INVESTIGATION OF CHILDREN WITH SPEECH DEFECT, WITH PARTICULAR REFERENCE TO NASALITY*

BY

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[WITH SPECIAL PLATE]

To discuss this subject within the limits of a short paper without appearing to be dogmatic is almost impossible, for I wish to present a scheme or method for the investigation of defective speech and proceed in logical sequence through it. This will require the acceptance as facts of certain statements which have not yet been incorporated into general surgical teaching although there is ample support for their veracity. These statements are in no way schismatic, but are the result of the newer knowledge concerning the speech mechanism.

First, the term "nasality" needs to be defined, for it is in popular use in this subject and commonly thought to be a definite word with an exact meaning. On the contrary, it is used to describe a feature of speech when the nose is pinched (complete nasal obstruction), when the speaker has a cold in the nose (partial nasal obstruction), when he has a cleft palate (nasal escape of air), or when he is from the Mid-Western States of the U.S.A. (increased nasal resonance). It is illogical that one word should be applied to diametrically opposing conditions, and so the more exact terms of "nasal escape" and "nasal resonance" must be used.

Nasal Escape During Speech

In the English language nasal escape is a feature of only the two consonants "n" and "m." It also occurs during the vowel "a," but is not a particular feature of that sound, although in some languages, such as Portuguese, nasal escape is a feature of vowels, especially when they occur as diphthongs.

According to the returns of the Registrar-General, in England and Wales in 1955 there were just under 665,000 live births, and in 1956 just over 699,000. Also it is known from several surveys in different countries (Fogh-Andersen, 1942; Ivy, 1952) that the incidence of congenital cleft lip and palate is around 1 in 700 live births, and that 70% of these infants will have palate clefts of one type or another. Allowing for 5% who will have died before their first birthday, this means that approximately 630 infants were born in 1955 with palate clefts, and all are potentially defective speakers. If all have been operated on at 1 year of age, 30% or more will still have excessive nasal escape during speech. To these 190 children each year can be added a lesser number who have other congenital speech defects associated with nasal escape, some who suffered from diphtheria, poliomyelitis, or encephalitis, and finally those who have their speech defect thrust on them by radical adenoidectomy. I have estimated the total at 250 children per year, but this is a very conservative guess, and the actual number is probably double this.

The causes of nasal escape can be summarized briefly as follows:

Causes of Nasal Escape During Speech

A. ANATOMICAL DEFECTS OF PARTS (Mechanics)

Congenital: Cleft palate; submucous cleft; absence of part of soft palate or pillars of fauces

Acquired: Trauma—surgical, gunshot wounds; disease—lues, cancer

B. DEFECTIVE MOTOR POWER (Dynamics)

Neurological: Nuclear and peripheral:

Congenital: Nuclear hypoplasia

Acquired: Trauma; disease—diphtheria, poliomyelitis

Supranuclear: Congenital paresis; encephalitis; meningitis

Cortical: Gross mental deficiency; hysteria

Muscular: Congenital agenesis—unilateral, bilateral; scar tissue

C. DISPROPORTION OF PALATE AND PHARYNX (Kinetics)

Repaired cleft palate

Congenital short palate

Congenital large pharynx

Adenoidectomy

There are numerous medical and moral reasons for treating patients with nasal escape and a sound economic motive for spending public money on them. The £8-a-week labourer who moves up into the £20-a-week skilled operator class pays back to the State as income-tax an additional £187 yearly. He has therefore paid for his treatment and rehabilitation within one year, and continues for the rest of his life to pay enormous interest for the privilege of attaining normal speech. That such a successful investment may occur has been seen in our department often enough to be impressive.

Diagnosis of Speech Defects

"Diagnosis before treatment" is as important in speech defects as it is in any other complaint, and an examination of the available aids to diagnosis is well worth while. We live in an age of great opportunity where diagnostic tools increase in quantity and quality almost daily, but they will not be produced for medical use unless they are demanded. Using the available diagnostic equipment, a scheme for the examination of patients is suggested as follows:

Investigation of a Speech Defect

History:

Present complaint

Past: "milestones," illnesses

Family: of C.L. and P.; of congenital deformities; of neurological complaints

Examination:

Inspection: palate, pharynx, tongue

Palpation

Auscultation: speech—articulation; nasal escape; resonance

Special Investigations:

Audiograms

Neurological

Psychological; intelligence

Social report; school reports

Dental

Lateral radiographs of palate

Cineradiography during speech and swallowing

Endoscopy

Faradic stimulation of palate and pharynx

Measurements of nasopharyngeal isthmus

Biopsy

The greatest advances in diagnosis have been achieved with the help of radiology, the employment of which has enabled us to clear away many of the false ideas that hitherto prevented deliberate and conscious attempts to reconstruct our ideas concerning the physiology of the palato-pharyngeal mechanism. Roentgen announced his discovery of x rays in 1895 and indicated their value in medicine. Between 1895 and 1935 a total of 98 papers had been written on their use for the study of the speech mechanism, but it is only in the last few years that it has been possible to use x rays in the routine examination of our patients. One can indeed make a fairly complete palato-pharyngeal analysis on a simple lateral radiograph (Calnan, 1956) or, if even more accurate information is required, on a cephalometric tracing (Ricketts, 1954).

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Palato-pharyngeal Analysis

1. Soft palate: (1) length; (2) thickness; (3) site of levator eminence; (4) mobility; (5) position at rest.
2. Hard palate: (1) length; (2) relationship to arch of atlas.
3. Nasopharynx: (1) antero-posterior diameter; (2) lateral diameter; (3) movements.
4. Adenoids: size, extent, importance.
5. Tongue: control.
6. Eustachian cushions: (1) relationship to hard-palate level; (2) relationship to posterior pharyngeal wall.

Perhaps it might be as well now to see how this diagnostic scheme works in practice. I have tried to show that the object is to be a keen clinician but a somewhat reluctant surgeon, and would also point out that radiological aids may help to control treatment. Three patients with speech defects are described (Special Plate, Fig. 1) and the investigations compared and tabulated (Tables I, II, and III).

Case 1

William T. M. was born on November 23, 1922, with a unilateral complete cleft of the lip, gum, and palate on the right side (Group III 1). The lip cleft was repaired in infancy. His parents were averse to further operation and the palate cleft was never repaired. He got on well at school and was in the Army from 1941 to 1946, where as an N.C.O. he used to drill men on the barrack square. He is

TABLE I.—Results of Conventional Investigation of Speech Defects

	Case 1	Case 2	Case 3
Name	William T. M.	Janet M.	Marion T.
Age (years)	34	12	8
Work	Market gardener (self-employed)	Normal school	Normal school
"Milestones"			
Speech began	About 2 years	Normal At 1 year. Normal until aged 6, when T's and A's removed	Normal At 1 year
Previous operation	C.L. and P. III I.R. Lip repair at 7 days	Usual	Nil
Previous illness	Usual	Usual	Usual
Previous speech therapy	None (Drill sergeant 1939)	5 years	2½ years
Speech: Articulation	Normal	Normal	Incorrect
Nasal escape	Medium	Gross	Gross; almost unintelligible
Palate	Cleft; elevates well	Normal; no notch; elevates well	Shapely; no notch; immobile
Diagnosis	Cleft palate	? short palate; ? old polio	? old polio; ? lack of use of S.P.
Proposed treatment	Repair palate	Speech therapy	Speech therapy

now 34, self-employed as a market gardener, married with one child. He wishes to improve his speech so that he may take an active part in local political affairs. He is no longer a child, but presents a problem occasionally seen in older children where there is quite good speech despite an un-repaired cleft palate, and one wonders whether this can be improved materially (Special Plate, Fig. 2).

Case 2

Janet M., a healthy girl aged 11, spoke normally until she was 6, when her tonsils and adenoids were removed. Immediately after this operation speech was so nasal as to be barely intelligible even to her mother. For the past five years she has received speech therapy once weekly with some improvement. Her articulation is correct, but all sounds are masked by gross nasal escape of air (Special Plate, Fig. 3).

Case 3

Marion T., aged 8, never spoke quite normally. She passed the developmental milestones at the normal times, but from the age of 3 her parents noted nasal escape in her speech, with articulatory faults which have improved only a little following 2½ years' speech therapy. She went to a normal school at 5 years and has become less shy. She is said to be in the right school class for her age and more methodical than her older sister (Special Plate, Fig. 4).

From Table I, the conventional method of investigation, it can be seen that in two cases the diagnosis is unsatisfactorily vague. In Table II further information has clarified the diagnoses, and to some extent indicated the appropriate line of treatment (Table III).

TABLE II.—Additional Information

	Case 1	Case 2	Case 3
Name	William T. M.	Janet M.	Marion T.
Age (years)	34	12	8
School	Got on well	Doing well	Normal standard
Social	Well adjusted	Sociable	Shy
Hearing	Normal	Normal	Normal
Dental	Arches reasonable. Instanding 4½	Normal arches	Normal arches; high palatal vault
Neurological	Nil	Poker face	Nil
I.Q.	Average	103	Average
Reading age	Normal	12.1 years	Normal
Psychological	—	Anxiety—hospitalization (Friends and mother primitive)	Anxiety
Lateral radiographs: Rest: S.P. Pharynx	Reasonable length Deep	Long and fleshy Deep	Long and thin Normal
Saying "ee"
Blowing
Cineradiography	Confirms incompetence	Confirms	Confirms immobility
Faradic stimulation	—	—	S.P. no reaction; pharynx brisk response
Biopsy	—	—	Scanty degenerate muscle fibres
Moulds of isthmus	35 mm. 59 mm.	31 mm. 19 mm.	35 mm. 19 mm.
Diagnosis	Cleft palate, deep pharynx	Deep pharynx following adenoidectomy	Congenital absence of levator palati muscle

TABLE III.—Diagnosis, Prognosis, and Treatment

	Case 1	Case 2	Case 3
Name	William T. M.	Janet M.	Marion T.
Age (years)	34	12	8
Diagnosis	Cleft palate; deep pharynx	Deep pharynx following adenoidectomy	Congenital absence of levator palati muscle
Operation	4 flap V-Y palate repair	Hynes (1950) pharyngoplasty	Passavant (1879) pharyngoplasty
Moulds	28 mm. 24 mm.	31 mm. 19 mm. 17 mm. 7 mm.	35 mm. 19 mm. No. 10 catheter
Lateral radiographs
Prognosis	Some improvement	Excellent	Fair
Speech therapy	No	No	Yes
Result	No change in speech	Normal speech	Less nasal escape; better projection of voice
Second operation	Hynes (1950) pharyngoplasty	—	—
Moulds	13 mm. 10 mm.	—	—
Lateral radiographs		—	—
Prognosis	Excellent	—	—
Speech therapy	No	—	—
Final result	Normal speech, pitch raised	—	—

Discussion

The whole object of treatment is to provide the patient with normal speech. In some cases this is impossible, and improvement is all that can be attained (Case 3). In others a surgical operation may so eliminate nasal escape that the speech therapist can complete treatment by eradicating old faults and training the patient in the correct use of the lips, tongue, and teeth during articulation. The most favourable cases to treat are naturally those with normal articulation, for in them the correct surgical treatment produces a dramatic result (Cases 1 and 2).

One could dilate further on what is normal speech and discuss the classification of defective speech. Reduced to the simplest terms, our patients may have defects of articula-

tion, of nasal resonance, or of nasal escape. This paper is concerned only with the last defect. There is, however, a fair amount of evidence to indicate that if nasal escape is reduced or, better still, eliminated, then the correction of the other two faults becomes a speedier and easier task. The reverse, the correction of articulatory faults before dealing with nasal escape, is common practice in many parts of this country and abroad; that it may be desperately hard work many patients will acknowledge.

Perhaps to understand more fully what has happened in these three patients the progress of each individual may be plotted as a graph. This form of recapitulation often points out salutary lessons for the various medical advisers. I say medical advisers in the plural, because it has been our experience at Oxford that every patient has been seen by one or two general practitioners and speech therapists, a paediatrician, an otolaryngologist, and a plastic surgeon before the diagnosis has been settled.

In Case 1 there were minor articulatory faults ("th" = "f," "v" = "w") which are common enough in normal speakers and so did not justify a course of speech therapy at his age (Graph 1). He has done well in life, despite his cleft, mainly because he has learnt to control the amount of nasal escape during speech. The radiographs (Special Plate, Fig. 2) suggest that the latter has been accomplished by keep-

GRAPH 1.—Speech progress in Case 1: treatment of a patient with nasal escape in speech in relation to time. The amount of nasal escape fell from "medium" to "slight" after repair of palate cleft. The increase in nasal escape immediately after pharyngoplasty was probably due to oedema, limiting movement of the parts.

GRAPH 2.—Speech progress in Case 2. Gross nasal escape in speech started immediately after adenoidectomy. Five years' speech therapy did not produce improvement. Normal speech returned within a few weeks of the pharyngoplasty operation.

ing his tongue low in the mouth, thus not deflecting air up into his nose and, at the same time, making the mouth a larger resonating cavity than the nose.

The patient in Case 2, on the other hand (Graph 2), was given speech lessons at weekly intervals for five years, although her articulation was correct and could not be improved further. From the radiographs (Special Plate, Fig. 3) it is equally obvious that speech therapy could never eradicate the nasal escape, and only reimplantation of the adenoid pad or some substitute could restore normal speech.

Case 3 is quite a different problem (Graph 3). Here articulation is markedly abnormal and nasal escape gross,

GRAPH 3.—Speech progress in Case 3. Gross nasal escape with gross faults in articulation had been present from the beginnings of speech. A pharyngoplasty, reducing the size of the nasopharyngeal isthmus, has already reduced the amount of nasal escape. By increasing the intraoral pressure, articulation will probably improve, but it is likely to take many years of patient speech therapy.

giving the patient unintelligible speech. In spite of 2½ years' speech therapy little progress has been made. Unfortunately, because the soft palate is immobile it is technically not possible to eradicate the nasal escape during speech without producing nasopharyngeal stenosis. One can only expect to reduce it, thereby increasing the intraoral pressure of air, which should allow the patient to take greater advantage of the help provided by the speech therapist.

These last two patients illustrate conditions which are not generally recognized but which are by no means rare. Cases similar to Case 2 are relatively easy to treat and the prognosis is good, but what the final result of treatment in cases like Case 3 will be is still unknown.

Case 2 illustrates what, to my mind, is the common clinical picture when permanent nasal escape occurs after adenoidectomy. Removal of the adenoids is the cause of the nasal escape; it is doubtful whether tonsillectomy alone ever gives rise to trouble. Unfortunately both operations are so often combined as "T's and A's," and only in special cases is either performed alone. Nasal speech immediately after operation is probably due to oedema tracking up the faucial pillars (after tonsillectomy), thereby restricting the mobility of the soft palate. Oedema is usually dissipated within three weeks, and so nasal escape must be due to some other factor—that is, loss of tissue (the adenoids) in the nasopharyngeal isthmus. This establishes disproportion between the soft palate and pharynx. Minor degrees can be overcome by greater elevation of the soft palate, so that nasal escape may be manifest only when the patient is tired, as at the end of the day; a major disproportion causes permanent and often gross nasal escape during speech, as in Case 2. The x-ray findings in Special Plate Fig. 3 are very typical: the soft palate, of normal length, has elevated to well above the level of the hard palate (on saying "ee..."), but is unable to meet the posterior wall of the pharynx, which is just too far away for contact. In a discussion on nasal speech after tonsillectomy in the *British Medical*

Journal (1956) various cures were proposed, but disappointingly little was mentioned about diagnosis. In our patient much time was wasted in conservative treatments of such kind: the logical and obvious cure is shown in Special Plate Fig. 3 and Graph 2.

In these cases one began by knowing little and believing much, and ended by inverting the quantities. The adenoids, long ago entered in the Gilbertian classification of things "that never will be missed," deserve some respect, if not a reappraisal, of their worth.

Finally, I would like to emphasize the advantages of early diagnosis and treatment in patients with speech disorders. These patients are invariably backward in reading and in their general education because of their speech defect. Some of them are of below-average intelligence and so require special help. Correcting the speech defect allows their education to proceed normally, and will also allow them to take advantage of extra coaching at school so that they may ultimately earn a living, as normal citizens, within their capacity for work and responsibility.

Summary

It is conservatively estimated that in England and Wales 250 children per year are born with potential speech defects or acquire such defects as the result of disease or of radical adenoidectomy. The mechanism of production of such defects by tonsil and adenoid operations is described.

A scheme for the investigation of children with defective speech is outlined, with particular reference to nasality. "Nasal escape" is defined and a summary of its causes given. The advantages of early diagnosis and treatment are stressed. A fairly complete palatopharyngeal analysis can be made on a simple lateral radiograph; even more accurate information can be obtained with a cephalometric tracing.

Children with speech disorders are invariably educationally backward, and correction of the defect allows their education to proceed normally so that ultimately they may earn their living as normal citizens.

Three illustrative cases are reported. These are discussed and their progress is charted.

REFERENCES

- Brit. med. J.*, 1956, 1, 760, 1060.
Calnan, J. (1956). *Brit. J. plast. Surg.*, 8, 265.
Fogh-Andersen, P. (1942). *Inheritance of Harelip and Cleft Palate*. Busck, Copenhagen.
Hynes, W. (1950). *Brit. J. plast. Surg.*, 3, 128.
Ivy, R. H. (1952). *Plast. reconstr. Surg.*, 9, 121.
Passavant, G. (1879). *Arch. klin. Chir.*, 23, 771.
Ricketts, R. M. (1954). *Plast. reconstr. Surg.*, 14, 47.

Lecturing at the Royal Institution on March 21, Professor C. D. DARLINGTON, F.R.S., said that man was already controlling his own evolution, as Francis Galton had predicted he would. The present methods of control operated at several levels. Irradiation induced mutations; cousin-marriage exposed them. Selective migration and education changed our breeding groups. The successes of medical treatment increased the proportion of individuals requiring medical treatment for birth or survival. These successes also favoured a differential fertility between social classes which tended to lower the proportion of educable individuals in the next generation. Artificial insemination and medical sterilization might be used to work in the opposite direction. On a higher level we could see that the development of class structure, and the rise and decline of nations which were connected with it, were genetic and evolutionary processes. These processes were influenced or controlled to-day by the action of departments of governments, especially in health, education, and taxation. We easily forgot that, though governments could provide us with education, only parents could endow us with the ability to profit by it.

LOCALIZED LEISHMANIASIS OF LYMPH NODES

REPORT OF FOUR CASES

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[WITH SPECIAL PLATE]

It is well known that lymph nodes may be affected in cases of kala-azar and that Leishman-Donovan (L.D.) bodies may be demonstrated in material obtained by lymph-node aspiration or biopsy (Cochran, 1912; Hu, 1936; Kirk and Sati, 1940; Lipscomb and Gibson, 1944; Rigg, 1957), but few records are available of human cases with lymph-node enlargement alone, and in which no other clinical manifestations of the infection could be found.

Chung (1944) described the case of a male student from Peiping who presented with an enlarged cervical lymph node. Microscopical examination of the node obtained by biopsy revealed the presence of L.D. bodies. Subsequent inoculation of a hamster with material obtained from an epitrochlear node gave rise to generalized leishmaniasis in the animal. The patient showed no other evidence of visceral disease over a period of four years' observation, at the end of which time he received a course of treatment ("neostibosan"—diethylamine-*p*-aminophenyl stibinate) following the development of a positive complement-fixation reaction.

Angevine *et al.* (1945) described two cases in U.S. Servicemen from the Mediterranean area who were admitted to hospital for investigation of lymph-node enlargement unassociated with any other abnormal physical signs. In both cases L.D. bodies were demonstrated in sections of the cervical lymph nodes, and, as in Chung's case, in the liver and spleen of a hamster which was inoculated with a culture made from a subsequent lymph-node biopsy from one of the cases. No other relevant case reports have been found in the literature.

Four cases are now described of British Servicemen serving in Malta and Cyprus in whom the only clinical abnormality has been the presence of enlarged lymph nodes and in three of whom L.D. bodies have been demonstrated in material obtained by lymph-node biopsy or aspiration.

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FIG. 1.—Photographs of palate in cases given as examples of speech defect. A: Wide cleft palate and deep pharynx (Case 1). B: Normal soft palate but nasal speech following adenoidectomy (Case 2). C: Normal-looking soft palate, but quite immobile (Case 3).

ORIGINAL CONDITION

FIG. 2.—Case 1. Lateral radiographs of palato-pharyngeal region. *Above:* Original condition, when palate was cleft (as in Fig. 1A). Essential outlines have been overdrawn with indian ink for easier interpretation. H.P.=hard palate; S.P.=soft palate; T=tongue; P.P.W.=posterior pharyngeal wall. Arrow indicates site of levator eminence. A.A.=anterior tubercle of arch of atlas, an important bony landmark. *Below:* Progress towards normal speech. Soft palate has not elevated fully in last radiograph and so did not quite meet pharyngoplasty prominence (P.H.), but later did become competent.

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ORIGINAL CONDITION

SAYING "EE..."

"Ee..."

Blowing

Original

Two weeks after pharyngoplasty

FIG. 3

Six months later

ORIGINAL CONDITION

SAYING "EE..."

At rest

"Ee..."

Blowing

Original

Two weeks after pharyngoplasty

FIG. 4

FIG. 3.—Lateral radiographs of palato-pharyngeal region in Case 2. *Above:* One must presume that the adenoids filled the gap between the elevated soft palate and the posterior pharyngeal wall before adenoidectomy, for the patient's speech was normal. *Below:* Stages in competence of the soft palate following pharyngoplasty.

FIG. 4.—Lateral radiographs of palato-pharyngeal region in Case 3. *Above:* The soft palate, being immobile, hangs dependent when the tongue moves forward. *Below:* Pharyngeal flap pharyngoplasty has hitched the soft palate up, as well as partly occluding the oro-nasal communication.

(Landmarks in Figs. 3 and 4 as for Fig. 2)