examination was, however, carried out and a greatly hypertrophied pylorus was found with complete stenosis.

Case 3.—This occurred in July, 1948. The child was a male and the projectile vomiting started towards the end of the second week. Visible peristalsis and a palpable mass were again found after feeding. Treatment with 1/750 gr. (0.875 mg.) of eumydrin before feeds was begun, and from the outset the condition was good. On this occasion co-operation from the parents was forthcoming and it was possible to carry out a Rammstedt operation in the early stages. Recovery was rapid and uneventful.

J. L. ADLINGTON, M.B., Ch.B.,
Que Que, Southern Rhodesia.

Congenital Pyloric Stenosis in Binovular Twins

The aetiology of congenital pyloric stenosis is quite obscure. The constant pathological findings and the onset a few weeks after birth point to some defect in the development of the pylorus, widely assumed to be dependent on a genetic factor.

Individual reports of the disease in two or more siblings have appeared from time to time (Parker, 1927; Bilderback, 1928; Cockayne, Moncrieff, and Sheldon, 1928; Thompson and Gaisford, 1935; Fabricius and Vogt-Müller, 1937; Donovan, 1938; O'Donnell and Klein, 1941; Cathala and Cler, 1944; Marquezy, 1944; Kaufman, 1947). On the other hand, Akin and Forbes (1947) found only three instances in 147 patients, Nafe (1947) six cases in 129 patients, and Gavois and Poyanne (1944) one case in 67 patients. It is clear that it is unusual for siblings to be affected, although Cockayne and Penrose (1943) concluded, after a most exhaustive investigation, that there was a definite though small familial incidence.

Occasionally pyloric stenosis has occurred in successive generations. The father of the female patient reported by Donovan (1938) had undergone operation for stenosis when a few weeks old; one of Nafe's (1947) patients had an uncle who had had the disease, as had the maternal aunt of the pair of twins reported by de Lange (1936). Schrieber (quoted by Laubscher and Smith, 1947) described a patient whose father and grandfather both suffered from pyloric stenosis in adult life, which was believed to be of congenital origin. Caulfield (1926) cited a further case that was even less convincing. Cockayne (1934) reported two pairs of twins who were first cousins, all of whom had the disease. The absence of any definite hereditary characteristics does not lend support to the genetic theory, but if a Mendelian recessive was concerned it would remain concealed for long periods, showing a tendency to exposure particularly on intermarriage; and in respect of this Cockayne and Penrose (1943) found the proportion of patients with consanguineous parents exceeded random expectation.

Laubscher and Smith (1947), in a recent review on congenital pyloric stenosis in twins, found it recorded in 13 monovular twins; in 11 of these both twins were affected, while Sheldon (1938) and Lasch (1925) each noted an exception. In 24 sets of binovular twins there were two instances in which both were affected. Laubscher and Smith (1947) reported binovular male twins, both of whom underwent operation five weeks after birth, and O'Donnell and Klein (1941) recorded a similar occurrence; Nafe (1947) mentioned that he had recently seen binovular twins operated on for pyloric stenosis on the same day.

CASE REPORTS

The mother, a 2-para aged 33, gave birth to a male child on June 29, 1948; simultaneously the membranes of the twin

were artificially ruptured, but the uterus became inert and the second child, a female, was not born until July 1. There was an elder child, a girl aged 6. No history was elicited of any similar trouble either in the mother's or in her husband's family, and the parents were not related.

First Twin.—Male, born June 29, 1948; birth weight, 6 lb. 2 oz. (2.78 kg.). He was breast-fed for the first fortnight, but then changed to a proprietary food. He thrived until July 21, when he vomited after one of his feeds. Three feeds later he vomited again, and thereafter following every bottle: the vomitus became large in quantity and projectile in nature. On July 23 he was admitted to Queen Mary's Hospital in a dehydrated condition, and weighing 5 lb. 3 oz. (2.35 kg.). A palpable tumour lay under the right costal margin, with visible peristalsis in the epigastrium. On July 24 the abdomen was explored under local analgesia. A firm pyloric muscle tumour was found, and a typical Rammstedt's operation was performed. The child made an uneventful and rapid recovery.

Second Twin.—Female, born July 1, 1948; birth weight, 4 lb. 2 oz. (1.87 kg.). She was maintained in an oxygen tent for five days, with three-hourly oesophageal feeds of a proprietary food. After a further three weeks in the premature baby nursery she was discharged home, on July 25, weighing 5 lb. 8 oz. (2.49 kg.). For a few days she continued to progress favourably, but on August 1 suddenly vomited after a feed, and thereafter shortly after every bottle; with each vomitus a large quantity was ejected in projectile fashion. On August 3 she was admitted to the Connaught Hospital because of an outbreak of whooping-cough at Queen Mary's Hospital. She weighed 4 lb. 1 oz. (1.84 kg.), but was less dehydrated than her brother had been. Peristaltic waves crossing the epigastrium were easily visible; and a mobile tumour was palpable under the right costal margin. She was given a feed and shortly after it was forcibly vomited. On August 3, under open ether anaesthesia, a Rammstedt's operation was performed for a typical congenital hypertrophied pylorus. Her post-operative progress was quite uneventful and she rapidly gained in weight.

COMMENT

A study of the published data, together with such cases as the one reported here, reveals the following: (1) the disease may occur in siblings, but with relative infrequency; (2) it is rare for two or more generations to be involved; (3) whilst each twin is usually affected when of monozygotic origin, authentic exceptions have been recorded; (4) there have been several instances in which each twin of dissimilar pairing has been affected, such as the example reported here.

E. S. R. HUGHES, F.R.C.S.

H. A. D. SMALL, F.R.C.S.

J. W. F. MACKY, F.R.C.S.

Queen Mary's Hospital, Stratford, E.15,
and Connaught Hospital, E.17.

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