PROGRESS IN PEDIATRICS

REVIEW ON DISEASES OF THE NEWLY BORN

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PHYSIOLOGY OF THE STOMACH OF THE NEW-BORN: THE TONUS AND HUNGER CONTRACTIONS OF THE STOMACH OF THE NEW-BORN

Carlson and Ginsburg¹ show tracings of their observations on a number of new-born infants with the results showing that the empty stomach at birth exhibits the typical periods of tonus and hunger contractions of the adult, the difference between the infant and the adult being the greater frequency and the relatively greater vigor of these periods in the young. Observations were made in some of the infants before the first nursing and it becomes clear that the gastric hunger mechanism is complete physiologically and is probably active some time before birth. They come to the conclusions that the periods of gastric tonus and hunger contractions are in evidence shortly after birth and before any food has entered the stomach. When the gastric hunger contractions become very vigorous the sleeping infant may show some restlessness and even wake up crying. If the infant is awake the very vigorous hunger contractions frequently induce crying and restlessness. The writers call attention to the fact in two of the tracings the gastric hunger periods end in incomplete tetanus which is an index of youth and vigorous stomach.

FETAL ERYTHROBLASTOSIS: FETAL ERYTHROBLASTOMATOSIS

Woolley² reviews the causes of congenital general edema in the new-born. Woolley says that in its characteristic form congenital general edema is evident at birth and appears to develop during the period in the uterus. Concerning the cause of this condition something is known of some cases, but nothing of others. He groups the causes as follows: (1) Disturbance in the portal circulation; fetal peritonitis; absence of the ductus venosus Arantii and in certain anomalies of the intestinal tract; (2) anomalies of the heart, of the vessels and of the uropoietic system of the fetus; (3) associated with maternal disease, chiefly nephritis; (4) some metabolic anomaly of the fetus develops which finds expression in abnormal blood production and edema.

1. Carlson, C. J., and Ginsburg: *Am. Jour. Physiol.*, 1915, xxxviii, 29.

2. Woolley, Paul G.: *Jour. Lab. Clin. Med.*, February, 1916, i, 347.

Woolley regards this last group as the most interesting, for in it belong the cases which have been called fetal erythroblastosis. Schridde characterizes the condition as follows: (1) The fetuses, premature, as a rule show universal edema. (2) Most of them show also edema of the placenta and cord. (3) The liver and spleen are markedly enlarged. (4) Microscopic examination of the liver and spleen show unusually large number of erythroblasts with large number of other myeloid cells inside and outside vessels. The liver cells are atrophic and in the spleen the follicles are absent. In other organs extramedullary erythroblastic nodules appear. Mitotic and rhectic figures appear and the liver cells and pulp cells of the spleen are colored with iron-containing pigment. These characteristics Woolley says were shown in their entirety in a case that he had access to. This case was more than usually interesting in that the anasarcaous fetus was one of a pair of twins. The other twin was apparently normal. Schridde believed that this fetal disease was a result of a severe fetal anemia and the anemia it was assumed was of toxic origin. Schridde did not believe it to be syphilitic.

In one of Schridde's cases the mother was nephritic, and in this instance he believed that the fetal condition was coincidental, and not the result of the maternal condition. It is generally supposed that this disease is of toxic origin and that the toxic materials, while causing destruction of the erythrocytes, at the same time stimulate the blood-forming tissue. The origin of the poisons is not known. However, the poisons certainly do not come from the maternal blood.

Woolley says that the anasarca is the expression of the anemia, which in turn is due to the disturbed production, and perhaps also to increased destruction, of erythrocytes. When anatomic defects occur in the body, anasarca also occurs, because the blood, capable though it may be, is not properly oxygenated for obvious reasons.

Woolley suggests that there is something of an analogy between this condition and leukemia, and if this conception has any force, then erythroblastosis may be looked upon also as belonging to a group of tumors. In this case it is the erythroblastic tissue which is involved.

SCLEREMA NEONATORUM

Paterson³ takes up the conflicting views of this disease, which is one of comparative rarity. He writes an interesting historical survey from the first recorded case in 1718 up to the present time. He calls attention to the early errors in the description of the disease.

Paterson says that this disease is most likely to be found where there are general conditions inimical to health. There is no direct proof

3. Paterson, W. S.: *Quar. Jour. Med.*, 1915, viii, 317.

that syphilis plays any part in the causation of this condition. Paterson thinks that the reason this disease has not been known more extensively and described more carefully is from the fact that the disease is so soon fatal, usually in poverty stricken and squalid surroundings, that the disease is not recognized, but is often ascribed to other obvious disabilities. He quotes freely from Parrot and gives him the credit for differentiating clearly the two conditions, sclerema and edema of the new-born.

Finkelstein says that in some cases this condition of sclerema and edema may be associated together. Paterson thinks that the serous infiltration is not an essential feature of the sclerema, but only a secondary sign. Paterson takes Ballantyne's definition of the disease as reasonable, that it is "a rare disease occurring in the new-born, characterized by induration of the subcutaneous tissue and little amenable to treatment." Mensi has reported on eighty-two such cases and he divides sclerema into two types, the first occurring in premature and new-born infants and in the cold season of the year. It follows a previous edema. The induration appears first in the buttocks, never or seldom in the hands and feet. It spreads more or less over the body. The temperature is usually low, seldom febrile and the disease usually ends fatally. Autopsy shows that the skin is atrophied with absence of the granular layer and the cells of the cutis are closely packed together. There was dilatation of the vessels with hemorrhage into the cutis. The second type that Mensi describes is characterized by condensation, thinning and drying of the skin. It occurs less often in premature children but mostly in warm weather and it may begin with edema. On examination postmortem there was found to be atrophy of the dermis, condensation of the fibers, scantiness of the cells and vessels of the skin and abnormal development of fibrous tissue around the fat. Paterson thinks that the large number of cases which Mensi claims to have seen raises some doubts whether they were all true cases of sclerema neonatorum.

The temperature shows some very striking records, Henock reporting one temperature of 83.3 F. and Roger one of 72 F., yet depression of the temperature is not an infallible sign. Considerable variation in the color of the skin appears, yet nothing is characteristic. In the differential diagnosis the condition of edema neonatorum is the most important disease to be differentiated from it. Edema neonatorum Paterson regards as a sign of a disease rather than a definite self-existing condition. Edema neonatorum is found usually, if not invariably, with other recognizable affections, and its distribution is as one would expect to find it, in those situations where the influence of gravity would be most favorable to its appearance. In edema neonatorum

pitting on pressure always occurs, though it is possible that in extreme cases the infiltration may be so firm that no pitting can occur, while in sclerema neonatorum there is no true pitting. Paterson says that he believes sclerema neonatorum to be a well-defined disease, not necessarily associated with or secondary to any recognizable morbid condition. It may present conditions varying widely in degree as to the extent of the local lesions and the severity of the constitutional symptoms and therefore the gravity of the ultimate prognosis.

He reports a case which he had personal observation of, a child born of healthy parents after a normal, quick labor. Thirteen hours after birth the nurse noticed that the child's face was of a dark, bluish color. This cyanosis soon passed off, but recurred in the afternoon five or six hours later. The following night these attacks came on more frequently and more violently. Each attack began with screams, the hands became rigid and fingers clenched, with marked cyanosis of the face. Each seizure lasted about ten minutes. Paterson said he could not detect any cardiac abnormality in this patient. In the morning after the birth of the child Paterson noticed that the buttocks and dorsal region had a rubber-like consistency. There was no pitting even on firm pressure and the skin could not be picked up from the underlying tissue, and even when freely handled did not appear to cause the child any pain. When it was first noticed this firmness was limited to the buttocks and the legs below the knees. Within seventy-two hours after birth the induration was found over the dorsal and lumbar regions, also about the shoulders, face and jaws, with patches over the front of the abdomen and the lateral and costal regions.

During the first week the child was extremely ill and was nourished with difficulty; but after the sixth day the stiffness left the facial muscles and the child became able to swallow. After this stiffness of the face left, it gradually faded away from the thorax and the abdomen, then from the dorsal region and lastly from the outer aspect of the thighs. This process of gradual resolution occupied fourteen or fifteen weeks. Two years after this child was born it was seen again by Paterson and it was found that the child was not in normal condition. It had a reasonably intelligent facial expression, but could not walk or even stand alone and showed an ataxic movement of the feet. There were no signs of rickets, but Paterson felt that the condition in general was suggestive of some cerebellar lesion. The second case that Paterson saw was an illegitimate child born in miserable surroundings with well-marked congenital syphilis. On the thirteenth day after the baby was born it developed a well-marked and extensive rubber hardness over the face and chest, clearly marked on the buttocks, thighs and calves. The child rapidly went to pieces and died when fourteen days old. Sections of the skin in this case do not show any increase of the

fibrous tissue, but there is a well-marked thickening of the rete malpighii.

Paterson goes into the morbid anatomy of this condition, but does not come to any definite conclusions about it because of the marked variations that the different physicians report, and in the etiology Paterson finds no satisfactory explanation of the condition.

The prognosis is always guarded, usually grave, but not invariably hopeless, and it depends to some extent on the local lesions and the presence or absence of complications. The general circumstances of the case will have much to do with the ultimate outcome.

The treatment is mostly symptomatic and nothing is as yet known to be specific for the condition.