

revealed no satisfactory explanation. Indeed, all gradations exist between the congenital idiopathic dilatation and that developing after several years.

Dr. Rolleston, in his interesting paper on this subject (to which I am indebted for several references), differentiates between acquired and congenital cases, admitting, however, the impossibility of absolute divisions. He includes under the latter group only those where the bowels have never acted naturally and distension appeared soon after birth, while all other cases are classed as acquired. In the statistical table, however, which is included in his article, this classification is scarcely adhered to, inasmuch as the case of Little and Callaway above mentioned, is regarded as congenital in origin, although the symptoms developed only late in adult life, and if such a case be regarded as antenatal, it would seem unjustifiable to exclude at least six or seven others where the condition has arisen even in earlier childhood, though perhaps not co-incident with birth. There can be no question that in the majority of these cases some mechanical hindrance to the evacuation of the large bowel is the main immediate etiological factor, and though these may frequently be antenatal, it could scarcely be regarded as associated with changes which have arisen in the earliest days of life in the embryo.

The cases of idiopathic dilatation of the colon or sigmoid which occur, might perhaps be classified as follows :

1. Those in which the symptoms occur at birth, either as constipation alone, or combined with distension of the abdomen (Rolleston's congenital cases). Of these there are very few on record, apparently only four cases about which there can be no doubt, viz.: those of Generisch, Osler, Hirschsprung and Peacock ; my own case is also to be herein included.

2. Those in which the symptoms develop shortly after birth (*i.e.*, within a few months), or where it is stated that "from earliest infancy" there was one or other sign present.

These cases are more numerous, and though they cannot be definitely called congenital, they so closely resemble them as to be practically identical, and are more than probably of congenital origin. These are recorded by Walker and Griffiths, Eisenhart, Hughes, Formad, Osler, Bristowe, Gee (2 cases) and Rolleston.

3. Those developing after several years and associated with no pathological lesion. Such is Gee's, subjoined, in which the symptoms appear to have commenced only after 4 years of age.

4. Those cases which occur only in adult life, which can likewise be explained only as idiopathic (*i.e.*, with no organic lesion to suggest a clue as to the cause). Of these, there are many cases too numerous to mention, *e.g.*, those of Herringham and Bruce Clarke, Little and Callaway, Hadden, Lewitt and many others.

The following table will explain these various cases and include a few details concerning each :