SCIENTIFIC PROCEEDINGS.

ABSTRACTS OF COMMUNICATIONS.

Sixty-third meeting.

Rockefeller Institute for Medical Research. President Lusk in the chair.

33 (965)

Observations upon the so-called food intoxication of infants with especial reference to the alveolar air.

By JOHN HOWLAND and W. McK. MARRIOTT.

[*From the Department of Pediatrics, Johns Hopkins University.*]

There is a condition which has been described under the heading of "Food Intoxication" and "Toxicose" in infants, which is a very fatal one. It is found in the course of severe diarrhoeas and as the terminal stage of various disturbances of nutrition. It is characterized by several striking symptoms, chief among which are dyspnea and excitement, changing into stupor and eventually coma. In its early stages, the condition is difficult to recognize. When it is marked it is unmistakable.

The observations which we have to report were made with the desire of learning more with regard to the essential causes of the condition and of developing some method by which it could be recognized in its incipiency when successful therapy is possible.

It was suggested many years ago by Czerny that acids might have some part to play in it for the reason that the dyspnea resembles that seen in rabbits when given mineral acids and the condition has been often spoken of as acidosis, meaning thereby an increase in the acetone substances. These, however, have not been found to be in excess.

For the last two years we have examined the blood of children

suffering from this condition according to the method described by Sellards which consists in evaporating to dryness, with phenolphthalein, the blood serum from which the proteins have been removed by absolute alcohol. A colorless residue indicates an almost complete absence of carbonates from the blood. Marked cases of intoxication show the carbonates to be very greatly reduced in every single instance. In mild cases this is not found to be the case.

Sellards' other method of determining acidosis,—that is, proving a great tolerance to alkali before the urine becomes alkaline, is also striking with these children.

As a more delicate test we have determined the carbon dioxide in the alveolar air. This has never before been determined in infants or young children. It is obvious that the Plesch method, or the rebreathing of air from a bag, is the only available method with infants. The Haldane or the Krogh-Lindhard methods that require either coöperation on the part of the patient or slow breathing are impossible. It is a matter of indifference whether we obtain the true alveolar air or not, so long as we obtain figures which with the individual patients are constant and so long as they fall within reasonable limits with a group of normal patients.

NORMAL

	CO_2 Mm.		CO_2 Mm.		CO_2 Mm.
I	44.6	VII	44.7	XIV	38.4
II	43.5	VIII	41.0	XIV	40.4
III	44.5	IX	39.0	XIV	38.4
III	44.3	IX	38.0	XV	40.4
III	44.5	IX	38.2	XVI	41.3
IV	39.0	X	38.9	XVI	39.6
IV	38.4	XI	40.3	XVI	40.0
V	40.2	XII	39.6	XVII	39.8
V	43.6	XII	41.7	XVIII	39.6
VI	40.8	XII	42.2	XVIII	38.7
VI	43.5	XIII	43.4	XIX	41.0

We have found that if a known amount of air is rebreathed for between 28 and 32 seconds and if the respirations are deep and the bag has been well ventilated, that we obtain results from day to day which differ very slightly. We have found that with children that are nearly normal the results are very close to those that Peabody found for adults, that is, that the extreme limits for the

carbon dioxide are 38 and 45 mm., and that the great majority fall between 39 and 42 mm. It is possible to obtain with the same child comparable results from day to day. The difference may be 2 mm.—sometimes more, but usually not so much. Occasionally, one determination is very much too low, but after a certain amount of experience, good results are obtained almost every time.

In contra-distinction to the regularity with the normal patients are the results with those suffering with "intoxication." There is no danger of confusion.

INTOXICATION.

No.	Date.	CO_2 Mm.	Dyspnea.	Alkali.
I	Sep. 22	21.2	+++	given
I	" 23	42.0	0	"
I	" 24	54.0	0	stopped
I	" 24	55.0	0	
I	" 25	41.3	0	
II	Sep. 28	18.0	+++	given
II	" 28	17.8	+++	"
II	" 29	21.3	++	"
II	" 30	32.8	+	"
III	Sep. 27	27.0	++	given
III	" 28	34.0	+	"
III	" 29	36.0	0	"
IV	Nov. 16	32.8	+	given
IV	" 17	36.8	+	"
IV	" 20	59.0	0	stopped
IV	" 24	48.2	0	
IV	Dec. 2	44.8	0	
V		27.8	++	
VI		30.4	++	
VII		24.4	+++	
VIII		27.0	++	

The chart shows single observations on four patients and a series of observations on four other patients. It will be noticed that the carbon dioxide is almost always very low. The highest that we have found is 33 mm.; in one it was 18 mm. Associated with the low pressure there is almost always great dyspnea. We have given, therapeutically, alkali in large doses by mouth, by rectum and subcutaneously. If the alkali is absorbed there is a rapid improvement in the dyspnea and coincident with this a marked rise in the carbon dioxide tension,—in several instances to a point much above normal (up to 59 mm. in one case). When

the alkali is stopped, the carbon dioxide sinks again to normal limits. We have found this method of much assistance in diagnosis and consequently of great value in the institution of treatment.

A report upon further phases of intoxication will subsequently be made.

34 (966)

Fibrinogen deficiency in hemophilia.

By ALFRED F. HESS, M.D.

[*From the Research Laboratory, Board of Health, New York City.*]

The amount of fibrinogen of the blood seems to vary within wide variations both in man and in animals. In hemophilia there have been some estimations of the percentage of fibrinogen based upon the amount of fibrin obtained after coagulation. However, these data are so divergent as to allow of no satisfactory deduction, quite apart from the fact that they give no information as to the quality of the fibrinogen.

In the estimations of fibrinogen here reported, a functional method has been made use of. Precipitated fibrinogen, made approximately according to the method of Hammarsten, has been added to the whole blood of cases of hemophilia, of purpura, and of normal adults and children. To ten drops of blood, one, two, and three drops of fibrinogen have been added; a fourth tube serving as a control. In this way we are able to ascertain whether the fibrinogen had a complementary action in hemophilia, as compared to the other cases, and also whether it brought the clotting time of the blood close to the normal. In all cases, the fibrinogen had been previously tested with calcium and found not to clot over night upon the addition of a few drops of a ½ per cent. solution of calcium chloride. In three cases of typical hereditary hemophilia, repeated tests showed that the addition of one drop of the fibrinogen solution to the whole blood markedly hastened the coagulation time. In one of these instances, a case of severe hemophilia, the clotting time was reduced by fibrinogen in four consecutive tests from 90 to 13 minutes, from 55 to 14 minutes,