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PROCEEDINGS OF THE PALACE OF
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PRECOCIOUS PARALYSIS OF THE PALATE IN DIPHTHERIA.

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SINCE first diphtheritic paralysis was systematically studied, the occurrence of palatal palsy at an early stage of the disease has been well known. Trousseau and Lasègue, and many subsequent writers of the pre-antitoxin era, such as Sanné, Squire, Morell Mackenzie, and Henoch have recorded illustrative cases. Until within recent years, however, no mention is to be found of the significance of the early affection. Some writers, indeed, such as Ruault, Sevestre, and Martin regard the paralyses that develop early as the benign forms, in that they show a tendency to be localised, unlike the late forms which tend to become generalised. An exactly opposite view is held by Baginsky and Romberg in Germany, and in France by Variot, Marfan, Deguy, Berthelot,

Petit, and Babonneix. These authorities hold that precocious paralysis of the palate is a mark of malignancy. The present writer has recently reported some cases that illustrate the truth of this view. The following remarks are based on observations made on 1000 consecutive cases of diphtheria that have been under the present writer's care at the Grove Fever Hospital during the course of the last four years.

Paralysis of the palate occurred in 162 cases (16·2 per cent.). The average date of occurrence was towards the end of the third week (18·3 day among 162 cases).

The term "early" or "precocious" has therefore been applied to such palsies as developed before the beginning of the third week. Fifty such cases among the 162 were so termed, the dates at which they were first observed being as follows:—

Table I. Showing Date of Occurrence of Precocious Palatal Palsy.

5th day of disease, 2 cases.			
6th	"	"	3 "
7th	"	"	6 "
8th	"	"	7 "
9th	"	"	7 "
10th	"	"	5 "
11th	"	"	7 "
12th	"	"	3 "
13th	"	"	6 "
14th	"	"	4 "

The relation of the onset of the paralysis to the subsidence of acute symptoms is shown in the following table:—

Table II.

						Cases.
Palatal palsy noted	3	days before the throat became clean,				4
"	"	2	"	"	"	3
"	"	1	"	"	"	5
"	"	same day as	"	"	"	10
Death before disappearance of membrane,						3
Palatal palsy noted after throat became clean,						25

Thus in 12 cases the palatal palsy developed before the throat became clean, in 10 the two events took place on the same day, in 3 death occurred before the membrane disappeared,

and in the remaining 25 the palsy arose after the throat had become clean. Most of the cases presented after the membrane had left the throat an extensive superficial necrosis of the epithelium of the tonsils, palate, and uvula, manifested by an opaque appearance of the mucous membrane which sometimes took three weeks or more to be completely regenerated. As will be seen from the following figures, the incidence of precocious palatal palsy is higher in childhood than in adult life:—

Table III.

Ages.	Cases of Precocious Palatal Palsy.	Percentage.
0- 5 years	17	4.5
5-10 "	27	5.9
10-15 "	4	4.4
15-20 "	1	3.07
20-30 "	1	

This corresponds with the incidence of post-diphtheritic paralysis at the various ages, as is shown by Table IV.

Table IV. Ages of 238 Paralysis Cases among 1000 Cases of Diphtheria.

Ages.	Cases.	Percentage.
0- 5 years	99	26.5
5-10 "	119	26.2
10-15 "	16	17.9
15-50 "	4	4.7

No case of precocious palatal palsy was met with above the age of twenty-nine, though sixteen of the 1000 patients were above that age, 7 of whom had severe, 4 moderate, and 5 mild faucial attacks.

The two sexes were almost equally affected, 22 being males (4.6 per cent.) and 28 females (5.3 per cent.). How frequent an associate early palatal palsy was of the severe forms is illustrated by the fact that of the 50 early cases 20 died, while among the remaining 112 cases of non-precocious palatal palsy there was only one death, due to diaphragmatic paralysis, on the 52nd day.

The mortality of the total 1000 cases was only 78, or 7.8 per cent.

The relation of the frequency of precocious palatal paralysis to the character of the initial faucial attack is shown in the following table:—

Table V. Showing relation of Precocious Palatal Paralysis to Character of Initial Attack.

Very severe faucial	.	.	29 cases = 29.2%
Severe faucial	.	.	17 cases = 8.8%
Moderately severe faucial	.	.	1 case = 1.1%
Moderate faucial	.	.	1 case = 0.3%

From this it will be seen that though more frequently found in the severest cases, precocious palatal palsy occasionally follows an angina of only moderate intensity. Berthelot's experience was similar. Four of his twelve cases were of moderate intensity, the remaining eight were severe forms. Petit and Deguy, on the other hand, regard precocious palatal palsy as the exclusive appanage of severe forms. The severity of the attacks might be attributed in the majority of cases to neglect of antitoxin treatment during the first few days of the disease; in a smaller number of cases the disease was precociously malignant. The truth of this statement is borne out by the following table, which shows that the majority of cases were admitted in the second half of the first week. Only one of the fifty cases had received antitoxin before admission to hospital. This was a child who, after having had small doses of antitoxin at home on the fifth and sixth days of disease, was admitted to hospital on the seventh day and died on the eleventh day.

Table V. Showing the Days of Disease on Admission to Hospital.

1st day	.	.	.	0 cases
2nd "	.	.	.	7 "
3rd "	.	.	.	10 "
4th "	.	.	.	10 "
5th "	.	.	.	14 "
6th "	.	.	.	5 "
7th "	.	.	.	1 "
8th "	.	.	.	2 "
11th "	.	.	.	1 "

That the concomitant symptoms were severe is shown by the following facts. Fatal cardiac paralysis occurred in 19 cases, the signs of which first developed within a few days of the palatal affection. One case died of diaphragmatic paralysis on the fortieth day. Fourteen were hæmorrhagic cases, *i.e.* presented purpuric spots, with or without hæmorrhages from the mucosæ. Eleven of these were fatal. In all but one of the cases albuminuria was present. In 9 it persisted for three weeks or more. All but 6, or 80 per cent., of the survivors developed other paralyses, which are classified as follows: generalised paralysis, 8 cases; ocular paralysis, 14 cases; labial paralysis, 2 cases. The incidence of further paralysis among the non-precocious forms of palatal palsy was much less. Sixty-two cases (53·5 per cent.) occurred, only one of which, as already stated, was fatal.

Among the survivors, cardiac disturbance of some kind occurred in 14 cases. In 4 it was severe, and was associated with vomiting. Enlargement of the liver, a very grave sign, was present in 26 of the 50 cases. Seventeen of the 26 were fatal.

All the cases received antitoxin, but in spite of the massive doses which were injected the sequelæ were, as a rule, less marked than in milder cases in accordance with the law enunciated by the author that the frequency and intensity of serum phenomena are in direct relation to the size of the dose and in inverse relation to the character of the diphtherial attack. Babinski's sign, which Kiroff has recently noted in malignant diphtheria, was present in 5, or 51 per cent., out of 9 cases in which it was sought for. It is noteworthy that, as in Kiroff's cases, the extensor response co-existed with sluggish or absent knee-jerks. Among the thirty survivors, the duration of the paralysis considerably exceeded the average, being 43·3 days, as compared with 24·8 days, which was the average duration of the palsy in the 112 cases. In 3 cases the palatal palsy was short-lived, the duration in each case being six, seven, and eight days respectively. No other paralyses subsequently occurred in these cases.

In the majority of cases the paralysis, as is the rule in diphtheria, was incomplete, and was manifested only by a change in the voice. Regurgitation seldom occurred at a very early

stage, except in young children. Inspection of the fauces showed that the motility of the velum was only slightly impaired on phonation. In a few cases the palsy was unilateral, when it was subsequent to an angina that had been unilateral or predominant on that side.¹

Diagnosis.

During the first fortnight of the disease, especially during the second week, the sound of the voice should be tested daily in all severe cases. The preservation of a clear voice will be found to coincide with a normal heart, an absence of liver enlargement, and a good general condition; while a nasal twang shortly precedes or accompanies the signs of cardiac involvement and the apathy or restlessness usually associated with it. It is sometimes difficult to distinguish a nasal intonation from the thick character of the voice due to faucial œdema and abundant membrane. As a rule, however, a nasal voice does not develop till the œdema has subsided.

When regurgitation occurs early in diphtheria it must be distinguished from that due to mechanical obstruction produced by hyperæmia and faucial œdema, such as may occur in any form of sore throat. In diphtheria, though the membrane may still be present, the œdema has usually subsided by the time that regurgitation occurs.

Pathology.

Maingault, the writer of the first monograph on diphtheritic paralysis, attributed the paralysis to a modification of nutrition of the palate under the influence of inflammation, and compared the paralysis of the palate to the similar phenomena that follow inflammation of the bladder and intestines. It was soon pointed out, however, that local inflammation did not account for cases of paralysis occurring where there had been no initial angina, nor for the paralysis attacking other parts than those which had been the site of membrane, *e.g.* the eyes. A purely local cause was therefore set aside in favour of systemic intoxication by Trousseau. The histological investigations of Charcot and Vulpian in 1862 in a fatal case of palatal paralysis showed

¹ Similar cases were reported by Gubler in 1861, by Gee in 1864, and more recently by Aubertin and Babonneix.

that the motor nerves alone were affected. Later observers, such as Oertel and Leyden, pointed out that other tissues besides the nerves were involved. Hochhaus, by his histological examination, proved that the morbid anatomy of paralysis of the palate was mainly an interstitial myositis. This view was adopted by Baginsky and Romberg, who distinguish early paralyses (*Frühlähmungen*) from post-diphtherial palsies. The former, according to them, arise from disease of the musculature, which in many ways is analogous to the change in the heart. Post-diphtheritic paralysis, on the other hand, depends on a degenerative change in the peripheral nerves, with occasional affection of the anterior cornual cells. The most elaborate histological researches yet published on the paralysis of the palate in diphtheria are those by Deguy, after researches carried on in Marfan's laboratory at the *Hôpital des enfants malades*.

Sections of the palate from cases that had died with early paralysis showed very marked inflammatory lesions. The presence of a large number of diplococci, both in the leucocytes and in the thrombosed capillaries of the part, made Deguy regard the condition as a diplococœmia superadded to diphtheritic intoxication.

From this brief survey it will be seen that recent authorities agree with the early writers in regarding the affection of the palate as due to a local change. The extensive superficial necrosis of the fauces accounts for the unusually long duration of the paralysis, owing to the long time that elapsed before the tissues are completely regenerated. A further proof of the influence of the local inflammation in determining the palsy is furnished by the fact that ocular paralysis in diphtheria is never precocious. In the present series of cases it never started before the beginning of the fourth week, and sometimes was not noted till the sixth week, although the vision had been tested carefully at frequent intervals until then.

Summary.

1. Precocious palatal palsy in diphtheria is almost invariably associated with malignant forms, as is shown by the high mortality, the association of other grave symptoms during the acute stage, and subsequent more frequent development of paralysis in convalescence in the cases in which it occurs.

2. It resembles the ordinary forms of diphtheritic palsy in its tendency to be frequently incomplete, and by its higher incidence among young persons.

3. It is, as a rule, of much longer duration than the palatal affection which occurs at a later date.

REFERENCES.

1. Aubertin. *Arch. gén. de méd.*, fév. 10, 1903.
2. Aubertin et Babonneix. *Gaz. des Hôp.*, 1902, p. 1285.
3. Babonneix. *Nouvelles recherches sur les paralysies diphtériques*, Thèse de Paris, 1904.
4. Baginsky. "Diphtherie und diphtheritischer Croup," 1898.
5. Berthelot. "De la gravité des paralysies diphtériques précoces," Thèse de Paris, 1904.
6. Deguy. *Rev. mens. des mal de l'enf.*, juin 1903.
7. Gee. *Med. Times and Gaz.*, 1864, p. 148.
8. Grancher, Boulloche, and Babonneix in Brouardel and Gilbert's "Traité de méd.", 2nd ed., 1905, art. "Diphtérie."
9. Gubler. *Gaz. méd. de Paris*, 1861, p. 704.
10. Henoch. "Lectures on Children's Diseases," *New Syd. Soc.*, 1889, vol. ii. p. 306.
11. Hochhaus. *Virchow's Archiv*, Bd. 124, S. 226, 1891.
12. Kiroff. *Rev. Neurol.*, Nov. 30, 1905; Abstract in *Review of Neurology*, Feb. 1906, p. 151.
13. Mackenzie. "Diphtheria," 1879.
14. Maingault. "De la paralysie du voile du palais à la suite d'angine," Thèse de Paris, 1854.
15. Marfan. *Bull. et Mem. de la Soc. méd. des Hôp. de Paris*, July 11, 1902; and "Leçons cliniques sur la diphtérie," 1905.
16. Moynier. "Compte rendu des faits de diphtérie dans le service de Trousseau," 1859.
17. Petit. *Rev. mens. des mal de l'enf.*, fév. 1897.
18. Rolleston, J. D. *Practitioner*, Nov. and Dec. 1904; *Ibid.*, May 1905; *M.A.B. Annual Reports*, 1904; *Review of Neurology*, Nov. 1905.
19. Romberg. "Lehrbuch der inneren Med.," 1905, art. "Diphtherie."
20. Ruault in "Traité de Méd.," par Charcot, Bouchard, et Brissaud, art. "Diphtérie."
21. Sanné. "Diphtérie," 1877.
22. Sevestre et Martin in Comby's "Mal de l'enf.," tom. 1, 1904, art. "Diphtérie."
23. Squire in Reynold's "System," vol. i., 1866, art. "Diphtheria."
24. Trousseau. *Union médicale*, 1851, p. 471; *Gaz. des Hôp.*, 1860, Nos. 1 and 5; *Clinique méd.*, 1st ed., 1861.
25. Variot. "La diphtérie et la sérumthérapie," 1898.

