OESOPHAGEAL SPEECH

BY

G. H. BATEMAN, B.M., F.R.C.S.

A. C. DORNHORST, M.D., M.R.C.P.

AND

G. L. LEATHART, M.B., M.R.C.P.

(From St. Thomas's Hospital and St. Thomas's Hospital Medical School)

It has been known for many years that speech may be regained after complete excision of the larynx and the establishment of a permanent tracheostomy; but, although efforts are often made to encourage and assist the patient to develop his "oesophageal" voice, little appears to be known of the mechanism of this remarkable accomplishment. The role of the cricopharyngeal sphincter was discussed by Negus (1938), and our observations largely support his suggestions.

FIG. 1.—Oesophageal speech; counting four. The top tracing indicates the amount of air in the chest; the next the rate of airflow in or out of it. Inspiration causes a downward deflection of both. The other tracings are the oesophageal pressure record and the sound-track.

FIG. 2.—As in Fig. 1, but recorded at greater paper speed.

This paper describes observations on three patients who had undergone complete laryngectomy, and who had well-developed oesophageal speech.

Methods.—Intra-oesophageal pressures and trunk movements were measured by the technique previously described by two of us (Dornhorst and Leathart, 1952). Sound (for timing purposes only) was recorded with a commercial amplifying stethoscope unit. The patients were examined by fluoroscopy, using barium paste to outline the oesophageal walls, and the appearances were in two cases checked by taking films.

Results

All three patients gave substantially similar results. During breathing the oesophageal pressure behaved normally, being maintained at a sub-atmospheric level and dropping further in inspiration. The pressure also behaved normally during swallowing.

During speech the oesophageal pressure mounts steeply and rises well above the atmospheric pressure (Fig. 1). The sound is formed during the rise, and one may sometimes see a judder in the pressure trace corresponding to it (Fig. 2). The positive-pressure wave is accompanied by an expiratory movement of the chest and by an obvious expulsion of air through the tracheostomy. The patient can repeat the process about ten times, each effort producing one syllable; he must then pause and take a breath, as during this time his lungs have been progressively emptying.

Fluoroscopy shows that the oesophagus is usually empty and that swallowing is normal. Just before the patient utters a sound the oesophagus suddenly fills with air from the pharynx to the diaphragmatic opening (Fig. 3). During speech this air is expelled by a series of short expiratory efforts, several syllables being possible on one "fill." If air is left in the oesophagus the next swallow carries it into the stomach, which thus tends to become distended, but with experience the patient learns to avoid this.

For comparison, records taken during speech in a normal person are shown in Fig. 4. It will be seen that the pressure rise is less than in the patient and the chest is emptied less. The normal process is clearly more economical of both air and effort.

Discussion

From the data described, the mechanism may be inferred with some confidence. The cricopharyngeal sphincter is kept closed until the patient is about to speak. He then releases it, allowing the oesophagus to fill, which it readily does because of the negative intrathoracic pressure to which it is exposed, the rate of filling being further enhanced by a simultaneous inspiratory movement. The patient then closes the sphincter and raises the intrathoracic pressure by a sharp expiratory effort. The rise in oesophageal pressure forces some air through the sphincter, causing a sound which is then modulated by tongue movements, etc., in the usual way. The effective lower limit of the air reservoir is the diaphragm and not the cardia. Although some air enters the stomach this is a side-effect and there is no evidence that stomach air is belched out to form the speech sound. The rise in oesophageal pressure during speech is always accompanied by a sharp expiratory effort and loss of air from the lungs. There is therefore no need to postulate any

FIG. 3.—Tracing of a radio-graph taken during oesophageal speech. The oesophagus, whose walls are outlined with barium paste, has filled down to the diaphragm.

FIG. 4.—Normal speech: counting ten. The method of recording is similar to that used in Figs. 1 and 2.

activity of the oesophageal smooth muscle. We believe that the whole knack lies in the control of the cricopharyngeal sphincter, and that the oesophagus behaves as a passive tube.

It is surprising to find that a patient with no glottis can raise his intrathoracic pressure in the way shown. Such patients can, however, achieve positive pressures of 30 to 50 mm. Hg during sustained expiratory strain and considerably more during coughing. It appears that the effective resistance of the bronchioles to a suddenly applied expiratory pressure can be very high, so that in a sense air is trapped in the alveoli. Anyone who has witnessed a bronchoscopy will recall that the veins of the head and neck congest during coughing and straining even with the bronchoscope in place.

In acquiring a voice after laryngectomy the patient has to gain control of the cricopharyngeal sphincter so that he can first relax the sphincter and allow air to enter the oesophagus during inspiration, and, secondly, control the degree of contraction during speech so that there is maximum economy in the use of the air in the oesophagus and the loss of air from the lungs. To acquire a good oesophageal voice therefore comprises two stages: (1) finding the voice, and (2) learning to use the new technique economically so that many syllables can be uttered without taking a fresh breath. Finding the voice usually takes a month or two, and the skill in the use of this voice increases over several years.

Summary

Measurements of oesophageal pressure and chest movements during oesophageal speech have been made, and the oesophageal movements observed by fluoroscopy.

The mechanism is discussed. Air enters the oesophagus down to the diaphragm. It is expelled by expiratory effort, the airway resistance allowing the intrathoracic pressure to rise above the atmospheric pressure despite the presence of a tracheostomy.

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OVERFEEDING IN EARLY INFANCY

BY

IAN G. WICKES, M.D., M.R.C.P., D.C.H.

*Late Chief Assistant, Children's Department,
St. Bartholomew's Hospital; Paediatrician, North Middlesex
Hospital and South-east Essex Group*

The purpose of this paper is to record the number of times a diagnosis of overfeeding was made in the first two months of life in a series of healthy infants fed along orthodox lines, and to assess, by observing subsequent events, how valid this diagnosis proved to be.

Review of Literature

Overfeeding has been regarded as the main source of dyspepsia in infancy ever since Aristotle attributed convulsions to this cause. Pemell (1653), amongst others, handed down the classical views, but Cadogan (1748) was the first to appreciate that the breast-fed baby should "have as much as it will take out of both breasts at each time," whereas he pitied those who were "stuffed with pap till they spue." The word "overfeeding" has been used indiscriminately to mean faulty feeding or

overloading with a suitable food—two very different conditions. The prevalence of gastro-enteritis in the nineteenth century stimulated Rotch (quoted by Morse, 1935) to dispense graduated milk mixtures for healthy babies as carefully as he prescribed a dangerous drug. For the same reason Cautley (1897) advised two-hourly feeds, but in 1915 Truby King objected that this method caused overfeeding. King's unbalanced views, which still influence many people to-day, are exposed in the following passage from his daughter's book (Mary King, 1941): "Overfeeding, especially in the breast-fed baby, is the commonest and most frequent cause of digestive and nutritional disturbances in early infancy. It occurs far more often than is realized in the first weeks. Overfeeding is much more serious than underfeeding, because the results are very far-reaching, and may gravely undermine the baby's digestion and nutrition."

Many modern textbooks still state that overfeeding may lead to vomiting, diarrhoea, loss of weight, and death, yet there is not a single authentic case history to be found in the literature illustrating this unlikely sequence of events. Budin (1907) described five non-fatal cases, four of which were probably really infections. Pritchard (1929), Brackett (1947), and others have tried unsuccessfully to link pyloric stenosis with overfeeding. Smith (1939), determined to denounce overfeeding, provided questionable evidence. Many warning statements are to be found, but few supporting facts. In contrast, there is ample published work on self-regulatory feeding (Weinfeld and Floore, 1930; Davis, 1935; Gesell and Ilg, 1937; Aldrich and Hewitt, 1947) demonstrating the safety of this method. Indeed, 20% of Aldrich and Hewitt's series demanded only four feeds daily when orthodox methods would have provided five. They make no mention of overfeeding in their paper, which seems to become a problem only for those who take active steps to avoid it. If the infant is allowed to determine his own requirements overfeeding takes care of itself.

Present Investigation

The mothers of a series of healthy infants were given an appointment, on leaving the maternity ward, to attend the "well babies' follow-up clinic" during the second month for a check-up. Four-fifths (503) of those given appointments kept them, and 80% of the babies born to primiparae and 66% of the others had been taken to an infant welfare clinic on at least one occasion in the meantime.

At the first interview at the follow-up clinic each mother was asked whether her baby had been diagnosed as overfed since leaving hospital; a definite diagnosis was considered to have been made if steps had been taken to reduce the size of the feed. On the basis of this definition, no fewer than 71 infants (14%) had already been treated for this condition. In a further 47 cases the possibility of overfeeding had been discussed, and 12 artificially fed infants had been changed from a full-cream to a half-cream formula on the grounds of qualitative rather than quantitative overfeeding. The question of overfeeding was therefore raised for 130 (26%) of the babies before they were 2 months old. None of the 503 infants attending the follow-up clinic were regarded as overfed at the time, but it was learned that several were subsequently so diagnosed on returning to their own welfare clinic.

For the 71 definite cases the mode of feeding at the time the diagnosis was made was as follows: Breast-fed, 51 infants; bottle-fed, 16; and breast+bottle, 4. Thus 72% of these were wholly breast-fed, which is a little higher than the incidence for the whole series at the age of 2 weeks. There had therefore been no tendency to attribute