Their Children's Teeth

We also examined seven children born of these women ; all of them had severe rickets. The results in Table II show that in 144 teeth of these rickety children only two cavities were present, confirming the results obtained in the group of children with rickets previously recorded.

TABLE II.—*Children with Severe Rickets Born of Women with Severe Osteomalacia*

Cases	Age	Clinical Rickets	Total Teeth Present	Cavities	Hypoplasia
1 (Child of osteomalacia case 1)	2 years	+	18	—	—
2 (" " " 11)	4 "	++	20	—	—
3 (" " " 14)	2 "	+++	15	1	—
4 (" " " 18)	3 "	+++	20	—	+
5 (" " " 18)	9 "	+++	23	1	—
6 (" " " 18)	15 "	+++	28	—	—
7 (" " " 22)	2½ "	+++	20	—	++
Total			144	2	—

Note.—In the 7 children only 2 cavities were present in a total of 144 teeth.

One of us (C. D. M. D.) has been studying dental fluorosis in the Punjab in recent years. In the plains of this province dental fluorosis is very common ; in some districts all children have mottled enamel. It has been recently suggested in America that mild degrees of fluorosis prevent dental caries. In the Kangra District of the Punjab dental fluorosis is conspicuous by its absence. An occasional child in whom we saw it there, among the many hundreds examined, had come from another district and had not been brought up in Kangra. Fluorine intoxication need not therefore be considered as a possible immunizing factor.

Conclusions

The above results in these twenty-two women, who appear to be typical of many thousands in this district, show that the teeth may be nearly perfect although severe decalcification of the bones has occurred as the result of osteomalacia. It is a common assumption, at the present time, that during pregnancy women's teeth show a greater susceptibility to dental caries ; and much money is spent on calcium and vitamin treatment in Western countries as a protective measure. In the light of our findings we consider, therefore, that the question of the association of dental caries with pregnancy needs more careful study.

We also conclude that as we found nearly perfect teeth in children and women with severe rickets and osteomalacia the calcification and decalcification of the bones of the body are not necessarily directly connected with the same processes in the teeth.

At a later date it is hoped that biochemical work on these cases of severe calcium deficiencies will be done in the Kangra District.

Suzanne Rossier (*Thèse de Paris*, 1940, No. 214), who records a fatal case in a woman aged 45 (the diagnosis was confirmed by a virulence test), states that diphtheritic septicaemia presents a characteristic clinical picture. The blood culture is positive, while cardiac signs are inconstant, but an acute malignant endocarditis is always found post mortem. The issue is always fatal. Diphtheritic septicaemia is the consequence of a previous bacteraemia, which would probably be found to be less rare than is supposed if minute and systematic investigations were made such as examination of the blood and urine during life and of the organs after death. The pathogenesis is still obscure, as an anatomical study does not show under what conditions the diphtheria bacillus, which is usually confined to the initial lesion, can pass into the blood. Possibly the necrotic ulcerative character of malignant diphtheria favours this passage, or a special virulent form of the organism may be responsible.

ETHER CONVULSIONS

BY

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Although I have been observing anaesthesia by ether almost daily for twenty-five years it was not until seven months ago that I saw a case of serious ether convulsions. I happened to go into the theatre ; an operation for acute appendicitis upon a girl of 13 had just been completed and artificial respiration was in progress. The patient was having convulsions of the most violent character ; at the height of the spasm the lower limbs literally banged upon the table. These fits, I learned, had been going on with increasing severity for a quarter of an hour. It was not long before the cardiac centre failed, and in spite of the combined efforts of the anaesthetist and surgeon resuscitation was ineffectual.

These ether convulsions reminded me exactly of a case of novocain poisoning which had been engraved indelibly upon my memory. Convulsion followed convulsion until the respiratory centre failed, but the pulse remained good. CO₂ and oxygen was administered, but the convulsions continued. The cardiac centre then gave out. I performed cardiac massage and the heart restarted, and three hours later the patient's condition was tolerably satisfactory. Approximately three hours later still, during the night, the convulsions restarted and proved fatal.

My interest in anaesthetic convulsions having been awakened, I consulted the literature. I could find no satisfying explanation of the phenomenon. It appeared that most cases of ether convulsions have occurred during the hot weather. (The case of ether convulsions I had observed occurred on a cold winter night.) The treatment recommended was to keep the lungs inflated. Ten c.cm. of a 10 per cent. solution of calcium gluconate intravenously had been advised, and in several instances the intravenous injection of evipan had put a stop to the convulsions. Momentary compression of the common carotid arteries had also been put forward as a suggested method of treatment. In discussing the subject with colleagues I learned that one had seen a successful issue from douching the patient with ice-cold water.

Recent Cases

Case 1.—Six weeks later, during the exceptionally cold weather of January, 1940, I had just removed an early acutely inflamed appendix from a stout but athletic man of 30. Ether convulsions started. First of all there were twitchings of the muscles of the face, and later spasms of the abdominal muscles, making closure of the incision difficult. So great were the spasms that the sutured internal oblique tore out and the peritoneum was pulled asunder. My attention was directed mainly to closing the abdomen, but I suggested to the anaesthetist calcium gluconate. There was none in the theatre. I then suggested evipan, and this was got ready. By this time, in spite of difficulty, I had succeeded in closing the abdomen. The spasms continued with increasing severity ; the legs banged upon the table just as in the case I have described above. The respiratory centre then failed, and artificial respiration was begun. The anaesthetist advised against giving the evipan which had been prepared, saying the patient was already deeply anaesthetized. We persevered with artificial respiration and the pulse remained good. Ten minutes later the heart failed. I carried out intracardiac injection of adrenaline and later performed cardiac massage, but without avail.

The bottle of ether was sealed and sent to Messrs. Duncan, Flockhart for analysis. It had been purchased recently and kept in the dark. Messrs. Duncan, Flockhart had the contents analysed and it was

found in perfect condition. Their representative told me that they had kept a record of all cases of ether convulsions brought to their notice. Most of these had occurred in the hot weather and the patients were often obese. They could offer no specific instructions for treatment. I also discussed the case with several professional anaesthetists, who were in agreement that evipan should not be administered to a patient who was already deeply anaesthetized.

Two weeks ago I saw two further cases of ether convulsions.

Case 2.—This patient was a woman of 40, with gallstones, on whom cholecystectomy was being performed. The convulsions were just beginning, and at this early stage a capsule of calcium gluconate was administered intravenously forthwith. After three minutes no further twitchings occurred.

Case 3.—The next day, to my dismay, while operating upon a case of stone impacted in the common bile duct, ether convulsions started. Calcium gluconate was given at once, this time without effect. Convulsion followed convulsion in the sequence I have described. This patient, unlike the others, was a man in extremely poor condition, and I knew too well that unless a powerful antidote was forthcoming immediately the case would certainly end fatally. I remember thinking that the patient was deeply anaesthetized and, further, that evipan is especially contraindicated where the liver function is depressed. Even so, I insisted on the administration of evipan. The fits by now had reached alarming proportions, and when 3.5 c.cm. of evipan had been injected a fit was at its height; the patient gave a long inspiration—almost a yawn—and from that second the fit ceased, and no more followed. I completed the operation while the anaesthetist administered CO₂ and oxygen. The pulse was steady but the volume poor. As soon as the CO₂ and oxygen was discontinued respirations became feeble. They were not exactly of the Cheyne-Stokes variety, but rather a series of weak inspirations followed by a feeble sigh, during which the lower jaw twitched. After waiting a quarter of an hour there was no substantial improvement. I therefore suggested an injection of lobeline. Within five minutes there was some improvement, and a second injection was given with a remarkable increase in respiratory ventilation. The patient recovered.

Commentary

No doubt exists in my mind, or in the minds of those who were present with me in the theatre, that *the* treatment for ether convulsions is the early administration of even a small dose of evipan.

So universal is the opinion that ether convulsions are in some way connected with hot weather that it would be impudent for me to suggest that this theory is but idle conjecture. Yet, seeing that two cases occurred during the coldest weather within living memory, my experience is diametrically opposed to this theory. I therefore put forward a suggestion. Overheating it may be, but this overheating is due neither to the outside temperature nor to the temperature in the theatre, but it might be due to the temperature of the water which heats the ether in the modern anaesthetic apparatus. Are anaesthetists in agreement as to the temperature at which the water which surrounds the ether bottle should be kept? I have not noticed that this is regulated by a thermometer.

A. NANSOT (*Thèse de Paris*, 1940, No. 312), who records five cases in men aged from 44 to 56, states that the complications of lumbar puncture may be classified in four groups according as they are due to hypotension, hypertension, inflammatory reaction of the meninges, and shock. Fatal cases seem to be due to the last cause and generally occur in subjects with arterial hypertension, in whom there is a sudden vasodilatation with a perivascular exudation. Prophylaxis consists in a gradual reduction of the spinal decompression and a limitation of the amount of fluid withdrawn.

Clinical Memoranda

An Unusual Ileo-caecal Membrane

The discovery of peritoneal membranes complicating the usual anatomy of the ileo-caecal region is not uncommon during appendicectomy. In the case described a membrane not previously reported was found.

CASE RECORD

A boy of 14 had suffered from five attacks of subacute appendicitis during the preceding two years. In a quiescent interval the abdomen was opened by a gridiron incision, when considerable difficulty was found in delivering or even visualizing the appendix. It was soon clear that a semi-transparent membrane covered the caecum. At first this was thought to be an example of Jackson's membrane, but its attachments and the direction of its fibres were quite different. Proximally it originated from the antimesenteric border of the terminal six inches of ileum, forming a broad sheet the fibres of which ran downward and outward in front of the caecum and appendix, blending with the posterior parietal peritoneum lateral to and below the caecum and attached to the pelvic brim medially (see illustration). A cul-de-sac

was thus formed into which the caecum descended more deeply with each inspiration. No other anomaly was found; the caecum was completely peritonized, the appendix was retrocaecal and singularly free of adhesions, and a Meckel's diverticulum was not present. The membrane was well supplied with long parallel vessels, and on division was seen to consist of two serous layers separated by a thin plane of areolar tissue.

DISCUSSION

A number of different types of band or membrane may be found in this region. Excluding those associated with a Meckel's diverticulum, there are, first, those which are anatomically normal—the meso-appendix, the ileo-caecal fold, and probably also the ileo-colic fold. Secondly, there is a structure which, though anatomically normal, can only appear with a certain type of caecum. The retroperitoneal caecum is fixed and the wholly peritonized type quite free; but when the degree of peritonization just falls short of the latter the caecal fold is produced, running laterally from the posterior aspect of the caecum and bounding the caecal fossa. Thirdly, it is probably more common than not to find short fibrous adventitious bands connecting two or three members of the caecum-appendix-ileum triad, and these mostly run on the front of the organs concerned. Fourthly come the adhesions of inflammatory origin, not difficult to recognize, associated with other evidence of past inflammation, irregularly vascularized, and commoner towards the appendix tip.

Finally, there are the definite membranes of which I have described an example—wide structures, well vascularized with leashes of long parallel vessels, and usually independent of the appendix itself. Of these, two are common enough to have been named. Jackson's membrane is a broad filmy sheet attached to the posterior peritoneum lateral to the ascending colon sweeping downward and in over the caecum towards the pelvic brim; some authors describe it as also having a superior origin from the greater omentum. Lane's band is short and thick, connecting the antimesenteric border of about two inches of terminal ileum to the pelvic brim. The one I describe should be classed with these as a well-