Survey comparing the treatment of central retinal artery occlusion with hyperbaric oxygen in Australia and New Zealand with the recommended guidelines as outlined by the Undersea and Hyperbaric Medical Society

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Abstract
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Introduction: Central retinal artery occlusion (CRAO) presents suddenly causing painless loss of vision that is often significant. Meaningful improvement in vision occurs in only 8% of patients with spontaneous reperfusion. Hyperbaric oxygen treatment (HBOT) is considered to be of benefit if commenced before retinal infarction occurs. The Undersea and Hyperbaric Medical Society (UHMS) guidelines on the management of CRAO were last amended in 2019. This survey questioned Australian and New Zealand (ANZ) hyperbaric medicine units (HMUs) about the incidence of CRAO cases referred and compared their subsequent management against the UHMS guidelines.
Methods: An anonymous survey via SurveyMonkey® was sent to all 12 ANZ HMUs that treat emergency indications, allowing for multiple choice and free text answers regarding their management of CRAO.
Results: One-hundred and forty-six cases of CRAO were treated in ANZ HMUs over the last five years. Most (101/146) cases (69%) were initially treated at a pressure of 284 kPa. This was the area of greatest difference noted in CRAO management between the UHMS guidelines and ANZ practice.
Conclusions: Few ANZ HMUs strictly followed the UHMS guidelines. We suggest a more simplified management protocol as used by the majority of ANZ HMUs.

Introduction

Insufficient blood supply to the inner layers of the retina from retinal artery occlusion (RAO) (either central or branch) is rare but serious. The incidence has been reported as 0.85 cases per 100,000 but may be significantly higher due to under-reporting of this condition.[1] Central retinal artery occlusion (CRAO) presents acutely with sudden onset painless, unilateral vision loss. Vision to the affected eye is often significantly reduced, typically with no useful vision remaining if the central retinal artery is occluded. Limited field vision is common when branch retinal artery occlusion occurs. Whilst over a few days there will typically be recanalisation of the artery, by this time the retina is often irreversibly damaged from hypoxia. Meaningful improvement in vision is estimated to occur in only 8% of patients with spontaneous reperfusion.[2] Vision impairment is known to have a profound impact on a patient's quality of life.[3] For convenience, the term CRAO will be used for all cases including branch RAO.

The central retinal artery is a branch of the ophthalmic artery. An ophthalmic artery originates from each internal carotid artery. The retina has a dual blood supply, with the inner layers supplied with blood from the central retinal artery and its branches, while the choroidal circulation supplies the outer layers. Retinal cells exhibit the highest oxygen (O_2) consumption in the body by weight (13 $mL \cdot 100g^{-1} \cdot min^{-1}$), making the retina highly susceptible to ischaemia.[4] Variation in visual acuity from CRAO occurs because partial perfusion of the retina may persist in some cases. The choroid supplies 50–60% of the retina with O_2, provided there is normal ophthalmic artery perfusion.[5] In addition, 15–30% of the population has a cilioretinal artery, supplying blood to the area around the fovea.[6]

There are multiple possible causes for CRAO including thrombosis, embolus, dissection, arteritis and vasospasm. The Undersea and Hyperbaric Medical Society (UHMS) guidelines on CRAO state that an ophthalmologist should be consulted emergently in cases of suspected CRAO.[7]

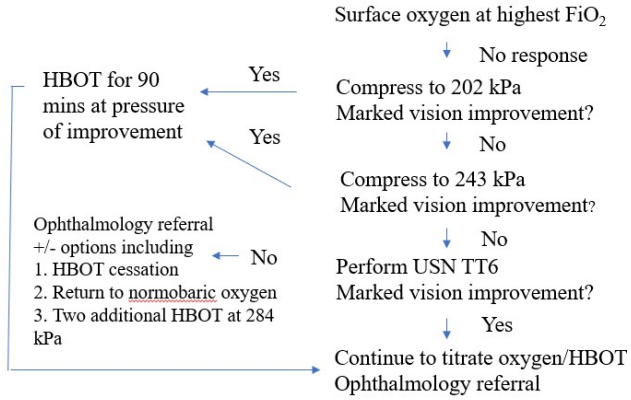
To arrive at a diagnosis of CRAO, decreased vision without improvement with pinhole examination needs to be confirmed, as well as a fundoscopic exam preferably using dilatation if there are no contraindications. Moreover, alternative diagnoses including retinal detachment or vitreous haemorrhage must also be excluded. Full work-up for CRAO includes: a full blood count (to screen for platelet disorders or infective causes); erythrocyte sedimentation rate (ESR) and C-reactive protein (to screen for giant cell arteritis); coagulation profile (fibrinogen, prothrombin time/ partial thromboplastin time [PT/PTT] , antiphospholipid antibody); lipid panel; electrocardiogram (ECG); carotid ultrasound; brain magnetic resonance imaging (MRI) and echocardiography. Of note, however, hyperbaric oxygen treatment (HBOT) should not be delayed accomplishing these diagnostic measures. Moreover, if arteritis is the suspected cause of CRAO, HBOT should still be undertaken in addition to intravenous corticosteroids.

Multiple treatments for CRAO have been reported including ocular massage, haemodilution, anterior chamber paracentesis, intravenous acetazolamide, transluminal Nd:YAG laser, intra-arterial thrombolytic therapy and intravenous fibrinolytic therapy. No significant benefit has been clearly demonstrated with any of these treatments, and moreover serious haemorrhagic sequelae may result from thrombolytic and fibrinolytic therapy, and surgical embolectomy.

Hyperbaric oxygen is considered to be of benefit for CRAO as the higher partial pressure of O_2 in arterial blood allows the peripheral collateral circulation to meet the retina's demands for O_2 while time passes before the central retinal artery recanalises. In animal models, HBOT has demonstrated the capacity to reduce both tissue oedema and ischaemia-reperfusion injury after recanalisation.[8]

Hyperbaric oxygen for CRAO is classified as American Heart Association (AHA) class IIb level of evidence.[9] Class IIb implies that the benefit of treatment is deemed to outweigh the associated risks but usefulness/efficacy is less well established by the evidence/opinion. Retrospective controlled case series have shown fair to good evidence supporting the use of HBOT for CRAO. The UHMS review reported that 66% of the 927 patients treated with HBOT experienced vision improvement after treatment.[7] A recent retrospective study from the Royal Brisbane and Women's Hospital continues to support HBOT as being beneficial and safe for CRAO.[10] There remains great difficulty in performing prospective randomised controlled trials for CRAO and HBOT, on account of the ethical considerations for a proposed trial when no alternative therapy with a similar outcome exists, and because of the relatively low incidence of the condition. The UHMS guidelines for management of CRAO offer at present the only widely available protocol. The most recent Hyperbaric textbook has used these UHMS criteria.[11] Acknowledging that there are limitations on the evidence as to how these guidelines were developed, we have used them as the best available option from which to consider the management offered by other Australian and New Zealand (ANZ) hyperbaric medicine units (HMUs).

Figure 1
Undersea and Hyperbaric Medical Society Guidelines for the acute management of central retinal artery occlusion (CRAO); FiO_2 – Fraction of inspired oxygen; HBOT – Hyperbaric oxygen treatment; mins – minutes; USN TT6 – United States Navy Treatment Table 6



UHMS GUIDELINES

The UHMS guidelines for management of CRAO (Figure 1) advise considering patients for HBOT if they present within 24 hours (h) of symptom onset.[7] This guideline however does note a few case reports where patients have had benefit from HBOT after the 24-h window had passed.

The UHMS guidelines advocate immediately commencing the highest possible fraction of inspired O_2 (FiO_2) at 1 atmosphere. If there is significant improvement within 15 minutes (mins), the patient should then have intermittent normobaric O_2 for 15 mins every hour, alternating with 45 mins of breathing room air. Visual acuity should continue to be checked after each air-breathing period, with this regimen continuing until either a fluorescein angiogram shows patency, the patient's vision remains stable on room air for 2 h, or a maximum of 96 h on intermittent supplemental O_2 therapy had been reached.

If there is no response to high fraction normobaric O_2 within 15 mins, the UHMS guidelines advocate that HBOT can be delivered for 90 mins at the pressure of the return to vision, with a maximum of United States Navy Treatment Table 6 (USN TT6) – which begins at 284 kPa / 2.8 atmospheres absolute (atm abs) for a first treatment. Initially their recommendation is compression to 2 atm abs (203 kPa) on 100% O_2. Should there be no improvement in vision at 2 atm abs by the first air-break period (or 30 minutes), they advise progressing to a pressure of 2.4 atm abs (243 kPa). If no response at 2.4 atm abs, the guidelines advise compressing

to 2.8 atm abs (284 kPa). If there were still no improvement after the first 20 mins period at 2.8 atm abs, the guidelines suggest proceeding to USN TT6. If vision had improved at 2.4 atm abs, the guidelines suggest conducting a United States Navy Treatment Table 9 (USN TT9).

Should there have been no response following completion of USN TT6, options at this point would be to either discontinue treatment, continue with normobaric O_2 at the highest possible FiO_2, or give two additional 90-minute treatments at 2.8 atm abs (284 kPa) with air-breathing periods, on a twice-daily schedule.

If the patient had return of vision during HBOT, the UHMS guidelines recommend considering inpatient monitoring and intermittent supplemental O_2. Should vision loss recur, the UHMS guidelines suggest aggressive use of intermittent normobaric O_2 as described in the initial treatment for CRAO. Alternatively, a customised HBOT protocol would be indicated to preserve retinal function until central retinal artery recanalisation occurs.

The UHMS guidelines also state that HBOT twice or three times daily may be necessary until the angiogram normalises or the patient has no further improvement for three treatments.

It should be noted that the UHMS mentions an exception to this advised regimen when CRAO results from cerebral arterial gas embolism (CAGE). The recommended treatment regimen for CAGE should be followed with a minimum of USN TT6.

For the purpose of this survey, we have used the UHMS guidelines for the management of CRAO as the benchmark against which the management by the ANZ HMUs can be compared. The UHMS guidelines were written based on what its authors considered at the time to be the best HBOT management of CRAO. With ongoing evidence adding weight to the body of knowledge already supporting HBOT for CRAO, it is important that ANZ HMUs are aware of CRAO and its management.

Methods

Approval was obtained for data review and extraction by Governance, Evidence, Knowledge and Outcomes (GEKO) at Fiona Stanley Hospital (Approval Number 42155).

All 12 Australasian HMUs that treat hyperbaric emergencies were emailed a SurveyMonkey® questionnaire for completion. The survey included a pool of nine questions that asked about their frequency of CRAO referral, their use of HBOT for CRAO, the methods by which this was delivered, as well as their ongoing management of CRAO (*Appendix 1). The survey allowed for a multiple-choice response as well as free text for further clarification or comment. Responses were analysed using SurveyMonkey® software (Momentive Inc, San Mateo, CA) for quantitative and qualitative results.

Results

CASES TREATED

There were 146 CRAO cases treated in ANZ HMUs in the 5-year period surveyed between 2017 and 2021 (Table 1 and Table 2).

TIME WINDOW FOR CRAO TREATMENT WITH HBOT

Nearly all institutions agreed that offering HBOT for CRAO patients presenting sub-acutely offered little benefit. Christchurch Hospital (CHCH) and Fiona Stanley Hospital (FSH) had a cut-off time of within 24 h but would ideally prefer < 12 h. The Royal Hobart Hospital (RHH) also had a cut off time < 24 h but preferred presentation within 8 h. Other units that had a cut off time < 24 h included: the Alfred Hospital (AH), the Royal Adelaide Hospital (RAH), the Royal Darwin Hospital (RDH), the Royal Brisbane and Women's Hospital (RBWH), the Townsville Hospital (TH) and the Wesley Hyperbaric Medicine Unit (WHMU). The RDH reported they offered HBOT if it could be initiated < 5 days from symptom onset. The Prince of Wales Hospital (POWH) offered HBOT if initiated < 24 h from symptom onset but would treat later presentations if the patient had

Table 1

Australian and New Zealand hyperbaric facilities and the corresponding number of central retinal artery occlusion cases treated with hyperbaric oxygen over five years; AH – The Alfred Hospital; CHCH – Christchurch Hospital; FSH – Fiona Stanley Hospital; LTPH – La Trobe Private Hospital; POWH – Prince of Wales Hospital; RAH – Royal Adelaide Hospital; RBWH – Royal Brisbane and Women's Hospital; RDH – Royal Darwin Hospital; RHH – Royal Hobart Hospital; SHMU – Slark Hyperbaric Medical Unit; TH – Townsville Hospital; WHMU – Wesley Hyperbaric Medical Unit

HMU	AH	CHCH	FSH	LTPH	POWH	RAH	RBWH	RDH	RHH	SHMU	TH	WHMU	Total
Cases	1	56	17	0	15	0	27	1	16	0	10	3	146

Footnote: * Appendix 1 are available on DHM Journal's website: https://www.dhmjournal.com/index.php/journals?id=336

Table 2

Australian and New Zealand hyperbaric facilities (excluding La Trobe Private Hospital) and their responses to the central retinal artery occlusion (CRAO) questionnaire. La Trobe treated no patients and only provided a response to the time window question (< 48 hours in their case). atm abs – atmospheres absolute; BD – twice daily; Contraindications – contraindications; ED – emergency department; HBOT – hyperbaric oxygen treatment; h – hours; m – metres; NA – not applicable; Opththal – ophthalmologist or ophthalmology service; S/B – seen by; SMO – senior medical officer; Sx – symptoms; T5 – United States Navy Treatment Table 5; T6 – United States Navy Treatment Table 6; UHMS – Undersea and Hyperbaric Medical Society; VA – visual acuity; See Table 1 for hyperbaric unit name abbreviations. See text for explanation of HBOT table abbreviations (18-60-30, 18-60-35, 14-90-30, 14-90-20, 10-90-30); in this nomenclature, the first number refers to the pressure expressed in metres of seawater, the second number refers to the duration of 100% oxygen breathing (minutes), and the third number refers to the decompression time (minutes) while still breathing 100% oxygen

Question	AH	CH	FSH	POWH	RAH	RBWH	RDH	RHH	SHMU	TH	WHMU
How many CRAO cases have you treated in the last five years?	1	56	17	10 to 15	0	27	1	16	0	10	3
Is there a time window from onset of symptoms beyond which your facility will not offer HBOT	24 h	24 h. Prefer within 12 h	Occ 24 h though usually 12 h	24 h (maybe unless "*last*" eye)	24 h	> 24 h	Nothing official (no written policy) but unlikely if > 5 days	Usually if > 24 h vision loss (unless clinical evidence that vision loss is still changing). Prefer to start HBOT within 8 h of symptom onset, if possible	No	24 h	> 24 h
What are the diagnostic criteria / minimum requirements by your facility before commencing HBOT?	Diagnosis confirmed by ophthal, duration since onset < 24 h, no response to normobaric oxygen	Diagnosis of CRAO by ophthal and within 24 h, no contraindications to HBOT	Clinical. Acute loss of vision with consistent retinal findings on fundoscopy/ absence of other cause	S/B opthal. Confirmed recent CRAO	Referral from ophthal after exam	CRAO confirmed by ophthal within 24 h of onset, no contraindications to HBOT	Low threshold to treat given what patient has to lose – so anyone referred by ophthal with diagnosis or suspicion	Diagnosis should be confirmed by an ophthal prior to HBOT. We are happy to take both CRAO and branch retinal artery occlusions. (Have also treated a couple of retinal vein occlusions)	Diagnosis and referral by ophthal. Onset within last 24 h. No response to 15 mins normobaric oxygen	Ophthal referral < 24h after onset	CRAO diagnosis by ophthal. No contraindications to HBOT
What initial treatment table do you offer for CRAO? 18-60-30 or similar, TT5, 140-90, other (please list)	60.5 (284 kPa table)	18-60-30	18-60-35 multiplace 18-60-10 monoplace	Standard 14-90-20	T6?	Sliding based on UHMS[7]	18-60-30	Tend to use Beiran's regime – 284 kPa for all treatments, 18-60-30 BD for 3 days then daily until no further improvement for 3 consecutive days	10-90-30 progressing to 14-90-30 if no improvement after 30 minutes. If no improvement after 90 mins at 243 kPa, progress to T6.	18-60-30	UHMS guidelines 14th edition (T6)[7]

Table 2 continued.

What follow-up HBOT schedule and tables do you use?	AH14 (243 kPa table)	18-60-30 or 14-90-20	18-60 x 3 over 1st 24 h then daily until plateau 3/7 or resolution	Join other patients in standard tables	10-90-30	Proportional to the initial treating pressure.	14-90-14	See answer to question 5 (above)	Would use 18-60-30 BD until plateau if responding or max 8 sessions if not	3 in 1st 24 h, then daily until plateau or resolution reached	18-60-30
Are they on high flow oxygen initially?	Yes	From diagnosis to HBO 1st treatment if possible	Usually	Pre HBOT? Don't think so	Don't know	Yes	No	Possibly – depends on whether they've come via ED or direct from ophthalmologist's rooms	They should be	Variable, unknown often.	Yes
Are they on high flow oxygen between treatments?	Maybe on oxygen between treatments	Only if vision deteriorates following treatment	No	No	No	Yes for the first 24 h with 15 mins on and 45 mins off with VA check	No	No	They should be	Yes if HBOT has produced benefit	Yes
Would the hyperbaric treatment schedule vary between physicians at the department or is there a unit consensus on treatment schedule?	Varies. In our 1 case in 5 years, we sought advice on protocol from another unit who had treated 36 cases in 3 years	Consensus on treatment now generally 18-60-30 but hyperbaric SMO retains discretion if having difficulty getting to depth etc	Unit consensus	We don't do a separate HBOT	Maybe. Don't know what others do	Unit consensus	Likely vary as limited/ no experience so little driver for policy development thus far	Generally adhere to Bieran's regime; very occasionally Murphy-Lavoie's strategy of 100% oxygen urgently at 1 ATA. Compress to 2 atm abs if no benefit. If no improvement within 30 minutes at 2 atm abs compress to 2.4. If no improvement after 20 minutes compress to 2.8 and if not improved, consider USN T6. No clear endpoint – "*until angiogram normalises*"	Unit consensus	Unit consensus	Probably variance but try to stick to UHMS 14th edition guidelines[7]
Is there a specific number of hyperbaric treatments that get offered, or does it vary according to patient response?	Varies based on response	Varies based on response. Christchurch protocol[14]	Until resolution or plateau- no benefit over 3 HBOT	Usually 5 – depends on response	3	Varies based on response – aim for at least 72 h for reperfusion	Dependent on patient response, but if no improvement after 5 then would likely cease	Varies depending on patient response and time to plateau in symptoms. Generally averages out at about 10–11 treatments per patient (167 treatments given to 16 patients over 5 years)	Should vary with patient response (see Q6)	No – according to response	Varies based on response

been affected in their only eye that had vision.

DIAGNOSTIC CRITERIA/MINIMUM REQUIREMENTS BY FACILITY BEFORE COMMENCING HBOT

All HMUs agreed on requiring an ophthalmology review before accepting CRAO cases for HBOT. Ophthalmologists have the essential role of expediently confirming a diagnosis of CRAO before HMUs will accept a patient for treatment. Of note, many HMUs responses stated there needed to be no contraindication to HBOT.

INITIATION OF HIGH FLOW O_2

Not all HMUs reported using high flow O_2 as initial treatment. The RDH said they do not. Most HMUs appeared to recognise that patients should be treated with normobaric first aid O_2 in as high a fraction as possible, but some were also realistic in recognising that when first presenting for HBOT they may not yet have received O_2.

INITIAL TREATMENT TABLE

This survey has shown there are at most only three HMUs which follow the UHMS protocol strictly regarding the initial treatment table. This accounted for 30 of the 146 patients treated over the five-year period (21%). Every other HMU chose a higher initial pressure (79% of the 146 cases). The most common initial treatment pressure (used by six of the 12) was 284 kPa, used in 101 out of the total 146 cases treated (69%).

The most frequently used initial treatment schedule was 18-60-30 or similar (284 kPa / 18 metres of seawater equivalent pressure for 60 mins breathing O_2 with a 30-min decompression). Five of the 12 HMUs said they used an 18-60-30 regimen and another unit used the very similar 18-60-35 (a 35-min decompression instead of 30-min). Two HMUs used a USN TT6, and two HMUs made specific reference to the UHMS 14th edition guidelines for the management of CRAO.[7] The Slark Hyperbaric Medicine Unit (SHMU) has not treated a CRAO case within that last five years, but their proposed management stated they start with a 10-90-30 table (203 kPa / 10 metres of seawater equivalent pressure for 90 mins breathing O_2 with a 30-minute decompression) and progressed to 14-90-30 (243 kPa / 14 metres of seawater equivalent pressure for 90 mins breathing O_2 with a 30-minute decompression) if no improvement after 30 mins. If still no improvement after 90 mins at 243 kPa, they would then progress to USN TT6. This is in keeping with UHMS guidelines. The POWH's initial treatment was a 14-90-20 (243 kPa / 14 metres of seawater equivalent pressure for 90 mins breathing O_2 with a 20-minute decompression).

O_2 BETWEEN TREATMENTS

Six of 15 HMUs answered "*no*" to the question of whether patients were on high flow O_2 between treatments, with another unit saying that O_2 treatment would "*probably not*" be offered. Additionally, one unit responded "*unknown*" and another "*NA*". The AH may offer high flow O_2 between treatments, and two other HMUs offered high flow O_2 between treatments conditionally. The TH offered "*if HBOT had produced improvement*" and the CHCH offered "*only if vision deteriorates following treatment*". Three of the units offered high flow O_2 between treatments unconditionally, with the RBWH reporting that they offered for the first 24 hrs with a 15 mins on and 45 mins off regime along with visual acuity checking.

FOLLOW-UP HBOT SCHEDULE AND TABLES USED

Follow up treatment pressures varied between 203 kPa and 284 kPa. A 243 kPa exposure was the most frequently utilised treatment pressure for follow-up. The RBWH reported that their follow-up pressure would be proportional to the initial treating pressure as per the UHMS guidelines. The TH's treatment schedules included three treatments at 284 kPa during the first 24 h and then one treatment per day subsequently until a plateau or resolution reached. The SHMU instead offered two treatments per day until plateau or resolution up to a total of eight treatments maximum. The FSH utilised three HBOTs at 284 kPa in the first 24 h then daily until plateau for three days or resolution. The RHH treated with Beiran's regime with twice daily treatments for three days then daily until no further improvement for three consecutive days.[12]

DEPARTMENTAL POLICY REGARDING HYPERBARIC TREATMENT SCHEDULE

Seven of the 12 units answered that there would be unequivocal consensus between physicians on the treatment regime chosen for managing acute RAO. When variance was mentioned (as by RAH, RDH and WHMU), it seemed mostly because of low case numbers. The AH mentioned seeking advice from an international unit that had more experience treating CRAO.

NUMBER OF HYPERBARIC TREATMENTS OFFERED

Most units offered a varied schedule depending on the response of the patient to treatment. The CHCH treatment varied according to patient response and have a published protocol outlining a clear treatment regime. The FSH treatment end point was resolution or plateau of symptoms (no improvement over three HBOT). The RBWH's treatment also varied based on response, and specifically mentioned aiming for at least 72 h to allow for recanalisation. The RDH's treatment schedule varied based on response but if no improvement after five HBOT then they would likely

cease. The RHH's detailed answer was that they also varied treatment number depending on patient response and time to plateau in symptoms. They added that it generally averaged out at about 10–11 treatments per patient (167 treatments given to 16 patients over five years).

Discussion

Great variability exists in the number of CRAO cases treated with HBOT over the last five years in ANZ HMUs. This broad range of cases treated is not expected to have resulted from geographical variability in the incidence of CRAO. It is expected that there be a roughly similar incidence of CRAO for all regions. Perhaps what varied was the rate of ophthalmology referral to hyperbaric units and this in turn would depend upon this specialty's regional support of HBOT for acute CRAO. Future work could elucidate whether this is the case.

The responses regarding the time window from symptom onset for which HMUs provide HBOT identify that CRAO is a time critical emergency. Perhaps offering HBOT up to five days from symptom onset is on account of the few case reports noted in the UHMS guidelines demonstrating benefit despite late treatment. Considering the physiology, these cases may represent those that had partial retinal artery occlusion, and so irreparable damage to the retina had been spared. Some HMUs also offer HBOT beyond 24 h of symptom onset if a patient has been affected in their only eye that had vision. Certainly, preservation of vision offers significant quality adjusted life year benefits,[3] and therefore a short trial looking for any improvement may be reasonable.

If we support the theoretical basis of how HBOT works acutely for CRAO, then initial high flow O_2 should be commenced. Also, while HMU specialists may initiate this treatment after being involved in a CRAO patient's care, the emergency department must be considered the best site for initiating immediate normobaric high flow O_2 as this is where many patients will initially present.

We must note that the UHMS recommendations for the intermittent highest flow normobaric O_2 schedule of 15 min·h^{-1} was arrived at based on only three patients treated with normobaric O_2 received continuously for several hours . Patients who received interrupted high FiO_2 normobaric O_2 in fact received carbogen (5% carbon dioxide 95% O_2) which is more vasodilatory than plain O_2, theoretically improving retinal O_2 delivery. Given these issues, and with the aim of providing a simple and achievable protocol for which to follow, it may be suggested as an alternative to provide continuous high flow O_2 to patients. The initial treatment pressure of 284 kPa chosen by most ANZ HMUs has advantages over the UHMS guidelines which are complicated. Moreover, their lower recommended initial treatment pressure and subsequent increments based on treatment response may result in longer times before retinal oxygenation and therefore a delayed time until return of vision. We propose to use a more simplified approach with a higher starting pressure and less subsequent adjustments, such as that used presently at some Australasian HMUs. Starting at a higher initial pressure potentially may result in faster return of visual acuity and a minimisation of retinal ischaemic time.

It should be noted that data from 20 years' experience of O_2 toxicity seizures in patients undergoing HBOT from a single HMU demonstrate higher rates of OTS associated with higher treatment pressures. At 203 kPa, seizures occurred 2/17,512 (0.01%) or 1/8,756 treatments. The event rate for treatment at 243 kPa was 12/20,633 (0.06%) or 1/1719 treatments. At a pressure of 284 kPa, seizures occurred in 7/2,371 (0.3%) or 1/339 treatments.[13] This increase in seizure occurrence at higher treatment pressures necessitates appropriate consenting of patients as well as vigilance during treatment.

Variance in treatment schedule by specialists within a HMU seemed to correlate with infrequency of exposure to HBOT for CRAO.

Determination of the best HBOT schedule for CRAO requires ongoing research. It is our hope that this survey can serve to raise awareness of CRAO and its management with HBOT, as well as allow HMUs to consider other institutions' management and compare it against their own.

We propose a management guideline consistent with the majority of practice in Australasia as well as adapted from the UHMS guidelines and from the published CHCH as follows.[14]

- Any patient with sudden, painless vision loss suspicious for CRAO should be commenced on the highest fraction / flow of normobaric O_2 immediately and seen by an ophthalmologist urgently.
- If diagnosed with CRAO by an ophthalmologist and within the 24 h window from symptom onset, they should be immediately referred to a HMU and assessed for contraindications to HBOT. Patients affected in their only eye that has vision should be referred up to 5 days post symptom onset.
- An initial 18:60:30 treatment table or similar 284 kPa treatment should be the initial HBOT.
- With no improvement in vision after three 20 min O_2 breathing periods at 284 kPa, progression to a USN TT6 may be considered.
- Treatment should continue two or three times daily or until either resolution, clinical plateau or an angiogram confirms recanalisation / reperfusion.
- Ideally, patients should be admitted for at least the first 24 h with regular visual acuity checks.
- Visual acuity should be monitored following treatments.

Should visual loss recur, high flow normobaric O_2 should be administered continuously until repeat HBOT can be arranged.

- The HMU should closely liaise with the referring ophthalmologist throughout the patient treatment schedule.

Conclusions

This survey has shown that in those centres where CRAO is treated more frequently there exists agreement in how it is managed, with most having diverged from the UHMS guidelines specifically in the initial treatment schedule offered. The more simplified approach of initially treating with a 284 kPa table offers a more pragmatic way of treating CRAO and may potentially result in a reduced retinal ischaemic time thereby increasing the chances of restoring and preserving visual acuity. Our belief is that this benefit would outweigh the small increased O_2 toxicity seizure risk associated with the higher treatment pressure.

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