

of these cases showed the presence of pus in the antra when lavage was undertaken. The chest lesions were of all grades, varying from a minor radiological change, usually at the base of the lung, to an advanced bronchiectasis.

It will be noted that 65 (89%) of these children were found to have either a sinus suppuration or a chronic respiratory infection or both.

### 3. Management

When the implication of the presence of a wax keratosis had been realized a routine procedure was followed in all cases.

Under general anaesthesia both ears were inspected, and the wax present in the deep part of the external auditory canal was removed. This was usually a most difficult procedure owing to general narrowing in the outer part of the canal and the very firm impaction of the wax in the depths beyond. Often this came out as a complete cast of the canal even to the imprint of the handle of the malleus, but occasionally difficulty was found in separating the epithelial envelope from the canal wall, and especially from the tympanic membrane. In these cases only the inner core of wax was removed, leaving the envelope to be extruded later by the normal physiological process of desquamation. Any granulation tissue present was removed by suction at the same time. Proof puncture and antral lavage of both maxillary antra were then performed, revealing in most cases an underlying suppuration requiring further treatment.

Many children had a productive cough which obviously needed treatment, but in those children in whom a chest condition was not so obvious a routine examination, including a chest x-ray examination, was carried out. In this manner about half of the children were found to have an unsuspected chest lesion.

### 4. Treated Prognosis of the Ears

It is not our intention to discuss the results of treatment of the sinusitis or of the coexistent chest lesion. This treatment is essential, and it has been evident that in children in whom respiratory suppuration remains a wax keratosis can re-form. Probably an important result of this investigation has been to emphasize the likely primary nature of the respiratory infection and the great difficulties experienced in its treatment. Many cases of unsuspected sinusitis and bronchitis have been discovered which would otherwise have continued untreated.

With regard to the ears, they will usually return to normal. When a non-infected wax keratosis has been removed, the tympanic membrane may appear opaque and thickened at first, especially when the outer envelope of the wax keratosis remains attached to it. Gradually this envelope is thrown off and the tympanic membrane becomes perfectly normal in all respects. Any deafness which was present has normally been due to the simple action of the wax keratosis as a foreign body, and the hearing returns to normal when it has been removed.

When the wax keratosis has become infected and granulation tissue has formed, local antiseptic drops may have to be used after the wax keratosis and the granulations have been removed. Once the pressure of the epithelial lining of the external auditory canal has been relieved there is no stimulus to the formation of granulation tissue, and in these children as well the ear soon returns to normal.

So long as the associated respiratory suppuration is treated and cured there will be no re-formation of the wax keratosis, and thus we hope that keratosis obturans will be prevented.

### Summary

Wax keratosis, if looked for, will be found in about 3% of children attending an E.N.T. department for any reason.

It is almost universally bilateral.

In half the cases there will be no suspicion of any lesion in the ears until they are examined.

Respiratory suppuration (sinusitis, bronchitis, bronchiectasis) will be found, if looked for, in nearly all cases.

The wax keratoses must be removed to prevent the possibility of any future infection, with local suppuration or progression to keratosis obturans. General anaesthesia will be required for this removal and will allow the opportunity for antral lavage.

Chronic suppuration with polypus formation can occur and simulate chronic suppurative otitis media.

Early removal, associated with concurrent and subsequent treatment of any respiratory suppuration present, will result in an ear normal in appearance and in function, with no likelihood of recurrence.

### REFERENCES

- Black, J. I. M. (1954). *Newc. med. J.*, **24**, 284.  
Collins, E. G. (1951). *J. Laryng.*, **65**, 14.  
Morrison, A. W. (1956). *Ibid.*, **70**, 317.

## PHYSIOTHERAPY FOR BELL'S PALSY

BY

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Bell's palsy can be defined as unilateral paralysis of the facial muscles resulting from an intrinsic lesion of the seventh cranial nerve (Cawthorne, 1951). There is general agreement that 70 to 80% of these patients recover completely within one to three months, while the remainder develop various sequelae (Taverner, 1955). There is no agreement about treatment, although many methods have been tried. At present electrical stimulation of the paralysed muscles is widely used, at least until voluntary movement reappears. This practice is supported by many of the standard textbooks (Collier, 1948; Evans, 1951; Nielsen, 1951; Elliott, Hughes, and Turner, 1952; Wilson, 1954; Brain, 1955; Wechsler, 1958), but Walshe (1952) states that galvanism is useless.

There is apparently no record in the literature of a controlled trial of the value of electrotherapy in facial palsy, and the present investigation was undertaken because daily electrotherapy is expensive and time-consuming. It was felt to be justifiable to withhold physiotherapy from the control patients because previous experience has shown that lack of physiotherapy does not necessarily prejudice recovery from peripheral nerve injuries (Henderson and Taverner, 1949).

### Design of the Trial

All patients with Bell's palsy of less than 14 days' duration seen in the department of electromyography of the General Infirmary at Leeds from October, 1954, to December, 1956, were included in the trial. The diagnosis of Bell's palsy was made on the basis of the following

three criteria: (1) complete or partial paralysis of the muscles of expression of the whole of one side of the face of sudden onset; (2) absence of any symptom or sign of any other disease of the central nervous system; and (3) absence of any symptom or sign of any disease of the ear or posterior fossa. The patients were examined clinically and electromyographically throughout the course of their disorder. The degree of paralysis was estimated visually as a percentage of the function of the normal side. This method proved reasonably consistent from visit to visit, but was supplemented by photography under standard conditions in many patients.

The electrical examination consisted of concentric needle electrode exploration of the facial muscles without local analgesia. The electrical activity was amplified and recorded with conventional electromyographic equipment. In some patients supplementary studies of neuromuscular excitability were made with a Ritchie-Sneath stimulator.

Denervation of the facial muscles was established by the presence of fibrillation activity in the electromyogram, by abnormalities in the strength-duration curves, and by the subsequent development of abnormalities of the facial musculature during recovery (cf. Taverner, 1955).

All the patients were instructed to massage the face daily, but about half of them, by random allocation from a prepared list, were given daily galvanism by one of us (J. M.). Each patient received infra-red radiation to the affected side of the face at 2 ft. (60 cm.) distance for 10 minutes. The skin was prepared with soap and warm water, and interrupted galvanism was then applied from a Ritchie-Sneath stimulator, using saline electrodes. Each facial muscle in turn was stimulated to 30 minimal contractions with a pulse of 100 msec. duration. This was repeated three times, giving 90 contractions a muscle. In some patients with severe denervation of the facial musculature the muscles tired quickly and the number of contractions was then reduced to not fewer than 30 in three groups of 10. The muscles stimulated were frontalis, corrugator supercillii, orbicularis palpebrarum, levator labii superioris alaeque nasi, levator labii superioris, levator anguli oris, risorius, orbicularis oris, depressor anguli oris, depressor labii inferioris, levator menti. Treatment was given daily until active contractions returned and then thrice weekly until recovery was virtually complete or, in the event of denervation, until the condition seemed stationary. Treatment for patients with denervation lasted from two to six months.

### Findings

Of the 86 patients admitted to the trial, three were eventually rejected—two because of failure to reattend, and one because of failure to complete treatment owing to illness. Of the 83 patients studied, 43 received physiotherapy, and 40 were used as controls. The age, sex, and duration of facial palsy did not differ significantly between the two groups (Table I).

TABLE I.—Mean Values for Age, Sex, and Duration of Facial Palsy When First Seen. Range in Parentheses

	Whole Group	Treated	Control
Age .. ..	37.5 years (3-79)	39.5 years (3-79)	35.6 years (7-73)
Sex .. ..	M.40; F.43	M.22; F.21	M.18; F.22
Duration ..	5.2 days (1-14)	5.2 days (1-14)	5.2 days (1-14)

It is known that when facial palsy is due to conduction block of the facial nerve no sign of denervation is found and recovery is complete in from one to three months. If denervation of the facial muscles develops, improvement is slower and complete recovery never occurs (Taverner, 1955). The patients in this trial were divided, for purposes of analysis, into those with and those without electromyographic evidence of denervation (Table II). Proportions with conduction block and denervation did not differ significantly between the two groups.

TABLE II.—Proportions with Denervation and Conduction Block in Treated and Control Groups

	Conduction Block	Denervation
Treated .. ..	23 (54%)	20 (46%)
Control .. ..	27 (68%)	13 (32%)

All the patients with conduction block recovered completely, and the time taken is shown in Table III. The clinical course of patients with conduction block was almost identical in treated and control groups, both in the time taken to initiate movements and in the time to complete recovery.

TABLE III.—Mean Time in Days From Onset of Facial Palsy to First Visible Movement and to Full Recovery in Treated and Control Groups. Range of Observations in Parentheses

	Initial Movement	Full Recovery
Treated .. ..	10 (3-21) days	43 (14-90) days
Control .. ..	11 (2-21) ..	39 (14-134) ..

The patients with denervation never recovered completely, and most of them were treated for much longer periods than those without denervation. The times from onset of palsy to first movement were 53 days (range 10-104) in the treated group and 66 days (range 14-90) in the control group. These patients were observed for at least a year or until no further recovery could be detected. The final degree of recovery of voluntary power was assessed visually as a percentage of that of the normal side. An overall estimate of facial movement was made by halving the sum of the percentage estimates of function for the frontalis and orbicularis oris muscles. The last assessments of percentage recovery in the two groups are presented in Table IV. Proportions showing varying degrees of recovery do not differ significantly between the two groups.

TABLE IV.—Estimated Voluntary Power After Maximal Recovery of Denervated Facial Musculature. Figures in Parentheses are Percentages of Respective Totals

	Total	<25%	25-75%	>75%
Treated .. ..	20	3 (15%)	11 (55%)	6 (30%)
Control .. ..	13	3 (23%)	6 (46%)	4 (31%)

The presence of contracture of the facial muscles was carefully recorded. This appears only after denervation, and its occurrence could not be related to the electrical treatment. Eleven (26%) of the treated group and eight (20%) of the control group developed contracture.

### Discussion

The treated and control groups are similar enough to enable useful comparisons to be made. The three defaulters would have added two to the control group and one to the treated group and could scarcely have affected the issue. The results in the two groups are so similar that it is unlikely that electrical treatment can be of importance in the management of facial palsy. The cost to the community and to the patient of daily visits to hospital for electrotherapy does not seem justified on the present evidence. It has been suggested that physiotherapy is valuable for maintaining the morale of the patient. It has been found, in the management of several hundred patients with facial palsy, that if a firm prognosis and careful explanation and reassurance are given then full confidence is maintained.

### Summary

A controlled trial of the value of galvanic stimulation in the management of Bell's palsy is reported. Eighty-six successive patients were randomly allocated to treatment and control groups. Eighty-three of them were studied until recovery was complete or for at least one year.

Treatment consisted of infra-red radiation followed by interrupted galvanism to 11 individual facial muscles on the affected side. Treatment was given daily at first and later thrice weekly.

No significant advantage could be demonstrated from the use of galvanic stimulation for Bell's palsy.

## REFERENCES

- Brain, W. R. (1955). *Diseases of the Nervous System*, 5th ed. Oxford Univ. Press.  
 Cawthorne, T. (1951). *Proc. roy. Soc. Med.*, 44, 565.  
 Collier, J. (1948). *British Surgical Practice*, 4, 1. Butterworth, London.  
 Elliott, F. A., Hughes, B., and Turner, J. W. A. (1952). *Clinical Neurology*. Cassell, London.  
 Evans, G. (1951). *Medical Treatment*. Butterworth, London.  
 Henderson, W. R., and Taverner, D. (1949). *Lancet*, 1, 1084.  
 Nielsen, J. M. (1951). *Textbook of Clinical Neurology*, 3rd ed. Cassell, London.  
 Taverner, D. (1955). *Brain*, 78, 209.  
 Walshe, F. M. R. (1952). *Diseases of the Nervous System*, 7th ed. Livingstone, Edinburgh.  
 Wechsler, I. S. (1958). *Textbook of Clinical Neurology*, 8th ed. Saunders, Philadelphia.  
 Wilson, S. A. K. (1954). *Neurology*, 2nd ed. Butterworth, London.

## LAXATIVE EFFECT OF SORBITOL

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As sorbitol is now available commercially and is being used increasingly in food products, its metabolism and other physiological characteristics are of some importance. Apart from its value as a diabetic sugar it is useful in confectionery as a humectant and plastifier and for increasing the shelf life of the products. Its metabolism has been studied extensively and further work is in progress. In the course of this work it has been observed that, like other sugars of its kind, it exerts a laxative effect, particularly noticeable in large dosage. An attempt to define the threshold dose was made by Ellis and Krantz (1941), who found that by trial on 12 subjects the minimum dose to produce soft or watery stools was about 50 g. a day for the crystalline product and 20-30 g. a day for the 70% syrup (the crystalline compound being much superior in quality to the syrup).

In view of the small number of subjects previously used in these tests the results can hardly be regarded as representative for the general population. We therefore decided to apply tests to a larger number of volunteers to determine the effects at a specified dosage.

## Method

**Dosage Consideration.**—A prerequisite of the tests was that they should not unduly affect the majority of subjects, but should indicate exceptional susceptibility if such should exist. A trial approach was made on three apparently normal subjects, one at a dosage of 50 g., and the other two at 25 g. Only the higher dosage showed spectacular results, and the test level chosen was therefore 25 g. a day.

Of the 101 volunteers taking part, 15 failed to complete the test for reasons unconnected with the effect of sorbitol. Control was obtained by the provision of a glucose solution with an addition of saccharin to match the sweetness of the sorbitol. The volunteers were accepted *en bloc* and no planned distribution of age group, sex, etc., was arranged.

The test consisted in taking 12.5 g. of either sorbitol or glucose after meals twice daily for seven days, half of the panel taking sorbitol and the other half glucose, and then changing over to the other substance for a second week. So far as the subjects were concerned, the solutions were identical in appearance, colour, and taste.

The solutions were: (1) a 50% w/v solution of glucose in water and (2) a 50% w/v solution of sorbitol in water to which was added 0.04% w/v of saccharin (based on the sorbitol content).

Each subject was given a supply of test solution sufficient for one week and invited to complete a questionnaire day by day. The 101 volunteers are accounted for as follows: completed tests both weeks, 84; tests not completed owing to alleged effects of sorbitol, 2; tests not completed for reasons not connected with sorbitol, 8; tests not started for personal reasons, 7.

## Results

Analysis of the factors, such as rotation of dosage, method of dosage (as supplied or in a beverage, etc.), age group, occupation, marital status, and some others, not all of which are analysed in the accompanying Table, did not

Table of Results

Occupation	Status	Solution Used	Age Groups				Totals	
			Under 30		Over 30		Sub.	Mot.
			Sub.	Mot.	Sub.	Mot.		
Active	Single	Glucose	3	21	1	11	4	32
	" Married	Sorbitol	3	21	1	9	4	30
		Glucose	4	25	30	236	34	261
	" "	Sorbitol	4	23	30	262	34	285
Single		Glucose	12	94	2	13	14	107
Sedentary	" Married	Sorbitol	12	90	2	17	14	107
		Glucose	7	48	25	223	32	271
	" "	Sorbitol	7	53	25	273	32	326
		Totals	Glucose	26	188	58	483	84
	Sorbitol	26	187	58	561	84	748	

Sub.=Subjects. Mot.=Motions.

reveal any significant effects. In view of this the significant differences between sorbitol and glucose (standard deviation, 4.99 and 2.95 respectively; DF, 83) must be attributed to some other factor not disclosed in the group allocations.

By inspection three results were seen to be outstandingly divergent from the general run. A recalculation of standard deviation of the glucose and sorbitol series after eliminating these cases gave a standard deviation of 2.90 for glucose and 2.95 for sorbitol (DF, 80) indicating that the two series show no significant difference.

A fuller statistical analysis of the factors and interactions was found to be beyond our competence owing to the complications of disproportionate class numbers.

Inspection of the observations on "wind" showed a definite tendency to flatus due to sorbitol.

## Comment

Olmsted (1953) commented on the results of Ellis and Krantz (1941) and suggested that sorbitol may be a physiological laxative in the sense that it is a substrate for the colonic bacterial populations. It has been shown, however, that, in general, sorbitol is a poor nutrient for the more common bacteria, and it seems more likely that its humectant properties are the cause of the laxation observed at higher dosages.

We conclude that sorbitol administered in a split daily dosage of 25 g. does not cause laxation in the general population, but specific reactions may be expected from about 5% of the subjects. In practice it has been noticed that the presence of fats in foodstuffs allows much larger doses to be tolerated without laxative effects. In view of the increasing use of this product in the confectionery trade, it might be well to bear in mind the possibility of a sorbitol sensitivity as a cause of diarrhoea in an otherwise healthy subject.