BRITISH MEDICAL JOURNAL

LONDON SATURDAY FEBRUARY 10 1962

CAUSES OF SEVERE DEAFNESS IN SCHOOLCHILDREN IN NORTHUMBERLAND AND DURHAM

BY

M. E. BARTON,* M.A., M.B., B.Chir., M.R.C.P.Ed.

S. D. COURT, M.D., F.R.C.P.

AND

W. WALKER, M.D.

From the Department of Child Health, Durham University Medical School, King's College, Newcastle upon Tyne

In 1948 Lande recognized that deafness as well as choreo-athetosis and mental deficiency could follow severe neonatal jaundice. Crabtree and Gerrard (1950) reported that the degree of deafness bore no constant relationship to the extent of the extrapyramidal lesion nor to the level of intelligence and could be the only sequel to neonatal jaundice. Various authors (Goodhill, 1950; Gerrard, 1952; Johnsen, 1952; Cavanagh, 1954; Fisch and Osborn, 1954) have studied the incidence of neonatal jaundice among children with loss of hearing, and as premature infants are particularly liable to develop severe jaundice even in the absence of haemolytic disease of the newborn the incidence of prematurity is given for most of these series. They cannot, however, be compared with each other, because, although most of the children concerned were severely deaf and attending schools for the deaf, some, less severely affected, were in normal schools (Johnsen) while others were attending hospital clinics (Goodhill; Fisch and Osborn). Goodhill's series was the only one in which all the children were resident in a defined area, but in his series the incidence of prematurity was not stated.

We therefore thought it worth while to carry out a retrospective study of the family background and early medical history of all severely deaf children of school age in Northumberland and Durham in order to determine to what extent severe deafness may develop in early infancy either because of neonatal jaundice or in other ways.

Material and Method

We obtained the names of all children resident in Northumberland and Durham and born in the ten years 1945-54, who had been classified as deaf in grades II B and III.† All of these children were attending schools for the deaf and were therefore regarded as educable. Children with various handicaps in addition to deafness

were attending other special schools and institutions and therefore do not come within the scope of this study. Children of school age were selected because we felt that serious deafness if present would have been recognized.

The majority of the children were attending the Northern Counties School for the Deaf in Newcastle upon Tyne, and a pilot study was first conducted on these cases, the headmaster giving us free access to the school records for this purpose and making it possible for us to interview most of the parents during their visits to the school. For the remaining children and those attending other schools‡ we sent a questionnaire to the parents. We asked for information concerning deafness in other members of the family, the mother's health in pregnancy, and any abnormalities associated with delivery. We also asked for the birth weight of the infant, and information about the infant's behaviour and condition immediately after birth and in the neonatal period, the health of the child, and any drugs administered in the years preceding school age; also the diagnosis of deafness. In many instances parents could give information of only limited value, and in such cases, if indicated, we obtained additional details from the records of the family doctor or hospital concerned.

Results

There were 270 severely deaf children in Northumberland and Durham who were born during 1945-54. The parents of 90 were interviewed, 148 questionnaires were completed satisfactorily, and in a further 18 cases adequate information was obtained from school and hospital records. There remained 14 children of whom adequate details were not available, and these have not been considered further.

In only two instances was there a clear history of maternal rubella during the first three months of pregnancy, and deafness was attributed to this cause.

There remain 254 cases, which we have divided into three groups depending on the presumed cause of deafness. Group 1 consists of 64 children in whom there was a strong family history of deafness or other good reason to suppose the deafness was genetically

‡The names of these children were obtained from the chief medical officers of the area concerned.

*During the tenure of a Luccock Research Fellowship, University of Durham Medical School.

†Grade II pupils can hear and understand conversation between 20 and 2 ft. (6.1 and 0.6 m.) away in ordinary classroom conditions. Grade II B are those in need of education in schools for the deaf. The remainder, grade II A, are in normal schools. Grade III pupils can hear and understand conversation at not more than 2 ft. (0.6 m.). All require education in schools for the deaf.—Board of Education (1938) "Report of the Committee of Inquiry into Problems Relating to Children with Defective Hearing." H.M.S.O., London.

determined. Group 2 consists of 69 children in whom deafness followed an infective illness. Group 3 consists of 121 children who have been deaf from a very early age, but in whom there is neither a history of deafness in the family nor of preceding illness.

Group 1. Hereditary Deafness

We included in this group 54 children who had been deaf from birth, and where the same was true of either parent, grandparent, or sibling, aunt, uncle, or first cousin.

Sixteen families having two or more affected siblings accounted for 34 cases. Ten additional children had deaf siblings who were born in years other than those of this study. The remaining 10 had one or more deaf relatives as defined above.

Ten children were also included in this group although no relatives were deaf—two because the parents were first cousins and eight because the child had a genetically determined syndrome of which deafness is a feature—for example, Waardenburgh's syndrome.

Group 2. Post-infective Deafness

Thirty children in this group gave a history of pyogenic meningitis, and in some it was clear that deafness dated from this illness. In 18, because meningitis occurred in the first year of life, it was not possible to say this with certainty. Only five children were more than 3 years of age at the time of the illness. The causal organism was known in only 14 cases, in seven it was the pneumococcus, in five the meningococcus, and in two *Haemophilus influenzae*.

Tuberculous meningitis was regarded as the cause of deafness in 13 children, and it is of interest that in contrast to the cases of pyogenic meningitis all occurred after the age of 18 months—seven after the age of 3 years.

Eight other children became deaf following meningitis, but the available data did not enable it to be classified as pyogenic or tuberculous.

In retrospect we judged from the clinical details that 10 additional children had probably suffered from meningitis prior to deafness, although no specific diagnosis was made at the time of the illness. In eight of these the illness occurred in the first year of life.

Of the remaining eight children in this group the illness preceding deafness had been diagnosed as bilateral otitis media in three cases, as pneumonia in two cases, as measles in one case, as virus labyrinthitis in one case, and as renal disease in one case.

It is possible that drugs used in treatment, especially streptomycin and dihydrostreptomycin, might have

caused the deafness rather than the illness itself. Unfortunately accurate records were not available in many instances, but it is certain that not all received these drugs. Three cases of tuberculous meningitis, however, were known to have received intrathecal streptomycin for five weeks, six weeks, and four months respectively, and at least one case had received intrathecal dihydrostreptomycin. Four other children were also known to have received streptomycin, two with influenza meningitis, one with pneumococcal meningitis, and the child with renal disease.

Group 3. Unexplained Deafness

Two of the 121 cases in this group were known to have suffered from severe neonatal jaundice due to haemolytic disease of the newborn, and this was accepted as the cause of their deafness. One weighed 5 lb. 10 oz. (2,550 g.) at birth and was not admitted to hospital until the fifth day, when evidence of kernicterus was already present. The other weighed 9 lb. 4 oz. (4,195 g.) at birth, the cord haemoglobin was 9 g./100 ml. and bilirubin 8.6 mg./100 ml. Despite exchange transfusion carried out at eight hours, severe jaundice developed, but the bilirubin level was not estimated. In the remaining 119 cases we have examined the available data in an attempt to discover possible causal factors. We have compared our results either with accepted normal values or with our findings in groups 1 and 2, for in these groups the cause of deafness was adequately explained on other grounds.

Maternal Age.—We were unable to demonstrate any relationship between maternal age and the occurrence of deafness in the child.

Birth Weight.—In the Table we have shown the birth-weight distribution for the three groups of deaf children, and also for children attending welfare clinics (Ministry of Health, 1959). As birth weights for school-children were not available these figures were used because they exclude neonatal deaths, which would have increased the proportion in the small-weight groups. There is an excess of premature infants in groups 2 and 3. In group 2 this can be explained on the grounds that many cases followed meningitis in infancy, and premature infants are particularly susceptible to infections (Drillien, 1958). If such cases are excluded the prematurity rate is reduced to 4%, which corresponds with group 1 and with the normal cases. The increased frequency of small infants in the cases with unexplained deafness (group 3) cannot be accounted for so easily. There is a general shift towards the small-weight groups, and the disproportion is especially evident in the very small infants. Thus 21.0% (S.E. 3.75%) weighed 5½ lb. (2,495 g.) or less, compared with 5% of the normals, and

Birth-weight Distribution of Deaf Schoolchildren

	<4 lb.	4-5 lb.	5-5½ lb.	5½-6 lb.	6-7 lb.	7-8 lb.	8-9 lb.	>9 lb.	Not Recorded	Total
Group 1 ..			1 (1.7%)	7 (11.9%)	19 (32.2%)	20 (34%)	7 (11.9%)	5 (8.4%)	5	64
.. 2 ..	1 (1.8%)	2 (3.7%) 1.7%	3 (5.6%)	—	9 (16.7%)	24 (44.4%)	9 (16.7%)	6 (11.1%)	15	69
.. 3 ..	9 (7.6%)	10 (8.4%) 11.1%	6 (5.0%)	11 (9.3%)	33 (27.7%)	31 (26%)	15 (12.6%)	4 (3.4%)		119
Normal*	(0.2%)	21.0% (1.4%)	(3.4%)	(4%)	(25%)	(36%)	(21.7%)	(8.3%)		19,616
		5%								

* Ministry of Health (1959).

in fact 7.6% (S.E. 2.41%) weighed less than 4 lb. (1,815 g.) compared with 0.2% of the normals.

Birth Rank.—Fifty-nine per cent. of the children with post-infective deafness (group 2) and 49% with unexplained deafness (group 3) were first-born, compared with 33% with hereditary deafness (group 1) and 39.9% in the general population of England and Wales (Registrar-General, 1959). First-born children have lower birth weights than subsequent infants, and the association between birth rank and deafness may largely be due to the more marked association between deafness and low birth weight.

Place of Birth.—A comparable number of first-born children in the three groups were born in hospital—namely, 50% of those in group 1, 64.5% of those in group 2, and 60.5% of those in group 3. Among subsequent children, however, only 32% of those in group 1 and 20% of those in group 2 were born in hospital, compared with 55% of those in group 3. Of the 32 children in group 3, 18 were delivered in hospital because of multiple pregnancy, prematurity, pre-eclamptic toxæmia, or difficulties associated with delivery. These factors themselves may have been responsible for the deafness.

Mode of Delivery.—The incidence of abnormal deliveries was 19.5% among children with unexplained deafness, compared with 9% of those with hereditary deafness and 4% of those with post-infective deafness. Caesarean section was uncommon in all groups, but other difficulties associated with delivery—for example, forceps delivery, precipitate delivery, or strangulation by the cord—occurred in 13.5% of cases of unexplained deafness but in only 2% of the cases in the other two groups combined. Similarly, there was a higher incidence of breech deliveries among children with unexplained deafness (5%) than in the other two groups combined (2%). In nearly half of these abnormal cases prematurity was also a feature and may have been a factor of the nature of the delivery, although the converse is also possible. It is not possible to be certain that any of these difficulties were factors in the causation of deafness.

Neonatal Jaundice.—Excluding the two cases of haemolytic disease of the newborn, there was a history of neonatal jaundice in 16 (13.5%) children with unexplained deafness but in only 7 (5.25%) in the other two groups. In these seven and in nine of the children with unexplained deafness, the jaundice was mild and of short duration, and was not considered a probable factor in the causation of deafness. However, the remaining seven children with unexplained deafness were all severely jaundiced, and it was thought probable that jaundice had played a part in the development of deafness. Six of the seven were premature, with birth weights ranging from 1 lb. 12 oz. to 5 lb. (795 to 2,270 g.), and in two the onset of respiration was delayed for 15 minutes and 30 minutes.

Anoxia at Birth.—Thirty children had some delay in establishing regular respiration; 10 in group 1, 2 in group 2, and 18 in group 3. However, in the majority such delay was of only short duration, but in four cases in group 3 there was severe anoxia of at least 30 minutes' duration. One of these children was premature, and also had severe neonatal jaundice; the other three were mature infants. It was thought probable that anoxia had been a factor in the causation of deafness in these four cases.

Discussion

This is a retrospective study of 270 children resident in Northumberland and Durham, born in the years 1945–54, who were so deaf as to require special schooling, but without other handicaps that necessitate institutional care. This represents an incidence of severe deafness of 0.7 per 1,000 population, which agrees with the estimate of 0.7–1.0 for the whole country (Ministry of Education, 1946). Details of the family background and health prior to deafness have enabled us to divide the cases into three broad groups. Approximately 25% had a strong family history of deafness or a syndrome of which deafness is a feature. In a further 25% deafness followed an infective illness, usually meningitis; but as this often occurred in the first year of life it is impossible to be sure that normal hearing was present before the illness. The remaining 50% had no obvious cause for deafness, and it is mainly with these children that this study is concerned.

Rubella in the first three months of pregnancy was a factor in only two cases. In four additional cases a virus infection had probably occurred at this time—measles in one, influenza in one, and an unidentified virus in the other two. Even assuming a causal relationship in all six cases, this would account for only 2.2% of deafness. The contribution of rubella to deafness in various series has varied greatly—2.55% in that of Cavanagh (1954), 7.7% in that of Fisch and Osborn (1954), but as high as 20.5% in the cases reported by Goodhill (1950). These differences can probably be accounted for on the method of selection of the cases of deafness, but in Goodhill's material the very high incidence was probably because a local epidemic of rubella occurred at the time of conception. It has been estimated (*Lancet*, 1960) that if rubella occurs during the first 12 weeks of pregnancy the risk of deafness in the infant is 19%, but 12% if occurring during the thirteenth to sixteenth week of pregnancy. In any event, maternal rubella accounts for only a very small proportion of deafness.

Haemolytic disease of the newborn was judged to be the causal factor in two cases, but in one case exchange transfusion had not been performed, while in the other, although exchange transfusion had been performed early, subsequent bilirubin estimations or repeated exchange transfusions were not carried out. In other words, the management of both these cases was unsatisfactory, judged by present standards. In the present series haemolytic disease of the newborn accounted for less than 1% of cases, a lower incidence than reported by other workers—1.4% by Gerrard (1952), 2.5% by Cavanagh (1954), and 3% by Fisch and Osborn (1954). These differences are probably explained on the grounds that the material in these series differed, but with present-day management severe deafness as a result of haemolytic disease of the newborn should be extremely rare. Lesser degrees of high-frequency deafness may be more common, however, and probably explains the 10% incidence of haemolytic disease of the newborn in children who were attending municipal schools (Johnsen, 1952).

Jaundice of prematurity, not associated with blood-group incompatibility, may also lead to kernicterus and deafness (Aiden *et al.*, 1950; Govan and Scott, 1953; Crosse *et al.*, 1955). The prematurity rate in our material was high, 12.5%, compared with the expected

5%, but the proportion varied, depending on whether deafness was regarded as hereditary, post-infective or unexplained. In fact, 21% of the children in whom deafness was unexplained weighed 5½ lb. (2,495 g.) or less at birth, and, moreover, many were very small. Thus 7.6% weighed 4 lb. (1,815 g.) or less at birth, whereas the expected incidence is only 0.2%. A history of severe neonatal jaundice, however, was obtained in only 18% of the premature infants.

We think that severe jaundice may have passed unrecognized in some, because of the six in whom severe jaundice was recorded five were born in one or other of two large maternity units where observation was probably more complete.

The prematurity rate is significantly high in the group of children with unexplained deafness. Only a small proportion can be accounted for by the occurrence of severe jaundice, and only one on the basis of anoxia. Although 50% of the premature infants had abnormal deliveries, it is not possible to say whether this played any direct part in the cause of deafness.

There is little doubt, however, that an association occurs between deafness and prematurity, an observation also recorded by Gerrard (1952) in 18.3% of cases and by Johnsen (1952) in 23%. Fisch and Osborn (1954), on the other hand, recorded a prematurity incidence of only 7.6% but took into account only those cases where they regarded prematurity as the cause of deafness. Corner (1960) in a follow-up study of 174 infants with a birth weight of less than 3 lb. 4 oz. (1,473 g.) found seven with cerebral palsy, of whom three were deaf. All three were cases of prematurity kernicterus. Five additional children had deafness as the only stigma. Severe jaundice was a factor in only two of the others (Corner, 1960, and personal communications).

The headmaster of Longwill School, which serves South Birmingham, has kindly provided comparative information from his school. Of the 120 pupils, 11 were premature and four gave a history of neonatal jaundice; this corresponds closely with our observations in Northumberland and Durham. We calculate that severe deafness is present in 1.7 out of every 1,000 premature infants who survive, and the incidence is higher for infants with a birth weight of 4 lb. (1,815 g.) or less.

One mature infant in our series had deafness which we attributed to neonatal jaundice, although blood-group incompatibility was excluded; and other workers have also regarded physiological jaundice in mature infants as the cause of deafness (Gerrard, 1952; Johnsen, 1952). Gerrard in fact suggested that, whereas premature infants who develop kernicterus will probably die, bigger infants may survive with sequelae.

We regarded anoxia as the cause of deafness in three mature infants, a 1% incidence, lower than the 19% and 9% in the series of Johnsen (1952) and of Fisch and Osborn (1954) respectively. A quarter of all our cases apparently followed an infective illness, nearly always meningitis. In Goodhill's series 10.3% of deafness was attributable to meningitis, and in Cavanagh's 13.5% followed pyogenic meningitis and 5% tuberculous meningitis.

Streptomycin used in the treatment of infection, especially tuberculous meningitis, may itself cause deafness, and Sita Lumsden and Powell (1953) calculate that in adults streptomycin in a dosage of 1 g. daily is two to three times as likely to cause vestibular

disturbance as dihydrostreptomycin in similar dosage, but the converse is the case with regard to deafness. Baqai *et al.* (1960), however, reported that prolonged administration of streptomycin to children rarely produced either effect. However, if the intrathecal route is employed the risk is increased. Thus Nickerson and MacDermot (1961) reported that seven out of nine children who had received 50 mg. of streptomycin intrathecally for a period of 200 days because of tuberculous meningitis became deaf, but when the dosage was reduced to 25 mg. no deafness occurred in 35 surviving children so treated.

Conclusion

Approximately 27 infants born in Northumberland and Durham each year will require education in schools for the deaf.

In 25% of these deafness will be hereditary in origin, and at present there is little that can be done to prevent this.

A further 25% will develop deafness as a sequela to severe infections, often meningitis, especially in the first year of life. The early diagnosis and prompt treatment of meningitis is imperative if deafness is to be prevented. Hospital admission is indicated in all such cases, and the follow-up should be planned so that, if deafness does occur, early recognition is achieved and suitable education instituted.

The remaining 50% of cases of deafness arise from other causes, and only isolated cases are due to the occurrence of maternal rubella during pregnancy, severe neonatal jaundice, or anoxia. The majority remain unexplained, although we have demonstrated an association with prematurity in many such cases. Whether prematurity *per se* is a factor or whether the factors causing prematurity are responsible is not as yet established. In the meanwhile the most practical way to help deaf children is by achieving early diagnosis (*British Medical Journal*, 1961), and to do this early hearing tests on children who are known to be "at risk" are indicated.

Summary

A total of 270 children born in Northumberland and Durham in the years 1945-54 are receiving education in schools for the deaf. This represents an incidence of severe deafness of 0.7 per 1,000, the same as the national incidence.

We have studied the early medical history and family background of these children: 25% were classified as having hereditary deafness, in 25% deafness followed meningitis or some other infection, but in 50% the cause of deafness was unknown.

Of the unexplained cases there was a history of maternal rubella during the first three months of pregnancy in two instances, while two more children had suffered from severe haemolytic disease of the newborn. Of the remainder, 21% were "premature" infants, 7.6% having a birth weight of 4 lb. (1,815 g.) or less. Only 18% of the premature infants gave a history of severe jaundice, and it would appear that prematurity even in the absence of jaundice may be an important cause of deafness.

We thank all who made this inquiry possible, particularly Mr. W. Wearmouth and the staff of the Northern Counties School for the Deaf, Newcastle upon Tyne; Mr. H. H. Shorrocks, of the Longwill School, Birmingham; the Chief

School Medical Officers of Northumberland and Durham ; and all hospital staff and general practitioners who gave us access to their records. We also thank Dr. Beryl Corner for additional information of her premature-baby follow-up, Dr. George Knox for statistical advice, and Mr. J. I. Munro Black for helpful criticism. Secretarial assistance was financed by a grant from the Dowager Countess Eleanor Peel Trust.

REFERENCES

- Aiden, R., Corner, B., and Tovey, G. (1950). *Lancet*, **1**, 1153.
 Baqai, M. A., Lorrman, G., and Bentley, F. J. (1960). *Brit. med. J.*, **2**, 306.
Brit. med. J., 1961, **2**, 163.
 Cavanagh, F. (1954). *J. Laryng.*, **68**, 444.
 Corner, Beryl (1960). *Prematurity*, p. 504. Cassell, London.
 Crabtree, N., and Gerrard, J. (1950). *J. Laryng.*, **64**, 482.
 Crosse, V. M., Meyer, T. C., and Gerrard, J. W. (1955). *Arch. Dis. Childh.*, **30**, 501.
 Drillien, C. M. (1958). *Ibid.*, **33**, 10.
 Fisch, L., and Osborn, D. A. (1954). *Ibid.*, **29**, 309.
 Gerrard, J. (1952). *J. Laryng.*, **66**, 39.
 Goodhill, V. (1950). *Ann. Otol. (St. Louis)*, **59**, 1123.
 Govan, A. D. T., and Scott, J. M. (1953). *Lancet*, **1**, 611.
 Johnsen, S. (1952). *Acta oto-laryng. (Stockh.)*, **42**, 51.
Lancet, 1960, **2**, 800.
 Lande, L. (1948). *J. Pediat.*, **32**, 693.
 Ministry of Education (1946). "Special Educational Treatment," Pamphlet No. 5. H.M.S.O., London.
 Ministry of Health (1959). *Reports on Public Health and Medical Subjects*, No. 99. H.M.S.O., London.
 Nickerson, G., and MacDermot, P. N. (1961). *Pediatrics*, **27**, 68.
 Registrar-General for England and Wales (1959). *Statistical Review for the Year 1957*, Part II, p. 162. H.M.S.O., London.
 Sita Lumsden, E. G., and Powell, R. J. (1953). *Tubercle (Lond.)*, **34**, 324.

MYRINGOPLASTY

SURGICAL REPAIR OF TYMPANIC MEMBRANE PERFORATIONS

BY

STUART MAWSON, F.R.C.S., D.L.O.

Consultant E.N.T. Surgeon, King's College
 Hospital and Queen's Mary's Hospital,
 Roehampton, London

AND

BRIAN PICKARD, F.R.C.S., D.L.O.

Consultant E.N.T. Surgeon, St. George's Hospital,
 London ; late Senior Registrar in E.N.T.,
 King's College Hospital, London

The tympanic membrane is a diaphragm capable of being set in vibratory motion by sound waves. The movement takes place at the periphery of the membrane, the central portion moving in and out like a plunger. The plunger action is transmitted to the oval window by the ossicular chain with a hydraulic effect ; that is to say, the disparity in sizes between the two membranes results in augmentation of energy per unit area. In addition, there is a slight augmentation by the lever action of the ossicles. The handle of malleus (integral with the membrane) is longer than the long process of the incus, and the two bones together act as a bent lever round an axis running roughly through their bodies. The ratio of advantage derived from the hydraulic and lever effects is about 18 to 1. Impairment of the tympanic membrane therefore is inseparable from some impairment of auditory function. The degree of impairment, however, will not be a simple function of the size of perforation, but will depend also upon the position of the defect and the integrity of other portions of the conducting mechanism.

The passage of a sound-wave through the cochlea demands a sound pressure differential between the oval and round windows ; the sound "flows," as it were, down the pressure gradient between them. For optimal function the sound must be concentrated at one window and "kept" from the other. The round window is normally "kept" from sound, paradoxically, by the tympanic membrane. A defect in the membrane may therefore allow exposure of the round window to sound, with reduction of the differential and impairment of hearing additional to that sustained by loss of hydraulic and leverage action at the oval window. It goes without saying that these hydraulic and leverage actions will be lost anyway if there is any disruption or fixation of the ossicular chain. It may also be accepted that part of the essential conducting mechanism is an air-transmitting eustachian tube : free vibration of tympanic membrane and inner-ear membranes requires air in the middle ear at atmospheric pressure.

Finally it is a vital condition of perfect hearing that the cochlea and its central connexions should be unimpaired. Hearing cannot be made better than the cochlear potential.

The auditory effect of a breach in the integrity of the tympanic membrane is therefore variable and somewhat unpredictable, as is the auditory prognosis associated with repair. Repair will be efficacious in restoring hearing loss in proportion as the hearing loss can be attributed to the defect. Where other factors are involved—for example, discontinuity of the ossicular chain—the result of repair may be ineffective as regards the hearing. The selection of cases for myringoplasty for this and other reasons is of great importance.

The Safe Ear

Persistent perforations of the tympanic membrane are for practical purposes legacies of either otitis media or traumatic rupture. Renewed interest in the investigation and treatment of perforations has been partly due to the post-war appearance of greater numbers of traumatic rupture in which the defect appeared to be the only barrier in many cases to normal hearing and a normal ear (Fox, 1945 ; Baron, 1948 ; Derlacki, 1953). Persistent cases of traumatic rupture are deserving of every effort towards closure and generally constitute cases presenting no inherent difficulty in selection for operation.

It is when the perforation is a legacy of otitis media that difficulty in selection arises. Prior to the advent of antibiotics it was an accepted and strongly held principle of otology that free drainage of infections of the middle ear and mastoid was the essential condition for safety. The control of infection without drainage (witness the spectacular decrease in myringotomy and mastoidectomy for acute infections) is a post-war phenomenon. Acute infections are now invariably treated with antibiotics, drainage being reserved only for those cases where pus is under tension or persistent, or where complication is threatened. Antibiotics, however, did not and do not allow the same control in chronic as in acute infections,