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Esophageal Atresia / Tracheoesophageal Fistula Overview – RETIRED CHAPTER, FOR HISTORICAL REFERENCE ONLY

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Summary

NOTE: THIS PUBLICATION HAS BEEN RETIRED. THIS ARCHIVAL VERSION IS FOR HISTORICAL REFERENCE ONLY, AND THE INFORMATION MAY BE OUT OF DATE.

The purpose of this overview is to increase the awareness of clinicians regarding esophageal atresia / tracheoesophageal fistula and its genetic causes and management.

The following are the goals of this overview.

Goal 1

Describe the clinical characteristics of esophageal atresia / tracheoesophageal fistula.

Goal 2

Review the genetic causes of esophageal atresia / tracheoesophageal fistula.

Goal 3

Provide an evaluation strategy to identify the genetic cause of esophageal atresia / tracheoesophageal fistula in a proband (when possible).

Goal 4

Inform genetic counseling of family members of an individual with esophageal atresia / tracheoesophageal fistula.

Goal 5

Review management of esophageal atresia / tracheoesophageal fistula.

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1. Clinical Characteristics of EA/TEF

Clinical Description

Esophageal atresia (EA) is a developmental defect of the upper gastrointestinal tract in which the continuity between the upper and lower esophagus is lost. EA can occur with or without tracheoesophageal fistula (TEF), an abnormal connection between the trachea and the esophagus.

Infants with congenital forms of EA/TEF usually present shortly after birth with copious oral secretions, coughing, gagging, cyanosis, vomiting, and/or respiratory distress.

The following five EA/TEF configurations have been described [Clark 1999] (see Figure 1):

- EA with a distal TEF (the most common; 84%)
- Isolated EA (i.e., without TEF) (8%)
- H-type TEF with no EA (4%)
- EA with proximal and distal TEF (3%)
- EA with a proximal TEF (1%)

A proband with EA/TEF can present in one of the following clinical settings:

- **Isolated.** Not associated with other congenital anomalies (40% of probands)
- **Non-isolated.** Associated with other congenital anomalies (60% of probands):
 - **Syndromic.** In a subset of non-isolated EA/TEF cases, a specific genetic diagnosis can be made based on the constellation of other congenital anomalies and/or molecular testing (~10% of probands; see Genetic Causes of EA/TEF) [Brosens et al 2014b].
 - **VACTERL association** (~10% of probands), the non-random association of vertebral, *a*nal, cardiac, tracheoesophageal fistula, renal, and limb defects that occur together more often than would be expected by chance, but are not known to be caused by a unifying genetic defect. Infants with EA/TEF and at least two of the other defects included in the VACTERL acronym can be said to have VACTERL association if a specific genetic diagnosis cannot be reached. Note: Maternal diabetes mellitus has been suggested as a possible risk factor for development of VACTERL association [Castori et al 2008].

Establishing the Diagnosis of EA/TEF

EA/TEF may be suspected prenatally if ultrasound examination reveals polyhydramnios, absence of a fluid-filled stomach, a small abdomen, lower-than-expected fetal weight, and a distended esophageal pouch [Kronemer 2015]. Fetal MRI may be used to confirm the presence of EA/TEF.

EA may be detected postnatally by:

- Failure to pass a nasogastric (NG) tube and radiographs that demonstrate coiling of the NG tube in the pouch;
- Tracheal compression and deviation on plain chest radiographs;
- Absence of a gastric bubble on plain radiographs, which may suggest EA without a TEF or EA with a proximal TEF;
- Three-dimensional chest CT examination [Kronemer 2015].

Note: Administration of barium into the esophagus followed by chest radiographs can confirm the diagnosis but is seldom required.

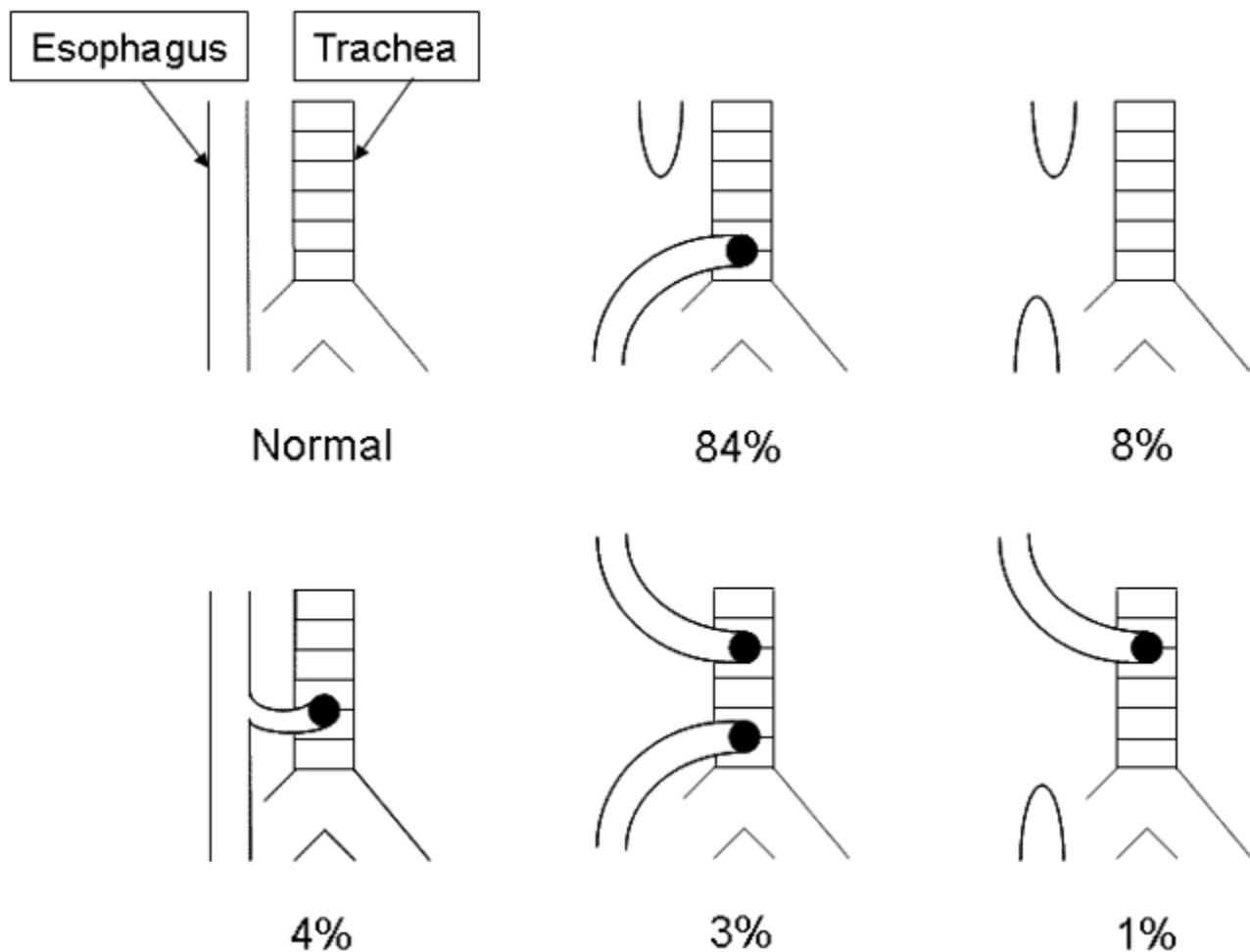


Figure 1. EA/TEF configurations and their frequencies

Differential Diagnosis of EA/TEF

Laryngotracheoesophageal cleft. Midline defect between the posterior larynx and trachea and the anterior wall of the esophagus. This entity can present prenatally with polyhydramnios and a small or absent stomach bubble and postnatally with aspiration after feeding. Laryngotracheoesophageal clefts are commonly associated with other anomalies including TEF.

Esophageal webs/rings. Circumferential partial obstruction of the esophageal lumen caused by membranous or diaphragmatic tissue that can be associated with TEF. Many cases are asymptomatic. Symptomatic esophageal webs/rings typically present with recurrent vomiting, dysphagia (solid > liquids), and sometimes aspiration. These symptoms tend to occur later in life than those caused by EA.

Esophageal stricture. A narrowing of the esophageal lumen caused by a variety of intrinsic and extrinsic disease processes. Congenital esophageal strictures typically present after the newborn period in a manner similar to that seen in esophageal webs/rings.

Esophageal diverticulum. A sack or pouch arising from the esophagus. Esophageal diverticula can be present at birth but most often arise and/or become symptomatic in adulthood, presenting with a history of dysphagia, chest pain, vomiting, and sometimes aspiration pneumonia.

Tubular esophageal duplications. Tubular channels that lie parallel to the esophagus and often connect to the main esophageal lumen or the stomach. These lesions are often asymptomatic and are most commonly identified serendipitously at autopsy. Symptoms typically result from inflammation and/or distention secondary to food entrapment and typically involve intermittent dysphagia.

Congenital short esophagus. An abnormally short esophagus accompanied by an intrathoracic location of part of the stomach. Symptoms are often present at birth and include gastroesophageal reflux and vomiting.

Tracheal agenesis/atresia. Lack of communication between the larynx and the alveoli of the lungs. This can be associated with other anomalies including TEF. Prenatal findings can include hyperechoic lungs, flattened diaphragms, oligo- or polyhydramnios, and large breathing movements. Postnatal symptoms include severe respiratory distress, cyanosis, absent cry, and failure to ventilate despite tracheal intubation.

2. Genetic Causes of EA/TEF

The genetic causes of esophageal atresia / tracheoesophageal fistula (EA/TEF) include syndromes that result from mutation of a single gene (see Table 1) or chromosome anomalies.

Table 1. Esophageal Atresia / Tracheoesophageal Fistula: Syndromic Causes and Distinguishing Clinical Features

Syndrome ¹	Gene	MOI	Distinguishing Clinical Findings
Anophthalmia-esophageal-genital syndrome	SOX2	AR	<ul style="list-style-type: none"> • Anophthalmia/microphthalmia • EA/TEF • Urogenital anomalies
CHARGE syndrome	CHD7	AR	<ul style="list-style-type: none"> • Coloboma of the eye • Cardiac anomalies • Choanal atresia • Intellectual disability • Growth restriction • Genital anomalies • Ear anomalies • Hearing loss • EA/TEF
Feingold syndrome	MYCN	AR	<ul style="list-style-type: none"> • Esophageal & duodenal atresias • Microcephaly • Learning disabilities • Syndactyly • Cardiac defects
Fanconi anemia	≥20 genes	AR AD	<ul style="list-style-type: none"> • Bone marrow failure • Malignancies • Short stature • Abnormal skin pigmentation • Radial ray defects • Eye anomalies • Renal anomalies • Cardiac defects • Abnormal ears • Central nervous system anomalies • Hearing loss • Developmental delay • Gastrointestinal anomalies incl EA/TEF

Table 1. continued from previous page.

Syndrome ¹	Gene	MOI	Distinguishing Clinical Findings
VACTERL-H (OMIM 300515)	<i>FANCB</i>	XL	<ul style="list-style-type: none"> • Vertebral anomalies • Anal atresia • Cardiac malformations • Tracheoesophageal fistula • Renal anomalies • Limb anomalies • Hydrocephalus

1. EA/TEF has also been reported in individuals with Opitz G/BBB and Pallister-Hall syndrome, although it is not a major feature in these disorders.

Chromosomal Causes of EA/TEF

Chromosome anomalies have been reported in approximately 6%-10% of individuals with EA/TEF. EA/TEF is found in the following aneuploidy syndromes [Felix et al 2007, Brosens et al 2014a]:

- Trisomy 21 (~0.5%-1.0% of affected individuals)
- Trisomy 18 (~25% of affected individuals)
- Trisomy 13
- Trisomy X

Copy number variant (CNV) analysis in individuals with both isolated and non-isolated EA/TEF can reveal rare gene-containing CNVs, a subset of which can be shown to have arisen *de novo* [Brosens et al 2014b].

Recurrent duplications of 3p25-pter and 5q34-qter suggest that these regions may harbor one or more genes in which overexpression causes or predisposes to the development of EA/TEF [Felix et al 2007].

Recurrent deletions of 2q37.2-qter, 4q35-qter, 5p15-pter, 6q13-q15, 14q32.3-qter, and 17q22-q23 suggest that haploinsufficiency or decreased expression of one or more genes in these regions may cause or predispose to the development of EA/TEF [Felix et al 2007].

Recurrent deletions of 13q34-qter and 22q11 are also associated with EA/TEF, but typically with findings consistent with VACTERL association [Walsh et al 2001, Felix et al 2007].

3. Evaluation Strategies to Identify the Genetic Cause of EA/TEF in a Proband

Establishing a specific genetic cause of EA/TEF:

- Can aid in discussions of prognosis (which are beyond the scope of this *GeneReview*) and genetic counseling;
- Usually involves a medical history, physical examination, laboratory testing, family history, and genomic/genetic testing.

Medical history. A history of neurodevelopmental defects, other structural birth defects, abnormal growth patterns, bone marrow failure, malignancies, and hearing loss may provide evidence in support of a specific cause of EA/TEF (see Table 1).

Physical examination. The identification of other structural birth defects (e.g., anal anomalies, limb defect) and/or abnormal growth patterns may provide evidence in support of a specific cause of EA/TEF (see Table 1).

Family history. A three-generation family history should be taken, with attention to relatives with manifestations of EA/TEF and documentation of relevant findings through direct examination or review of medical records, including results of molecular genetic testing.

Imaging studies may identify additional anomalies of the skeleton, heart, and/or kidneys.

Cytogenetic testing. If aneuploidy is suspected in an individual with EA/TEF, a G-banded karyotype can be performed (see Chromosomal Causes of EA/TEF).

Molecular genetic testing approaches can include a combination of **gene-targeted testing** (single-gene testing or multigene panel), **chromosomal microarray analysis** [CMA], and **comprehensive genomic testing** (exome sequencing, genome sequencing, or exome array). Gene-targeted testing requires the clinician to hypothesize which gene(s) are likely involved, whereas genomic testing does not.

- **Serial single-gene testing** can be considered if clinical findings and/or family history indicate that pathogenic variants in a particular gene are most likely causative (see Table 1).
- **A multigene panel** that includes some or all of the genes listed in the Table 1 is most likely to identify the genetic cause of the condition while limiting identification of variants of uncertain significance and pathogenic variants in genes that do not explain the underlying phenotype. Note: (1) The genes included in the panel and the diagnostic sensitivity of the testing used for each gene vary by laboratory and are likely to change over time. (2) Some multigene panels may include genes not associated with the condition discussed in this *GeneReview*. (3) In some laboratories, panel options may include a custom laboratory-designed panel and/or custom phenotype-focused exome analysis that includes genes specified by the clinician. (4) Methods used in a panel may include sequence analysis, deletion/duplication analysis, and/or other non-sequencing-based tests. For this disorder a multigene panel that also includes deletion/duplication analysis is recommended (see Table 1).

For an introduction to multigene panels click [here](#). More detailed information for clinicians ordering genetic tests can be found [here](#).

- **Chromosomal microarray analysis (CMA)** using oligonucleotide or SNP arrays to detect genome-wide large deletions/duplications that cannot be detected by sequence analysis may be considered in individuals with distinguishing phenotypic features suggestive of a contiguous gene deletion (see Chromosomal Causes of EA/TEF).
- **Comprehensive genomic testing** (which does not require the clinician to determine which gene[s] are likely involved) may be considered. **Exome sequencing** is most commonly used; **genome sequencing** is also possible.

Exome array (when clinically available) may be considered if exome sequencing is not diagnostic.

For an introduction to comprehensive genomic testing click [here](#). More detailed information for clinicians ordering genomic testing can be found [here](#).

4. Genetic Counseling

Genetic counseling is the process of providing individuals and families with information on the nature, mode(s) of inheritance, and implications of genetic disorders to help them make informed medical and personal decisions. The following section deals with genetic risk assessment and the use of family history and genetic testing to clarify genetic status for family members; it is not meant to address all personal, cultural, or ethical issues that may arise or to substitute for consultation with a genetics professional. —ED.

Mode of Inheritance

Esophageal atresia / tracheoesophageal fistula (EA/TEF) can occur as an isolated finding, as part of a genetic syndrome, or as part of a non-isolated (but not syndromic) set of findings. Most individuals with EA/TEF represent simplex cases (i.e., the only affected member of the family). A small subset of families are multiplex (i.e., ≥ 2 relatives have EA/TEF).

Nonsyndromic EA/TEF is generally considered to be inherited in a multifactorial manner.

If an affected individual is found to have an inherited or *de novo* chromosome abnormality, or a specific syndrome or association with EA/TEF, genetic counseling for that condition is indicated.

Risk to Family Members

Table 2. Risk to Family Members

EA/TEF Classification	Recurrence Risk	
	To sibs of a proband	To offspring of a proband
Isolated w/o a clear etiology ¹	Empiric risk: 1% ²	~2%-4% ^{2, 3, 4}
Non-isolated & part of a known genetic syndrome	Depends on: <ul style="list-style-type: none"> • MOI assoc'd w/the genetic syndrome; & • Genetic status of proband's parents 	Depends on MOI assoc'd w/the genetic syndrome
Non-isolated & not part of a known genetic syndrome ⁵	<ul style="list-style-type: none"> • In most families risk is likely low. ⁶ • In the minority of families risk is higher (~25%-50%). ⁶ 	~2%-4% ^{2, 3}

EA/TEF = esophageal atresia / tracheoesophageal fistula; MOI = mode of inheritance

1. Nonsyndromic EA/TEF is generally considered to be inherited in a multifactorial manner. The twin concordance rate for EA/TEF is also low (~2.5%) [Robert et al 1993].

2. Shaw-Smith [2006]

3. Recurrence risk for EA/TEF and/or malformations in the VACTERL association.

4. Since these estimates were generated by studying the offspring of individuals with both isolated and non-isolated EA/TEF, the risk to offspring in isolated cases may be lower [Shaw-Smith 2006].

5. Recurrence risk counseling for individuals with non-isolated EA/TEF – in whom additional anomalies have been identified and for whom a specific genetic disorder is not recognized – is problematic:

- Some cases of non-isolated EA/TEF are probably caused by *de novo* autosomal dominant mutations, and therefore, pose a low recurrence risk to the sibs of the proband.
- Some are probably unrecognized or private autosomal recessive conditions.
- Some may be multifactorial disorders with a low recurrence risk.
- Non-genetic causes including stochastic events, epigenetic modifications, or teratogenic/environmental exposures are possible as well.

6. Counseling in this setting should be as for other multiple congenital anomaly disorders of unknown etiology.

Related Genetic Counseling Issues

DNA banking. Because it is likely that testing methodology and our understanding of genes, pathogenic mechanisms, and diseases will improve in the future, consideration should be given to banking DNA from probands in whom a molecular diagnosis has not been confirmed (i.e., the causative pathogenic mechanism is unknown). For more information, see Huang et al [2022].

Prenatal Testing and Preimplantation Genetic Testing

Molecular genetic testing. If the pathogenic variant(s) have been identified in an affected family member, prenatal testing for a pregnancy at increased risk and preimplantation genetic testing are possible.

Ultrasound examination

- Prenatal ultrasound examination of a fetus affected by EA/TEF may reveal an absent or small fetal stomach bubble in combination with maternal polyhydramnios [Houben & Curry 2008]. The diagnostic accuracy is increased if an anechoic area is present in the middle of the fetal neck, as it can help differentiate EA from diseases with possible swallowing impairments [Kronemer 2015]. In one study of 87 fetuses with a small (n = 53) or absent (n = 34) stomach bubble, the positive predictive value of an absent stomach bubble and polyhydramnios was 56% and the sensitivity of prenatal sonography in the diagnosis of EA was 42% based on the outcome of all fetuses scanned during the same period [Stringer et al 1995]. The positive predictive value is likely to be even lower among fetuses with multiple anomalies [Choudhry et al 2007].
- The presence of a dilated blind-ending esophageal pouch on a sonogram is suggestive of EA. It is the most reliable sonographic sign indicative of EA and has been seen in cases with and without TEF [Kronemer 2015].
- Polyhydramnios can be seen in a wide variety of disorders and alone is a poor indicator of EA: only one in 12 fetuses with polyhydramnios has EA [Kronemer 2015].
- When EA/TEF is suspected on routine prenatal ultrasound examination:
 - A high-resolution ultrasound examination is indicated;
 - Fetal MRI should be considered to help confirm the diagnosis of EA/TEF and to evaluate for additional structural anomalies;
 - Chromosome analysis, an array-based copy number detection assay, and/or molecular testing using fetal cells/DNA should be considered.
- All fetuses suspected of having EA/TEF should be evaluated for the presence of additional major malformations that could be part of an underlying syndrome because such malformations and/or the underlying diagnosis may significantly affect the prognosis. Involvement of a clinical geneticist in the evaluation of these families can be helpful.

Resources

GeneReviews staff has selected the following disease-specific and/or umbrella support organizations and/or registries for the benefit of individuals with this disorder and their families. GeneReviews is not responsible for the information provided by other organizations. For information on selection criteria, click [here](#).

- **National Library of Medicine Genetics Home Reference**
[Esophageal atresia/tracheoesophageal fistula](#)
- **TOFS**
St. George's Centre
91 Victoria Road
Netherfield Nottingham NG4 2NN
United Kingdom
Phone: +44 (0)115 961 3092
Fax: +44 (0)115 961 3097
Email: info@tofs.org.uk
www.tofs.org.uk
- **Medline Plus**
[Esophageal atresia](#)

5. Management of EA/TEF

Evaluations Following Initial Diagnosis

After an individual has been diagnosed with esophageal atresia / tracheoesophageal fistula (EA/TEF), the following evaluations should be considered as a means of identifying associated anomalies:

- X-ray evaluation for vertebral anomalies
- Echocardiogram
- Renal ultrasound examination

Treatment of Manifestations

Initial postnatal intervention, aimed at minimizing the risk of aspiration pneumonia, typically includes the elimination of oral feeds, placement of a suction catheter to allow continuous drainage of secretions, and elevation of the head of the bed to minimize reflux [Clark 1999]. Intravenous (IV) glucose and fluids should be provided; supplemental oxygen should be administered as needed. When intubation cannot be avoided, a possible complication is the collection of air in the stomach, which in severe cases can be removed only by gastrostomy.

Surgical repair consists of closure of the TEF and anastomosis of the esophageal segments [Sharma 2017]. Surgical repair may need to be delayed in infants with low birth weight, pneumonia, or other major congenital anomalies. When surgical repair is delayed, infants may be treated with parenteral nutrition, gastrostomy tube placement, and upper pouch suctioning until they become surgical candidates.

The most common complications after surgical repair include leakage at the site of the anastomosis, recurrent fistula, structure formation, and gastroesophageal reflux [Clark 1999, Kronemer 2015, Sharma 2017].

Evaluation of Relatives at Risk

See Genetic Counseling for issues related to testing of at-risk relatives for genetic counseling purposes.

Therapies Under Investigation

Search [ClinicalTrials.gov](https://clinicaltrials.gov) in the US and [EU Clinical Trials Register](https://european-clinical-trials-register.eu) in Europe for information on clinical studies for a wide range of diseases and conditions. Note: There may not be clinical trials for this disorder.

Chapter Notes

Author Notes

Baylor College of Medicine Molecular and Human Genetics [web page](#)

Revision History

- 20 April 2023 (ma) Chapter retired: chapter does not reflect current use of genetic testing
- 20 September 2018 (sw) Comprehensive update posted live
- 12 June 2014 (me) Comprehensive update posted live
- 12 March 2009 (me) Review posted live
- 14 October 2008 (das) Original submission

References

Literature Cited

- Brosens E, de Jong EM, Barakat TS, Eussen BH, D'Haene B, De Baere E, Verdin H, Poddighe PJ, Galjaard RJ, Gribnau J, Brooks AS, Tibboel D, de Klein A. Structural and numerical changes of chromosome X in patients with esophageal atresia. *Eur J Hum Genet.* 2014a;22:1077–84. PubMed PMID: 24398799.
- Brosens E, Ploeg M, van Bever Y, Koopmans AE, Ijsselstijn H, Rottier RJ, Wijnen R, Tibboel D, de Klein A. Clinical and etiological heterogeneity in patients with tracheo-esophageal malformations and associated anomalies. *Eur J MED Genet.* 2014b;57:440–52. PubMed PMID: 24931924.
- Castori M, Rinaldi R, Capocaccia P, Roggini M, Grammatico P. VACTERL association and maternal diabetes: a possible causal relationship? *Birth Defects Res A Clin Mol Teratol.* 2008;82:169–72. PubMed PMID: 18181216.
- Choudhry M, Boyd PA, Chamberlain PF, Lakhoo K. Prenatal diagnosis of tracheo-oesophageal fistula and oesophageal atresia. *Prenat Diagn.* 2007;27:608–10. PubMed PMID: 17457956.
- Clark DC. Esophageal atresia and tracheoesophageal fistula. *Am Fam Physician.* 1999;59:910–6, 919-20. PubMed PMID: 10068713.
- Felix JF, Tibboel D, de Klein A. Chromosomal anomalies in the aetiology of oesophageal atresia and tracheo-oesophageal fistula. *Eur J Med Genet.* 2007;50:163–75. PubMed PMID: 17336605.
- Houben CH, Curry JI. Current status of prenatal diagnosis, operative management and outcome of esophageal atresia/tracheo-esophageal fistula. *Prenat Diagn.* 2008;28:667–75. PubMed PMID: 18302317.
- Huang SJ, Amendola LM, Sternen DL. Variation among DNA banking consent forms: points for clinicians to bank on. *J Community Genet.* 2022;13:389–97. PubMed PMID: 35834113.
- Kronemer KA. Imaging in esophageal atresia and tracheoesophageal fistula. Medscape. Available [online](#) (registration required). 2015. Accessed 4-11-23.
- Robert E, Mutchinick O, Mastroiacovo P, Knudsen LB, Daltveit AK, Castilla EE, Lancaster P, Källén B, Cocchi G. An international collaborative study of the epidemiology of esophageal atresia or stenosis. *Reprod Toxicol.* 1993;7:405–21. PubMed PMID: 8274816.
- Sharma S. Tracheoesophageal fistula. Medscape. Available [online](#) (registration required). 2017. Accessed 4-11-23.
- Shaw-Smith C. Oesophageal atresia, tracheo-oesophageal fistula, and the VACTERL association: review of genetics and epidemiology. *J Med Genet.* 2006;43:545–54. PubMed PMID: 16299066.
- Stringer MD, McKenna KM, Goldstein RB, Filly RA, Adzick NS, Harrison MR. Prenatal diagnosis of esophageal atresia. *J Pediatr Surg.* 1995;30:1258–63. PubMed PMID: 8523220.
- Walsh LE, Vance GH, Weaver DD. Distal 13q deletion syndrome and the VACTERL association: case report, literature review, and possible implications. *Am J Med Genet.* 2001;98:137–44. PubMed PMID: 11223849.

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