

The Mechanism of Recurrent Vomiting after Mild Head Injury in Children

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I. Introduction

It is a common experience that infants and children with mild head injury show recurrent vomiting and somnolence in the early posttraumatic period.

When the children become somnolent and show facial pallor and vomiting soon after a head injury, their parents bring them to a hospital as emergency cases, with anxiety about traumatic intracranial hematoma, on not a few occasions. However, in a great majority of those cases, the clinical findings and adjunctive examinations rule out the existence of traumatic intracranial hematoma, and vomiting rapidly subsides on IV fluid therapy. The children get well by the following day, without remaining neurologic symptoms.

Therefore, recurrent vomiting after mild head injury in children occurs through a mechanism different from that of vomiting after head injury in adults (i.e. vomiting as increased intracranial pressure symptoms due to traumatic intracranial hematoma and cerebral edema or as meningeal irritation due to traumatic subarachnoid hemorrhage).

Even now, however, there is no established theory as to the mechanism of vomiting after mild head injury in children. The present study was undertaken to elucidate the mechanism of its onset.

II. Clinical Materials and Methods

To characterize this peculiar type of head injury, the patients were chosen in whom recurrent vomiting and somnolence occurred in the early stage of trivial and mild head injury and for whom no surgical treatment was subsequently necessary.

The head injury was defined as mild when there was no initial loss of consciousness or only brief loss of consciousness just after the trauma and when there was neither convulsion nor lobe syndrome. Cases with depressed fracture and intracranial hematoma were excluded.

Sixty-eight children, whom we treated over a period of 3 years, were categorized as having this peculiar type of head injury and studied.

Fig. 1 shows the age distribution, ranging from 11 months to 13 years. The boy to girl ratio was 47 : 21.

Key words: Head injury, Recurrent vomiting, Somnolence, Central ketosis, Children.

索引語: 頭部外傷, 反復性嘔吐, 傾眠, 中枢性ケトン症, 小児.

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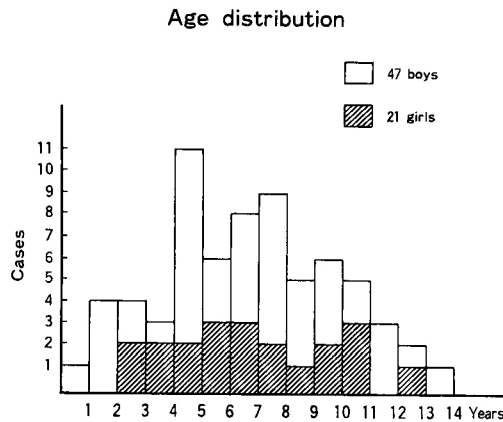


Fig. 1. Age distribution

The causes of trauma were a simple fall in 25 cases (36.8%), a fall from a height in 25 cases (36.8%), sports in 10 cases (14.7%) and a fall from a bicycle in 8 cases (11.7%). Thus, the trauma was not due to violent external force in any case.

The site of injury, which was known in 62 cases, was as follows: occipital region, 26 cases; frontal region, 16 cases; temporal region, 12 cases; and parietal region, 8 cases. Occipital impact was most frequently seen.

Patients were examined with special attention to the state of consciousness, facial pallor, neurologic findings, fundoscopic findings, pulse rate and blood pressure. In addition, laboratory examinations, including urinary ketone bodies, blood glucose and electrolytes, skull X-rays, CT scan and electroencephalography were performed.

III. Results

The clinical course of mild head injury in children is characterized, in many cases, by recurrent vomiting, facial pallor, transient somnolence and ketosis several minutes or several hours after the sustained injury. Recurrent vomiting started within 1 hour after head injury in about half of the cases (36/68, 52.9%).

Fig. 2 shows the interval between head injury and onset of vomiting and the frequency of vomiting. This figure shows that vomiting commenced in 5 to 10 minutes after injury in early cases, and after 30 to 60 minutes in many cases. Facial pallor and somnolence occurred in about half of the cases (35/68, 51.5%). Ketonuria developed in 43 out of 68 cases (63.2%). Ketonuria tended to appear more frequently in the children who vomited frequently (Ketonuria was observed with higher frequency in those who vomited 5 times or more.). However, there was no relationship between the time of onset of vomiting and positive ketonuria.

Fig. 3 represents pulse rate and blood pressure. Bradycardia or pressure pulse was not seen. Hypotension was not found, either.

Fig. 4 shows blood sugar levels. The patients showed normal blood sugar levels or slight

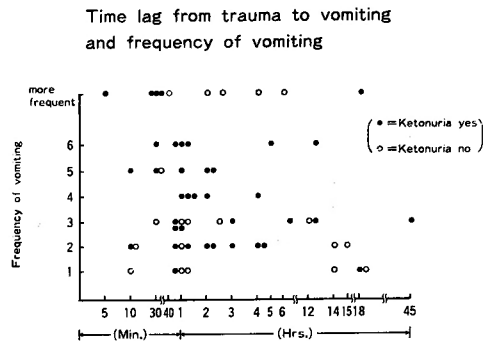


Fig. 2. Time lag from trauma to vomiting and frequency of vomiting

hyperglycemia; no hypoglycemia was encountered. Serum electrolyte values were within normal limits.

Table 1 lists clinical and laboratory data in children with mild head injury as compared with those found in children with acetonemic vomiting^{3,7)}. Both groups have much in common, i.e. both groups have the same age distribution and show frequent occurrence of somnolence, facial pallor, vomiting, ketonuria and leukocytosis. In other words, clinical manifestations after

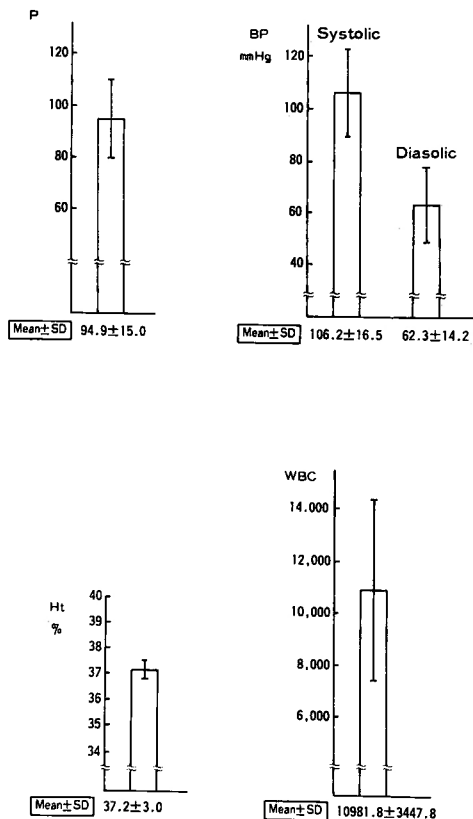


Fig. 3. Pulse rate, blood pressure, Ht and WBC

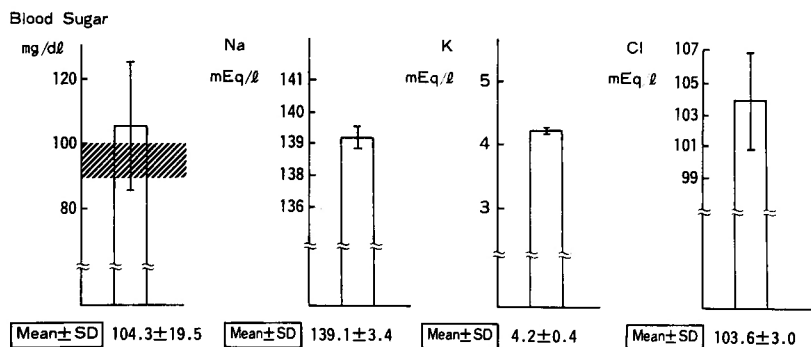


Fig. 4. Blood sugar and serum electrolytes

mild head injury in children seem to be similar to those seen in acetonemic vomiting.

Optic fundi were all normal without retinal hemorrhage or papilledema.

CT scans were all normal with no traumatic intracranial hematoma.

In 68 patients who underwent plain roentgenography of the head, 13 children suffered from skull fracture. In all cases, it was a linear fracture. Ketonuria was found in 10 of 13 cases with skull fracture and in 33 of 55 cases without skull fracture.

Table 2 summarizes EEG findings in 18 cases as recorded within 24 hours after initial vomiting. The EEG was abnormal in 17 of the 18 cases. The main findings were polymorphous focal slow waves.

Table 1. Comparison of clinical manifestations and laboratory findings between mild head injury and acetonemic vomiting

	Age	
	Mild head injury 11 mos → 12 yrs	Acetonemic vomiting (Cyclic vomiting) 1 yr 6 mos → 8 yrs
Symptoms and signs	Somnolence	⊕ 34.2%
	Pallor	⊕ 57.1%
	Headache	⊕
	Vomiting	⊕ 100%
	Motor and sensory disturbances	⊖
	Convulsions	⊕
	Papilledema	?
	Retinal hemorrhage	?
	Pathological reflex	⊖
	Concave abdomen, Bruit of femoral artery	⊕
Laboratory examinations	P	94.9 ± 15.0
	BP	Systolic 106.2 ± 16.5 Diastolic 62.3 ± 14.2
	Ketonuria	⊕ 63.6%
	Ht	37.2 ± 3.0
	WBC	10981.8 ± 3447.8
	Blood sugar	104.3 ± 19.5
	Serum electrolytes	Normal
	EEG	Focal slow
		Tachycardia & feeble pulse
		Lowering of diastolic BP

Table 2. EEG findings of 18 cases within 24 hours after recurrent vomiting.

Normal	1 (5.6%)		
		Focal slow	15
		Monorhythmic theta ...	1
		Spike	2
			Parietal
Abnormal	17 (94.4%)		
		Occipital ...	12
		Temporal ...	4
		Frontal ...	2
		Parietal ...	1
		Hemisphere ...	1

Fig. 5 represents tracings of a 6-year-old boy with mild head injury, who suffered a blow on the occipital region. Polymorphous focal delta waves appear in the left occipital region.

Clinically, recovery was rapid and the symptoms disappeared within 2 or 3 days. However, the EEG abnormalities remained in many cases for 2 or 3 weeks.

Meanwhile, out of 10 children who were subjected to a follow-up EEG, 7 showed normal EEG findings within one month after injury and 3 had persisting abnormalities for over one

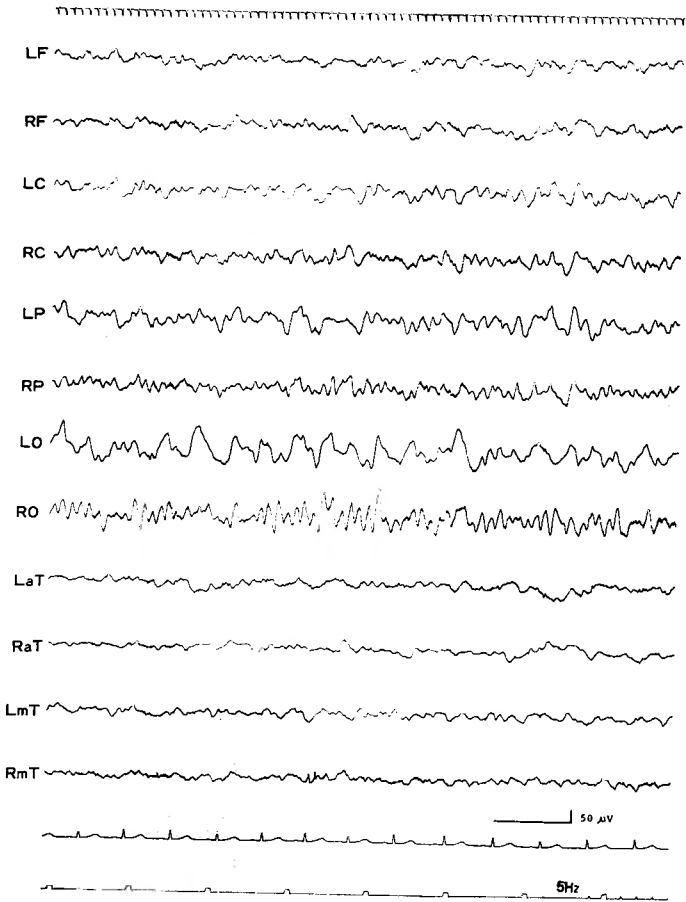


Fig. 5. Poymorphous focal delta in left occipital area from a 6-year-old boy with mild head injury

month.

These patients were observed for the clinical course, as inpatients on the whole. Because recurrent vomiting made it impossible to take water orally, a solution of sugar with electrolytes (e.g. dextrose with lactate-Ringer's solution) was infused in order to inhibit the β -oxidation of fat and to promote the circulation of the TCA cycle. On this occasion, hypertonic solutions of urea, mannitol and glycerol, which were used for the treatment of cerebral edema, were contraindicated. In many cases, 200 ml to 500 ml of infusion settled vomiting. The children got well on the day following the treatment without neurologic sequelae. Urinary ketones became negative.

IV. Discussion

It has been said that vomiting occurs in approximately one third (32%)⁸⁾ of children with head injury. In the present study, we selected patients with mild head injury accompanied by vomiting to elucidate the mechanism of vomiting.

So far the following theories have been advocated as to etiology of recurrent vomiting after mild head injury in infants and children.

1) Traumatic cerebral edema theory

Such an early onset of vomiting as one hour after injury does not usually occur with cerebral edema. From the fact that infusion of hypertonic solutions was ineffective, while electrolytes with sugar solution was effective, the cerebral edema theory could not be advocated. Considering the time of onset of vomiting and the response to treatment, therefore, there is little possibility that vomiting is due to traumatic cerebral edema.

2) Traumatic spreading depression syndrome theory

Recently, Oka et al.¹¹⁾ have launched a traumatic spreading depression syndrome theory.

This syndrome is also seen in children with mild head injury. Although this syndrome has much in common with the symptoms in our cases, it involves convulsions and transient hemiplegia. Reference is not made to ketosis. On the basis of clinical status, we classify this syndrome into a different category from our cases.

3) "Syndrome of cerebral concussion in children" theory

This theory was published by Schnitker¹²⁾, who considered that cerebral edema caused by external force extended to the midbrain, resulting in the occurrence of autonomic nervous system symptoms. However, Masuzawa⁸⁾ critically stated that cerebral concussion was a disease state characterized mainly by a disturbance of consciousness immediately after trauma and was therefore unsuitable for explaining a condition which appears after a lag.

4) Juvenile head trauma syndrome theory

This is a theory advocated by Haas et al.⁴⁾, who ascribed vomiting and somnolence to angiospasm of intracranial blood vessels induced by trauma. However, Masuzawa⁸⁾ gave

a comment that it appeared unthinkable that the angiospasm developed primarily.

5) Autointoxication theory

After Masuzawa⁸⁾ criticized Schnitker's and Haas et al.'s theories, he pointed out that autointoxication played a great part in the mechanism of vomiting after trauma in children.

6) Shock-induced metabolic disorder theory

Uetsuhara et al.¹³⁾ reported that shock at the time of head injury produced metabolic disorders. Inhibited glycolysis and increased fat metabolism ensued together with clinically increased ketones and its associated metabolic acidosis. It is noteworthy that they pointed out that metabolic acidosis seen at shock was ultimately the same metabolic disorder occurring in cyclic vomiting peculiar to children, i.e. ketonemia with metabolic acidosis.

From the above-mentioned theories of etiology, the relation to acetonemic vomiting (cyclic vomiting=autointoxication) has been in the limelight.

Taking into consideration various theories of etiology together with our clinical findings, we describe our opinion on the vomiting mechanism.

1. Mechanism of vomiting

As shown in Table 1, clinical symptoms and laboratory data of mild head injury in children accompanied by frequent vomiting are similar to those of acetonemic vomiting^{3,7)}. When the mechanism of vomiting was considered from these clinical findings, the following conclusions were drawn: frequent vomiting seen after mild head injury in children was not due to vasovagal collapse or to increased intracranial pressure, judging from the pulse rate, blood pressure and fundusoscopic findings; they were not a sequel of electrolyte imbalance or hypoglycemia, either.

It has already been described that in 43 (63.2%) of 68 cases, ketonuria was seen.

Ketosis is a condition in which ketone bodies, i.e. acetone, aceto-acetic acid, β -hydroxybutyric acid, are increased in the blood and excreted in the urine⁵⁾.

Ketosis appears in the following two pathologic metabolic processes^{5,7,9)}.

First, a deficiency of sugar supply into the TCA cycle (e.g. diabetes mellitus, starvation) produces ketosis (Fig. 6).

Second, although sugar supply is not deficient and TCA cycle functions normally, hypermobilization of fat and accelerated β -oxidation⁵⁾ leads to excessive acetyl CoA, not used sufficiently, thus resulting in ketosis (Fig. 6). In other words, when acetyl CoA supply exceeds the treating capacity, viz, the circulation rate for TCA cycle is relatively exceeded, excessive formation of ketone bodies appears.

The conditions which are known to involve ketonuria are: a) acetonemic vomiting, b) diabetes mellitus, c) starvation and d) ketotic hypoglycemia. From the laboratory data and clinical course, b), c) and d) are excluded, and a) remains as a possibility.

When emphasis is laid on ketosis found in about two-thirds of the cases, ketosis found after mild head injury is not due to a deficiency of sugar supply judging from blood sugar levels. The ketosis may be central ketosis¹⁾ like that of acetonemic vomiting, which appears as a result

Schema of Ketone Body Metabolism

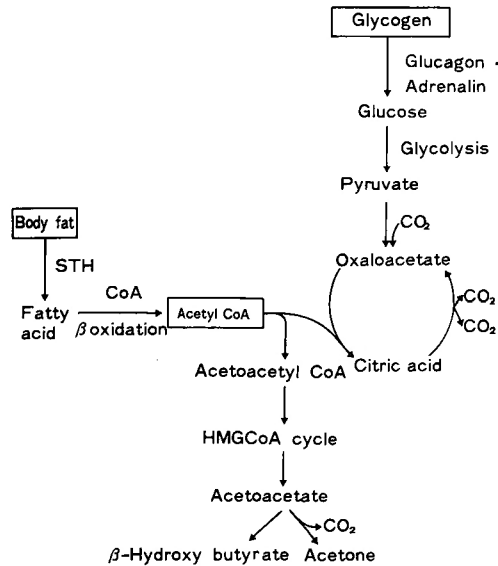


Fig. 6. Schema of ketone body metabolism [cited from Harper (1971)]

of hypermobilization of fat and acceleration of β -oxidation and fatty acid formation.

Ogawa^{9,10}) reported that central ketosis was caused by stimulation of the parasympathetic zone in the anterior hypothalamic region. It is thus found that the hypothalamus plays an important role in fat metabolism.

Next, we discuss that characteristic symptoms seen after mild head injury, i.e. somnolence, facial pallor, vomiting and ketonuria are seen not in adults but only in children. Is it not possible that clinical progression of these symptoms in children only is attributable to immaturity of the brain in childhood? It has been said that in children, as shown in Fig. 7^{9,10}), the neocortex of the cerebrum is underdeveloped, and its inhibitory action on the limbic system is insufficient and unstable. When trauma (i.e. stress) takes place, functional separation or disharmony⁹) occurs between the cerebral neocortex, on the one hand, and the hypothalamus and limbic system, on the other. Consequently, vomiting, autonomic disturbance and ketone metabolic disorder, in which the hypothalamus and limbic system play the leading role, may be induced.

It is known that the center which regulates ketosis is located in the parasympathetic zone of the anterior hypothalamus^{9,10}). The sleep center⁶) is also located in the anterior hypothalamus. Moreover, the vomiting center is said to be present in the hypothalamus. Hess^{7,13}) found experimentally the existence of a site in the hypothalamus adjacent to the mammillary body which when stimulated caused vomiting. Arima²) reported that stimuli to the area in the vicinity of the amygdaloid nucleus and hippocampus, which are closely related to the autonomic center in the hypothalamus, produce movement of the intestinal tract and diaphragm, leading to vomiting. That the vomiting center is present in the hypothalamus or hypothalamus/limbic system appears to be the case.

Neocortex and limbic system in children

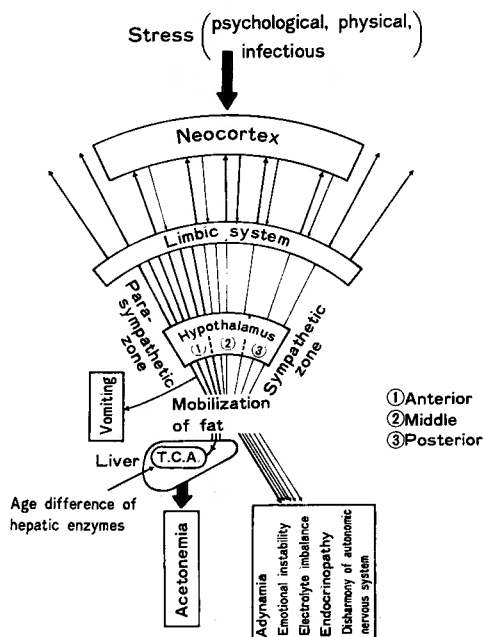


Fig. 7. Neocortex and limbic system in children [cited from Ogawa (1965, 1972)]

As described above, Ogawa^{9,10)} reported that ketosis seen at the time of disorders of the central nervous system, particularly autonomic disturbance, is a result of hypermobilization of fat and accelerated β -oxidation and described the involvement of the parasympathetic zone of the anterior hypothalamus in the primary mechanism of ketosis. It is of interest that the sleep center⁶⁾ and vomiting center^{7,13)} are also located in the hypothalamus, as well as the center producing ketosis.

If somnolence, facial pallor, vomiting and central ketosis occur as a result of a lesion in a particular site of the brain, the region which can provide a consistent explanation for these symptoms is only the hypothalamus.

It is presumed that a stress, trauma, laid on the underdeveloped brain causes, through the hypothalamus and limbic system, somnolence and metabolic disorder of ketone bodies and vomiting as well.

In a further study, we hope to elucidate the mechanism of vomiting using adjunctive diagnostic aids, such as CT scan and endocrine function tests.

2. EEG

In 17 out of 18 cases where electroencephalography was performed within 24 hours after injury, EEG abnormalities were found. The main abnormal findings were polymorphous focal slow waves.

Considering waveforms, it appeared that EEG abnormalities in our cases were not due to

central ketosis but caused by the impact of head injury itself. External symptoms are at times misleading, when children are involved into head injury. It is known that children are not inclined to lose consciousness even when an impact of considerable force has been sustained to the head. Even when loss of consciousness is not seen immediately after injury, there is a possibility that an impact of considerable force has been applied to the brain. EEG abnormalities (polymorphous focal slow waves) seen in the present study suggested local brain injury at the site of impact sufficient to act as a stress may be applied to the underdeveloped brain.

3. Treatment

The treatment for mild head injury in children accompanied by vomiting begins with the recognition of characteristic symptoms and signs.

Fortunately, in recent years, the availability of CT scan has permitted easy exclusion of traumatic intracranial hematoma.

The children cannot take water orally. In addition, vomiting rapidly puts the children into dehydration. Therefore, for a period when oral ingestion is impossible, it is necessary to bear in mind the water, electrolyte and acidbase balances.

In the present study, the hematocrit value and electrolytes were within normal limits, and hypoglycemia was not seen. However, in order to inhibit the β -oxidation of fat and to promote the circulation of the TCA cycle, water and sugar were supplied. On the other hand, an infusion of electrolyte-free dextrose solution alone occasionally causes fatal side effects in dehydrated children. Because vomiting causes somewhat a loss of electrolytes, a drip infusion of dextrose with electrolyte solution (e.g. dextrose with lactate-Ringer's solution) is indicated.

If vomiting persists after drip infusion, antiemetic agents should be administered, after excluding intracranial organic disease.

V. Conclusion

From the clinical and EEG findings, it is postulated that head injury acts on the immature neocortex of children as a stress.

Functional disharmony may then develop between the neocortex and the limbic system, resulting in (1) central ketosis by stimulation of anterior hypothalamic area, (2) somnolence by stimulation of the "sleep center" in anterior hypothalamus and (3) recurrent vomiting by stimulation of the "vomiting center (Hess)" in hypothalamus.

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和文抄録

小児軽症頭部外傷後の嘔吐の発現機序

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山本 豊城, 尾形 誠宏

1) 小児軽症頭部外傷後早朝に、反復性嘔吐をきたした68症例について検討し、嘔吐の発現機序の解明を試みた。

2) 嘔吐と同時に、傾眠、顔面蒼白、ケトン症がしばしばみられた。このケトン症は、中枢性ケトン症と考えられる。

3) 視床下部には、ケトン症をおこさせる中枢が存

在し、また、睡眠中枢と嘔吐中枢の存在も知られている。

4) 以上から、対象症例のなかには、外傷 (stress) によって小児の未発達な新皮質と視床下部・辺縁系との間に機能的な不調和が生じ、視床下部・辺縁系をとおして、ケトン体代謝異常、傾眠、自律神経系異常が起こり、さらには嘔吐をきたすものと推論される。