There may remain cases of acute uraemia which do not belong to any of the groups referred to, but they cannot be numerous. On the other hand we may assume that the great majority of all possible cases of acute uraemia have been met with and been grouped, and in the result extrarenal uraemia has been found to account for most of the cases in question.

There is general agreement that contracted kidney is the pattern for *chronic* uraemia. It may not be an exaggeration to assert that extrarenal uraemia is, to say the least, the most frequent pattern for *acute* uraemia. As to the pathological processes typical of extrarenal uraemia, the following triad of specific features—increased endogenous breakdown of protein, disturbance in the blood supply to the kidneys and certain morphological changes in those organs—has so far been recognized. They, all or in part, form the background for the renal failure, the nature of which, however, even if recognized as functional, is not as yet clearly defined. There is, for instance, no certainty whether the chief cause of the functional disturbance is glomerular filtration or tubular reabsorption or whether it comprises both of these basic processes; nor is it certain what the peculiarities of these processes are. These questions certainly open a wide field for investigation.

It cannot be denied that the term "extrarenal uraemia" (and also "prerenal uraemia") may give rise to some misunderstanding in so far as it may be considered to infer that there exists a state of uraemia where renal insufficiency does not represent the basic process. Such a conception, of course, does not fit in with the facts. In "extrarenal" uraemia renal insufficiency does not play a smaller part than in any other condition determined as "uraemia." The term "extrarenal" refers to the primary disease, which in extrarenal uraemia is not necessarily a renal affection as in the classic form of uraemia. The eventual misunderstanding will certainly be avoided by accepting the term "renal anoxia" as proposed by Macgrath *et al.* (1945). But, even admitting that the uraemia in question may actually be caused by renal anoxia only, the two processes can hardly be identified with each other: the fact that this uraemia is caused by renal anoxia certainly does not justify the conclusion that in turn renal anoxia necessarily leads to renal failure. For comparison, the subject of cardiac pathology may be referred to. Though acute cardiac failure can be caused by coronary failure, the latter does not necessarily involve the former; therefore the term "coronary failure" cannot replace that of "cardiac failure." For the same reason the term "renal anoxia" can hardly be applied when a state of renal failure is to be expressed.

In a previous publication "extrarenal uraemia" was designated also as "functional renal failure"; but there may be arguments against this term also: first, in view of the morphological renal changes found in cases of extrarenal uraemia the functional nature of the latter might not be regarded as absolutely proved; secondly, other forms of uraemia, such as that which occurs in cases of glomerulonephritis, may be functional too. If "extrarenal uraemia" for the reason given above should no longer be regarded as wholly appropriate, the term "acute non-specific uraemia" might best fit in with all the facts.

Summary

Acute uraemia occurring in cases of chronic renal disease may be of the type of extrarenal uraemia.

Extrarenal uraemia represents the pattern for most of the cases of acute uraemia.

The term "extrarenal uraemia" may be replaced by "acute non-specific uraemia."

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FIBROSITIS

BY

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Fibrositis has been divided into primary and secondary. This is a separation with which I am in the fullest agreement; for in my opinion primary fibrositis is an imaginary disease and secondary fibrositis is a real entity.

Primary Fibrositis

Controversy has gone on for many years about the nature and identity of the different disorders included by common consent under this heading. The existence of fibrositis is affirmed by most clinicians, denied by most pathologists, but in the absence of an alternative explanation for the symptoms and signs purely negative views have carried little weight.

It is my purpose to draw attention to the ready solution to the problem of the existence or not of fibrositis that can be obtained by going back to first principles. This involves taking a detailed history, making a clinical examination of the patient, and drawing deductions on accepted lines from the physical signs discovered in each case. If this is done the conclusion is forced upon the unprejudiced observer that the symptoms so readily ascribed in the past to "rheumatic fibrositis" (i.e., fibrositis coming on for no apparent reason) are all in fact the result of articular lesions. I say "unprejudiced observer" advisedly; for I started my professional life as the very reverse, and it is only recently that I have been able to convince myself that the condition has no real existence.

Historical Note

In 1816 Balfour described fibrous thickenings occurring in the muscles in chronic rheumatism; Froriep, in Weimar, again drew attention to them in 1843. Sir William Gowers, in an article on lumbago in 1904, coined the term "fibrositis" to denote inflammatory changes in the fibrous structure of the sacrospinalis muscles. He went on to point out—perfectly correctly, as time has shown—that subsequent sciatica is "primarily an affection of the fibrous sheath of the nerve." Ever since, the disease has been recognized by medical men as a true entity. For example, Stockman (1920) shows photomicrographs of inflammatory changes detected in excised fibrous tissue from patients with fibrositis. Steinberg (1942) illustrates polymorphonuclear infiltration and proliferation of fibroblasts in a piece of the trapezius muscle excised from a woman who for 14 years had had generalized muscular stiffness ascribed by several

other physicians to a functional disorder. Another excellent photograph shows loss of cross-striation, disappearance of nuclei, and fibroblastic activity in a section of the latissimus dorsi muscle excised during the second week of recurrent lumbago.

Copeman and Ackerman (1944, 1947) carried this work a stage further; they searched for abnormalities of the trunk muscles in soldiers, whatever the cause of death. They began by postulating that fibrositic pain in the back was a well-known clinical entity and described lumbo-ilio-sacral trigger-points, myalgic spots, and tender rheumatic nodules as part of the disorder. Nothing suggesting inflammation of the soft tissues was found at post mortem; therefore pain experienced in this area was correlated with herniation of fat-lobules present in the muscles. These studies received widespread approval and were followed by a confirmatory paper by Hench (1946). He sought and excised such lumbar herniations, with complete relief from symptoms lasting 6 to 36 months in 34 out of 37 cases of back-ache. In a book completed in 1945 but not published till 1947, though I stigmatized diffuse fibrositis in the lumbar region as an imaginary disease I had not yet taken the logical step of discarding it as a possible cause of cervico-thoracic symptoms.

These authoritative opinions on a disorder whose existence has been widely affirmed for more than 40 years, backed by the demonstration of pathological changes in excised tissue, provide a formidable array of seemingly relevant fact. No one denies, of course, that these changes occur; it is their bearing on symptoms experienced in that region which is called into question in this paper. My views serve to illustrate the time-worn truth that anatomical diagnosis must precede pathological investigation. Until the tissue at fault has been singled out microscopy is out of place.

Preconceptions to be Discarded

1. *That Muscle Spasm Denotes a Disorder of Muscle.*—Spasm of muscles about some point is a secondary phenomenon designed to protect a painful structure. Common events are prevention of a joint from being moved beyond a certain range (e.g., in arthritis) or about an inflamed viscus (e.g., in appendicitis). Generalized muscle spasm, apart from congenital myotonia, is also a secondary disorder caused by fear, cold, upper-motor-neurone lesions, tetany, toxins, etc. Temporary spasm of a single group of muscles can be brought about by unaccustomed exercise—e.g., in the adductor muscles of the thigh after riding. *Thus the existence of spasm of several muscles provides a strong indication that the muscles themselves are normal.* Its detection should lead to a search outside the muscles for the cause of the spasm.

2. *That Muscles in Spasm are Tender.*—At the knee or ankle, for example, where muscles and joints do not overlap, it is obvious that the pain and tenderness lie at the joint, not in the muscles, however wasted they may be and however ready to spring into spasm to protect the joint.

3. *That Tenderness of Muscle Indicates a Muscle Lesion.*—Many muscles are normally tender at only one point in their extent—e.g., the deltoid insertion at the humerus, the extensor bellies overlying the head of the radius. Moreover, in cases of root pressure in lumbar and cervical disk lesions genuine unilateral deep tenderness of muscle is often found. It was tempting to ascribe this phenomenon to small areas of fasciculation secondary to the lower-motor-neurone lesion, but it occurs in muscles situated where the pain is felt but not supplied by the damaged root—e.g., the trapezius and levator scapulae muscles in seventh cervical root pressure. The chief assistant in the department, Dr. M. Woodhouse, has kindly carried out electromyograms on

the vertebro-scapular muscles in these cases and has established that fasciculation is absent there. Hence the hope once entertained that the cause of referred tenderness was to be found in a number of small areas of neurogenic spasm within a muscle has had to be abandoned. *It is never tenderness of a muscle, but pain elicited by the appropriate resisted movement that identifies a muscle lesion.*

4. *That Nodules or Crepitus at a Muscle are Significant.*—When excised, the nodules so often felt at the lumbo-gluteal region are found to consist of collections of lobulated fat lying superficially to the muscles. Copeman and Ackerman have shown that lobules of fat occur also in the substance of the sacrospinalis muscles. Correct interpretation of the physical signs serves to demonstrate that they are not the cause of symptoms felt in the lumbo-gluteal region. It merely so happens that pain and tender fat deposits are both common there. Yet for generations deep massage to these innocent little swellings has been given under the best auspices. The same applies to muscular crepitus, to which I used to attach importance when the idea of nodules had to be abandoned. It was puzzling to find that stringiness and coarse grating in a muscle did not alter when a patient's symptoms ceased (Cyriax, 1941). The answer is obvious now: muscular crepitus is a perfectly normal phenomenon, felt most easily at the cervico-thoracic extent of the erector spinae muscle.

5. *That Limitation of Movement in More than One Direction can Result from a Muscle Lesion Alone.*—Myotendinous lesions are usually associated with a full range of movement at the affected joint—e.g., tennis-elbow, supraspinatus tendinitis. A muscle lesion may, however, limit movement in one direction—e.g., adherence of the quadriceps muscle to the shaft of the femur limits the range of flexion at the knee-joint; dorsiflexion of the foot is limited for a few days after a minor rupture in the belly of the gastrocnemius muscle. Though the movement that stretches the damaged muscle may be limited, all the other movements of which the joint is capable remain of full range.

6. *That Examination of Conduction Along a Nerve Suffices.*—When pressure is applied to a nerve, pain results at first from interference with its sheath. If this continues, signs of involvement of the parenchyma may in due course appear, but this is by no means certain (Cyriax, 1942). Hence, when pressure is exerted on a nerve by, for example, a prolapsed intervertebral disk or a cervical rib many cases will remain unrecognized if the examiner awaits the appearance of clear evidence of parenchymatous involvement. At present dural and nerve-sheath lesions unaccompanied by evidence of parenchymatous change are usually labelled fibrositis and provide a large number of diagnostic errors.

Clinical Examination

In suspected fibrositis, as in any other condition, the physical signs must be sought and properly interpreted. Deductions can also be drawn on the nature of the disorder from the signs that in due course appear in patients who get worse.

The following uncontroversial principles should be borne in mind: (1) Passive movements indicate only the state of the joint. Limitation of movement in more than one direction indicates an articular disorder. (2) Resisted movements indicate only the state of the muscles. Pain felt when the muscle contracts, but no weakness, implies a minor myotendinous lesion; weakness without pain implies complete rupture or nervous disease. (3) A painful arc, or pain felt only on the relaxed side when a joint is moved passively, indicates that the lesion is pinched where it lies between two bony surfaces.

Fibrositis at the Neck and Trunk

Let us now consider the physical signs and the subsequent history in patients hitherto regarded as suffering from rheumatic fibrositis.

Acute Rheumatic Torticollis.—The patient often wakes up and finds himself prevented by severe lower cervical pain, more marked on one side than the other, from moving his neck at all. Examination shows: perhaps lateral deviation of the neck; limitation of movement in all directions at the cervical joints, more marked in some than in other directions; and pronounced muscle spasm. These signs clearly indicate an articular lesion. In fact, the condition is due to any attack of internal derangement—lumbago in the neck, as it were—and the signs are of the same order. If spontaneous reduction does not soon occur the pain usually shifts to one side only of the base of the neck. The pain is now felt solely in the scapular area; yet what possible lesion of the muscles hereabouts (trapezius, levator scapulae, rhomboid, latissimus dorsi, spinati, serratus anterior) could conceivably prevent nearly all active and passive movement at the neck while leaving the voluntary scapular and arm movements free and painless?

Vertebro-scapular Fibrositis.—The patient may start with bilateral pain that shifts to one scapular area, or the symptoms may come on gradually and be unilateral from the first. Examination usually shows a full range of movement at the neck, the extremes of some movements hurting, of others not. The resisted movements of the neck and scapula are painless. Can one imagine any unilateral lesion of the paravertebral neck muscles or of the vertebro-scapular muscles, however nodular or crepitant they may appear to be (before, during, and after!), that leads to pain felt at the extremes of range at the cervical joints but to no discomfort when the resisted neck and scapular movements are tested? Spontaneous reduction may take place or the condition may become stationary. If the displacement increases, the pain begins to radiate down the upper limb; tingling may be felt in the fingers.

Brachial Neuritis.—In most cases the pain is severe, worse at night, and uninfluenced by use or rest of the painful limb. Examination shows that one or other of the neck movements increases the pain in the arm; another perhaps eases it; limitation of movement in one or two directions may be noted. If, as is usual, the sixth cervical joint is affected, weakness of the triceps muscle eventually becomes apparent in most cases; occasionally the power of flexion at the wrist is slightly reduced. Sluggishness of the triceps-jerk is a rarity. A small patch of cutaneous analgesia may be detected at the dorsum of the index or long finger; often the patient complains of pins-and-needles and numbness with nothing objective to show for them. The analogy with sciatica has become complete, and it is now generally agreed that brachial neuritis of the type described above is the result of a cervical disk lesion. Do not the precursors, described under the two previous headings, form part of the same pathological process, but lesser in degree?

Pleurodynia.—The pain is felt in the thorax, posteriorly at first but usually spreading anteriorly if it becomes more severe; sometimes the symptoms are purely abdominal or anterior thoracic. It is difficult to prove that all these cases are caused by thoracic disk lesions, since the physical signs are in some cases equivocal. However, examination may show that trunk side-flexion towards the painful side causes pain. This indicates either that active contraction of the muscle hurts or that the lesion is squeezed. When, as is usual, resisted side-flexion proves painless the muscle is exculpated. This means that the lesion lies intra-articularly and is pinched between two vertebral bodies. The same reasoning holds good when pain on active but not on resisted trunk extension is discovered. Cutaneous analgesia at an intercostal space is seldom discernible. Further proof that unilateral thoracic pain of the sort labelled fibrositis arises from a disk lesion may be obtained on attempted manipulative reduction; cases are met with in which this results in a click, whereupon suddenly the pain is wholly transferred to the other side of the chest. This must at least indicate that the lesion lies loose within a cavity situated at the middle of the body, and only one such exists—the intervertebral joint.

Acute Lumbar Fibrositis (Lumbago).—I regard lumbago as caused by internal derangement at a low lumbar intervertebral

joint (Cyriax, 1945). The patient bends forwards and is suddenly immobilized in the flexed position by severe pain in the lower back, usually bilateral. Examination shows muscle spasm to limit lumbar movements. If this event was described as occurring at the knee-joint, even if it was found that spasm of the hamstring muscles was limiting extension at the knee, no one would hesitate to ascribe the accident to internal derangement. Were lumbago really caused by acute fibrositis of the sacrospinalis muscles, with spasm, opisthotonos would result; in fact, the typical posture involves some degree of trunk flexion. Examination of a patient with acute lumbago shows marked limitation of movement in each direction at first; later the range of flexion and side-flexion gradually returns, only trunk extension remaining impossible. Limitation in each direction indicates, as always, an articular lesion; the sudden onset and the persistence of limitation of trunk extension only show that internal derangement took place. Posterior displacement of part of the cartilaginous disk provides a block at the back of the joint preventing full approximation of the posterior articular margins—i.e., trunk extension. Once a patient has had lumbago he is subject to recurrences, as would be expected in loose-body formation within a joint.

A few patients with lumbago display the lumbar signs of sciatica (lumbar deviation, generally away from the painful side, and limitation of trunk flexion) and a few patients with sciatica have the signs of lumbago, but this is not surprising when one remembers that they are essentially the same disorder.

Chronic Lumbar Fibrositis (Backache).—The patient may feel his back give way or click during, say, heavy lifting, and thereafter he finds himself subject to backache. Or he may develop pain more gradually, for no obvious reason or even after a period of rest in bed; recumbency in flexion is the common cause of the backache coming on during the puerperium. The pain is usually central, becoming unilateral later; the reverse sequence also occurs.

In the early stages examination of the patient standing often reveals the existence of a painful arc, especially on coming up from trunk flexion. This means that trunk flexion has allowed a slight degree of backward movement of the fragment of disk to take place while the joint gapes posteriorly. At the moment when the joint surfaces reverse their inclination as lordosis is regained the disk is sharply squeezed. Alternatively, pain may be felt on the side towards which the trunk is bent, associated with absence of pain when the same movement is tested against resistance; this phenomenon also implies that the lesion is so placed that it can be squeezed between the vertebral bodies. I used to think that painful pinching could occur between the lumbar transverse processes on side-flexion of the trunk towards the painful side, but this attractive idea has had to be abandoned. In other cases the signs are less distinctive, but it is my experience that, judged by the effect of manipulative reduction and of epidural local analgesia and by the later development of sciatica, there is no difference between cases showing clear signs of a disk lesion from the first and those with equivocal signs.

Gluteal and Sacro-iliac Fibrositis.—The history may be of central lumbar pain, perhaps recurrent, which later settles in one buttock. Sometimes the pain starts in the buttock without any premonitory backache. Examination often shows that full trunk extension reproduces the pain in the buttock. Since the lumbar muscles and every structure at the sacrum or the buttock, apart from the joints themselves, are relaxed by this movement, it follows that gluteal pain reproduced thus arises in the lumbar, sacro-iliac, or hip joints, not locally. If clinical examination then shows that the hip and sacro-iliac joints are normal and that resisted contraction of each of the buttock muscles tested in turn is painless, the lesion must lie at a lumbar joint. Epidural local analgesia quickly confirms the fact that the symptoms arise from pressure exerted at the side of the dura mater. Moreover, most patients with this symptom are on the brink of sciatica; hence the mere passage of time often makes the diagnosis obvious.

Sciatica and Anterior Crural Neuritis.—When neurological signs are present it is generally agreed that disk protrusion is responsible. But it must be remembered that pressure exerted on a nerve root from without may not be sufficient in degree to affect conduction. Thus the tests for the sheath of the nerve

must also be given due weight. Hence the arrival at a diagnosis in cases of diffuse pain felt in the lower limb involves clinical examination of the lumbar spinal joints, the limb itself, the sheath of the nerve roots, and the parenchyma of the nerve roots. If patients with sciatica due to a disk lesion proved at laminectomy are questioned about the past they nearly all describe attacks of backache or lumbago preceding for many years the onset of sciatica. Has lumbar fibrositis then changed to a disk lesion, or was it a disk lesion from the first?

Treatment of Primary Fibrositis

The underlying principle is simple: to secure reduction of the intra-articular displacement causing the symptoms. The actual techniques of reduction have already been described and illustrated (Cyriax, 1947).

Neck.—This is usually easy, whether the patient has pain in the neck, the scapular area, or the upper limb: a few sessions of manipulation seldom fail to secure reduction. Occasionally continuous traction is indicated. Operation is required in 1% of all cases. Evidence of pressure on the spinal cord contraindicates manipulation.

Thorax.—In simple cases one manipulation may result in full reduction; but eventual relapse is common. The difficult cases are very difficult, and it is easy to make the patient worse. If attempted manipulative reduction—even during traction—fails, rest in bed is indicated.

Backache.—Recovery follows a few sessions of attempted manipulative reduction in about half of all cases. Rest in bed relieves some others but may lead to aggravation. Epidural local analgesia has a lasting effect on some of the remainder. Once well, the patient must be shown how to avoid further attacks of internal derangement (Cyriax, 1945). The passage of time, particularly in young patients, may bring relief if the protrusion erodes the body of the vertebra.

Lumbago.—Two-thirds of all cases are considerably, one-third fully, relieved by one manipulation. Those whom manipulation does not affect should receive an epidural injection at once. Rest in bed ensures recovery in the end; but this consumes much more time and can often be avoided if these measures are tried first.

Sciatica.—Manipulation is particularly apt to be effective in the elderly; in patients under the age of 50 it is likely to succeed in only one case in four. It is always worth trying, however, if the neurological signs are inconspicuous. Epidural local analgesia effects lasting improvement in others. Rest in bed eventually brings about spontaneous reduction in most cases. If sustained pressure results in atrophy of the affected nerve root the symptoms slowly disappear, though the signs of parenchymatous involvement increase. Operation is required in about one case in eight.

Secondary Fibrositis

There is no important controversy about the existence of four categories of this disorder: traumatic, rheumatoid, infectious, and parasitic.

Traumatic Fibrositis.—This results from overuse or a single strain. Perhaps the best example is a tennis-elbow. A minor rupture occurs at the origin of the common extensor tendon from the lateral humeral epicondyle. Very little aching is set up at first, but, as the result of the torn edges beginning to join and then being pulled apart again each time the muscle is used, excess scar-tissue is laid down in the healing breach. Within one to three weeks the elbow has become quite painful from the development of chronic traumatic fibrositis at the site of the lesion.

Scarring in an intercostal or in the gastrocnemius muscle. golfer's elbow, teno-periosteal lesions at the wrist, crepitating teno-synovitis caused by overuse, abnormal scarring binding down a ligament after a sprain, capsular adhesions

at the shoulder after injury—all these and a number of other similar conditions can well be regarded as caused by post-traumatic fibrositis—more exactly, fibrosis. Ischaemic contracture, since the fibrosis affects the whole of the muscle equally, does not cause chronic pain. Treatment consists of deep massage, manipulation, or local analgesia, depending on the lesion present (Cyriax, 1947).

Rheumatoid Fibrositis.—A number of observers (Curtis and Pollard, 1940; Freund *et al.*, 1942, 1946; Gibson, Kersley, and Desmarais, 1946) have obtained clear evidence from microscopy of tissues excised from patients with rheumatoid arthritis that nodular perineuritis and polymyositis complicate this disease. This is in full accord with clinical findings, which show that, in addition to the joint lesions, the tendon sheaths thicken, the tendons become rough and nodular (particularly in the palm), and bursae swell and fill with fluid. "The inference may be drawn that rheumatoid arthritis is a generalized affection of the fibrous tissues of the body in which the chief and most obvious incidence is on the capsule of the joints." (Cyriax, 1947).

Infectious Fibrositis.—Epidemic myalgia (Bornholm disease) is an infectious disease the virus of which has been identified. It is characterized by fever, severe pain in the abdominal and thoracic muscles, and speedy recovery.

Parasitic Fibrositis.—Infestation with *Trichina spiralis* sets up fever and painful swelling of the affected muscles, the skin over which may become red. The tendons may also be invaded. The disease comes on some ten days after eating infected pork. Active contraction of the affected muscle increases the pain. The symptoms and signs subside in the course of some weeks, after which the patient becomes completely unaware of the foreign bodies in his muscles.

Generalized Fibrositis

Rheumatoid arthritis is the only condition to which the term "generalized fibrositis" properly applies. By contrast, the disorder to which this name is often given is osteoarthritis of the spinal joints. This may lead to considerable aching over part or the whole of the trunk—areas where it so happens that muscular crepitus and fatty nodules are commonly detectable. Unrecognized osteitis deformans or spondylitis deformans is repeatedly called fibrositis.

Another disorder often called "generalized fibrositis" is psychoneurotic pain. The idea of generalized fibrositis has led to such concepts as "non-articular rheumatism" and "the psychological basis of rheumatism"—notions in which the cart is put before the horse. Clearly, functional pain is not rheumatism, and the discovery of the real cause should lead to revision of that ascription, not to an attempt to fuse two incompatible diagnoses.

Osteopathy

It is not infrequent that cases of pain felt at the trunk are mistakenly ascribed to various internal disorders but are in fact set up by root pressure from lumbo-thoracic-cervical disk lesions. Subsequent events in such cases, if the true diagnosis is missed, may appear to corroborate osteopaths' unfounded claim that their spinal treatments cure visceral disease. In a number of cases of obscure pain a disk lesion has proved responsible for the symptoms, and manipulative reduction has been successfully undertaken.

Summary

Primary fibrositis, both local and generalized, is an imaginary disease. The symptoms erroneously ascribed to this condition are all the result of articular disorders (largely internal derangement) at the spinal joints.

Secondary fibrositis (traumatic, rheumatoid, infectious, and parasitic) is a real entity.

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Medical Memoranda

Two Cases of Volvulus of the Caecum

In the *British Medical Journal* of Jan. 18, 1947 (p. 83), Mr. Ralph H. Gardiner reported three cases of volvulus of the caecum. He pointed out the embryological basis for the non-fixation of the caecum and right half of the colon which occurs in some cases as a failure of its embryological mesentery to disappear. This mesentery acts as a hub around which torsion occurs. He also pointed out that the onset of volvulus of the caecum may be sudden, the patient suffering from violent abdominal pain, vomiting, constipation, and abdominal distension—signs of acute intestinal obstruction. On the other hand the onset may be slow and insidious, with pain in the right iliac fossa, tenderness, and varying degrees of gaseous distension. The case may simulate an attack of appendicitis.

The following is a report of two cases with a mobile caecum and ascending colon due to the persistence of the embryological mesentery, around which torsion of the caecum had occurred.

CASE 1

On Oct. 7, 1946, at 10 a.m., the patient, aged 60, was taken ill with a sudden attack of pain of a continuous nature, aching in character and "like frightful indigestion," and a feeling of fullness. She felt nauseous but was unable to be sick. The pain became so severe that she rolled in agony on the floor. She had trouble in passing flatus also, and she heard her "stomach roll." Next evening she kept complaining of great thirst, and had a whisky-and-soda, which she returned. She was given morphine without effect and was then admitted to hospital. Her bowels had been opened the day before admission. An enema was without result and she did not pass any flatus.

Her past history showed that thirty years ago she had suffered from constipation and a persistent high temperature. She was seen by the late Lord Moynihan, who treated her with paraffin, 4 oz. daily for six months. At the end of this time she underwent a laparotomy and "decayed intestine from birth" was excised.

On examination the patient appeared to be in great agony. She was very dehydrated; her tongue was dry and furred and the abdomen distended. Intravenous drip was set up and a right paramedian incision was made. Haemorrhagic free fluid was present in the abdomen, and an adhesion extended from the lateral wall over the ascending colon and caecum to the line of anastomosis of the old resection of ileum mentioned above. The caecum had twisted round the axis of the adhesion and the ascending colon. There was a hiatus in the mesentery into which the small bowel had looped, causing further obstruction. The ascending colon was very much narrowed. The adhesion was divided, and the caecum, which was folded anti-clockwise, was unfolded clockwise, establishing free communication with the ascending colon. The proximal end of the adhesion (the adhesion adherent to the mesentery) was stitched to the region of the omentum, thus straightening the axis. No attempt was made to obliterate the hiatus.

The patient made an uneventful recovery and is to-day feeling better than she has felt for the past thirty-five years.

CASE 2

This patient was first admitted on July 16, 1941, when he was 46 years old. He had a sudden attack of colicky pain in the early morning, and more frequent and intense attacks after admission. His bowels had been opened the day before admission.

Examination revealed visible peristalsis, but nothing abnormal could be palpated abdominally. Examination per rectum was negative. Laparotomy disclosed volvulus of the caecum, which was mobile and grossly distended. Aspiration was performed; the volvulus was untwisted and appendicectomy carried out at the same time. The patient was subsequently discharged in a satisfactory condition.

On Oct. 29, 1945, he was readmitted complaining of several attacks of abdominal pain and distension. An attack just prior to admission was very severe and he had had no motions or flatus for several days. A Ryle's tube was passed, with aspiration, and the patient was given a soapy enema followed by one of turpentine. He appeared to have settled down during his stay in hospital, and a radiograph on Nov. 5 was reported on as follows: "Barium enema has revealed a rather unusual appearance in connexion with the large bowel, which appears to be displaced towards the left; there is no definite evidence of any obstructive lesion. Some irregularity in the region of the caecum suggests the possibility of adhesions; no definite evidence of any malignant infiltration has been discovered." The patient's general condition cleared up and he was discharged.

He was again readmitted on Feb. 10, 1947, with a history of abdominal pain "at the bottom of the stomach" that was colicky in nature. It had become continuous during the last hour, but was not as severe as when of a colicky form. He had vomited five times since the onset of the pain. His bowels had been opened on the day before admission and on the day when admitted. There was no associated loss of weight. On examination the patient's tongue was clean. He pointed to the mid-hypogastrium as the site of the pain. The abdomen moved on respiration and he complained of tenderness just lateral to the scar of the right lower paramedian incision. There was a tympanitic note on percussion of the abdomen, peristalsis was heard, and the abdomen was soft with voluntary guarding. The patient moved about without undue stress or strain. Aspiration was carried out and clear fluid obtained. A flatus enema was given with a fair result.

Several symptoms which had cleared up during the first day or two in hospital recurred, and when he began to vomit laparotomy was carried out under spinal analgesia. At operation the previous scar was excised. On opening the abdominal cavity the caecum was found to be lying across the lower abdomen and left iliac fossa. It had rotated once, clockwise. On undoing the caecum a strong adhesion was found at its upper end, and this made the ascending colon adherent to it. There was narrowing of the ascending colon at this point. The ileum was also adherent to the lower part of the caecum. Other adhesions were present; these were separated. The caecum was replaced in the right iliac fossa and stitched to the lateral abdominal wall. An adhesion from the descending colon to the above mass of adhesions was also found. There was much gaseous distension in the caecum.

The patient made an uneventful recovery and remained well until July 31, 1947, when he was readmitted with bilateral bronchopneumonia and abdominal distension, from which he died.

COMMENT

As pointed out by Mr. Gardiner, volvulus of the caecum occurs more often than has been reported, and should be kept in mind in arriving at a diagnosis in cases of intestinal obstruction. As can be seen from the two cases, the cause of the volvulus was failure of the caecum and ascending colon to become attached to the posterior abdominal wall—in other words, caeco-colic mesentery. The onset of symptoms, as previously mentioned, were those of acute obstruction—namely, sudden onset, violent abdominal pain, vomiting, and constipation. The onset can also be chronic intestinal obstruction, as mentioned in the review of the history of the second case.

I wish to thank Mr. W. M. Morrison and Mr. I. G. L. Ford, honorary surgeons, for permission to publish these cases, and Mr. Morrison also for guidance.

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New regulations entitled the Clay Works (Welfare) Special Regulations, 1948 (No. 1547), which come into operation on Oct. 1, supersede the Clay Works (Welfare) Order, 1932. The regulations extend the provisions of the order with regard to washing facilities, clothing accommodation, and welfare in factories in which clay, shale, sand, lime, or similar materials are made into bricks, tiles, blocks, slabs, pipes, stilts and spurs, nozzles, or similar articles. They also specify that an ambulance room should be equipped with a glazed sink with hot and cold water always available while people are at work, a table with a smooth top, means of sterilizing instruments, a supply of suitable dressings, a couch, a stretcher, and a foot-bath.