ORIGINAL ARTICLES

THE EFFECTIVENESS AND COST OF OXYGEN THERAPY FOR DIABETIC FOOT WOUNDS

Lyndsae Wheen

Summary

Diabetic foot ulcers present a major healing problem due to the combined effects of hypoxia and infection. The social and financial costs of the problem diabetic foot are substantial. Experimental work has proved that hyperbaric oxygen therapy will improve tissue oxygenation and thus wound healing. Oxygen also has direct and indirect effects in antisepsis. Clinical hyperbaric oxygen therapy has been shown to improve the healing of diabetic foot wounds significantly and to decrease the rate of consequent amputation. In addition, the financial costs involved are lessened when hyperbaric oxygen therapy is used in combination with conventional management of the problem diabetic foot. Further properly randomised and controlled prospective trials are, however, required to evaluate different oxygen therapy regimes and to obtain accurate cost-benefit data.

Introduction

Individuals with diabetes mellitus are predisposed to foot ulceration because of neuropathies, vascular disease and a susceptibility to infection. Loss of sensation from a peripheral neuropathy may lead to unrecognised foot trauma. Diabetic vascular disease slows healing and predisposes to infection. Infections are common in diabetics because of a combination of tissue hypoxia, resulting from ischaemia,[1] and the pathophysiology of diabetes. The latter has not been completely explained, but diabetics have often been shown to have impaired neutrophil phagocytosis and/or intracellular killing.[2]

The prevalence and cost of diabetic foot ulcers

Severe foot disease is present in about 10% of diabetics, with gangrene resulting in one third of these.[3] A retrospective survey of hospitalised diabetics with foot lesions showed that 34% of the patients required a major amputation.[4] Diabetic foot problems account for an average of 44% (range 26-56%) of all amputations.[5-9] Amputations occur at a rate of 59.7/10,000 diabetics/year[7] or 12.8/100,000 individuals/year.[9] Although Swedish investigators have claimed that the risk of amputation in diabetics is no different from that of non-diabetics,[9,10] this is contrary to other surveys. In North America the risk of amputation is 15 times greater in diabetics.[7]

The level of amputation in diabetics is usually below the knee,[6,10] perhaps due to the predominantly small vessel disease that is seen in diabetes. Although diabetes as such does not influence the morbidity or the mortality associated with amputation,[9] the level of amputation affects the rehabilitation and the consequent mortality. Both are improved with lower level amputations.[5,6,8,10]

In the United States of America (USA) the mean hospital stay for amputations is 29.6 days,[7] but in Denmark it is 68 days overall and 81 days for below knee amputations.[5] This difference may perhaps be explained by the different health funding policies in these countries. The frequency of successful healing of below knee amputations also varies between 39% in Sweden[9] and 79% in the USA.[6] A below knee amputation requires subsequent conversion to an above knee amputation in 22% of cases.[8] A survey of amputees in the USA showed that only 77% were mentally and physically fit enough to be given a prosthesis and, of these, successful rehabilitation was achieved in only 90% of unilateral below knee amputees.[8] The success of rehabilitation dropped to 76% with unilateral above knee amputees, to 40% in those who had needed a below knee to above knee conversion and to 45% among all bilateral amputees.

The mortality associated with amputation is also high. In-hospital mortality is between 11 and 13% in the USA[6,8] and 18% in Denmark.[5] The survival of all amputees drops to between 46 and 49% after three years and to between 30 and 31% after five years.[5,8] The incidence of subsequent amputation of the opposite leg varies from between 25 and 33% in Sweden,[9,10] to 45% in the USA.[4]

It is clear that the morbidity and mortality rates of diabetic foot problems are high because of the frequency at with they occur and their common progression to amputation. Consequently, there are associated significant monetary costs. The cost of care of a patient with an infected ulcer requiring prolonged hospitalisation and eventual minor amputation (of three toes) was US$50,000 in 1988.[11] A prospective study of the costs of care of patients with limb-threatening ischaemia treated with vascular reconstruction or amputation in a hospital in the USA in 19781981, with follow-up through 1982, reported mean hospital costs of US$28,374 for successful vascular reconstruction, US$40,563 for primary amputation and US$50,809 for failed reconstruction.[12]

The costs of care of diabetic foot ulcers cannot be considered in context unless the prevalence of diabetes is described. In the USA, it is estimated that 6% of the population have diagnosed diabetes and that another 6% have undiagnosed diabetes.[13] Although the overall

incidence of diagnosed diabetes in New Zealand (NZ), estimated at 3% (personal communication, NZ Department of Statistics), is lower than that in the USA, diabetes and its complications are of importance and cost in New Zealand, particularly in the non-European populations. The prevalence of diabetes in Maori, Pacific Island and Asian populations is up to five times that of a similar European population (9.9%, 8.9%, 7.5% and 1.9% respectively).[14] A study of diabetic admissions to Middlemore Hospital in Auckland, New Zealand, during 1987, showed that 49 of the 357 (14%) diabetic patients were admitted for foot disease.[15] These 49 patients accounted for 64 separate admissions, of which 35 (55%) were for foot ulcers and 14 (22%) were for gangrene. The mean length of stay was 33 days, the amputation rate was 31%, the in-patient mortality was 12% and only 64% of the patients had survived two years later. Of the survivors, 24% had undergone further amputations and half of them had become bilateral amputees. The average hospital cost per patient was reported to be NZ$ 12,500.

The significance of hypoxia in diabetic foot wounds

Diabetic foot ulcers are often refractory to conventional treatment because diabetics heal poorly and have both a compromised immune response to infection and inadequate peripheral perfusion. Poor peripheral perfusion and infection will both lead to tissue hypoxia which is associated with poor wound healing.[16,17] Both macrovascular and microvascular diabetic disease leads to hypoperfusion of the capillary bed.[18] Macrovascular disease (atherosclerotic narrowing) causes ischaemia due to decreased blood perfusion. In diabetics, atherosclerosis is typically peripheral, occurring in the tibial and pedal vessels,[18] thus tissue perfusion is least in the foot. Increased arteriovenous shunting has also been seen in diabetics with neuropathic foot ulcers. This is thought to result from a local sympathetic component of the autonomic neuropathy caused by microvascular disease. The increased arteriovenous shunting leads to redirection of blood away from the skin such that the venous oxygen tension approaches that of arterial blood and so the arterio-venous oxygen gradient is greatly reduced.[19]

In the diabetic person there are several mechanisms by which infection and hypoxia are linked. Microvascular disease is seen as thickening of the basement membrane and functional abnormalities which interfere with the transfer of nutrients and the migration of leucocytes out of the capillary.[20] Thus infection control is diminished. Diabetes also leads to decreased resistance to infection via, at least in part, neutrophil dysfunction.[2,21] Infection causes increased oxygen consumption because of the metabolic requirements for oxygen of aerobic micro-organisms and the oxygen dependant killing of certain micro-organisms by neutrophils. This compounds the local hypoxia and predisposes to further infection.[22] Hypoxia decreases resistance to both aerobic and anaerobic infections. In vitro studies of splenic macrophages have shown decreased phagocytosis in hypoxia.[23] Human polymorphonuclear leucocyte (PMN) phagocytosis and killing of certain aerobes (*Staphylococcus aureus, E coli, Klebsiella* sp, *Proteus* sp and *Salmonella* sp) in culture is markedly reduced in hypoxic conditions, but that of other aerobes (*Streptococcus epidermidis,* viridans Streptococci, enterococci and *Pseudomonas aeruginosa*) and anaerobes (peptostreptococci, *Bacteroides fragilis* and *Clostridium perfringens*) is unaffected.[24] Similar inhibition of *S aureus* killing by rabbit PMNs occurs in hypoxia.[25] Rats exposed to hypobaric hypoxia show reduced PMN neutrophilic granulocyte phagocytosis.[26] In addition, an hypoxic environment is ideal for the growth of anaerobic organisms. Hypoxia also affects the action of the aminoglycoside class of antibiotics which require oxygen for their uptake by bacteria. A member of this class, tobramycin, has been shown to be ineffective at killing *P aeruginosa* in an anaerobic environment.[27] The activity of other antibiotics which need oxygen for their action, such as nitrofurantoin[28] and vancomycin,[29] may also be diminished in hypoxic conditions.

Hypoxia, at the levels frequently measured transcutaneously in the non-healing diabetic foot ulcer,[1] retards wound healing. The initiating events in wound healing are coagulation, inflammation and local hypoxia. These are followed by macrophage migration to the wound and phagocytosis of debris and bacteria.[22] Diabetics appear to have an intrinsically decreased inflammatory response and leucocyte function.[30] Hypoxia results in a further reduction in phagocytosis by macrophages.[23,25] Although some investigators believe that macrophages release an "angiogenesis factor", which may be lactate, in response to hypoxia,[31] others argue that the oxygen gradient across the wound is the signal for angiogenesis.[32] Hypoxia appears to stimulate in vitro growth and sprouting activity of arterial endothelium.[33] Whichever stimulus predominates, wound healing continues by fibroblast proliferation with collagen synthesis by fibroblasts and capillary growth.[22] Fibroblasts and capillary buds move together across the wound as cross-linked collagen is established as a "scaffolding" for new capillaries. Hypoxia inhibits in vitro fibroblast proliferation and the hydroxylation of proline to hydroxyproline (procollagen).[34] The tensile strength of healing skin wounds is proportional to the collagen-hydroxyproline content and is seen to be lowered in hypoxia. [17]

The role of oxygen therapy in diabetic foot wounds

Correction of any hypoxia may improve otherwise slowed wound healing. Tissue oxygenation can only be improved by increasing the partial pressure of oxygen in the inspired gas and hence the arterial oxygen pressure.[35] The oxygen tension in non-healing wounds has been

measured at between 10 and 20 mm Hg, compared with an oxygen tension in normal tissue of between 40 and 52 mm Hg.[35,36] Breathing 100% oxygen at 1 atmosphere (bar) has been shown to raise the oxygen tension in such a wound from 19 mm Hg to 85 mm Hg. Administration of hyperbaric oxygen (HBO) to a series of patients with ischaemic and hypoxic wounds showed locally increased oxygen tension, from between 10 and 20 mm Hg to over 30 mm Hg.[36] The oxygen tension in infected bone (between 19 and 23 mm Hg) can be increased by HBO to a level (between 96 and 111 mm Hg) above that in normal bone (between 44 and 46 mm Hg).[25]

Raising the wound oxygen tension has beneficial effects on the local microcirculation. Studies of an experimentally induced compartment syndrome in dogs demonstrated that HBO reduced the oedema present.[37] Oedema, resulting from injured and ischaemic tissue, increases the diffusion distance from capillaries to cells. Diabetic microangiopathy also causes oedema because affected vessels show an abnormally increased permeability.[20] Between 1 and 2.5 bar, oxygen acts directly on blood vessels to cause vasoconstriction.[38] This lowers the transmural pressures acting across capillaries and so fluid leakage and oedema are reduced. It appears that at pressures above 2 or 2.5 bar, oxygen causes vasodilation.[38] In addition, the microcirculation is improved by the effect of hyperoxygenation on red blood cell rigidity. Red blood cell flexibility, measured as an ability to pass through a 3m filter, was shown to increase significantly with HBO.[39] Red cell deformability is important in decreasing the viscosity of the blood in the microcirculation.

The cellular events leading to wound healing are also improved with hyperoxygenation. In culture, maximal fibroblast proliferation occurs at a tissue oxygen tension of 80 mm Hg.[34] Tissue oxygen tensions above and below this level progressively decrease fibroblast growth. The hydroxyproline content, a measure of collagen synthesis, is also maximal at tissue oxygen tensions of 80 mm Hg.[34] Using an in vitro model of wound healing (fibroblast cells cultured in a chronic hypoxic environment), the effect of simulated clinical HBO treatments was assessed. The cultures were given a schedule of different HBO treatments for 90 minutes a day for four days.[40] Intermittent hyperoxia led to significantly increased numbers of fibroblasts from an hypoxic environment compared with those from a normoxic environment. The hydroxyproline content, however, was not significantly changed by this length of treatment.

The stimulus for new capillary growth appears to be either hypoxia as such, or the oxygen gradient across a wound. Neovascularisation and fibroblast proliferation are closely linked. Capillary growth is required to provide the perfusion, and thus the oxygen, for fibroblast proliferation and synthesis and cross-linking of collagen. The cross-linked collagen is required to form the scaffolding for the new capillary buds. Thus oxygen is required, at least indirectly, for the growth of new capillaries. The revascularisation of full thickness burn wounds in rats was significantly improved, both angiographically and histologically, by intermittent HBO.[41]

Hyperoxia also improves wound healing by its effects in infection control. Hyperoxia leads to increased intracellular and extracellular production of superoxide, hydrogen peroxide and other toxic oxygen radicals. These oxygen radicals are lethal for strict anaerobes, as these organisms lack the detoxifying enzymes superoxide dismutase and catalase.[29,42] Although some investigators have found that aerobic organisms (*S aureus, P aeruginosa, Candida albicans*) are able to detoxify the extra oxygen radicals formed in hyperoxic conditions,[46] others have shown decreased growth of some aerobes (*E coli, P aeruginosa*) in hyperoxic culture.[28] The number of viable *E coli* surviving in experimental wounds in guinea pigs is significantly decreased after two days of 45% inspired oxygen compared with room air (21% oxygen).[43] The in vitro and in vivo growth of *Vibrio* sp is decreased in hyperoxic conditions.[44] The size and number of experimental *Fusobacterium* sp and *Bacteroides* sp abscesses in mice are decreased with HBO.[45] Although some investigators have found that aerobic organisms (*S aureus, P aeruginosa, Candida albicans*) are able to detoxify the extra oxygen radicals formed in hyperoxic conditions,[46] others have shown decreased growth of some aerobes (*E Coli, P aeruginosa*) in hyperoxic culturre.[28]

Oxygen therapy also has indirect effects in infection control. As previously described, PMN bacterial killing is impaired in hypoxic conditions. PMNs use both oxygen-dependent and oxygen-independent mechanisms to kill micro-organisms. The oxygen-dependent killing is initiated by a respiratory burst, which produces superoxide and other potent oxygen radicals and oxidised halide ions, all of which are highly effective microbicidal agents.[29] A study of an experimental *S aureus* osteomyelitis showed that HBO increased PMN killing in osteomyelitic bone to, if not above, that seen in normal bone. HBO had no direct effect on *S aureus* survival.[25] In addition, hyperoxia potentiates the action of certain antibiotics (Table 1).

The mean inhibitory concentration (MIC) and the mean bactericidal concentration (MBC) of nitrofurantoin (which cycles through reduction and oxidation and so requires oxygen for its action) for *E coli* have both been shown to be significantly reduced by the concomitant administration of HBO.[28,47] Although the action of sulphamethoxazole against *E coli* is unchanged,[28,47] the MBC of another sulphonamide, sulphisoxazole, for *Vibrio anguillarum* is significantly reduced by HBO.[44] Hyperoxia potentiates the activity of trimethoprim against *E coli*[28] and *Vanguillarum.*[44] The synergistic action of trimethoprim and the sulphonamides is further enhanced by the use of

TABLE 1

EFFECT OF OXYGEN ON ANTIBIOTIC EFFICACY AGAINST SELECTED MICRO-ORGANISMS

Antibiotic	Organism	Effect of oxygen
nitrofurantoin	*E coli*	potentiation[28,47]
sulphamethoxazole	*E coli*	no change[28,47]
sulphisoxazole	*V anguillarum*	potentiation[44]
trimethoprim	*E coli*	potentiation[28]
	V anguillarum	potentiation[44]
sulphamethoxazole & trimethoprim	*E coli*	potentiation[28]
sulphisoxazole & trimethoprim	*V anguillarum*	potentiation[44]
cephalothin	*S aureus*	no change[28]
cephazolin	*S aureus*	potentiation[48]
gentamicin	*E coli*	no change[28]
	P aeruginosa	no change[28]
tobramycin	*E coli*	no change[28]
	P aeruginosa	no change[28] potentiation[47,49]

HB0.[28.44] Cephalosporins also have an antibiotic-specific response to HBO. The anti-*S aureus* effect of cephalothin is unchanged,[29] but that of cephazolin is enhanced by HBO.[48] Aminoglycosides have an oxygen-dependant uptake into bacterial cells; and although some studies indicate that hyperoxia has no effect on the action of tobramycin and gentamicin (against *E coli* and *P aeruginosa*),[28] others have shown that the in vitro and in vivo activity of tobramycin (against *P aeruginosa*) is potentiated.[27,49]

The clinical use of oxygen therapy for diabetic foot wounds

Many reports of the benefit of HBO therapy in the treatment of diabetic foot wounds now exist. Davis et al. presented an historical and pictorial series of seven diabetic patients whose ulcers and underlying osteomyelitis were healed or grafted using HBO.[1] A recent paper reported the successful use of HBO in a complex wound healing problem in a diabetic patient.[50] Barr and Perrins used HBO in the management of 24 diabetic patients with non-healing ulcers.[51] Over an average of about seven months, healing occurred in 67% and amputation was avoided in 18% of these patients. An Italian study showed that, in the treatment of diabetic gangrene, HBO therapy decreased the amputation rate from between 39 (from 1979 to 1982) and 33% (from 1983 to 1987) to only 5% (from 1983 to 1987).[52] Without HBO the healing rates of these major ulceronecrotic lesions dropped from 96% to 67%. Two reports of ten years' experience with HBO in the management of the problem diabetic foot have recently been published.[53,54] In one series, 151 patients with extensive ulceronecrotic lesions were treated with an average of 40 HBO sessions; 130 (86%) achieved complete healing or a more minor amputation than originally planned and only 12 (14%) had worsened clinical findings or a below knee or above knee amputation.[53] The second paper compares an HBO-treated group of 67 patients with a control group of 33 patients.[54] The healing rate was 80% and the amputation rate was 20% in the HBO-treated group, but in the control group the healing rate was only 40% and the amputation rate reached 60%.

There is, as yet, only one prospective and controlled, though not randomised, trial of HBO in the management of diabetic foot wounds.[55] This study included 28 patients, 23 with gangrene and 5 with a perforating ulcer. The treatment group of 18 patients showed a significantly increased healing rate (89% versus 10%) and a decreased amputation rate (11% versus 40%) compared with the control group of 10 patients. Interestingly, the mean hospitalisation period was 20 days shorter (62 versus 82 days) for those patients receiving HBO than those not receiving HBO.

HBO is most successfully used in conjunction with other more conventional measures such as surgical debridement, local wound care, appropriate antibiotic therapy and good metabolic control.[56,57] Not all diabetic patients with foot wounds will benefit from HBO therapy. Adequate peripheral perfusion is necessary, therefore vascular assessment (including palpation, Doppler evaluation of peripheral pulses, ankle perfusion pressure studies, angiography and transcutaneous oximetry) is crucial. Vascular surgery to bypass any large vessel occlusion may be required before HBO therapy can be of any use.[56] Ankle perfusion pressures in the range of 75 to 90 mm Hg are associated with healing with oxygen therapy.[58] Transcutaneous partial pressures of oxygen ($TcP0_2$) can be used to predict healing with HBO. Although some authors believe that only TcP02 measurements made during HBO treatment are predictive,[59] others argue that those made while breathing ambient air or 100% oxygen at ambient pressure can be used to predict outcome.[58] The ratio of wound to reference TcP02, measured in air, is particularly useful in predicting healing outcome: a ratio in the range of 0.20 to 0.85 indicates probable healing with oxygen therapy.[58]

The risks to the patient from HBO are those due to pressure and those due to oxygen. Overpressurisation of any gas trapped in a cavity such as the lungs, sinuses, ears and teeth, can result in barotrauma.[60] Oxygen at higher pressures than 0.21 bar is a toxic gas and so HBO therapy has the potential to produce toxic effects. The manifestations of oxygen toxicity are seen in the central nervous system at partial pressures of over 2 bar. Lung changes develop more slowly and at lower pressures. The risk of toxic effects from oxygen becomes greater as inspired partial pressures of oxygen and time are increased.[60] The

HBO treatment regimens used for wound healing (generally 100% oxygen at 2 to 3 bar for 1.5 to 2 hours daily) avoid serious and irreversible toxic and pressure effects.[61] None of the patients treated with HBO in the studies reviewed suffered from treatment-related problems.[51-55,62]

The costs of HBO therapy for diabetic foot wounds

Cianci et al. published an economic analysis of HBO therapy for problem foot wounds in the USA in 1988.[62] There were 19 diabetic patients in the study; 13 (68%) with limb-threatening lesions. To provide adequate peripheral perfusion for HBO therapy to be used, vascular surgery was required for 8 patients, 42% of total. These 8 were 62% of those with more serious disease. Patients with limb-threatening wounds had a longer mean hospital stay (42 days versus 35 days) and a greater mean number of HBO treatments (40 versus 38) than the group of diabetics as a whole. Successful salvage of the limb was achieved in 17 (89%) of all the diabetic patients and in 11 (85%) of those that had been potential amputees. The charges for those patients with more severe disease (mean HBO cost US$13,456 and mean total hospital charges US$40,697) were higher than the whole group average (mean HBO cost US$12,668 and mean total hospital charges US$34,370). These costs compare favourably with the cost of an acute amputation in 1986 (US$40,563).[12] Rehabilitation expenditures are an additional major cost following amputation. Cianci et al. report that the costs of rehabilitation approached US$40,000[62] and hence the total cost of amputation and rehabilitation in 1988 was in the range of US$50,000 to US$80,000.

Cost analysis of HBO and conventional management of diabetic foot wounds in New Zealand

In order to assess the comparative costs of HBO and conventional management of diabetic foot wounds in New Zealand, the trial of Baroni et al.[55] was used to provide patient outcomes and costed for New Zealand. Bed stay and amputation costs were obtained from a New Zealand public hospital. Information from a public hospital's occupational therapy and physiotherapy departments allowed only crude estimates of in-hospital rehabilitation costs to be made. The Artificial Limb Centre provided costs involved in prosthetic supply and training. Costs of prostheses and other equipment are based on those for a below knee amputation and for the first year only. Prostheses and crutches are usually replaced every year. Data from the Royal New Zealand Navy (RNZN) Hospital, which has the only Hyperbaric Unit in New Zealand, were used for bed stay and treatment costs for HBO therapy. The costs of vascular assessment, medical management of diabetes and daily debridement were not included as these procedures were carried out on both groups of patients.

The calculations show that average cost per patient would be significantly less for the group treated with HBO at the RNZN Hospital (NZ$10,565) than for the control group (NZ$38,359).

Average bed stay cost per treatment group patient (NZ$7,440) is approximately one-fifth that per control group patient (NZ$36,900). This is because of two factors. The first is the shorter hospitalisation period of the treatment group (62 days) compared with the control group (82 days). The second factor is the much lower (approximately one-quarter) bed stay cost of the RNZN hospital (NZ$120/day) than the public hospital (NZ$450/day). Even if the bed stay costs of the two hospitals were the same, the shorter period of hospitalisation in the treated group would result in a 19% saving. (Table 4)

The average per patient cost of amputation and first-year rehabilitation is NZ$405 for the treatment group and

TABLE 2

COSTS OF CONVENTIONAL MANAGEMENT OF DIABETIC GANGRENE IN NEW ZEALAND

Control Group 10 patients

Outcomes **Healed** 1 patient **Amputation** 4 patients **No change** 5 patients

Mean hospitalization period 82 days

Costing item	**Number**	**Cost/item (NZ$)**	**Total cost (NZ$)**
Bed stay	820 days	450	369,000
Theatre costs amputation	4	593	2,372
Occupational therapy input	4	113	452
Physiotherapy input	4	64	256
Walking frame	4	100	400
Crutches	8	89	712
Prosthesis supply and training	8	1,300	10,400
Total			**NZ$383,592**

Average cost per patient NZ$38,359

TABLE 3

COSTS OF HBO WITH CONVENTIONAL MANAGEMENT OF DIABETIC GANGRENE IN NEW ZEALAND BASED ON RNZN HOSPITAL BED DAY COSTS.

Treatment Group l8 patients

Outcomes **Healed** 16 patients **Amputation** 2 patients **No Change** None

Mean hospitalization period 62 days

Mean HBO treatments 34

Cost item	**Number**	**Cost/item (NZ$)**	**Total cost (NZ$)**
Bed stay	1,116 days	120	133,920
HBO treatment	612	80	48,960
Theatre costs amputation	2	593	1,186
Ocuupational therapy input	2	113	226
Physiotherapy input	2	64	128
Walking frame	2	100	200
Crutches	4	89	356
Prosthesis supply and training	4	1,300	5,200
Total			**NZ$190,176**

Average cost per patient NZ$10,565
Saving per patient NZ$27,794

TABLE 4

COSTS OF HBO WITH CONVENTIONAL MANAGEMENT OF DIABETIC GANGRENE IN NEW ZEALAND BASED ON PUBLIC HOSPITAL BED DAY COSTS.

Treatment Group l8 patients

Outcomes **Healed** 16 patients **Amputation** 2 patients **No Change** None

Mean hospitalization period 62 days

Mean HBO treatments 34

Cost item	**Number**	**Cost/item (NZ$)**	**Total cost (NZ$)**
Bed stay	1,116 days	450	502,200
HBO treatment	612	80	48,960
Theatre costs amputation	2	593	1,186
Ocuupational therapy input	2	113	226
Physiotherapy input	2	64	128
Walking frame	2	100	200
Crutches	4	89	356
Prosthesis supply and training	4	1,300	5,200
Total			**NZ$558,456**

Average cost per patient NZ$31,026
Saving per patient NZ$7,333

NZ$1,459 for the control group. This difference is due to the significantly lower amputation rate in the treatment group (1.1/10 patients) than in the control group (4/10 patients).

Despite the additional cost of HBO therapy (an average of NZ$2,720/patient) for the treatment group, the lower bed stay, amputation and rehabilitation costs result in lower total average cost per treatment group patient compared with the control group. These calculations, using known treatment outcomes,[55] and applying NZ hospital and HBO treatment costs, shows that HBO therapy is a cost-effective adjunctive treatment to the conventional management of diabetic foot wounds. Further properly randomised and controlled prospective trials are required to evaluate different oxygen therapy regimes and to obtain

accurate cost-benefit data.

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This paper is the thesis submitted for the South Pacific Underwater Medicine Society's Diploma of Diving and Hyperbaric Medicine, which was awarded to Dr Wheen.

Dr Lyndsae Wheen's address is 6 Wilson Road, Glen Eden, Auckland, New Zealand.

THE WORLD AS IT IS

SAFE LIMITS SYMPOSIUM

This meeting was was held in Cairns from 21/10/94 to 23/10/94 by the Diving Workplace Health and Safety Committee of the Queensland Department of Employment, Vocational Education, Training and Industrial Relations, which makes recommendations on workplace health and safety standards for the Queensland Diving Industry.

The symposium aims were to explore the health and safety implications for the Queensland diving industry of the risks associated with multiple dives during multiple days of diving, post-diving altitude exposure and of Resort Diving. From these discussions the participants were to produce conclusions which were to be internationally valid, relevant to Queensland and form a basis for recommendations by the Diving Industry Committee to the Division of Workplace Health and Safety.

It was the first time in Australia that Diving Doctors had met with representatives of the Recreational Diving Industry to discuss the problems of diving accidents, their frequency and the best ways to cope with making diving as safe a recreation as possible. Two full days of presentations were followed by some hours discussion to arrive at the conclusions. There was a remarkable degree of consensus about these in spite of the widely differing viewpoints from which diving safety was being discussed by the more than 100 registrants.

The Safe Limits symposium papers are available as a bound volume available, while stocks last, from

Ms Sylvie Munson, Council Secretariat,
Division of Workplace Health and Safety,
PO Box 69, Brisbane, Queensland 4001, Australia.

The SPUMS Journal has asked permission to reprint the symposium papers to bring this important initiative to the attention of the membership around the world. The papers will appear over the next year or so, depending on space available. The first one, the Official Summary appears in the adjacent column.

SAFE LIMITS: AN INTERNATIONAL DIVE SYMPOSIUM OVERVIEW AND CONCLUDING SESSION

Des Gorman

Introduction

A comprehensive range of subjects were discussed over the 2 days of formal symposium sessions. The most notable feature of this debate was the friendly context and the considerable consensus. This is noteworthy in that previous gatherings of this type have been recipes for "bun-fights".

The attendance at the Symposium was impressive in its breadth, Government Agencies (Queensland, Victoria, South and Western Australia were represented), commercial divers and their union, recreational divers and members of their support industries, "technical" recreational divers and medical practitioners (with delegates from all the Australian States and New Zealand and all of the members of the SPUMS, South Pacific Underwater Medicine Society, Executive).

Similarly, the presentations were of a general high standard and it is reasonable to conclude that the interest of delegates was sustained throughout the program.

Special appreciation must be expressed here for all the officers of the Queensland Government Division of Workplace Health and Safety involved in conduct of the symposium and in the preparation and introduction of the Code of Practice for Diving. The agreement at the concluding session of the symposium on the suitability of this Code to act as a template for the rest of Australasia is testimony to the merits of the Code and its authors.

This review of the concluding session of the symposium will address each of the 5 principal debating points and briefly mention other topics of concern.