intensive care society

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Management of Patients With Gas Embolism:

Guidance For Intensive Care And Resuscitation Teams

Professional Endorsements

Society of British Neurological Surgeons
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British Hyperbaric Association

British Society of Interventional Radiology
Registered Charity No: 1084852

The Faculty of Intensive Care Medicine

Management of Patients With Gas Embolism:
Guidance For Intensive Care And Resuscitation Teams

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Preface

The Intensive Care Society requested assistance to develop national guidance on the detection, referral and treatment of air or gas embolism. The term gas embolism should be used in preference, as gases other than air can be involved under certain circumstances.

I would like to express my gratitude to the following colleagues who contributed substantially to various sections:

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Dr M Edsell, Consultant Anaesthetist, St George's University Hospitals NHS Foundation Trust.

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Dr K Valchanov, Consultant Anaesthetist, Royal Papworth Hospital NHS Foundation Trust.

Dr L Hudson, Research Fellow London Hyperbaric Unit, Anaesthetist

Mr Bruce Mathew, Consultant Neurosurgeon and Medical Director of North of England Medical Hyperbaric Unit, Hull.

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- Standards and Guidelines Committee, Intensive Care Society
- Professional Affairs and Safety Committee, Faculty of Intensive Care Medicine

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1. List of Abbreviations

AGE Arterial Gas Embolism
ASAP As soon as possible
BHA British Hyperbaric Association
CAGE Cerebral Arterial Gas Embolism
CGE Cerebral Gas Embolism
CPR Cardio pulmonary resuscitation
ECHO Echocardiography
HBO Hyperbaric Oxygen Therapy
HBU Hyperbaric unit
ICU Intensive-care unit
IPPV Intermittent Positive Pressure Ventilation
RCVGE Retrograde Cerebral Venous Gas Embolism
TOE Trans Oesophageal Echocardiogram
TTE Transthoracic Echocardiogram
VGE Venous Gas Embolism

Explanatory Terms:

1. **Durant manoeuvre**
   Placing the patient in a partial left lateral decubitus position.

2. **Millwheel Murmur**
   A characteristic splashing auscultatory sound due to the presence of gas in the cardiac chambers.

3. **Coroner's Reports to Prevent Future Deaths – PFD**
   In certain circumstances a person's death is reported to HM Coroner to determine whether an inquest must be held. The Coroner's Report to Prevent Future Deaths will set out the concerns and request that action should be taken.

2. Summary

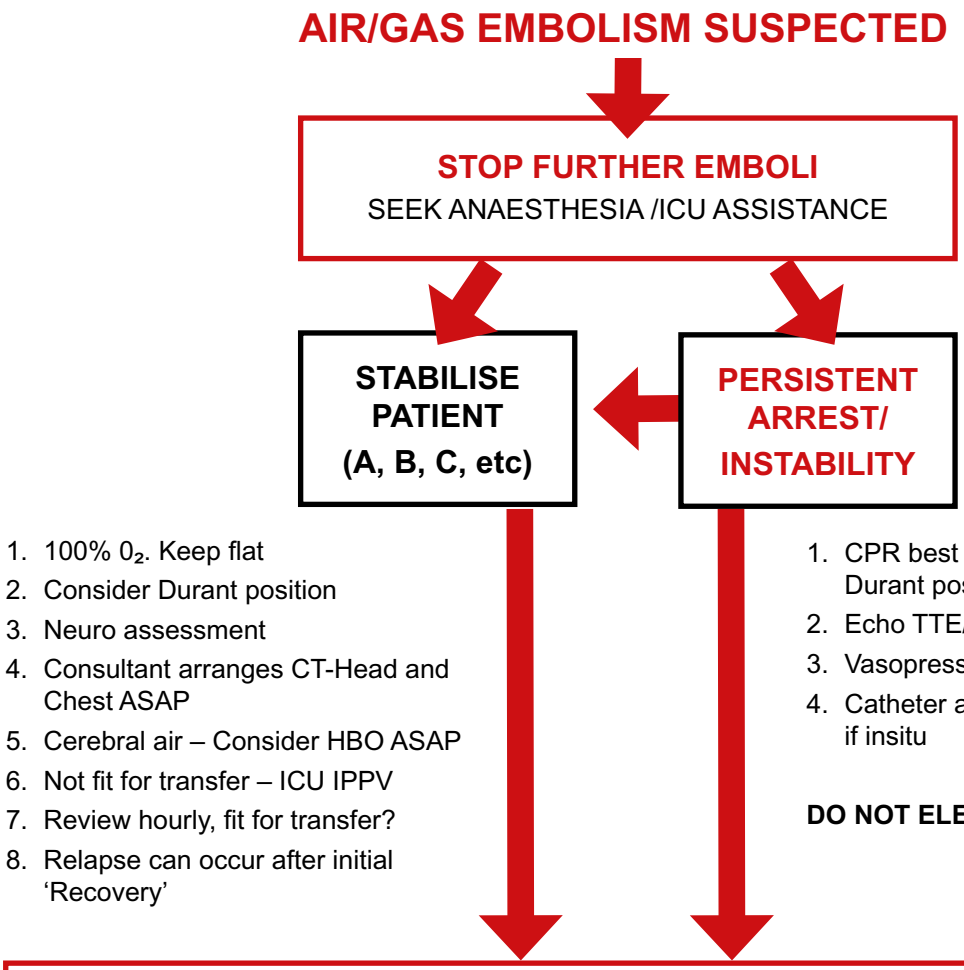
Vascular gas embolism is the entrainment of air (or exogenously delivered gas) from the environment into the venous or arterial vasculature, producing systemic effects.

The immediate management is to stop further entry of gas, life support according to the situational need, stabilisation of the patient and then removal of gas in the most appropriate way and with the facilities and skills available.

KEY POINTS

- Administer 100% Oxygen as part of patient resuscitation until gas embolism has been excluded or resolved.
- Always consider Vascular Gas Embolism in periprocedural stroke or neurological events, or if unexpected haemodynamic changes occur.
- Patients may deteriorate rapidly and develop irrecoverable brain damage or cardiac arrest. Occasionally there may be some improvement with subsequent relapse.
- Consider early discussion with the local/nearest Hyperbaric Unit.

Summary flowchart of approach to suspected gas embolism:



Not all HBUs have consultant anaesthetists in the unit available 24/7

Most units have a 1h callout time after hours

Early contact will facilitate ICU/ anaesthesia staffing arrangement in the nearest unit or transfer further afield.

Aberdeen	0345 408 6008
Chichester	01243 330 096
Great Yarmouth	01493 452 452
Hull	01482 659 471
London (Whipps Cross)	07999292999
Rugby-Midlands	07940 353 816
Oban	0345 408 6008
Plymouth	07831 151 523
Wirral	0151 648 8000

CONTRAINDICATIONS

Bullous lung disease

Undrained – Pneumothorax

Others are relative (discuss risk/benefit)

**The aim of Urgent CT imaging is to rule out another cause for the clinical picture; should not delay urgent Hyberbaric referral in obvious cases.

Air bubbles may disappear but damage persists in many cases!

3. Introduction

Arterial gas embolism involves gas entering the arterial circulation with embolism to distal capillaries, resulting in ischaemia. This is possible to every organ system but the effects are particularly profound when embolism occurs in the cerebral or coronary circulation. (1-3)

Venous gas embolism involves gas entering the venous circulation with gas passing into the right atrium, then into the right ventricle and onwards to the lungs where some or all of it can be filtered. Large amounts of gas over a short period can lead to catastrophic right-sided cardiac failure and cardiovascular arrest.

All causes potentially result in a life-threatening condition and should be regarded as a dire emergency. Intra-cardiac air may lead to cardiac arrest or, if the quantity allows ongoing cardiac output in a supine patient, embolisation usually to the right coronary artery, (but occasionally the left) leading to arrhythmia or cardiac ischaemia. In the sitting position (e.g. during neurosurgery) it would be more likely to embolise to the cerebral circulation.

It is also essential to keep in mind that most episodes of VGE are preventable and no effort should be spared to put in place essential preventative steps.

4. Aetiology

A. Arterial Gas Embolism (AGE)

Gas can enter the arterial system through:

1. Cardiovascular Procedures

a. Imaging
b. Interventional
c. Surgical

2. Gas Entering Pulmonary Veins

a. Barotrauma during mechanical ventilation
b. Chest trauma
c. Rapid ascent from diving activities
d. Rapid decompression of a pressurised aircraft
e. Blast injuries

3. Paradoxical Arterial Gas Embolism (4)

a. Gas entering arterial circulation through an intracardiac right-to-left shunt
b. Arteriovenous malformation in the lungs
c. Overwhelming of the pulmonary capillary filter mechanism

B. Venous Gas Embolism (VGE)

Gas can enter the venous system through:

1. Venous line insertion, management and removal
a. Central lines
b. Peripheral Lines
2. Open venous channels after trauma or during surgery when there is a pressure gradient between the wound at atmospheric pressure and negative intrathoracic pressure
3. Insufflation of gas in a body cavity with open venous channels

C. Retrograde Cerebral Venous Gas Embolism (RCVGE) (5, 6)

Due to buoyancy of gas in the venous system, gas bubbles can migrate upwards into the cerebral venous system where the neurological effect depends on which area of the brain is affected.

Relative risk of Gas Embolism (7):

In this review, Mirski et al. quote a large number of reports which concur with other authors that certain conditions are clearly categorised as high-risk as opposed to others in a medium or low risk.

Individual authors obviously have different experience in their units. The following give an indication of the variability. Percentage of all gas embolism reported by various authors: The highest group varying from 9-80%, medium risk from 5-12%, and lower risk 2-10% of referrals.

• Sitting position craniotomy	High
• Posterior fossa/neck surgery	High
• Laparoscopic procedures	High
• Total hip arthroplasty	High
• Caesarean delivery	High
• Central venous access–placement/removal	High
• Craniosynostosis repair	High
• Spinal fusion	Medium
• Cervical laminectomy	Medium
• Prostatectomy	Medium
• Gastrointestinal endoscopy	Medium
• Contrast radiography	Medium
• Blood cell infusion	Medium
• Coronary surgery	Medium
• Peripheral nerve procedures	Low
• Anterior neck surgery	Low
• Burr hole neurosurgery	Low
• Vaginal procedures	Low
• Hepatic surgery	Low

Open Heart Surgery e.g. aortic valve replacement

CT-guided percutaneous lung biopsy

Ingestion of Hydrogen Peroxide

The latter 3 were not included in Mirski's table, but in this author's experience are at least a medium risk.

5. Pathogenesis

Venous Gas Embolism (VGE):

Large volumes of gas entering the venous system rapidly leads to circulatory collapse due to cessation of cardiac output. This can happen extremely rapidly and patients may not respond to resuscitation.

Smaller volumes of VGE can lead to temporary haemodynamic disturbance and then depends on what happened to the emboli – filtered by the lung, with full recovery or retrograde flow to the brain or becoming arterialised with subsequent organ ischaemia.

Arterial Gas Embolism (AGE):

Large volumes of gas entering the arterial system will result in immediate coronary or cerebral embolism and death.

Smaller volumes may reach peripheral capillary beds with less severe consequences. The work done on intra-coronary artery gas embolism in an animal model has demonstrated that as little as 0.02ml/kg can lead to death, so all gas embolism should be regarded as a serious issue (4).

The bubble surface acts as a foreign substance and activates the coagulation cascade with the following effects:

- Increased levels of C3a and C5a
- Prostaglandin and leukotriene synthesis
- Platelet and leukocyte activation, leading to ongoing impairment of microcirculation
- Fibrin release and adhesion to endothelium
- Vasospasm followed by vasodilatation
- If cerebral vessels involved: Damage to the blood brain barrier. Cerebral oedema and raised intracranial pressure, which can ultimately cause cardiorespiratory arrest and death if not interrupted
- Despite this, some cases sometimes recovered spontaneously but should not detract from active management.

6. Diagnosis

The diagnosis is often difficult, particularly in the early stages, when a small amount of gas is involved; or if the patient is anaesthetised or sedated. A constant awareness of the possibility is necessary, particularly in situations that are not classically associated with a high risk of gas embolism.

The clinical diagnosis may be clear-cut with catastrophic cardiac arrest but in some instances the symptoms may be subtle.

1. **Symptoms:** the awake patient can become restless, appear anxious and may express a feeling of impending doom. Dyspnoea, chest pain and a variety of neurological symptoms may be elicited from the patient.
2. **Cardiovascular signs:** a variety of arrhythmias, from tachycardias to asystole. Blood pressure variation with hypotension and profound cardiogenic shock is occasionally seen. Jugular venous distension can sometimes be seen as a manifestation of pulmonary hypertension and a 'mill wheel' murmur may be audible
3. **Neurological signs:** may be very subtle or obvious, with altered mental status, seizures, hemiparesis or coma.
4. Some very subtle but **diagnostic clinical signs** may occasionally be seen, e.g. cutis marmorata, a marble skin rash. Careful fundoscopy may demonstrate gas bubbles in the retinal vessels and examination of the tongue may show Liebermeister's sign, pallor of the tongue due to arteriolar obstruction with gas bubbles (8).
5. A sucking noise is sometimes reported when air enters an open vessel or vascular access device.

A periprocedural stroke or neurological event should always be regarded as gas embolism with cerebral involvement until proven otherwise.

In many cardiac procedures a trans-oesophageal echo probe will be in place and will pick up air bubbles at an early-stage and help to prevent or reduce the impact and progression of air embolism. In other cases where air embolism is suspected, a quick transthoracic echo may show microbubbles entering the heart or in some instances bubbles or collections of air may be seen in the cardiac chambers, major thoracic vessels and even high up in the jugular venous system. Lack of such findings does not exclude air embolism though.

Imaging, e.g. CT/ MRI of the brain may indicate gas bubbles, or rarely in the acute phase ischaemic injury. These are often not localised to single vascular territories. The use of contrast will also help to differentiate between arterial and venous involvement. Absence of bubbles does not exclude gas embolism if the clinical picture was clear. CT imaging is a very easy modality to rule out cerebral haemorrhage to avoid having a patient committed to a long treatment if potential surgical intervention is required. The urgency of CT imaging needs to be made very clearly by consultant to consultant referral.

7. Immediate management

Goals

A. Minimise further air entrainment

B. Maintain oxygenation and provide haemodynamic support

C. Reduce the size of embolism

D. Overcome mechanical obstruction caused by embolism

E. Stop or minimise further air entrainment immediately:

F. Check all intra vascular access devices for their integrity, proper connections, valve function, air in lines and correct any defects

G. Check sites of recently removed central lines, haemodialysis line etc for signs of patency

H. Notify the surgeon to identify and eliminate a surgical port of gas access

I. Lower the surgical field to below the level of the heart

J. Deflate the viscus or body cavity if under pressure

K. Maintain oxygenation and provide haemodynamic support according to the ABC approach

L. Reduce the size of embolism by using the highest possible FiO_2

Traditionally the Trendelenburg position or the Durant manoeuvre were suggested to relieve the air-lock in the right side of the heart, but adequate CPR is not easy in this position. In the Trendelenburg position cerebral oedema is more likely to develop quickly and should be avoided except for an initial short period to avoid bubbles entering the cerebral circulation.

Another controversy has arisen around inserting a central line to aspirate air from the heart in the event of an “air lock” in the right ventricle. When there is already a catheter near the right atrium or ventricle aspiration could be attempted.

Chest compressions may help to force air out of the pulmonary outflow tract into the smaller pulmonary vessels, thus improving forward blood flow.

8. Further management: HBO for persistent symptoms, particularly neurological symptoms

Although there are case reports of gas being aspirated percutaneously, the most effective treatment is urgent HBO (3, 9, 10) and should always be considered as urgent management. The ideal therapeutic window is 6-7h (9, 11), however delayed treatment should still be considered and discussed with experts in the field.

The pathophysiology of gas bubbles in the cerebral vascular system and the mechanism of HBO has been clearly described (2, 4, 12). Once the diagnosis is made, every minute counts. For that reason, clear guidance is essential. Air transport as low as safely possible is ideal, preferably lower than 1000 ft.

For hyperbaric assistance contact your nearest available BHA member chamber, at the address and phone number in the list below. If you do not know the location of your nearest BHA member chamber then call:

National Diving Accident Helpline on 07831 151523 (24 hrs).

MECHANISM OF ACTION OF HBO

- Reduce the size of emboli (Boyle's Law)
- Remove nitrogen from emboli by removing nitrogen from the blood and tissue (The hyperoxia produces enormous diffusion gradients for oxygen into the embolus and nitrogen out of the embolus)
- Improve oxygen delivery to tissues in the ischaemic penumbra
- Reduce intra-cranial pressure by causing constriction of cerebral arteries
- Hyperbaric oxygen inhibits membrane guanylate cyclase, which in turn inhibits β2 integrin adherence and decreases leukocyte 'stickiness', improving the microcirculation.

9. Further research is required to resolve uncertainties and improve outcome

Animal research has helped a lot to understand the pathogenesis of CGE but has unfortunately also led to the perception that the body can safely deal with large volumes of gas. A very important caveat is not mentioned in any of these estimates - the animal model is usually in the horizontal position when exposed to the intervention. In such situations the preferential route of venous gas embolism in the animal model would be from the site of entry to the right atrium, then the right ventricle and then the lungs. The lungs provide a filter mechanism for intravascular gas and probably explain why large quantities are survivable.

Quoting the large volumes tolerated by animals ignores the work done by Schlimp et al on the alternative RCVGE mechanism of cerebral injury (5, 6, 13). They have shown that through the buoyancy of venous gas bubbles, gas can bypass the heart by ascending against the venous blood flow to the cerebral venous system in patients that are in the Fowlers or in the semi-Fowler's position. These cases have been shown to lead to venous infarcts and even death (14). These cases are not protected by the filter mechanism of the lungs and have not been studied in animal models.

In humans it is hardly ever possible to calculate the quantity of gas in the circulation. This should be an area of focus if a registry or a prospective study is put in place. Another important issue to be investigated prospectively is to see if RCVGE has a longer window for hyperbaric treatment.

10. Prevention of Vascular Access Device related Gas Embolism

Training of the staff involved with insertion, management and removal of vascular lines is an important step in reducing the risk of this serious complication of modern medicine. Attention to detail is essential. A recent Coroner's Report to Prevent Future Deaths mandates urgent action to prevent gas embolism.

https://www.judiciary.uk/wp-content/uploads/2016/05/2016-0201-Response-by-NHS-Improvement.pdf

Careful explanation to conscious and cooperative patients how the procedure is done and what is expected of them is important. Unfortunately, most patients that are critically ill cannot be relied upon to follow and remember the instructions. For such patients it is important to be extra vigilant and have extra measures in place to reduce the risk.

Preparation, De-airing (Priming)

All central lines and their connections, as well as all administration tubing, transducer sets and syringe drivers should be carefully de-aired. Preparation is usually done by nursing staff who are used to cross-checking administration of drugs with a colleague. Checking of complete de-airing or 'priming' with a colleague will add minimal extra workload and contribute to safety, along the same lines as checking intravenous drugs.

When setting up arterial line pressure transducers, it is essential to remove even the small amount of air normally present in a 500 ml Saline bag. Eliminating all air from the drip chamber by keeping it inverted when priming the line also decreases the risk of arterial air embolism. When a bag of intravenous fluid is removed from the administration set, air is sucked into the bag and should not be re-used. (Ruby's rule)

Before starting the insertion process, all lumens of central lines should be flushed with saline. Ensure that all self-sealing valves are functioning properly, correctly connected and that the clamps are applied. The clamps should also remain applied when the CVC is not in use. Only the lumen to the distal tip should be left open for the guidewire, but as soon as insertion is completed, it should also be fitted with a valved needleless connector and securely clamped.

Insertion of CVC

The use of ultrasound has become mandatory in most departments and also allows the operator to assess the degree of hydration and the need for steep Trendelenburg position to fill the veins in dehydrated patients or to allow slight head up positions in patients who can't tolerate the Trendelenburg position but who have well filled veins. When possible, it is worthwhile to avoid a short subcutaneous path to the central vein. This method decreases the risk of air embolism during catheter removal as a longer tract is less likely to remain open. Securing central access devices to the skin needs special care, as accidental removal or partial removal has frequently led to air embolism.

Safe management of lines

Unused taps attached to CVCs should be closed to air and capped off with an appropriate device. Some central lines may be packaged with non-occlusive caps pre-attached to facilitate sterilisation. These must be removed and replaced with occlusive caps when the line is first flushed. All CVCs should also have clamps associated with each lumen of the line, and these should be used to clamp any lumens that are not in use as an additional safety measure.

Infusion pumps should have air-in-line sensors for all continuous infusions as very small amounts of air could have serious effects in neonates and infants(15). Fluid warmers, high volume resuscitation devices and extra-corporeal circuits also should have very efficient bubble removal /warning systems. Pre-filled syringes also have a small amount of air and these particularly, should always be kept with the plunger vertically above the IV connector and not emptied completely. Extra care is required when turning or mobilising a patient with a central line, to avoid partial dislodgement and allowing one orifice of a multiorifice catheter to be outside the skin.

Removal of CVCs

Intravascular access devices should be removed when they are no longer required or are causing problems. Nursing guidelines universally indicate the need for the patient to be in the Trendelenburg position with the exit site below the heart to reduce risks of air embolus. Firm digital pressure should be applied for at least 5 min beyond haemostasis, followed by an occlusive dressing, and instructions for the patient to remain lying flat for 60 minutes after removal of the catheter. This is based on the recent guideline after fatal haemorrhage seen after removal of a dialysis catheter.

https://renal.org/patient-safety-alert-response-reported-death-blood-loss-following-removal-temporary-femoral-dialysis-catheter/

It is important to keep in mind that in some instances a patient may be discharged home shortly after lines have been removed. Some patients may not remember instructions and may allow the occlusive dressing to get wet and may remove it. Some patients will not tolerate lying flat even for 5 min and many are unable to breath-hold or do a Valsalva manoeuvre. In such patients and those where a patent tract may exist, it is advisable to seal the orifice with tissue adhesive (Cyanoacrylate). This adds only a few seconds to the removal procedure but yields significant additional security. This is of importance particularly when larger catheters have been used, or lines were in situ for longer periods and in older patients with less elasticity in surrounding tissue, or in dry, scarred skin (16-18). The case can be made to routinely seal all central access line tracts like this.

Peripheral lines

Peripheral venous lines are not innocuous and the same amount of care with priming, secure connections, etc. should be taken (19, 20).

Prefilled glass syringes are not compatible with all needle-free devices, as the internal pathway of the glass syringe tip is significantly narrower than a standard plastic syringe. The size of the glass syringe internal pathway can also vary considerably, even with syringes from the same batch. This smaller than normal internal pathway of the glass syringe and inconsistency in its diameter, may cause an incomplete connection with the IV fluid pathway inside some needle-free devices, and has been identified as a risk of air embolism.

Constant vigilance when using vascular access devices is essential.

11. Conclusion

This is an uncommon but serious complication. When it occurs, emergency medical management should be followed by Duty of Candour as soon as possible. For the recommended approach to suspected gas embolism please refer to the summary flowchart page 7. Discussions with clinical governance leads should take place to share learning about cases of gas embolism when they are presented. When a patient dies from gas embolism, the Coroner will be involved and the death certificate will be used to collate data. This data collection will help to provide better statistics about the prevalence of gas embolism and our understanding in the long term.

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13. Contact details of Hyperbaric units:

Aberdeen

Aberdeen Royal Infirmary,
Foresterhill,
Aberdeen
AB25 2ZN
Daytime & Emergency Telephone: 0345 408 6008
Clinical Lead NHS Grampian Hyperbaric Service: Dr Ruth Stephenson
Email: ruth.stephenson@nhs.net
Web Site: www.hyperchamber.com

Chichester

Hyperbaric Medicine Unit,
St Richard's Hospital,
Spitalfield Lane,
Chichester,
West Sussex
PO19 6SE
Daytime Telephone: 01243 788122 Ext 2504
Emergency: 01243 330096
Principal Medical Director: Dr Mark Glover.
Email: Mark.Glover@qinetiq.com

Great Yarmouth

East of England Hyperbaric Unit,
Lowestoft Road,
Gorleston,
Great Yarmouth,
Norfolk
NR31 6LA
Daytime Telephone: 01493 452452
Emergency Telephone: 01493 452452 or 01493-603 151
Principal Medical Director: Dr Pieter Bothma.
Email: pabothma@gmail.com
pieter.bothma@nhs.net (for patient related correspondence)

Hull

North of England Medical Hyperbaric Unit,
BUPA Hospital,
Lowfield Road,
Anlaby,
Hull
HU10 7AZ
Principal Medical Director: Dr Bruce Mathew
Daytime Telephone: 01482 659471
Emergency Telephone: 01482 659471

London (Whipps Cross)

London Hyperbaric Medicine,
Whipps Cross University Hospital NHS Trust,
Leytonstone,
London
E11 1NR
Principal Medical Director: Dr Pieter Bothma
Daytime Telephone: 020 8539 1222
Emergency Telephone: 07999 292999
Email: mail@londonhyperbaric.com
Web Site: www.londonhyperbaric.com

Midlands Diving Chamber

Redwood House,
Hospital of St Cross,
Barby Road,
Rugby,
Warwickshire,
CV22 5PX
Principal Medical Director: Dr Michael Gonevski
Other contact: Mathew Wormleighton - mat@midlandsdivingchamber.co.uk
Daytime Telephone: 01788 579555
Emergency Telephone: 07940 353816
Email: doctor@midlandsdivingchamber.co.uk
Web Site: www.midlandsdivingchamber.co.uk

Oban

West Scotland Centre for Diving and Hyperbaric Medicine
Tritonia Scientific Ltd.,
Dunbeg,
Oban,
Argyll
PA37 1QA
Principal Medical Director: Dr C.M. Wilson
Daytime Telephone: 01631 559211
Emergency Telephone: 0345 408 6008
Email: colinwilson@tiscali.co.uk or martin.sayer@tirtoniascientiic.co.uk

Plymouth

DDRC Healthcare, Plymouth Science Park,
Plymouth,
Devon
PL6 8BU
Principal Medical Director: Dr Clair Ashford
Daytime Telephone: 01752 209999
Emergency Telephone: 07831 151 523
Email: info@ddrc.org
Web Site: www.ddrc.org

Wirral

N W Emergency Recompression Unit,
Murrayfield Hospital,
Holmwood Drive,
Thingwall Wirral
CH61 1AU
Principal Medical Director: Dr Tristan Cope
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Emergency Telephone: 0151 648 8000
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