KETOSIS IN SEA-SICKNESS*

BY

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The theory of the presence of a metabolic disturbance in sea-sickness is mainly based on the work of Oriel (1927); his observation that ketosis may occur before the onset of vomiting suggests the possibility that this ketosis may either itself be the cause of the sickness or evidence of a metabolic disorder which is the actual cause. It is therefore of interest to find how frequently ketosis appears before vomiting begins, and to measure the degree of ketosis when it does occur, in order to decide whether it is sufficiently severe to account for symptoms.

The subjects of this investigation were mainly passengers and crew on two Cunard liners during two rough passages from Liverpool to New York; a few observations were also made on two calm passages from New York to Southampton.

Incidence

With one exception, ketosis occurred in all subjects within twenty-four hours after actual vomiting began. This one exception was a woman, aged 54, who stated that she suffered from "stomach trouble," associated with vomiting at other times; it is possible that her sickness was not "sea-sickness."

On the other hand, considerable disturbance apart from actual vomiting may occur without ketosis; one subject, J. M., had continuous nausea for two days on one voyage without ketosis, although on a later voyage, when actual vomiting occurred, ketosis appeared after twelve hours. Another subject, A. M., had ketosis immediately after vomiting on one voyage, but on a subsequent one ketosis was never detected, although she felt unwell throughout the voyage and her blood pressure was low.

Relation of Ketosis to Time of Vomiting

Figure 1 shows the relation between the intensity of the nitroprusside reaction and the time before or after vomiting at which the observation was made in a series of cases. A pink colour developing in about ten minutes

FIG. 1.—Relation between the intensity of the nitroprusside reaction and the time at which the observation was made before or after vomiting.

was classed as (1), and an almost black colour as (4); (2) and (3) were intermediate degrees. As Oriel found, ketosis may appear before the onset of vomiting. The subject with a (4) reaction before vomiting appeared quite well at the time. Otherwise the cases with early ketosis did not appear different in any way from other cases. However, the figure shows that as a general rule ketosis developed *after* vomiting had started, and might still be moderate after twenty-four hours' vomiting.

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Amount of Acetone Bodies Excreted and Relation to Carbohydrate Metabolism

Figure 2 shows the ammonia excretion of two sea-sick subjects; A. M. took a small amount of carbohydrate food, A. S. was starved. The ammonia affords a fair measure of the ketone bodies in the urine, as is shown by the estimates of ketone bodies and organic acids made on some of the urines. Although the ketosis in the first case was slight it is difficult to regard it as due to carbohydrate starvation, as it was detectable at the

FIG. 2.—Diagram to illustrate the ammonia excretion in two sea-sick persons.

onset of vomiting only six hours after a solid meal taken in comfort and retained; and the small amount of food (mainly carbohydrate) taken and retained after vomiting began should have been sufficient to prevent ketosis. Again, another subject (J. M.) developed ketosis although he retained all but one meal, and the meals taken were mainly carbohydrate.

In Case A. S., which is a fair sample of three cases in which no food was taken, the ketosis was more severe than is usual in starvation in adults. It is true that Folin (1915) found a similar degree in a fat woman who was starved for therapeutic reasons, but that case was quite exceptional, and we have not seen anything comparable in epileptics, nor in a fat woman who was starved to reduce her weight. However, in young children ketosis of this degree may occur (Ross and Josephs, 1924).

In the three cases in which we estimated blood sugar (see Table I) we did not find the rapid fall noted by Oriel: the figures are such as are usual in normal persons before breakfast.

TABLE I

	Blood Sugar	Nitroprusside Reaction
Case A. M.	Per cent.	
At onset of vomiting ...	0.14	+
Seventeen hours later ...	0.09	++++
Twenty-seven hours later ...	0.12	++++
Case J. M.		
At onset of vomiting ...	0.10	0
Fourteen hours later ...	0.10	++++
Case J. T.		
Seven hours after onset of vomiting	0.08	++++

It appears, therefore, that carbohydrate starvation alone will not account for the ketosis.

Relation of Ketosis to Symptoms

Ketosis may produce symptoms either owing to the toxicity of the ketone bodies, particularly aceto-acetic acid, or by a disturbance of the acid-base balance such as may be produced by any acid—that is, an acidosis in the strict sense of the word. A good deal is written still about ketosis and “acidosis” in relation to sickness, especially in children. In order to appreciate the effects of ketosis it is well to consider cases occurring in diabetes mellitus. We find here that patients may excrete large quantities for long periods without nausea, vomiting, or mental changes. Joslin mentions a patient who excreted 3 grams of ammonia (=2,000 c.cm. decinormal solution, normal amount) daily for a year, and was able to travel.

It appears, then, that the effects of moderate amounts of ketone bodies are apt to be exaggerated. It might be argued that such patients have gradually become accustomed to large amounts of ketone bodies; we give, therefore, an example (Case F., in Figure 2 and Table II)

TABLE II

Days:	1	2	3	4	5
Case F.					
Ammonia	970	618	990	2,580	1,930
Ketone bodies	500	384	860	2,030	1,380
Case A. M.					
Ammonia	308	290	—	—	—
Ketone bodies	—	136	—	—	—
Case A. S.					
Ammonia	348	983	1,190	—	—
Free acid	334	544	385	—	—
Organic acid	567	1,576	1,184	—	—
Ketone bodies	175	—	—	—	—
Case A. E.					
Ammonia	225 285	—	—	—	—
Free acid	150 273	—	—	—	—
Organic acid	328 537	—	—	—	—
Ketone bodies	— 353	—	—	—	—
Case J. S.					
Ammonia	—	364	—	—	—
Free acid	—	286	—	—	—
Organic acid	—	568	—	—	—
Ketone bodies	—	257	—	—	—

The excretion of ammonia, organic acid, and ketone bodies in one diabetic and four sea-sick subjects; all urinary constituents reckoned in cubic centimetres of decinormal solution. Except in Case A. M. the estimations on the sea-sick subjects began twelve hours after vomiting started. Estimations in Case A. E. made in two twelve-hour periods.

of ketosis of sudden onset without symptoms. This was a young diabetic patient with a short history of illness who had been treated with insulin; the insulin was temporarily stopped and ketosis appeared. Although the ketosis was more severe even than in the subject A. S., there was no nausea or vomiting. Compared with such a case the ketone bodies excreted by A. M. are trivial. When we take into account the fact that nausea and depression may last for several days, and vomiting begin without ketosis, it is impossible to regard ketosis as the cause of the symptoms.

In Case F. we can see the degree of disturbance of acid-base balance that may be expected in such a ketosis of short standing; on the fifth day the plasma bicarbonate had fallen to 49 (normal 55–66); such a degree of acidosis occurs in normal persons after exertion, and is accompanied by no unpleasant sensations—rather the reverse. It is improbable that more than slight disturbance of acid-base balance occurred even in the cases

of sea-sickness that were starved. We examined one patient (J. W.), who had vomited repeatedly for two days, but had retained some food. His urine gave a +4 nitroprusside reaction; his plasma pH was 7.33 and his plasma bicarbonate was 62; no acidosis had developed in this case.

There is no evidence that acidosis, in the strict sense of the word, will produce vomiting or any other symptoms but hyperpnoea. In fact, the symptoms of sea-sickness suggest far more the collapse seen with alkalosis. The possibility that an alkalosis might be produced either by hyperpnoea or by loss of acid in the vomit is refuted by the fact that all the urines examined were acid. In Case J. W. the plasma chloride was normal (0.63 per cent.), so that there was no evidence of excessive loss of hydrochloric acid by vomiting. Ketosis therefore appears to be an incident in sea-sickness which is not itself the cause of any of the symptoms, but is evidence of some metabolic change which may itself be the cause. What this disturbance is, is not clear; it cannot be frank carbohydrate starvation. There are, however, other conditions in which ketosis appears—for example, alkalosis, pregnancy, and in occasional liver diseases. It is unfortunate that the nitroprusside reaction is not more commonly used in the routine examination of urines, as much knowledge about the occurrence of ketosis might be gained. Children seem particularly prone to ketosis, both in starvation and in fevers. The exact relation of ketosis to cyclic vomiting in children is still unsettled, but the position seems to be very similar to that in sea-sickness.

During a calm voyage we examined the urines of patients who were liable to sea-sickness on rough voyages, but found ketone bodies in none. Also in Case A. M., although ketosis appeared at the onset of vomiting, it was not detected on previous examinations nor during a subsequent voyage when the subject was nauseated and her blood pressure low.

We have therefore found no evidence of a special liability to ketosis in persons subject to sea-sickness.

Conclusions

Ketosis is usual in sea-sickness after vomiting has begun, and occasionally appears earlier.

Ketosis is not the cause of sea-sickness nor of any of the symptoms of sea-sickness.

It may be regarded as evidence of some metabolic disturbance the nature of which is not known.

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Memoranda**MEDICAL, SURGICAL, OBSTETRICAL***** A LARGE OCCIPITAL MENINGOCELE**

The following case, which presents certain features of interest, seems worthy of record.

The patient, a Hindu female child aged 3 months, was brought to hospital on June 2nd, 1930. There was a tumour, rather larger in size than the child's head, springing from the occipital region, to which it was attached by a thick pedicle. The tumour was fluid and fairly flaccid. The skin covering it was healthy, though beginning to show signs of malnutrition at the most dependent part. Pressure on it did not lead to any signs of increased tension within the skull, nor did it lead to any decrease in the bulk of the tumour. The child was fairly well nourished and had, the parents alleged, increased in weight at a normal rate since birth. The tumour had been present at birth, when it was about the size of an orange, and had steadily increased in size since then; but the general health of the child had been good, and, apart from