Decapsulation not only interferes with the physiologic vasculization in the volume of the organ incident to its vasculating functional requirements, but subjects it to the damaging influence of vibrations incident to walking and riding and to shocks incident to more violent exercise of the body. In this way, as well as by the use of transfusion sutures, pathologic changes are induced in the organ.

Dr. Row, of Huntington, W. Va., brought me a case a year or so ago in which twenty months after operation by decapsulation and transfusion at the hands of another surgeon, I operated for relief of pain. I found that the kidney had been converted into three lobes by the transfusion sutures, and that it had a thin inelastic cicatricial envelope as the result of the decapsulation.

T. Carwardine reports a similar case in which the kidney was indistinguishably blended with the surrounding parts, but in which the kidney substance was paler and firmer than usual, and scarcely bled when cut as if a certain fibrosis had developed from the capsule along the trabeculae.

Another case in point was brought to me a couple of years ago by Dr. Hefflin of Newport, Ky., for fixation of the left kidney. The right one had been anchored several years before by one of the most distinguished operators in the United States, who employed the decapsulation-transfixion method, with the result that the kidney had been a constant source of pain from the date of operation. I anchored the left kidney by the method that I have just described with the result that it has been absolutely painless from the date of convalescence. When the patient was last seen, less than a month ago, both kidneys were firmly anchored, but the right one—the decapsulated and transfixed one—remained painful. The contrasting results of the two procedures were never more strikingly illustrated.

Paul F. Freiherich of Greifswald asserts that transfixion sutures always cut through, sometimes completely, and interfere with function.

Conclusions

In conclusion permit me to add that I have been induced to evolve and practice extracapsular fixation of the kidney because: (1) it is surgically feasible, (2) it is effective primarily, (3) it is general, and indeed, as far as my experience goes always practicable, and (4) it comes nearer than any other procedure to restoring the pathologically movable kidney to its normal anatomic relations and to the exercise of its normal physiologic functions. I urge it as the operation of choice as against certain other procedures that are yet prevalent because, among the latter, (5) decapsulation is liable to induce and frequently does induce wholly unnecessary pathologic changes resulting either in its loss or in the invalidism of either the kidney or the patient or both; (6) transfusion sutures in the parenchyma may and often do induce similarly disastrous consequences; (7) treatment of the surface of the kidney by irritating substances induce adhesions and tissue changes that are pathologic in character; and (8) in consequence of the greater liability to hemorrage and infection following all invasions of the true capsule and the parenchyma of the kidney and for other obvious reasons these procedures are more liable than extracapsular fixation to be followed by primary complications and final failure.

The Groton.

Abstract of Discussion

Dr. Samuel Lloyd, New York City: My criticism of Dr. Reed's operation would be that he fails to recognize the fact that a very large number of the patients with movable kidney have chronic nephritis. Dr. Edelbohs called attention to this fact some years ago, and it was his observation in these cases that he will perform decapsulation of the kidney for chronic nephritis. Often we do not discover that these patients are afflicted with nephritis until we are operating on them. Dr. Reed, by using the fibrous bands in the fatty capsules for his fixation and leaving the capsule proper intact, would deprive the patients of the very best feature of their operative treatment, the cure of the nephritis by the decapsulation. It has been my experience in following up Dr. Edelbohs' operation, that a great deal of benefit is derived from the decapsulation of the kidney and its fixation through the capsule, not through the kidney. I have not seen the difficulties Dr. Reed speaks of following exercise, such as pain or discomfort. The transfusion was given up by Dr. Edelbohs many years ago, because fixation by means of the capsule gave better results.

Dr. Charles A. L. Reed, Cincinnati: I recognize the fact that pathologic conditions in an individual case may necessitate division of the capsule.

Further Report on Ileocolitis Complicated by Acidosis *

Thomas D. Parke, M.D.

Birmingham, Ala.

Three years ago I presented to this Section a series of cases that had occurred under my observation during the preceding four years, and I now wish again to call attention to them by the relation of a few histories and by a brief discussion of the problems involved.

I have been unable to find descriptions in the textbooks of the symptom-complex presented by the series, and my object now, as three years ago, is to focus attention on and bring recognition of this distinctly marked symptom-complex. It is not reasonable to suppose that such cases are confined to one limited area. It is only reasonable to conclude that they have not been recognized elsewhere.

Thirteen cases have come under my observation since the report of three years ago, mostly in consultation, but this does not embrace a number of other cases that have come to my notice, directly and indirectly, in the practice of other physicians of our district. A large percentage of the whole series of thirty-two cases has occurred in breast-fed, well-nourished children, previously healthy and flourishing. The oldest of the series was 8½ years of age.

Symptoms

Loose bowels, caused by a mild grade of ileocolitis and lasting from one to four days, precede the onset of the typical symptoms. Then laboring breathing develops, often suddenly, followed by prostration, restlessness, obstipation, enlargement of the liver. The temperature

* Read in the Section on Diseases of Children of the American Medical Association, at the Sixty-first Annual Session, held at St. Louis, June, 1910.
in some cases becomes subnormal, in others stands around 100 F.; in a few runs high. Vomiting with acetone odor of breath occurs in a good proportion and is absent in others. The urine is limpid and free. Albumin has been present in some and absent in others of the few cases from which urine could be obtained. Urine from three cases was submitted to chemists for nitrogen estimation, and in each instance the report was that the ammonia nitrogen was high. Jaundice was absent in every case.

During the period of labored breathing the breath feels cold to the hand held in front of the nostrils. This has been interpreted, whether rightly or wrongly, to mean volatile substances thrown out in the expired air. During this period of labored breathing, too, soot, in our locality, lines the interior of the nostrils, being, of course, mechanically deposited by the air as it rushes in. Some of the children noticed little unless their attention was especially attracted, but the impression given was that they were directing attention rather to getting air, and deep labored breathing without interruption, hour after hour, calls for attention on the part of any conscious breather.

**OUTCOME**

In fatal cases death occurs in from 48 to 70 hours after the onset of labored breathing, 55 hours being about the average.

In the cases recovering, the duration of the stage of labored breathing is about the same. Death seems to occur from exhaustion. Convulsions have not been noted and coma has not developed till within a few hours of death.

The mortality in a series of thirty-two cases, occurring in my own practice or in consultation, has been 71 per cent.

**POST-MORTEM APPEARANCE**

Post-mortem examinations have been made in nine of the cases. The findings have been negative, as to the gross appearance of the organs, with the exception of the liver and the mucosa of the intestine; these were injected in some cases, in others not. The liver usually showed yellowish areas on the surface, extending downward to varying depths. The gall-bladder in all cases has been distended with dark, green,ropy bile. Microscopic examination of the organs has only shown fatty degeneration of the liver. Neither the heart, the kidney nor the muscles have been found fatty in any instance.

**TREATMENT**

Treatment has varied from eliminative to symptomatic, with the employment of strychnin in good doses, epinephrin and hypodermoclysis of normal salt solution. I am under the impression that treatment avails little when a sufficient injury has been done the system, but that hypodermoclysis is of decided value when the injury is not sufficient to be lethal.

**CASE HISTORIES**

Of the fourteen cases encountered during the last three years only three will be cited.

**Case 1.**—Infant L., male, white, aged 2½ years. The child was breast-fed until weaned at the end of the first year. April 30 the child had two bloody actions, for which castor oil was prescribed by the attending physicians. Friday the mother reported the child so much better that he was not seen. On Saturday the attendant again called to find him with labored breathing, but only thirty to the minute. Vomiting began at midnight.

After twenty-four hours' vomiting of the water given, coffee grounds vomitus began. The patient was seen in consultation Sunday afternoon at six. Respirations 35, labored and deep; pulse feeble and irregular, 135; liver one inch below free border of ribs; abdominal masses were palpable on the left side of the abdomen; the liver was not absent. Anemia was of a marked degree; temperature 99.5; mind clear. Hypodermoclysis of normal salt solution, strychnin and epinephrin were recommended. The child reached the height of a very severe condition on April 30 but continued to improve on May 2, 1910.

**Case 2.**—Infant B., male, white, aged 15 months; breast-fed and still nursing. Lately the child had been given buttermilk in addition. It was a well-nourished infant when in consultation April 21, 1910, a history was obtained of slight colitis from the seventeenth, four days previously. The child was not sick enough to cause any concern till the day previously when he developed labored breathing and vomiting. There was obstruction from the onset of symptoms; respirations were 34, labored and deep; pulse 125, easily compressible; abdomen moderately distended with gas, liver an inch below free border of ribs prominent; tender to pressure; acetone odor of breath quite marked; breath cool to hand; temperature 99.8; mind clear. Hypodermoclysis of normal salt solution, strychnin and epinephrin were recommended. Next day the respirations were 44, temperature 100.4, pulse 140. Respirations were not so labored as on preceding day, but on least exertion child was badly winded; acetone odor less marked; liver extends two inches below free border of ribs.

On the next day, April 23, the respirations were 33 and not labored, temperature 100.4, pulse 140; liver retraced some what from day before, and edge less sharp to finger. On April
ILEOCOLITIS AND ACIDOSIS—PARKE

24 the child was better in every way; liver not palpable below border of ribs; barley gruel recommended.

A good report was received April 25, except that the barley water had disappeared, causing nausea and had been discontinued. April 26, in afternoon, there was a slight tendency to laboring breathing noted on any exertion. Barley gruel and orange juice were given once, followed by vomiting, which kept up during the night and showed coffee-ground material. When seen April 27, condition was apparently better and had been so during the preceding night and that morning. The breathing could be heard as soon as one entered the room during the early morning hours. Respirations 40, pulse 100, child very restless; but vomiting had ceased; liver was felt one and a half inches below ensiform; tympany quite marked over abdomen.

On April 28 respirations 28 and not laboring; pulse 150, temperature 100.2; tympany lessened. The child's condition was better and nourishment was again given, consisting of a half ounce of buttermilk. Medication consisted of strychin and ephedrine. After the obstipation of the period of extreme illness, muceous stools reappearance and persisted during a tedious convalescence not complete to date.

This child received no nourishment from April 18 to 24, when barley water was given and digested. It was given barley water again on April 26, when vomiting was precipitated which came nearly being fatal. Buttermilk was given on the 28th and the child was nourished regularly thereafter. This recovery from a condition showing a mortality of over 71 per cent., and other recoveries from similar conditions under enforced starvation of from five to seven days, would seem to merit some attention, in view of the rôle assigned to starvation in the production of acidosis.

As to the nature of these cases, I have come to hold, tentatively, that they belong in the acidosis group; that a colitis, usually of mild type, through the production of some toxin, cripples the liver cells and, as a consequence of impaired liver function, products of fatty, incomplete metabolism are swept into the circulation and carried to the fixed tissue cells, which are dependent, of course, on the blood for proper nutritive and oxidative substances.

Dr. James Ewing¹ has presented elsewhere a thorough discussion, in which he freely admits the difficulties inherent in a question that involves the many unsettled problems of metabolism, and I think every one who has made any study of the question will fully agree with him. He divides cases of acidosis into two main groups: one without fatty degeneration, as represented by diabetic acidosis, and one with fatty degeneration, represented by the toxemia of pregnancy, delayed chloroform poisoning, Eck's fistula, cyclic vomiting in children and poisoning by hematoxic immune serum.

Only recently a professional neighbor was relating the history of a case of toxemia of pregnancy in a woman who had passed successfully through five pregnancies. With my own case in mind, he was asked if dysentery had preceded the onset of toxemic symptoms, and he replied in the affirmative. This question was further suggested by the history of a non-pregnant adult female, in whom a most serious acidosis was precipitated apparently by a mild dysentery.

If we are right in assigning our series of cases to the acidosis group represented by toxemia of pregnancy or by delayed chloroform poisoning, we have traveled only a short way on the road of explanation. We have not explained why the liver enlarges so rapidly; we have not explained the fatty degeneration of the liver, the changes in the bile, the obstruction, nor the factors involved in the air hunger.

Hematoxic immune serum injected into an animal produces fatty degeneration or necrosis, according to the dosage. A ligated hepatic artery results in increase of fats in the liver cells, according to Holmes and Pearce, but the explanation of these results leads to as many theories, practically, as there are investigators. So long as physiologic chemistry fails to account for all the processes involved in the functions of the liver, so long as physiology fails to agree on the functions of the liver, so long as the rôle of the internal secretions is undetermined and the intimate processes of cell nutrition, including tissue respiration, are matters of conflicting theories, it is not to be expected that definite conclusions will be formulated, either by the laboratory worker or the clinician.

Considerable effort was made to discover the explanation of the air hunger met with in all of this series of cases.

Haldane and his coworkers have apparently demonstrated that hyperpnea, labored breathing, results from carbon dioxide tension in the respiratory center, and that the threshold at which the carbon dioxide excites the center may be lowered by acid or other products resulting from the want of oxygen.

No cyanosis exists in any of the cases, as would pertain if the blood contained excess of carbon dioxide, and there is no lack of oxygen in the alveolar air inspired, so that some other factor must be involved beside want of oxygen in the alveolar air.

An original worker writes in a personal letter:

There may be incomplete oxidation in the presence of abundant oxygen in the blood, because the factors—enzymatic or otherwise—which normally cause oxidation are defective. This would, of course, be defective metabolism. Just what the factors of oxidation are, is the dark problem. The intermediate acids combine with the bases of the respiratory center (Na2CO3, NaHCO3, NaHPO4, etc.) and the free CO2 resulting, remains in the center, causing increased tension and hyperpnea.

And thus we seem to be carried back to the original question involved in acidosis—the production of intermediate acids. Whatever the ultimate explanation of the air hunger, it appears reasonable to think that this explanation will mean the settlement of the controversy between those physiologists who hold respiration to be accounted for by the laws governing diffusion of gases and those who hold vital cell action to be involved.

ABSTRACT OF DISCUSSION

Dr. John Zahorsky, St. Louis: I have seen altogether five cases that fitted this syndrome exactly. All were fatal. I have studied this syndrome from a variety of standpoints, but a year ago I had a patient that put me on entirely new track. This little boy, two and a half years old, had recurrent attacks of cyclic vomiting and in one of these attacks there seemed a little indigestion and the mother gave him a dose of calomel. This deep breathing developed and the liver swelled enormously, but gaining too fast, the child was very sick and I was afraid it would die. That suggested how this syndrome might be explained, i. e., by acute dilatation of the right side of the heart. I could not explain how this liver could swell twice its normal size except by edema or congestion, and passive congestion seemed to be the cause in this case, and that could most easily be explained by an acute dilatation of the right ventricle. We know that deep breathing is one of the symptoms of dilatation or weakness of the right heart. It is true

¹ Ewing, James: Acidosis and Associated Conditions, Arch.
that there was no marked cyanosis or swelling of the veins of the upper extremity, but as there is diarrhea and vomiting with the loss of a large quantity of fluid, we can understand how close might be this accumulation of blood in the liver and yet not such an accumulation of bile in the upper extremities. One boy had spasmodic croup with severe suffocative attacks. I was asked to intubate the child and on examination no marked obstruction in the larynx was found, but he had this very deep breathing which was mistaken for obstruction. The liver was enormously enlarged, reaching almost to the umbilicus. Another child had cardiac disease. He suddenly had some severe symptoms and developed this deep breathing and also had this swollen liver. I have concluded that this degeneration was found enlarged livers are due to an acute cardiac dilatation which may be incidental to any acute disease, such as acute gastritis, acute gastro-duodenitis or colitis, or severe vomiting forcing the blood into the heart and stretching the right ventricle. So, while I originally considered Parke's syndrome a definite disease, I believe it is merely a group of symptoms occurring in many diseases. The acidosis is due to faulty metabolism in the absence of carbohydrates, and is not the cause of the disease.

Dr. Isaac A. Arr, Chicago: I am already on record as having said more than once that in almost every one I have had an autopsy performed. In the first case, the child of a physician, the symptoms were pretty much as outlined by Dr. Parke. The child falls suddenly ill, vomiting, rapid respiration, rapid, small pulse, and goes gradually into a coma and last of all develops symptoms of obstruction of the bowel, due probably to extreme atony. They have all shown at autopsy, uniformly, fatty degeneration of all the organs. Dr. Ricketts—who did the autopsy work in the first case, thought that possibly the child might have eaten matches, so profound and extensive was the fatty degeneration. We found not only acetone but lactic and tyrosin in the urine. Dr. Ricketts found that the peri¬cle and the intima of the vessel walls showed fatty changes. I think this is a syndrome that is a pathologic entity and depends on some grave toxemia. The acidosis is merely a symptom, and it is of no more importance than the rapid breathing, the rapid pulse, or the mechanical obstruction of the bowels, and it would be wrong to ascribe too much importance to it. I discussed these cases with a pioneer physician of Illinois and he suggested their close resemblance to the disease described as milk-sickness in cattle. Inquiring more closely into the subject one finds that milk-sickness or trembles bears a striking similarity in symptoms to those which Dr. Parke noticed in his patients.

Dr. Thomas D. Parke, Birmingham, Ala.: There was no coma in any of these cases and there was no mechanical obstruction. There was dysentery and poor digestion and though the bowels did not act during the period of laboring breathing, after this stage of laboring breathing passed off, the bowels became loose again. I am sure there was no mechanical obstruction in any single case. I am sure not only from the post-mortems that I made but from the patients that recovered. There is a good deal more in the acidosis problem yet to be elaborated and I think you will agree with me if you will study the paper by Dr. Ewing in the December, 1908, issue of the Journal of Experimental Medicine. My conception is that the injury done by the toxins of this intestinal trouble cripples the liver-cells and when you get crippling of the liver-cells, the products of intermediate metabolism are carried into the general circulation and produce the air hunger. Dr. Ewing attaches immense importance to this fatty degeneration. He says that these studies of the last few years show the immense importance of fatty degeneration. But we do not know just what works the liver has to do. I have been unable to find any physiologist who could tell me the complete function of the liver. I do not know whether the trouble is in the liver-cells or in the blood that is in the liver, and until we can get thoroughly worked out the full physiology of the liver we shall not know its pathology.