemesis or haematuria may increase the resemblance to an intra-abdominal crisis. But the visceral vesicles will be accompanied by the typical cutaneous vesicles which, like the preceding pain, are strictly unilateral and of girdle distribution. In practice herpes more usually mimics thoracic than abdominal disease.

In the preparalytic phase of ACUTE ANTERIOR POLIOMYELITIS abdominal pain and tenderness may accompany the more usual limb pain. A physiotherapist in the Middle East developed a headache and vomited. When I saw her she was acutely ill with pain and tenderness over the right iliac fossa. Fortunately her headache continued and as this is extremely unusual in my experience in appendicitis I decided to defer operation. Twenty-four hours later leg pains appeared and on the fourth day she developed paralysis of the lower limbs and of the lower abdominal muscles on the right side.

Another classical impersonator of intra-abdominal crisis is CHRONIC LEAD POISONING. The onset of symptoms is acute with colicky pains and sometimes with reflex vomiting. Although uncommon, we have encountered two cases in the Surgical Out-Patient Department of the Royal Infirmary during the past year. Neither showed the tell-tale dropped wrist ("The Dangles") but both had the black lead-line on the teeth.

Intra-cranial disease

Acute abdominal crises may follow a variety of intra-cranial lesions. Many of these have involved the hypothalamus but severe gastric disturbances have been reported after operative procedures confined to the frontal lobes. Haematemesis, increased intestinal secretion and disordered peristalsis are a frequent complication of head injuries and often constitute a major problem in supportive therapy.

Among the PSYCHIATRIC CAUSES of abdominal crisis the bizarre symptom patterns of the psychoneurotic, embellished with a wealth of minute detail are usually self-evident but it has to be remembered that such patients may have gleaned some medical knowledge and present quite a plausible story. Acute symptoms due to the gastro-intestinal hyperactivity of an anxiety state are usually equally obvious. Psychotic patients, on the other hand, are particularly liable to certain types of abdominal disaster such as perforation of stomach or bowel by foreign bodies or intestinal obstruction due to the swallowing of un-

chewed material and their mental attitude may greatly complicate the diagnosis. Undoubtedly the most intriguing of the psychiatric patterns is that which is responsible for the so-called "Munchausen syndrome." There are a number of individuals wandering round the country from hospital to hospital, always gaining emergency admission with an apparently authentic picture of head injury, haematuria, perforation or some other such crisis. The symptoms settle immediately and after 24 hours or so they become difficult and take their own discharge only to repeat the performance elsewhere.

ENDOCRINE AND METABOLIC DISEASE

The endocrinopathies provide many pitfalls in the diagnosis of abdominal crises. By far the most important are those associated with diabetes. Apart from the additional hazards of any acute illness in a diabetic patient (and it must never be forgotten that 15 per cent of all the surgical diabetics registered in the Royal Infirmary are first diagnosed after admission to hospital for some unrelated illness) the occurrence of abdominal pain in diabetic ketosis represents the commonest problem. More rarely acute urinary infections, so common in diabetic patients may cause abdominal pain which suggests the possibility of an intra-abdominal crisis. The syndrome of the "diabetic abdomen" is never seen in stable diabetics. The pain is of gradual onset and is often preceded by vomiting associated with the ketosis. The pain and tenderness are usually diffuse and the abdomen moves freely on respiration. A marked polymorphonuclear leucocytosis may be present even in the absence of any infective process. The air-hunger of acidosis with the aroma of acetone in a dehydrated drowsy patient should lead to a suspicion of the diagnosis, but the final exclusion of primary intra-abdominal disease depends upon the resolution of the abdominal symptoms following energetic treatment of the diabetic state.

Acute adrenal failure is usually characterised by a shock-like state without pain but it is occasionally marked by the sudden onset of abdominal pain, vomiting and diarrhoea. The abdominal muscles are flaccid, like those of the limbs but there may be vagus tenderness, sometimes most marked near the adrenal glands in cases of infarction. The presence of generalised extreme asthenia coupled with pigmentation should not escape the alert doctor.

Whereas diarrhoea is common in thyrotoxicosis the muscular hypotonicity of myxoedema results in constipation which in severe cases is so complete as to produce the picture of intestinal obstruction. Indeed death from ileus used to be a common outcome in untreated myxoedema. The other features of the disease and the fact that peristalsis is diminished rather than increased should avoid diagnostic errors.

Hypercalcaemic crisis in patients with hyperparathyroidism is a somewhat exotic cause of acute abdominal symptoms. A high proportion of patients with this disease suffer from dyspeptic symptoms and constipation due to intestinal atony but if the serum calcium rises suddenly to levels above 15 or 16 mg%, severe vomiting with vague upper abdominal pain may develop. These patients may be drowsy or comatose, and death is imminent unless immediate operation is undertaken to remove the parathyroid tumour. Like urea and sugar, excess calcium in the urine acts as a diuretic and a history of polyuria and nocturia in the drowsy, vomiting patient who is not a diabetic should rouse interest in the serum calcium level. Two such patients have been admitted to the Royal Infirmary in recent years; both died—one undiagnosed until

autopsy revealed a parathyroid carcinoma with pulmonary metastases and the other despite emergency parathyroidectomy.

The vomiting and intestinal distension of uraemia may mislead the unwary and I am aware of at least two laparotomies on uraemic patients with an ill-founded diagnosis of intestinal obstruction. In the confused elderly man with hiccup and a dry tongue an emergency blood urea estimation is well worth while.

Over the past few years a number of patients, like those just mentioned, have been operated upon in the Royal Infirmary because of a clinical picture which resembled organic intestinal obstruction. I have called this condition "intestinal pseudo-obstruction." In some of these patients the abdominal disorder was secondary to disease elsewhere, as in the uraemic patients or those with intracranial disorders, but the most interesting group were patients who had suffered from haemorrhage. Three of these patients had bled severely over more than a week from a peptic ulcer; the fourth was an old woman who had cut her scalp in a fall and, living alone, had sustained considerable blood loss before help arrived.

In each the features of intestinal obstruction developed without any demonstrable organic cause. We know that intestinal contractions cease in profound hypotension and that the local hypoxia produced by distension may result in lack of contraction. Experimentally, hypoxia in animals can cause incoordinate contraction of both small and large bowel. In man the motor innervation of the blood vessels is upset by minor degrees of hypoxia and it is my belief that the motor activity of the gut may also suffer under similar circumstances.