

# South Australian Perinatal Practice Guidelines

# Amniotic Fluid Embolism

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## Note:

This statewide guideline has been prepared to promote and facilitate standardisation and consistency of practice, using a multidisciplinary approach.

Information in this statewide guideline is current at the time of publication.

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The clinical material offered in this statewide standard/policy provides a minimum standard, but does not replace or remove clinical judgement or the professional care and duty necessary for each specific patient case. Where care deviates from that indicated in the statewide guideline contemporaneous documentation with explanation must be provided.

This statewide guideline does not address all the elements of clinical practice and assumes that the individual clinicians are responsible for:

- > Discussing care with consumers in an environment that is culturally appropriate and which enables respectful confidential discussion. This includes the use of interpreter services where necessary,
- > Advising consumers of their choice and ensuring informed consent is obtained,
- > Providing care within scope of practice, meeting all legislative requirements and maintaining standards of professional conduct, and
- > Documenting all care in accordance with mandatory and local requirements

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## Background

- > Amniotic fluid contains various concentrations of fetal squamous epithelial cells, lanugo hair, vernix, mucin, zinc coproporphyrin, prostaglandins, and platelet activating factor
- > Amniotic fluid embolism (AFE) occurs only rarely, and on many occasions material from the amniotic fluid may pass into the maternal circulation without causing symptoms

## Pathogenesis

- > The pathogenesis of AFE is poorly understood. The entrance of amniotic fluid into the maternal circulation, possibly under a pressure gradient, is the principal mechanism invoked in the pathogenesis of AFE. This triggers the clinical manifestations of the condition (mainly maternal cardio-respiratory collapse), but how it does so is less clear. Some have suggested that it represents an anaphylactic reaction to the fetal material, which stimulates a cascade of endogenous-immune mediators (Conde-Agudelo & Romero 2009)

## Prevalence

- > The estimated frequency of AFE is between 1.25 / 100,000 and 12.5 / 100,000 (RCOG 2011). A United States national registry for AFE found that 70 % of cases occurred during labour, 19 % during caesarean section and 11 % immediately after vaginal birth (Clark et al. 1995)
- > AFE has also been reported during first-trimester surgical termination of pregnancy, second trimester termination, abdominal trauma and amniocentesis
- > Survival rates have improved significantly over time (14 % in 1979; 30 % in 2005; 80 % in 2010), mostly due to improvements in resuscitation. However, hypoxia-induced neurological impairment is also a major issue (RCOG 2011)

## Maternal mortality / morbidity

- > AFE is a leading cause of direct maternal death in Australia (Sullivan et al. 2007) as it is in other high-income countries. The maternal fatality rate is reported as 35 % (Roberts et al. 2010), within the range of that reported in national registries in the United Kingdom and the United States, respectively 37 % and 61 %. The United Kingdom registry reported neurological impairment in 7 % of survivors (Dawson in CMACE 2011)
- > Neonatal survival was 79 % in the US registry and 78 % in the UK registry

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## Risk factors

- > No definite risk factors have been established in the literature and AFE is such a rare event that no risk factor is likely to be of prognostic value

## Diagnosis

- > Presumptive - based on clinical presentation after considering other causes of haemodynamic instability
- > Premonitory symptoms, such as restlessness, numbness, agitation, tingling, may have been present
- > Currently, there is no definitive diagnostic test.
- > The United Kingdom Obstetric Surveillance system (UKOSS) and the United States registry recommend the following criteria, all of which must have occurred in the context of labour, caesarean delivery, dilation and evacuation, or within 30 minutes postpartum with no other explanation of findings, to make the diagnosis of AFE
  - > Acute hypotension or cardiac arrest
  - > Acute hypoxia
  - > Coagulopathy or severe haemorrhage

OR

- > Women in whom the diagnosis was made at post-mortem examination with the finding of fetal squames or hair in the lungs

## Differential diagnosis

- > Anaphylaxis
- > Aortic dissection
- > Cholesterol embolism
- > Myocardial infarction
- > Pulmonary embolism
- > Septic shock

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Clinical features of AFE compared to pulmonary embolism		
	AFE	PE
Timing of onset	Most likely to occur during delivery	Any time
Early symptoms	Dyspnoea, restlessness, panic, feeling cold, paraesthesiae, pain less likely	Dyspnoea, cough, haemoptysis
Collapse	Highly likely	May occur
DIC	Highly likely	Absent
ECG	Non specific	Non specific
CXR	Pulmonary oedema, ARDS, right atrial enlargement, prominent pulmonary arc	Segmental collapse, raised hemidiaphragm, unilateral pleural effusion
ABG	Non specific	Non specific
CTPA	Negative	Positive

## Presentation

- > AFE typically occurs during labour or delivery, or immediately postpartum, but has occurred during termination of pregnancy, after abdominal trauma, and during amnioinfusion or amniocentesis (Moore & Smith 2009)
- > The woman is often conscious at the onset of symptoms. Not all AFE is rapidly progressive and early diagnosis and supportive treatment may result in better outcomes
- > Acute shivering, sweating, anxiety and coughing, followed by respiratory distress, altered mental status, seizure or seizure-like activity and cardiovascular collapse (profound hypotension, tachycardia and possible arrhythmias)
- > More than 80 % of women with AFE experience cardio-respiratory arrest within the first few minutes (Perozi & Englert 2004)
- > Disseminated intravascular coagulation (DIC) can occur quickly, causing massive maternal haemorrhage

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## Management

- > Ask for help early (Code blue or MET call, depending on hospital)
- > Prompt resuscitation whilst considering the differential diagnosis
- > The most senior person should take charge and assign roles and responsibilities to all other individuals with good communication and organisation
- > Recruit as many people as possible to assist during resuscitation e.g. to record events, drugs given, someone to make urgent phone calls, to organise transport of laboratory samples, to bring blood (products) to the site of resuscitation, and additional staff to support family members and significant others
- > Commence monitoring immediately, including SpO<sub>2</sub>, automated blood pressure recording, insert indwelling catheter as soon as possible without hindering basic life support
- > Avoid delays once a decision is made about the following:
  - > Perimortem caesarean section (for more information on perimortem caesarean section, follow link to [Chapter 14b maternal collapse](#): perimortem caesarean section)
  - > Urgent release of resuscitation products to treat signs of coagulopathy (liaise with haematologist)

## Medical Care

### Treatment is supportive (ABC)

- > Get help: obstetrician, anaesthetist, intensivist, midwife(s), paediatrician, haematologist on hand
- > Administer oxygen via mask to maintain normal saturation 100 % O<sub>2</sub>: via bag and mask may be required
- > Apply lateral tilt (preferably left) to the woman or manually displace the uterus
- > Support ventilation and intubate if necessary
- > Initiate cardiopulmonary resuscitation (CPR) if the woman has a cardiac arrest. If she has not responded to resuscitation after 4 minutes, perform a so-named perimortem caesarean section (For further details on advanced life support [ALS] measures and perimortem caesarean section, follow link to [chapter 14b maternal collapse](#))
- > IV access with 2 x 16 gauge cannulae
- > Urgent bloods as below
- > Treat hypotension with warmed crystalloid, colloid and blood products. Consider vasopressors
- > Insert indwelling urinary catheter
- > Maintain body heat with warmer or space blanket
- > Consider pulmonary artery catheterisation in patients who are haemodynamically unstable
- > Continuous CTG. Fetal bradycardia is common and may not be an indication for immediate caesarean section

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## Investigations

Take urgent bloods:

- > Group and match 6 units
- > Complete blood picture (CBP) with platelets
- > Coagulation profile including D-dimer
- > Arterial blood gases (ABG)

## Coagulopathy

- > Activate local massive transfusion protocol, anticipate and treat coagulopathy, in collaboration with a specialist haematologist, including:
  - > FFP
  - > Cryoprecipitate
  - > Transfuse platelets
  - > Seek haematological advice for any further treatment as necessary

## Surgical Intervention

- > Perform perimortem caesarean section in women who have suffered a cardiac arrest and who are unresponsive to resuscitation (follow link to [chapter 14b maternal collapse](#): perimortem caesarean section) It may be appropriate to perform a caesarean section for other maternal or fetal indications before cardiac arrest
- > Consider mechanical and / or surgical techniques to control uterine haemorrhage (Bakri balloon, B-Lynch suture)

## Imaging and ECG

- > CXR findings are usually nonspecific, but evidence of pulmonary oedema may be observed
- > ECG changes are not specific. They may show tachycardia, ST segment and T-wave changes, and findings consistent with right ventricle strain

## Resuscitation and perimortem caesarean section is successful:

- > Care in a hospital with adult intensive care facilities
- > Significant maternal / neonatal morbidity is associated with AFE
- > Provide adequate counselling to the woman / family as soon as possible and arrange further follow-up

## Complications

- > Pulmonary oedema is a common occurrence in survivors. Pay close attention to fluid input and output
- > Left heart failure may occur. Some sources recommend inotropic support
- > Treat ongoing DIC with blood components. In consultation with haematologist, consider activated factor VIIa for intractable haemorrhage
- > Consider further surgical techniques. Bilateral uterine artery embolisation has been successful in controlling blood loss in 2 reported cases (Goldszmidt and Davies 2003)
- > Hysterectomy if unable to control bleeding

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## Prognosis

- > Many survivors have neurological deficits
- > The intact infant survival rate is 70 %. Neurologic status of the infant is directly related to the time elapsed between maternal cardiac arrest and delivery
- > Risk of recurrence is unknown. Successful subsequent pregnancies have been reported

## Resuscitation and perimortem caesarean section unsuccessful:

- > A post-mortem will be required (any medical devices, such as intravenous lines or tubes, should not be removed)
- > Provide adequate counselling to the partner / family as soon as possible

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## Version control and change history

**PDS reference:** OCE use only

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