Preventable death in oesophageal atresia

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SUMMARY We report on experience gained in the treatment of 158 cases of oesophageal atresia presenting during a period of 10 years. The factors influencing mortality were analysed. During the period studied there was a slight improvement in survival, and this was probably due mainly to improved preoperative preparation of those babies undergoing primary repair. At best, 'staging' was thought to have had little influence on the survival of poor risk cases. Midwives, obstetricians, paediatricians, surgeons, and general practitioners did not do all that they could have done to prevent morbidity and mortality in these babies. At least one-third of the 79 deaths could have been prevented, and several deaths were caused solely by lack of awareness of the possible complications and associated abnormalities.

Although much progress has been made in the treatment of infants with oesophageal atresia since 1943 (Haight and Towsley), before which time practically all cases died, there is still an unacceptably high mortality rate for 'poor risk' cases. Formerly, technical errors were responsible for many deaths, but advances in operative techniques, preoperative management, and paediatric anaesthesia have succeeded in substantially reducing the overall mortality rate. Since it is unlikely that any new operation of major importance will be developed in the foreseeable future, it is necessary to look to other factors in order to improve survival in these infants, particularly the early nursing and paediatric care, the preoperative management and choice of operation by the surgeon, and the subsequent community and environmental care.

Patients and methods

A retrospective review was carried out on a consecutive series of 158 infants with oesophageal atresia admitted to the Neonatal Surgical Unit of the Sheffield Children's Hospital in the 10-year period from January 1965 to December 1974. Cases of oesophageal stenosis and isolated tracheo-oesophageal fistula without atresia were excluded.

Initial surgery consisted of one of three approaches.

(i) *Primary end-to-end anastomosis* of the oesophageal ends after ligation and division of the tracheo-

oesophageal fistula was the treatment of choice. Occasionally an end-to-side anastomosis was used. This group included all repairs during the first week after admission. A transpleural approach was used initially, but it was abandoned in favour of an extrapleural technique. A transanastomotic feeding tube was used in most cases, but a few infants had a gastrostomy constructed.

(ii) *'Staged repair'*, i.e. gastrostomy followed several weeks later by a primary anastomosis. The upper pouch was kept empty by sump drainage (Replogle tube). Lateral oesophagostomy was used in a few early cases but proved to be unsatisfactory and hazardous.

(iii) *Gastrostomy and end-oesophagostomy* followed by ligation of the tracheo-oesophageal fistula when the infant was judged to be fit for thoracotomy. This method of treatment was mainly restricted to babies of low birthweight in poor general condition, and those cases with isolated oesophageal atresia without a fistula, but was used in some cases where there was a considerable gap between the oesophageal ends.

One of these three methods of treatment was used in 141 cases. 11 cases were treated in various ways, e.g. neonatal colon interposition or the surgery was modified because of other associated anomalies, and 6 cases were thought to be unsuitable for surgery because of multiple congenital abnormalities combined with poor general condition.

The results were compared by the χ^2 test with use of Yates's correction for small numbers.

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Results

The details of 156 of the 158 cases were available for analysis. The only information about the remaining 2 cases was that death occurred within the first few weeks of life.

Groups. The cases were allocated to 'risk groups' according to the criteria of Waterston *et al.* (1962). *Group A:* birthweight >2500 g and in good general condition, 50 cases (32%). *Group B:* birthweight

Fig. 1 Results of all forms of treatment in groups A, B, C, according to Waterston *et al.* (1962), for 156 cases.

1800–2500 g, or with 'moderate' pneumonia, or with 'moderate' associated anomaly, 35 cases (22%). *Group C:* birthweight <1800 g, or with 'severe' pneumonia, or with 'severe' associated anomaly, 71 cases (46%).

The survival rates according to this classification are shown in Fig. 1. There were an inordinate number of cases in group C and this had an adverse effect on the overall survival rate.

Anatomy of the lesions. The classification used was that of Vogt (1929) as modified by Ladd (1944) and Roberts (1958), but types V (E) and VI (F) were excluded from this series. The exact anatomy in 145 cases is shown in Fig. 2. An upper pouch fistula was found in 5 (4%), and a distal fistula in 135 (93%).

Sex ratio. There were 91 boys (58%) and 67 girls (42%) in this series, and the initial survival rate was 59% in boys and in girls 64%.

Age on admission. This was determined in 156 cases, no information being available for 2 cases, 1 being admitted in the first 5 years of the survey and 1 in the second. In the first 5-year period, 35 of the 81 cases (43%) were admitted after the first 24 hours of life, compared with only 15 of the 75 cases (20%) in the second 5-year period. (This is a significant difference: $\chi^2 = 8.61, 0.01 > P > 0.001$.)

Comparative survival rates show no significant increase in mortality attributable to delay in diagnosis. In the first 5-year period survival rates were 54% and 51% according to whether the child was admitted within 24 hours of birth or later. In the second 5-year period the respective survival rates were 68% and 87% ($\chi^2 = 1.19, P > 0.10$).

Fig. 2 Anatomy of the oesophageal lesion and the effect on survival for 145 cases.

Preoperative contrast studies and pneumonia. 13 infants had preoperative studies of the upper oesophageal pouch, a variety of contrast materials being used. 6 babies died, and 7 survived (survival rate 54%); thus the chance of survival in this group was not significantly different from that of 50% for the series as a whole.

The pulmonary complication rate in the 'contrast-studied group' (54%) was not significantly greater than that for the noncontrast group (51%) ($P > 0.50$).

Initial treatment and survival. The 'initial survival' was calculated at the time of the first discharge from hospital.

(i) **Primary repair.** Primary repair of the abnormality was carried out in 84 cases, 21 (25%) of whom died during the initial admission. There was a significant improvement in survival rate in the second 5-year period (86%) when compared with the first 5-year period (58%) ($\chi^2 = 7.34$, $P > 0.001$) (see Fig. 3). The causes of death can be seen in Table 1.

Fig. 3 Comparison of the results of the three main types of treatment for each method as a whole and for each 5-year period, 1965–1969 and 1970–1975. Overall total of 141 cases.

Table 1 Causes of 'initial death' in cases of primary repair, 1965–1974

Cause of death	No. cases
Anastomotic leak followed by sepsis	5
Congenital heart disease	4
Recurrent tracheo-oesophageal fistula	2
Multiple congenital abnormality	2
Anastomotic leak + sepsis + recurrent tracheal fistula + congenital heart disease	1
Pneumothorax	1
Ruptured bronchus	1
Pneumonia + sepsis	1
Gastric trans-section + peritonitis	1
Enterocolitis	1
Choanal atresia	1
Immaturity	1
Total	21

At the time of review there were 56 survivors, 7 of the children having died after the initial discharge from hospital. Of the survivors, 12 (21%) had developed a stricture (defined as a symptomatic narrowing requiring more than 3 dilatations) which nearly always improved with the passage of time and a bougie, 2 (4%) had had a recurrent fistula ligated, 1 had had a stricture and a recurrent fistula treated, and 2 had had the primary anastomosis abandoned because of gross leakage, an oesophagostomy being constructed in each case. 39 (70%) of the survivors made an uneventful recovery.

(ii) **Staged primary repair.** No such repair was done before the 46th day of life, and the longest interval between birth and anastomosis was 161 days (mean 88.5 ± 33.5 SD days). This method of treatment resulted in success in only 5 of the 34 cases (15%) and had an initial mortality rate of 53%. The repair or attempted repair was abandoned in 29 cases (85%) and 24 of these died eventually, 6 after the first discharge. The other 5 survivors in this group were salvaged by oesophagostomy and later underwent successful colon interposition. There was no change in mortality over the 10-year period.

In addition to the 34, there was one unusual case of a girl who had had a disrupted primary anastomosis treated by oesophagostomy and gastrostomy and then had a successful delayed or 'secondary' anastomosis constructed; although a stricture developed which required numerous dilatations, the child could swallow normally at the time of review.

(iii) **Oesophagostomy and gastrostomy.** This method was used in 23 cases and the 15 (65%) survivors subsequently required colon interposition to restore oesophageal continuity. There was no change in mortality rate during the period of the review.

Table 2 shows that treatment by staged primary repair seemed to carry the greatest mortality.

Table 2 Initial mortality rates of each of the three main types of initial surgery, according to Waterston's groups for 141 cases from 1965 to 1974

Group	Mortality rate as % of 141 cases			
	Primary repair	Oesophago-gastrostomy-gastrostomy ligation fistula	Staged primary repair	Total
A	4.9	0	16.7	6
B	16.7	12.5	25.0	17.6
C	64.0	58.3	75.0	66.7
Total	25.0	34.8	52.9	33.3

However, the differences in mortality rates were not significant, except that overall primary repair gave the best results ($0.01 > P > 0.001$).

The figures for initial survival are given in Fig. 1 for 156 cases; and 2 additional cases had died during the first few weeks, giving an overall initial survival rate of 61% (97 of 158 cases). The initial survival rate for 1965/69 was 52% and for 1970/74 was 71%; this was probably a significant improvement ($\chi^2 = 5$, $0.05 > P > 0.02$). However, there was no significant improvement in survival in any one of the risk groups A, B, or C, and the apparent improvement probably occurred only in those undergoing primary repair. Also there was a decrease in the number of poor risk cases in the second half of the series.

All 3 deaths in group A were due to surgical error, as were 6 of the 7 deaths in group B. Enterocolitis of the terminal ileum was responsible for the remaining death in group B. In group C, associated major congenital anomalies contributed greatly to the mortality. In 6 cases these anomalies were so severe and the general condition of the children so poor that death was inevitable, 4 babies being admitted in the first 5 years and 2 in the second. None of these cases had surgical operations and obviously, all died. The postoperative survival rates are given in Table 3.

Delayed mortality. At the time of review exactly 50% of the children were alive. After first discharge from hospital a further 18 children had died (23% of the total deaths). Table 4 lists the causes of these late

Table 3 Initial survival rates in 150 cases undergoing surgery excluding 2 cases with insufficient detail

Group	% survival		
	1965-1969	1970-1974	1965-1974
A	90.9	96.4	94
B	77.8	82.4	80
C	24.3	46.4	33.8
Total	55.8	74	64.7

deaths. No deaths followed colon interposition in cases admitted as neonates after 1967, except for 1 infant in group C in whom the operation was performed in the neonatal period in 1969.

Associated anomalies (Table 5). Some children had several anomalies and each is listed separately. A total of 64% of the infants had at least one associated defect. The effect on survival is shown in Table 6.

Table 4 Causes of late deaths (18 cases)

Cause of death	
<i>Admissions 1965-69</i>	
Group A	
1	Uncertain; died at home with oesophagostomy + gastrostomy
2	Respiratory failure after colon transplant
3	Gastropleural fistula after colon transplant
4	Pneumonia, empyema, bilateral hydronephrosis
5	Cerebral haemorrhage and anoxia; subglottic stenosis
Group B	
6	Pyocyanus septicaemia after colon transplant
7	Gastrostomy diarrhoea + pneumonia from measles
8	Mongol; cause of death uncertain
9	Pneumonia from spastic quadriplegia
10	Aspirated vomitus after colon transplant
11	'Asphyxia' + bilateral hydronephrosis
Group C	
12	Uncertain; sudden death at home
13	Mongol; viral pneumonia
<i>Admissions 1970-74</i>	
Group A	
14	<i>E. coli</i> peritonitis and septicaemia from gastrostomy
Group B	
15	Otitis media, meningitis, and cavernous sinus thrombosis
16	Uncertain
Group C	
17	Pneumonia and congenital heart disease
18	Mastoiditis, meningitis, and hydrocephalus

Table 5 Associated anomalies in 156 cases

Anomaly	No. cases	%
Renal (details known in only 84 cases)	25	30
Skeletal (excluding cleft palate and sacral agenesis in anorectal cases)	27	17
Cardiovascular (excluding isolated right-sided aorta)	26	17
Anorectal	22	14
Ear defects	18	11.5
Cleft lip and palate	10	6.5
Abnormal penis or vulva	8	5
Hydrocephalus; microcephalus	8	5
Duodenal atresia	7	4.5
Undescended testis (testes) in 89 boys	4*	4.5 boys
Malrotation	5	3
Right-sided aorta	5	3
Eye defects	5*	3
Defects of jaws other than cleft lip	5	3
Meckel's diverticulum	3	2
Volvulus	3	2
Down's syndrome	3	
Pyloric stenosis or atresia	3	

*Almost certainly underestimates of true figures. In Liverpool, Brereton (1976) found a 13% incidence of undescended testis.

Table 6 Associated anomalies present in 64%

Group	Living	Early death	Late death	Total
<i>No other anomalies present</i>				
A	28	1	1	30
B	11	3	3	17
C	5	4	0	9
Total	44	8	4	56
<i>Other anomalies present</i>				
A	13	2	5	20
B	11	4	3	18
C	11	45	6	62
Total	35	51	14	100

Two cases excluded as details were insufficient.

Discussion

Delay in diagnosis in this series did not affect prognosis, although Hamilton (1969), Swenson *et al.* (1962), and Myers (1974) reported a progressive rise in mortality associated with increasing age at diagnosis, presumably due to pulmonary complications. The review of the American Academy of Pediatrics (Holder *et al.*, 1964) found the worst prognosis in those cases diagnosed on the first day of life. This could have been due to two factors. Firstly, that infants of low birthweight, or with pulmonary complications and obvious severe associated anomalies, tended to be diagnosed at an early stage. Secondly, that group A cases were better able to withstand the ensuing pulmonary complications which accompany late diagnosis. In our series, 32% of cases were not diagnosed until after the lapse of at least 24 hours, but there was an inordinate number of group C cases (46%) and these two factors counterbalanced one another, differences between the survival rates for group C cases discovered early and group A cases discovered late clouding the advantages of early diagnosis.

Because of feeding problems several of the babies had been 'tube fed' before diagnosis. This could have been avoided by the passage of an inflexible wide bore tube (10F) followed by testing for acid reaction with litmus paper in all infants with dysphagia or respiratory symptoms (particularly those born to mothers having had polyhydramnios, Hamilton, 1969). Optimum care during transportation to a surgical unit could also minimise respiratory complications and reduce the overall mortality and morbidity (Koop *et al.*, 1974; Blake *et al.*, 1975). Sometimes the possibility of oesophageal atresia is not considered in cases of imperforate anus, cleft lip, cardiac anomaly, duodenal atresia, and 'respiratory distress'. On occasion such infants have received inappropriate treatment until the diagnosis has become embarrassingly obvious.

Some paediatricians send neonates suspected of having oesophageal atresia for radiological contrast studies. Although no danger seems to have come to the babies so treated in this series, the practice is condemned as unnecessary (Freeman, 1969; Hamilton, 1969) and dangerous should the material be inhaled (Koop *et al.*, 1974; Saron *et al.*, 1975), especially in studies using the hyperosmolar 'gastrografin' which may cause severe pulmonary oedema.

The operative repair of oesophageal atresia should not be considered to be a surgical emergency. Many infants with respiratory complications could benefit from preoperative intensive physiotherapy and antibiotic chemotherapy, as well as correction of acid-base and fluid and electrolyte imbalances and hypothermia (Haight, 1944; Gross, 1953). A period of 24-48 hours of such therapy may be life-saving. In the early part of this series 10 infants were subjected to surgery when, in retrospect, the preoperative resuscitation had been inadequate as judged by present standards. None of these survived.

The use of preoperative tracheobronchial lavage and aspiration (Hertzler, 1965) and particularly using miniature fiberoptic bronchoscopes during anaesthesia (G. Bush, personal communication, 1976) may be of considerable value in improving the pulmonary status. The latter technique allows the study of the trachea for the position of fistulae, tracheomalacia, and extrinsic compression by vessels.

Accurate elucidation of associated anomalies (Gruchalski *et al.*, 1976) in group C infants, with special reference to those lesions incompatible with survival, such as severe intracranial haemorrhage, bilateral renal agenesis, gross cardiac abnormalities, and various trisomies may influence the management of the affected child (Myers, 1974; Cozzi and Wilkinson, 1975). Low birthweight has been shown to be a significant factor in the mortality (Haight, 1944; Holder *et al.*, 1962; Martin, 1965; Abrahamson and Shandling, 1972), and Hertzler (1965) reported a 52% overall survival rate for infants with low birthweight, falling to 22% in the presence of other congenital anomalies (see Table 6).

Pneumonia was directly responsible for only 7% of the deaths in this series, all in group C, although 50% of the cases developed pneumonia at some stage during the initial admission. This contrasts with the finding of Holder *et al.* (1964) and Hamilton (1969) who reported respectively that 62% and 50% of the deaths in their series were attributable to pneumonia and that the prevention of this complication would be the single most significant factor leading to improved survival rates.

The overall survival rate of 50% and initial survival rate of 61% compare fairly well with those

reported by others (Waterston *et al.*, 1962; Koop *et al.*, 1974; Cozzi and Wilkinson, 1975). We cannot claim 100% survival for group A cases (Holder *et al.*, 1962; Koop *et al.*, 1974; German *et al.*, 1976), nor can we match the 97% success rate reported by Martin (1965) for all cases with a birthweight greater than 2000 g. However, Martin was able to perform a primary anastomosis in the majority of his cases, whereas in this series primary anastomosis was feasible in 82% of group A cases, in only 51% of group B, and in 35% of group C. From Philadelphia (Hamilton, 1969) it was reported that the two ends of the oesophagus were in partial contact in half the cases, but this occurred in only 4% of our cases.

There is no doubt that primary repair via the retropleural approach produces the best results (Holder *et al.*, 1964). In cases where the oesophageal ends are far apart following ligation of the fistula, due consideration should be paid to the possibility of freeing the stomach (Zachary, 1965), or to pulling up the stomach by means of a Foley balloon catheter (Hertzler, 1965), or to lengthening the upper pouch by serial myotomy (Livaditis, 1973; Eraklis *et al.*, 1976) in order to gain extra oesophagus for an anastomosis under less tension. Hamilton (1969) reported that anastomotic leaks occurred in 17% of his primary repairs and that in the presence of a large leak, oesophagostomy (and gastrostomy, if not already done) should be performed. In this series four operations other than oesophagostomy and gastrostomy were performed for anastomotic leaks, and as a result all 4 infants died.

When primary anastomosis was technically impossible, particularly for infants in groups B and C, the best overall initial survival rate was obtained by ligation of the tracheo-oesophageal fistula, cervical oesophagostomy, and the construction of a feeding gastrostomy. However, the construction of an oesophagostomy condemns the child to a colon interposition, gastric tube replacement, or to intrathoracic oesophagogastric anastomosis (Vaage *et al.*, 1975), the long-term results of which are usually inferior to those from direct oesophageal anastomosis. We would agree with Cozzi and Wilkinson (1975) that 'staging' has not produced the benefits that had been anticipated (Holder *et al.*, 1962; Koop and Hamilton, 1965; Martin, 1965; Glasson *et al.*, 1971). Abrahamson and Shandling (1972) showed that 'staging' could be harmful except for desperately ill infants with severe respiratory problems or with several gross associated anomalies. There is no need to stage treatment on grounds of birthweight alone (Myers, 1974). The policy of 'staged primary repair' was associated with the most danger in this series and should not be

used unless optimal facilities are available, and the surgeon must be willing to abandon this form of treatment at the first sign of deterioration. Cervical oesophagostomy will not save a neglected, moribund infant.

In this series 13 (24%) of the 55 postoperative deaths were assessed to be preventable, and all of those in groups A and B. Cardiopulmonary pathology was responsible for 25% of the deaths, intra-abdominal causes for 18%, and multiple associated anomalies including chromosomal defects for 11%. Tension pneumothorax caused the death of 5 (9%) infants and was a major contributing factor in a further 2.

Early recognition and prompt treatment could have prevented many of these deaths. It is particularly distressing that on occasion junior medical staff did not appreciate that a gasping, blue infant could be suffering from the effects of tension pneumothorax, although in one or two cases the operated side was aspirated and the pneumothorax was on the contralateral side. Other complications that tended to cause confusion and were associated with delay in diagnosis included Meckel's diverticulitis, 'enterocolitis' of the stomach or intestine, malrotation and volvulus, duodenal stenosis, pyloric stenosis, and peritonitis due to leakage from a gastrostomy. Several of these complications have been recorded (Schwartz and Dale, 1955; Berg, 1956; Ahmed, 1970; Glasson *et al.*, 1973), and in particular those consequent upon gastrostomy (Connor and Sealey, 1956; Holder *et al.*, 1964; Cozzi and Wilkinson, 1967; Glasson *et al.*, 1971; Koop *et al.*, 1974). 3 babies in our series died during the initial admission because of complications arising from the gastrostomy.

The incidence of associated abnormalities varies according to the diligence with which they are sought. Holder *et al.* reported a 52% incidence and claimed that 43% of the deaths were caused by these abnormalities. R. J. Brereton (1976, unpublished) and German *et al.* (1976) report figures as high as 68% and 70% respectively. In this series 64% of cases had an associated anomaly of varying severity.

There may be an appreciable mortality rate between the first discharge from hospital and final accomplishment of normal swallowing. This late mortality may be related to secondary attempts at the restoration of oesophagogastric continuity, recurrent respiratory tract infections, the late effects of associated anomalies, or be due to unrelated factors. Many of these children, particularly those with gastrostomies, tend to be retarded in development and are prone to diarrhoea with failure to thrive, subject to infection, especially in the respiratory tract, middle ear, and urinary tract, or less

commonly, may be obese and unhealthy from 'forced feeding'. Sometimes the care of these infants at home or in 'convalescent institutions' leaves something to be desired, and on occasion it seems that those giving advice to the mother are unaware of the risks and dangers to which these infants are exposed. Several of the infants in this series developed diarrhoea and were readmitted in states of extreme dehydration. Sudden death from unexplained causes seemed unusually common and this could probably have been prevented by more adequate community care (Oakley *et al.*, 1976). Of the late deaths in this series at least 10 (56%) could have been prevented (Table 4).

Overall, at least 23 (29%) of the 79 deaths were preventable. In group A only 1 death was not wholly preventable. In group B all of the early deaths could have been avoided, and at least 30% of the late deaths.

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