BCG Vaccine: Nine Years' Experience at a Chest Clinic [*Summary*]

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BETWEEN July 1950 and the end of 1958 BCG vaccine was given to 664 persons attending the Lewes Chest Clinic, which serves a mixed urban and rural population of some 50,000 persons. Of those vaccinated 642 were under the age of 16. Follow-up to the present time has been carried out wherever possible—though 90 of the 664 have left the area at varying intervals following vaccination.

None of those vaccinated has, so far as is known, contracted tubercle.

Reversion to tuberculin anergy has occurred in 30 cases—in 8 cases only after five or more years of allergy, and in 7 cases after two years or less. Inadequate vaccination is thought to be the cause of 2 of the cases where there was only a short-term conversion period: in the other cases no explanation has been found.

The only complication encountered has been enlargement of regional lymph nodes. That occurred in 22 children, of whom only 5 required treatment. No constitutional upset was seen after vaccination.

There was a family or a close contact history of tubercle in 643 of the cases and 113 of those vaccinated came from homes where there had recently been an infectious case. 29 of the vaccinees were exposed to infection *after* immunization.

As nearly all the child contacts attending the clinic were vaccinated, there has been no adequate unvaccinated group to serve as controls. Primary tubercle has been found amongst the unvaccinated few, however, and it is considered that BCG vaccination has been a useful and simple protective measure.

Pathology of Infantile Hypertrophic Pyloric Stenosis

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THIS enquiry was stimulated by the apparent ignorance of the cause of infantile hypertrophic pyloric stenosis. The macroscopic anatomy (Hirschsprung, 1888), the clinical course, and the management are well understood. The modern operative treatment is that of Ramstedt (1912), guided by Nicoll (1906) and Frédet and Dufour (1908). The results of treatment are very good.

Material.—From August 1955 until April 1959, 70 pyloromyotomies were performed at the Royal Alexandra Hospital without a death. In one of these cases the diagnosis was not confirmed at operation, but the vomiting was relieved, as in a case reported by Cameron (1925). In one further case the diagnosis was proven, but no operation was performed. Biopsy was done in 28 cases, and the segments of the abnormal pyloric muscles were examined.

Post-mortem material has not been available, and such museum specimens as have been examined have been too old to show cell structure.

The normal pylorus.—Cunningham (1906) described well the normal pyloric musculature. The longitudinal coat is uniformly dispersed around the pyloric canal. The more superficial fibres continue over the pyloroduodenal junction, but the deeper fibres leave the surface, penetrate the pyloric sphincteric ring in distinct fasciculi, and end in the bundles of the inner circular coat, or deep to them. The inner coat is a concentration of the circular muscle fibres of the stomach in the length of the pyloric canal, a concentration comparable only to the arrangement of the internal sphincter of the anus. There is no continuity between the circular muscle of the stomach and of the duodenum.

Muscular co-ordination is maintained by Auerbach's myenteric plexus of nerve fibres and ganglia, which lies between the longitudinal and circular muscles. The development of this plexus has been examined by Friesen *et al.* (1956). At the 12th week of fœtal life there is a continuous layer of immature nerve cells, which by the 26th week is organized into definite ganglia. The nerve cells are still immature, but a few show vesicular nuclei. Mature nerve cells do not appear until two to four weeks after birth; these are recognized by their abundant cytoplasm, prominent cell and nuclear membranes, and distinct nucleoli.

The abnormal pylorus.—The biopsies were taken during Ramstedt's operation by incising the muscle parallel to the pyloromyotomy, and deepening this to meet the original incision as near the submucous layer as possible. Biopsy included the two layers of muscle and the enclosed Auerbach's plexus.

The gross hypertrophy of both muscle layers was well shown—a true hypertrophy due to overwork.

Auerbach's plexus was carefully examined and compared with normal ones from a similar age group. No special staining methods were used. Two aspects were considered, the quantity of the ganglia and the quality of the contained cells. The size and number of the ganglia varied widely in both the normal and the abnormal pylorus. In both, two to three ganglia could be seen in most low-power fields, but in the abnormal ones the size of the ganglia tended to be smaller, and the intervals between them greater. In the normal pylorus it was easy to find ganglia arranged in large continuous sheets occupying almost the entire low-power field; such ganglia were never found in the biopsy specimens. In general, therefore, the quantity of ganglion tissue was less in the abnormal pylorus than in the normal.

A ganglion contains nerve cells, supporting cells of Schwann, and nerve fibrils. In the abnormals these cells were more tightly packed. Fewer well-differentiated nerve cells were to be found. The majority of the nerve cells took more stain, and contained less cytoplasm, although normal cells were to be found in varying numbers. There was no obvious relation between the number of normal nerve cells and the age of the subject at the time of operation.

Discussion.—The background of any enquiry into infantile hypertrophic pyloric stenosis must be a consideration of the natural history of the disease. This is not congenital, but is acquired in the first few weeks of life. If the obstruction to the flow of food from the stomach is not so severe as to kill the infant, or if it is relieved by medical or surgical treatment (other than a by-pass operation), the pylorus returns to normal. There is a genetic factor in the ætiology; 5 of these 70 cases had a family history and 1 was of the second generation.

It has been postulated for many years that the cause of the work hypertrophy of the pylorus is a neuromuscular inco-ordination (Cameron, 1925). Changes in Auerbach's plexus may account for this inco-ordination, and such changes have been reported by others.

Belding and Kernohan (1953) reported changes in the nerve cells of the plexus, which they believed to be due to degeneration. They used autopsy material, which may not be reliable because the great killers have been gastro-enteritis and the results of sepsis. Alarotu (1956) studied biopsies, and confirmed this finding. He suggested that the degeneration was due to necrobiosis as a result of overstimulation by the vagus nerves. If this were the cause, however, it is difficult to understand how the process could be reversible, and normal peristalsis resumed.

Friesen *et al.* (1956) believed the changes to be evidence of delayed maturation of the nerve cells of the plexus. If this were so, one would expect a greater incidence of hypertrophic pyloric stenosis in premature infants. No such increased incidence is reported, and there is none in this series.

Conclusions.—In infantile hypertrophic pyloric stenosis there are quantitative and qualitative changes in the ganglia of Auerbach's plexus of variable degree.

These changes are the reason for a phase of neuromuscular inco-ordination when the infant starts to feed. The inco-ordination results in ineffectual contractions of the pyloric muscles and a work hypertrophy. This in time blocks the pylorus in most cases.

The disturbance of function may be relieved by the natural adaptive processes of the gastro-intestinal tract, in which case the tumour will soften and relax as normal peristalsis is successfully achieved. Recovery is accelerated by surgical decompression, or sometimes by pharmaceutical means.

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CASES

Recurrent Hypoglycæmia, Hæmatemeses and Ketonuria in Identical Twins.—TREVOR P. MANN, M.D.

Twin I.—C. A., female, born 17.5.55.

Normal delivery; birth weight 5 lb. 10 oz. Three cyanotic attacks between fourth and seventh days—rigid and staring during one. Thereafter healthy until aged 17 months when she and identical twin (L. A.—*see below*) both started vomiting altered blood shortly after rising one morning; both normal within twenty-four hours.

Just before the third birthday she (and her twin) again vomited altered blood shortly after waking; both were promptly admitted to hospital. Full recovery by following day. Barium meal normal; no cardiac enlargement. Iron deficiency anæmia. Still no precise diagnosis.

Aged 3 *years* 2 *months* further attack of vomiting (bile at first; then blood) at beginning of the