Faults in the maxilla: Inward collapse of the lateral maxillary element frequently occurs, and growth of the whole maxilla is sometimes deficient. Once the inward collapse of the lateral element has occurred, nothing can be done about it until the eruption of the first molar teeth which are required for the fitting of stable and effective orthodontic appliances. It is not worth while attempting to correct malalignment of deciduous teeth. Nevertheless it is of prime importance to preserve the baby teeth for as long as possible because of the part they play in the growth of the maxilla. For suitable cases osteotomy and bone graft procedures may be required, but extensive bone work is not usually attempted till after the age of 16 years.

Faults in the speech: If the speech is not developing normally by the time the child has reached school age, the surgeon and the speech therapist should investigate the cause jointly. Speech therapy should never be attempted without a full investigation to find out the cause of the trouble.

The faults will be due to a defect of the palatopharyngeal sphincter allowing nasal escape of air, and/or due to defects in the maxilla, tongue, teeth or lips. The failure of the palate to reach the posterior pharyngeal wall is the chief cause of faulty speech in cleft palate cases. To observe what the palate does X-rays are essential. It is quite impossible to say, by looking into the mouth, whether the palate and pharynx meet during speech. Three still films are usually all that are needed to give adequate information. One is taken with the palate at rest, one on saying 'ee' and one on blowing. If the sphincter is incompetent on saying 'ee', then the surgeon will have to do something further to make complete closure possible. The palate can be lengthened only a little by a subsequent procedure and it is necessary, therefore, to bring forward the posterior pharyngeal wall by a pharyngoplasty procedure. The Hynes type of pharyngoplasty (Hynes 1950), whereby a mucomuscular mound is built up, is the operation of choice. This operation cannot be done in the presence of adenoids, and before it can be attempted the adenoidal pad must be removed. This is the only reason, apart from the most compelling E.N.T. ones, for removing the adenoids in cleft palate cases. The adenoidal pad is usually an essential structure in the effective closure of the palatopharyngeal sphincter during the childhood of cleft palate cases, and it must never be lightly discarded.

Until the nasal escape of air has been cured, speech therapy will not meet with much success, although some patients with a naturally high speech skill can overcome the most severe defects in the speech mechanism.

Speech training can be begun at home at quite an early age and the parents may be encouraged to help the child without calling on the special skill of the speech therapist to begin with. Usually skilled therapy becomes available after the child goes to school. Speech therapists should be encouraged to attend the plastic surgery clinic with cleft palate patients, and indeed with all patients who have speech faults due to nasal escape of air, in order that a precise diagnosis and adequate treatment plan can be made. Joint examination and discussion would save much time and disappointment in many cases.

REFERENCES

Burston W R (1960) *Trans. int. Soc. plast. Surg.* (2nd Congr., London, 1959) p 28
Davis J S & Ritchie H P (1922) *J. Amer. med. Ass.* **79**, 1323
Fogh-Andersen P (1957) *Trans. int. Soc. plast. Surg.* (1st Congr., Stockholm, 1955) p 156
Hynes W (1950) *Brit. J. plast. Surg.* **3**, 128
Kernahan D A & Stark R B (1958) *Plast. reconstr. Surg.* **22**, 442
McNeil C K (1954) Oral and Facial Deformity. London
Peer L A (1960) *Trans. int. Soc. plast. Surg.* (2nd Congr., London, 1959) p 15

Conductive Deafness Associated with the Cleft-palate Deformity

by Christopher Holborow FRCS FRCSEd (*London*)

Introduction: The association of conductive deafness and the cleft-palate deformity has been known for some time. The deafness may not be noticed until school age as it is often of a moderate degree only and so it is possible that these cases are seen in the ear, nose and throat department more often than elsewhere. There has been surprisingly little work on this problem but nearly all authors agree that about 50% of cleft-palate children are recognizably deaf and that this deafness is always of a conductive nature (Drettner 1960, Halfond & Ballenger 1956, Miller 1956). In the series of children that I have seen about half had some hearing loss. Even when this hearing loss is small it must be an additional handicap to these already handicapped children.

Various suggestions have been made to account for the occurrence of this deafness: (1) Frequent upper respiratory infections. (2) Recurrent attacks of otitis media (Holmes & Reed 1955). (3) Lymphoid hyperplasia in the nasopharynx (Masters *et al.* 1960). (4) Failure to remove tonsils and adenoids due to fear of damage to speech (Miller 1956). (5) Imbalance of the pharyngeal muscles (Skolnik & Fornatto 1956). All these may be of importance but I suggest that the primary reason for the deafness is related to the anatomy and function of the eustachian tube.

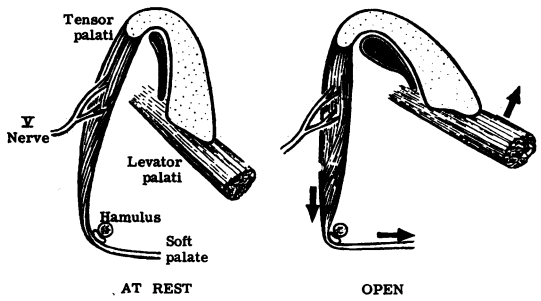

Fig 1 *Transverse section of eustachian tube*

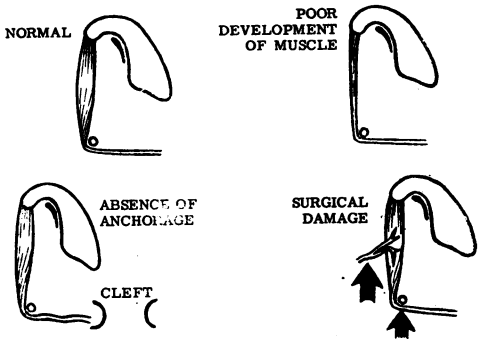

Fig 2 *Causes of malfunction of tensor palati*

Anatomy and function of the eustachian tube: The eustachian tube connects the nasopharynx and the middle ear and is normally closed. It opens during swallowing and this allows the air pressure in the middle ear to equalize with that of the outside environment. If the tube fails to open, air is slowly absorbed from the middle ear and a conductive deafness develops. The functional portion of the eustachian tube is partly enclosed by the tubal cartilage (Fig 1). The levator palati muscle lies under the medial lamina. The tensor palati muscle arises from the lateral lamina and the fibres converge on a tendon which passes round the hamulus of the medial pterygoid plate to be inserted into the palatal aponeurosis. The salpingopharyngeus muscle has little action on the tube (McMyn 1940). The tube is opened by the combined action of the tensor and levator, the tensor pulling the lateral lamina downwards and the levator pushing the medial lamina medially. The curve of the cartilage is widened and the tube opened. Closure is passive and is due to the elasticity of the cartilage.

In infancy the eustachian cartilage lies more horizontally and the tensor palati is therefore relatively more important than in adult life (Aschan 1955, Terracol *et al.* 1949).

Experimental work: To demonstrate the importance of the tensor palati muscle some experiments were performed upon dogs. These animals were used as their pharyngeal muscles are very similar to those of man (Rich 1920).

The dogs were anæsthetized and it was possible to make direct recordings of the middle ear air pressure on a smoked drum kymograph and to vary the pressure artificially. The pharyngeal muscles were stimulated electrically using an instrument giving simple electrical pulses of variable intensity, frequency and duration. Stimulation caused the eustachian tube to open and the air pressure in the middle ear could be seen to alter. The recordings showed that with normal palatal muscles it was possible to return the air pressure in the middle ear to normal from an artificially produced positive pressure and, more importantly, from a negative pressure. The tendon of tensor palati was then isolated as it passed over the hamulus and was cut. The recordings then showed that, using the same degree of electrical stimulation, the air pressures remain unchanged. Partial opening of the tube occurred when there was a positive middle ear air pressure but none when there was a negative pressure.

Conclusions: It is possible to conclude from these experiments that the tensor palati is the most important muscle acting on the eustachian tube and that without its action the tube will not open. I suggest that deafness in cleft-palate children is related to malfunction of this muscle and that it may occur in various ways (Fig 2):

(1) Poor development of the muscle.

(2) Before closure of the cleft, it may be due to the absence of a firm anchorage for the muscle. This is corrected by union of the two halves of the palate and is an argument in favour of early surgery.

(3) The mechanism may be damaged during cleft-palate surgery: (*a*) The hamulus of the pterygoid plate may be deliberately infractured to enable the cleft to be closed. This damages the pulley for the tendon or alters the angle of pull. (*b*) The nerve of supply (a branch of the trigeminal nerve *via* the otic ganglion) may be damaged by dissection lateral to the pterygoid plate. (*c*) There may be scarring and fibrosis around the muscle.

I think that attention to the eustachian tube and preservation or reconstruction of this important and delicate mechanism will prove more profitable than the usual search for sepsis in tonsils, adenoids and sinuses, though these may play a secondary part in some cases.

REFERENCES

Aschan G (1955) *Acta Soc. Med. upsal.* **60**, 131

Drettner B (1960) *Acta otolaryng., Stockh.* **52**, 131

Halfond M M & Ballenger J J (1956) *Arch. Otolaryng., Chicago* **64**, 58

Holmes E M & Reed G F (1955) *Arch. Otolaryng., Chicago* 62, 620
McMyn J R (1940) *J. Laryng.* 55, 1
Masters F W, Bingham H G & Robinson D W (1960) *Plast. reconstr. Surg.* 25, 503
Miller M H (1956) *Laryngoscope, St Louis* 66, 1492
Rich A R (1920) *Bull. Johns Hopk. Hosp.* 31, 206
Skolnik E M & Fornatto E J (1956) *J. int. Coll. Surg.* 25, 356
Terracol J, Corone A & Guerrier Y (1949) La Trompe d'Eustache. Paris

The Indications for Cardiac Surgery under One Year

by R E Bonham-Carter MB FRCP (*London*)

The indication for cardiac surgery in any patient, whether under or over 1 year, must be judged against two risks. The first risk is the prognosis of the unoperated patient. The second is the risk of the operation in any given patient.

The prognosis of the unoperated patient may be looked at, in a biased way, from post-mortem figures. Two-thirds of those who die from congenital malformations of the heart in childhood do so under the age of 6 months. The mortality of acute cardiac illness in infants under 1 year at the Hospital for Sick Children, Great Ormond Street, between 1947 and 1953, when little cardiac surgery was done in this age group, was 73%.

The risk of surgery in any given patient cannot be viewed without considering surgical results and the type of surgery offered. The types of operation available are corrective, and 'salvage' or temporizing.

Corrective surgery at this age is available for patent ductus arteriosus, coarctation of the aorta, pulmonary stenosis with normal aortic root and aortic stenosis. There are no 'salvage' procedures worth while for these lesions.

'Salvage' surgery, which has as its object the preservation of the patient's life until corrective surgery can be carried out with a reasonable surgical risk, is available for transposition of the great arteries, Fallot's tetralogy and its variants, and ventricular septal defect. The rarer forms of congenital heart disease are omitted here.

Analysis of the surgical results in corrective surgery show that in patent ductus arteriosus the mortality in the youngest group under 6 weeks old is largely due to multiplicity of cardiac anomalies. But the indication for surgery is the presence of cardiac failure. This is also the indication for corrective surgery in coarctation of the aorta, and our series shows no death occurring in uncomplicated coarctation of the aorta. The cause of the high mortality rate in pulmonary stenosis is late diagnosis, and the important factor here is the myocardial state, which is shown histologically later to be very poor. Any infant with pulmonary stenosis and normal aortic root who shows increased heart size with increasing electrocardiographic right ventricular hypertrophy is a candidate for surgery before cardiac failure supervenes. The same is true for aortic stenosis in which the left heart is involved, and the ECG shows increasing left ventricular strain.

Transposition of the great vessels is still the commonest cause of early death in cyanotic heart disease. The Blalock-Hanlon procedure of making an atrial septal defect has shown itself to be the best temporizing procedure for these patients. The indication is the presence of congestive cardiac failure, and it is hoped that correction by the Senning type of operation later may be possible.

The surgical results of the Blalock-Taussig type of anastomosis are good and the operative risk is low. The indications for early surgery of this sort are 'blue turns' of increasing frequence or severity, and also anginal attacks.

Ventricular septal defects frequently cause cardiac failure within the first year, which is the indication for palliative surgery until the mortality for complete correction is greatly lowered in this age group. If, after the full treatment for cardiac failure has been tried, the baby still cannot feed or thrive because of breathlessness, surgery must be considered. If the anatomical diagnosis is correct, and the ventricular septal defect is uncomplicated by transposition of the great arteries, or corrected transposition, the surgical results of banding the pulmonary artery are uniformly good. They are also good in our hands in persistent atrioventricular canal. We have not yet corrected these defects in babies after this operation, but this has been successfully done elsewhere. Clearly the object of this surgery is to raise the right ventricular pressure to that of the left, if there is any difference to start with, and so reduce the shunt, but what is so interesting is that even if these pressures are balanced at the start, improvement results if the pulmonary artery pressure is reduced to 20–30 mm Hg. This effect may be due to increased compliance of the lungs with better ventilation resulting.

Our surgical results throughout both 'salvage' and corrective surgery show the same trend; that is, that after the age of 3 months there is a drop in mortality to a level which becomes a reasonable surgical risk. Exceptions to this in our experience are pulmonary stenosis with normal aortic root, and aortic stenosis in which our experience, though small, has been unhappy. The high risk in these two conditions under the age of 1 is due not only to late diagnosis but also to the myocardial state to which the severity of the lesion has also contributed.