Transcutaneous Oxygen Measurements Predict a Beneficial Response to Hyperbaric Oxygen Therapy in Patients with Nonhealing Wounds and Critical Limb Ischemia

RODNEY E. GROLMAN, M.D., DONALD K. WILKERSON, M.D., JAN TAYLOR, P.T., C.P., PETER ALLINSON, M.D., MICHAEL A. ZATINA, M.D.

From the Department of Surgery, St. Agnes HealthCare, Baltimore, Maryland

Hyperbaric oxygen (HBO) therapy may be a useful adjunct in the treatment of patients with wounds associated with critical limb ischemia. These patients either cannot undergo a successful bypass or may not heal after vascular reconstruction alone. Identification of patients likely to benefit from HBO is essential before treatment, as this therapy is time-consuming, costly, and not without risk. Transcutaneous oxygen measurements (TCOM) can be used to evaluate the degree of hypoxia in ischemic tissue. In this study we evaluated whether TCOM could be used to identify those patients who would or would not benefit from HBO therapy. Our hypothesis is that a difference in transcutaneous oxygen tension readings measured near the ischemic lesion with the patient breathing room air and while breathing 100 per cent oxygen at ambient pressure may be predictive of wound healing with adjunctive hyperbaric oxygen therapy. Thirty-six patients with critical limb ischemia and nonhealing ulcers were referred for HBO therapy. They were deemed either nonreconstructible from a vascular surgical viewpoint, had failed prior revascularization attempts, or could not achieve complete wound healing even after a successful revascularization. Pretreatment assessment included a room air and post-100 per cent-O_2 challenge TCOM reading obtained in the vicinity of the open wound. Hyperbaric oxygen treatments at 2.0 to 2.5 atm were then administered until healing occurred or failure was confirmed. All patients undergoing HBO had a baseline TCOM of <40 torr. Twenty-seven patients had an increase in TCOM of >10 torr with oxygen inhalation at initial evaluation. Of these patients, 19 (70%) healed their wounds with HBO therapy. Conversely the increase in TCOM was <10 torr in nine patients, and only one of these patients (11%) ultimately healed ($P < 0.01$). Patients with nonhealing ischemic extremity wounds may heal with adjunctive HBO therapy. We can predictably identify patients who are likely to benefit from this modality using TCOM at the time of initial evaluation. An increase of tissue O_2 tension of ≥10 torr when breathing pure O_2 suggests that the patient may benefit from HBO therapy. Those patients with an increase of <10 torr are unlikely to receive benefit from this treatment modality.

PERIPHERAL ARTERIAL OCCLUSIVE disease of the extremities can result in nonhealing ulceration and gangrene. Arterial reconstructive surgery remains the cornerstone of therapy. The restoration of pulsatile arterial flow into a patent distal runoff bed combined with proper debridement of devitalized tissue is usually sufficient to effect wound healing and subsequent limb salvage. Conversely in some patients limb-threatening ischemia persists after the successful reconstruction of large-vessel occlusive disease secondary to the absence of a competent tibiopedal circulation. In such instances ischemic lesions and minor amputations may not heal even with a patent and functioning bypass graft.

Hyperbaric oxygen (HBO) therapy is the inhalation of 100 per cent oxygen in a pressurized environment typically at two to three times the atmospheric pressure experienced at sea level. It may be a useful adjunct in the treatment of these patients having wounds associated with critical refractory limb ischemia. Identification of patients likely to benefit is essential before

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Address correspondence and reprint requests to Michael A. Zatina, M.D., Department of Surgery, St. Agnes HealthCare, 900 Caton Avenue, Baltimore, MD 21229.

treatment, as therapy can be associated with complications and is costly.[1] Typically patients are subjected to this HBO environment in two different settings. A monoplace chamber is large enough for only one patient and is pressurized with the oxygen itself. This was the type of chamber used in our center. Alternatively larger centers often use multiplace chambers, where several patients are treated simultaneously. In this setting, the environment is pressurized with air, and patients breathe oxygen through a mask or other device to assure that only oxygen is being inhaled while they are exposed to this elevated pressure.

Transcutaneous oxygen measurement (TCOM) has been used to assess the degree of hypoxia present in ischemic tissue and has been proven to be satisfactory for objective risk stratification.[2, 3] Adapting a criterion originally introduced to assess skin flap viability and exploited to successfully predict amputation success we tested the response of TCOM to 100 per cent oxygen inhalation in an effort to improve our ability to predict the benefit of adjunctive HBO therapy.[4, 5] We tested the hypothesis that a ΔTCOM value of ≥ 10 is predictive of healing with adjunctive HBO therapy and that a ΔTCOM of <10 is predictive of failure of healing with HBO therapy.

The purpose of the present study was twofold. We first wished to determine whether HBO is in fact a useful adjunct in the treatment of patients with these ischemic wounds and to what extent it is useful. Secondly we wished to evaluate whether these changes in TCOM values with 100 per cent oxygen inhalation in a normobaric environment can assist in identifying those patients who may heal with HBO therapy and those who are unlikely to benefit.

Materials and Methods

Between December 1997 and March 2000 we assessed 36 patients with wounds in critically ischemic extremities at our wound center. This service is staffed by surgeons including those from the vascular surgery service. Eighteen of these patients (50%) had undergone recent lower-extremity revascularization including 20 bypass grafts, four arterial angioplasties, and two arterial endarterectomies. There were 21 men (58%) and 15 women (42%). The average patient age was 68.9 ± 1.8 years. Twenty-four patients (67%) had diabetes mellitus, nine patients (25 per cent) had hypercholesterolemia, 23 patients (64 per cent) had hypertension, 21 patients (58%) had significant coronary artery disease, and eight patients (22%) smoked cigarettes. Dialysis-dependent renal failure was present as a coexisting condition in nine patients (25%). These patients had one or more of the following nonhealing wounds. There were 28 foot or leg ulcers, 18 digit amputation sites, one above-knee amputation, and one transmetatarsal amputation. All patients underwent lower-extremity segmental pressure assessment and waveform analysis—either Doppler and/or pulse volume—before HBO therapy. In all of these patients noninvasive testing documented some element of distal extremity ischemia.

All of these patients underwent baseline evaluation using TCOM during evaluation for HBO therapy. In each patient the two TCOM readings were determined at time of the initial evaluation. With the patient in a relaxed supine position and after local skin preparation baseline measurement was taken with the patient breathing room air. Skin site preparation included gentle desquamation and preparation with isopropyl alcohol. The transducer was then applied. A Clark Polarographic Electrode and oxymonitor (SM 361, Litton Medical Electronics, Elk Grove Village, IL) were used. A second site was simultaneously measured for calibration purposes. The site used for the actual TCOM measurement was the healthy skin just proximal to the nonhealing wound. After successful measurement at baseline the patients were administered 100 per cent O_2 via a nonrebreathing mask for 10 minutes until equilibration occurred, and the TCOM values were reassessed. In each patient ΔTCOM was calculated as the difference in TCOM values obtained during inhalation with 100 per cent oxygen by mask *versus* that value obtained at baseline when the patient was breathing room air. This value was calculated for each patient only at the time of the initial patient evaluation, and these data were used for this study. We did not measure TCOM during the actual hyperbaric treatments.

A Sechrist (Sechrist Industries, Anaheim, CA) monoplace hyperbaric chamber was used for all patients undergoing HBO therapy. Sessions were conducted once a day usually five days per week and typically lasted a total of 90 minutes each. Compression ranged between 2.0 and 2.5 ATA (atmospheres absolute) pressure. Pressurization was performed with pure oxygen. There was a single "air break" totaling 10 minutes in the middle of each session. This is routinely used to interrupt the continuous exposure to the oxygen to minimize pulmonary toxicity. During this time the patients breathed room air through a tube while still pressurized in the chamber. Patients were carefully monitored during each treatment session by either a registered nurse or respiratory technologist under the supervision of the staff physician. All patients received daily wound care including but not limited to wet to dry dressings, sharp debridement, hydrotherapy, and topical antimicrobials and/or topical growth factor application. Wound healing was defined

TABLE 1. *Data for Patients in Group A (ΔTCOM <10 mm Hg)*

Patient		Depth of HBO Dive	Total No. of Dives	TCOM 21%*	TCOM 100%†	ΔTCOM (mm Hg)‡	Complications	Wounds	Prior Vascular Reconstruction	Results of Adjunctive HBO Therapy	Final Outcome
Age	Sex										
75	M	2.4 ATA	7	2	2	0	Pleural effusion	R heel, L heel, L first and second toe ulcers	R POP–peroneal bypass	Failed	R AKA
77	F	2.4 ATA	23	15	15	9		Plantar abscess	ANT TIB-DP bypass	Failed	R BKA
62	M	2.4 ATA	35	10	10	−5		R first toe ulcer	R Profundaplasty	Failed	
62	M	2.4 ATA	7	3	3	4		L lateral leg ulcer		Failed	
72	M	2.5 ATA	32	3	3	−1	Severe anxiety	R first toe gangrene	R FEM-peroneal bypass with AV fistula	Healed	R forefoot amputation
60	M	2.5 ATA	57	9	9	2		L foot ulcer, R leg ulcer		Failed	L AKA
64	F	2.5 ATA	27	2	2	0		L ankle ulcer, necrotic R middle finger		Failed	
85	F	2.4 ATA	18	8	8	−5		L foot gangrene	L FEM ANT-TIB bypass	Failed	L AKA
60	F	2.4 ATA	27	1	1	2		R forefoot gangrene, L first and second toes gangrene	L SFA and TIB angioplasties; R FEM-POP bypass; R-POP-PT bypass; L POP-PT bypass	Failed	R BKA

Patients are listed individually by age and sex; individual patient data are shown. ATA, atmospheres absolute; LAT, lateral; POP, popliteal; ANT, anterior; TIB, tibial; DP, dorsalis pedis; FEM, femoral; AV, arteriovenous; SFA, superficial femoral artery; PT, posterior tibial; AKA, above-knee amputation; BKA, below-knee amputation; R, right; L, left.

* Initial TCOM on room air (21%).

† TCOM response to 100 per cent oxygen challenge.

‡ Calculated ΔTCOM value [ΔTCOM = TCOM (100%) − TCOM (21%)].

as complete wound closure with epithelialization of the wound surface, healed suture lines, or viability of split-thickness skin grafts.

The patients were divided into two groups for analysis on the basis the values of ΔTCOM calculated. Given previous reports about the use of this value[4, 5] patients were placed in Group A if ΔTCOM was less than 10 torr. Patients were placed in Group B if ΔTCOM was greater than or equal to 10 torr. Treatment protocols were the same for all patients regardless of the ΔTCOM value calculated at the time of the initial evaluation.

Pooled data from each group were expressed as mean ± standard error of the mean. Data were analyzed using unpaired Student's *T* test and chi-square evaluations. Significance was assumed if $P < 0.05$.

Results

Data are summarized for the two groups in Tables 1 and 2. There were nine patients in Group A having ΔTCOM values of less than ten. There were 27 patients in Group B having ΔTCOM values of greater than or equal to ten. The gender distributions of the two groups were similar with males comprising 55 per cent of patients in Group A and 59 per cent in Group B. The average patient age in Group A was 68.6 ± 3.0 years and in Group B 69.1 ± 2.2 years. Patients underwent HBO treatments until healing was complete or until the attending physician determined that there was little or no improvement to justify further therapy. The total numbers of HBO treatment sessions were similar in Group A (25.9 ± 5.1) and Group B (28.2 ± 2.3). Ten complications of HBO were observed. These included severe claustrophobia requiring presession sedation (one), seizure (one), severe myopia (one), middle ear barotrauma (five; with four patients requiring myringotomy and tube placement), and congestive heart failure exacerbation in two patients.

The average TCOM at baseline for patients in Group A was 5.8 ± 1.6 torr. The TCOM value for patients in this group after the inhalation of 100 per cent oxygen was 6.50 ± 2.4 torr. The mean ΔTCOM value in this group was 0.7 ± 1.5 torr. In some Group A patients ΔTCOM was actually a negative value, as the TCOM value was less after breathing 100 per cent oxygen than when they were breathing room air.

In Group B patients the average TCOM value at baseline when breathing room air was higher than those in Group A (16.0 ± 2.5 torr; $P < 0.05$). The TCOM value after inhalation of 100 per cent oxygen was dramatically higher (75.9 ± 11 torr; $P < 0.05$) than those patients in Group A. Finally, the ΔTCOM value of patients in Group B was 59.9 ± 10 torr, again significantly higher than the corresponding value in Group A patients ($P < 0.05$).

As seen in Table 1 only one of nine (11%) ischemic wounds healed after HBO therapy in Group A patients. Nineteen of 27 (70%) patients having ΔTCOM values ≥10 mm Hg healed ischemic wounds with HBO (Table 2). This difference proved significant ($P \leq 0.01$), which confirms that this distinction was suggestive of the likelihood of success or failure. Three of eight treatment failures in Group B were secondary to refractory bone and joint infection. Limb amputation was performed in these patients despite granulating wounds. Initial (room air) TCOM values did not predict outcome. Patients who went on to heal did not have baseline TCOM values (15.8 ± 2.8 torr) that differed significantly ($P < 0.05$) from those who did not ultimately heal (10.7 ± 2.9 torr). Patients who went on to heal did, however, have a higher TCOM after breathing 100 per cent oxygen than those who did not heal (77.4 ± 14.7 *vs* 35.0 ± 8.9 torr; $P < 0.05$). We could not identify a specific comorbid factor or race or gender to be associated with a different likelihood of predicting success or failure of wound healing with HBO therapy. The influence of ΔTCOM on healing can be seen in Fig. 1.

Discussion

Unfortunately because of its past indiscriminate usage (as treatment for arthritis, wrinkles, impotence, aging, baldness, epilepsy, and many other maladies), HBO is thought by many to be "a therapy in search of a disease." More recently the documentation of significant beneficial biocellular and clinical effects have established HBO as primary therapy in patients with decompression sickness, arterial gas embolism, or carbon monoxide poisoning. It has also proved useful as an adjuvant therapy in the management of osteoradionecrosis, clostridial myonecrosis, crush injury, radiation-induced soft-tissue injury, certain problem wounds, and compromised skin grafts and reconstructive flaps.

HBO therapy is not inexpensive and does involve some risk.[12] Our charge for a typical 90-minute session with wound care was approximately $408. Given the total number of treatments used for patients in this study the average cost ranged between $10,200 and $12,240. Known adverse effects of HBO therapy include: claustrophobia; myopia, which is usually reversible; pain in the middle ear, cranial sinuses, or teeth; and pulmonary barotrauma. In addition this therapy can be associated with precipitation of generalized seizures, the exacerbation of congestive heart failure, and in the critically ill requiring prolonged high concentrations of normobaric oxygen a risk of pulmonary toxicity. The most catastrophic complication is the risk of a fire or explosion developing be-

TABLE 2. *Data for Patients in Group B (ΔTCOM >10 mm Hg)*

Patient											
Age	Sex	Depth of HBO Dive	Total No. of Dives	TCOM 21%*	TCOM 100%†	ΔTCOM (mm Hg)‡	Complications	Wounds	Prior Vascular Reconstruction	Results of Adjunctive HBO Therapy	Final Outcome
76	F	2.4 ATA	30	1	21	20		R second and third toe ulcers	R FEM-POP bypass	Healed	R second and third toe AMP
74	F	2.5 ATA	39	22	173	151		R leg eschar		Healed	STSG
63	F	2.4 ATA	20	26	132	106		Nonhealing L AKA	STSG	Healed	
84	F	2.4 ATA	26	27	131	104	Myringotomy tubes	R first toe gangrene	R FEM-peroneal bypass	Healed	R first toe AMP
75	M	2.5 ATA	17	29	117	88		R leg ischemic ulcer		Healed	
82	M	2.5 ATA	21	1	27	26	Ear pain	R LAT maleolar ulcer		Healed	STSG
65	M	2.5 ATA	29	36	104	68		L heel ulcer	L FEM-peroneal bypass, R FEM-ANT TIB bypass	Failed	L BKA
55	M	2.5 ATA	11	38	69	31	Myringotomy tubes	R first toe ischemic ulcer		Healed	
76	F	2.0 ATA	27	5	33	28		R second toe gangrene	R POP-plantar bypass	Healed	R second toe AMP
56	M	2.4 ATA	21	13	36	23		R foot plantar ulcer	R POP-peroneal bypass	Healed	Open R third ray AMP
83	F	2.5 ATA	25	2	34	32		L leg wound		Healed	
67	M	2.4 ATA	35	27	48	21		R forefoot gangrene due to emboli from CABG	Profundaplasty, R FEM-POP bypass, peroneal embolectomy	Healed	R TRANSMET AMP
45	M	2.4 ATA	35	8	26	18		L foot wound		Failed	L BKA
74	M	2.0 ATA	16	32	47	15	Myringotomy tubes	Nonhealing R TRANSMET		Healed	STSG
63	M	2.5 ATA	42	12	89	77		L heel ulcer		Failed	L BKA
72	F	2.4 ATA	16	12	185	173	CHF	L second toe ulcer	TIB angioplasty	Healed	

75	F	2.4 ATA	24	14	36	22	Myringotomy tubes	R leg stasis ulcer	R ANT TIB angioplasty, L ANT TIB-ANT TIB bypass	Healed	STSG
60	F	2.0 ATA	32	37	56	19	Seizure	B/L forefoot gangrene, POST calf ulcers	R POP ANT TIB bypass	Failed	R BKA
82	M	2.5 ATA	13	2	35	33		R first toe ulcer, R SAPH vein harvest site wound	R FEM-peroneal bypass, peroneal-DP bypass	Failed	R BKA
69	M	2.4 ATA	28	3	43	40		L first toe open AMP-first ray	L POP-peroneal bypass, peroneal-DP bypass	Failed	L BKA
81	F	2.5 ATA	58	35	241	206		L maleolar ulcer		Healed	
69	M	2.4 ATA	54	7	24	17		R fifth toe gangrene		Healed	R fourth and fifth toe AMP, STSG foot
52	M	2.4 ATA	33	4	58	54		R fourth toe gangrene		Healed	Healed open fourth ray AMP site
85	F	2.5 ATA	27	16	26	10		R great toe ulcer		Healed	R first and second toe AMP
48	M	2.5 ATA	11	2	52	50		L plantar ulcer		Failed	L BKA
76	M	2.4 ATA	24	1	108	107		L lower LAT calf wound		Healed	STSG
58	M	2.4 ATA	48	21	98	77	Visual changes	Necrotic L heel	Aorto-FEM bypass, profunda-ANT TIB bypass	Failed	L AKA

Patients are listed individually by age and sex; individual patient data are shown. ATA, atmospheres absolute; AKA, above-knee amputation; LAT, lateral; TRANSMET, transmetatarsal; B/L, bilateral; POST, posterior; SAPH, saphenous; AMP, amputation; FEM, femoral; POP, popliteal; STSG, split-thickness skin graft; ANT, anterior; TIB, tibial; DP, dorsalis pedis; BKA, below-knee amputation; CHF, congestive heart failure; CABG, coronary artery bypass graft; R, right; L, left.

* Initial TCOM on room air (21%).

† TCOM response to 100 per cent oxygen challenge.

‡ Calculated ΔTCOM value [ΔTCOM = TCOM (100%) – TCOM (21%)].

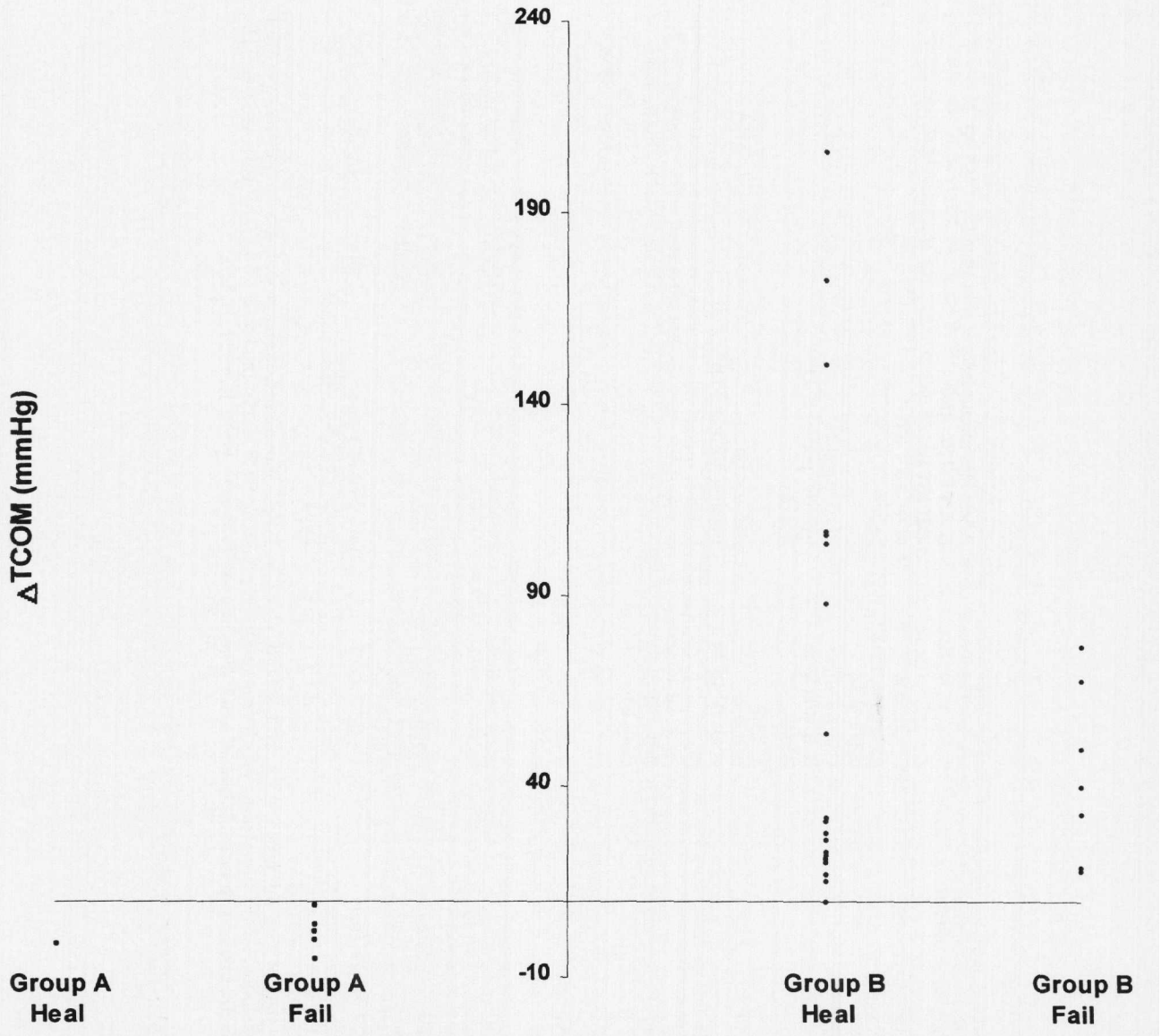

FIG. 1. ΔTCOM *versus* Groups A and B subdivided by healing and failure. *x*-axis from left to right: Group A healed, Group A failed, Group B healed, Group B failed. Only one of nine patients in Group A healed. Nineteen of 27 patients in Group B healed. *y*-axis: ΔTCOM (mm Hg). Note: The *x*-axis intercepts the *y*-axis at ΔTCOM of 10 mm Hg. Points below left and above right of the *x*-axis therefore represent patients in Groups A and B, respectively. Three points for Group A Fail represent two patients each, including one signifying two patients exhibiting a negative ΔTCOM value. In addition there are two other patients with negative ΔTCOM values.

cause of the high concentration of pressurized oxygen in use.[6] Although this has occurred, with catastrophic consequences, with strict attention to the details of proper utilization, this is a very rare occurrence; it was not experienced in our center.

A prerequisite for the healing of any wound is adequate local oxygen tension.[4, 7–10] Fibroblasts located beyond native capillary beds require a critical tissue oxygen tension for the formation of a supportive collagen matrix, which is essential for angiogenesis. The rate of neovascularization is directly proportional to O_2 concentration. An oxygen environment of at least 40 torr is required to initiate normal collagen production.[7, 11] In addition collagen crosslinking, which is crucial to wound tensile strength, is driven at pO_2 values in this range. Additionally infection is promoted through impaired leukocyte bacterial killing within a hypoxic environment.[12, 13] HBO treatment can rapidly reverse local hypoxia in most wounds by causing dramatic increases in the dissolved-oxygen content in blood. It was because of this effect that we considered this therapy to be a likely adjuvant in this patient population. We are unaware of any objective data supporting the use of HBO therapy in patients having documented peripheral arterial occlusive disease and problem extremity wounds.

There has been significant scrutiny used in evaluating requests for reimbursement for HBO therapy. The

various intermediaries for Medicare have denied payments for this therapy without documentation that it is in fact beneficial and not experimental. These data clearly give support to the use of HBO therapy as an adjuvant for the healing of difficult ischemic wounds in which all conventional therapy has been exhausted.

In this series 18 patients were found to have arterial occlusive lesions that were amenable to some form of vascular reconstruction. They were treated aggressively with both suprainguinal and infrainguinal vascular reconstruction, angioplasty and/or endarterectomy, or combinations thereof. Arterial reconstruction patency was objectively documented intraoperatively and either before or during HBO therapy. We suspect that similar patient groups are found in all busy vascular surgery practices. Furthermore the persistence of ischemia after aggressive reconstruction is explained by the severe nature of their arteriosclerotic disease.

Noninvasive TCOM determination is an established predictor of the severity of tissue hypoxia associated with peripheral arterial occlusive disease. This objective measure, which was previously used to quantify the healing potential in ischemic extremity wounds,[14] has also been documented to assist in the prediction for likelihood of healing or failure in lower-extremity amputation sites.[4] Unlike other noninvasive studies TCOM assessment uniquely provides a demonstration of the adequacy of local tissue oxygenation. We believe that in ischemic extremities (TCOM <40 mm Hg), increases in TCOM after oxygen inhalation reflect microcirculatory reserve. Moreover ΔTCOM ≤10 mm Hg, which is a limit previously exploited to discriminate skin flap viability and to predict limb amputation levels, proved to be a strong negative predictor of wound healing.[4, 5]

In our study population we used this difference in ΔTCOM to determine whether patients were likely to heal with adjuvant HBO therapy. Patients in Group B with ΔTCOM ≥10 mm Hg had a 70 per cent chance of healing their ischemic wounds when HBO therapy was used in addition to aggressive vascular reconstruction and appropriate local wound care. Conversely patients in Group A with ΔTCOM values of <10 torr are very unlikely to benefit when treated with HBO, as 89 per cent failed to heal in this study. We feel that this value can be used to discriminate between patients who should be considered for adjuvant HBO therapy. Excluding patients in Group A from therapy seems very cost-effective, whereas referring patients in Group B is most appropriate and can be defended as good use of valuable health care dollars.

Conclusion

Wound healing requires a critical level of tissue oxygen tension below which healing is compromised. HBO therapy is a treatment modality currently in use but of disputed efficacy. We have identified a group of patients with peripheral ischemia and nonhealing wounds who benefited from HBO therapy. Specifically using an objective and reproducible measurement (ΔTCOM) and a corresponding limit (≥10 torr) statistical significance regarding healing could be predicted. These patients should be treated with HBO therapy. Of equal or possibly even greater importance is the fact that those patients with ΔTCOM values of <10 torr are unlikely to benefit and should not undergo this costly and prolonged therapy.

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DISCUSSION

DANIEL DIAMOND, M.D. (Washington, PA): There are many other factors that might affect TCOM changes. A few that come to mind are: bronchospasm, atelectasis, the timing of the last albuterol treatment. Did you make any attempts to factor in these and other potential variables?

CHRISTOPHER GATES, M.D. (Augusta, GA): You mentioned that HBO is controversial in the management of indolent wounds. I would say it is controversial only among those who are unfamiliar or untrained with its use and that in itself is probably controversial. Did you break down your successes and failures *in* those who had vascular reconstruction *versus* those that had not? You mentioned that you use 2.0 to 2.5 ATA; why not use 1.8 or 1.6 or why not use 3.0? What gave you your range of 2.0 to 2.5? Could it be lower or could it be higher and could you do some studies to verify that?

RODNEY E. GROLMAN, M.D. (Closing Discussion): Dr. Diamond, in response to your question, patients were given inhalant bronchodilator therapy prior to our measuring TCOM both at baseline and after O_2 inhalation if their clinical condition warranted it. All patients were standardized to the extent that all breathed oxygen through a nonrebreather mask. None of the patients reported on in this series were considered to be in end-stage chronic obstructive pulmonary disease, and none required chronic home O_2 therapy.

Dr. Gates, all patients that required bypass surgery underwent bypass if it was anatomically possible. Our goal has always been to employ vascular reconstruction for limb salvage aggressively, with debridement of devitalized tissue being used as indicated. We did not use hyperbaric oxygen therapy as a replacement to vascular reconstruction, but as an adjunctive therapy. Having said that, we could not find a clear association based on the use of prior vascular reconstruction alone. Failure of limb salvage occurred in patients in both groups. Some of the patients in Group B that failed had very extensive soft and boney tissue damage or loss prior to evaluation that ultimately precluded salvage despite evidence of hyperoxygenation. Others in this group just failed aggressive attempts at salvage despite the response to oxygen inhalation at the time of initial evaluation.

Finally, we used a range of 2.0 to 2.5 ATA per our protocols for wound care that have been developed over years of hyperbaric oxygen therapy use. Higher pressures are in fact used for treating, for example, gas gangrene, air embolism, and decompression illness. Note however that there is more risk of pressure-related complications with exposure to these higher pressures. Use of the lower pressures may not give outcomes as beneficial as those that were seen in this study.