oedema of the feet was present. This we considered was probably due to hypoproteinaemia.

The fatality rate was low (2.3%). Of the five patients who died four were infants. All were bacteriologically proved cases, four being of Flexner and one of Shiga type. This low fatality rate is in keeping with our finding that the majority of cases were mild in character.

Diagnosis

The most difficult disease to differentiate is salmonellosis, in which also blood is often found in the stools at onset. The only accurate means of diagnosis is by bacteriological culture. Amoebic dysentery may be identified by finding the cysts or vegetative forms of *Entamoeba histolytica* in the stools, or from knowledge that the patient comes from an area where such infection is endemic. Infections of the central nervous system may be diagnosed in shigellosis, especially in the early, pre-diarrhoeal stage of the fulminant type of infection, when the picture suggests an encephalitis. Diarrhoea due to parenteral infection or alimentary upset may be differentiated by finding a focus of infection elsewhere—for example, otitis, nasopharyngitis, pneumonia, or pyuria—or by the response of the patient to correct dietary regime.

Conclusions

The younger the child the more difficult is the diagnosis of shigellosis, because in infancy the disease more often suggests gastro-enteritis than dysentery, and because diarrhoea of other aetiology (alimentary, parenteral, or salmonella infection) may present the same clinical features as the dysenteric form of shigellosis. The disease is an important one in summer, especially in Yugoslavia, where standards of hygiene are low, and should be regarded as the most probable cause of enteric illness in infants and young children. The disease has an acute onset in children and lasts longer and is more severe in infants than in the older group. The most efficient method of diagnosis is the finding of shigellae in the stool.

Spread of the disease is by means of infected food. This is suggested by the fact that only small numbers of cases are seen in infants in the first three months of life when breast-feeding is usual, whereas older children are frequently affected. As the child becomes older and receives cow's milk and solid foods, especially when these are uncooked, opportunities for infection increase. In addition, with increasing activity the child himself may put infected objects into his mouth.

The small number of infections we saw during the winter months were probably exacerbations of chronic shigellosis, an uncommon condition under the age of 2 years, but more frequent in adults. The survival of shigella organisms in adult cases throughout the winter months ensures that there is a reservoir of infection from which epidemics may appear in children in the summer. As the number of cases of chronic shigellosis in adults increases, greater opportunity of infection in childhood occurs.

Summary

An analysis has been made of 221 cases of shigellosis in children up to 2 years of age in Zagreb, Yugoslavia. In 59% there was bacteriological proof, and in the remainder the epidemiological data suggested shigella infection as the cause of illness. The Flexner type of organism was most frequently found.

The majority of cases were mild in character. Five children in the series died—a fatality rate of 2.3%.

Four clinical forms of the disease were noted, of which the most severe were seen in infancy. The importance of the variation of the disease in infants from the classical description, and the importance of routine stool plating in this group, are emphasized.

The diagnostic difficulties are discussed.

ABDOMINAL MIGRAINE IN CHILDREN

BY

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Considerable interest has recently been shown in the condition variously known as cyclical vomiting, abdominal migraine, abdominal epilepsy, or the periodic syndrome. By whatever name it may be called the clinical manifestations are of a remarkable constancy, consisting in recurrent attacks of abdominal pain, vomiting, and headache, occurring alone, consecutively, or in combination. Laboratory investigations and radiological examination seldom help in the diagnosis, which remains in doubt unless this syndrome is recognized. The importance of the condition is indicated by its frequency—one case in ten patients attending a paediatric clinic (Kempton, 1956)—and by the number of children who are unnecessarily subjected to operation because of it. The aetiology remains in doubt, but an inherited constitutional abnormality is admitted by most writers on the subject, and a close association between migraine and, more recently, epilepsy has been recorded.

Hurst (1924) thought that bilious attacks in children were most often due to migraine or appendicitis, and could detect no difference between these attacks and so-called "psychical vomiting." Riley (1937) emphasized the importance of suspecting migraine as the cause of obscure abdominal symptoms in children. Piper (1951) described a case of abdominal migraine, and regarded it as indicating the similarity between cyclical vomiting in childhood and the abdominal variant of migraine. Krupp and Friedman (1953) analysed the case histories of 50 children suffering from all forms of migraine and found that they were weighted with attacks of biliousness, vomiting, and abdominal pain. In their series 84% of the parents suffered from headaches and 70% from migraine.

This association between migraine, cyclical vomiting, and abdominal pain seems to be so common that records have been kept of the incidence of familial headaches and migraine in a series of cases. It is the purpose of this paper to describe the series, to quote typical cases, and to put forward the evidence in favour of a migrainous aetiology for the symptoms.

Mechanism of Attack

It is necessary first to define the term "migraine." There is no known method of proving the diagnosis of migraine, though certain investigations, such as electroencephalography, may be suggestive. It is therefore fortunate that the classical syndrome is so precisely delineated that little doubt about the diagnosis need arise. The cause of migraine itself is still not known, but the mechanism of an attack is now fairly well established. There is good reason to believe that the onset of the attack is due to a vasoconstriction of the arteries supplying the head and brain. This vasoconstriction leads to anoxia of the cortex and so to the aura of migraine, which is often visual. This is followed by a reactionary vasodilatation of the vessels that can sometimes be seen in the superficial vessels of the scalp. It is this stretching of the blood vessels which is believed to cause the headache, and since this vasodilatation can be confined to one carotid artery it is often unilateral.

To be accepted as migrainous in this series headaches must have had at least three of the following characteristics: they must have been cyclical, and in women often aggravated at the menses and becoming less severe at the menopause; they must have been severe enough at times to cause the sufferer to lie down in the dark and seek relief by sleep; on occasion they must have been unilateral and preceded or accompanied by nausea or vomiting. Teichopsia, scotomata, fortification spectra, paraesthesiae, or temporary palsies accompanying them have been accepted as diagnostic.

The series under investigation consisted of 112 patients (61 boys, 51 girls) between the ages of 18 months and 15 years. Of the parents, 72 suffered from migraine (61 female, 11 male). A further 32 suffered from recurrent headaches which may well have been migrainous but which did not meet the essential criteria: in these the sexes were equally distributed. The analysis of headaches in the grandparents was naturally less accurate, but in 12 maternal grandparents and 4 paternal grandparents an adequate history of migraine was obtained. Nine more distant relatives suffered from migraine, six from recurrent headaches.

Symptomatology

The series can be divided into six separate groups according to the predominance of the symptoms shown, but each merges into the other. The younger the child the more frequent were the abdominal symptoms, particularly vomiting. With advancing age the abdominal symptoms became less marked and headache more prominent.

1. *Attacks of Vomiting*.—This group represents those cases which would otherwise have been diagnosed as suffering from cyclical vomiting. There were five boys and two girls. Four mothers and four fathers suffered from migraine. The mean age at onset of symptoms was 2.75 years, but it was of interest to note that many had persistently vomited throughout infancy. Vomiting usually occurred early in the morning, before breakfast. Sometimes it was only once, more often it was repeated throughout the day, and more rarely it persisted for from two to three days up to a week. In these latter cases prostration might be extreme and require admission to hospital for intravenous therapy. It was the cause of the only death in the series. The vomit at first contained the food of the last meal, even if this had been taken a considerable time previously. In most cases it early became bilious and continued so. In many cases pallor of the stools was reported during the attacks and, not rarely, some looseness of the bowels. Fever, sometimes quite pronounced, was not a rare accompaniment. The attacks varied in frequency from once a week to once every six months, but usually they occurred at least once a month. Between attacks the health was quite normal.

Case 86.—A boy aged 8 had had recurrent attacks of vomiting every two to three weeks since the age of 2. From the age of 5 occasional headaches accompanied the vomiting. These usually affected the left frontal region. Both parents suffered from migraine. The attacks ceased completely for one year while he was receiving phenobarbitone, but when this was discontinued the symptoms recurred. Further treatment with this drug again completely controlled his attacks.

2. *Recurrent Headaches and Vomiting*.—There were 14 boys and 7 girls in this group; the mean age at onset was 7 years. Eight mothers and two fathers suffered from migraine. The headaches of 10 of these children were typical of migraine. Vomiting usually occurred at the peak intensity of the headache but sometimes at the onset. In other respects it was similar to that of the first group. The headaches varied in intensity from an insignificant discomfort to a pain of such intensity that the child could do nothing but lie down until it passed away. Not infrequently it was described as throbbing in character and steadily increasing in amplitude to an intolerable zenith. One mother was able to recognize an attack by the increased pulsation of the superficial arteries of the child's scalp. By far the commonest site of origin was just above one eye, spreading

thence across the forehead. Temporal headaches were not uncommon, but occipital headaches were rare. Teichopsia was quite frequent, and one child had ophthalmoplegic migraine. In one child temporary loss of consciousness occurred with the more severe headaches, and in another slight twitching of the face. Most of the attacks came once every two weeks, but it was not rare for some children to complain of almost daily headaches. In such cases periods of relief of a month or more might occur between bouts of headache.

Case 65.—A boy aged 14 was admitted to hospital at the age of 9 because of sudden complete loss of vision; this was followed by a severe headache. Vision returned in half an hour. He gave a history of previous recurrent weekly headaches accompanied by vomiting. The headaches were usually preceded by the appearance of bright dancing spots in front of his eyes. His mother had severe headaches once a year accompanied by vomiting, and had had bilious attacks all her life. His sister was known to suffer from migraine.

3. *Recurrent Abdominal Pain*.—There were four boys and five girls in this group. The mean age at onset of symptoms was 6 years. Eight mothers suffered from migraine, but none of the fathers. The abdominal pain was usually localized to the umbilicus or, less frequently, to the epigastrium; in only one case was it not central. It did not radiate, and had no relation to bowel habits. Rarely it was said to be made worse by food, but most children would not eat when it was present. Most often it was described as an ache or a feeling of fullness, but sometimes it was of great severity and resembled a colic. It usually recurred throughout one or more days, and if severe a continuous ache persisted. One child had an appendicectomy and another was operated upon for a suspected intussusception, both without relief.

Case 89.—A girl aged 6 had suffered from periumbilical pain of increasing frequency for 18 months. The attacks were preceded by pallor of the face of some hours' duration and occurred daily. Her mother suffered from migraine and her sister from travel sickness. Treatment as for migraine has completely controlled her symptoms.

4. *Abdominal Pain and Vomiting*.—There were 12 boys and 12 girls in this group. The mean age at onset of symptoms was 5½ years. Fourteen mothers and three fathers suffered from migraine. The combined symptoms had the same character as in the previous groups.

Case 50.—A girl aged 5 had had monthly periumbilical pain for the previous eight months. Vomiting often accompanied the pain, nausea always. She suffered from travel sickness. Her mother and sister suffered from migraine. An appendicectomy was performed without relief of symptoms.

5. *Abdominal Pain and Headaches*.—In this group there were 9 boys and 12 girls. The mean age at onset of symptoms was 7½ years. Thirteen mothers and two fathers suffered from migraine. Ten of these children had migrainous headaches.

Case 38.—This boy, aged 10, had had recurrent abdominal pains from the age of 7, accompanied by headaches twice weekly. The headaches were always unilateral, the side varying. They spread across the frontal region. On occasion they were preceded by the appearance of bright-coloured wavy lines in front of the eyes. His mother had migraine; his sister was renowned for the number of headache tablets she consumed. Treatment with phenobarbitone completely controlled his symptoms.

6. *Abdominal Pain, Headache, and Vomiting*.—This, the largest group of the series, represents the complete syndrome of abdominal migraine. There were 17 boys and 13 girls. The mean age at onset of symptoms was 5 years. Fourteen mothers suffered from migraine, but no fathers. In 14 of the children the headaches complained of were typical of migraine. The symptoms were otherwise as previously described. Four of these children had had their appendices removed without relief of symptoms.

Case 109.—This boy, aged 5, was first admitted to hospital at the age of 6 months for persistent vomiting. No cause was found. He was next admitted at the age of 4, on three occasions, because of monthly attacks of abdominal pain and vomiting.

An appendectomy was undertaken, but the pain returned within 12 days of the operation. At this time a history was obtained of migraine in the mother and of headache accompanying each attack of abdominal pain in the child.

7. Headaches Only.—This last group, consisting of six boys and three girls, has not been included in the series, as no abdominal symptoms were complained of. It was in this respect alone that these cases differed from those previously described. The mean age at onset of symptoms was 9 years. In five of the children the headaches were typical of migraine. Four mothers suffered from migraine, but none of the fathers.

Analysis of Cases

The nature of these children was so uniform as to deserve some comment. Though measurements of their intelligence were not undertaken, they were repeatedly described as above the average at school. It was almost universal for them to be described as conscientious, hard-working, and worriers. Tidiness and obsessional tendencies were common. The most frequent precipitating cause of an attack, whatever form it might take, was nervous tension. O'Neill (1951) emphasizes this factor in a series which he investigated from the psychiatric point of view. It was common, therefore, for the attacks to come just before the school term, and examinations were a prolific and regular cause. It has been well called "*une maladie scolaire*" (Woringer, 1953). This timing of the attack led at times to misunderstanding of its cause and unsympathetic handling. It was not only unpleasant sources of nervous tension which led to an attack. Birthday and Christmas parties were often spoiled by vomiting at a time before a more likely cause of this symptom could possibly be held to blame.

Unfortunately an accurate analysis of the frequency of travel sickness was not undertaken, but this seemed a very common complaint both in the patients themselves and in their relatives. Recurrent febrile attacks without obvious cause also seemed common, and made difficult the assessment of the importance of infection as a precipitating cause. These, however, have been emphasized by other writers (Kempton, 1956). Those children who suffered from alimentary symptoms almost always found these to be aggravated by fatty foods and to be relieved by abstinence from them. Chocolate in particular seemed to offend.

Extensive investigations were not undertaken on these children, in view of their nervous nature. A barium-meal examination of the alimentary tract was undertaken in 20 cases—in 13 it was normal and in 7 spasm in varying parts of the alimentary tract was reported, most commonly in the duodenum. It is interesting that Piper (1951) reports dilatation of the stomach during an attack. These findings are non-specific, but were made by one radiologist who does not usually report such findings. Of greater interest are the results of electroencephalography. It was undertaken in 20 cases. Five of these patients were reported as normal, two as having very slow activity, possibly within the normal limits for their age, and 12 as having excessive slow activity well outside the range of normal for their age—in three this was paroxysmal, in two brought out only on hyperventilation. One child showed abnormal spiking in the parietal areas. The excessive slow waves were often of high voltage. Michael and Williams (1952) found only one normal E.E.G. in 11 when investigating children with migraine, and demonstrated the same slow high-voltage waves.

Treatment

It is not the purpose of this paper to discuss treatment; indeed, in a condition with so large a functional element it is impossible to assess how much benefit is due to suggestion and how much to drug therapy. Without a control series it is therefore possible to give only a clinical impression. The results have, however, been so clear-cut that this may be of some value. In no case has treatment entirely failed to

relieve symptoms, and in most cases the relief has been either complete or to such a degree that the symptoms no longer interfere with the child's life.

Treatment has been directed first to the relief of all sources of nervous tension and to an explanation of the child's symptoms. This has often elicited the comment that these were of course identical with those suffered by the mother in childhood. Such a realization diminishes greatly her own fears concerning the prognosis of the attacks and lessens the tension with which they are handled. In those children in whom vomiting or abdominal pain was prominent a low-fat diet with the total exclusion of chocolate and cocoa has been advised. So far as these symptoms are concerned, this appears to be an almost specific remedy. Woringer (1953) is so convinced of the value of this method of treatment that he considers the basic disorder to be a dysfunction of the liver caused by ingestion of fat—"une dyshépatie lipidogène." He claims uniformly good results with a low-fat diet and the use of methionine, choline, and inositol. Graham (1951) is opposed to this line of therapy but cites no cases. The relationship between migraine and liver dysfunction has been long recognized; it is discussed in detail by Gorse (1952), who emphasizes the frequency of atony of the gall-bladder as demonstrated radiologically during an attack. It may be that the finding of pale stools in so many of these childhood attacks of migraine is evidence of some failure in liver function.

So far as drug therapy is concerned phenobarbitone would seem to be pre-eminent in this condition, and has been given in this series to all those whose lives have been seriously limited by the affliction. Moncrieff (1951) emphasizes the importance of regular dosage with this drug. It has never been found necessary to give more than $\frac{1}{4}$ gr. (16 mg.) three times a day. In milder cases β -pyridyl carbinol has been given in a dosage varying from 8.25 to 12.5 mg. three times a day with considerable benefit. Anti-epileptic drugs other than phenobarbitone have been found of value by other workers (Moore, 1950; Wallis, 1955), a fact which gives some credence to the theory that an underlying epileptic mechanism is involved in the attacks.

Discussion

In this series well over half the children (64%) had one or both parents who suffered from migraine. Just under one-third (30%) suffered themselves from migraine in association with the other symptoms of the syndrome. If these attacks are not attacks of migraine they can be said with confidence to occur in children with a migrainous heredity, and to be precipitated by the very same factors that bring on an attack of migraine. It would seem more reasonable to consider that all the manifestations of this disease are but part of the clinical picture of migraine in childhood.

The classical description of this syndrome is to be found in an article by Samuel Gee (1882), on "fitful or recurrent vomiting." Little that has been written since has added anything to the clinical picture so brilliantly described. He noted the presence of "megrim" in three parents and severe headaches in two others. He also noted the early onset in infancy—an observation little emphasized in later writing. A full discussion of the syndrome took place in the Royal Society of Medicine (*Proc. roy. Soc. Med.*, 1951). Kempton (1956) has again drawn attention to its importance. Of recent years the suggestion has been made that these attacks should be regarded as epileptiform in nature (Moore, 1950; Wallis, 1955). To differentiate them from epilepsy is a difficult problem. Indeed, the borderline between migraine and epilepsy has always been a narrow one, and nowhere more so than in this condition. Chavany *et al.* (1954) describe the case of a child aged 13 suffering from classical migraine with teichopsia who occasionally had the same visual aura followed by a generalized convulsion. Jewesbury (1954) discusses the same problem and quotes an identical condition in a woman, and two cases of migraine in which temporary loss of consciousness occurred at the

onset of the attack. In the present series three children had symptoms suggestive of epilepsy accompanying their headaches. One child had attacks of unconsciousness lasting half an hour at the onset of her more severe headaches; one had slight twitching of the face, and a sense of not knowing where he was; and a third had twitching movements of the fingers of the left hand.

Moore (1950), referring to this problem, puts forward a theory which goes far to reconcile the two points of view. He proposes that the vasoconstriction of the onset of a migrainous attack may lead to such anoxia of the cortex as to cause the synchronous discharge of the affected neurones. If these be in the visual cortex, then the usual visual symptoms will occur; if in the motor area, motor symptoms; and if in the areas affecting the innervation of the gut, then visceral symptoms will occur. Though proof of this theory is lacking, it fits well with the observed facts. The finding of abnormal electroencephalograms in these children may lend some support to it. To call the condition abdominal epilepsy is, however, to cause the parents unnecessary anxiety (*Lancet*, 1955) and would seem to ignore the primary migrainous onset of the attacks.

It is to the surgeon that the recognition of this condition is of prime importance. Throughout the years the inadvisability of surgical treatment has been emphasized. Buchanan (1921) and Riley (1937) both stressed the importance of searching for a personal or familial history of migraine before undertaking surgery. Lennox (1941), referring to adults, considered that they were fortunate if they did not have either the appendix or the gall-bladder removed for this complaint. In the present series six children, or over 5%, were operated on. That this condition is a frequent cause of abdominal pain is little recognized, and few references can be found in the standard paediatric textbooks. A recent review (Hindmarsh and Court, 1955) of the acute abdomen in children referring to the medical differential diagnosis does not mention it.

Summary

A series of children suffering from recurrent abdominal symptoms and headaches are described. Reasons for suspecting a migrainous aetiology are discussed. The close relationship with epilepsy is pointed out. The danger of surgical treatment is emphasized.

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REFERENCES

- Buchanan, J. A. (1921). *J. nerv. ment. Dis.*, **54**, 406.
 Chavany, J. A., Lobel, G., and Hagenmuller, D. (1954). *Sem. Hôp. Paris*, **30**, 1626.
 Gee, S. (1882). *St. Bart's Hosp. Rep.*, **18**, 1.
 Gorse, J. M. (1952). *Gaz. méd. France*, **59**, 183.
 Graham, S. (1951). *Proc. roy. Soc. Med.*, **44**, 720.
 Hindmarsh, D., and Court, D. (1955). *British Medical Journal*, **2**, 899.
 Hurst, A. F. (1924). *Ibid.*, **2**, 60.
 Jewesbury, E. C. O. (1954). *Ibid.*, **2**, 1518.
 Kempton, J. J. (1956). *Ibid.*, **1**, 83.
 Krupp, G. R., and Friedman, A. P. (1953). *Amer. J. Dis. Child.*, **85**, 146.
Lancet, 1955, **2**, 913.
 Lennox, W. G. (1941). *Science and Seizures*, p. 212. Harper, New York.
 Michael, M. I., and Williams, J. M. (1952). *J. Pediat.*, **41**, 18.
 Moncrieff, A. (1951). *Proc. roy. Soc. Med.*, **44**, 724.
 Moore M. T. (1950). *Ann. intern. Med.*, **33**, 122.
 O'Neill, D. (1951). *Proc. roy. Soc. Med.*, **44**, 721.
 Piper, D. W. (1951). *Med. J. Aust.*, **2**, 468.
Proc. roy. Soc. Med., 1951, **44**, 719.
 Riley, H. A. (1937). *Bull. neurol. Inst. N.Y.*, **6**, 387.
 Wallis, H. R. E. (1955). *Lancet*, **1**, 70.
 Woring, P. (1953). *Arch. franç. Pédiat.*, **10**, 770.

AN INNOVATION IN TECHNIQUE FOR DENTAL GAS

BY

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It would have been thought, as "dental gas" ushered in the first attempts at anaesthesia, that by this time no innovation in technique would be possible or desirable.

However, our concept of anaesthesia with nitrous oxide has of late years altered so remarkably that on reading a communication by Klock (1955) on a technique of "amalgasia" for dental surgery I decided to try it out at some of the Gloucestershire County Council dental clinics. After using it on some 200 consecutive unselected cases the results have been so uniformly and strikingly successful that I consider a preliminary report on the method worth while. These clinics are the so-called school dental clinics and the patients are mostly children of school age (5 to 15 years), but we also get quite a number of pre-school age (usually from about 3 years old), and also a sprinkling of nursing and expectant mothers.

The technique is revolutionary in that it postulates using a high percentage of oxygen (20%) with nitrous oxide. The condition of amalgasia is defined by Klock as "a plane of surgical anaesthesia between the so-called analgesia plane and the second or excitement stage, wherein surgical operations may be performed without pain or memory."

Technique

As in these clinics the patients are mostly children, and as an average of some 15 are dealt with at a session which limits the time for each, I have departed in detail from Klock's technique. For the children under about 13 years I reckoned that, owing to their higher metabolic rate and to the greater possibility of their reverting to mouth-breathing and the difficulty of an adequate fit for the nose-piece, one should aim at about 15% oxygen. In addition, as it is necessary to gain their confidence, and as any delay in induction might cause them to be frightened, I start them off with a few (not more than six) breaths of pure nitrous oxide and then immediately switch over to the high-percentage oxygen. The details of this technique are as follows.

1. Jectaflo machines are used at these clinics.
2. Premedication is not used and the children and adults are handled in a way to secure their confidence and co-operation. This is usually not difficult to achieve.
3. The patient is seated in position in the dental chair, a suitable mouth prop is inserted in the mouth (this can be omitted until later, if thought advisable), and the child is instructed to blow out and in through the nose.
4. The machine is set to deliver nitrous oxide just off the pressure flow, a suitable-sized nose-piece is placed over the child's nose, and he is told to blow into it with his nose ("You will then hear yourself blowing a tune with your nose" being a good inducement). The expiratory valve should not be too loose. After about two breaths the pressure is slightly increased and the breathing-bag switched in to the circuit. If, with this apparatus, the expiratory valve is not too loose and the pressure is adequate, the bag distends satisfactorily at each expiration and converts the machine into almost an intermittent-flow apparatus. This not only makes for economy but gives valuable visual indication of the effectiveness and character of the nasal respiration. After some four more breaths the machine is