

ANAPHYLAXIS: ASSESSING PATIENTS WITH ALLERGIES

By referring to a case study, KATHERINE TSANG reports on the results of an initial respiratory assessment of patients with severe food allergies



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Anaphylaxis is a common presentation seen in emergency departments (EDs), yet due to a lack of consensus over definition its true prevalence remains unknown (Moneret-Vautrin *et al* 2005). There is agreement however that the presence of the life-threatening features of respiratory difficulty and hypotension constitute a severe reaction (Hathaway 2005, Lieberman *et al* 2005, Moneret-Vautrin *et al* 2005).

Anaphylaxis deserves attention because it can progress from mild to fatal within minutes unless appropriate assessment and care are provided. Early recognition and understanding of the symptoms, along with rapid and accurate assessment, are therefore crucial.

The main focus of this article is the initial respiratory assessment of patients presenting with severe allergic reactions to food substances. It refers to a case study of a 15-year-old male who presented to a 24-hour paediatric ED staffed by nine paediatric trained nurses. To maintain his confidentiality, an alias, Adam, has been used and no information that could identify him has been included. The procedure described is in accordance to Nursing and Midwifery Council (NMC) (2002) guidelines.

CASE HISTORY

Adam was brought by paramedics to the ED after suffering an anaphylactic reaction.

After Adam's history was taken it was found that he was 15 years old and a known asthmatic, with an allergy to uncooked eggs. His family has a history of food allergy, asthma and eczema.

Adam had spent the evening at home with his family. He had eaten muesli and within minutes developed a mild allergic reaction. He was given an antihistamine by his father, but as his symptoms did not settle, he was given a second antihistamine. In less than an hour, Adam deteriorated and the emergency services were contacted. Although Adam had an adrenaline pen, his father was anxious about using it and it was withheld.

When the paramedics arrived, Adam had collapsed but was rousable.

The paramedics found that he had a Glasgow coma score (GCS) of E3 + V1 + M6 = 10/15. His airway was protected but showed signs of respiratory distress. He had equal air entry, but had wheeze and recession, and he seemed to be breathing quite deeply. Pulse oximetry revealed significantly reduced oxygen saturation levels at 80 per cent in air, but he was peripherally cold and clammy, and his capillary refill time was about three seconds. His blood pressure (BP) was 100mmHg/60mmHg, with a pulse of 120 beats per minute. He also had a rash on his chest.

On the way to the ED the paramedics administered 0.5ml of 1:1000 adrenaline intramuscularly, followed by 5mg nebulised salbutamol. During the journey the rash on Adam's chest spread to his limbs. On arrival to the ED, the paramedics reported that Adam was improving throughout the journey.

INITIAL PATIENT ASSESSMENT

As the paramedics had prioritised Adam, the paediatric team was ready to receive and assess him on arrival. Adam was assessed using the airway, breathing, circulation, disability, or ABCD, approach as identified by the Advanced Life Support Group (ALSG) (2005). This approach takes less than a minute and so allows rapid clinical assessments (ALSG 2005).

The purpose of any triage assessment is to identify a process of injury or illness and prevent or minimise potential detrimental effects through rapid assessment and decision making (Cone and Murray 2002).

The ABCD approach is most effective in managing potentially life-threatening conditions because it allows patients to be assessed and stabilised before proceeding to other aspects and management of underlying conditions. Zimmermann (2002) supports the approach, stating that triages should always start with the ABCD assessment regardless of presenting complaint.

Adam's assessment identified a protected airway, with spontaneous breathing at 24 breaths per minute (BPM). He spoke in monosyllables and had a notably hoarse voice. An absence of wheeze and crackles was noted on chest auscultation.

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The use of accessory muscles was evident with the presence of intercostal recession, and diaphragmatic contraction was noted to be increased. Oxygen saturations were recorded at 100 per cent with 12L of oxygen. No cyanosis was observed. He had a BP of 70mmHg/37mmHg, and a weak pulse rate of 117. His capillary refill time was three seconds and his peripheries were very cold and clammy.

Since he had presented at the ED, Adam's GCS had improved to E3 + V5 + M6 = 14/15 and he was observed to have normal pupil response and normal posture. His body temperature was 34.8°C and blood glucose was 8.0mmols. He had an urticarial rash over his trunk and limbs, and facial swelling that was most noticeable over his eyelids and lips.

A diagnosis of anaphylaxis was made. Adam's vital signs are listed in Table 1.

ALLERGIC FOOD REACTIONS

Anaphylaxis has no universal definition (Hathaway 2005) but McKevith and Theobald (2005) define it as 'an inappropriate reaction to a food component by the immune system'. Other writers on the subject, such as Moneret-Vautrin *et al* (2005), offer a more specific definition, describing it as a 'severe systemic reaction affecting two or more organs or systems'.

Lack of uniformity in the definition of anaphylaxis is partly due to the large number of symptoms associated with it, and debate about which of these are essential features of the condition (Ewan 1998). It is generally agreed however that such life-threatening features as respiratory difficulty and hypotension constitute a severe allergic reaction (Hathaway 2005, Lieberman *et al* 2005, Moneret-Vautrin *et al* 2005).

Anaphylaxis can be mild, yet can progress rapidly to severe symptoms resulting in fatal outcomes (Lieberman *et al* 2005). An awareness that allergic reactions occur as part of a continuum must be borne in mind during patient assessments because clinical situations can evolve during and after initial assessment (Crusher 2004, Lieberman *et al* 2005). The potential danger of any anaphylaxis reaction is that asphyxia, shock or respiratory arrest can occur, and can be fatal (Ewan 1998).

Beyer and Teuber (2004) say that allergic food reactions affect 8 per cent of children and 2 per cent of adults in Westernised countries. The exact figure is unknown due to a lack of consensus on the definition (Ewan 1998, Moneret-Vautrin *et al* 2005).

After reviewing relevant literature from various countries however, Moneret-Vautrin *et al* (2005) found that between one and three people in 10,000 have severe anaphylaxis, and between one and three people in a million die of anaphylaxis.

Childhood food allergies to milk, eggs, wheat and soya tend to be outgrown in 85 per cent of cases, while allergies to peanuts, tree nuts and seafood are not commonly outgrown (Sicherer and Teuber 2004).

Adolescents and young people are at greatest risk of severe anaphylaxis (Moneret-Vautrin *et al* 2005). Fatalities are particularly high in people with asthma and existing food allergies to peanuts, tree nuts and seafood (Sicherer and Teuber 2004).

Hathaway (2005) describes anaphylaxis as a 'known clinical response to a type 1 hypersensitivity immunological reaction between a specific antigen and an antibody'. Immunoglobulin E is the antibody specifically responsible for the majority of anaphylaxis reactions, because it is attracted to foreign proteins and activates the immune system. Immunoglobulin E is found in small numbers attached to mast cells and basophils (Henderson 1998).

Following the ingestion of an allergen, the body immediately reacts by mounting the inflammatory response. This ensures that defensive mediators such as antibodies, phagocytes and clot-forming chemicals are deployed to the injured site to remove the toxins from the body and to prevent their spread to other areas (Totora and Grabowski 1996).

Following ingestion of the offending food item, the allergen quickly gains access to the circulatory system through the digestive tract, activating mast cell release in the mouth, throat, lungs, skin, abdomen and other tissues and organs (Crusher 2004). Mast cells are found in the loose connective tissue, bronchi, gastrointestinal mucosa and around blood vessels (Henderson 1998). They contain an extensive array of mediators that are responsible for the anaphylaxis response, such as histamines, spasmogens, inflammatory activators and a potent bronchodilator.

Basophils are circulating white blood cells that contain histamine and heparin. On entering tissues, basophils become mast cells. Once an antigen attaches to an antibody, it causes degranulation of the mast cells and basophils, leading to the clinical picture of an allergic reaction.

Table 1

Expected vital signs for Adam and vital signs on arrival.

	Normal vital signs expected in a 15-year-old	Adam's vital signs on arrival to ED
Respiration	18 breaths per minute	24 breaths per minute
Pulse	55-90 beats per minute	117 beats per minute
Blood pressure	114mmHg/65mmHg	70mmHg/37mmHg
Temperature	36.6°C	34.8°C

The release of chemical mediators, specifically histamines, tryptase and leukotrienes into the extracellular fluid, causes attraction of eosinophils, vasodilatation, smooth muscle contraction, increased glandular secretion and capillary permeability (Henderson 1998).

The manner in which the antigen gains entry into the body determines the main clinical features (Henderson 1998). Cutaneous signs and angioedema tend to occur before bronchiolar constriction and laryngeal oedema.

The final manifestation is shock, indicating the most severe type of reaction, involving multiple systems (Wong 1997). A summary of the effects of anaphylaxis is shown in Table 2.

The immediate inflammatory response of vasodilatation and increased permeability of the blood vessels results in increased blood flow to

the damaged area. This causes large amounts of defensive materials to be carried to the area to rid the body of toxins, and large amounts of clotting factors to be carried to the tissues to localise and trap the invading allergens.

The increase of blood vessel diameter and permeability results in redness, heat and swelling to the affected area. Smooth muscle contraction and increased mucus secretions result in the symptoms of a runny nose and difficulty breathing (Totora and Grabowski 1996). The common initial symptoms of an allergic reaction are the cutaneous signs with angioedema (Ewan 1998).

Adam's urticarial rash, for example, was due to fluid leakage into the upper dermis, while fluid leakage into the deep epidermis resulted in the angioedema (lip and eye swelling).

Localised responses are limited only to a small area, whereas a systemic reaction affects several or all parts of the body. Wong (1997) states that the involvement of multiple systems would encompass the cardiovascular, respiratory, gastrointestinal and integumentary systems.

As a result of the anaphylaxis process, Adam's cutaneous, respiratory and cardiovascular symptoms indicated multi-system involvement, indicative of a severe reaction. The key findings of Adam's assessment established the presence of respiratory difficulty and shock, both of which are potentially fatal.

CIRCULATORY SHOCK

Shock is defined as a circulatory failure that results in inadequate tissue perfusion with cellular dysfunction and eventual organ failure (Wong 1997).

In anaphylaxis, the movement of large volumes of fluid from the intravascular space into the interstitial space, with a net loss, results in hypotension and impaired cardiac output with poor perfusion (Henderson 1998).

Lieberman *et al* (2005) state that the severity of anaphylaxis is such that up to 50 per cent of the intravascular fluid can move into the extravascular space within ten minutes, leading to haemodynamic collapse with little cutaneous or respiratory manifestations.

In anaphylaxis, the shock is caused by the vast quantities of mediator release which encourages fluid loss and capillary permeability (Ewan 1998).

Once the patient is in shock, there is reduced venous return, low cardiac output and hypotension, caused in part by the initial response of vasodilatation (Wong 1997). In an attempt to compensate, the rate and force of cardiac contractions increase along with the constriction of arterioles and veins, which result in increased peripheral vascular resistance.

Table 2

Summary of the potential effects on body systems of anaphylaxis

Body system	Effects	Symptoms
Integumentary	Vasodilatation	Angioedema Lip and eyelid swelling Urticarial rash Pruritus and sensation of warmth Watery eyes
	Cyanosis	Cold, clammy skin
Cardiovascular	Vasodilatation	Hypotension
	Capillary permeability	Impaired cardiac output
	Movement of large volumes of fluid	Poor perfusion, hypotension
	Hormone secretion to decrease blood flow to skin, kidneys, muscles and heart	Tachycardia
Gastrointestinal	Mediator release	Nausea
	Vasodilatation	Nausea
	Smooth muscle contraction	Abdominal cramps
	Glandular secretion	Vomiting, diarrhoea
Respiratory system	Mucus secretion	Rhinitis, sneezing
	Capillary permeability	Dyspnoea
	Smooth muscle contraction	Stridor, hoarse voice, dysphagia
	Decrease of peripheral airway diameter	Wheeze, asthma
	Hypoxia	Cyanosis, behavioural change
	Use of accessory muscles	Dyspnoea, diaphragmatic contraction

Adam was clearly in shock because his BP, of 70mmHg/37mmHg, should have approximated 114mmHg/65mmHg. His tachycardia was caused by his body's autonomic compensatory mechanisms (Henderson 1998). Adam's heart rate while awake should have been approximately 55 to 90 beats per minute, but Adam's body was attempting to compensate for the low BP and his heart rate was 117 on arrival. Adam's weak pulse volume was also indicative of hypotension (ALSG 2005).

Simultaneously, hormones are secreted to help conserve body fluid, with the effect of reduced blood flow to the skin, kidneys, muscles and heart (Wong 1997).

This physiological process resulted in Adam's skin feeling cold and clammy with a poor capillary refill time and reduced body temperature due to the peripheral shutdown (Henderson 1998).

RESPIRATION DURING ANAPHYLAXIS

If perfusion is impaired because of oxygen depletion, the anaerobic respiratory pathways can be initiated. This can lead to lactic acidosis, which places an extra burden on the lungs in an attempt to remove excess carbon dioxide by increasing the respiratory rate (Wong 1997).

Behrman *et al* (1996) explain that the respiratory rate remains the best indicator of respiratory effectiveness and severity of respiratory distress because oxygen administration can maintain near normal blood gas levels while the compensatory mechanisms are sustained.

At 24 BPM, compared with a normal breath rate of 18 BPM, Adam's respiratory rate was elevated. His blood gas results were unavailable during the initial assessment.

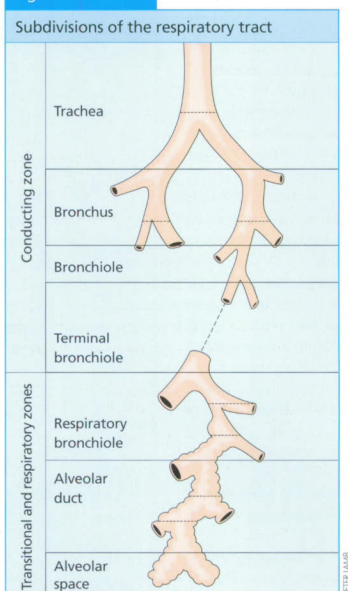
The effect of anaphylaxis on the respiratory system is such that increased capillary permeability and smooth muscle contraction result in the commonly seen symptoms. The smooth muscles of the airway are located throughout most of the tracheo-bronchial tree up to the level of the respiratory bronchioles (Wilkins *et al* 1996) (Figure 1).

The effect of smooth muscle contraction is most significant distal to the trachea and large bronchi, and leads to a significant decrease in the diameter of the peripheral airways (Wilkins *et al* 1996).

Hepner (2005) states that the bronchospasm in anaphylaxis requires the presence of previous pulmonary inflammation and mediator release, and for this reason asthmatics have an increased likelihood of suffering pulmonary manifestations after antigen exposure.

Mediators of anaphylaxis are potent constrictors of smooth muscle and provoke mucous hypersecretion and increase vascular permeability.

Figure 1



The effect of the mediators is such that it leads to respiratory difficulty and could cause asphyxia or respiratory arrest.

Flenley (1990) says that those who die from asthma are found to have the precise symptoms which can result from anaphylaxis such as inflammation, vasodilatation, oedema and hyperplasia of smooth muscle. He also states that tachypnoea of more than 20 BPM is typical in pulmonary disease, identifying pulmonary oedema as a cause.

In the case study, these aspects are especially relevant and competent assessment of Adam's respiratory effort was therefore extremely important. Adam's respiratory rate, at 24 rather than 18 BPM, was only mildly elevated, yet due to the increased capillary permeability and mucus secretion with bronchial constriction, the presence of pulmonary oedema could adequately explain his increased respiratory rate and effort.

Behrman *et al* (1996) believe that tachypnoea is an early sign of the increased physical effort required to provide adequate ventilatory support, along with use of accessory muscles.

Respiration normally provides the mechanism for gas exchange between the circulating blood and the atmosphere (Behrman *et al* 1996).

As Figure 2 shows, the airways comprise the diaphragm, chest wall, pharynx and larynx (Behrman *et al* 1996).

The accessory muscles of respiration, namely the sternomastoid, scalene, trapezius, intercostal, rhomboid and abdominal muscles, assist by increasing the thoracic volume, and are located in the neck and upper part of the chest (Behrman *et al* 1996, Wilkins *et al* 1996) (Figure 3). These muscles are not active during relaxed breathing but are activated when respiratory effort is increased (Wilkins *et al* 1996), but the interaction between them determines the respiratory effect (Behrman *et al* 1996).

The use of accessory muscles is largely due to the increased resistance to air flow in the thorax, as seen during episodes of lung inflammation (Wilkins *et al* 1996). They can generate enormous pressures and sustain large increases in work for a long time.

However, being skeletal in origin, the muscle can undergo fatigue, leading to respiratory failure. Children of all ages with decreased perfusion are particularly susceptible to this (Behrman *et al* 1996).

Adam was susceptible to the risk of respiratory failure, so accurate respiratory assessment was vital in preventing respiratory failure.

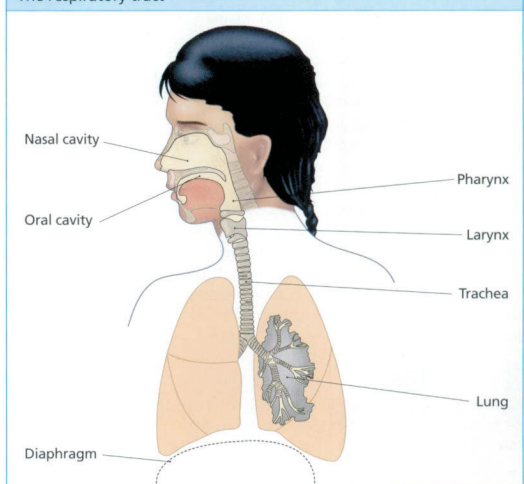
Behrman *et al* (1996) believe that assessment of physical signs can give many clues to the nature of the respiratory dysfunction. They also believe that these assessments should take into account: the presence and vigor of respiratory movements; the presence of cyanosis, upper airway obstruction and retractions; the extent of respiratory movements and use of accessory muscles; and the breathing rate, with attention being paid to each patient's state of consciousness.

The depth of breathing is difficult to quantify and therefore the respiratory rate can provide an excellent and reliable guide, as the respiratory rate increases as the muscles become less effective (Behrman *et al* 1996).

Lissauer and Clayden (2007) believe that the degree of subcostal, intercostal and sternal recession are more reliable indicators than respiratory rate of

Figure 2

The respiratory tract



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the severity of upper airway obstruction, and the ALSG (2005) supports this physical finding as a good indicator for the severity of respiratory difficulty.

In the case study, it was noted that Adam had an increase in respiratory rate, which required a considerable increase in effort, witnessed through observation of depth and use of the accessory muscles. Cyanosis was not present and Adam had a sound state of mind, which ruled out the presence of hypoxaemia.

Intercostal recession was present in Adam because the intercostal spaces have no bony support, and as a result move inwards under the effects of negative pleural pressure during inspiration.

Intercostal recession is exaggerated whenever the pleural pressure is more negative than normal (Behrman *et al* 1996, Flenley 1990). Recession may be revealed in patients with respiratory difficulty (ALSG 2005).

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Although Adam displayed signs of recession, their severity was assessed as mild to moderate.

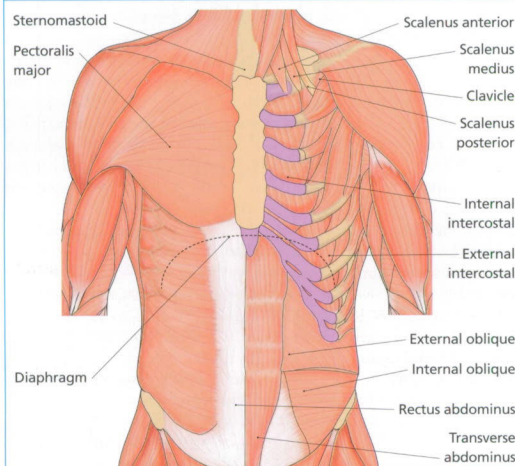
In anaphylaxis, the main respiratory symptoms are wheezing or asthma, dyspnoea, rhinitis, laryngeal obstruction causing stridor and hypoxia (Henderson 1998, McKeivith and Theobald 2005).

Laryngeal angioedema, either isolated or in conjunction with facial angioedema, must be considered as serious, because asphyxia can occur (Moneret-Vautrin *et al* 2005). Laryngeal oedema as a result of the anaphylaxis response would frequently cause stridor (Henderson 1998).

Adam did not have an obvious audible stridor but spoke in monosyllables with an especially hoarse voice. Despite lack of stridor, hoarseness is an indication of inflammation to the vocal cords (Flenley 1990, Lissauer and Clayden 2007), and Adam's inability to speak effectively was another demonstration of respiratory difficulty.

Figure 3

Front view of the muscles of ventilation



Where hypotension is present, patients should be asked to lie down with their legs elevated to slow the progression of haemodynamic compromise (Hathaway 2005, Henderson 1998, Lieberman *et al* 2005).

Due to the increase in respiratory effort, Adam was placed in an upright position to aid his respiratory effort, and oxygen was administered. On arrival at the ED, he sat upright and was not repositioned. Allowing Adam to sit upright gave the accessory muscles improved leverage, which allowed for easy diaphragmatic movement (Behrman *et al* 1996). In light of Pumphrey's (2003) finding that death can occur from repositioning patients in shock due to sudden changes in the circulatory system, the decision to allow Adam to sit up was also relevant to his care.

When respiration is difficult, it is recommended that patients should sit up, even when hypotension is present (Crusher 2004, Ewan 1998, Sicherer and Teuber 2004).

SUMMARY

Adam's assessment identified the presence of the most severe symptoms of anaphylaxis, respiratory difficulty and shock. The actions of the ambulance crew ensured that on arrival at the ED, Adam's condition was improving. However, it was found that despite the interventions already undertaken, Adam was suffering significant respiratory difficulty due to the cascade of events that occur in anaphylaxis.

Adam was seen on arrival and the ALSG's (2005) assessment method proved to be the most effective to ensure that Adam was assessed and treated rapidly.

The most beneficial aspect of this assignment is that the author can return to practice with this knowledge and will be more confident when faced with other patients with anaphylaxis.

Understanding the underlying processes and symptoms has decreased the fear of 'not knowing' and will aid accurate assessment. It will also provide a good basis for educating parents of affected children and members of nursing teams.

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