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ACUTE LESIONS OF THE UPPER ABDOMEN*

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From the practitioner's point of view the most interesting and important aspect of this subject is that of diagnosis. We shall therefore concern ourselves almost exclusively with this side of the subject, while, for reasons of time, limiting our consideration to non-traumatic lesions.

It is always useful, when a problem of diagnosis arises in any localized area, to run over in one's mind the various normal anatomical structures present within the region, otherwise important possibilities may be easily overlooked. In the present case, for example, this method of approach will ensure that we do not forget the sympathetic ganglia, the suprarenals, and the large blood vessels. Next we consider the probable and possible lesions of the various organs which can give rise to the type of symptomatology presented by our patient. Here there is no reason to scorn the use of a list which may prevent the oversight of some of the less common conditions. A list having any pretensions at completeness would be a formidable one, and my purpose will be better served if I include only those lesions which in my experience have caused difficulty. It is probably fairly representative of the cases met with by the general surgeon, and may be taken as a guide to most of the conditions in which the question of an acute upper abdominal lesion will arise. They are grouped together under the various organs, but the list gives no indication of the relative frequency of the lesions.

1. *True Acute Lesions of the Upper Abdomen.*

Perforation of gastric ulcers, either simple or malignant.
Leakage from a gastric ulcer.
Acute gastritis.
Acute dilatation of the stomach.
Acute volvulus of the stomach.
Perforation of duodenal ulcers.
Acute duodenal ileus.
Perforation of anastomotic ulcers.
Acute cholecystitis and perforation of the gall-bladder.
Biliary colic.
Abscess of the liver, and infection of echinococcal cysts.
Acute pancreatitis.
Haemorrhage into pancreatic cyst.
Splenic infarct.
Spontaneous rupture of the spleen.
Strangulation of a paraduodenal hernia.
Obstruction of diaphragmatic hernia.
Dissecting aneurysm of the aorta or coeliac axis.
Retroperitoneal haemorrhage and infections, especially in the vicinity of the sympathetic ganglia.

2. *Conditions Arising Lower in the Abdomen, but Sometimes Resembling Upper Abdominal Lesions.*

Appendicitis with general peritonitis.
Ruptured ectopic gestation.
Primary peritonitis, usually pneumococcal.
Perforation of the colon, stercoral or malignant.
Mesenteric thrombosis.
Intestinal obstruction of the upper jejunum.

3. *Other Conditions Simulating Abdominal Lesions.*

Pneumothorax.
Pleurisy and basal pneumonia.
Angina, either a true abdominal type or by reference from angina pectoris.
Tabes, spinal tumours, and tuberculous spine in infants.
Hypoglycaemia and hyperglycaemia.
Uraemia.

Some of these lesions are, of course, so uncommon and so rarely diagnosed preoperatively that we feel no disgrace and no reflection upon diagnostic acumen if they are misdiagnosed; nevertheless, to have considered the possibility appears to be some mitigation for most of us.

Diagnostic Principles

In reaching a diagnosis, especially of acute abdominal lesions, there are two distinct methods of approach which we all apply with varying success. In the first place we usually try to arrive at a conclusion on a purely anatomical and physiological basis, and then we modify and amplify this according to our experiential knowledge and what, for want of a clear understanding of its mechanism, we call instinct. No one would be so foolish as to deny the value of experiential knowledge, and few are willing to place themselves in the hands of an inexperienced person, no matter what his academic attainment may be; yet it is the constant aim of the whole of medicine to make the subject more scientific and therefore controlled by definite laws which can be learned and applied by the novice. In the teaching of students one has this continually in mind, for it is expected of the newly qualified doctor that he shall be a competent practitioner, despite the fact that his experience must of necessity be rather scanty.

If medicine were a pure science, and diagnosis depended upon the exhibition of unalterable laws, it should be possible to teach it upon an entirely theoretical basis. We are all of us aware, however, that the practice of medicine still remains as much an art as a science. I thought that it might be interesting in considering this question of abdominal diagnosis to have the scientific aspect constantly in view, and to see how far it might be divorced from experiential knowledge.

Pain is the outstanding feature of most of the cases with which we have to deal, and so, as a preliminary, some attention must be paid to the significance of the nervous distribution to the upper abdomen. The parietal peritoneum is supplied from the somatic thoracic nerves 6, 7, 8, and 9. This supply extends posteriorly as far as the roots of the mesenteric reflections. The peritoneum of the under aspect of the dome of the diaphragm and certain other areas in the immediate vicinity is supplied by the phrenic trunk from the somatic cervical nerves 3, 4, and 5. The deep visceral supply is derived via the splanchnic nerves and the large sympathetic ganglia from the thoracic segments 5, 6, 7, 8, 9, and 10. Finally, the vagus supplies the intestine with its *accelerator* (a much

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more accurate description than *motor* fibres, and also provides special sensory fibres to the stomach mucosa.

Pain of the somatic type will arise from impulses affecting the anterior abdominal wall or the diaphragm; it is well defined and localized, and is usually associated with muscular rigidity and some hyperaesthesia. The patient has little difficulty in describing this quality of pain, for it is one with which we are all more or less familiar, or have comparable experiences, so that there are adequate terms in the language to express its character, such as burning and cutting. Splanchnic pains arise from any condition giving rise to increased tension in the muscular walls of the viscera—that is, over-distension or excessive muscular contraction. They are usually of a vague type even when severe, poorly localized, and badly defined; there is no overlying rigidity, and little or no hyperaesthesia. The patient has great difficulty in describing their character, and, for want of adequate terms in the language, he uses analogies to somatic pains and speaks of "burning" or "stabbing" pains. Obviously these are pains concerning which we have no opportunities for experiential comparison, seeing that their origins are hidden, and it is for this reason that we have no special words to describe their character (except the word colic, which is meaningless to the average patient).

Stimulation of the vagus gives rise to the special sensation of nausea. Investigation of patients under spinal anaesthesia gives the impression that no true pain impulses pass via this route.

Perforation of Gastric Ulcer

We are now in a position to take a concrete example of the application of these anatomical and physiological points. One of the commonest causes of acute abdominal pain is the perforation of a gastric or duodenal ulcer. This may be a mere leak through a small aperture which is almost immediately sealed off by omentum, or a large perforation causing a massive invasion of the abdomen. It is only the latter type which constitutes a major abdominal catastrophe. Here, a most important point, as would be expected theoretically, is the extreme suddenness of the onset; there are few other conditions in which the patient can state to the minute the exact time of the commencement of the pain. The perforation is related to increased intragastric pressure, produced either by exertion or food, so that it rarely occurs at night.

Almost immediately after the onset there may be a little vomiting; this is of no special significance from the diagnostic point of view, occurring in any condition causing intense pain; it is probably related to the vomiting associated with faintness. But for this, vomiting is usually absent, and a moment's thought will show that this would be expected. Stimulation of the sensory endings of the vagus leads not only to the sensation of nausea but also to the actual contractions of the stomach which initiate the act. The presence of irritating substances in the stomach, either taken in the food or arising from the small intestines below, as in cases of obstruction, is sufficient to start the reflex.

It must not be overlooked, however, that, even if the reflex arc is intact, vomiting cannot occur if the motor nerve endings are out of action, as they are in cases of peritoneal irritation. Added to this, the rigidity of the muscles of the abdominal wall limits their use as accessory means of emesis. We are all familiar with the absolute stillness of the intestine in peritonitis, which indicates a complete inhibition of the motor mechanism. For this reason, even although the sensory nerve endings of the vagus may be stimulated, and the patient experience nausea, the reflex will not be effective. When the stomach becomes filled with intestinal contents these will

merely overflow up the oesophagus without any force, and give the very typical regurgitant vomiting (although here the word vomiting is really a misnomer) of peritonitis. In cases of intestinal obstruction, on the other hand, there is no such inhibition of the motor mechanism, at least until the late stages, and so forcible vomiting occurs, helped by the musculature of the abdominal wall.

In a case of perforation in the early stages the motor mechanism is inhibited from peritoneal irritation, but there is also nothing in the stomach to stimulate the sensory endings of the vagus, even if the motor mechanism were not affected. Thus no vomiting occurs except in the later stages, when a peritonitis has supervened, and it is then of the regurgitant type.

The sudden pain is somatic in origin, due to irritation of the parietal peritoneum; this is an exceedingly sensitive membrane, hence the intensity of the pain. Later there is added to this a muscular pain due to the extreme contraction, which produces rigidity; this is described by the patient as a cramping pain. As with all somatic sensations, the peritoneal pain is very accurately localized in the early stages, a point of great practical importance. As extension of the exudation occurs the pain due to peritoneal irritation follows its advancing margin and tends to disappear from those regions already invaded; thus it may be found in the pelvic region, and may be mistaken for an appendicitis or a ruptured ectopic gestation if the patient is not seen in the initial stages.

This shift in the localization of the peritoneal pain is, however, largely obscured by the intensity of the muscular pain, which, of course, remains distributed over the wider area, where the muscles are rigid. A particularly useful diagnostic sign arises from the fact that the phrenic is a somatic nerve, so that, according to the general rule, pain arising from irritation of its deep branches will be referred to its superficial cutaneous distribution—that is, to a small saddle-shaped area over the acromio-clavicular region and the lower part of the neck. This is the "shoulder-tip" pain; it is almost invariably present in cases of perforation. Often the abdominal pain is so intense that there is a psychical repression of the lesser shoulder-tip pain, but that it is actually present is easily demonstrated by the administration of a spinal anaesthetic. As the anaesthetic cuts off the pain impulses travelling via the splanchnic and thoracic nerves, the shoulder-tip pain engages the patient's consciousness and he now complains bitterly of it. Of course, any source of irritation of the deep phrenic branches will produce this reference, so that its presence is not diagnostic of perforation, unless the abdominal signs agree.

Since any movement on the part of the patient will produce contraction of the already painfully cramped muscles, and pressure on the sensitive peritoneum, it is characteristic of these patients that they lie absolutely still, without movement other than that necessary for respiration. This contrasts strongly with the restlessness of the patient with colic or an intraperitoneal haemorrhage.

When the case is first seen the pulse is commonly slow; it is not easy to provide the student with an adequate explanation of this. There is, of course, as yet no toxæmia sufficient to raise the pulse centrally, but the effect in many cases seems to be more than a mere absence of acceleration, and, in the absence of any vagus stimulation, may be attributed to a sympathetic inhibition such as occurs in many cases of severe pain. For the same reason the blood pressure falls, although the compensatory mechanisms are so efficient that it is not usually very low when the patient is first seen. The intensity of both these effects varies a good deal, and is

perhaps dependent upon the patient's general make-up—that is, vagotonic or sympathetotonic. Obliteration of the liver dullness and the presence of free fluid in the peritoneum are found a little later, and are signs due to such obvious physical conditions as to need no elaboration.

Enough has been said to show that most of the symptoms and signs of perforation can be explained upon an entirely theoretical basis without any appeal to experiential knowledge. It is particularly in the differential diagnosis that we shall need the help of the latter.

Acute Pancreatitis

A condition very likely to be mistaken for a perforation is acute pancreatitis. I would emphasize that there are many types of pancreatitis, and that only the fulminating variety gives the picture described in the textbooks which resembles a perforation. The onset, although rapid, is not quite so sudden as that of a perforation, but, as in that condition, it often occurs immediately after a meal. On the whole the patient is more obese, and any previous history of indigestion is less distinct and clear-cut. The initial pain is even more severe than that of a perforation; in the latter case there is some degree of pallor, but there is a cyanosis added to this in the case of pancreatitis which makes the colour a peculiar ashen grey.

The patient is rather more restless than in the case of ulcer, and there is frequently some dyspnoea. The abdominal rigidity, however, is less intense, and for this reason tenderness is easier to elicit. The local signs, indeed, stand midway between those of a perforation and an acute cholecystitis, and one frequently hesitates between these two diagnoses without thinking of pancreatitis. Exudation behind the lesser sac irritates the left phrenic nerve, and there is well-marked shoulder-tip pain on this side in many cases. In contrast with the rather good pulse in the initial stages of a perforation, the pulse in pancreatitis is very poor and the degree of shock is very profound. This is probably due to the setting free of histamine-like bodies, but the proximity of the semilunar ganglia and the adrenals makes a deficiency of adrenergic effect a possible additional factor. At a later stage there may be some discoloration in the left loin; even in the early stages there may be tenderness in this loin such as is rarely found in a perforation.

Acute Cholecystitis

Acute cholecystitis also enters into this differential diagnosis. The gall-bladder is developed as a mid-line structure, and its splanchnic innervation reflects this origin, so that pains due to muscular tension (biliary colic) are referred right across the epigastrium, and are not of necessity limited to the right side. We have already mentioned how badly localized are splanchnic pains, but once the patient's somatic localizing sensibility has been aroused by the doctor's (or the patient's) elicitation of tenderness over the gall-bladder, there is a great tendency to "fix" the splanchnic pain to this spot. This secondary localization of splanchnic pains under the guidance of somatic tenderness is a phenomenon very frequently met with in all types of abdominal case.

When the gall-bladder is inflamed the surrounding peritoneum is irritated and a true somatic pain results. The reference in this case is over the tip of the ninth costal cartilage, but the region of the foramen of Winslow receives some fibres from the right phrenic nerve, and this gives a reference to the tip of the right shoulder. This is to be carefully distinguished from the so-called shoulder pain of biliary colic. Here the reference is to the lower part of the scapula, and the impulses travel via the splanchnic nerves. In gall-bladder lesions there may thus be both a shoulder-blade and a shoulder-tip pain.

The latter contrasts with the left shoulder-tip pain in pancreatic disease and in most cases of perforation.

As would be expected, there is a rise of temperature in cholecystitis, whereas in the other conditions the temperature is initially subnormal. The pulse, although raised, is not up in proportion to the temperature; the history of a rigor is very much in favour of a cholecystitis. While not definitely jaundiced, the patient often has a slight icteric tinge; rigidity and tenderness are much more localized than in a perforation.

Biliary colic is less likely to cause difficulty in diagnosis. It is to be noted, however, that the patient is inclined to sit up; he complains of difficulty in breathing, and feels as if his chest were held in a vice—all symptoms of angina, with which this condition may be muddled. There is no true abdominal rigidity, and only very localized tenderness. Since there is much vagal stimulation, and no interference with the motor mechanism of the stomach, vomiting is frequent and violent, but gives little relief. The pain shoots through to the shoulder-blade but not to the shoulder-tip. In some cases inflammation is present at the same time or supervenes; then the picture of a cholecystitis is added and the diagnosis becomes much more obscure.

To return to the question of acute pancreatitis, there are some who do not think the differentiation from the other lesions is of great importance, since, they argue, in any event the patient should be submitted to operation. There is, however, an increasing school which believes that expectant treatment is correct in acute pancreatitis, so that it clearly becomes necessary to attempt the differentiation. Naturally, in such difficult cases mistakes most frequently arise, and it is still true that the great majority of cases of acute pancreatitis are operated upon under a mistaken diagnosis.

In my experience an estimation of the urinary diastase has proved a very useful guide. Since a few hours are required to complete the test it is obviously of no value in the most acute cases, where the supposed perforation must be dealt with immediately. For the large group where one hesitates over the diagnosis, and for those other cases seen early, where it is decided to wait an hour or two for recovery from initial shock, the test becomes possible. The index rises from a normal of 7 or 8 units to prodigious numbers in the region of 400 to 500. Any rise above 50 can be taken as indicating a serious disturbance of the pancreas, and in the presence of acute abdominal signs indicates a pancreatitis. I have on five occasions accurately established the diagnosis in this way.

It is impossible in a short lecture to discuss the diagnosis of all the conditions given in the list, but the individual signs and symptoms occurring in some of the commoner diseases have been dealt with in detail. Many of the points mentioned can obviously be applied to the diagnosis of the other lesions.

Differential Diagnosis

A word may be added concerning the differential diagnosis between abdominal and chest conditions. The most likely thoracic lesion to be mistaken for an acute abdominal condition is a spontaneous pneumothorax. Here the onset is very sudden, and is frequently related to an attack of coughing; if this occurs immediately after a meal, perhaps as a result of smoking, the picture may be very misleading. The chest feels as if held in a vice, and there is for the time great difficulty in breathing, two signs which may be present in both biliary colic and angina. The onset of colic is never quite so sudden, nor is it related to coughing. Even when the initial severe dyspnoea of a pneumothorax has subsided the respiration rate continues to be raised, and there is a mild cyanosis not unlike that seen in pancreatitis.

Rigidity occurs, but is not so intense as that of a perforation; it is limited to the upper abdomen, and may be much more marked on one side than on the other. There is, of course, no loss of liver dullness nor evidence of free fluid, and no previous history of indigestion. The slight bulging of one side of the chest and its limited respiratory excursion are suggestive signs, and the diagnosis is established as soon as a stethoscope is applied to the chest. This will be completely confirmed by radiography.

In pneumonia and pleurisy the onset is much less sudden, so that they are not usually mistaken for a perforation. As we all know, the differential diagnosis in these conditions is more difficult in the case of children than in adults, owing to the greater frequency of abdominal reflex rigidity and tenderness in the former. I find in practice that the distinction is usually made instinctively rather than on the basis of physical signs, for these are notoriously misleading in these cases. A really high temperature, some increase in the respiration rate and disturbance of the pulse-respiration ratio, a flushed face with bright eyes, a hot skin, and respiratory expansion of the nostrils combine to paint a picture

which makes one hesitate to suggest an abdominal origin for the condition. In some cases there is a pneumococcal peritonitis as a complication, when early accurate diagnosis becomes almost impossible. There is no need to call attention to the fact that diminished air entry at the base is of little diagnostic value, for it is frequently found in cases of upper abdominal lesions due to diaphragmatic inactivity. Nor is it to be expected in the early stages of chest conditions, where mistakes are likely to arise, that the characteristic physical signs in the lungs will be well marked.

Conclusion

Despite the rapidly advancing knowledge concerning the mechanism of production of signs and symptoms, diagnosis in the case of upper abdominal lesions remains difficult. That the newer knowledge is of very real value, however, is indicated by the fact that operations of a purely exploratory nature are now much less common than they were in the past. When the average acute abdominal case comes to the operating table a reasonable anticipation of what is likely to be found has already been made.

EPIDEMIC NAUSEA AND VOMITING

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In March, 1936, there occurred in an English boarding school an epidemic which presented some novel features, and resembled the widespread epidemics recently reported in Denmark under the title of "epidemic nausea."* Its symptoms consisted either of vomiting of sudden onset or of nausea; to which was added in a quarter of the cases the sensation of giddiness, sometimes of considerable severity. The illness ran a short afebrile course, all the patients, with one exception, being fully restored to health within forty-eight hours; yet in spite of its apparent mildness the epidemic attacked within a period of fifteen days no less than one-half of the number of pupils and one-third of the adults in the school. Altogether among 117 residents there were fifty-two cases, this total including four pupils who became ill at their own homes within a week of leaving the school.

It is proposed here to describe this epidemic with special reference to the Danish outbreaks, and to discuss the problem of its possible nature.

English School Epidemic

The epidemic developed in a high-class boarding school for girls in Thanet on March 23rd, 1936, within a week of the end of term.

During the term there had been a certain number of colds but no serious epidemic or other illness, and on March 22nd the health of the school was excellent. On that day there were in residence 117 people: seventy-eight girls between the ages of 7 and 17 years, and thirty-nine adults, including resident mistresses, matrons, and eighteen maids. Visiting the school were some masters and mistresses, but no day girls. On the first day of the epidemic fourteen people (eleven girls

and three adults) were affected, of whom three experienced nausea only and eleven suffered from vomiting. Immediate inquiries were made into the question of the food supply for the day, the water supply, and drainage, but nothing of moment could be discovered. The epidemic developed as is shown in the accompanying table:

	Vomiting		Nausea Only		Total
	Girls	Adults	Girls	Adults	
March 23rd ...	10	1	1	2	14
" 24th ...	5	4	1	—	10
" 25th ...	6	1	—	—	7
" 26th ...	3	2	1	1	7
" 27th ...	3	—	—	—	3
" " ...	1 (h)	—	—	—	1
" 28th ...	3	1	2	—	6
" 29th ...	—	—	—	1	1
" 30th ...	—	—	—	—	0
" 31st ...	Term ended: school disbanded				
April 1st ...	1 (h)	—	—	—	1
" 2nd ...	1 (h)	—	—	—	1
" 6th ...	—	—	1 (h)	—	1
Totals ...	33	9	6	4	52

The table shows that four girls, marked (h), developed the disease after leaving the school. Their inclusion brings the length of the epidemic up to fifteen days, during which period fifty-two of the possible 117 people were attacked. There was no spread of the disease in the girls' homes, nor were any of the visiting masters and mistresses affected.

Symptoms.—The chief symptom was that of vomiting, which occurred in forty-two of the fifty-two cases. The sickness came on quite suddenly, so that several of the girls were sick wherever they happened to be in the school or grounds; but a great many, having gone to bed apparently well, woke up in the morning and were sick before they could get out of bed. Many of the patients were sick only once, and in no case did the vomiting go on for more than four to six hours. The onset of the vomiting is well shown in the following case.

A. R., aged 10. As subject to vomiting attacks she was removed from the school on the morning of March 25th to her home in London. On the next day at noon she was

* It appears that the Danish authors use the term "nausea" in its original meaning of "sea-sickness." It would thus include vomiting and giddiness, as well as the feeling of sickness, to which the meaning of the term is limited in the present paper.