others. Face powders, silk, linseed, and other causes of sensitization turn up from time to time.

A particularly interesting protein is that of house dust; patients often give a reaction to the dust from their houses and to nothing else. American observers and van Leeuwen have also noted this. On one occasion I found I was obtaining an unusually large number of reactions to flour. Patients would react to it when they would not do so to anything else. In comparison with this we may note that van Leeuwen finds in Holland many cases of asthma due to the habit they have there of keeping flour and grain in the house and to its becoming infected with mites and moulds. I have added this infected wheat and the house dust to my routine tests. I have noted that patients in whose sputa moulds are detected suffer from a particularly pernicious type of asthma.

Having discovered proteins to which the patient is sensitive, three methods of treatment are available.

(a) *The avoidance of the protein.* The only proteins which it is difficult to remove from the patient's surroundings are the pollens, horse hair, house dust, and the proteins amongst which he works.

(b) *Specific desensitization.* Provided the correct dosage is arrived at by the dermal reactions using dilutions of the protein great success often follows this method.

(c) *The non-specific method of desensitization.* In certain cases peptone given intravenously is very valuable, in others I prefer the Danyasz or mixed coliform vaccine, after which a particularly permanent cure may result. Autohaemotherapy and colloidal manganese, either separately or together, have cured many cases.

Many patients are benefited by small doses of vaccine and made much worse with large doses. If a dose grossly upsets a patient, especially if it gives him an attack of asthma, a dose ten times smaller should be tried.

With perhaps half a dozen exceptions every one of this series of 1,000 cases of asthma has been tested to all those proteins which may be causing the asthma. Removal of those proteins to which the patient is sensitive cannot always be expected to be successful, because the patient may be sensitive to the other proteins or have asthma also from other causes which are not so easily discovered.

A young lady sensitive to wheat grew progressively worse instead of better after giving up wheat, until I found that she was also sensitive to compound extract of liquorice, which was contained in a prescription I had given her.

Type 2.—Clinically many of these patients are exactly like those of the sensitive type, to which they will be transferred as fresh causes of sensitization are discovered.

Type 3.—These patients are often sensitive to feathers.

Type 4.—These patients have asthma at school, at home, and away on holiday, periodically, perhaps once a fortnight. Very often nothing will cure them but complete enucleation of the tonsils. More rarely the nidus of infection is to be found in a nasal sinus, an appendix, or even a single tooth abscess. One woman lost this type of asthma after the removal of an unhealthy uterus.

Types 5, 6, and 7 will be dealt with in some future article.

Type 8.—Patients of the periodic type suffer from four or five tremendous waves of asthma each year. The attack may take a month reaching its height and the same in going again. After a month of freedom the whole thing occurs over again.

Type 9.—These patients bring up some sputum every day. A patch of bronchitis is often found in the lungs. An autogenous vaccine from the sputum is often entirely successful in curing this somewhat rare type.

Type 10.—The only symptom peculiar to this type is that the patients have a high blood pressure, usually round 200. Apart from that we find them distributed here and there throughout all the other types, and the asthma should be treated quite independently of this symptom.

Type 11.—Of the whole series of 1,000, 22 per cent. had eczema in early life; 30 per cent. of the sensitive type had had eczema and 34 per cent. of those sensitive to foods.

Type 12.—Asthma in this type is due to an intake of too much food, especially the last thing at night, and insufficient exercise. Though the patient has asthma from other causes, a strict diet will do much to help the majority

of cases. This type is very fully described by Dr. James Adam of Glasgow in his books. I have many patients who keep entirely free by following his régime of starvation and exercise.

Type 13.—Typical of this is, of course, hay asthma. But I also find a number of patients who have pronounced asthma strictly limited to the months of August and September. Christmas is often a bad time; so many unusual proteins are met at that season. Recently I had a patient sensitive to holly.

Type 14.—A large number of patients are worse at the seaside, on clay soil, or in woods, especially if damp, or when in houses surrounded by trees.

Types 15, 16, and 17.—I hope to discuss these in a future article. Type 17 is quite the worst that I see. Naturally these patients never touch aspirin, but their asthma is almost continuous. They nearly all suffer from nasal polypi, a rare thing in ordinary asthmatics.

Results of Treatment.

In order to obtain statistics on the subject, 300 patients were written to. These had been seen by me between September, 1925, and December, 1926, and included every single case of asthma seen during that time. The answers received denoted the condition of the patient's health during the one to two years since coming to me. Replies were received from 223; of the rest two had died, both from strokes, and 14 could not be traced; the remainder have not answered. Many of the latter, to my knowledge, are abroad, and there is no reason to suppose the results obtained in these would be less favourable than from those who answered.

Replies received from 223 Cases.

(a) "No better"	...	...	...	...	44
(b) "A little better"	...	...	...	...	31
(c) "Better"	...	...	...	...	32
(d) "Much better"	...	...	...	...	17
(e) "Very much better indeed"	...	...	...	...	34
(f) "Practically free"; "Occasional attacks from overfeeding"; or "Perfectly well"	...	...	...	...	65

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Taking those patients in (e) and (f) their average age was 27 and the duration of their asthma thirteen years. The average age of (b), (c), and (d) was 37 years, with a duration of asthma for fifteen years. Those "no better", (a) were 40 years of age and had had asthma for seventeen years on the average.

In conclusion, and considering that these 300 cases written to were in series and not picked cases, and that by far the larger majority were only seen once, I think the results obtained are sufficient to prove that these new methods of treating asthma have more than justified their trial.

CONVULSIONS OCCURRING DURING SURGICAL ANAESTHESIA.

BY

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THAT patients completely under the influence of a general narcotic, both children and adults, should develop general convulsions, and die of them, is extraordinary, but several instances have come to my notice. I have been an anaesthetist for nine years, and have made over 11,000 administrations. The first case occurred only two and a half years ago; I have been able to cut short the last few. The seizure is epileptiform, comes on at any time during the progress of the operation, and may continue until the end and after. It may quite cease, but start again later on, perhaps after the patient has reached the ward (without, however, having regained consciousness). In other cases, after some time, it stops of its own accord, or in response to treatment.

It begins with twitching of the eyelids, or arms, or about the mouth, and extends rapidly all over the body, suddenly almost; but before this, I think, some jerkiness

of the breathing can be detected, inspiration is cut into two, and later on is so interfered with by spasms that ventilation becomes quite inefficient, and it may be difficult or impossible to keep a good colour even with oxygen. The convulsion is unremitting and severe, though there is not gross or violent movement, and it is obvious that it will quickly wear out the heart, even apart from the increasing respiratory insufficiency.

All the instances have occurred under ether, and nearly all in young people who have had an acute infection with more or less pyrexia. There have been fifteen in all, of which I will describe two or three typical ones.

The third case was perhaps the most singular of all, because when I saw the patient last he seemed on the way to a normal recovery from the anaesthesia, yet four hours later he was dead.

This was a boy, aged 7, with a gangrenous appendix, operated on at 6 p.m. in October, 1925, in hospital. From my own notes at the time: "Respiration very difficult owing to breath-holding in going under. Got generalized convulsions later on; these lasted till end of operation, but soon after stopped. Three hours later (in the ward) started again (after I had left the hospital). Artificial respiration; good colour; not round; respiration failed." Here I was impressed that the laboured breathing was the chief cause of the convulsions, the various muscle movements accessory to respiration overflowing apparently into the rest of the limb, face, and trunk muscles. There was no general peritonitis, and the boy was in good condition, but with some elevation of temperature; before he died, however, it went up to 107° F. The bomb method was here, as usually, employed. At the *post-mortem* examination there was nothing found that afforded any good explanation, and the coroner's verdict was in accordance with the medical evidence—that the fatal issue was due to some unknown effect of the ether.

It is most extraordinary that when I saw him, half an hour after the end of the operation, he should look perfectly well, though still unconscious, his colour, pulse, and breathing good, and so tranquil after the storm that there seemed not the least cause for anxiety.

The fifth case was at the same hospital, and the same method was employed. The date of the event and of my notes is April 13th, 1926.

"Strong healthy woman of 25; went under quite well; full blooded and rather muscular. Operation for removal of 'tarry' cyst difficult; moderate Trendelenburg position. Respiration full, unobstructed, good colour, and oxygen all the time. Suddenly, without the least warning, general convulsions—very severe; continued forty minutes. Anaesthetic stopped immediately, artificial respiration to regain regular breathing—no good; pressure of carotids seemed to give a few seconds quiet. Began to get bluish, and pulse not so good; also temperature rose to 101.8°, and then to 103°. Very little sign of recovery from anaesthesia. Suddenly became quiet when mouth was gagged open preparatory to passing an intratracheal tube and insufflating with air, which was therefore not done. Hewitt's airway tube has been in all the time. Respiration still rather jerky. In ward, pulse quite good, looking very slightly blue, breathing quiet, all right afterwards."

In this case there was no infection or toxæmia, the woman was in perfect general health, and the only exceptional features were the Trendelenburg position, and that the breathing was full. Immediate discontinuance of the drug did not, at any rate during forty minutes, relieve the spasms. But I will return to this matter. I then began to be haunted by the affection, for on two consecutive days in June of 1926 I had a patient develop it.

The eighth case was a girl, aged 19, in hospital with a temperature raised to 102.5° F. by an acute pyosalpinx, who became convulsed under deep narcosis. But I seem now to know what to do. The notes are:

"All right on ether (bomb) till stitching up peritoneum, deep now, sudden onset of general convulsions. Immediately stopped anaesthetic. Lasted (severe) until three or four minutes after bandaging, when I noticed blueness due to oxygen being discontinued. However, convulsions soon stopped, and colour became good again. N.B.—No trouble except some struggling during induction; respiration quiet, nice, but when deep at end was shallow, regular, but jerky. Temperature 101.2° F.; in bed immediately afterwards."

Of the eleventh case I have notes kindly written out for me by the administrator, Mr. R. L. Holt, when in residence.

W. B., aged 21, admitted September 15th, 1926, with acute appendicitis spreading peritonitis, and a high temperature (103.2° F.); 1/60 grain atropine given shortly before operation (which all the previous cases, too, had in doses suitable to their ages). Ethyl chloride and ether induction; cyanosis observed soon after, but improved with oxygen, the airway being quite

free. Though fully anaesthetized, spasm, followed by twitching of the eyelids, and then general convulsions, came on, and a good colour could not be kept. By the time the operation had been speedily concluded, the convulsions, though less severe, had brought him to the point of death. Stimulants, artificial respiration, and cardiac massage were alike unavailing, and he expired while still in the theatre. *Post mortem* there was found a distinctly enlarged thymus.

All but four of these cases occurred in my own practice, and in all these ether was given by the "bomb" method. In the last six of them the convulsions, in response to my treatment, lasted only a few minutes, or did not become general. The onset is definitely recognizable and easily to be distinguished from "ether clonus," which is of a coarser and at the same time a more rhythmical form. Notes were made of them the same day in all except two.

It appears from these cases that the convulsions are similar and typical. Five of them ended fatally, and in the first and fourteenth the patients were already in an extremely grave state before operation; in most cases there was pronounced infection, but in some, particularly the fifth (quoted here), this was not so.

All but one, the first, were in young people and children. All had ether, most of them nothing else, so that this appears so far as a causal factor, but no particular brand, as several kinds had been given. No history of epilepsy, convulsions, or chorea was at any time obtained.

Why is it that it is only recently that these things have been observed—only recently that they have happened, since they scarcely could have escaped notice?

Two matters suggest themselves in explanation—the gradually increasing use, especially by beginners, of ether, and the adoption, as a consequence, of many coverings over the frame and face to concentrate the vapour. Neither of these is very convincing, as little or no change in either direction, that I am sensible of, has taken place in my own routine during the past two or three years.

It is to be seen that in most cases the breathing is first affected: it becomes jerky, insufficient, or stertorous, or there is cyanosis, and in some, from the beginning, has been difficult, laboured, or uncomfortably deep and full. In my own cases I have not been satisfied in every one that the breathing could in any way be taken exception to; but in most I have, and always I have felt that it would not be unreasonable to blame an invisible increase of CO₂ for its effects, in the way of deep and energetic breathing, have, as I say, been obvious enough. Even without this it is fair to think that instead of resulting in steady and deep inspirations and in perfectly co-ordinated action of important muscles, it may produce only partly co-ordinated and jerky efforts, which can easily be supposed to extend to the whole body in conditions where, for some reason or another, there may be abnormal irritability of other muscles—such conditions, I mean, as pyrexia, toxæmia, and ether narcosis. Quiet respiration is regular enough—in fact, when breathing is quiet, it is the more regular—but under general anaesthesia it is frequently but a short step from the accustomed rhythm to various irregularities, both of a rhythmical and an arrhythmical sort; so that I have come to regard the convulsion as largely the outcome of the extensive action of the CO₂ in patients who are sensitive to this naturally, or by reason of toxæmia, pyrexia, and so on, and who are under the influence of ether, a substance itself having a marked effect on breathing.

I have on occasion seen persons inhaling ether take breaths constantly increasing in depth, vigour, and rate, until in a few moments the whole frame is in tonic spasm, where they remain for some moments quite unable to move or breathe at all. Moreover, those so-called "athetotic" movements come particularly to those patients who, by reason of rigidity during the struggling stage, have difficulty, often lasting some minutes, in drawing their breath—being, as it seems to me, overflow movements of muscles other than accessory muscles of respiration, and lasting in a disorderly manner for an unexpectedly long time. Such cases throw light on the subject of this report, and however it may be, treatment based on this assumption has so far been successful. It is directed to the riddance of excess CO₂, and must be prompt. Luckily some moments' warning is given of the impending seizure

by twitching of the eyelids and mouth, jerkiness of the breathing, especially inspiration, or some strange movement or sound, and so the essential thing can be accomplished—the immediate removal of all coverings from the face before the convulsions have set in. Frame, towels, everything, are taken right away, and the air of the room given free access; any obstruction must be completely relieved, and a gentle stream of oxygen allowed to flow towards the mouth. In this way the CO₂ will diminish as much as possible by natural means. Recovery from the anaesthetic, so delayed in most of these cases, is permitted to proceed, until it is utterly impossible to have the patient come round any more, when pure chloroform may be given in small quantities and by a quite open method, though, indeed, this will often not be needed. The success of this procedure depends on the promptitude with which the onset of spasms is foreseen.

A field for investigation is here; but the most important thing for the time being is that as many as possible should know how to recognize the condition and treat it.

THE APPENDIX REFLEX.*

BY

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THE appendix reflex, giving rise to pyloric spasm, followed by hyperacidity and symptoms suggestive of gastric or duodenal ulcer, is now well recognized.

The object of this paper is to suggest that in certain subjects, with unstable nervous system, a diseased appendix may be the exciting cause of symptoms of an entirely different character. I propose to illustrate this suggestion by reference to a series of cases which have come under my observation, in most of which the possibility of an appendical origin had never been suspected.

CASE I.—*Migraine.*

In 1912 a country farmer, aged 45, was sent to me with a view to operation on his stomach, owing to repeated attacks of vomiting.

His history appeared to be a typical one of migraine. As long as he could remember he had suffered from violent headaches, associated with the appearance of spots before his eyes and followed by severe vomiting. At first the attacks occurred only two or three times a year, then once a month. When I saw him they came once a fortnight. He wakened with them, and was completely prostrated for a whole day, and unfit to work on the following day. He had no history of abdominal pain at any time. The stomach appeared normal. I regarded the case as one of migraine, and said I could not give any guarantee of relief by operation. We might possibly find a chronic appendix, but operation would be largely exploratory.

I saw no more of him for four years. He came back stating that his attacks had become intolerable. They now lasted four days. He was five days in bed. He had to sell his farm as he was quite incapable of looking after it. He asked me to "open him up" and see if anything could be done.

At operation, which was carried out through a right paramedial incision, admitting of a thorough examination of the whole abdomen, we could find nothing abnormal except a very large, chronically inflamed and distended appendix, dipping into the pelvis and adherent there.

He made an uneventful recovery. He had no recurrence of headache for a month after operation. When I last heard of him, a year later, he still had occasional headaches, but they never lasted more than a day, and he was again farming.

I have no doubt whatever that if this man had had the appendix removed in early life the attacks of migraine would have disappeared completely.

CASE II.—*Migraine.*

Shortly after operating on Case I, in 1916, a school teacher, a lady aged 40, came to me complaining of steadily increasing migraine. Her attacks, which at first had been monthly, had now become weekly. They were very severe, and unfitted her for her duty for two days at a time.

All the usual investigations had been made. The eyes had been thoroughly tested, and reading glasses supplied, without relief. The uterus was normal in size and position, functions regular. Urine normal in every respect.

In view of the previous case I suggested the possibility of relief by operation, at the same time explaining that no guarantee could be given. She at once agreed, saying she could carry on no longer as she was.

* This paper was read before the Section of Surgery of the Royal Academy of Medicine in Ireland on February 18th; the discussion which followed was published in the JOURNAL of March 19th (p. 517).

At operation we found (1) a well marked chronic appendix, adherent to the caecum; (2) a cholesterine gall stone, the size of a small pea, in an apparently healthy gall bladder. The appendix and the gall stone were removed, and the gall bladder drained. She made an uneventful recovery.

Even after the operation, with knowledge of the pathological condition, we could elicit no history suggestive of either appendix or gall-bladder trouble.

I had a letter from her a year later informing me that she had now only a very occasional headache, which never interfered with her work.

CASE III.—*Migraine, with Glycosuria.*

In January, 1919, I operated on a young medical man, whose own account of his case speaks for itself.

"For several years previous to the onset of glycosuria (1917) I had periodic attacks of severe headache, associated with indigestion and loss of appetite, but no vomiting. The attacks lasted from two to four days, and were always in the frontal region, on one side or other, but never both sides together. The most tender spot was the exit of the supratrochlear nerve from the orbit. At times I could not use my eyes for the pain.

"The attacks continued till you operated on me in January, 1919. I think you found a chronic retrocaecal appendix, which you removed. This was associated with some cholecystitis, so you drained the gall bladder for a few days.

"Since that time I have been practically free from headache. I am also quite free from glycosuria, and am on an ordinary carbohydrate diet."

CASE IV.—*Cyclical Vomiting: Acidosis.*

Early in 1920 I saw, with one of our leading medical men, a little girl, aged 10. Since she was 2 years old she had suffered from attacks of acidosis with violent vomiting, occurring every two or three months, as the result of which she was confined to bed for some days, but was always relieved by large doses of alkalis. She never had any definite abdominal pain till the attack in which I had seen her. She then had definite symptoms of appendicitis. Immediate operation was advised, but it was with considerable difficulty that her parents could be persuaded to have this done. They said she was terribly nervous, and the very idea of operation would put her out of her mind. However, they finally consented.

At operation we removed a distended, congested appendix. Recovery was uneventful. Her attacks of vomiting and acidosis disappeared completely. Her nerves quieted down. In a short time she became a normal child and has remained so ever since.

CASE V.—*Cyclical Vomiting: Acidosis.*

On January 1st, 1923, I saw a little girl, aged 6. She was quite well till 2½ years of age. She then began to have severe "bilious attacks" once a month or oftener. The attacks increased in frequency and severity until 1923, when I saw her. They commenced with sneezing, running of the nose, then cough, lasting for a day, next day violent and persistent vomiting, which lasted twelve hours and completely prostrated her, leaving her greatly exhausted all the following day. She had some acidosis with each attack, but no headache. She never had any abdominal pain, except some muscular pain after the vomiting.

A finger in the rectum elicited some tenderness high up when pressure was made with the other hand in the right iliac fossa—that is, a bimanual examination of the ileo-caecal region, which is so useful in children. Otherwise nothing could be made out.

She was referred to a specialist for a thorough examination of the nose and nasal pharynx. He reported that there was nothing to account for her attacks. We accordingly operated, and removed a long, distended, corkscrew-like appendix. A month later she had one of her old attacks, then another slight one, after which they completely ceased. Her doctor reported on January 20th, 1925, that she had remained perfectly well ever since.

Osler says: "In young children acetone acidosis (cyclical vomiting) may give difficulty in diagnosis from appendicitis. There may be severe vomiting, with abdominal pain, and the caecum may be distended and tender. The smell of acetone from the breath and the urinary findings should distinguish." (Tenth edition, p. 533.)

I desire to suggest that in some of these cases appendicitis may be the real disease, the acidosis merely a reflex effect.

Appendicitis in children is very common, much more so than is generally recognized. I believe the majority of cases of so-called colic are primarily appendical, and that a large proportion of the cases of so-called chronic appendix which are met with to-day during abdominal operations have originated in early childhood. In the "nervous child" such an appendix may give rise to a variety of reflex symptoms, even in the absence of any local signs.

CASE VI.—*Epilepsy.*

In July, 1919, I was asked to see Mr. H., aged 37, who for years had been the subject of epileptic attacks. In 1904 he had an attack of what seemed to have been appendicitis, and was