especially if signs of a focal lesion exist (e.g. hemiparesis or hemianopia). The difficulty is increased by the facts that (i) in cerebral arteriopathy some increase in the protein content (up to 0.08 per cent. or more) of the cerebrospinal fluid is often found and (ii) a cerebral tumour may coexist with arteriosclerosis. Although an optic neuritis may occur in the latter, changes in the disc itself are usually absent. Retinal arteriosclerosis is nearly always present, the state of the retinal vessels being the best guide to the condition of the cerebral arteries. The presence of a high blood pressure favours arteriosclerosis; on the other hand, in the presence of extensive atheroma of the cerebral vessels the blood pressure may often be quite low. A history of transient palsies or of a sudden onset is more indicative of arteriopathy. In some cases it is necessary to keep the patient under observation for some time in order to exclude cerebral tumour.

(6) *Cerebral abscess*. The diagnosis is made mainly on the presence or history of discharge from the ear, sinusitis or other source of infection. The commonest sites are the temporo-sphenoidal lobe and the cerebellum. Papilloedema if present at all is usually slight. The cerebrospinal fluid may show a slight increase in the number of lymphocytes—even a few polymorphonuclear cells—as well as in the protein content. The presence of polymorphs in the cerebrospinal fluid does not necessarily indicate abscess as they may occur in association with some gliomas (e.g. glioblastoma). Also, a leucocytosis in the blood

may occasionally be found in cerebral tumour as well as in abscess. In some cases a definite differential diagnosis can only be made by exploration.

Treatment

In general, if the tumour can be localized, operation should be performed with a view to its possible removal. If extirpation is not practicable, as much as possible of the tumour can be removed and the remainder subjected to diathermy. Subsequently deep X-ray treatment can be applied. In deep-seated tumours, radon seeds have been placed around the tumour with occasional benefit. If the tumour cannot be localized and papilloedema is approaching 4 dioptries, decompression should be carried out, either a right-sided subtemporal decompression or one over the suspected site of the tumour.

In cases of pituitary tumour it is a safe rule not to operate for purely endocrine symptoms.

If operation is impracticable or undesirable for any reason the only resort is deep X-ray treatment.

As a temporary measure for reducing increased intracranial tension, the intravenous injection of 50 to 100 cc. of a 50 per cent. solution of sucrose is often successful and has superseded the hypertonic saline (15 to 30 per cent.) formerly used. A more slowly acting method of reducing intracranial tension is by the rectal administration of a 25 per cent. solution of magnesium sulphate, according to the quantity the patient will retain, up to a total of 250 cc.

RECURRENT UMBILICAL PAIN IN CHILDHOOD

With special reference to non-specific mesenteric adenitis

By JOHN APLEY, M.D., M.R.C.P.

Department of Child Health, Bristol University

Partisanship incurs both rewards and penalties. Of its rewards, the most obvious is increased opportunity for exploitation; of its penalties, the outstanding one is the hindrance of progress in other directions. In many branches of medicine the results of over-emphasis have proved in this way detrimental, and among them is the subject under consideration. The wide variety of causes to which 'umbilical colic' has been attributed, will be illustrated during this discussion. They range between such extremes as 'neuropathic constitution' and 'chronic constipation,' accord-

ing to the bias of the observer due to variations both in opportunity and in the method of approach.

The main purpose of this paper is to survey the existing extensive contributions in the light of information derived from cases investigated personally.

Causes of chronic or recurrent abdominal pain in childhood

As a first step it will be helpful to set out a comprehensive list of possible causes of pain which may be localized to the umbilical region. It is

the more important to do so because umbilical and other abdominal pains are often loosely grouped together. The reasons are many: the child may be incapable of giving an accurate account, or its parents may superimpose their own ideas, or the history-taking may not be sufficiently meticulous.

No scheme of classification can be perfect, but the semi-anatomical one given here is preferred as that most helpful in diagnosis. For comprehensiveness it includes many rare causes.

TABLE I

POSSIBLE CAUSES OF CHRONIC OR RECURRENT ABDOMINAL PAIN IN CHILDHOOD.

I. EXTRA-ABDOMINAL

1. *Psychological.*
2. *Metabolic.* Cyclical vomiting, diabetes mellitus, coeliac disease, menarche.
3. *Thoracic.* Pleurisy, pericarditis. (These are more frequently acute than chronic or recurrent.)
4. *Abdominal wall.*
 - (i) Skeletal: Postural defects, spinal caries or tumour, hip disease.
 - (ii) Muscular: Hernia of linea alba, 'fibrositis.'

II. INTRA-ABDOMINAL

1. *Peritoneum and mesentery.*
Tuberculosis, adenitis (whatever the pathogenesis), pancreatitis, bands, tumours.
2. *Stomach.*
(The child's stomach is rarely the seat of organic disease, but most dietetic causes are included here for convenience.)
Defective mastication (grossly defective teeth, or bad eating habits), excessive food, dietary indiscretions (e.g. 'green-apple-colic').
3. *Small intestine.*
Duodenal ulcer, Meckel's diverticulum, chronic intussusception, the Henoch-Schönlein syndrome, regional ileitis.
4. *Large intestine*
Chronic constipation, Hirschsprung's disease, chronic appendicitis, diverticulitis, chronic colitis, neoplasms, lead poisoning, tuberculosis, infestation with worms.
5. *Other abdominal organs*
Kidneys and uro-genital tracts (calculi, recurrent pyelitis or nephritis, intermittent hydronephrosis, urethral or ureteral obstruction), spleen (recurrent splenitis), liver (recurrent engorgement or hepatitis), biliary colic, undescended testis.

Umbilical Colic

In the nineteenth century cases presenting 'umbilical colic' were grouped rather loosely among the dyspepsias, and the symptoms were commonly attributed to dietary defects. Constipation and infestation with worms were also

invoked as common causative agents, a tendency which persists even today in some modern textbooks. Later, in the heyday of intestinal surgery, such diagnoses as partial intestinal obstruction due to bands or adhesions, and chronic appendicitis came to the fore. In the first quarter of this century the diagnosis of abdominal tuberculosis became fashionable. Psychologists introduced the conception of the 'neuropathic constitution' as an explanation for umbilical colic, and with this diagnosis such conditions as 'spastic intestine,' 'chronic colitis' and 'defective muscle tone,' were, and still are, frequently linked. In the 1920's attention became focused on infection outside the alimentary system; for example, umbilical colic has been attributed to low-grade liver infection.

It is nowadays considered that upper respiratory infection is the common causative agent, mediated through non-specific mesenteric lymphadenitis. The evidence for and against this conception will be considered.

Clinical Features

Many clear descriptions of the common type of umbilical colic, in which no obvious cause is to be found, have been recorded. That of Short (1928) may be quoted: 'Sudden centralized pain, severe enough to make the child cry, lasting 15 minutes or less, relieved by pressure or hot applications, recurring perhaps two or three times a day, and stopping as suddenly as it began, so that in the intervals the child is quite free.'

This description may be amplified. The pain may be severe or mild. It is often exclusively periumbilical, but may, in addition, be located in the right iliac fossa. During an attack the temperature may be slightly raised, the tongue is clean or moderately coated, there is no abdominal wall rigidity, and abdominal tenderness is absent or vaguely localized to the umbilical area and/or right iliac fossa. Relief is sometimes afforded by external applications, but rarely if ever by internal medication. The bouts of pain tend to be brief, but pain occasionally persists in a mild form between the acute exacerbations. In the intervals between attacks some children have a poor appetite, tire easily, and show other signs of subnormal health. These vague manifestations tend, in my own cases, to be lost after the initial stages, as also the mild pyrexia and the bouts of nausea, or vomiting, which may occur.

Critical Discussion

(1) *Pain.* The study of pain is beset with difficulties. So long as it depended only on the interpretation of clinical data results were limited, but with the transfer of physiological methods

from animals to man, and with improvements in surgery increased precision has become possible. (Morley, 1931; Kinsella, 1940; Ray and Neil, 1947.)

Certain facts have been demonstrated. The absence of muscular rigidity and superficial tenderness suggests that the typical case of umbilical colic is concerned with visceral and not somatic pain. Disturbances of the digestive tract, embryologically a mid-line structure, may be referred as pain in the anterior mid-line of the body surface within roughly defined anatomical limits (Fig. 1). The mesentery as well as the intestine appears to be sensitive to certain stimuli; they may be referred as pain localized to areas that correspond to the part of the intestine to which the mesentery is attached.

Site of pain. On the basis of these statements the anatomical classification of causes of abdominal pain proves of use. As possible causes of umbilical pain certain conditions can clearly be eliminated, such as extra-abdominal diseases (e.g. pleurisy and diseases of the hip). Of the intra-abdominal causes, disorders of viscera such as the spleen, liver and gall-bladder can also be excluded if the history is accurate. In young children an accurate history may not be obtainable, but umbilical colic is believed to occur, as a rule, in older children who are quite capable of localizing the pain if both they and their parents are questioned with care and sympathy.

With accurate localization of the pain, diseases confined to the large gut can also be excluded as causes of umbilical pain. In such cases as Hirschsprung's disease, with recurrent abdominal pain, it is found that the pain is not predominantly periumbilical.

Disease of the urogenital system should also be excluded if the history is clear, but certain qualifications must be made. Such conditions as intermittent hydronephrosis can usually be separated with little difficulty; recurrent pyelitis, however, may give rise to doubt. It may be because the kidneys possess a low-grade visceral sensitivity to pain, or because, owing to the relative paucity of perinephric fat in early life, the kidneys are intimately related to the peritoneum; but vaguely localized or central abdominal pain is, in my own experience, not infrequently associated with pyelitis. Other disturbances in the urogenital system may also cause abdominal pain, and must be similarly considered.

Character of pain. The pain is commonly described as colicky, though a constant, dull ache may in some cases persist between the bouts. Colic is usually attributed to disordered peristalsis, but with our present knowledge this does not sufficiently account for all instances. Several relevant

examples may be cited: there is the pain associated with an incompletely descended testicle; that associated with a small hernia of the linea alba; and the intermittent colicky pain due to tuberculous mesenteric adenitis. The last example is particularly relevant to the present discussion. The occurrence of colicky pain does not, therefore, necessarily localize the underlying lesion to hollow viscera such as the intestine, and additional evidence is required before precise localization becomes possible.

(2) *Additional localizing evidence.* One of the characteristic associations of disordered intestinal peristalsis, especially in childhood, is *diarrhoea*. It is significant that diarrhoea is a rarity in cases with umbilical colic.

When it is considered also, as is clearly shown in my own case records, that gross dietary indiscretions are rarely concerned, that there is no correlation between eating and the occurrence of pain, that anti-spasmodic drugs afford no relief, and that radiological evidence of disordered peristalsis is absent, it becomes tempting to suppose that the cause of the pain is likely to be outside the intestinal wall.

(3) *Evidence of infection.* In many instances moderate *pyrexia* occurs, even between bouts of pain. This is true even in those cases where infection outside the alimentary system is not found. In my own cases it appears that pyrexia is less likely to occur once the disease has passed the initial stages.

Changes in the *blood* are equivocal and unhelpful, though some observers (Fitzsimons, 1946; Postlewait, 1942) have described a slight relative lymphocytosis.

The incidence of *concomitant infection* is difficult to assess. Postlewait (1942) places it as high as 77 per cent. of cases as seen at the first examination, while other observers find it no more commonly than among the normal population. More than half my own cases undoubtedly had some upper respiratory infection, such as tonsillitis, at the first examination. A seasonal incidence, probably in association with upper respiratory infections, has been described (Rosenburg, 1937). It appears probable from this evidence that a high incidence of upper respiratory infections is likely to be found only in the early stages, but becomes diminished when the chronic stage is reached.

The possibility of *tuberculous infection* presents an interesting problem. Earlier generations of physicians tended to explain all cases of chronic umbilical colic as tuberculous in origin. It is difficult in some cases to differentiate clinically between chronic non-tuberculous and tuberculous mesenteric lymphadenitis, but certain features are of help (see Table 2).

TABLE 2

DIFFERENTIATION OF CHRONIC MESENTERIC LYMPHADENITIS

	<i>Tuberculous</i>	<i>Non-tuberculous</i>
Wasting	may be marked	slight or absent
Pyrexia	often considerable	slight or absent
Recurrent colic	frequent	often long intervals
Continuous pain	frequent	unusual
Palpable abdominal glands	common	rare
T.B. in stools	may be found	absent
Mantoux test	positive	may be negative

It has been claimed (Rosenburg, 1937) that in non-specific mesenteric lymphadenitis the incidence of positive tuberculin reactions is significantly low, but in my own cases there has been no appreciable divergence from normal expectation.

(4) *Age incidence.* It is significant that generalized lymphatic hypertrophy in response to localized infections predominates in the age-group in which umbilical colic most commonly occurs. At this same period of life the lymph nodes are liable to hypertrophy to a degree which is considerably exaggerated when compared with adult standards.

(5) *Laparotomy.* From the above evidence, it appears that in the typical case of umbilical colic the cause is some disease process, probably infective, which is localized to the mesentery mainly of the small intestine and probably involves the regional lymph nodes.

These inferences lend themselves to direct confirmation at laparotomy. With modern criteria, laparotomy is more and more to be condemned as a sign of failure in diagnosis ; nevertheless, the surgical temerity which led to 200 laparotomies for diagnostic purposes (Fitzsimons, 1946) appears to have served its purpose. Fitzsimons observed enlarged mesenteric or pre-aortic glands in all cases with umbilical colic which came to operation.

From his description, and that of other observers, the differentiation from tuberculous glands appears fairly clear.

Laparotomy has led also to more precise diagnosis of cases previously diagnosed as ‘chronic appendicitis.’ Though clinical certainty cannot always be achieved, it should now be the rule and not the exception (see Table 3).

The dictum that ‘the appendix never “grumbles” ; it screams or remains silent’ has been justified. Laparotomy reveals that in the large majority of cases the symptoms of so-called ‘chronic appendicitis’ are due, in childhood at least, to mesenteric lymphadenitis. But dangerous exceptions occasionally arise, since generalized hyperplasia affecting the abdominal lymphoid tissue sometimes includes the lymphoid tissue of the vermiform appendix (Malloy, 1945), with the production of a genuine appendicitis.

TABLE 3

DIFFERENTIATION OF APPENDICITIS AND MESENTERIC LYMPHADENITIS

	<i>Appendicitis</i>	<i>Non-specific mesenteric lymphadenitis</i>
Spasms of pain	May start centrally, and later be sharply localized to R.I.F.	Pain usually central, but may also be present in R.I.F.
Muscular rigidity	Common	Rare
Abdominal tenderness	Acute and sharply localized to R.I.F.	Slight or absent, vaguely localized
Movement of patient during spasms	Uncommon	Common
Pain between spasms	Persistent	Usually absent
History of previous attacks	Uncommon	Common
Associated with upper respiratory infection	Occasional	More frequent

(6) *Evidence against adenitis.* By no means all observers are agreed that non-specific mesenteric lymphadenitis accounts for the majority of cases of umbilical colic, and some alternative explanations will be discussed.

Psychological disturbances. This possible explanation evokes more fervent partisanship and antagonism than almost any other. On the one hand, such observers as Cameron (1946) with a wealth of clinical and psychological data, attribute almost every case of umbilical colic to psychological aberrations. On the other hand, we have equally strong expressions of disapproval, such as : ‘The abdominal expression of psychological discomfort is unknown in the child’ (Maitland-Jones, 1947). From my own cases I find it difficult to accept the explanation put forward by the psychologists. It is impossible to deny that psychological disturbances exist in some children with umbilical colic: the difficulty lies in accepting them as causative. In most instances the patients appear not to differ psychologically from other children who have suffered from recurrent pain, and often the onset of psychological disturbance does not precede that of the pain. Moreover, there does not appear to be any greater incidence of umbilical colic in only children, or in children at ages when psychological disturbances occur most commonly. Obviously psychological cases must, of course, occur, as in one of my own patients who suffered from recurring, rather vague, abdominal pain for several months, with complete cure which coincided with the discharge from hospital of his father who had been treated for peptic ulcer. On the whole, it seems more probable that such psychological disturbances as occur are the result and not the cause of the pain ; if the patients have a ‘neuropathic constitution’ it is, surely, an acquired one.

Postural defects. Some observers (Talbot and Brown, 1920) have attributed umbilical colic to postural defects. In this contention they share

some common ground with the psychological school, who also describe characteristic postures in these cases. Talbot and Brown described as the typical postural defects a flat chest, rounded dorsal spine, and prominent lower abdomen; they were able to relieve the pain by correcting the posture. In a few of my own cases considerable spinal deformity was present. In one of these the umbilical pain had originally been recurrent, but had become constant, and in this child correction of the deformity (scoliosis) certainly coincided with relief of the pain. In other cases the posture was not appreciably improved by exercises and symptoms persisted. Wider experience should permit more precise differentiation of pain associated with postural defects. The following criteria are tentatively put forward:

- (1) The postural defect should be considerable.
- (2) The pain should be constantly, or frequently, present, and not recur only in short bouts with long intervening periods of freedom.
- (3) The pain should be abolished with remedy of the postural defect.

Chronic constipation. To chronic constipation many diseases have, on some occasion or other, been attributed. Following on older descriptions, Toverud (1925) has recorded a series of cases of umbilical colic associated with constipation and/or diarrhoea, and other symptoms which were attributed to bowel disfunction. By correcting the diets he improved the symptoms. Nevertheless, the conception that symptoms arise from chronic constipation is nowadays considered to be a legacy inherited by the lay public from the medical profession, after a delay customary with such bequests.

In my own cases it did not appear that dietary deficiency could be causally implicated. Enquiry fails to reveal any dietary inadequacy, nor had changes in the diet or its fortification with vitamin extracts produced any amelioration of symptoms. Moreover, umbilical colic was by no means confined to children of large families or in the lower income groups.

Infestation with worms. The bed-pan is a happy hunting ground not only for worms but for theories. Worms are frequently found in the stools if the search is sufficiently assiduous. In two of my cases *Giardia lamblia* was found, but umbilical pain continued after cure just as inevitably as if they had been the more prosaic *Oxyuris vermicularis*. Many cases of umbilical colic are referred for further opinion long after infestation with worms has been eradicated. A colleague from the Near East assures me, however, that in that part of the world roundworms present a much more serious problem, and that when infestation is cured many abdominal symptoms, such as pain, do in fact clear up.

Calcified abdominal glands. In a small proportion of cases with umbilical colic calcified abdominal glands can be demonstrated radiologically. Among 20 consecutive cases they were, however, seen in only one instance. It is theoretically possible that, by producing adhesions, they may provoke some degree of obstruction of the intestinal tract, but in the only proved cases of which I have experience the obstruction increased progressively.

Investigation of a Case

The investigation of umbilical colic should be based on two primary considerations which are apparently contradictory: first, *to eliminate serious and remediable causes*; and second, *to spare the child unnecessary hospitalization and investigation*.

History. Experience teaches that in only a very small proportion of cases is serious disease present. Moreover, in these cases a precise history nearly always provides clear indications of the gravity of the underlying lesion. Examples are: Pott's disease, chronic intussusception, abdominal tuberculosis and abdominal neoplasm. The two most important points to be clarified in the history are: (i) whether the pain is, in fact, peri-umbilical, and (ii) whether the symptoms indicate a lesion which is progressive.

General physical examination. Wasting, severe anaemia, a high temperature and the presence of considerably enlarged intra- or extra-abdominal glands are obvious indications of serious disease. In borderline cases difficulty will be experienced: for example, in almost every child small glands may be palpable in the neck, axillae or groins. The spine should be carefully examined; postural deformities of any significance should be observed, and can with advantage be corrected, even if they are unrelated to the symptoms. Thorough examination will reveal any serious disease outside the abdomen, and many inside it; laparotomy must be considered if there is muscular rigidity, or if tenderness is sharply localized to the right iliac fossa. Such conditions as a hernia of the linea alba, or undescended testicle, should be carefully sought. The upper respiratory tract deserves special attention; previous tonsillectomy does not preclude the diagnosis of non-specific mesenteric lymphadenitis.

Investigations. The urine should be examined chemically and microscopically in all cases, but only in the presence of precise indications need further investigations, such as examination of the urine for tubercle bacilli, be attempted. If the opportunity arises the stools may be inspected, but in the absence of a history of abnormality, melaena for example, further examination is unnecessary. I have found barium meal examina-

tions a waste of time and effort. Examination of the blood is, perhaps, a wise precaution; but serological tests and E.S.R. estimations are very rarely helpful. In my patients radiography of the chest and tuberculin tests contributed nothing towards the elucidation of the problem, though the case is arguable for carrying them out on all children suffering from any symptoms whatsoever. Such procedures as blood-sugar estimations, sigmoidoscopy, and intravenous pyelography (which were carried out in a few cases) have proved unjustifiable in the absence of positive indications.

It will be seen from the above discussion that in the vast majority of cases simple investigations, which are readily carried out in one interview, alone prove necessary.

Conclusions

'Umbilical colic,' the recurrent peri-umbilical pain of childhood is, in the large majority of cases,

due to non-specific mesenteric lymphadenitis. Nevertheless, umbilical colic may be the expression of other, and more serious lesions, and these should be excluded. The process of elimination can almost always be completed during the course of one clinical examination together with an accurate history, and aided by a few simple ancillary investigations.

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THE CHANGING NATURE OF GASTRIC AND DUODENAL ULCERATION

By J. DONALDSON CRAIG, M.D., M.R.C.P.

Research Fellow in Medicine, St. Mary's Hospital, Medical School

'There are gastric ulcers and there are duodenal ulcers. The differences between them are not merely geographical. In my opinion no real advance in our knowledge of the aetiology, clinical manifestations or treatment of these two diseases, gastric ulcer and duodenal ulcer, will be made if we speak of them as one.'

MOYNIHAN. (1928).

Introduction

Acquired disease is the product of disharmony between constitutional and environmental factors. It is therefore to be expected that in the course of the years there should be considerable change in the nature, prevalence and distribution of many diseases. In the present survey, an attempt has been made to trace the evolution of gastric and duodenal ulceration from earliest times to the present day.

From this review, it emerges that not only have the total incidence, the age and sex distribution and the clinical picture of peptic ulceration changed very considerably, but that the evolution of gastric and of duodenal ulcer have been along

very different lines. It is therefore suggested that although, as postulated by Hurst, individual constitutional variations may play a large part in the genesis of peptic ulcer, environmental factors are of even greater importance, and it is to such factors that attention should be directed in an attempt to control these diseases. Further, the history of the development of gastric and duodenal ulceration through the ages being so distinct, one is inclined to suspect that the differences between the two principal types of ulcer are more than geographical. It should not therefore be too readily assumed that gastric and duodenal ulceration are of entirely similar pathogenesis.

The Classical Period and Middle Ages

The earliest reference to gastric disease is found in the Ebers Papyrus (circa 1500 B.C.) in which cancer of the stomach is mentioned. Hippocrates was familiar with haematemesis and melaena, both of which he regarded as of serious import, particularly if associated with fever, but there is nothing in his writings to suggest that he recognized the ulcer syndrome. Gastric ulceration as such is first