which was stopped two days before the operation, a cup arthroplasty on the hip. At necropsy the adrenals were atrophied; there were multiple haemorrhages in the lungs and in the interventricular septum; a few petechiae were present in the brain.

Proctor and Rawson (1951) described a similar case: adrenal cortical failure occurred on completion of a 24-day course of cortisone followed by corticotrophin for 6 days. There was cytolytic destruction of the adrenal cortex, most pronounced in the fascicular zone.

A state of shock and hyperpyrexia followed by death has been reported after a minor operation on a patient receiving cortisone for rheumatoid arthritis (Lewis *et al.*, 1953) and also after a course of cortisone given to a patient with ulcerative colitis (Rachet *et al.*, 1951).

Sprague *et al.* (1950) report the death of two patients, on treatment with cortisone for rheumatoid arthritis, who after operation became shocked, comatose, and anuric. In both cases the adrenals showed atrophic changes.

Norymberski *et al.* (1953) have shown that 50 mg. of cortisone daily will completely suppress the endogenous output of hydrocortisone-like steroids, while Sprague *et al.* (1950) suggest that a dose of cortisone as low as 20 mg. a day for five days may be suppressive. These authors suggest that patients who develop extensive hypercortisonism may be more liable to persistence of impaired adrenal and pituitary function.

Our patient, who had been on cortisone for 18 months and had mild hypercortisonism, abruptly stopped taking cortisone and developed coma, hyperpyrexia, tachycardia, and hypotension, the serum electrolytes being normal apart from a slightly low serum sodium. She thus had many of the features mentioned in the cases previously described. We think it likely that she had acute adrenal insufficiency, although as she recovered we do not know the nature of the pathological lesion.

With regard to the neurological lesions our patient had a series of cerebral disturbances during the acute phase and was left with serious incapacitating sequelae similar to Parkinsonism. It is possible that she had a coincidental encephalitis of bacterial or virus origin, but the normality of the C.S.F. is against this. The widespread nature of the cerebral damage suggests multiple small vascular lesions.

Cosgriff (1951) described a series of thrombo-embolic episodes which followed the cessation of cortisone. These he ascribed to the hypercoagulability of blood which is present during and following cortisone therapy. However, only two of these were cerebral vascular accidents and both occurred in elderly patients aged 69 and 70. Neither case resembled ours.

The withdrawal of cortisone from patients suffering from rheumatoid arthritis may be followed by panmesenchymal and panangiitic reactions which, when severe, may resemble acute disseminated lupus erythematosus or polyarteritis nodosa (Slocumb, 1953). In our case there was no evidence of vascular lesions outside the brain.

In the case described by Fraser *et al.* (1952) a number of petechial haemorrhages were found in the brain. It is possible that our patient had similar lesions. However, the exact pathology of the cerebral lesions must remain in doubt.

Summary

A patient with rheumatoid arthritis was treated with cortisone for 18 months. She inadvertently took a double dose for one week and then discontinued the drug. Coma, hyperpyrexia, hypotension, and, later, dysarthria, Parkinsonian tremors, and muscular weakness ensued. The patient has only partially recovered from these disabilities and is still bedridden after 30 months. We consider that this syndrome may have been due to acute adrenal insufficiency complicated by multiple cerebral vascular lesions.

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ROVSING'S SIGN

BY

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In 1907 Professor Thorkild Rovsing, of Copenhagen, first described the sign which now bears his name. The following is a translation of his description. "The left hand is applied over the healthy colon in the left iliac fossa, the right hand applies pressure over it in an anti-peristaltic direction; because the ileo-caecal valve is competent, pain is produced in the right iliac fossa with inflammation of the appendix and caecum. Where there is muscular rigidity in the right iliac fossa and therefore accurate palpation is impossible, it will give a clue to the diagnosis—that is, it will differentiate between a lesion of the caecum and the appendix in the right iliac fossa from another lesion giving inflammation in the right iliac fossa."

This sign, with its suggested mechanism, has since found a place in the minds of countless medical students, and it receives tacit approval in surgical textbooks—for example, Aird (1949) and Bailey and Love (1956). The following note and the accompanying well-known diagram are those of Hamilton Bailey (1954): "Even pressure is exerted over the descending colon. This forces gas into the caecum. If, when the left iliac fossa is pressed, pain is appreciated in the right iliac fossa, the case is probably one of acute appendicitis."

I have been intrigued by this sign, and for this reason, with the following questions in mind, a series of observations were carried out: (1) Does Rovsing's sign occur? (2) Is the suggested mechanism correct? (3) If not, what is the explanation?

Does It Occur?

During a series of 303 consecutive cases of acute appendicitis confirmed at operation, Rovsing's sign was found to be positive on five occasions. The findings in these patients were confirmed by at least two other surgeons.

Case 1.—A woman aged 34. The right lower quadrant of the abdomen was blocked by 1/2000 amethocaine hydrochloride. After this, Rovsing's sign was no longer present, although the rebound phenomenon still remained. A grid-iron incision with gentle retraction of the wound edges revealed an acutely inflamed retro-ileal appendix; the proximal part of the appendix between the caecum and the ileum was visible, and was found to be in direct contact with the overlying parietal peritoneum, which showed signs of inflammation.

Case 2.—A man aged 37. This patient presented with a classical history of appendicitis. The right lower abdomen was blocked by local analgesia as in Case 1. Rovsing's sign

was completely abolished, although rebound tenderness still remained. A grid-iron incision revealed an appendix with patches of gangrene lying in the paracaecal gutter; the adjacent parietal peritoneum of the flank of the anterior abdominal wall was rough and red.

Case 3.—A woman aged 32. The findings in this case were almost identical with those in Case 1. The appendix was retro-ileal in position, and partially in contact with the anterior abdominal wall. Removal was carried out under local analgesia, which was found to abolish Rovsing's sign.

Rovsing's sign. Photograph reproduced from Hamilton Bailey's *Physical Signs in Clinical Surgery*, published by Wright, Bristol.

Case 4.—A man aged 30. Under general anaesthesia, through a grid-iron incision, the appendix, which was acutely inflamed and surrounded by a fibrinous reaction, was found lying in the paracaecal position. The appendix was intra-peritoneal, and there were marked signs of inflammation in the adjacent parietal peritoneum of the abdominal wall and caecum. Appendicectomy was performed.

Case 5.—A woman aged 75. As the diagnosis was in doubt the abdomen was opened through a right paramedian incision under general anaesthesia. Free purulent fluid was found, and the appendix, which was gangrenous, was seen to be lying with its tip half an inch (1.3 cm.) above the symphysis just to the right of the midline. It was attached to the parietal peritoneum with fine fibrinous adhesions. The caecal wall involving the base of the appendix was gangrenous.

During the above series of 303 appendicectomies, three patients were found to have a positive Rovsing's sign which at operation was not found to be associated with acute appendicitis. The details are as follows.

Case A.—A man aged 59. At operation through a grid-iron incision, 6 in. (15 cm.) of ileum, starting 18 in. (46 cm.) from the terminal ileum, was found to be strangulated in the retrocaecal fossa. The bowel was viable and easily reduced. There was much redness and oedema of the peritoneum in the right iliac fossa and the mesentery of the small intestine, which was in direct contact with the parietal peritoneum of the anterior abdominal wall.

Case B.—A boy aged 11. A grid-iron incision revealed a normal appendix, and the condition was found to be a well-marked inflammation of the ileo-caecal mesenteric lymph nodes.

Case C.—A man aged 22. A grid-iron incision showed on the antero-medial wall of the caecum, just above the ileo-caecal junction, a rounded hemispherical mass the base of which was 9/10 in. (2.3 cm.) in diameter and its length $\frac{1}{4}$ in. (1.9 cm.). The surface was inflamed and covered with peritoneum, and over it were running several tortuous vessels. Invagination of the lateral wall of the caecum demonstrated that it was a paracaecal diverticulum. It was removed and the caecal wall closed in layers.

From a study of these patients it is seen that Rovsing's sign in association with acute appendicitis is uncommon—only 5 cases out of 303. Yet in all eight cases where the sign was found to be positive an inflammatory lesion was present in the right iliac fossa.

Is the Suggested Mechanism Correct?

The suggestion that pressure on the colon as it lies in the left iliac fossa causes an increase of pressure in the caecum by forcing gas from the distal colon to the caecum sounds plausible, but in fact does it stand investigation?

A series of manometric experiments were carried out on a colon removed at necropsy, the recto-sigmoid junction and the terminal ileum having been ligated. A glass cannula connected to a U-shaped manometer containing water was inserted into the caecum through the appendix stump. It was found that no intracaecal rise of pressure occurred with compression of the distal colon unless there was a positive pressure in the caecum initially. This positive pressure was obtained only when the entire colon was moderately distended with air. The presence of faeces in the colon, it was noted, greatly decreased or eliminated the intracaecal rise in pressure, following pressure on the distal colon. Yet constipation is the usual finding in acute appendicitis, and a continuous column of gas in the large intestine has not been found in the two pre-operative radiographs of the abdomen in two patients who were shown to have a positive Rovsing's sign.

Viewed from the anatomical point of view, the colon is so constructed that, if inflated from the collapsed state, the haustrations between the taeniae fill out first; this is a lateral distension, and only when these haustrations are moderately filled is the air inflated available to open up a further segment of the colon. Finally, only when all the haustrations are moderately distended, does the air inflated cause a generalized rise of pressure in the colon. This state of distension must be extremely rare except in cases of obstruction of the distal colon with a competent ileo-caecal valve. In function the colon which lies in the left iliac fossa is normally contracted and empty, and on palpation can be felt to be so, except during the act of defaecation, so that it is extremely unlikely that pressure on this segment of the colon would initiate a wave of gaseous pressure along the colon to the caecum. In view of the possible invalidity of the manometric evidence of the colon outside the body, further evidence was sought radiologically.

On looking at a large number of films of the abdomen a continuous column of gas has not been seen to be present. Following barium-enema examinations and inflation of the colon with air, pressure exerted over the left iliac fossa, much greater than could be exerted in Rovsing's sign, failed to produce any change in the gaseous shadows in the caecal area.

From these observations it seems reasonably certain that the present explanation of Rovsing's sign is not correct.

What Is the Explanation?

Rovsing's sign in association with acute appendicitis is rare. It occurred in only 1.7% of the series observed (303). Has it anything to do with the anatomical position of the appendix? In Cases 1 and 3 the proximal part of the appendix was in contact with the anterior abdominal wall, in Cases 2 and 4 the appendix was in the paracaecal gutter and in contact with the anterior abdominal wall in the flank. In Case 5 almost all of the appendix from base to tip was in contact with the anterior abdominal wall. In all five cases showing a positive Rovsing's sign there was one thing in common—namely, direct contact between the parietal peritoneum of the abdominal wall and the inflamed appendix.

In the other three cases (not acute appendicitis) there was also an inflammatory lesion in the right iliac fossa in contact with the parietal peritoneum of the anterior abdominal wall.

In an analysis of the position of the appendix Wakeley and Gladstone (1928) found only 1% to occupy the pre-ileal

position, where we might expect direct contact with the abdominal wall, and a further 0.5% were described as post-ileal, in which position there is a possibility that part of the appendix might be in contact with the abdominal wall. They do not describe the paracaecal position.

What, then, is the connecting link between pressure on the left iliac fossa and an inflammatory lesion in the right iliac fossa? Is it a central reflex? It seems unlikely. So it must have a peripheral explanation. In the three cases explored under local analgesia Rovsing's sign was abolished after the analgesic was inserted, and yet the rebound phenomenon remained. The most likely explanation therefore seems to be some movement which causes a temporary friction between the inflammatory lesion and the parietal peritoneum (cf. dry pleurisy), such as a wave of vibration passing across the lower abdominal wall, or a temporary displacement of the abdominal wall or contents, such as one might expect from pressure on the left iliac fossa. Indeed, two of the patients with a positive Rovsing's sign described the pain as "shooting across to the right side," and one said, "I can feel the vibrations on my right side."

In all cases where Rovsing's sign was present, rebound tenderness was present also. When Rovsing's sign was abolished in the three patients explored under local analgesia, rebound tenderness remained. It is considered that the mechanism of pain production in the rebound phenomenon is similar to that in the Rovsing's sign but that the rebound phenomenon is more easily produced, as the displacement of the abdominal wall is more abrupt and therefore provides a more efficient stimulus.

In rebound tenderness it may well be that the peritoneum lining the right iliac fossa, as well as the peritoneum on the posterior aspect of the abdominal wall opposite the right iliac fossa, is moved and stretched. This would explain the reason why it is not abolished by an anterior field block.

Conclusions

Rovsing's sign is not common.

When positive it indicates that an inflammatory lesion in the right iliac fossa lies in direct contact with the parietal peritoneum lining the anterior abdominal wall in that region.

The probable explanation is a momentary vibration or displacement of the abdominal wall or abdominal contents initiated by pressure on the left iliac fossa and passing across to the right iliac fossa, causing friction between the inflamed viscus and the overlying anterior peritoneum with resulting pain.

It is considered that there are no grounds for Rovsing's statement that the sign is of help in differentiating between inflammation of the appendix or caecum from other lesions causing inflammation in the right iliac fossa.

When Rovsing's sign is present, and the diagnosis is thought to be acute appendicitis, that organ is probably lying in such a position that part of it at least is in direct contact with the anterior abdominal wall.

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The British Empire Leprosy Relief Association has received nearly £1,500 from the pharmacists of Great Britain in response to an appeal launched by Mr. E. A. Brocklehurst, Sheriff of Kingston-upon-Hull, when president of the Pharmaceutical Society.

Medical Memorandum

An Example of Painful Subcutaneous Tubercle

Picard's aphorism that recognition of a rarity demands only that it be borne in mind was exemplified recently in a clinical diagnosis of subungual glomangioma. Although glomangiomas were described in 1812, histological identification by Masson was delayed until 1924.

CASE REPORT

A man attended at the out-patient department complaining of a painful finger. As he put it, "there seemed no reason for the severe pain in the nail" of his right index finger. The slightest change in position of the hand caused agonizing shooting pain from the tip of the index to the hand.

On examination a bluish circular area the size of a small pea was visible beneath the hard nail. Pressure on this area produced the agony of which he complained. The colour of the area did not alter with change of posture. An x-ray film was not taken pre-operatively, but at operation a tumour on the under surface of the nail-bed was found to have caused pressure erosion of the phalanx. Histological examination verified the clinical diagnosis of glomangioma.

COMMENT

Origin and Site.—The normal glomus body is a convoluted arteriovenous communication with a characteristic perivascular cuffing of cuboidal epithelium-like cells. The narrow channel with its thick coat of cells is also known as the Sucquet-Hoyer canal. Although it is recognized that such glomera occur in the integuments of peripheral parts of limbs their function has not been explained satisfactorily. The undoubted occurrence of tumours of the glomus body in the face, trunk, muscles, and even in the stomach shows that glomera are of wider distribution than was at first supposed.

There are two types of glomangioma: (1) the vascular type with relatively few intervacular and perivascular cells; and (2) the cellular type (Masson's pauci-vascular form) where masses or sheets of large epithelium-like cells occur with relatively few blood spaces in the field. The typical glomus cell is uniform in size, is polyhedral, and contains a rounded darkly staining nucleus. The cells have distinct walls. In addition to blood spaces and glomus cells there are varying amounts of connective tissue, smooth muscle, and nerves.

The *glomus cell* is the cause of great controversy, for it is suggested that it is akin to the muscle cell in an arteriolar wall. The ordinary muscle cells of the afferent vessel to a glomus body change gradually to typical glomus cells. The *glomeric arterial segment* makes a sudden appearance and differs from the afferent vessel by the disappearance of the internal elastic lamina. Glomus cells appear where normally muscle cells would be found and, congregated there, form a broad cuff of cells.

CONCLUSION

Pain due to a glomangioma may be described as agonizingly acute in spite of there being very little to see.

The nail-bed and pulp are the commonest sites of occurrence, and because there is little room for expansion the nail-bed site is particularly painful.

Autonomic disturbances have been associated with glomangiomas, but no Horner syndrome was present in this case.

The pain of a glomus tumour is not related to its neural content.

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