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# Australian paediatric hyperbaric oxygen therapy 1998–2011

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SUMMARY

For a large number of ischaemic, infective, inflammatory or traumatic conditions, hyperbaric oxygen therapy is either the only treatment or an adjunct that significantly reduces morbidity and mortality. The primary aim of this review is to identify clinical conditions treated in a paediatric population referred to Australian hyperbaric units. Secondary aims are to describe outcomes of treatment and detail any complications occurring during treatment or during transfer between units.

This was a retrospective cohort study (January 1998–December 2011) of children treated at four Australian hyperbaric medical units. A total of 112 children underwent 1099 hyperbaric treatments for 14 indications. Ages were not normally distributed with a median age of 14 years (interquartile range 11–16; range 0.25–16 years). Treatments were completed as planned in 81.5% of cases with 25 patients' treatment terminated at the request of physicians, parents or patients. Complications relating to hyperbaric oxygen therapy occurred in 58 treatments (5.3%). Central nervous system oxygen toxicity occurred in 1:366 treatments. Our findings indicate that provision of hyperbaric oxygen therapy to children is feasible in major regional hyperbaric units and is associated with low complication rates. Management of children in an adult hyperbaric facility, however, requires significant cooperation between paediatric, intensive care and hyperbaric consultants, as the need for transfer to another hospital and prolonged transports often impacts on optimal ongoing surgical and intensive care management.

Key Words: hyperbaric oxygenation, child, infant, intensive care unit, paediatric, complications, treatment outcomes

Hyperbaric oxygen therapy (HBOT) is the primary medical treatment for decompression sickness and arterial gas embolism and is also an adjunct treatment for necrotising soft tissue infections, clostridial myonecrosis (gas gangrene), necrotising fasciitis, acute traumatic ischaemias, chronic diabetic and non-diabetic hypoxic problem wounds, chronic refractory osteomyelitis, prophylaxis for prevention of osteoradionecrosis, radiation tissue damage, compromised skin grafts and some types of intracranial abscesses. The Undersea and Hyperbaric Medical Society defines HBOT as "a treatment in which a patient breathes 100% oxygen while inside a treatment chamber at a pressure higher than sea level pressure (i.e. >1 atmosphere absolute [ATA])"[1]. Hyperbaric chambers are typically operated at pressures above 2 ATA for periods of 60–120 minutes per treatment session. Such doses of oxygen have a number of beneficial biochemical, cellular and physiologic effects.

Paediatric patients are treated infrequently at hyperbaric units in Australia due to a number of factors. First, Australian hyperbaric medical units are located in adult hospitals and treatment of children often requires inter-hospital transfer. Second, there are very few paediatric consultants with appropriate hyperbaric experience and third, there are few published series to guide paediatricians, neonatologists and paediatric intensivists in balancing the risks of transferring a critically ill child with the benefits of hyperbaric oxygen therapy. In addition, there is a perception that hyperbaric oxygen therapy is associated with significant risk of barotrauma related side-effects. This may also have limited the initial referral of children for treatment[2].

The primary aim of this review is to identify clinical conditions treated in paediatric patients referred to

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TABLE 1
*Indications, demographics and number of treatments for children presenting to Australian hyperbaric units*

| Indication | n | Age, y (SD) | Gender, male:female | Treatments (SD) |
|---|---|---|---|---|
| *'Bubble injury'* | | | | |
| Decompression illness | 9 | 15.1 (0.6) | 8:1 | 4.1 (4.3) |
| Cerebral arterial gas embolism | 4 | 7.6 (8.5) | 3:1 | 1.6 (0.9) |
| *Acute arterial ischaemia* | | | | |
| Compartment syndrome | 13 | 12.7 (4.3) | 6:5 | 7.2 (3.5) |
| Crush injury | 14 | 12.4 (4.2) | 11:5 | 7.2 (7.0) |
| Iatrogenic | 7 | 12.1 (2.1) | 4:3 | 11.5 (8.9) |
| *Acute severe infection* | | | | |
| Clostridial infection | 4 | 11.3 (6.8) | 4:0 | 4.5 (2.4) |
| Necrotising fasciitis | 10 | 12.1 (3.7) | 6:5 | 8.3 (10.5) |
| Fungal sepsis | 3 | 15 (0) | 1:1 | 8 (8.5) |
| Purpura fulminans | 3 | 10.6 (8.5) | 1:2 | 13.7 (12.4) |
| CO poisoning | 9 | 9.1 (5.4) | 7:2 | 1.7 (0.7) |
| *Non-healing wounds* | | | | |
| Surgical wounds | 6 | 13 (4.6) | 4:2 | 11.6 (9.9) |
| Mycobacterium ulcerans | 2 | 13.5 (0.7) | 2:0 | 14.5 (6.4) |
| Crohn's disease | 3 | 16.3 (0.3) | 2:1 | 17 (14.7) |
| *Radiation-induced injury* | | | | |
| Radiation soft tissue injury | 8 | 13.5 (1.9) | 2:6 | 30.4 (13.2) |
| *Refractory osteomyelitis* | 13 | 12.9 (1.8) | 10:2 | 11.2 (10.4) |
| *Miscellaneous* | | | | |
| Cerebral palsy | 1 | 14 (0) | 1:0 | 20 (0) |
| Optic neuropathy | 1 | 7 (0) | 0:1 | 3 (0) |
| Chronic regional pain sysndrome | 1 | 16 (0) | 0:1 | 33 (0) |
| Oxycephalus | 1 | 0.5 (0) | 0:1 | 1 (0) |
| *Total* | 112 | 12.6 (4.3) | 72:39 | 11.6 (10.7) |

y=years, SD=standard deviation, CO=carbon monoxide.

Australian hyperbaric units. Secondary aims are to describe outcomes of treatment and to determine which complications are specifically related to treatment of children in adult hyperbaric units and which are a consequence of their underlying clinical condition.

There are 15 hyperbaric facilities in Australia: eight in major public hospitals, five in private facilities and two in military facilities. Most patients, and especially critically ill patients, are treated in multi-place recompression chambers with a limited number of treatments being carried out in single occupant (mono-place) hyperbaric oxygen chambers.

## METHODS

Approvals from four hospitals' human ethics research committees were obtained (Alfred HREC 372/09, Prince of Wales Hospital HREC 10/178, Wesley Hospital HREC 2012-07-46 and Fremantle HREC 10/190). Following approvals, a retrospective study of consecutive patients aged 16 and under, treated in Australian hyperbaric units from July 1998 to December 2011, was undertaken. Cases were identified by searching the databases of participating hyperbaric facilities using age at presentation as a criterion for inclusion. Data collected included demographic details, clinical indications for HBOT, hyperbaric treatment tables used and complications during either treatment or transfer. Missing or incomplete data were obtained from case notes and hospital pathology databases. A review of each patient's notes was undertaken to determine clinical characteristics, sites of infection, causative pathogens, comorbidities, clinical management and

outcome not recorded on the hyperbaric database. Data were collected for each indication to determine precipitating events, disease severity, ancillary therapy used and complications. Primary outcomes recorded were survival with complete resolution of the condition, survival with minor morbidity (repeated debridements or grafting), survival with major morbidity (major debridement or amputation), deaths and hyperbaric-related complications. A review-specific data extraction form was used so that the same data was extracted for each patient and so missing data were clearly apparent. These data were then exported to an Excel spreadsheet for comparison and analysis.

Continuous data were described as either mean (standard deviation) or median (interquartile range) if not normally distributed. Descriptive statistics alone are presented. All analyses were performed using the statistical package STATA (College Station Texas, USA).

## RESULTS

Four hyperbaric units provided data on the children they treated. Three other hyperbaric units indicated they had not treated children in the timeframe of this study, and the five private and two military units indicated they had never treated children.

From 1998–2011, 112 children aged 16 years of age and under were treated with HBOT for a range of conditions (Table 1). The median age was 14 years (interquartile range 11–16; range 0.25–16 years). Treatment tables used were identical to those used in an adult population with the same condition. Decompression illness and cerebral arterial gas embolism were treated with a Royal Navy table 62, which involves treatment at a maximum pressure of 2.8 ATA for four hours and 45 minutes. Follow-up treatment was with a Royal Navy table 61 (2.8 ATA for two hours and 15 minutes). Clostridial myonecrosis was initially treated with a 3 ATA table for two hours and 11 minutes in one patient and a 2.8 ATA table in three other patients. Acute necrotising fasciitis, crush injury and compartment syndromes, including purpura fulminans, were treated with a 2.8 ATA table for one hour and 35 minutes. Radiotherapy-induced soft tissue injury, osteoradionecrosis, osteomyelitis and complex regional pain syndrome were all treated with a 2.4 ATA table for one hour and 40 minutes. One patient with oxycephalus received a single 2.8 ATA treatment using heliox (80/20 $He/O_2$) for one hour and 35 minutes.

The 112 patients received a total of 1099 treatment sessions (mean 9.3, standard deviation 11, 95% confidence interval 7.3–11.3). In this series, 93 patients (81.5%) successfully completed treatment, with 19 patients having treatments terminated prematurely by patients, parents or referring clinicians. Of the six patients whose treatments were terminated by referring clinicians, five were ceased because the clinicians decided that hyperbaric treatment was impeding medical and surgical management. One child with optic neuropathy demonstrated a sudden deterioration in eyesight following a third treatment and HBOT was ceased by the ophthalmologist. Of the four patient-generated early cessation of treatment, two children became acutely unwell and could not complete HBOT, one child with muscular dystrophy did not tolerate therapy due to respiratory compromise and one was ceased due to aggressive patient behaviour during treatment. Seven patients withdrew from treatment because of increasing anxiety and claustrophobia during treatment. For two patients a parent requested termination of treatment.

Thirteen children were treated while still receiving ventilation support. Eight were referred from a paediatric intensive care unit and five from an adult intensive care unit. During the period 1998–2011, a total of 18,284 patients received 300,443 treatments in Australian hyperbaric units[3]. Children 16 years of age and under therefore represented 0.64% of patients treated and 0.36% of all treatments.

### *Indications*

Indications for treatment were classified into eight broad categories ('bubble injury'), acute arterial ischaemia, acute severe infection, non-healing wounds, carbon monoxide (CO) poisoning, radiation-induced soft tissue injury, refractory osteomyelitis and miscellaneous (Table 1). Thirty-four children (30.4%) were treated for acute ischaemia (27.7% of total treatments). This included 11 compartment syndromes, 16 crush injuries, four ischaemic surgical flaps, one dogbite-induced femoral artery ischaemia, one severe frostbite and one arterial ischaemia secondary to iatrogenic alcohol injection during an interventional radiology procedure.

There were 20 cases of acute severe infection including ten necrotising soft tissue infections, four clostridial infections, three meningococcal purpura fulminans and three systemic fungal infections. This represents 17.8% of patients and 13.4% of treatments. Ten paediatric patients were treated for necrotising fasciitis. There were seven patients with type 1 (polymicrobial) necrotising fasciitis and three patients with type 2 (monomicrobial or group A streptococcal) necrotising fasciitis. Precipitating events included acute lymphocytic leukaemia with neutropenia (four

cases), acute myeloid leukaemia (one case), spider bite (two cases) and trauma (three cases). The infective organism was *Pseudomonas aeruginosa* in six cases; group A beta haemolytic streptococcus in three cases and *Staphylococcus aureus* with toxic shock syndrome in one case. Four patients were treated for clostridial infection including clostridial myonecrosis of the abdominal wall, clostridial myonecrosis of the thigh and gluteal muscles, clostridial cellulitis of the knee and enterocolitis necrotans of the jejunum (Pigbel). Organisms isolated were *Clostridium perfringens* type A (two), *Clostridium septicum* (one) and *Clostridium perfringens* type C. Invasive fungal sepsis occurred in three patients involving the jejunum, lungs and orbit. The causative agent was *Mucor indicus zygomycosis* (one intra-abdominal mucor, one rhinocerebral) and one *aspergillus* and *rhizopus* pneumonia.

Three patients with severe meningococcal disease with significant peripheral arterial compromise (purpura fulminans) were transferred for treatment. The causative organism was *Neisseria meningitidis* serotype B (two patients) and type C (one patient).

Bubble injury was the indication in 11.6% of patients and 3.8% of treatments. Included in this group were nine teenagers treated for decompression illness and four patients treated for cerebral arterial gas embolism. Cerebral arterial gas embolism occurred during cardiac surgery in two patients,

TABLE 2
*Outcomes of hyperbaric oxygen therapy by indication*

| Indications | n | Complete recovery | Recovery with minor disability | Recovery with major disability | Death |
|---|---|---|---|---|---|
| *'Bubble injury'* | | | | | |
| Decompression illness | 9 | 9 | | | |
| Cerebral arterial gas embolism | 4 | 4 | | | |
| *Acute arterial ischaemia* | | | | | |
| Compartment syndrome | 13 | 4 | 7 | 2 | |
| Crush injury | 14 | 5 | 8 | 1 | |
| Iatrogenic | 7 | 6 | 1 | | |
| *Acute severe infection* | | | | | |
| Clostridial infection | 4 | 2 | 2 | | |
| Necrotising fasciitis | 10 | 2 | 5 | 3 | |
| Fungal sepsis | 3 | | | 2 | 1 |
| Purpura fulminans | 3 | | | 3 | |
| CO poisoning | 9 | 9 | | | |
| *Non-healing wounds* | | | | | |
| Surgical wounds | 6 | 4 | 2 | | |
| Mycobacterium ulcerans | 2 | 2 | | | |
| Crohn's disease | 3 | 3 | | | |
| *Radiation-induced injury* | | | | | |
| Radiation soft tissue injury | 8 | 6 | 2 | | |
| *Miscellaneous* | | | | | |
| Refractory osteomyelitis | 13 | 10 | 3 | | |
| Cerebral palsy | 1 | | | | |
| Optic neuropathy | 1 | | 1 | | |
| Chronic regional pain syndrome | 1 | | 1 | | |
| Oxycephalus | 1 | 1 | | | |
| *Total* | 112 | 67(59.8%) | 34(30.4%) | 9(8%) | 1(0.9%) |

Major disability is defined as persistent neurological injury, major debridement or major amputation. Minor disability is defined as fasciotomy, escharotomy, minor amputation or skin grafting. Data are patient numbers and percentage of total. The one death occurred as a complication of fungal sepsis in an immune-compromised patient and was not thought to be related to hyperbaric therapy. CO=carbon monoxide.

following damage to a central venous catheter in one and following scuba diving, also in one. Twelve patients were treated for osteomyelitis involving the femur (eight cases), pelvis (one case), foot (one case), humerus (one case) and mandible (one case). Eleven patients were treated for chronic non-healing wounds following failure of conventional surgical approaches and multi-pronged antibiotic therapy. This included six surgical wounds, two patients with biopsy proven *Mycobacterium ulcerans* (Bairnsdale ulcer) and three patients with severe perianal fistulous Crohn's disease.

Nine children were treated for CO poisoning including four members of the same family who were retrieved from a bogged four wheel drive vehicle. All were treated with 100% oxygen at the scene and had no overt neurologic deficit after HBOT treatment. Four patients (7.4% of patients and 20.4% of treatments) were treated for radiation soft tissue injury (two wound breakdowns, one vesico-vaginal fistula and one bladder perforation). Two patients were treated as prophylaxis for osteoradionecrosis of the mandible and two patients for avascular necrosis of the femur or tibia secondary to dexamethasone chemotherapy for acute myeloid leukaemia.

Four miscellaneous indications were reported. One patient with Leber's optic neuropathy and deteriorating vision developed a sudden deterioration in eyesight following the third treatment. Hyperbaric treatment was ceased and a review by the ophthalmologist involved at a three month check-up demonstrated no further deterioration or slight visual improvement. One patient with cerebral palsy received 20 HBO treatments without any objective improvement in neurodevelopmental state. One patient with complex regional pain syndrome (complex regional pain syndrome type 1) involving the left leg underwent 33 treatments following failure of amitriptyline, gabapentin, pregabalin, lumbar plexus blocks, intravenous guanethidine block and behavioural modification techniques. One infant developed oxycephalus after a nasal oxygen catheter placed after recovery from congenital cardiac surgery was found to have traversed the cribriform plate and deposited a large oxygen bubble in the anterior cranial fossa.

*Outcomes*

Outcomes of hyperbaric treatment are detailed in Table 2. Complete functional recovery occurred in 67 (59.8%) cases. Minor disability secondary to fasciotomy, escharotomy, minor amputation or skin grafting occurred in 34 (30.4%) cases. Major disability including persistent neurological injury, major debridement or major amputation occurred in nine (8%) cases. Major and minor disability was considered to have occurred as a result of the underlying disease process or surgical procedures necessary to control the progression of injury. There were no deaths during hyperbaric treatment, but one child died of complications of fungal pneumonia prior to completion of hyperbaric therapy.

*Complications*

Hyperbaric side-effects were defined as those events specifically related to increased atmospheric pressure and/or oxygen concentrations. There were 58 adverse events in 1099 treatments (5.3%). Adverse events occurring during HBOT therapy included four Teed grade[4] I or II middle ear barotraumas (0.4%), 22 episodes of anxiety (2.0%), three oxygen toxicity convulsions (0.3%), three pulmonary oxygen toxicity events (0.3%), 18 episodes of nausea (1.6%) three episodes of progressive hypoxaemia (0.3%), four episodes of brief hypotension (0.4%) and one episode of symptomatic hypoglycaemia. No hyperbaric side-effects caused ongoing morbidity or disability. Prophylactic myringotomies were performed in 13 patients (11.6%) prior to treatment.

Three oxygen-induced convulsions occurred (an incidence of 1:366 treatments). The first occurred in a patient with necrotising fasciitis of the thigh during the third HBOT treatment at 2.8 ATA. The second occurred during the second treatment at 2.4 ATA in a patient with a chronic non-healing facial wound overlying a large arteriovenous malformation. The third occurred during the first treatment at 2.4 ATA in a 14-year-old boy with a lower limb crush injury.

## DISCUSSION

The most common indications for paediatric hyperbaric oxygen in Australian hyperbaric units were acute arterial ischaemia, intravascular air, acute severe infection, CO poisoning, chronic wounds and chronic osteomyelitis. These indications are consistent with indications approved by the Hyperbaric Oxygen Therapy Committee of the Undersea and Hyperbaric Medical Society. The Undersea and Hyperbaric Medical Society approved use of hyperbaric oxygen therapy in decompression illness, air or gas embolism, clostridial myositis and myonecrosis, necrotising soft tissue infections, CO poisoning, crush injury, compartment syndrome and other acute traumatic ischaemias, arterial insufficiencies, enhancement of healing in selected problem wounds, compromised grafts and flaps, severe anaemia, intracranial abscess, refractory osteomyelitis, delayed radiation soft tissue injury and acute thermal burn injury[1]. There are no randomised controlled trials in children of hyperbaric oxygen supporting any of

the above conditions; however, referral for treatment was based on physiological principles taking into account evidence from adult trials.

The conditions treated in this series are significantly different to those described in two other series of children referred for hyperbaric oxygen therapy. Waisman et al[5] reported on 139 paediatric patients aged two months to 18 years of whom 79% were treated for acute CO poisoning and 9.2% for traumatic ischaemia. Keenan et al[6] reported a case series of 32 children aged between three days and 11 years of whom 66% were treated for necrotising infections, 28% for CO poisoning and 6% for iatrogenic arterial air embolism.

Hyperbaric oxygen therapy has an important role in the management of acute severe infections, especially clostridial myonecrosis and necrotising fasciitis[7,8]. The primary mechanisms of hyperbaric oxygen in acute severe infections include restoration of normoxia or achievement of hyperoxia in previously hypoxic tissues, inhibition of exotoxin production, enhancement of neutrophil function and synergistic enhancement of antibiotic activity[9]. Necrotising fasciitis is rare in childhood and is often caused by group A beta haemolytic streptococci but also by other aerobic and anaerobic bacteria including clostridia, pseudomonas, *Bacteroides fragilis, Staphylococcus aureus, Enterobacter cloacae* and *Klebsiella pneumoniae*. Successful treatment of necrotising fasciitis with hyperbaric oxygen in children has been reported in four small series[10–13]. Johnston et al reported seven cases of necrotising fasciitis with myonecrosis in neutropenic paediatric oncology patients and demonstrated a synergistic effect of surgical debridement, HBOT and granulocyte colony stimulating factor[10]. Golger and Brogan reported low mortality rates in children with group A streptococcal necrotising fasciitis secondary to varicella[11,13]. For clostridial myositis and myonecrosis (gas gangrene) or spreading clostridial cellulitis with systemic toxicity the preferred treatment is a combination of hyperbaric oxygen, surgery and antibiotics. At 2.8–3 ATA hyperbaric oxygen produces tissue $pO_2$ values of 250–300 mmHg[14]. These tissue values are bacteriostatic to clostridia and also impedes α-toxin production[15]. Three clinical presentations of clostridial disease (clostridial myonecrosis, clostridial cellulitis and enterocolitis necrotans) were demonstrated in our series[16]. The incidence of *Clostridium perfringens* gas gangrene in children in Australia and New Zealand is unknown but presumed to be rare. In a ten year period (1971–1981), Unsworth[17] reported 73 patients (22% mortality) with gas gangrene of which 17 cases were less than 19 years of age, with two less than two years of age. Hyperbaric oxygen therapy has been reported as effective in treating clostridial infection or gas gangrene in paediatric case reports after penetrating injury to the head and neck (three cases) and spontaneous clostridial myonecrosis (three cases)[18–23]. Spontaneous clostridial myonecrosis is usually associated with *Clostridium septicum* infection and often has a markedly fulminant nature despite intervention due to its association with malignancy and immunological abnormalities. Enteritis necroticans (Pigbel) is caused by the beta toxin produced by *Clostridium perfringens* type C. It is an often fatal illness characterised by haemorrhagic, inflammatory or ischaemic necrosis of the jejunum.

Purpura fulminans is a life-threatening disease of children characterised by progressive purpuric lesions of the skin, primarily on the lower limbs, that eventually become necrotic[24]. Acute ischaemia can occur as a result of disseminated intravascular coagulation or secondary to compartment syndromes. Hyperbaric oxygen may be of benefit in purpura fulminans during the phase of acute ischaemia, during reperfusion and during the healing phase. The use of HBOT for paediatric purpura fulminans has been reported in seven case reports with a total of 23 patients[5,25–30]. In these case reports, limb salvage was possible in 21 of the 23 patients. In this series, hyperbaric oxygen was used in 34 cases of acute peripheral arterial ischaemia. The use of hyperbaric oxygen in compartment syndromes and crush injuries is supported by a number of clinical studies and one randomised controlled trial[24]. The mechanism of benefit in acute compartment syndrome is threefold[31]. First, HBOT has an osmotic effect to reduce tissue oedema. Second, HBOT inhibits neutrophil beta-2 integrin function and modifies endothelial dysfunction in ischaemia reperfusion injury. Third, HBOT influences wound healing by mobilising growth factors, growth factor receptors and endothelial stem cells from the bone marrow via induction of nitric oxide[9].

Hyperbaric oxygen is recommended for acute severe CO poisoning in all but one Australian hyperbaric unit[32–34]. Hyperbaric oxygen may improve outcome by reducing the binding of CO to haemoglobin, inhibiting pathological endothelial leukocyte adhesion, inhibiting lipid peroxidation, improving mitochondrial oxidative processes, reducing brain reperfusion injury and preventing intracranial hypertension. The half-life of carboxyhaemoglobin is reduced under hyperbaric conditions (249 minutes breathing air at 1 ATA, 47 minutes breathing oxygen at

1 ATA and 22 minutes at 2.5 ATA on 100% oxygen)[35,36]. Despite several randomised controlled trials and a systematic review of published papers[32,33,37,38], the issue remains contentious. The Alfred Hyperbaric Unit is the only unit which recommends normobaric oxygen rather than hyperbaric oxygen to treat patients with CO poisoning and has never treated childhood CO poisoning with HBOT.

The use of HBOT for paediatric perianal Crohn's disease has not previously been reported. The pathogenesis of Crohn's disease is thought to be a genetic susceptibility triggered by environmental factors. An excessive inflammatory response involving activated immune cells results in persistent inflammation and tissue injury. Hyperbaric oxygen therapy may control infection, restore tissue hypoxia, down-regulate inflammation and promote tissue repair.

Two children were diagnosed with *Mycobacterium ulcerans* skin ulcers. This condition is variously known as Bairnsdale ulcer, Daintree ulcer or Buruli ulcer and is due to elaboration of a lipid toxin, mycolactone, which causes necrosis of fat and subcutaneous tissue. There has only been one other report of the use of hyperbaric oxygen to treat *Mycobacterium ulcerans* wounds[39].

A number of uncommon indications were treated in this series including complex regional pain syndrome and oxycephalus. Treatment was based on clinical consensus and a physiological basis for action as no randomised controlled trial exists to support the use of HBOT in paediatrics. Kiralp et al reported benefit in adults with complex regional pain syndrome in a double-blind, randomised, placebo-controlled study[40]. The use of hyperbaric heliox to treat an oxygen or air pneumocephalus was based on the effect of hyperbaric pressure on counterdiffusion of inert gases. Hyldegaard et al[41] described a more rapid resolution of injected air bubbles in rat spinal white matter with a heliox (80:20) mixture than with 100% oxygen.

*Outcomes and complications*

Outcomes from our series are not directly comparable to two other paediatric case series[5,6]. Both these series were drawn from a different era (1980–1997) and had a different case mix. In a series by Waisman et al, 79% of children treated had CO poisoning. In contrast, Keenan et al's series consisted mainly of patients less than 12 years of age with necrotising soft tissue infection (66%) and CO poisoning (28%). Waisman et al's series were children under 18 years of age and reported complete recovery in 93% with two deaths (1.4%) and morbidity in 7.1% (amputation in six patients, persistent neurological injury in three patients)[5]. In contrast, Keenan et al reported that 43% of patients were considered normal at discharge, 37.5% had major morbidity as a result of their illness and treatment, and mortality was 12.5%[6]. The reported mortality rates for paediatric meningococcal sepsis-associated purpura fulminans is 23%[42], for necrotising fasciitis[18] is 25% and clostridial gas gangrene[11] is 25%. In our series, by contrast, there were no deaths in the 17 children with these illnesses.

The overall rate of complications in this series (5.3%) is low, with most complications being minor and self-limiting. The incidence of oxygen-induced seizures (1:366 treatments or 0.3%) in our series is consistent with the incidence reported in the two paediatric series but higher than the incidence of central nervous system toxicity in adults. The central nervous system toxicity incidence in adults treated at the Fremantle hyperbaric unit during the same time frame was 1:1651 treatments (0.06%), with a higher incidence at higher treatment pressures (1:339 at 2.8 ATA) and in patients treated for decompression illness (1:178 or 0.56%) and CO poisoning[15]. Waisman et al reported two episodes of oxygen toxicity; one pulmonary and one central nervous system[5]. In Keenan et al's series there were 47 complications or events occurring during HBOT therapy including hypotension (63%), bronchospasm (34%), haemotympanum (13%) and progressive hypoxaemia (6%)[6].

## CONCLUSIONS

In selected conditions, HBOT for children is a safe and effective therapeutic option. Complications related to hyperbaric oxygen therapy are largely related to the underlying disease process with barotrauma side-effects being uncommon. Management of children in an adult hyperbaric facility, however, requires significant co-operation between paediatric, intensive care and hyperbaric consultants as the need for transfer to another hospital and prolonged transports often impact on optimal ongoing surgical and intensive care management. The lack of well designed, randomised controlled paediatric studies is an impediment to our ability to quantify the benefit of hyperbaric oxygen in acute severe infections and should be an area of further investigation.

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