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ESOPHAGEAL ATRESIA: RESULTS OF 108 CASES IN AN 11 YEAR PERIOD

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ABSTRACT

From October 1977 to December 1988, 108 neonates born with esophageal atresia (EA) and/or a tracheoesophageal fistula (TEF) were treated at the Bristol Royal Hospital for Sick Children.

An incidence of 1:4000-4500 live births was noted. 82.4% had the common-type anomaly, 5.5% had pure esophageal atresia, and 6.5% had an H-type anomaly. 2.8% had upper and lower fistulae and 2.8% had upper fistulae.

Forty-nine patients (45.3%) had associated anomalies of which the cardiovascular system (16.6%) was the most commom. With consideration of the urogenital system as a unique system, urogenital anomalies were the most common associated anomalies (20.3%). Thirty (27.7%) of 108 neonates had VACTERL associated anomalies, which were more frequent in the common-type anomaly. Seven of 9 deaths in the VACTERL associated group were because of associated anomalies and cardiac anomalies were a common cause of late death in this study. In full-term and well babies with common-type anomalies, transanastomotic tubes significantly decreased hospital stay.

Every effort was made to maintain the neonate's own esophagus, and in pure esophageal atresia 5 of 6 neonates were successfully treated by spontaneous growth and anastomosis and only 1 neonate underwent gastric tube formation as an esophageal replacement. Anastomosis was done in one layer by 5/0 silk , and 17.14% developed leak, 29.62% strictures, 1.90% recurrent fistula, and the rate of anastomotic complications was markedly higher in delayed and staged operations. Fifty-three neonates (50.47%) had respiratory complications which were the most common complication. Thirty-three (31.4%) had gastroesophageal reflux (GER), all but one of which were treated medically. The routine policy now is that all babies are put on Gaviscon until the child can adopt an upright position.

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Low birth weight and pneumonia are not contraindications for surgery, and with the improvement of surgical technique and postoperative care, do not affect survival. There was 12.03% mortality and the main cause of death was associated anomalies.
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INTRODUCTION

Quick development and improvement of the surgical treatment of esophageal atresia in recent decades is said to be "the great triumph of modern paediatric surgery".[1]

According to surgical philosophy: "the best esophagus is the patient's own esophagus".[2] Therefore, the routine policy in this study was to maintain the neonate's own esophagus and a high degree of success attests the good result of this study. However, medical management of gastroesophageal reflux in the first year of life can avoid most early and aggressive surgical therapies of this problem.

MATERIAL AND METHODS

One hundred and eight neonates born with esophageal atresia and/or a tracheoesophageal fistula were admitted to the Bristol Royal Hospital for Sick Children in the 12 years from 1977 to 1988. Surgery for type A, C and E anomalies was primary repair with transanastomotic feeding tube (TAT) or primary repair with gastrostomy and transpyloric feeding tubes (TPT). If there was a long gap, a delayed repair was undertaken. This delayed repair can be further subdivided into two groups:

1) Anastomosis undertaken within seven days of initial gastrostomy and TPT (short delay).

2) Anastomosis undertaken after seven days of initial gastrostomy and TPT (long delay).

If an esophageal anastomosis could not be undertaken in spite of a delayed approach, esophageal replacement using a gastric tube was performed as a staged repair.

RESULTS

Incidence and type of anomaly

The annual incidence of esophageal fistula in the South West Region is 1:4,000-4,500 livebirths with a male to female ratio of 1:1. The anatomical classification with relative incidence is shown in Table I. Three patients (2.8%) had an atresia with a proximal fistula. Only one was diagnosed preoperatively, and the other two were diagnosed as having an atresia without a fistula and the proximal fistula was identified during a delayed repair. Three patients (2.8%) had an upper and lower pouch fistula which were identified during a primary repair in two patients, but in the third patient the upper pouch fistula was diagnosed 40 days after the initial surgery and was repaired via a cervical approach. Seven patients (6.5%) had a fistula without an atresia. All were diagnosed by contrast study between two and ninety days after birth.

Anatomically, there were two atypical cases. One had a double fistula, with the distal fistula connected to the distal esophagus by an atretic segment. The other patient had a common-type anomaly with double distal fistulae, the upper one connecting to a bronchogenic cyst.

Operative treatment

Seventeen neonates who had a type A anomaly underwent ligation of the fistula and end to end anastomosis with TAT feeding. One neonate developed

Table I. Type of anomaly

Type of anomaly	Number	Percentage
E. Atresia* + lower TEF** (Type A)	89	82.4
E. Atresia + upper TEF (Type B)	3	2.8
E. Atresia + upper and lower TEF (Type C)	3	2.8
E. Atresia (Type D)	6	5.5
TEF (Type E)	7	6.5
Total	108	100%

* E. Atresia: Esophageal atresia
** TEF: Tracheoesophageal fistula

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Table II. Long delayed operation.

Type of anomaly	Reason for long delayed repair	Method of treatment	Time of delayed repair	Mortality
A	duodenal atresia	repair of EA+G+TPT+ division and repair of TEF	53 days	-
A	severe RDS	G+TPT+division and repair of TEF	33 days	-
A	RDS	G+TPT+division and repair of TEF	30 days	-
A	long gap	G+TPT+division and repair of TEF	43 days	-
A	long gap	G+TPT+division and repair of TEF	30 days	-
A	long gap	G+TPT+division and repair of TEF	40 days	-
A	RDS+ perforation of bowel	G+TPT of TEF	40 days	-
B*	long gap-upper fistula	G	60 days	-
C	upper fistula+ atretic distal fistula-clinically like type D	G	42 days	-
D	isolated atresia	G+TPT	43 days	-
D	isolated atresia	G	58 days	-
D	isolated atresia	G	50 days	-
D	isolated atresia	G	50 days	-
D*	long gap isolated atresia	G	45 days	-
total 14				0

*In two cases long delayed repair failed and approximation of the two segments was done and later anastomosis was performed.

G: Gastrostomy TEF: Tracheoesophageal fistula TPT: Transpyloric feeding tube EA: Esophageal atresia

a severe stricture due to an inadequate anastomosis and required a reanastomosis. Survival was 100%.

Sixty neonates had a primary repair, gastrostomy, and TPT feeding. Fifty-one had a type A anomaly, 2 a type C (upper and lower pouch fistula) and 7 type E anomaly (tracheoesophageal fistula). Two patients with type A developed a major leak and required a staged repair with a gastric tube. Survival of these patients was 86.6%.

Twenty-four neonates had a gastrostomy, TPT and delayed repair, of whom 17 had a type A anomaly, 1 type B, 1 type C and 5 type D. Ten of these 24 neonates had a delayed repair at less than 7 days of age for a type A anomaly. Six of these ten patients had an associated high anorectal anomaly and 4 had severe pneumonia. There were 2 deaths in this group.

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Table III. Staged operation

Type of anomaly	Reason for staged operation	Type of staged operation	Time of staged operation	Mortality
A	Major leak after primary repair - cervical esophagoscopy	Gastric tube	age 1 year	-
A	Major leak 2 days post-operative cervical esophagoscopy	Gastric tube	age 1 year	-
A	Long gap (fistula in carina) G*+TPT*+C.E.*	Gastric tube	age 10 months	-
B	Long gap, perforation of lower pouch, C.E.	Gastric tube	age 1 year	-
B	Long gap, failed delayed repair, C.E.	Gastric tube	age 1 year	-
D	Long gap, G+C. E.	Gastric tube	age 1 year	-
Total 6				0

*C.E.: Cervical esophagostomy G: Gastrostomy TPT: Transpyloric feeding tube.

Fourteen of the 24 neonates had a delayed repair at greater than 7 days of age. Seven had a type A anomaly, 1 type B, 1 type C and 5 type D. The reason for the long delay in Type A anomaly was a long gap (3), RDS (3), and duodenal atresia (1). There were no deaths in this group (Table II). Six patients had a staged repair using a gastric tube replacement. Two of these patients developed a major anastomotic disruption and the others had a long gap preventing a primary or delayed repair (Table III). Survival in this group was 100%.

Associated anomalies

Forty-nine of the 108 patients (45.3%) had a total of 89 other anomalies. Forty of these patients had type A esophageal atresia. The most common associated anomaly was of the genitourinary system (20.3%), followed by cardiovascular (16.6%) and anorectal (12.0%) malformations (Table IV).

Thirty of the 108 neonates (27.7%) had a VACTERL anomaly. Nine of these patients died, 3 because of severe multiple anomalies without undergoing definitive surgery, 2 following cardiac surgery at the age of 6 months and 5 years, one because of sudden death post primary repair possibly due to a mucous plug causing respiratory arrest. There were 2 late deaths at 6 and 9 months of age due to chronic respiratory failure and sepsis. Patients that had a higher number of individual components of the VACTERL syndrome had a higher mortality. Thus, of the 6 patients with 4 components, there was a 50% mortality rate due mainly to cardiac anomalies. Of 10 patients with 3 components, 3 patients died , again of cardiac anomalies. Of 14 patients with 2 components, only 2 died of other causes. Twenty-six of the 30 patients with VACTERL anomaly had a type A esophageal abnormality (Table V).

Deaths and complications

There were 13 deaths in the 108 patients (12.03%). Three patients had no operation because of severe associated anomalies incompatible with life. Two patients died following cardiac surgery, 2 suffered severe sepsis and 3 early deaths post primary and delayed repair from 6 hours to 7 days of age, one because of severe Fallot's tetralogy and cardiac and respiratory failure and the other two because of respiratory arrest due to an early apneal attack and mucous plug.

Three late deaths occurred from 6 months to 2.7 years of age; one because of respiratory failure following severe RDS and apnea following prolonged ventilatory support, one because of sudden death due to an

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Table IV. Analysis of associated anomalies.

Type of anomaly	No. and % of anomaly	Type of anomaly	No. and % of anomaly
Vertebral:	9(8.3%)	**Limb:**	3(2.7%)
sacral defect	5	Extra digit	2
sacral defect+ 6 T hemivertebrae	1	Talipes equinovarus	1
		CNS:	4(3.7%)
Scoliosis + 3 T hemivertebrae	1	Aquaduct stenosis	1
3 thoracic hemivertebrae	1	Mild communicating hydrocephalus	1
		Erb's palsy	1
Congenital scoliosis	1	Facial palsy	1
Anorectal:	13(12.0%)	**GI excluding anorectal:**	3(2.7%)
High type	9	Duodenal atresia	1
Low type	4	Duodenal atresia + malrotation	1
Cardiovascular:	18(16.6%)	Antenatal ileal perforation+ meconium peritonitis	1
VSD	3	**Genitalia:**	13(12.0%)
PDA	3	Hypospadias	6
Fallot's tetralogy	2	Hypospadias + undescended testis	2
F.T.+Rt side aortic arch	1	Ambiguous genitalia-severe hypospadias	1
VSD + ASD	1	Unilateral undescended tests	3
VSD + PDA	1	Bilateral undescended testes + partial atretic urethra	1
Incomplete A-V canal	1	**Genetic:**	1(0.9%)
Complex cardiac anomaly	1	Down's syndrome	1
Type I truncus arteriosus	1	**Face:**	6(5.5%)
Double umbilical vein	1	Pre-auricular tag + low set ears	3
Insignificant systolic murmur	3	Unilateral mouth cleft + skin tag (Goldenhar's syndrome)	1
Renal:	9(8.3%)	Cleft palate	1
Rt. single kidney	1	Lt. choanal atresia	1
Lt. single kidney	1	**Others:**	10(9.2%)
Lt. dysplastic kidney	1	Pulmonary hypoplasia	1
Rt. hypoplastic kidney + megaureter	1	Bronchogenic cyst	1
Rt. duplex system	1	Subglottic stenosis	1
Bilateral reflux + Rt. hydronephrosis	1	Potter's syndrome	1
Bilateral hydronephrosis	2	Bilateral inguinal hernia	4
Rt. hydronephrosis	1	Umbilical and epigastric hernia	1
		Weakness of vocal cords	1

apneal attack one year after aortopexy for tracheomalacia (this patient had mild reflux) and one had sudden death following choking and developing a late apneal attack (this patient had severe gastro-esophageal reflux). The survival according to the Waterston classification is shown in Table VI. It should be noted that group C consists of not just low birthweight infants but also those with severe associated anomalies. However, survival when related purely to birthweight shows that low birth weight in itself is not a poor prognostic indicator. Thus, of 8 neonates who weighed less than 1500 grams, one died of severe sepsis and the second death was because of an associated cardiac abnormality. There was therefore only a 14% mortality in the 7 patients who were well without a major cardiac anomaly and were of low birth weight.

Reflux

Studious attempts to document reflux in this group were not performed, but all patients had a routine

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Table V. VACTERL-associated anomalies

No. of systems affected	Type of esophageal anomaly A,B,C,D,E	Waterston risk group A,B1,B2,C1,C2	Total No.& Percentage	Non VACTERL system involved	Mortality No. &%
4 systems involved	6,-,-,-,-	-,-,-,-,6	6(5.5%)	3	4(66.6%)
3 systems involved	7,-,-,2,1	-,-,5,1,4	10(9.5%)	5	3(30%)
2 systems involved	13,-,-,-,1	2,3,4,-,5	14(12.9%)	5	2(14.2%)
Total	26,-,-,2,2	2,3,9,1,15	30(27.9%)	13	10(20.4%)

Table VI. Waterston risk group classification

Risk group	Number		Percent	Survival		Percent	
A	44		40.74%	43		99.72%	
B1	23	41	21.29%	21	38	91.30%	92.68%
B2	18		16.66%	17		94.44%	
C1	7	23	6.48%	5	14	71.42%	60.86%
C2	16		14.81%	9		56.25%	
Total	108			95		87.96%	

Table VII. Patients with GER*

No. of patients 105 (%)	Pneumonia (%)	Apneal attack(%)	Stricture (%)	Leaks (%)	Failure to thrive
GER 33(31.4)	18 (54.5)	11 (33.3)	13 (39.3)	7 (21.2)	6 (18.1)
Non-refluxing 72 (68.5)	21 (29.1)	14 (19.4)	19 (26.3)	11 (15.2)	6 (8.3)

*G.E.R.: Gastroesophageal reflux

postoperative barium study at approximately 7 days after repair of the atresia. This study documented reflux in 33 of the 105 patients. Although it is highly likely that the vast majority of patients with esophageal atresia have gastroesophageal reflux, the documentation of reflux in these 33 patients showed a significant morbidity rate (Tables VII and VIII). There were two deaths in refluxing patients, one because of apnea one year following aortopexy, and the other because of sudden death following choking and respiratory arrest.

Leaks and strictures

18 patients (17.1%) developed anastomotic leak. Eleven minor leaks following primary repair were treated conservatively and healed spontaneously within two weeks. A further 4 leaks occurred after a

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Table VIII. Number of admissions in patients with and without G.E.R.*

No. of patients	One admission(%)	2 - 5 admissions (%)	>5 admissions (%)
33 patients with G.E.R.	13 (39.3)	11 (33.3)	9 (27.2)
72 patients without G.E.R.	42 (58.3)	20 (27.7)	7 (9.9)

*G.E.R.: Gastroesophageal reflux

Table IX. Operative complications

Type of complication	Type of anomaly	Technique of operation			Total number (%)
		primary(%)	delayed(%)	staged(%)	
Leak	A(13) B(2) D(3)	10(12)	4(18)	4(50)	18(17.14)
Stricture	A(23) B(3) D(6)	16(20.7)	11(50)	5(62.5)	32(29.62)
Recurrent fistula	A(1) C(1)	1(1.29)	1(4.54)	-	2(1.90)

staged repair and again healed but took up to 8 weeks to close. One patient leaked following esophagoscopy and dilatation and was treated by re-anastomosis. Two patients developed a major disruption of the anastomosis following primary repair of a type A anomaly and had an esophageal replacement performed. Eleven of these 18 patients developed stricture which was treated by dilatation in 7 and re-anastomosis in 4.

Stricture

Thirty-two patients developed a stricture (29.6%) of which 20 were early strictures, occurring within 3 months of operation time, and 12 were late strictures. Sixteen of 20 strictures were treated by dilatation and 4 required re-anastomosis. Ten of 12 late strictures were treated by dilatation and only 2 of them required refashioning of anastomosis. The last two were strictures of proximal gastric tube anastomosis (Tables IXand X).

Recurrent fistula

Two patients developed a recurrent fistula. One followed staged repair of a type A anomaly and was diagnosed 6.5 months after a delayed repair. The other occurred in a type C anomaly probably from the upper fistula and was repaired via a cervical approach 2 months after primary repair.

DISCUSSION

Transanastomotic tube versus gastrostomy and transpyloric feeding tube

Many surgeons advocate the use of either a gastrostomy alone or with a transpyloric feeding tube in the management of babies undergoing a primary repair of an esophageal atresia and/or tracheoesophageal fistula.[2,6,7,8,9,10] All surgeons use a gastrostomy and transpyloric feeding tube in those patients managed by a delayed or staged repair, or in the management of those patients having a major complication of a primary repair such as severe anastomotic leak, stenosis, or a recurrent fistula. Some authors have suggested that a TAT may lead to a leak or stricture formation as it acts as an irritant foreign body.[6]

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Table X. Complications in operated neonates

Complication	No.	Percent
No complication	36	34.3%
Total complications	69	65.7%
Early complications:		
Anastomotic leak	18	
Anastomotic stricture	20	
Recurrent fistula	1	
*Pneumothorax	12	
*Recurrent pneumonia	20	
*Apneal attack	20	
Late complications:		
Dysphagia	24	
Failure to thrive	11	
Foreign body obstruction	9	
Deafness	6	
Post-operative scoliosis	4	
Stricture	12	
Recurrent fistula	1	
*Apneal attack	8	
*Recurrent pneumonia	19	

* The total number of early and late respiratory complications was 53.

but a recent experimental study has shown that a TAT has no effect on the anastomosis[5] and many surgeons have recently felt that a gastrostomy is unnecessary in the management of those neonates who are otherwise well and have undergone a primary repair of an esophageal atresia.[3,4,9,10] Some authors believe that the gastrostomy not only fails to protect the anastomosis, but also increases the incidence of gastroesophageal reflux.[4] In our experience, there was no increased incidence of gastroesophageal reflux (GER) in the gastrostomy group (Table XI), and there was no increased incidence in complications, but there was a significant decrease in hospital stay in the TAT group (Table XI). We must stress, however, that only those patients who were mature, of good birth weight, and otherwise well were managed without gastrostomy and transpyloric feeding tube.

Anastomotic technique and complications

One layer of full-thickness anastomosis by silk or other non-absorbable sutures has been accepted by many surgeons as a quick and simple method of anastomosis.[3,4,7,8,9,10,11,12] The effect of silk acting as a foreign body and leading to anastomotic complications has been stressed by some authors both experimentally and clinically.[4,15,10] In a comparative study between silk, dexon, and prolene, it was suggested that silk had three times the risk of anastomotic complications over the other suture materials.[4] We have found one layer end to end anastomosis simple and easy to carry out and have preferred silk for suture material because it is easy to handle and has a good tensile strength.[5] Anastomotic complications in this study in patients undergoing primary repair were leakage (12%), stricture (20%), and recurrent fistula (1.29%) (Table IX). The incidence of anastomotic complications in the delayed and staged groups was significantly higher than in those patients undergoing primary repair. We believe the incidence of anastomotic complications was not related to the suture material, but, as many other authors have suggested, depend upon technical causes,[4] vascularity of the distal segment,[6,3] and the distance between the two segments.[6] There is no doubt that accurate mucosal apposition is an important factor in obtaining uncomplicated anastomotic healing.

Gastroesophageal reflux and respiratory complications

Gastroesophageal reflux (GER) is common in ba-

Table XI. Hospital stay in TAT and gastrostomy + TPT groups

Type of anomaly & No. of patients	Methods of treatment	G.E.R.	Hospital sta				
			<2 Weeks	2-3 weeks	3-4 weeks	4-8 weeks	>8 weeks
Type A 16	Primary repair + TAT	4(25%)	7(43.7%)	7(43.7%)	2(12.6%)	0	0
Type A 28	Primary repair+ gastrostomy+ TPT	8(28.5%)	0	15(53.5%)	6(21.4%)	7(19.4%)	0

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bies with esophageal atresia and tracheo-esophageal fistula.[8,9,14,16] The incidence of reflux has been quoted as being from 24%[9] to 82%.[16] In this study 33 patients (31.4%) had gastroesophageal reflux diagnosed by a barium study. The exact cause of GER in EA and TEF is not clear[15] but the possibilities include congenital abnormality of esophageal nerve supply and motility, surgical damage to the vagus nerve and thus esophageal motility, and shortening of the esophagus and straightening of the gastroesophageal angle.[9,13,14,16,19,20] The symptoms of GER are the same as for those patients without an esophageal atresia, namely cough, apnea, recurrent pneumonia, failure to thrive, and recurrent anastomotic or distal esophageal stricture formation. In those patients with EA and TEF, other causes for these symptoms include an anastomotic stricture, a recurrent fistula, dismotility of the esophagus,[18] and tracheomalacia associated with compression of the dilated upper pouch posteriorly and vascular structures anteriorly.[4,21,22,23] The main cause for recurrent pneumonia and bronchitis in patients with EA and TEF has been shown to be disturbed motility of the esophagus, which is present in all cases.[17,18] We believe that because of this poor motility the symptoms of reflux in babies with an EA and TEF are more severe and treatment of the reflux both medically and surgically is less successful in this group. Treatment for reflux does nothing for the esophageal dismotility and intraesophageal reflux. LeSouef et al.[17] have shown increased lower esophageal sphincter pressures following surgery leading to increased respiratory complications. Despite this, aggressive surgical treatment of gastroesophageal reflux in patients with EA and TEF has been advocated by many authors.[4,9,12,14,15,16] In our series we had 33 neonates with GER (31.4%) of which all but one were treated medically and only one patient required operative treatment for GER because of failure to thrive and an associated esophageal stricture. We believe that GER is more common in babies with EA and TEF than has previously been reported and that almost all neonates after repair of an esophageal atresia have some degree of gastroesophageal reflux with associated symptoms, particularly respiratory, which are severe but do not necessarily require aggressive surgical treatment. In our study most of those patients with recurrent pneumonia and apneic attacks required admission in the first year of life. Six of nine patients requiring more than five admissions presented in the first year of life. Three were between the ages of two and five and only one of these last three had an admission after the age of five. It is now our policy to put all neonates on Gaviscon after repair of an esophageal atresia and tracheoesophageal fistula up to an age when they can adopt an upright position. If they develop apneic attacks the food is also thickened and positional treatment is started. In the future the evaluation of this conservative approach in comparison with other methods will result in the correct policy being determined.

Management of the long gap

The vast majority of neonates with an esophageal atresia can be successfully treated by a primary esophageal repair. In those patients with a wide gap many different techniques to enable a repair to be undertaken have been used in the past.[7] When this method of management fails, esophageal replacement using the colon[24,28] gastric tube[25] or gastric transposition[26,27] will be required. In this series the treatment of choice for patients with a long gap has been to allow spontaneous growth of the esophageal segments to occur leading to an anastomosis of the two esophageal segments at a later date. If this fails or there has been a major disruption following a primary repair a gastric tube has been carried out. We believe as do many other authors that the best esophagus is the patient's own esophagus,[2,3,7] and our experience shows that there is significant spontaneous growth of the two segments in the first six weeks to two months of life. After this period there is minimal differential growth between the vertical column and the intrathoracic viscera and little further length is ever achieved. This spontaneous growth is enhanced by using bolus feeding with air injected via a gastrostomy in those patients with a pure esophageal atresia to encourage reflux into the lower pouch and subsequent growth. Before operation in order to achieve an esophageal anastomosis in patients with pure atresia, mobilisation of the gastroesophageal junction is first performed via a laparotomy 48 hours prior to the subsequent thoracotomy to undertake the repair. Of six patients with pure esophageal atresia, five successfully underwent a delayed primary anastomosis by this method. Only one patient who had cloacal exstrophy required esophageal replacement via a gastric tube. In patients undergoing a long delayed and/or staged repair there was a 100% survival rate, but the incidence of anastomotic complications was significantly higher in the delayed and staged groups than in those undergoing a primary repair (Table IX).

Mortality and Waterston risk grouping

Waterston reported his risk factor classification in 1962[30] which has historically been an excellent method to compare the results of management of dif-

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ferent centers in the survival of high risk neonates with esophageal atresia and tracheoesophageal fistula. Survival among these high risk groups has improved recently because of better respiratory care, earlier referral, modern pediatric anesthesia, etc. Historically, therefore, the Waterston classification probably requires revision because associated anomalies are now the main cause of death in patients with esophageal atresia rather than just a low birth weight and pneumonia.[1,2,3,4,10,11,31,32] In this series, there was a 99.72% survival in risk group A, 92.7% in risk group B and 60.7% in risk group C. All babies in risk group C in this series had associated anomalies as well as low birth weight (Table VI).

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