CLINICAL DIAGNOSIS OF WHOOPING-COUGH WITHOUT THE WHOOP

BY

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Whooping-cough is a socially important disease, killing more children in this country than scarlet fever and diphtheria together.¹ Its diagnosis, like that of pregnancy, is difficult in the early stages, and obvious to the household in the late. It is made obvious by the onset of whooping. Earlier diagnosis is important, affecting as it does the treatment of the child, the prevention of yet further spread of the infection, and the reputation of the practitioner.

Two laboratory methods may be of great help in early diagnosis: (1) an absolute and relative lymphocytosis is usually present (Kolmer,² Crombie³), and (2) the bacillus of Bordet and Gengou may be isolated in the catarrhal stage of the malady. The former takes too much time in a busy hospital out-patient or general practice; the latter, the efficiency of which has been shown by Madsen in Denmark, and Gardner and Leslie⁴ in this country, should be of considerable value, but facilities for its performance are few and far between. Agglutinins are fugitive and inconstant in the blood; complement fixation develops too late; skin tests are controversial; while the discovery of specific radiological changes is a skilled and expensive procedure.⁵ So one is, in the end, generally driven back to clinical methods.

Of 156 cases in which there was finally no doubt of the diagnosis, personally seen at the Hospital for Sick Children, Great Ormond Street, from May to the middle of October, 1934, fifty-six had not whooped at their first appearance. The account of the symptoms given here results from the study of these latter cases. For the sake of convenience the number of children having any symptom is expressed as a percentage of those in whom the particular feature was investigated, despite the false impression of precision it gives in a small series. The average duration of the cough in the cases considered was just under two and a half weeks; all except one were in the paroxysmal stage.

Symptomatology

Whooping-cough may appear at any season of the year, but is probably commonest in the spring and autumn. It starts like a common cold, with catarrh of the nose and eyes, and slight fever; at this stage it cannot be diagnosed clinically. But a common cold dies away, the catarrh of pertussis persists a while, and a short, dry cough appears. If there has been contact with a case one's suspicions are aroused, as one finds the cough becoming increasingly severe, and, to a more or less extent, paroxysmal.

Age and History of Contact.—The age of the child is significant. In Luttinger's 10,000 cases 80 per cent. were less than 6 years old. The malady may appear at any age, but is definitely uncommon below 3 months or over 7 years. In this series the ages were:

Under 1 year ...	15 per cent.	5-6 years ...	18 per cent.
1-2 years ...	9 " "	6-7 " ...	4 " "
2-3 " ...	17 " "	7-8 " ...	4 " "
3-4 " ...	15 " "	8-9 " ...	4 " "
4-5 " ...	13 " "	9-10 " ...	2 " "

The large proportion of cases less than 1 year old is to be expected, for many babies do not whoop throughout the whole of the attack. The slight difference in incidence between the sexes is of no value in any one case. A history of contact with another case is often helpful, for its frequency naturally varies in different groups of cases. In this group it reached the high figure of 74 per cent.

The Cough.—This is paroxysmal, increasing crescendo to the great distress of the child and the mother, who notices that the face is red and puffy (81 per cent.), particularly under the eyes. Before whooping actually occurs the child may be said to "catch its breath" after the paroxysm. The cough is hard, and those who witness the attack say that the child "cannot bring anything up." Nevertheless, careful inquiry usually elicits the fact that some sputum is coughed up and ejected by the child, with or without the aid of the mother's finger (70 per cent.). This is a valuable sign—a young child who coughs and spits has whooping-cough or frank bronchiectasis. It is of more frequent occurrence in older children than younger; the average age of those who produced sputum was 4.3 years, compared with 3.4 years for the whole series. It does, nevertheless, occur in babies as young as 3 months. The sputum is whitish in the early stages; in later cases with bronchitis it may be yellow. A classical feature of pertussis is that the violence of the cough excites vomiting. This is commoner in later cases, but often occurs before whooping (56 per cent.). It occurs at any age, the youngest in this series being 3 months and the oldest 9 years. It is an important cause of the wasting which is obvious in severe cases, but this is not of early diagnostic significance. Typically, the cough is worse at night (77 per cent.), and is a common temporary cause of insomnia in children and their parents. In a few cases (19 per cent.) it is equally bad in the day and in the night; in fewer still (4 per cent.) it is most troublesome in the daytime.

Other Symptoms.—**Appetite.** This is usually (69 per cent.) retained at first (though the onset of most acute illnesses in children is accompanied by anorexia), but it may be lost as the disease progresses. **Haemorrhage.** This is a feature of few cases (under 20 per cent.). Occasionally there may be a visible subconjunctival haemorrhage, but the two common types are epistaxis and haemoptysis. The latter is rather more common. About a third of these cases have frenal ulceration, and the blood probably comes from the ulcer. Others have not, but there seems no reason to assume that the lung is the source; the small necrotic lesion in the larynx is probably responsible. On examination of the child one may be struck at once by the well-marked puffiness of the face (7 per cent.) even between paroxysms. But most cases do not present this till later.

The Frenal Ulcer.—The search for this (18 per cent.) should never be omitted. It is simple, and may clinch a doubtful diagnosis. It occurs in no other acute illness, and is thus practically pathognomonic. It is small, between 1 and 3 mm. long, oval, with its long axis in the line of the frenum; grey-white in colour; painful at first, later painless; it occurs at any age, and not only, as is usually stated, in young children whose incisor teeth are particularly sharp. I have seen it in a baby of 10 weeks and in a girl 9 years old. It is presumably caused by the fretting of the frenum on the lower central incisor teeth when the tongue is protruded in a paroxysm; occasionally there is no ulcer, but a small, oval, grey-white area of raised epithelium.

Throat and Lungs.—Examination of the throat is of itself not helpful. Some faucial congestion is common, and many town children have inflamed tonsils. But it is useful in a doubtful case, for when the history is indefinite one may often elicit an unmistakable paroxysm by touching a tonsil with the spatula (17 per cent.). Absence of pulmonary signs which might explain the cough is a frequent (74 per cent.) and useful finding in these early cases. Signs of bronchitis are usually limited to an occasional rhonchus, but the presence of many rhonchi and rales does not invalidate the diagnosis.

Summary

To recapitulate. The diagnosis may be made on the history, in a child probably less than 7 years old who has not previously had whooping-cough, of a common cold that did not clear up but progressed steadily with the development of a dry cough, becoming paroxysmal; and at least one of the following points: a history of contact with a definite case; absence of any obvious cause of cough in the throat and lungs; a cough worse during the night than the day; sputum coughed up and not all swallowed; cough followed by vomiting; frenal ulceration; an unmistakable paroxysm during examination. Confirmatory points are: a red and puffy face; haemoptysis, epistaxis, or subconjunctival haemorrhage; absence of anorexia.

Skill is not required to elicit these points, and the difficulty lies in correct summing up of the relative importance of their presence or absence. It is hoped that the figures and the rather disjointed remarks in this paper will help in the diagnosis of pertussis before the onset of whooping betrays the condition to all who have ears to hear.

REFERENCES

- ¹ Registrar-General's Statistical Review, 1920-9.
- ² Kolmer: *Amer. Journ. Dis. Child.*, 1911, i, 431.
- ³ Crombie: *Edin. Med. Journ.*, 1908, i, 222.
- ⁴ Gardner, A. G., and Leslie, P. H.: *Lancet*, 1932, i, 9.
- ⁵ Stévenin, H.: *La Coqueluche*, Paris, 1926.
- ⁶ Luttinger, P.: *Amer. Journ. Dis. Child.*, 1916.

Clinical Memoranda

HEREDITARY SYNDACTYLISM AND POLY- DACTYLISM DESCRIPTIVE OF RECENT ADDITIONS TO PEDIGREE

(With Special Plate)

In 1915 I published in the *Journal of Genetics* a pedigree and description of a family showing syndactylism and polydactylism transmitted through five generations. This article, with a description of further additions to the family up to August, 1926, was republished in *Observations on Human Heredity* in 1928.

Altogether sixty-four descendants of the original affected parent, who was born about the beginning of the nineteenth century, were shown on the pedigree chart, and of these twenty-seven were affected, thirty-three unaffected, and four doubtful. During the past eight years—that is, since August, 1926—thirteen additions from five families have been made to the Warrington branch of the original family. Eight of these have come from two families of unaffected parents, five from three families with one affected parent. As the condition does not appear in families with unaffected parents, the eight do not require consideration here; but of the five an only child, a male, is normal—the offspring of affected IV 6 (male); and one female is normal, the offspring of affected IV 8 (female), now dead.

In the pedigree chart published it will be seen that IV 8 had three other unaffected children, so that she had altogether four unaffected offspring, although she herself had both syndactyly and polydactyly in the right hand and both feet. These families of IV 6 and IV 8 are small, and it is likely that had they been more numerous, affected offspring would have appeared. Their mother—affected III 2—had seventeen children, of whom eight were affected.

But the most interesting are the three offspring of IV 16 (female), who are all markedly affected in the hands, and to a slighter degree in the feet. It is remarkable that this should be so, because the mother herself, IV 16, had the slightest abnormality of all the affected members of the family. In her case the abnormality was limited to webbing of the third and fourth digits of the right hand without any bony abnormality whatever. Her husband is free from any abnormality of hands and feet. The children are J. (female),

aged $4\frac{1}{2}$; W. (male), aged $2\frac{1}{2}$; and B. (female), aged $1\frac{1}{2}$. Skiagrams show the following conditions.

J.—Right hand: webbing of third and fourth digits; duplication of terminal phalanx of third digit, which is fused with the terminal phalanx of fourth digit. Left hand: six digits, third, fourth, and fifth webbed; fusion of ends of the proximal phalanges of third and fourth digits. Left foot: fifth digit is broad, with broad nail, and is partially webbed to fourth digit. Right foot: normal.

W.—Right hand: six digits—third, fourth, and fifth webbed; fusion of third and fourth metacarpal bones. Left hand: similar condition to right hand. Left foot: fifth digit is broad, with double nail. Right foot: normal.

B.—Right hand: six digits—third, fourth, and fifth webbed; fusion of proximal phalanges of third and fourth digits. Left hand: six digits—third, fourth, and fifth webbed; fusion of third and fourth metacarpal bones. Feet normal.

DISCUSSION

Apart from the main feature—the persistence of the abnormality through five generations—there is the specially interesting point that, while the mother is but slightly affected, her children have abnormalities of the digits in an augmented form. Evidently somatic manifestation does not indicate the intensity of the hereditary factor present in the germ cells.

As to the nature of the hereditary factor, it might be inferred from the extent and variability of the abnormality that this was related to Spemann's embryonic regulators of organic growth. The hand, and to a lesser degree the foot, are among the most complex anatomical structures in the body, and it would seem that in this family a gene carrying a hyperactive "organizer" was transmitted through the generations, producing a dominant character of either a complete or partial extra digit in the hands and feet, with correlated changes in the skin and soft tissues.

The variable dominance shown in this family waxing and waning through the generations is a feature which is hardly likely to be confined to it, or to abnormalities of the hands and feet, and its presence in other hereditary or supposed hereditary conditions should be borne in mind.

I am greatly indebted to Dr. Edward Fox of Warrington for the x-ray photographs.

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RODENT ULCER IN THE YOUNG

Rodent ulcer is generally regarded as a disease of old age. Most textbooks on diseases of the skin so describe it, and rather ignore the possibility of its occurrence in the young. So far as I am aware the only book in which the occurrence in young people is stressed is Sir Norman Walker's *Introduction to Dermatology*, where a table is given showing the occurrence of rodent ulcer at different ages, many being in young people.

The following cases came under my notice at the Royal Infirmary, Edinburgh.

W. S., aged 15, first attended in January, 1933, with a rodent ulcer about the size of a threepenny-piece on the tip of his nose. The condition had been present for more than a year. The appearance of the rodent ulcer was typical, with the rolled edge and depressed centre. A piece was excised for microscopical examination and the diagnosis confirmed.

G. H., aged 25, was first seen in May, 1934, with a rodent ulcer on the left ala nasi. It was about the size of a sixpence, and had been present for several years. The patient was quite definite that he had had the lesion when he was 17, the size then being about half what it was when he first attended the Royal Infirmary. As the condition must have been present for more than a year before that, it brings the age of onset to about 15, and approximates it to that of the preceding case. Biopsy confirmed the diagnosis in this case also.