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Preface to the Australia and New Zealand fifth edition

The fifth edition of the APLS manual maintains the clarity and structure of the earlier editions. It adds a greater level of detail and explanation, based on the latest medical science and guidelines. The editors, working groups and contributors are to be congratulated for continuing to improve a book that was already one that you wished you could have with you at all times.

To achieve consistency with Australasian guidelines and clinical practice the manual has been adapted for Australia and New Zealand.

The chapters on Basic and Advanced Life Support have been rewritten to match the guidelines of the Resuscitation Councils of Australia and New Zealand. Local epidemiology replaces UK data. Information on management, including procedures and medications, follows current practice and availability. Illustrations and photographs have been added to complement the text.

This task has been achieved thanks to the diligence, knowledge and experience of the members of the Editorial Group of the Australia and New Zealand edition. Many thanks also go to Jane Andrew and all at Wiley-Blackwell for their invaluable assistance with this project.

Matthew O’Meara
Sydney, 2011
Preface to the first edition

Advanced Paediatric Life Support: The Practical Approach was written to improve the emergency care of children, and has been developed by a number of paediatricians, paediatric surgeons, emergency physicians and anaesthetists from several UK centres. It is the core text for the APLS (UK) course, and will also be of value to medical and allied personnel unable to attend the course. It is designed to include all the common emergencies, and also covers a number of less common diagnoses that are amenable to good initial treatment. The remit is the first hour of care, because it is during this time that the subsequent course of the child is set.

The book is divided into six parts. Part I introduces the subject by discussing the causes of childhood emergencies, the reasons why children need to be treated differently and the ways in which a seriously ill child can be recognised quickly. Part II deals with the techniques of life support. Both basic and advanced techniques are covered, and there is a separate section on resuscitation of the newborn. Part III deals with children who present with serious illness. Shock is dealt with in detail, because recognition and treatment can be particularly difficult. Cardiac and respiratory emergencies, and coma and convulsions, are also discussed. Part IV concentrates on the child who has been seriously injured. Injury is the most common cause of death in the 1–14-year age group and the importance of this topic cannot be overemphasised. Part V gives practical guidance on performing the procedures mentioned elsewhere in the text. Finally, Part VI (the appendices) deals with other areas of importance.

Emergencies in children generate a great deal of anxiety – in the child, the parents and in the medical and nursing staff who deal with them. We hope that this book will shed some light on the subject of paediatric emergency care, and that it will raise the standard of paediatric life support. An understanding of the contents will allow doctors, nurses and paramedics dealing with seriously ill and injured children to approach their care with confidence.

Kevin Mackway-Jones
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CONTACT DETAILS
APLS provides training courses to doctors, nurses and paramedics on Paediatric Life Support and Advanced Paediatric Life Support throughout Australia and New Zealand, with outreach work conducted in developing countries in the Pacific Rim and elsewhere.

This not-for-profit organisation is fully committed to promoting excellence in emergency care of ill and injured children through the provision of high-quality contemporary education programs for health care professionals.

For further information on future courses, visit the website or contact:

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UPDATES
This fifth edition of Advanced Paediatric Life Support: The Practical Approach has been fully reviewed and adapted for use in Australia and New Zealand by the Australia and New Zealand editorial committee.

REFERENCES
The APLS website contains all materials relating to APLS courses – visit www.apls.org.au. All algorithms used in this manual are freely available to download and reproduce at www.apls.org.au.
PART 1

Introduction
CHAPTER 1

Introduction

1.1 INTRODUCTION

Each year many millions of children around the world die from potentially preventable and treatable causes. Whilst the majority of these deaths would be prevented by attention to living conditions and public health measures, an improvement in the recognition of serious illness and delivery of initial medical treatment would undoubtedly save lives.

The training of health care practitioners and the resources available for health care delivery varies enormously among countries. It is possible, however, to improve the outcome of serious illness and injury in children with modest resources if the basic principles of resuscitation are adhered to. The structured sequential approach to the recognition and treatment of the seriously ill and injured child followed in this manual is applicable in many situations and circumstances.

1.2 MORTALITY RATES IN CHILDHOOD

The infant mortality rate is defined as the number of deaths of children under 1 year of age in one calendar year per 1000 live births in the same calendar year.

Worldwide mortality rates in children have fallen substantially and consistently over the last 100 years. The World Health Organisation has estimated that the global infant mortality rate has fallen from 180 in 1950 to around 50 in 2010. In some developed countries the fall has been even more dramatic. For example in Australia the infant mortality rate in 1902 was 107; 100 years later in 2002 the figure had reduced to 5 where it stayed for the next 5 years. Even with figures at such low levels, the rates in developed countries have recently continued to fall. In England and Wales the infant mortality rates have more than halved in the last 28 years, falling from 12 in 1980 down to 4.5 in 2008: the lowest on record.

These dramatic improvements in infant mortality are due largely to improvements in living conditions such as sanitation, shelter, quality of drinking water and better nutrition. Some medical measures such as better obstetric and neonatal care and the advent of mass vaccination have also played substantial roles. The delivery of better acute care for seriously ill and injured children is likely to assist in reducing mortality rates further.

The mortality rate decreases significantly with the increasing age of the child, with the highest death rate occurring in the first 28 days, and indeed most deaths occur on the first day of life. Male children are more likely to die than females in all age groups, a trend which is not reversed until much later in life.

1.3 CAUSES OF DEATH IN CHILDHOOD

The causes of death in childhood in any country vary with age. Figure 1.1 shows the top worldwide causes of death for children under 6 years of age. In the newborn period the most
In children aged 1–12 months, congenital abnormalities, conditions related to prematurity and sudden unexplained death each contribute around 20% to mortality. This is in contrast to a number of years ago when sudden infant death syndrome (as it was known then) was much more prevalent.

Congenital abnormalities contribute significantly to mortality rates during all stages of childhood. Complex congenital heart disease, central nervous system malformations, metabolic disorders and chromosomal anomalies are the commonest lethal disorders. After 1 year of age, trauma is a frequent cause of death and remains so until well into adult life. Deaths from trauma have been described as falling into three groups. In the first group there is overwhelming damage at the time of the trauma, and the injury caused is incompatible with life; children with such massive injuries will die within minutes whatever is done. Those in the second group die because of progressive respiratory failure, circulatory insufficiency or raised intracranial pressure secondary to the effects of injury; death occurs within a few hours if no treatment is administered, but may be avoided if treatment is prompt and effective. The final group consists of late deaths from raised intracranial pressure, infection or multiple organ failure. Appropriate management in the first few hours will also decrease mortality in this group.

In developing countries, infectious diseases are still major causes of death. Seven out of 10 childhood deaths can be attributed to just five main causes: pneumonia, diarrhoea, measles, malaria and malnutrition. Three out of every four children seen by health services are suffering from at least one of these conditions. Human immunodeficiency virus (HIV) and acquired immune deficiency syndrome (AIDS) have contributed to this and also been associated with increasing deaths from tuberculosis in countries affected. As these societies become more urbanised the mortality from trauma, especially from motor vehicle accidents, increases. In South Africa, a country that, although developing rapidly, has large areas of severe poverty, the under-fives mortality rate has recently been shown to include 40% (42,749) of deaths from...
HIV/AIDS, 11% (11,876) from low birth weight, 21% (22,680) from infections and 3% (3506) from trauma. In older South African children, trauma, especially road traffic accidents, homicide and suicide are leading causes of death. In Trinidad, children under 1 year of age accounted for 4% of deaths in 1997, with infant mortality at 17 per 1000 live births. In Trinidadian school children, the foremost cause of death was injury, with infections causing one-fifth of deaths.

In developed countries, many children with diseases that were once invariably fatal – such as complex congenital heart disease, inborn errors of metabolism, haematological malignancies or cystic fibrosis – are now treated with drugs, operations, diet, transplant or, soon, even gene therapy. In these children, common acute illnesses such as varicella or chest infections have potentially lethal consequences. They require a low threshold for rapid aggressive treatment delivered by a team with an understanding of their underlying disease.

Only a minority of childhood deaths, such as those due to end-stage neoplastic disease, are expected and ‘managed’. There should be timely discussions among the child, family and health carers to identify whether and in what manner resuscitation should be carried out to prevent unwanted and inappropriate resuscitation and interventions.

1.4 **PATHWAYS LEADING TO CARDIORESPIRATORY ARREST**

As the outcome from cardiorespiratory arrest in children is poor the only effective way to prevent death and permanent disability is to understand its antecedent events, and be able to recognise and treat them vigorously.

Cardiac arrest in children is rarely due to primary cardiac disease. This differs from the situation in an adult where the primary arrest is often cardiac, and circulatory and respiratory function may remain near normal until the moment of arrest.

In children, most cardiorespiratory arrests are secondary to hypoxia caused by respiratory pathology, including birth asphyxia, inhalation of foreign bodies, bronchiolitis and asthma. Respiratory arrest also occurs secondary to neurological dysfunction caused by such events as convulsion or poisoning. Raised intracranial pressure (ICP) due to head injury or acute encephalopathy eventually leads to respiratory arrest, but severe neuronal damage has already been sustained before the arrest occurs.

Whatever the cause, by the time of cardiac arrest the child has had a period of respiratory insufficiency that will have caused hypoxia and respiratory acidosis. The combination of hypoxia and acidosis causes cell damage and death (particularly in more sensitive organs such as the brain, liver and kidney) before myocardial damage is severe enough to cause cardiac arrest.

Most other cardiac arrests in children are secondary to circulatory failure. This will have resulted often from fluid or blood loss, or from fluid maldistribution within the circulatory system. The former may be due to gastroenteritis, burns or trauma, whilst the latter is often caused by sepsis or anaphylaxis. Because all organs are deprived of essential nutrients and oxygen as shock progresses to cardiac arrest, circulatory failure, like respiratory failure, causes tissue hypoxia and acidosis. In fact, both pathways may occur in the same condition. The pathways leading to cardiac arrest in children are summarised in Figure 1.2.
Figure 1.2 Pathways leading to cardiac arrest in childhood (with examples of underlying causes). ICP, intracranial pressure.

1.5 OUTCOME FROM CARDIAC ARREST IN CHILDREN

The outcome of cardiac arrest in children is poor. Of those who survive, many are left with permanent neurological deficits. The worst outcome is in children who have had an out-of-hospital arrest and arrive at hospital apnoeic and pulseless. These children have almost no chance of intact neurological survival, especially if cardiopulmonary resuscitation has been in progress for 20 minutes or longer. There has often been a prolonged period of hypoxia and ischaemia before the start of adequate cardiopulmonary resuscitation.

Earlier recognition of seriously ill children and paediatric cardiopulmonary resuscitation training for the public could improve the outcome for these children.

Figure 1.3 Advanced paediatric life support (APLS) in action.
CHAPTER 2

Why treat children differently?

2.1 INTRODUCTION

Children are a diverse group varying enormously in weight, size, shape, intellectual ability and emotional responses. At birth a child is, on average, a 3.5 kg, 50 cm long individual with small respiratory and cardiovascular reserves and an immature immune system. They are capable of limited movement, exhibit limited emotional responses and are dependent upon adults for all their needs. Fourteen or more years later at the other end of childhood, the adolescent is a 50 kg, 160 cm tall person who looks physically like an adult and often exhibits a high degree of independent behaviour.

Competent management of a seriously ill or injured child who may fall anywhere between these two extremes requires a knowledge of these anatomical, physiological and emotional differences and a strategy of how to deal with them.

Key differences to consider in children
- Weight
- Anatomical – size and shape
- Physiological – cardiovascular, respiratory and immune function
- Psychological – intellectual ability and emotional response

2.2 WEIGHT

The most rapid changes in weight occur during the first year of life. An average birth weight of 3.5 kg will have increased to 10 kg by the age of 1 year. After that time weight increases more slowly until the pubertal growth spurt. This is illustrated in the weight charts shown in Figure 2.1.

As most drugs and fluids are given as the dose per kilogram of body weight, it is important to determine a child’s weight as soon as possible. Clearly the most accurate method for achieving this is to weigh the child on scales; however, in an emergency this may be impractical. Often, especially with infants, the child’s parents or carer will be aware of a recent weight.

If weighing the child is not possible, various formulae or measuring tapes are available. The Broselow tapes use the height (or length) of the child to estimate weight. The tape is laid alongside the child and the estimated weight read from the calibrations on the tape. This is a quick, easy and relatively accurate method. Various formulae may also be used although they should be validated to the population in which they are being used.

If a child’s age is known the formulae given in Table 2.1 may be useful. The formula method has the added advantage of allowing an estimation of the weight to be made before the child arrives in hospital so that the appropriate equipment and drugs may be available.

Whatever the method used, it is essential that the carer is sufficiently familiar with it to be able to use it quickly and accurately under pressure.

Table 2.1 Weight formulae in different age groups.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–12 months</td>
<td>Weight (in kg) = (0.5 × age in months) + 4</td>
</tr>
<tr>
<td>1–5 years</td>
<td>Weight (in kg) = (2 × age in years) + 8</td>
</tr>
<tr>
<td>6–12 years</td>
<td>Weight (in kg) = (3 × age in years) + 7</td>
</tr>
</tbody>
</table>

Figure 2.1 (a) Weight percentile for girls aged 0–3 years, and (b) weight percentile for girls aged 2–18 years. (Data source: Centers for Disease Control and Prevention, www.cdc.gov/nchs/about/major/nhanes/growthcharts/datafiles.htm.)
2.3 ANATOMICAL DIFFERENCES

As the child’s weight increases with age the size, shape and proportions of various organs also change. Particular anatomical changes are relevant to emergency care.

Airway

The airway is influenced by anatomical changes in the tissues of the mouth and neck. In a young child the head is large and the neck short, tending to cause neck flexion and airway narrowing. The face and mandible are small, and teeth or orthodontic appliances may be loose. The tongue is relatively large and not only tends to obstruct the airway in an unconscious child, but may also impede the view at laryngoscopy. Finally, the floor of the mouth is easily compressible, requiring care in the positioning of fingers when holding the jaw for airway positioning. These features are summarised in Figure 2.2.

The anatomy of the airway itself changes with age, and consequently different problems affect different age groups. Infants less than 6 months old prefer to breathe via their noses. As the narrow nasal passages are easily obstructed by mucous secretions, and as upper respiratory tract infections are common in this age group, these children are at particular risk of airway compromise. In 3–8-year-olds, adenotonsillar hypertrophy may be a problem. This not only tends to cause obstruction, but also causes difficulty when the nasal route is used to pass pharyngeal, gastric or tracheal tubes.

In young children the epiglottis is horseshoe-shaped, and projects posteriorly at 45°, making tracheal intubation more difficult. This, together with the fact that the larynx is high and anterior (at the level of the second and third cervical vertebrae in the infant, compared with the fifth and sixth vertebrae in the adult), means that it is easier to intubate an infant using a straight-blade laryngoscope. The cricoid ring is the narrowest part of the upper airway (as opposed to the larynx in an adult). The narrow cross-sectional area at this point, together with the fact that the cricoid ring is lined by pseudo-stratified ciliated epithelium loosely bound to areolar tissue, makes it particularly susceptible to oedema. As tracheal tube cuffs tend to lie at this level, uncuffed tubes are preferred in emergencies and for use by non-experts in pre-pubertal children.

The trachea is short and soft. Overextension of the neck as well as flexion may therefore cause tracheal compression. The short trachea and the symmetry of the carinal angles mean that not only is tube displacement more likely, but a tube or a foreign body may be displaced into the left or right main-stem bronchus.

Breathing

The lungs are relatively immature at birth. The air–tissue interface has a relatively small total surface area in the infant (less than 3 m²). In addition, there is a 10-fold increase in the number of small airways from birth to adulthood. Both the upper and lower airways are relatively small, and are consequently more easily obstructed. As resistance to flow is inversely proportional to the fourth power of the airway radius (halving the radius increases the resistance 16-fold), seemingly small obstructions can have significant effects on air entry in children.
Infants rely mainly on diaphragmatic breathing. Their muscles are more likely to fatigue as they have fewer type I (slow-twitch, highly oxidative, fatigue-resistant) fibres compared with adults. Pre-term infants’ muscles have even less type I fibres. These children are consequently more prone to respiratory failure.

The ribs lie more horizontally in infants, and therefore contribute less to chest expansion. In the injured child, the compliant chest wall may allow serious parenchymal injuries to occur without necessarily incurring rib fractures. For multiple rib fractures to occur the force must be very large; the parenchymal injury that results is consequently very severe and flail chest is tolerated badly.

**Circulation**

At birth the two cardiac ventricles are of similar weight; by 2 months of age the left ventricle (LV) is twice the weight of the right ventricle (RV). These changes are reflected in the infant’s electrocardiogram (ECG). During the first months of life the RV dominance is apparent, but by 4–6 months of age the LV is dominant. As the heart develops during childhood, the sizes of the P wave and QRS complex increase, and the P–R interval and QRS duration become longer.

The child’s circulating blood volume per kilogram of body weight (70–80 mL/kg) is higher than that of an adult, but the actual volume is small. This means that in infants and small children, relatively small absolute amounts of blood loss can be critically important.

**Body surface area**

The body surface area (BSA) to weight ratio decreases with increasing age. Small children, with a relatively high surface area, lose heat more rapidly and consequently are relatively more prone to hypothermia. At birth the head accounts for 19% of BSA; this falls to 9% by the age of 15 years. Figure 2.3 shows these changes.

<table>
<thead>
<tr>
<th>Area indicated</th>
<th>Surface area at</th>
<th>0 year</th>
<th>1 year</th>
<th>5 years</th>
<th>10 years</th>
<th>15 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td></td>
<td>9.5</td>
<td>8.5</td>
<td>6.5</td>
<td>5.5</td>
<td>4.5</td>
</tr>
<tr>
<td>B</td>
<td></td>
<td>2.75</td>
<td>3.25</td>
<td>4.0</td>
<td>4.5</td>
<td>4.5</td>
</tr>
<tr>
<td>C</td>
<td></td>
<td>2.5</td>
<td>2.5</td>
<td>2.75</td>
<td>3.0</td>
<td>3.25</td>
</tr>
</tbody>
</table>

*Figure 2.3 Body surface area (%). (Reproduced courtesy of Smith & Nephew Pharmaceuticals.)*
2.4 PHYSIOLOGICAL DIFFERENCES

Respiratory rate
The infant has a relatively greater metabolic rate and oxygen consumption. This is one reason for an increased respiratory rate. However, the tidal volume remains relatively constant in relation to body weight (5–7 mL/kg) through to adulthood. The work of breathing is also relatively unchanged at about 1% of the metabolic rate, although it is increased in the pre-term infant.

In the adult, the lung and chest wall contribute equally to the total compliance. In the newborn, most of the impedance to expansion is due to the lung, and is critically dependent on the surfactant. The lung compliance increases over the first week of life as fluid is removed from the lung. The child’s compliant chest wall leads to prominent sternal recession and rib space indrawing when the airway is obstructed or lung compliance decreases. It also allows the intrathoracic pressure to be less ‘negative’. This reduces small-airway patency. As a result, the lung volume at the end of expiration is similar to the closing volume (the volume at which small-airway closure starts to take place).

At birth, the oxygen dissociation curve is shifted to the left indicating that haemoglobin binds oxygen more readily. This is due to the fact that 70% of the haemoglobin (Hb) is in the form of HbF; this gradually declines to negligible amounts by the age of 6 months.

The immature infant lung is also more vulnerable to insult. Following prolonged ventilation of a pre-term infant, bronchopulmonary dysplasia may cause oxygen dependence. Many infants who have suffered from bronchiolitis remain ‘chesty’ for a year or more. Table 2.2 shows respiratory rate at rest by age.

Table 2.2 Respiratory rate at rest by age.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Respiratory rate (breaths/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>30–40</td>
</tr>
<tr>
<td>1–2</td>
<td>25–35</td>
</tr>
<tr>
<td>2–5</td>
<td>25–30</td>
</tr>
<tr>
<td>5–12</td>
<td>20–25</td>
</tr>
<tr>
<td>&gt;12</td>
<td>15–20</td>
</tr>
</tbody>
</table>

Cardiovascular factors
The infant has a relatively small stroke volume (1.5 mL/kg at birth) but has the highest cardiac index (cardiac output relative to surface area) seen at any stage of life (300 mL/min/kg). Cardiac index decreases with age and is 100 mL/min/kg in adolescence and 70–80 mL/min/kg in the adult. Heart rate decreases with age (Table 2.3). Stroke volume increases with age as the heart gets bigger.

Table 2.3 Heart rate at rest by age.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Heart rate (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>110–160</td>
</tr>
<tr>
<td>1–2</td>
<td>100–150</td>
</tr>
<tr>
<td>2–5</td>
<td>95–140</td>
</tr>
<tr>
<td>5–12</td>
<td>80–120</td>
</tr>
<tr>
<td>&gt;12</td>
<td>60–100</td>
</tr>
</tbody>
</table>
Normal systolic blood pressures are shown in Table 2.4. Expected systolic blood pressure (BP) can be estimated by the following formula: 85 + (age in years x 2) for the 50th centile. BP varies within any age group by height and these values are for the average height at that age.

As the stroke volume is small and relatively fixed in infants, cardiac output is directly related to heart rate. The practical importance of this is that the response to volume therapy is blunted because stroke volume cannot increase greatly to improve cardiac output. By the age of 2 years myocardial function and response to fluid are similar to those of an adult.

Systemic vascular resistance rises after birth and continues to do so until adulthood is reached. This is reflected in the changes seen in blood pressure (Table 2.4).

### Immune function
At birth the immune system is immature and, consequently, babies are more susceptible than older children to many infections such as bronchiolitis, sepsicaemia, meningitis and urinary tract infections. Maternal antibodies acquired across the placenta provide some early protection but these progressively decline during the first 6 months. They are replaced slowly by the infant’s antibodies as he or she grows older. Breastfeeding provides some protection against respiratory and gastrointestinal infections.

### 2.5 PSYCHOLOGICAL DIFFERENCES
Children vary enormously in their intellectual ability and their emotional response. A knowledge of child development assists in understanding a child’s behaviour and in formulating an appropriate management strategy. Particular challenges exist in communicating with children and as far as possible easing their fear of the circumstances they find themselves in.

#### Communication
Infants and young children either have no language ability or are still developing their speech. This causes difficulty when symptoms such as pain need to be described. Even children who are usually fluent may remain silent. Information has to be gleaned from the limited verbal communication and from the many non-verbal cues (such as facial expression and posture) that are available. Older children are more likely to understand aspects of their illness and treatment and so be reassured by adequate age-appropriate communication.

#### Fear
Many emergency situations, and many other situations that adults would not classify as emergencies, engender fear in children. This causes additional distress to the child and adds to parental anxiety. Physiological parameters, such as pulse rate and respiratory rate, are often raised because of it, and this in turn makes clinical assessment of pathological processes such as shock, more difficult.

Fear is a particular problem in the pre-school child who often has a ‘magical’ concept of illness and injury. This means that the child may think that the problem has been caused by