the British Medical Association, the meetings of which have the reputation of being among the most successful and interesting of all societies, this use of the abstract has reached its highest terms, as yet. It is seldom wise to adopt wholesale the regulations of other organizations. It is usually better to let changes follow a more gradual and natural evolution under the needs of the individual assembly, but the methods of the English association may well be worth a passing consideration from you, and especially from those of you who must in their turn guide the destinies of our society.

A fellow of the British Medical Association who desires to present a paper at one of its meetings must put it in the hands of the secretary, complete and in the form in which he desires its publication, a number of weeks before the meeting, and the communication may be of any length he chooses. A paid secretary, a qualified and experienced medical author, then abstracts each paper into the form and length which he considers best fitted for spoken delivery. This official then reads the abstracts to the society, as they are called from the program. Such a reading inaugurates each discussion, and the member whose ideas have been thus succinctly set before his associates takes part in the discussion and closes it. It is said that their experience shows that the ideas of the individual member are by his method better and more intelligibly presented to the society than if he had read to them his complete paper. The time of the society is economized, full debate is encouraged, and at the same time the member has the advantage of publishing to the world a paper in which his points are set forth at the fullest length and without time limitation. Such a method is perhaps too far advanced for our present needs or possibilities, yet it has many advantages, and has seemed to me worth calling to your attention as one towards which we might well advance. A similar method has been adopted by common consent in the Association of American Physicians, and is spoken of with much enthusiasm by the members of that society. Would it not be wise for us to give this method, or a modification of it, a year's trial?

We must not underestimate the value of the other, the social, side of our meetings; but, indeed, none of us are likely to forget or undervalue the warm friendships and charming half hours which accrue to all of us from our membership in the society.

In limiting the remainder of my address to the expression of my opinion that in the now crowded state of our programs it would be better for the society that the conventional length of the presidential address should be curtailed, I feel that if I am right in this opinion, it will be evidenced by the conduct of my successors in this honored office. If their course should show me wrong, may I hope that you will at least believe that I have not been actuated by any unworthy desire to curtail my own labors, but rather that I have been willing to risk the possibility of

incurring such odium for what I believe to be the advantage of the society.

In conclusion, may I take this opportunity of expressing to each and all of you my thanks for the kindly personal feeling which must have actuated you when you elected me to your presidency, for which, and for the many personal pleasures which I have enjoyed in this society, I am, and must always gladly remain, your willing debtor.

Original Article.

ACIDURIA (ACETONURIA) ASSOCIATED WITH DEATH AFTER ANESTHESIA.

BY E. G. BRACKETT, M.D., J. S. STONE, M.D., H. C. LOW, M.D., BOSTON.

Presented for the Staff of the Children's Hospital, Boston.

THE attention of the surgical staff of the Children's Hospital has been challenged by the following facts:

On the 6th of October, a boy of ten years and a girl of eight years entered the hospital. Each had an extensive infantile paralysis of long standing. Otherwise each was well at the time of entrance. The boy remained perfectly well until four days after his entrance to the hospital, when he began to vomit persistently. The vomitus was watery, of light green color. Up to that time the bowels had moved freely. The abdomen was soft, but not distended. When the vomiting began, the pulse became very weak and increased in rate from 95 to 150. The temperature which had been normal rose gradually to 105.8. The child became unconscious, the condition gradually failed until death, twenty hours after the onset of the vomiting.

The girl had, on several occasions during the preceding year, had attacks of persistent vomiting, associated with collapse and rapid pulse. The child seemed delicate, but of sound health, except for the infantile paralysis. Four days after entrance, three tendon transplantations were done under ether anesthesia. The operations consumed one and one-half hours in time, but were insignificant in severity, and no blood was lost. The child took ether rather poorly, and during the operation had a rapid pulse. She vomited in coming out from the ether, as is usual, but seemed comfortable and in fair condition twelve hours later. She then suddenly began to vomit persistently. The pulse became very rapid, about 190, and of poor quality; the temperature gradually rose to 106. The vomiting lasted about thirty hours. The child then gradually sank into a delirious stupor and died forty-two hours after the operation, thirty hours after the vomiting began.

Six weeks later, on Nov. 16, a girl of five and a half years, also with an extensive infantile paralysis, entered the hospital. On the day after entrance, under ether anesthesia, the fasciæ on the upper outer part of both thighs were cut by open incision. The operation was short and very little ether was used. The child recovered well from the ether and was comfortable up to thirteen hours after operation. She then began to vomit. During the night she was very restless and noisy, sleeping only three hours during the night following the operation. Twenty-four hours after the operation, the circulation became very poor. In spite of the fact that there was no obstruction to respiration the color became livid blue, the face grew

cyanotic, the extremities were cold, the pulse weak and rapid, the expression was anxious. Thirty hours after operation the child became delirious. On the second morning after operation, the child became more dull and apathetic, until she gradually passed into a condition of coma, and died forty-eight hours after operation.

In this case the peculiar, sweet, chloroform-like odor of the breath led to the examination of the urine for acetone and diacetic acid, both of which were found to be present. No test for acetone was made in the two cases entering on the 6th of October; in one of them, however, the characteristic odor of the breath was remembered very distinctly.

On Dec. 17, two more patients, each with extensive infantile paralysis of long standing, entered the hospital. They were girls, aged six and eight years. One child began to vomit six hours after entrance; the other fifteen hours after entrance. Both became markedly dull and stupid. In each case the odor of the breath was characteristic, each had acetone and diacetic acid in the urine. Both fortunately recovered.

On Dec. 26, a girl four years old entered the hospital. For six days before she had been vomiting at frequent intervals. There had been no movement of the bowels since the vomiting began. The odor of acetone in the breath was very strong, acetone was present in the urine. An intestinal obstruction by Meckel's diverticulum was found and relieved by operation. The child died twenty-six hours later, having gradually become unconscious. In this case the presence of acetone seems probably to have been incidental to the intestinal obstruction; perhaps undue importance was attached to it because of the preceding six cases, in some of which acetone had been found. Operation was delayed a little because it was uncertain at first that obstruction existed.

After this, the sweet odor of the breath was noticed in a number of patients after operation. In these the urine was examined for acetone, which was found to be present. In most of them vomiting occurred, and some showed marked prostration, but in only the following case, however, were the symptoms alarming:

A girl of four, rather delicate and nervous, suffering from double congenital dislocation of the hip, had been operated upon by the Lorenz method five months before. Reduction of the hip had been easily effected, and the limb placed in the frog position with the usual spica bandage. Both hips were not reduced at once, as the child did not seem strong. The patient recovered from the operation without trouble and was returned home after a few days. The child was unable to learn to walk about, and returned to the hospital five months later in rather a poorer condition than on her first entrance, though her condition was not at all alarming. The child was quite nervous and frightened at her second operation. In manipulating the reduced hip, and changing it from the first to the second position, requiring but little force, as well as in the manipulative reduction on the unreduced hip requiring more force (though much less than is required in many cases), a separation of the epiphyses at the lower ends of both femora occurred, which indicated that the child was less strong than at the first operation. A double plaster spica was applied, and the child returned to the ward, the operation having been a short one, not requiring more than half an hour in all. The child recovered from the anesthesia well. There was a little nausea, there was absolutely nothing in any way unusual or alarming in the condition until six o'clock on the morning after operation, an interval of twenty hours. The pulse then grew suddenly weak and rapid, the

color became poor, but after a hypodermic injection of strychnine she improved. The child seemed a little dull, there was no vomiting, the breath was sweet. Acetone was found in the urine. Three hours later there was another sudden collapse. During the day the child grew rapidly worse, she sank into a restless, unconscious condition, the lips became very blue. Except for rallying somewhat during the evening, the child gradually failed, until she died forty-two hours after the operation.

These cases present certain features in common: Vomiting associated with collapse; a very weak and rapid pulse; an absence of fever until just before death; cyanosis, in the fatal cases, causing extreme dyspnea; apathy and stupor, alternating with periods of restlessness at first, but in the fatal cases gradually deepening into coma and death; and the presence of acetone in the breath and urine.

Excluding the case of intestinal obstruction, there remain 6 cases in which the symptoms came on without operation. In three of these cases the symptoms came on the day after entrance, and in one two days after. In one case the symptoms came on after the child had been in the ward for four weeks and was up and about. In the sixth case the symptoms came on four days after entrance. This child died.

There are 7 cases in which the symptoms followed operation. Three of these patients died, 4 recovered. In the 4 milder cases which recovered, no symptoms at all appeared within the first twelve hours after operation. The symptoms came on between twelve and twenty-four hours afterward. In the 3 fatal cases alarming symptoms came on after about twelve hours, but in none of the cases was the recovery from the operation quite normal. The condition of the children caused anxiety, although there was no one definite symptom which could be defined as its sufficient cause.

Those children in whom the symptoms were severe were of a high strung, nervous temperament. The symptoms coming on after operation were usually more severe than those coming on without operation, although in the latter group one rapidly fatal case is included.

The first symptom to attract serious attention was, except in one case, the vomiting. The vomitus was always bile stained, becoming dark, but never bloody. In one fatal case, however, vomiting occurred but twice, each time from an obvious cause. Otherwise, the symptoms differed in no way from those in the cases in which vomiting was a prominent symptom.

In one case the symptoms seem unquestionably to have been due primarily to acute intestinal obstruction. And yet, even in this case, the presumably secondary acetoneuria confused and masked the ordinary, or what might be termed the mechanical, symptoms of intestinal obstruction.

Restlessness at first was associated with the vomiting. Occasionally there was a certain amount of apathy either preceding or alternating with periods of restlessness.

In the milder cases there was a varying degree of prostration; in the severer cases marked collapse which was often repeated. The children would rally for a few hours only to fail again suddenly.

After a short time in some of the severe cases cyanosis and dyspnea became marked. None of the cases with marked cyanosis recovered. As the color changed, the restlessness was followed in some instance by delirium, but in all the fatal cases there gradually came on a stupor which became more profound, changing gradually to coma. Death in the fatal cases occurred from twelve to thirty-six hours after the onset of the symptoms, generally after about thirty hours. Death seemed to be due in some cases to a lack of oxygen. The struggle for breath seemed very striking till exhaustion came on.

Pathology.—The autopsy reports in the first two cases were made by Drs. Christian and Brinckerhoff. They were both cases of infantile paralysis with extensive muscular paralysis and degeneration of both lower limbs. In the first case there was a double intussusception of the jejunum, which was very evidently post-mortem. No important lesions were noted. In the second case no anatomical lesions were noted; no sufficient cause of death was found. There was a moderately fatty liver and large kidneys.

The other two autopsies occurred after the fact of the acetonuria had been fully appreciated. The third case was one of rather extensive infantile paralysis, dying within twenty-four hours after the comparatively simple operation of fasciotomy. The pathological interpretation of the cause of death in this case was practically negative, except for a very extensive fatty metamorphosis in the liver and less marked one in the kidneys. The fourth case was one of double congenital dislocation of the hips, which died within forty hours after operation. There was extreme fatty degeneration of the muscles of both thighs and a fatty liver and kidneys. In addition there was in this case a large number of eosinophilic cells in the spleen and the glandular epithelium of the stomach, which were only seen in small numbers in the intestine of the third case. These were in some cases phagocytic and similar to the cells which Mallory has shown in scarlet fever and acute infectious disease.

The only marked anatomical lesion which these cases showed was the extreme fatty degeneration of the liver and the muscles. This fact is more interesting when associated with the clinical knowledge that in the eight most severe of these cases there was either a paralysis or some disability resulting in the degeneration and atrophy of an extensive part of musculature.

Fatty liver is generally the result of the quality and quantity of the food and the general constitution. Diet rich in fat and carbohydrates may lead to it. These cases of paralysis in children often are the cause of an over-indulgence in sweets and an excess of milk and cod-liver oil which may induce the fatty liver. Gastro-enteritis in children, and in adults carcinoma, infectious disease, phthisis and drug poisoning are the causes of fatty liver. It will be noticed that these conditions are also associated with the increased excretion of acetone.

We can make no sharp distinction between

fatty infiltration and fatty degeneration (Taylor). Pathologic accumulation of fat may occur in the liver without degeneration and without functional impairment of the cells, or it may occur with serious cellular changes. Clinical symptoms are slight if functions remain intact, even with great fat deposit. And that these children were all in apparent good health is evidence that there was not previous fatty degeneration of the liver. The microscopical examination of the liver cells in these cases shows only slight degenerative process and extensive fatty infiltration.

A physiologically fat liver may arise within a few hours after the ingestion of fat, but the extreme fatty liver due to phosphorous poisoning or chloroform similar to the liver in these cases does not develop for two or three days. So it seems evident that in these cases there was nothing in the anesthetic or the operation to produce the extreme fatty infiltration of the viscera. At least, we have proven that in animals the administration of acetone does not produce fatty liver unless it is prolonged for some time.

It is probable, however, that the fatty liver in these cases was not a sufficient cause of death, but that it was only associated, as is the acetonuria, with an acid intoxication which may, through general toxemia, or an excessive process of oxidation in the blood causing lack of oxygen, have been fatal. In clinically healthy children, such as these appeared to be, the fatty liver is rare. Wollstein in 390 autopsies in children found fatty liver in 133 cases (28%) and an extremely fatty liver in 39 cases (10%). These conditions, however, were noticed especially in cases of suppurative inflammation, pneumonia, and intestinal disease.

The adipose tissue of the body furnishes the fat in the liver. This has been proven by fattening animals for some time on mutton fat, and then producing a fatty liver by the administration of phlorizin, and observing that the acute infiltration of the liver consisted of mutton fat (Rosenfeld). In starved chickens, phosphorus does not cause fatty liver. So it may be that the disturbed metabolism of these cases is sufficient to cause an intoxication similar to that of phosphorus, and, occurring in these individuals with very extensive fatty deposits in the muscles and subcutaneous tissues, has a great store of fat to draw upon to cause the hepatic deposits. Although the presence of these primary intestinal poisons has not been shown, it is probable that they do exist and are the cause of the formation of the acetone bodies (Kraus). In phosphorus poisoning there is a marked increase of acetone excretion, due to the destruction of fat tissue, and this is probably true of acetone excretion after chloroform and other forms of narcosis (Schwarz).

The first two of these cases died in October, 1903, and the odor of the breath in them aroused the suspicion of acetonemia. Since the first of November, 425 surgical cases have been admitted to the wards of the Children's Hospital, and over

300 of them have been tested for acetonuria. Acetone has only been found present in the urine in 11 cases, and it has always been associated with other more or less severe symptoms.

The urine was tested in these cases by the standard tests for acetone and diacetic acid. Five cubic centimeters of urine, a crystal of sodium nitro-prussiate and a sufficient quantity of sodium hydrate to make it strongly alkaline were shaken up in a test-tube. The addition of glacial acetic acid to this gives a purple color to the foam if acetone is present. The urine was tested for diacetic acid by the addition of the strong aqueous solution of ferric chloride. A Burgundy red color shows its presence. This test for acetone is a modification of Legal's, and is at least more delicate than Gerhardt's, Le Nobel's or the sulphuric acid tests. Of course, this is not by any means a quantitative method, but it nevertheless allows one to distinguish between the presence of a trace and a considerable excess. A slight modification was introduced by one of the internes which made it even more delicate. A piece of bibulous paper, dipped in the foam and then dried, showed the characteristic purple color more delicately in the presence of quite small amounts of acetone. The more difficult quantitative tests were not practical in the routine of hospital work. In only one case was a quantitative test of the urine made.

The presence of acetone in the urine of ordinary cases of *diabetes* was first recognized by Petters in 1857, and soon after this Kaulisch described the clinical picture known as acetonemia. Many physiologists have demonstrated the presence of acetone normally in the urine. An excretion in the urine of 0.02 gm. per twenty-four hours is physiological; but under pathological conditions this may be enormously increased. The lungs excrete more acetone than the kidneys (Gelmüyden, Schwarz, L. Müller), but there is only a very small amount carried off in the feces.

There are various conditions, mostly associated with alimentation, in which acetonuria has been noted. First, in diabetes, especially of long duration, acetone is generally present in the urine. In starvation, inanition and the cachexia of malignant growths it is not uncommon, and with but few exceptions, it occurs in pregnancy with dead fetus. In peritonitis, where there is often auto-intoxication, and in the fevers of acute infectious diseases, sepsis and pneumonia, acetonuria occurs. In various nervous lesions and mental disease there is often a digestive disturbance which results in acetonuria. The excretion of acetone has been much increased artificially by alimentation, by drugs as phlorizin, and in chronic morphinism; in experiment by extirpation of the solar plexus or pancreas, and after general anesthesia.

Not even with the recognition of an excess of acetone excreted in the urine under these pathological conditions, do we have sufficient evidence of the existence of acetonemia. It is far from sure if acetone is really the toxic substance in the blood as Kussmaul thought; it is more probable

that it is some of the various fatty acids from which acetone is obtained. Acetone may be in excess in the blood without producing marked symptoms or anatomical lesions. Considerable quantities can be exhibited with no harmful effects and very little increase in its secretion (von Jaksch, F. Müller, Kussmaul, Frerichs and Schwarz). We have injected, both intra-peritoneally and subcutaneously, large amounts into guinea pigs without causing anatomical lesions, unless the administration was prolonged for many days.

It is probable, as Naunyn says, that acetone *per se* is practically harmless; that the so-called picture of acetonemia is an acid intoxication or an acidosis due to the presence of diacetic and β -oxybutyric acids, and also probably lactic and various volatile fatty acids. At least, up to within the last two or three years the importance of acetone as a poison in the system has been overestimated. β -oxybutyric acid, the principal one of this group, is harmful only through its acid property of removing alkali from the body tissue. It may remove not only the fixed alkalis but also the bases of the proteids (Magnus-Levy) and thus destroy the efficiency of tissue very needful to life. However this may be, it is evident that the quantitative estimate of the acetone excretion is less important than the total acidity of the urine.

In one case of our series, the quantitative estimation of the acetone in the urine was made by Dr. Joslin. This was a child of eight years, dying twenty hours after the operation of fasciotomy. The total excretion of acetone for twenty-four hours was shown to be approximately 142 mgm. which is considerably more than normal. In cases of diabetes of long duration the daily excretion of acetone during the state of coma often reaches 100,000 mgm., but it may be true that the diet favors the secretion, and the accommodation of the system becomes gradually accustomed to the effects of this great formation of acetone.

Joslin has shown that the ingestion of 100 gm. of oleic acid will cause in a healthy man an excretion of over 1,700 mgm. of acetone in twenty-four hours without producing any other symptoms. He did not administer different amounts of the oleic acid to show whether the excretion of acetone was directly dependent on the amount ingested, but he did observe that those fatty acids that were only partially absorbed produced much less acetonuria. In these experiments the acetonuria was probably the direct result of fatty metabolism of the ingested fats taking place in the intestine, which, though of sudden inception and resulting in the formation of very large quantities of acetone, did not compare in its seriousness with a similar process taking place in the body fats of the liver and muscles.

The comparatively small amount of acetone excreted in one of our fatal cases shows that the quantitative estimation of the acetone excretion does not give a true index of the severity of the

condition, and that the presence of any considerable amount of acetone in the urine should be considered only as a probable sign of a more or less serious acid intoxication.

Now in these eleven cases showing acetonuria, there were only two in which diacetic acid was not found in the urine, and in one of these the test was not made for it. This is in accord with the idea that diaceturia and acetonuria cannot be separated (Lorenz). In most severe disturbances both are seen or they may alternate. This was clearly evident in case five in which the quantities of acetone and diacetic acid varied alternately from day to day. Gerhardt was the first to recognize this association of diacetic acid with acetone in the urine, which leads to the explanation of the origin of the acetone, from the acids, diacetic and β -oxybutyric.

Acetone has generally been said to have its source in the destruction of proteids, such as takes place in fevers and starvation. Though tissue destruction is conceded to be essential, it is not evident that acetone is the result of the breaking down of proteids (von Noorden, Honigmann). Acetone excretion may be diminished by a diet of proteids. Waldvogel administered to fasting men a diet of pure proteids and one of pure carbohydrates, but in neither case did acetonuria occur. Stadelmann and Minkowski showed an exception to this in the case of diabetes where, due to a sudden and complete administration of a meat diet there was a diminution of carbon dioxide in the blood, making it less alkaline, and coma and acetonuria resulted. A diet of proteids does not *increase* and a *diet of carbohydrates generally diminishes* the production of acetone. A reduced proteid diet causes the excretion of considerable acetone as a result of the destruction of body tissue (Schwarz). And in cases of poor nutrition we know that the fats of the body are drawn upon first. Bernert found acetone present eleven times in ninety-four cases of typhoid fever, but he says after the intestinal disturbance with the high fever had subsided, it was due to subnutrition and disappeared when the proteids were fed freely. Chemically there has never been success in producing acetone from proteid, but as it has been formed from gelatin (Blumenthal and Neuberg) it is probable that it may be obtained from proteid. It is, however, readily obtained from fats, and has been obtained from carbohydrates.

A constant excessive fat diet causes a large increase in the excretion of acetone (Waldvogel). When fats were fed to starving people acetonuria developed; but the subcutaneous injection of fat did not cause this result. So acetonuria is probably dependent on a fat metabolism generally associated with intestinal disturbance. That it can be induced artificially without intestinal disturbance is very true. In the normal person with full diet the addition of fat causes only a very slight increase of acetone excretion (Schwarz). A diet largely of fats,

especially the volatile fatty acids of butter, results in acetonuria (Schwarz, Satta, Joslin). In the experiments of Joslin the ingestion of large amounts of sodium bicarbonate greatly increased the acetone excretion, but the reason for this is not known, and it is opposed to the statement of von Noorden that alkalis frequently decreased the excretion of acetone.

The formation of acetone is due to fatty metabolism with the formation of free fatty acids, and is largely dependent on a reduction of the carbohydrates and proteid diet and general sub-nutrition. In most of the pathological conditions in which acetonuria is recognized clinically there is a sub-nutrition, or an intestinal disturbance which results in a defective nutrition of the body, and it is associated with waste of body tissue. It may be the body fat or the food fat which is the source of the acetone, but the immediate cause is the disturbance of the metabolism.

The recognition of the occurrence of fatalities after narcosis, in which acetone and its allied compounds have been present in the urine, is by no means new. In 1894 Becker, from the Bonn clinic, reported three fatalities, after operation on diabetic subjects, in which the clinical conditions were quite similar to those observed in these and other isolated cases. The clinical symptoms in the three cases of Becker were so similar as to suggest some common cause, the condition noticed beginning as a slight but increasing dullness after the anesthetic, and after twenty-four to forty-eight hours the occurrence of a stupor, passing into a complete unconsciousness and coma, with a lack of autopsy findings. All clinical conditions seemed to him to point to some form of poisoning through acetone, and his experience with these led him to investigate the literature, where he found reported twelve fatalities, under like conditions. These were all associated with marked acetonuria, but were in cases known at the time, or after found out to have been, diabetic subjects. The condition of the patients before operation had not been such as to give reason for apprehension for the outcome, nor were the operations in themselves such as to give cause for alarm. It led him to formulate, from his observations, the statement that diabetic patients, who, even up to the time of operation, had shown no unusual symptoms under ordinary conditions, were, through some form of disturbance of metabolism, liable to pass from the narcosis into a stupor, and finally into a fatal coma, and that this is due to a condition which, at the time, was known as acetonuria, the term then used to indicate the manifestation of the presence of acetone in the secretions, particularly in the urine. There were no pathological findings in his cases which came to autopsy, except that in some there were lesions in the organs, similar to those found in cases of severe phosphorous poisoning. His supposition was that in some way the effect of the anesthetic was such that this condition resulted. This series was, however, collected from the

literature in which they had been reported as unusual fatalities, and they had not been so thoroughly investigated as would have been the case, had special interest been brought to bear on them. Becker's three cases of the series were distributed over a period of a number of months.

Brewer, in *Annals of Surgery*, October, 1902, reported a fatal case of acetonuria occurring after an operation for appendicitis. The case had presented nothing unusual in the condition previous to the operation to intimate an unusual result, and the operation itself offered nothing of remarkable interest. In clinical symptoms, the condition following the operation closely resembled the cases of Becker, and those of the series here reported, the acetone being detected both in the urine and breath.

In 1903 Dr. Leonard Guthrie published an article on "Fatal Effect of Chloroform in Children Suffering from a Peculiar Condition of Fatty Liver," in which he describes exactly the clinical condition found in the cases here reported. In addition to his own, he collected a large number of cases of fatal results after chloroform, in which at autopsy there was found the same peculiar condition of fatty liver. In a number of cases the symptoms were what he called the "cerebral type," and these had so close a resemblance to the cases here under observation that a portion of his description could almost be taken as an illustration of some of those here reported. In it he says: "After recovery from the immediate effects of the chloroform, there was usually an interval of a few hours, at the end of which the child would begin to utter piercing cries at intervals, disturbing the whole building by its shrieks, grinding its teeth, tossing, struggling, and requiring constant attention lest dressings should be torn off or fractured bones displaced. The pupils were often dilated, sometimes unequally, the face being flushed or pale, with a look of wild terror and anxiety. Consciousness was sometimes lost early and never regained; sometimes there were intervals in which the child would be dull and apathetic, but would answer rationally when addressed, and usually deny being in pain. Vomiting was a marked feature in all but one case. It was copious, frequent and persistent, and the vomited matter sooner or later almost exactly resembled the dregs of beef-tea. In one case it was described as 'bloody,' and in another as 'coffee-ground.' Headache seemed to be absent in all cases. Pyrexia, was not a marked feature, but in some instances it rose to 104° or more. The screaming at length subsided, perhaps lulled by the morphia and other sedatives given, the vomiting became less frequent, unconsciousness led to coma, and the respiration and the pulse gradually failed, the latter becoming often imperceptible before breathing ceased. In some the vomiting and the restlessness continued almost till the end, and death occurred, with suddenness, from heart failure." In his cases the urine was examined and note made of albumin. It was reported as never suppressed,

but no mention was made of the presence of acetone or its allied compounds, or of any attempt to find it. He considers at length the probable causes of the condition, among them fat embolism and toxemic poisoning. The question of carbolic acid poisoning was considered, but this cause was found to be impossible. These cases seem to have particular interest, inasmuch as they so closely resemble, clinically, this series here reported, and also that in the pathological findings they so closely resemble two of this series. In others of his, as in this series, also, there was no pathological condition found. All of his reported cases occurred after a narcosis by chloroform, but it must be remembered that this was the agent used by him.

The conclusions which were given by Guthrie as to the cause of death in his cases were:

(1) In all but two cases due to auto-intoxication.

(2) Fatty condition of the liver, and therefore functional disturbance of that organ resulted before operation.

(3) Chloroform and operation shock combined aggravated the condition already present, and led to the formation of toxic alkaloids which the kidneys were unable to eliminate.

Guthrie, in support of the theory that it was due to fatty infiltration of the liver, quotes results by a number of men who experimented upon the effect of chloroform on animals for a greater or less period. These researches appeared to prove that long continued administration of chloroform produced fatty changes in body in general, and in the liver particularly. It was also found that the fat which appeared in the organs a few hours after the inhalation of chloroform did not disappear for several days.

With the exception of Becker's three cases which occurred in diabetics, and Brewer's isolated case, no mention is made of the seriousness of this condition, independent of its association with diabetes, but the cases were reported as being of interest, as they were fatalities after operation, under conditions which had given no cause for alarm. The phenomenon of acid auto-intoxication had then been thoroughly recognized, other than in its association with diabetes. The condition had been studied by von Jacksch, Lorenz, Baginsky and others. Baginsky, in 1888, from a thorough study of the subject, reported that acetone is frequently present in fever patients, the amount corresponding to the height of the fever; that it also came from disturbances of the metabolism, and could be induced by the cutting off of some forms of nourishment, as shown both in experiments and bedside observations.

Its occurrence after an anesthetic, and in individuals in whom there had been, up to the time of the operation, no unfavorable symptoms, although diabetic, led Becker to investigate as to whether this might have resulted from the use of the anesthetic, and whether the kind of anesthetic made a difference in the production of acetone, and he made between fourteen and

fifteen hundred observations, with narcosis by different agents, with reference to its effect in producing acetone in the urine. It was shown by these, that there usually existed, after a narcosis, for a longer or shorter time, and in healthy people, a distinct amount of acetone in the urine. This occurred independently of the form of anesthetic used, although ether being the usual form of anesthetic used in the Bonn clinic, the larger number of observations were made with this agent. It was also found that acetonuria appeared without being influenced by the length of the narcosis, the kind of disease, or operation, whether bloody or bloodless, or by age in the adult person, or sex. It was found, however, that children were far more susceptible in this respect; that in children, a larger number of cases of acetonuria appeared, and the amount of acetone was greater. The average duration was from two to three days. In some cases, however, it did not disappear until the eighth or ninth day.

Lorenz, in 1890, from his observations, was inclined to attribute it to gastro-intestinal disturbance, and recognized the different forms of allied intoxications. He also pointed out the fact that it was found in those forms of gastro-intestinal disturbance which were secondary to a nervous affection, but also showed that the amount of acetone was much less when the disturbance was of nervous origin. That it could be caused by intense nervous disturbance, as fright, was shown by the well known experiment, in thrusting a dog from a window at a high altitude, as if to throw him out, and the presence of acetone demonstrated in the urine after the conclusion of the experiment.

ETIOLOGY.

The question at once arises as to the cause of the condition in the cases here reported. It is obvious that something incident to entering the hospital induced the condition in some cases, while in other more serious cases it was induced by the operation or something incident to it, and yet it is plain that there must be some deeper underlying cause or causes. A number of conditions are suggestive as having an etiological bearing, and are to be considered.

In such a series of cases the first thought is that the conditions might have resulted from anesthesia, or that they were in some way the remote results of the anesthesia. In analyzing the cases, however, we find that in one of the fatal cases and five of the milder cases the symptoms came on without operation or even anesthesia. One of the fatal cases had taken ether and been operated upon several months before without any ill effects whatever. In one case ether had been given several years before without any ill effect, and ether was given again a few days after the acetone had disappeared from the urine without any ill effect and without any reappearance of the acetone.

It seems fair to conclude, then, that anesthet-

ization alone does not bring on the condition, or at least in a serious form.

Operative interference and operative shock cannot be regarded as the underlying cause, as is evident from the fact just stated, that in five milder and one fatal case the condition came on without operation. Furthermore, in none of these cases were the operations severe. None showed the ordinary symptoms of shock. None lost much blood, and in none was there any extensive injury of the tissues.

Ptomaine poisoning.—The question will be asked, Is not this condition one of ptomaine poisoning? The sudden onset, the nature and the severity of the symptoms, certainly suggest it, but in no case was there any diarrhea, which is scarcely ever absent in ptomaine poisoning if the patient lives long enough. There was no material rise of temperature except in the fatal cases soon before death. In one case the vomiting was slight, though otherwise the symptoms corresponded exactly to those in the other fatal cases. Furthermore, other children in the same ward at the same time and eating the same food have shown no symptoms to suggest ptomaine poisoning. These facts seem to bar out poisoning from this source.

Homesickness is suggested as a cause of the symptoms. The fact that the condition appeared usually about twelve hours after operation, no matter how long the child had been in the hospital before, would not necessarily militate against this suggestion. One patient, however, a boy with acute hip disease, four weeks after entering the hospital, while up and about the ward, was taken sick with vomiting and marked collapse. The vomiting lasted but one day, and acetone persisted for three or four days. In this case homesickness certainly was not a factor. The condition occurred also in other children who had been in the hospital before, who were old enough to know what they had been through, what they had to expect and who seemed absolutely happy. In one of the milder cases, however, a child who spoke no English and who cried constantly really seemed to bring on the condition.

Fright has been suggested as a possible cause. Two children had been in the hospital and knew what they were to expect. In some of the cases dread or homesickness or both did exist, but not to any greater degree than with many other children. The acute fright sometimes incident to taking ether did not seem to be more prominent in any of the cases than in many other children, though in the last fatal case it was quite noticeable.

One condition existing in the hospital at the time of the occurrence of these cases, though fortunately since removed, should be mentioned. Owing to the increased demands of the operative work at the hospital, the recovery ward for children after operation was taken as a service room. It had been the practice to return the patients to the wards immediately after operation. The children in the wards were necessarily

witnesses of the recovery from the intoxication of anesthesia with the noise and incoherent exaggeration of suffering. It is manifest that such a condition of affairs was calculated to increase the nervousness and anxiety of a nervous child and may have served as a contributing cause to the alarming symptoms under consideration.

The confinement of the hospital may possibly be regarded as a factor. Yet it seems that four days' confinement could not possibly have brought on a fatal condition, and it seems unlikely that the conditions would, if caused by confinement, come on after four weeks' stay, while the child was up and walking about the wards.

Change of food.—One very important factor suggests itself, especially in view of what is known in regard to the appearance of acetone in the urine after fasting, especially after cutting off of carbohydrate food. The change in diet on entering the hospital or the loss of appetite incident to it or the starvation and thirst incident to the taking of ether are factors present in every case except one, that of the child in whom the condition developed after four weeks' stay in the hospital. On the other hand it seems inconceivable that symptoms sufficient to cause death should come on so soon as a result of change or diminution in the amount of food, taken except in the presence of some other underlying conditions. In these cases the after effect of ether, the shock, or even the change in conditions and diet may have been sufficient to cause an intestinal disorder with disturbance of metabolism which set free from the excess of fats stored in the liver, muscles and subcutaneous tissues various fatty acids which caused the serious symptoms and were partly changed to acetone and so eliminated.

In looking over the cases one very striking fact appears. Barring out the fatal case of intestinal obstruction, there remain four fatal cases. Three of these had very extensive atrophy of the muscles of the lower extremities resulting from long standing infantile paralysis; the fourth had marked muscular atrophy, the result of confinement of one lower extremity in a plaster of Paris spica bandage for several months. Of the nine cases which recovered two had extensive infantile paralysis, with its consequent muscular atrophy; one had muscular atrophy; one had very acute hip disease associated with marked atrophy of the limb. Thus all the fatal cases and four of the nine cases which recovered had marked and extensive muscular atrophy. It seems a little unreasonable to maintain that two of the remaining five cases had muscular atrophy resulting from rickets, for the consequences of which they were admitted.

As Waldvogel says, acetone is certainly a production of fat, and it appears in abnormal amounts in the urine when the demand for energy must be met by tissue waste and the breaking down of body fats, and when there are large amounts of fat in the intestine. There was not in these cases any excess of fat ingested, but the store of body

fat was in nearly all of them exceptionally great, and though they were apparently well, it is evident that most of them were afflicted with some chronic wasting trouble, which made them more susceptible to a slight change of conditions, or this demand for energy which could only be met by breaking up the store of fats into nutritious and at the same time toxic products.

At least, it remains for experiment to prove whether an extensive fatty degeneration of the muscles may not take away the metabolic efficiency of so much muscle tissue, and leave a deposit of fat to co-operate with the fatty liver in the cause of the serious results associated with acetonuria.

There are many conditions in these cases which present questions of special interest. A number of similar cases have been presented by different observers, and cases having so close a resemblance that they must be considered to be of like etiology. This group of cases, however, has a bearing of special interest, in that it represents a series, occurring within a comparatively short time, under different conditions, but alike in clinical manifestations. In looking back over the history of the hospital, a number of cases are brought to mind, by all connected with the institution, which, clinically, so closely resemble these as to be considered by them as undoubtedly of like origin; but in no instance, however, have they occurred in such a way that they could be regarded as a series, or grouped as regards their relation to time or etiology.

No attempt is made in the presentation of this series to formulate a definite theory as to their causation. At the present time, not enough is known about the physiological condition which results in the appearance of acetone and its allied compounds, to formulate a complete working theory. In the present state of knowledge, one can offer only the suggestions which have been given in the clinical presentation of the paper, and which point to the different conditions under which they have made their appearance. Neither is the opinion offered that the condition as seen, or that the fatal results, are due to an intoxication with acetone from its presence in the blood, for it is known that the same symptoms are present when other allied compounds are found in the blood, urine and breath. It may be that the poison is not particularly injurious when acting on proteids in circulation, but becomes so when demanding its affinity from that which is fixed in the cells.

The object is to report, (1) The occurrence of these cases which give a distinct clinical evidence of being a form of acute intoxication, due in some way to a disturbance of the metabolism, and which has, among its peculiar manifestations, the presence of acetone and its allied compounds in the excreta. (2) The occurrence of these cases in a series or group, comprising twelve or more, in the course of a comparatively short period. (3) Their apparent occurrence under many conditions, but particularly fatal when following the administration of an anesthetic, and under cir-

cumstances which gave no reason to expect an untoward result.

This series of cases establishes certain facts. It can be stated positively that the symptoms are not the result of anesthesia, operation or shock, unless in the presence of certain underlying causes still undetermined. It is merely suggestive to say that most of the cases have occurred in children with a high-strung, nervous temperament. In such children the confinement, the changed habits, the changes in diet, the homesickness, the dread of operation, the anesthesia, and the operation itself, may lead to changes in metabolism which have not hitherto been taken into account. These changes may lead to serious or fatal consequences unless all the organs, including the muscular system, are not only sound, but performing their functions normally.

The fact that acetone is found in such very much larger quantities under various conditions without producing symptoms, and the fact that experimentally acetone can be injected into the blood of animals without producing symptoms, as has been shown above, prove that the presence of acetone is not the cause of the symptoms in the series of cases reported.

The fact that acetone had been very rarely found in the urine of the children in the hospital, except when there were the symptoms described in the cases reported, suggests that the toxic agent may be produced by the same conditions which cause the appearance of acetone in the urine, and that the appearance of acetone in the urine, in quantity sufficient to give the ordinary clinical reaction, is to be regarded as an indication of serious and possibly dangerous disturbance of metabolism.

In the cases reported, it is plain that the ordinary cardiac stimulants, brandy, strychnine, digitalis, proved useless. Acting on the assumption that the symptoms were due to an acid auto-intoxication, bicarbonate of soda was given to all the cases after acetone was found in the urine. Some of these cases died, though even to them the soda seemed to give sometimes temporary relief. Several of the children who recovered were seriously sick before the soda was given. So far as can be judged from the limited number of cases, bicarbonate of soda or some other alkali is the only remedy which promises to be of any decided benefit, and should be given in large amounts by mouth and in enemata. In some instances, the infusion of large quantities of salt solution under the skin or into the veins seemed to be of decided, though usually of only temporary, benefit.

This report is given with the hope that it may stimulate further and more careful observation of cases, for although one is not able to point to the definite origin of the disturbance, yet one may, in conclusion, give certain suggestions in regard to it which are gathered from a study of the different conditions under which the phenomena occurred. These suggestions are based upon circumstantial evidence, but point to the

need of paying more attention to certain conditions in the hospital care of children, both operative and nonoperative. These are: First, that greater attention should be paid to temperament, and to the conditions which influence it, realizing the anxiety and fright, with the accompanying physical disturbances, caused by entering a hospital, remembering that these are particularly noticeable in children of high-strung and apprehensive temperament. Second, that the absence of any gross evidence of a pathological condition, as shown by heart murmurs, evident changes in the lungs and kidneys, or abdominal tumor, may not constitute immunity from the danger of acetonemia, and possible death after operation. Third, decided caution should be paid to those cases in which there may be reason to suspect a fatty condition of the liver. The reported experience of many writers, as well as that derived from those cases here reported, warrant this view. Fourth, it is more than probable that unusual care should be exercised in those cases showing extensive degenerative change, and, particularly, degenerative muscular change, such as is seen in extensive infantile paralysis. The large percentage of cases in this series, which present this condition, seems to indicate that special stress should be laid on this point.

CASE I. L. R., a boy, aged ten years, of Roxbury, entered the Children's Hospital on Oct. 6, 1903.

Excepting for measles, the child had always been well until an attack of severe infantile paralysis nine months before. He had in part recovered the power to use his arms, but there remained paralysis of some muscles of the back and abdomen, and complete paralysis with marked atrophy of all the thigh and leg muscles on each side. The urine was acid, clear in color, and free from sugar and albumen. The pulse was 100; the temperature 99.5.

Four days after entrance, up to which time he had been perfectly well, the boy began to vomit persistently. The bowels had moved freely. The vomitus was watery, of light greenish color, not fecal. The abdomen was soft, and not distended. This lasted for two days, and on the third, the condition was improved, and the vomiting nearly ceased. Toward evening the pulse became very weak and rose from 95 to 150. The temperature, which had been normal, shot up to 105.8. There was no response to stimulation. The child became unconscious, and died in about twelve hours.

Autopsy, Oct. 11, 1903. Dr. W. R. Brinckerhoff.

Body of a well developed, fairly nourished white boy, twelve years of age. Rigor mortis present. Pupils equal, of normal size. Subcutaneous fat 1 cm.

Peritoneal cavity: Surface smooth and glistening. Two greenish black coils of jejunum lie on the left side of the cavity at the level of the umbilicus. These two coils, respectively each 10 cm. long, are somewhat distended, and in the upper end of each coil is an intussusception. The invaginated portion of the gut is 1.5 cm. in diameter; the greenish black distended portions being 3 to 4 cm. There is not fibrinous exudate in this region. Appendix normal. Mesenteric lymph nodes normal. Diaphragm at normal height.

Pleural cavities normal.

Pericardium contains a few centimeters of clear,

straw-colored fluid. The epicardium on the posterior aspect near the auricular-ventricular groove presents a few 1-mm., cherry-red spots. Other valves and cavities normal. Myocardium normal in color and consistency. Heart weight, 120 gms.

Lungs: Air containing, crepitant and pink.

Spleen: Weight 40 gms. Consistence flabby; color light red.

Pancreas normal.

Liver: Weight 1,130 gms. Light yellowish brown; markings distinct. Gall bladder contains much bile.

Gastro-intestinal tract: Mucosa presents no ulcerations at any point.

Stomach contains bile, stained fluid and the mucosa on the summit of the folds is somewhat injected. Duodenum and first portion of jejunum normal. Twelve centimeters of the intestine above the first intussusception contains a greenish black tenacious material (changed blood). The distended bowel below each intussusception is filled with the same material, which extends to a point 20 cm. or more below. The remaining portion of the intestine contains rather fluid, normal contents. The mesenteric veins tributary to the intussusception are normal.

Kidneys: Weight 130 gms. Pale, reddish brown; capsule strips readily leaving a smooth surface. On section cortex is pale brown; markings distinct. Adrenals normal. Bladder normal.

Cranium not opened. Cord: Consistency normal throughout. On section in the lumbar region is a pin-point, reddish area in one anterior horn.

Anatomical diagnoses. — Double intussusception of jejunum. Punctate ecchymoses in epicardium. Anterior poliomyelitis (clinical).

CASE II. M. J. B., a girl, aged eight years, of Fitchburg, entered the Children's Hospital on Oct. 6, 1903. She had an extensive infantile paralysis involving most of the muscles of both lower extremities which came on five years before, and necessitated the use of crutches in walking.

On several occasions during the preceding year, the child had several very acute attacks of severe and persistent vomiting, accompanied by marked prostration and rapid pulse, and the physician in attendance stated that the condition of the child at the time of these attacks, very closely resembled that described to him as existing on the first day of the fatal one. The attacks were severe enough to cause considerable alarm as to their outcome.

The child seemed rather delicate, but was well nourished, and physical examination showed nothing abnormal aside from the infantile paralysis already mentioned. The urine was acid, with specific gravity of 1,020, free from albumin and sugar. The pulse was 100; the temperature was 100.

Four days after entrance the child was etherized, and three tendons were transplanted. The loss of blood was insignificant, though the operation and the application of the plaster bandages kept the child under ether about an hour and a half. The child took ether rather badly, being rather blue, and having a rapid pulse, but at no time during or immediately after the operation, was there ground for anxiety in regard to the condition.

During the recovery from ether the child vomited as usual, and then seemed comfortable, and in good condition till twelve hours later. She then suddenly began to vomit persistently. The vomitus was at first watery and light green in color, but later dark. The pulse became very rapid, jumping to about 190, the temperature rose to 106. The vomiting lasted for about thirty hours. The child, who had been restless, gradually sunk into a delirious stupor, and

died forty-two hours after the operation, thirty hours after the vomiting began.

In this case no test was made of the urine for acetone or diacetic acid. It was clearly recalled afterward, however, that the peculiar odor of the breath was very strong. Stimulation had no effect.

Autopsy, Oct 12. Dr. H. A. Christian.

Body of a well-developed, well-nourished female child.

Pleural cavities are free from adhesions and contain a normal amount of fluid.

Pericardial cavity likewise normal. Heart: Weight 100 gms. Scattered over the surface of the left ventricle are about one dozen subpericardial ecchymoses from 1 to 2 mm. in diameter. Heart of normal appearance throughout.

Lungs: In the anterior portion of the mediastinum in the loose areola tissue there are numerous small gas bubbles, and on the surface of the anterior portion of the right lung there are several subpleural areas about 3 mm. in diameter, and 1 cm. in length, which are distended with gas. These suggest in course the distribution of some of the subpleural lymphatics. About these, as also about the gas bubbles in the mediastinum, there is no evidence of inflammatory reaction. The surface of both lungs is smooth. The lungs anteriorly are grayish pink; posteriorly darker red; are everywhere air containing. Anteriorly they are more evidently crepitant, though there is a distinct crepitation posteriorly. The bronchial mucosa is very slightly congested; there is no apparent exudation. Bronchial lymph nodes show no change.

The thymus is quite large, coming well down towards the base of the heart. However, it is not very thick, and there is no evidence of any pressure on the deeper tissues from this.

Abdomen: Subcutaneous fat well developed. Peritoneal cavity, particularly on the left side, contains a considerable amount of dark, sour-smelling fluid. This is seen to originate from the stomach, and greater curvature in the region of the spleen is completely digested away. (This is apparently a post-mortem change.)

Vermiform appendix normal, free from adhesions. Mesenteric lymph nodes show slight general enlargement. The intestines are contracted. Peritoneal surface everywhere smooth and glistening.

Spleen is of normal size; is smooth. Its surface is much discolored in places by the action of intestinal gas; it is soft and flabby; cut surface is grayish red. Malpighian bodies are not made out with distinctness. There is a moderate increase in the pulp. The spleen was exposed to the action of the gastric juice, and some of the softening may be due to this.

The stomach, with the exception of the area of self-digestion, appears normal, as do also the intestines.

Pancreas is normal.

Liver: Weight 510 gms. Surface is smooth and irregularly mottled with yellowish areas from 3 to 5 cm. in diameter, scattered over a reddish brown surface. Cross section is yellowish brown in color. Markings are indistinct. There are a few very small areas of a darker reddish color.

Kidneys: Of normal size; capsule strips readily leaving a smooth surface. From cut surface cortex appears to be of normal thickness, is greyish in color; glomeruli are visible as small reddish points; pyramids are redder in color, and their markings are quite distinct. The contrast between the two is quite sharp.

Adrenal glands normal. Bladder normal. Aorta normal. Genitalia normal.

Brain: Weight 1,360 gms. The meninges everywhere appear normal. There is no evidence of exu-

dation nor chronic thickening. There is no apparent increase of subpial fluid. The ventricles do not contain an increased amount of fluid and the ependyma is smooth. The choroid plexus are also congested, as are also the meningeal blood vessels. Section of the brain in the usual way reveals no lesion. There is no evidence, either by flattening of the convolutions, or pressure ring on the cerebellum, of increased cerebral pressure.

Spinal cord shows, particularly in the lower lumbar region, an atrophy of the anterior horn on one side. The blood vessels lie on the surface of the cord, are much congested. No other lesion found.

Anatomical diagnosis. — Chronic poliomyelitis, anterior.

Cultures. — One culture spleen — bacillus of colon group. Two culture liver — very slight growth of same.

None of three show any staphylococci or streptococci.

Histology. — Spinal cord: The anterior horn of one side shows marked atrophy with almost complete disappearance of the anterior nerve cells.

Anterior mediastinum: The loose tissues of the anterior mediastinum show an acute inflammatory process of slight degree, evinced by the presence of polynuclear leucocytes in the tissue, adjacent to the gas cysts described in the gross. Sections of this tissue stained by Gram-Weigert method show no micro-organisms. Sections of liver, spleen, kidneys and lungs are negative. From these sections it is evident that to the marked process in the cord there is added some slight acute inflammatory process in the anterior mediastinum.

CASE III. E. W., a girl, aged five and a half years, living in Fall River, entered the Children's Hospital Nov. 16, 1903. When a year and a half old she had a severe attack of infantile paralysis, which kept her in bed some time with a high fever. A very general paralysis of the muscles of the thighs and legs persisted with contracted fasciæ, causing permanent flexion of the thighs, for the section of which she entered the hospital. The child has been about on crutches for about a year. Otherwise, the history was unimportant and the physical condition showed nothing abnormal, except, as was afterwards found, a slight trace of diacetic acid in the urine. The pulse was 100; temperature 99.

On the day after entrance, early in the morning, fasciæ at the upper outer part of both thighs were cut by open incision. The operation was very short, and very little ether was used. A double plaster of Paris spica bandage was applied.

After operation the patient seemed comfortable and passed a little urine. At 11 P.M., about thirteen hours after operation, she vomited a small amount of dark-brown thin fluid, and during the next ten hours vomited similar material several times. She slept in all three hours during this time. While awake she was very restless and noisy.

Twenty-four hours after operation the condition of the patient was very poor. The extremities were cold, the pulse was weak and rapid, the expression was anxious. The peculiar sweet odor of the breath became noticeable during the morning. At noon, twenty-six hours after operation, five ounces of urine were passed. This contained large traces of acetone and diacetic acid.

The child had not responded well to stimulation by strychnine given subcutaneously. A pint of salt solution was then given intravenously, after which the child became more quiet, though the pulse did not improve. Thirty hours after operation the child

began to be delirious. The pulse became scarcely perceptible at the wrist. Attempt to move the bowels by enemata and cathartics were without avail, except for one small constipated movement.

Thirty-six hours after operation, twelve ounces of two-percent bicarbonate of soda solution was given intravenously, and also under each breast. Throughout this time there had been nausea, and occasional vomiting. Three hours later the child had an hour's sleep, and the pulse became more regular.

On the second morning after operation, the child became more dull and apathetic. She gradually lost consciousness. Coma came on. The odor of the breath became more and more marked as the condition became worse. The alæ nasi were working vigorously as though the child suffered from lack of air, and in the last few hours cyanosis was marked. The temperature after the operation remained about 102.

Autopsy, Nov. 19, 1903.

Body of female child, five and a half years of age. Well developed and nourished. No rigor mortis. No lividity. Pupils equal and normal, 3 mm. No edema. About 2 cm. subcutaneous fat.

Peritoneal cavity: No fluid nor adhesions. Serous membrane glistening and not congested. In mesentery of large intestine are numerous scattered small hemorrhagic points. Appendix is retrocecal, 7 cm. long. Mesenteric lymph nodes are not enlarged. Diaphragm at fifth rib or either side.

Pleural cavities negative. Pericardial cavity negative.

Heart: Fresh examination shows moderate amount of fat. Tissue is red, firm. Ventricles contracted, and the organ contains only a little fluid blood.

Lungs: Soft and crepitant. On section there is considerable white frothy exudation. The tissue is rich dark red, moist, crepitant everywhere. The bronchi are slightly injected. Bronchial lymph nodes are negative.

Spleen is firm and holds its shape well. Surface is dark red. The tissue is dark red, fairly firm, and only a moderate amount of pulp can be scraped off with the knife edge.

Gastro-intestinal tract shows nothing abnormal. The vessels are not congested. It contains little mucus, and a small amount of yellow feces. Pancreas is pale yellow and firm.

Liver: Surface smooth and pale yellow, and, in places, of reddish color. Stellate veins are in places well marked. Cut surface is pale yellow, oily and homogeneous. There are, in places, small, evenly distributed yellow areas corresponding with the centers of the lobules. The periphery of the lobules is pale red. The tissue is rather soft and friable. Fresh examination shows very marked fatty infiltration. Gall bladder moderately distended with brown bile.

Kidneys: Normal size. Capsule is thin and strips easily. Surface smooth and very pale yellow. In the section the lobulations are noticed, the cortex is very pale, yellowish, with some minute red points. The sub-cortical tissue is very dark red, and in marked contrast to the very pale red of the rest of the pyramids. Pelvis and ureters are normal. Fresh examination shows marked fatty infiltrations. Adrenals normal.

Bladder contains about 2 oz. of clear, straw-colored urine, which was drawn off in clean bottle with about 1 dr. of chloroform. Wall of bladder is negative.

Genital organs negative.

Aorta negative.

Organs of neck: Trachea and larynx are slightly congested. Thyroid normal. Thymus present, not large.

Brain: Calvarium thin, almost no diploe. Dura

only slightly adherent to bone. Slight congestion of meningeal vessels. No increase of fluid. Convulsions normal. Ventricles normal. Ependyma normal. Tissue not very moist.

Middle ears negative. Bone marrow red and soft.

Cultures from heart, spleen and liver were sterile. Cultures from kidney showed the colon bacillus.

DIAGNOSIS.

Microscopical description.—Heart negative. Lung and spleen show considerable acute congestion. Stomach negative. Small intestine shows many eosinophilic cells in the glandular epithelium. Pancreas negative.

Liver: Cystoplasm of liver cells is almost wholly replaced by vacuoles, both large and small, and what little there is stains poorly. The nuclei of the liver cells stain well. Many of the cells are phagocystic. Sections stained with Scharlach show very much fat.

Kidneys: The epithelium of the convoluted and descending tubules is swollen, vacuolated, and poorly stained. The nuclei stain well. Sections stained with Scharlach show considerable fat.

Sections of the thyroid, thymus, brain and pituitary body are negative.

Anatomical diagnosis.—Acetonuria. Very marked fatty infiltration of liver. Moderate fatty infiltration of heart and kidneys. Fatty degeneration of muscles of lower limbs.

CASE IV. M. S., a girl, aged eight years, of Boston, entered the Children's Hospital on Dec. 17, 1903. She had an extensive infantile paralysis of muscles of the right lower extremity, of seven years' standing. Otherwise, the child was in good physical condition. She entered at noon, was given dinner, and put to bed. In the afternoon she began to vomit, and continued to do so at intervals during the next forty-eight hours. On the evening of entrance the odor of acetone was detected in the breath. Next morning acetone and diacetic acid were present in the urine, the reaction being marked. The specific gravity was 1,020, sugar was absent; there was the slightest possible trace of albumin. During this time the patient had been restless, but on the second day after entrance, periods of stupor, from which the child could be easily roused, alternated with periods of restlessness, in which she cried out suddenly. A large part of the time she lay with her eyes open with a vacant stare.

Immediately after the onset of the vomiting nothing but hot water had been allowed by mouth. Enemata of salt solution and beef juice had been given every six hours. Peptonized milk and sodium bromide were later given by enema. Bicarbonate of soda was given in drachm doses every three hours from the day after entrance, at first by rectum, but after a week by mouth. The child slept nearly the entire third night, but on the following morning the vomiting began again and continued at intervals for the next three days, when it ceased entirely except for several times two days later. The vomitus was always watery and light green in color.

Aside from the facts mentioned, there were no other symptoms, and nothing abnormal could be found on physical examination. The abdomen was soft and not distended. The bowels moved freely. The temperature varied but little from 99. The pulse ranged about 100. The acetone and diacetic acid disappeared from the urine at the end of two weeks. The general condition had improved decidedly. Three weeks after entrance, the Achilles tendon was divided subcutaneously under local cocaine anesthesia, and a plaster bandage was applied. Ten days later this was changed under nitrous oxide anesthesia. The next

day the over-corrected position of the foot caused considerable pain, but nothing else.

Twenty days later the child vomited again several times, and acetone again appeared in the urine, but only for a day. She was discharged well nine weeks after entrance.

CASE V. M. McL., a girl, aged six years, of Cambridge, entered the Children's Hospital on Dec. 17, 1903. When a year and a half old she had an attack of infantile paralysis, which had left her unable to walk. Except for fair power in the psoas and glutie, there was almost complete paralysis and atrophy of the muscles of both thighs and legs. The pulse was 110; the temperature 99. The urine was acid, specific gravity 1,025, free from albumin and sugar.

On the morning after entrance the child vomited once. The vomitus was watery, of light green color. Acetone and diacetic acid were not then present in the urine, but on the following morning both were present, and the child was very dull. There were no fever or other symptoms. Bicarbonate of soda was given in half-drachm doses every two hours. The condition slowly but steadily improved. After twelve days the acetone and diacetic acid disappeared from the urine. The bicarbonate of soda was continued during this time. After a few days the urine became alkaline. The after history was uneventful. Splints were fitted. The child was discharged five weeks later.

CASE VI. M. G., a girl, age four years, residence, Roxbury, was admitted to the Children's Hospital Dec. 26, 1903. Previous history was negative except for an attack two years ago. This illness lasted three days. The prominent features were absolute constipation for three days and vomiting. This was finally relieved by enemata.

Dec. 21, 1903, vomiting again began. Constipation had existed for some time previously. From Dec. 21 to Dec. 26 there was only one defecation. The vomiting occurred during this time frequently. Nothing, even water, was retained. Patient had become weak. Enemata had failed to relieve the condition of constipation. There was great thirst.

Dec. 26. On examination the patient was found to be weak. The face was somewhat haggard and anxious. Marked "acetone" odor to the breath. Abdomen somewhat distended and tympanitic; not especially tender. The physical examination was otherwise negative. Temperature 98.6. Pulse 140. The urine was clear, acid, specific gravity 1,024, albumin and sugar absent, acetone (marked) and diacetic acid present. Blood examination showed white count of 24,000.

Dec. 27. The vomiting has continued. Is now thin, light brown, foul smelling, *i. e.*, fecal in character. Patient has not been relieved by treatment, and attempts at feeding have failed. The abdomen is somewhat more distended and peristaltic movement of the intestine can be seen. The urine is clear, acid, 1,023, albumin 0, sugar 0, acetone (slight), diacetic acid. The patient was etherized, and the abdomen explored through a median incision by Dr. H. W. Cushing. A band formed by a Meckels diverticulum was found which extended from the ileum, in the vicinity of the cecum to the anterior abdominal wall, to the left of the median line near the umbilicus, completely obstructing the small intestine. This band was resected. One end was inverted into the ileum, which was closed over it by a "purse string" suture. The abdominal end was ligated close to the peritoneal attachment on the abdominal wall. The distal collapsed portion of the intestine immediately filled, and the obstruction was relieved. Examination of the rest of the intestine showed no other obstruction and

the abdominal wound was closed by sutures. The patient's condition at the end of the operation was quite serious and required free stimulation. That the "shock" was more pronounced, and the danger of the patient increased by the use of an anesthetic in the "acetic" condition of the patient could not be definitely decided. There was good reason to expect marked shock in a case of this nature and duration, even had no anesthetic been used.

The patient reacted fairly well from the active stimulation, and during the afternoon and evening was quite bright. Temperature 100.4, pulse 150, fair quality.

Dec. 28. After 12.30 A.M. patient's condition became worse. Pulse was of poorer quality. There was gradually increasing stupor. No defecation. The abdomen rapidly became distended. At 9.45 A.M. (without anesthesia) the abdomen wound was opened, a loop of intestine found, and a rubber tube inserted for drainage, forming an artificial anus. Considerable liquid feces and gas drained off through this tube. The contents of the intestine had a marked acetone odor. The patient's condition became steadily worse. She was apparently unconscious, then comatose. The acetone odor in the breath increased. The pulse failed, and the patient died at 12.15 P.M. The urine (post-mortem specimen) showed a very marked reaction of acetone and diacetic acid. The temperature in this case was not especially elevated, while the pulse corresponded with the patient's general condition.

In this case two conditions existed: first, intestinal obstruction from mechanical cause; second, the "acetone" disturbance. This latter case indicated by the persistent vomiting, thirst, odor of breath, urine, stupor changing to coma without especial constitutional disturbance. The constipation, leucocytosis, abdominal distention, marked the condition of intestinal obstruction, which would cause the fecal character of the vomitus. The condition of the patient was quite serious immediately after the first operation, but after the reaction there seemed no reason why the patient should not recover, except for the disturbance from the acidosis; and it is probable that had not this existed the case would not have ended fatally.

CASE VII. T. D., a boy, aged seven years, of Melrose, entered the Children's Hospital on Jan. 2, 1904, with hip disease which had existed for nineteen months, but which had been very acute for only three weeks. An abscess had appeared. During the preceding two years there had been several attacks of diarrhea. The child also had measles and whooping cough and inflammation of the middle ear. Under rest and traction the acute symptoms about the hip subsided. The patient then got up and was walking about the ward with a Taylor hip splint. The pulse was 100; the temperature, 99. The urine was clear, acid, free from sugar, albumin and acetone and diacetic acid.

On Jan. 28, 1904, nearly four weeks after entrance, he vomited several times and was considerably prostrated. The characteristic odor of the breath was noticed. Acetone and diacetic acid were present for several days in the urine, which was rather high colored and had a specific gravity of 1.029. There were no further symptoms except a rise of temperature to 102.

CASE VIII. R. K., a boy, aged four years, of Boston, entered the Hospital on Jan. 27, 1904, for the

radical cure of a left inguinal hernia. Aside from the hernia there was nothing noticeable in the history or physical examination. The boy was a Russian Jew, who neither spoke nor understood English, and apparently, as a result of homesickness, he cried almost constantly after entrance for several days. The urine was of normal color, acid, reaction, and free from albumin, sugar, bile pigment, acetone and diacetic acid. The urates were increased.

Two days after entrance, under ether anesthesia, a Bassini's operation was performed. The child recovered from ether well. Next day a sweet odor of the breath was noticed. Acetone and diacetic acid were present in the urine. There were no other symptoms except the constant crying and pulling off the dressings. There was no vomiting. After two days the acetone disappeared from the urine. While there was acetone in the urine the child was kept on a fat diet, and was given bicarbonate of soda by mouth. The child was discharged well in three weeks.

CASE IX. C. K., a girl, aged four years, of East Boston, entered the Hospital on Feb. 15, 1904. She had a congenital dislocation of both hips. On Sept. 4, 1903, a little over five months before, the right hip had been reduced by the Lorenz method. The etherization and convalescence were without incident. Since that time the right thigh had been kept in plaster. The plaster was removed. The skin was in fair condition. There were no excoriations. Otherwise the child was in good physical condition. The urine was acid, clear, and of slightly high color, free from sugar and albumin.

Three days after entrance, on Feb. 18, 1904, the child was etherized. In attempting by manipulation to change the right thigh from the first to the second position, the lower epiphysis of the femur was separated in spite of the fact that but little force was used.

An attempt was then made to reduce the dislocation on the left, when the same accident happened on that side. No further attempts at reduction were made. A double spica bandage of plaster was applied reaching to the toes. The child was under ether between a half and three quarters of an hour.

The child recovered from the ether as usual. There was but little nausea. In the evening the pulse was not strong; the rate had increased from 130, before the operation, to 150. The color was good and the skin warm. The child slept well. She had taken liquids. The bowels moved. She passed urine. There was absolutely nothing in any way unusual in the condition until six o'clock in the morning after the operation, an interval of twenty hours. The pulse at that time grew suddenly weak and rapid. The color became poor. A hypodermic injection of strychnine was given, and a few minutes later the color and pulse improved. The child seemed a little dull. There was no vomiting. The breath smelled sweet.

The condition improved, the color grew better, the pulse became stronger during the next three hours. There was another sudden relapse. The pulse became weak and rapid, the lips blue, the face looked drawn. Three ounces of urine were withdrawn by catheter. Acetone and diacetic acid were present. An enema of brandy, coffee and salt solution was given. The pulse improved a little. A half a drachm of bicarbonate of soda was given every three hours by mouth, and two drachms every three hours by rectum from this time on. During the day the child grew rapidly worse. She sank into a restless coma. The lips were very blue in spite of oxygen given freely. The drawn expression of the face increased, the eyes became sunken. She seemed to rally a little after the hot enemata were given. Considerable liquid was given

by mouth and retained, with the exception of vomiting a little milk once and a dose of soda once.

At 6 P.M. an injection of salt solution was given under both pectorals. Marked improvement followed in the pulse and in the color, and the child came out of the coma. At that time the temperature was 104. Only an ounce and a half of urine was obtained with the catheter. Hot poultices were applied over the kidneys. Urine was passed naturally at midnight and at 2 A.M.

At 9.30 P.M. the child was able to recognize her mother. At midnight, however, the condition again grew suddenly worse. The pulse grew weaker and more rapid; the respiration became poor. The temperature rose to 106 at 3 A.M. The coma returned. Stimulation by digitalis and strychnine subcutaneously, and by oxygen were of no avail. The child died at 5.30 A.M., twenty-three hours after the first collapse, forty-three hours after the operation.

Autopsy, Feb. 20, 1904.

Well-developed and nourished girl of three and a half years. Skin translucent white with marked lividity of the back. Flesh of body and thighs quite flabby. Body length, 95.5 cm. Slight rigidity in hands and feet. Five hours post-mortem. The layer of subcutaneous fat in the abdominal wall is 2 cm. thick.

Peritoneal cavity: Omentum short and contains little fat. No adhesions or fluid. Peritoneal vessels not congested. Intestines are moderately filled with greenish fluid. Mesenteric lymph nodes are slightly enlarged. Appendix hangs into pelvis, 5 cm. long, with short mesentery. Diaphragm is at the fourth rib on the left, and third intercostal space on the right.

Pleural cavity negative. Pericardium negative.

Heart: Normal size, tissue pale, fairly firm. Ventricles contracted, blood dark and fluid. Fresh examination shows a small amount of fat in the tissue.

Lungs: Soft and crepitant except in very posterior parts where the tissue is darker red and moist.

Spleen: A little large, rather firm, dark red, and the cut surface is moderately pulpy. Malpighian corpuscles quite numerous and distinct.

Gastro-intestinal tract: Stomach is partly filled with greenish-yellow liquid. Vessels of mesentery are congested. Mucous membrane apparently normal. Intestines are not remarkable. Pancreas normal size, tissue firm and straw colored.

Liver: Quite large, very pale yellow with a pink color in places. Veins on the surface are well marked. The cut surface is pale yellow and the lobules are indistinctly marked. The knife edge scrapes up much fatty material. The tissue breaks easily on pressure.

Gall bladder distended with green bile.

Kidneys: A little large; capsule strips easily, leaving a smooth surface. Cortex is pale red and 4 mm. thick; pyramids dark red. Fresh examination shows considerable fat in the epithelium of the convoluted tubules. Adrenals negative. Genital organs negative. Bladder contains about 30 cc. cloudy urine.

Bone marrow bright red.

All the muscles of both thighs are small, soft and flabby, and pale yellow.

Organs of the neck, negative.

Brain: Vessels of meninges congested. Brain tissue firm, quite moist and apparently normal. Moderate amount of fluid in the ventricles. Ependyma normal.

Anatomical diagnoses.—Extensive fatty infiltration of liver. Fatty infiltration of kidneys. Eosinophilia of gastric mucous membrane and spleen. Fatty degeneration of muscles of the thigh.

Cultures of heart's blood and spleen are sterile.

Bacillus of the colon group in liver and kidneys.

Microscopical examination.—Sections of the heart,

lungs, pancreas, adrenals, ovary, thymus, thyroid, brain and pituitary body show nothing remarkable.

In the spleen there are some large phagocytic cells in the corpuscles, and there are numerous large cells, with generally one or two nuclei, which are filled with large eosinophile granules. Some of these are phagocytic.

In the epithelium of the peptic glands are many very large eosinophilic cells, in which there are numerous vacuoles and foreign bodies. In the small intestine these cells are very numerous, but there are few in the large intestine.

Liver: The nuclei of the liver cells are, in general, well stained, those of the connective tissue stain rather poorly. The liver cells are filled with vacuoles, which a section stained with Sharlach shows to be due to fat. Very little of the cytoplasm can be seen.

Kidneys: The epithelium of the convoluted tubules is broken down, stains poorly and is filled with vacuoles of fat droplets. The nuclei stain well. In the long tubules this is less marked.

In the bone marrow are numerous eosinophilic cells.

CASE X. R. H., a boy, aged six years, of Quincy, entered the Children's Hospital on Feb. 16, 1904. He had begun to walk at fifteen months. About the end of his second year it was noticed that he was lame, and as time went on he fell more and more frequently. When trying to rise he climbed up his own legs, grasping first an ankle, then by great effort a knee, and finally straightening up. Five months before entrance he recovered from pneumonia which came on after measles and whooping cough. Since then he had been unable to rise alone, although he could still walk when on his feet. During the three months before entrance his feet began to turn under him. He had been treated in the Out-Patient Department for sixteen months by massage.

On entrance he was only in fair physical condition. There was slight mental impairment. The expression was vacant; he was a mouth breather. The chest and abdomen showed nothing abnormal. The pulse was 100; the temperature 99; the respiration was 25. The muscles of the arm and shoulder girdles were much atrophied and weakened. The atrophy of the leg muscles was less marked. The feet were held in slight position of equino-varus. The gait was waddling and unsteady. The grip of the hands was weak. There were athetoid movements of the hands. The urine was acid with specific gravity of 1,018 to 1,028, and was free from albumin, sugar, acetone and diacetic acid and bile.

Three days after entrance, under ether anesthesia, both Achilles tendons were cut subcutaneously, and the feet were put in an over-corrected position in plaster-of-Paris bandages. Next day the child was comfortable and in no pain. The recovery from ether had been normal. Two days after the tenotomy the pulse was noticed to have jumped to 142, and the child seemed a little dull, with periods of restlessness. There was no pain, no nausea. The temperature and respiration were not altered. The sweet odor of the breath led to another test for acetone in the urine. It was found to be absent. Next day the child seemed to be better. The pulse dropped again to 100 within three days. Six days after operation the child was discharged to the Out-Patient Department without any unfavorable symptoms.

CASE XI. M.D., a girl, aged nine years, of Lowell, was admitted to the Children's Hospital on Feb. 18, 1904. She had been through measles and chicken-pox. She entered because of a valgus deformity of the foot, following an operation five years before for the correction of a congenital equino-varus. She was well

developed and fairly well nourished. Nothing abnormal was found on physical examination except the extreme valgus deformity of the right foot. The urine was acid, specific gravity 1,024 and free from albumin, sugar, bile, and acetone and diacetic acid. The temperature was 99; the pulse 100.

Two days after entrance the child vomited several times; complained that she felt sick, and seemed dull. The breath was sweet. Acetone was found in the urine.

She was at once put upon bicarbonate of soda, and given a diet as free from fat as possible. For the succeeding three days there was no acetone or diacetic acid in the urine. The child grew bright again. The vomiting ceased.

On the next day, the sixth after entrance, both acetone and diacetic acid again appeared in the urine, but there were no unusual symptoms.

Twelve days after entrance under ether anesthesia a complicated plastic operation on the foot was performed, and the limb part put up in plaster-of-Paris bandage. Considerable pain followed the operation, but there were no unusual symptoms and there was no reappearance of acetone or diacetic acid in the urine.

About three weeks later the child was discharged to the Out-Patient Department.

CASE XII. M. L., a boy, five years old, of Boston, entered the Children's Hospital on Feb. 23, 1904.

The child had been knock-kneed ever since coming to this country from Italy four years ago.

He was fairly well developed and nourished. There were a few râles in the back of both sides of the chest; otherwise the lungs and heart were normal. The abdomen was distended and tympanic. The rosary and the epiphyseal enlargements at the wrist and ankles were marked. There was a very prominent knock-knee due to a curve in the upper left tibia. The urine was normal in color, specific gravity 1,012, sugar, albumin, acetone and diacetic acid absent.

On the first two days after entrance the child vomited several times. The odor of the breath was sweet. The child complained of the throat and cried constantly. There was no collapse and no stupor. The temperature was elevated to 101 on the second evening, but at only one other time did it rise above 99.5. The urine showed both acetone and diacetic acid. This lasted for three days. There was no albumin or sugar at any time. The urine was acid except on one day after the soda had been given. The child was put on a fat free diet and given bicarbonate of soda.

The vomiting ceased in two days. There were no other unusual symptoms.

Twelve days later a Macewen's osteotomy of the femur was done, and the leg was put in a plaster-of-Paris spical bandage. The child was very noisy in coming out from ether. This was controlled by morphine. There was no recurrence of the acetone in the urine. Three weeks after the operation the bandage was renewed, and the child discharged to the Out-Patient Department.

CASE XIII. W. J., a boy, aged four years, of Roxbury, entered the Children's Hospital on Feb. 24, 1904, for the correction of double knock-knee. Three years before the child had chicken-pox and pertussis. Two years before had measles. He was markedly rachitic, but otherwise was in fair condition. Rosary and epiphyseal enlargements at the wrists and ankles were marked. The abdomen was distended and tympanic. There was knock-knee on each side with forward and outward bowing of the tibia and femur. There was a coxa vara on the left. The heart and

lungs were normal. The pulse was 90; the temperature 99, and respiration 30. The urine was acid, 1,026, normal in color and free from albumin, sugar, acetone and diacetic acid.

Two days after entrance a Macewen's osteotomy of both femora was done under ether anesthesia, and the legs were put in a plaster-of-Paris bandage from the toes to the umbilicus. The child recovered normally from the operation. Next day the odor of the breath led to the examination of the urine again. Both acetone and diacetic acid were present. There was no sugar or albumin. There was no rise of temperature. Within two days both the acetone and diacetic acid had disappeared. There were no other unusual symptoms at any time. The child was discharged three weeks later, still in plaster bandages, but with the legs in improved position and with good callus.

CASE XIV. E. P., a girl, aged three years, of Dorchester, entered the Children's Hospital on Feb. 27, 1904.

A year before she had pertussis. She came in because of pleuritic effusion following measles. She was well developed and nourished. The right chest was hyperresonant, but except for some moist râles in the lower back, the respiratory sounds were normal. The heart sounds were heard more plainly on the right than on the left.

On the left the intercostal spaces were bulging. The respiratory excursion was diminished. Respiratory and voice sounds and tactile fremitus were diminished except that there was bronchial breathing above the fourth rib. There was dullness in both back and front to the apex. The abdomen showed nothing abnormal. The temperature was 99.6; the pulse 122; the respiration 33.

The left chest was at once tapped and eight ounces of slightly cloudy, straw-colored serum were withdrawn. On account of coughing the needle was then removed. Next day she was transferred to the medical side. Afterwards two ounces more were withdrawn by a second aspiration. It was then found that there were signs of consolidation in the left apex,—dullness, broncho-vesicular breathing and numerous medium moist râles.

On the day after the tapping, the urine showed the presence of acetone though the reaction was not marked. There was no vomiting, no stupor, nor other unusual symptoms. She was given a diet without fat and was given cream of tartar and phosphate of soda, two drachms every four hours. The child was discharged from the hospital relieved, but still with dullness at the left apex two weeks after entrance.

Child showed no unfavorable signs or symptoms after aspiration of chest, no odor to breath noticed. The condition of acetone in urine would have been entirely overlooked except that in the course of routine examinations at that time acetone was found and in such small quantities that violet color not seen, or overlooked in foam, but urine gave a distinct violet reaction when dried on paper. This condition lasted through Feb. 29 (three days after entrance). Urine of March 1 showed not even the reaction on paper.

BIBLIOGRAPHY.

- Baginsky: Arch. f. kinderheilkund, 1888, 9, 1.
 Becker: Deut. Med. Woch., 1894, 16, 18; also Virchow's Arch., Vol. cxi, p. 1.
 Bernert: Zeitschr. f. Heilk., Bd. III, Heft. 2.
 Blumenthal and Neuberg: Deutsche med. Woch., Bd. xxvii.
 Brewer: Annals of Surgery, October, 1902.
 Edsall: Am. Jour. Med. Sciences, April, 1903.
 Frerichs: Deutsche Arch. f. klin. Med., Bd. xiv.
 Gelmuyden: Zeitschr. f. Phy. Chemie, Bd. xxiii.
 Guthrie: Lancet, July 4, 1903.
 Herter: Chemical Pathology, New York, 1902.
 von Jacksch: Zeitschr. f. klin. Med., Bd. viii.

- von Jacksch: Ueber Acetonurie und Diaceturie, Berlin, 1895.
 Joslin: Jour. Med. Research, 1904.
 Juffinger: Wein. Klin. Woch. schr., 1888, 1, 367.
 Kaulisch: Prager Vierteljahrsschr., 1857, Bd. xiv.
 Kraus: Alleg. Path. Lubarsch. and Ostertag., Heft 2, 1895.
 Kussmaul: Zeitschr. f. klin. Med., Bd. vi.
 Lane: Lancet, Jan. 27 and Feb. 17, 1894.
 Lorenz: Zeitschr. f. klin. med., Bd. viii.
 Lorenz: Zeitschr. f. klin. med., 1891, Vol. xix.
 Magnus and Levy: Arch. f. exper. Path. and Pharm., Bd. xlii.
 Marfan, Grancher, and Comby: Arch. de Med. des Enf., Vol. iv, No. 11.
 Müller, F.: Virchow's Arch., Bd. 131, Supplement.
 Müller, J.: Arch. f. Exp. Path. and Pharm., Bd. xl.
 von Noorden: Auto-intoxication, 1903.
 Offer: Zeitschr. f. innere Med., 1903, s. 245.
 Petters: Quoted by Kraus.
 Rosenfeld: Zeitschr. f. klin. Med., 1895, Bd., xxviii, s. 256.
 Satta: Quoted by von Noorden.
 Schwarz: Deutsche Arch. f. klin. Med., Bd. lxxvi.
 Stadelmann and Minkowski: Arch. f. klin. Med., Bd. xviii.
 Taylor: Am. Jour. Med. Soc., No. 107, p. 569.
 Taylor: Jour. of Exp. Med., 1899, p. 399.
 Waldvogel: Centralbl. f. innere Med., July 15, 1899.
 Waldvogel: Zeitschr. f. klin. Med., 1903, Bd. xlii.
 Wollstein: Am. Jour. Med. Soc., May, 1902.

Reports of Societies.

AMERICAN GYNECOLOGICAL SOCIETY.

TWENTY-NINTH ANNUAL MEETING, HELD IN BOSTON, MASS., MAY 24, 25 AND 26, 1904.

FIRST DAY.

THE Society met in the Boston Medical Library, under the presidency of DR. EDWARD REYNOLDS of Boston.

An address of welcome was delivered by DR. CHARLES M. GREEN of Boston, which was responded to by DR. HENRY T. BYFORD of Chicago.

THE TREATMENT OF GALLSTONES FOUND AS A COINCIDENCE IN ABDOMINAL OR PELVIC OPERATIONS.

DR. JOHN G. CLARK of Philadelphia stated that among the unsettled questions in abdominal surgery, the treatment of gallstones found as a coincidence in abdominal or pelvic operations might be considered a debatable one. He followed the plan at present of removing gallstones which were found in the course of another operation, if the patient's condition permitted of this extra operation. Although it was stated that 95% of gallstones produced no symptoms, he believed that this statement should not be applied to cases as one met with them at the time of an operation. In his review of recent literature he had been especially impressed with the fact that knowledge of the early stages of cholelithiasis was very indefinite, and that many cases which came to operation for more or less urgent symptoms did not have the clinical symptoms of colic and jaundice, as usually taught in medical schools. In view of this hiatus in the early history of this disease, he believed that many symptoms now attributed to gastralgia, indigestion, functional disturbances of the gastrointestinal tract, etc., would, as knowledge increased, be ascribed to the presence of gallstones with associated infection, which was so frequently found in cholelithiasis.

In referring to the etiology of gallstones, he said that three facts had been prominently established: (1) That the bile was not bactericidal; (2) that the micro-organisms in the gall bladder were predisposing, if not absolute, causative factors in the formation of gallstones; (3) when gallstones were present in the gall bladder, infection in that viscus was much more likely to take place.

He mentioned three general avenues through which infection might enter the gall bladder: (1) From

bacteria circulating in the general blood stream and reaching the liver through the hepatic veins; (2) by the direct passage of bacteria into the common bile duct from the duodenum; (3) by the transportation of bacteria from the intestine through the portal circulation.

He then recurred to the frequently quoted statement that 95% of gallstones did not produce symptoms, and showed from his own series of cases that in at least 50% there were varying symptoms from undoubted attacks of colic and jaundice to less pronounced gastro-intestinal symptoms. To justify operative intervention in cases which were not producing well-defined symptoms, the mortality and morbidity should be a very low one. In his own experience no death had occurred, nor had there been any serious complication referable to the secondary operation. In lieu of the fact that the additional operation did not seriously jeopardize the life of the patient, and also because he had seen two patients die from cholelithiasis a year or more subsequent to an abdominal operation, in which, had the routine exploration been made, the gallstones might have been easily removed, he believed that the best interests of the patient would be conserved if the gallstones were removed as a secondary part of another operation, in the event of their being found. He appended the history of twelve cases to his paper, in which the various operative points, as well as the significant facts in symptomatology, were elaborated.

DR. R. STANSBURY SUTTON of Pittsburg said that gallstones did not always produce symptoms which demanded or justified resort to operation. If they were encountered during the course of another operation, they had better be removed. There was a remedy, however, which obviated the necessity, in some instances, of surgical intervention, namely, kolalin. He had used kolalin in dozens of cases in which he had held operation in abeyance, and did not have to operate subsequently.

DR. GEORGE M. EDEBOHLS of New York had occasion at one time to operate on a woman who presented marked dyspeptic symptoms. In addition she had movable kidney, chronic appendicitis, and induration in the region of the gall bladder. He anchored the kidney, removed the appendix through a lumbar incision, pulled the gall bladder into the lumbar wound, and found the stone about four or five centimeters in length, pear-shaped, and nearly filling the gall bladder. The attending physician was positive that the gall bladder did not produce symptoms of stone in it. He would not let him remove the stone from the gall bladder. A year later he opened the woman's abdomen for some other condition, making an incision near the gall bladder. He investigated the gall bladder, found it was perfectly healthy, and that the large stone had either passed or had been dissolved. The treatment after the previous operation consisted of the use of olive oil for about a month, and whether this had anything to do with the passage of the stone, he did not know. At any rate, the stone had disappeared and had left no trace of its former existence.

DR. A. PALMER DUDLEY of New York emphasized the importance of looking beyond the gall bladder for trouble. He believed that stones were formed in the liver ducts themselves, and that from a stagnant circulation cholesterol nuclei formed, and that only a small proportion of stones were found in the gall bladder. He would not hesitate to go into the center of the liver. In fact, in the last case he had, in which the diagnosis of gallstones had been made by a medical confrère, he boldly went into the gall bladder, but found no stone. He found the duct dilated; he went