DECOMPRESSION SICKNESS

Extracted with permission from Diving and Subaquatic Medicine (1975)
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Following the development of the air pump by von Guericke in 1650, Robert Boyle was able to expose animals to decompression. In 1670 he reported these experiments and included the first description of decompression sickness - a bubble moving to and fro in the waterish humour of the eye of a viper. Hoppe-Seyler repeated these experiments n 1857, and correctly explained the mechanisms concerned. Admiral Sir Thomas Cochrane's pneumatic technique was used by Triger in France in 1839 in the development of a caisson for the sinking of coal shafts through the wet soils of Chalons. This was followed by the widespread use of the compressed air environment to allow for tunnelling, mining and caisson sinking. Trevessart and then Pol and Watelle observed Triger's subjects and the physiological and pathological manifestations of decompression.

Hoppe-Seyler repeated the experiments of Boyle. In 1857 he described the obstruction of pulmonary vessels by bubbles and the inability of the heart to function adequately under those conditions. He also recommended recompression to remedy this.

Le Roy de Mericourt in 1869, and Gal in 1872, described an occupational illness of sponge divers, which was also attributed to the breathing of compressed air and equated this with the caisson workers' disease. A host of imaginative theories was proposed during the 19th century to explain the aetiology of this disorder. It was followed by equally diverse mathematical models during the 20th century, also purporting to explain the same illness.

Paul Bert, in 1878, demonstrated in a most conclusive manner that decompression sickness is primarily the result of an inert gas (nitrogen in the case of compressed air divers and caisson workers) which had been dissolved according to Dalton's and Henry's Laws, and then released during or following decompression.

Haldane, Boycott and Damant in 1907 supported the concept of stage decompression where the diver ascends with a series of stops. It is based on the hypothesis of multiple tissue saturation rates and a critical super-saturation ratio necessary for bubbles to form. Most decompression tables still in use have their basis in this work, although the single critical ratio has now been replaced with multiple critical ratios for the different tissues at different depths.

Leonard Hill in 1912 produced both experimental and theoretical evidence questioning the value of stage decompression over continuous decompression, and this latter technique is now applied to decompression from saturation exposures.

Clinical Manifestations

Grading: A classification was presented as an attempt to differentiate non-serious and serious cases, so that identification prognosis and therapy could be more appropriately standardised. This clinical classification of Type I and Type II decompression sickness is not wholly satisfactory. The classification was neither defined nor applied in the same spirit. Type I is defined as (musculoskeletal) pain only bends. Type II included those presenting with symptoms other than pain, or with abnormal physical signs. The central neurological, cardiovascular, respiratory and gastrointestinal manifestations are potentially serious. Nevertheless in most series peripheral nerve symptoms are allocated to the same group as spinal and cerebral. Nor is there a differentiation made between vestibular and cerebellar presentations.

Although the appellation of Type I is reasonably clear when it is applied to acute decompression sickness affecting the musculoskeletal system ("joint bends"), the conglomeration of Type II manifestations, ranging as they do from an inner ear lesion to abdominal cramps to patches of hypoaesthesia to cardiovascular collapse, is of much less value. The classification is so commonly modified, even by its own proponents, that it appears to have lost any merit it originally had. The qualifying designation of the organs affected appears to be the only logical approach, eg. decompression sickness affecting the inner ear and musculoskeletal systems.

Onset. Decompression sickness develops after the subject has commenced decompression or ascent. Most cases present within 6 hours of the dive. In long or deep dives, cases may commence during the ascent. The delay in onset between initiating decompression and developing symptoms is influenced by individual and operational variations. The figures available must only be interpreted with an appreciation of the population being considered and the diving being performed. Some of the typical figures are presented in Table 1.

TABLE 1

POPULATION CHARACTERISTICS	TIME OF ONSET FROM SURFACING (percentage incidence)								
	0-30 mins. to 1 hour	30 mins. to 1 hour	2nd hour	3rd hour	4-6th hour	7-12 hour	13-24 hour	>24	Unknown
Royal Navy Divers	57	10	13	14		6	1		
Canadian Navy Divers	62	12	9	6	9	3	1		
US Navy divers (1)	54.7		12.1		19.5	6.6	2.3	0.3	4.5
US Navy divers (11)	43.9		27.4				3.9		
Hawaiian Civilians	66	6	4	3	2	5			14

The Naval series were heavily weighted for saturation exposures, whereas the civilian series was characterised by totally omitted decompressions. Thus the latter had few cases during decompression, the former had many. The civilian group was characterised by rapid and uncontrolled rates of ascent thus the incidence of early symptoms attributable to intravascular bubbles was greater. With an early presentation of symptoms, there are two diverging effects. The illness is potentially more severe, but treatment is more likely to be available.

Correlation of Symptoms

Although it is often stated that patients in the quoted series suffered from more than one symptom, the correlation between the different manifestations is not usually given. In some series, especially from cassion workers, the cumulative totals equal 100%, this suggests that not only the major symptom is documented. The two diver series which do show correlations between symptoms are fortunately the two extremes - one with US Navy controlled divers often in recompression chambers, and the other with uncontrolled diving civilians. The Navy divers represented a less severely affected group, the civilians more affected.

TABLE 2

N = 935

SIGN OR SYMPTOM	NO. OF INSTANCES	% OF INSTANCES	NO. OF INSTANCES MANIFESTED INITIALLY	% OF INSTANCES MANIFESTED INITIALLY
Localised Pain	858	91.8	744	79.6
Numbness or Paraesthesia	199	21.2	41	4.3
Muscular Weakness	193	20.6	8	0.8
Skin Rash	140	14.9	42	4.4
Dizziness or Vertigo	80	8.5	24	2.5
Nausea or Vomiting	74	7.9	8	0.8
Visual Disturbances	64	6.8	14	1.4
Paralysis	57	6.1	2	0.2
Headache	37	3.9	5	0.5
Unconsciousness	26	2.7	6	0.6
Urinary Disturbances	24	2.5	0	
Dyspnoea (“Chokes”)	19	2.0	4	0.4
Personality Changes	15	1.6		
Agitation or Restlessness	13	1.3		
Fatigue	12	1.2	2	0.2
Muscular Twitching	12	1.2		
Convulsions	11	1.1		
Inco-ordination	9	0.9		
Equilibrium Disturbances	7	0.7		
Localised Oedema	5	0.5		
Intestinal Disturbance	4	0.4		
Auditory Disturbance	3	0.3		
Cranial Nerve Involvement	2	0.2		
Aphasia	2	0.2		
Haemoptysis	2	0.2		
Emphysema - subcutaneous	1	0.1		

TABLE III

N = 100

Clinical Manifeatations	Incidence	Percentage incidence of associated clinical manifestations										
		Cerebral	Inner Ear	Spinal	Peripheral Nerves	Respiratory	Gastro-intesti-nal	Spinal or Back pain	Headache	Musculo-skeletal	Apathy and Malaise	Skin
Cerebral	24%		29	17	4	33	17	13	25	38	21	8
Inner Ear	18%	39		11	17	11	28	-	11	33	28	-
Spinal	22%	18	9		-	32	27	23	9	27	14	-
Peripheral Nerve	11%	9	27	-		18	36	-	9	64	9	9
Respiratory	21%	38	10	33	10		14	19	19	52	10	10
Gastro-Intestinal	16%	25	31	38	25	19		6	19	38	25	-
Spinal or Back Pain	11%	27	-	45	-	36	9		27	45	-	-
Headache	11%	55	18	18	9	36	27	27		27	36	-
Musculo-skeletal	54%	17	11	11	13	20	11	9	6		7	7
Apathy and Malaise	4%	38	38	23	8	15	31	-	31	31		-
Skin	3%											

Cutaneous

These manifestations range from being local and innocuous, to generalised and ominous, with a complete spectrum in between. They have been variously described as follows:

1. *Pruritis* A common complication of "diving" in compression chambers. It is often a transient effect, presenting very soon after decompression, and is not considered a systemic manifestation of decompression sickness.

   It is noticed mostly after short deep exposures, often with only one or two decompression stops. The areas affected are the forearms and wrists and hands, the nose and ears, and the thighs. The symptoms are transient and there is usually no objective sign available. In other cases there may be a slight folliculitis observed as red punctate areas, when this presentation merges with the next. The symptoms are attributed to small gas bubbles in the superficial layers of the dermis, and especially near its entry through the epidermis and the sebaceous glands.

2. *Scarlatiniform rash* This is an extension of the above manifestation. It is associated with pilo erector stimulation and perhaps a tissue histamine release and appears as a red punctate rash. The distribution is predominantly over the chest, shoulders, back, upper abdomen and thighs, in that order. The duration is from minutes to hours.

3. *Erysipeloid rash* This is an extension of the scarlatiniform rash and occurs over the same distribution, but with the involvement of endogenous gas interfering with venous drainage, it is a definite sign of systemic decompression sickness. Some of the skin appearance is thought to be a reflex vascular reaction. The lesions are collections of papules which may merge to form large plaques with flat and firm borders. Coughing or performing the Valsalva manoeuvre will accentuate the venous markings (Mellinghoff's sign).

4. *Cutis Marmorata Marbleization* This commences as a small pale area with cyanotic mottling. It may spread peripherally becoming erythematous with extension of cyanotic mottling. The area is warmer than the surrounding skin. Swelling and oedema result in a mottled appearance. Recompression gives dramatic relief. The area may become tender to palpation in a few hours, but the other signs may have diminished or disappeared by then. Marbling of the skin is a cutaneous manifestation of what is occurring elsewhere in the body, and thus is a serious sign of decompression sickness. Gas bubbles are present in both tissues and blood vessels.

5. *Subcutaneous emphysema* This has the typical crepitus sensation on palpation, either in localised areas or along tendon sheaths. It can be verified radiologically and should not be confused with the supraclavicular subcutaneous emphysema extending from the mediastinum and due to pulmonary barotrauma.

6. *Lymphatic obstruction* This presents as a localised swelling which may be associated with an underlying decompression sickness manifestation. If it involves hair follicles, a peau d'orange or pigskin appearance with brawny oedema is characteristic. It is common over the trunk, but is also seen over the head and neck.

7. *Counter diffusion of gases* There have been occasional reports of skin and mucosal swellings due to counter diffusion of gases. This results in bubbles forming at the interface between 2 gases diffusing at different rates, but with the total gas pressure at the interface exceeding the environmental pressure. This is only likely when the subject's body is exposed to a light readily diffusible gas, while breathing a heavier slower diffusing gas. The result is that the light gas moves rapidly through the body surface from the environment, while the inhaled gas maintains high tissue pressure and is reducing this slowly.

8. *Others* Formication may be the presentation in any of the skin manifestation described above, or due to involvement of the peripheral nervous system or the spinal cord. The neural involvement may also result in numbness, hypoaesthesia, paraesthesia and hyperaesthesia of the skin. These signs of inflammation may also occur over affected joints. Bruising is sometimes described over the chest and abdomen in serious cases, but this is not due to genuine tissue haemorrhages, as it blanches on local pressure.

Musculoskeletal

This is also termed "joint bends", "Type I", "pain only bends", "decompression arthralgia", etc. First, there is an ill-defined discomfort or numbness poorly localised to a joint, periarticular or muscular area. The subject may protect or guard the affected area, although in the early stages he may get some relief by moving the limb. Over the next hour or so the discomfort develops into a deep dull ache, then a pain with fluctuations in intensity, sometimes throbbing and occasionally with sharp exacerbations. Limitation of movement is due to pain, and the limb is placed in a position which affords the most relief. The duration of pain is often related to the severity of symptoms. The shoulder is the commonest joint affected in divers, in approximately one third of cases. Other joints, about equally affected, are the elbows, wrists, hands, hips, knees and ankles. Often, when two joints are involved, they are adjoining and frequently the localisation is between joints over the scapula, on tendon insertions etc. Rarely is the involvement symmetrical. The application of local pressure by means of a sphygmomanometer cuff, may result in considerable relief and may be of diagnostic value. It has been claimed that the site pain can sometimes be transferred by massage of the area.

In the mild cases, fleeting symptoms are referred to as "niggles", and may only last a few hours. The pain of the more severe cases usually increases over 12-24 hours and, if untreated, abates over the next 3-7 days to a dull ache. Local skin reactions may occur over the affected joint.

Although the musculoskeletal symptoms are the commonest presentation of decompression sickness in humans, the pathology of this is not well understood. Radiological evidence of gas both in joint spaces and periarticular lesions is available, but this is not necessarily the causative lesion. Extravascular bubbles in the subperiosteal area, tendons, ligaments, joint capsula tissue, fascia and muscles are thought to cause the pain of "bends". Bubbles in the articular vascular supply and referred neurological pain have also been incriminated.

Neurological

These presentations have produced a great deal of the past interest in decompression sickness. The areas affected can be subdivided according to the level of the nervous system affected.

The following clinical subdivisions of neurological presentations are:

Cerebral Any cerebral vessel may be occluded by gas bubbles, and this result in a great variety of manifestations, analogous to those of the cerebro-vascular accidents of general medicine. Especially noted are the homonymous scotomata, unilateral or bilateral, single or multiple. Other manifestations include hemiplegia, monoplegia, focal or generalised convulsions, aphasia, alexia, agnosia hemisensory or monosensory disturbances and confusional states. Raised intracranial pressure has been noted, and this may be associated with severe headache. In cases of homonymous hemianopsia electroencephalographic slow waves have been reported over the ipsilateral occipital cortex. Repeated EEG's are usually indicated during convalescence.

Serial, non-cultural, psychometric assessments of cognitive function may be of value if given before, during and after treatment. They provide measurement of mental impairment and response to treatment. Permanent mental impairment has been claimed as sequelae of cerebral decompression sickness. Brain stem involvement may also result in cranial nerve and pupillary abnormalities.

Cerebellar This results in ataxia, inco-ordination with typical neurological signs of hypotonia, diminished or pendular tendon reflexes, dysmetria, asynergia, tremor, dysdiadokokinesis, rebound phenomenon, scanning speech and nystagmus. The "staggers" which is variously described as vestibular, posterior column, spinal cord and cerebral decompression sickness, is probably more often due to cerebellar lesions.

Spinal Involvement of the spinal cord is most common in the lower thoracic or upper lumbar segments, although cervical lesions are by no means uncommon. It is often preceded by girdle pains, and commonly results in a paraplegic state. It is more common in patients who also have respiratory symptoms ("chokes"). The symptoms and signs are those of paraplegia or paraparesia, and include urinary retention with overflow incontinence. Often there is sparing of some sensory long tracts.

Peripheral nerve Bubble formation in the myelin of peripheral nerves will result in a patchy sensory deficit predominantly involving the lower limbs. The differentiation between this and an incomplete spinal lesion is important.

Pathological lesions in the neurological system include perivascular haemorrhages, oedema and demyelination in the cortex and subcortex, cerebellum, brain stem and spinal cord. The spinal cord changes are predominantly in white matter, and are most often observed in the mid-thoracic, upper lumbar and lower cervical areas, with the lateral, posterior and anterior columns suffering in that order.

The neurological manifestations of decompression sickness are explained in the following sequence of events. Gas bubbles form in the circulating blood after a short latent period after decompression. Most bubbles are filtered out by the lungs; some

bubbles, however, pass through the lungs, either by small arteriovenous anastomoses, or through the capillary lakes of Sjöstrand. These bubbles are small, about 25μ in diameter. They pass through the heart and reach the central nervous system and occlude arterioles of lesser calibre. The clinical manifestations, depending on the site of vascular obstruction and collateral supply are largely a matter of chance.

The multiple pulmonary gas emboli have secondary detrimental effects by raising pulmonary arterial pressure, and this may predispose to paradoxical cerebral embolisms through a patent foramen ovale, atrial septal defect or intra pulmonary arteriovenous shunts. Also of relevance is the interference with venous drainage of the spinal cord and subsequent damage to the cord. Some spinal cord and anterior horn damage may be due to spinal artery obstruction from an embolus. More importance is placed on the pulmonary hypertension interfering with venous drainage through the anastomoses of the spinovertebral-azygos system, subsequent engorgement and thrombosis in the vertebral venous system and oedema and infarction in the comparatively poorly vascularised area of the spinal cord - the mid-thoracic segment.

THOUGHTS ON RECORDS

“The Australian” recently quoted some recent additions and changes in the great wide wonderful world of record shattering achievements, where non-events become Guinness Crowned. Lest you are still awaiting the information, here it is. In Victoria, Texas, at the annual Armadillo Confab and Exposition, Elyira Rose Hunt has earned the title of Miss Vacant Lot of the World by stuffing 264 pennies into her mouth. As she wore a two piece purple hot pants outfit the spectators didn’t object to her speechless condition. Mr Joe Pena filled, licked and rolled 42 cigarettes in five minutes. The results of the belching, yelling and spitting competitions are not yet available, regrettably. However, the All-China Nationwide Peoples Liberation Army Games (Peking) hand-grenade throwing record of 63 metres, held by 16 year old Ma Li-Li was not equalled. The only Australian entry for the useless records section was the successful breaking of the Manly Marineland Underwater Endurance record, now held by their chief instructor and shark feeder. Few of the fish who shared the record attempt are thought to have suffered ill effects.

For the benefit of anyone seriously wishing to establish some sort of record for getting air through a hose it may be an advantage to know what the present state of the market is. In 1946 an American “hard hat” diver aged 26 set what could be an involuntary record, though his times may have been exceeded. The conditions are as follows: without any warning the diver is imprisoned in a mud tunnel, unable to move, in 40 ft water beneath a sunken barge. The only surface contact is by the air hose and telephone. Duration of dive must exceed 3 days and nights and no food or drink is allowed. The cold and a knowledge that there was a constant risk of the wreck settling harder onto the trapped man are factors included by serious contenders for the Record.

Intending contestants should contact “Project Stickybeak” first.

Gastro-Intestinal Manifestations

Patients mildly affected may present only with anorexia, nausea, retching, abdominal cramps and diarrhoea. When the condition is severe, local ischaemia and infarction of bowel, with secondary haemorrhages, may result.

Cardio Respiratory

Intravascular bubbles are more common in the venous system, and are associated with sudden or severe decompression sickness. Although many of these bubbles may be trapped in the pulmonary capillaries, some may pass into the arterial circulation, either through a patent ductus arteriosus, septal defects, or via the pulmonary plexuses. The clinical manifestations may be arbitrarily divided into three types as follows:

i. Local ischaemic effect. This may follow cerebral, coronary, renal or splenic occlusion, etc. The result of these occlusions may be tissue ischaemia and infarction. The clinical manifestations will vary according to the organs involved. Specifically, an infrequent but troublesome cardiac manifestation of decompression sickness is the development of a ventricular arrhythmia, which may not respond to recompression therapy. It is not clear whether these all represent coronary emboli, or whether they result from extravascular bubbles interfering with the myoneural conducting mechanisms of the heart.

ii. Pulmonary involvement (“chokes”). Clinical manifestations are noted when approximately 10% or more of the pulmonary vascular bed is obstructed. Tachypnoea and hyperpnoea are observed. The initial symptom is chest pain aggravated by inspiration, with an irritating cough precipitated by cigarette smoking. Interference with the pulmonary circulation can result in circulatory collapse in severe cases. Pulmonary effects appear early, and are followed by either rapid resolution or a progression of symptoms. Investigations are often not possible, but ECG evidence of right axis deviation, high peaked p waves and right ventricular strain may be obtained.

iii. Post decompression shock. In very severe cases, eg. in explosive decompression or following grossly omitted decompression, there may be a generalised liberation of gas into all vessels, resulting in rapid death. The presence of gas bubbles in the circulating blood results in a bubble/blood interaction which leads to all grades of vessel wall and haematological reactions from haemo-concentration disseminated intra-vascular coagulation. Even in subjects without clinical evidence of decompression sickness, there is observed to be an increase in packed cell volume (haematocrit), haemoglobin concentration, plasma free fatty acids and prothrombin time; decrease platelet counts, plasma cortisol, complement activity and prothrombin consumption time. Those with symptoms also had increased fibrin degradation products.

The effects of hypotension, especially with air platelet and lipid emboli cause secondary hypoxic damage to capillaries, increased capillary permeability and extravasation of fluid into tissues. The signs and symptoms of hypovolaemic shock, such as haemoconcentration, postural hypotension, syncope, low urinary output, etc. are not uncommon, but like the pulmonary manifestations, they are either resolved quickly or proceed ominously. Once the cycle leading to disseminated intravascular coagulation has commenced, it does not necessarily respond to recompression therapy.

Thus such cases may deteriorate even whilst at an initially adequate recompression depth. Attention to intravenous replacement, heparin usage, correction of coagulation defects, etc. follows general medical principles, and may succeed where the recompression has not.

TREATMENT

Pol and Watelle in 1854 recorded improvement of patients who returned to a compressed air environment, ie. they were recompressed. This has remained the mainstay of therapy, but does need to be supplemented by general medical support. Treatment is likely to be more readily available under operational situations when the symptom occurs during decompression or soon after. Alternately, the illness is potentially more severe. This may be explained by the continuing tissue to bubble pressure gradient, the more likely presence of intravascular bubbles as opposed to the extravascular bubbles from slower desaturating tissues, and the consequent haematological complication of disseminated intravascular coagulation. The late development of symptoms heralds a relatively simple therapeutic regime, as exemplified by joint bends near the termination of a uniform and linear decompression from a saturation dive. In both early and delayed cases of decompression sickness, the longer the symptoms are allowed to persist and extend the more difficult is the therapeutic procedure.

Recompression Therapy

The volume of intravascular and extravascular gas bubbles decreases in proportion to the absolute pressure applied, in accordance with Boyle's Law. Thus at a depth of 165 feet or 50 metres, the pressure is 6 ATA and the volume of the bubble will be reduced to one-sixth of its size at the surface. This may be sufficient to relieve the symptoms and signs of decompression sickness and restore circulation. In this particular example the surface area to volume ratio is almost double, thus aiding the resolution of the bubble by increasing the nitrogen gradient. The standard air therapeutic tables have as their most useful depth, 165 feet or 50 metres. This was rationalised as being a depth at which further volume change would become insignificant, and at which the increase in nitrogen saturation of tissues is becoming prohibitive by increasing subsequent decompression requirements. That depth was also consistent with the working medical attendants not being incapacitated by nitrogen narcosis. The air tables, 1 to 4 of the US Navy Diving Manual, had the counterparts in most other Navies, and they varied mainly in their duration, from 7 to 43 hours.

Recompression followed by a slow decompression is the basis for treatment. There are three problems to consider in deciding the necessary form of the recompression therapy. These are the depth required for therapy; the gas mixtures used; and the rate of decompression. The gas mixtures and decompression rate are partly reliable on the depth of recompression.

In deciding this there are three different approaches which may be made, and these are as follows:

A. Recompress to a pressure (depth) dependent upon the depth and duration of the original dive.

B. Recompress to a predetermined fixed depth - ie. according to standard tables of recompression therapy.

C. Recompress to a depth which produces a clinically acceptable result.

These are now elaborated further.

A. *Recompress to a pressure (depth) dependent upon the depth and duration of the original dive.* This not a particularly satisfactory technique, as it is designed to cope with the total quantity of gas dissolved in the body during the original dive. Because decompression sickness is the clinical manifestation of a gas bubble lodged in a vulnerable area, it is necessary to recompress in order to reduce the size of that particular bubble, irrespective of the total quantity of inert gas dissolved in the body. This approach was best typified by the now defunct concept of treating aviator bends by descent to ground level.

   The one advantage of this approach is when a diver develops decompression sickness soon after surfacing from a deep dive. Under those conditions a prompt return to the original depth will ensure that there is no tissue to bubble pressure gradient which could assist bubble growth at a lesser depth.

B. *Recompress to a predetermined fixed depth - ie. according to standard tables of recompression therapy.* The standard tables of recompression clearly state the gas mixture to be used (usually air or oxygen). The application of the standard recompression tables produces relief in 90% of cases, if treatment commences within half an hour or the onset of symptoms. This proportion falls to 50% if the delay exceeds six hours. After a 12 hour delay, the results are poor.

   As a general rule the short air table, taking just under seven hours, is only used for the Type I cases and the longer tables, needing over 42 hours, are used for the more difficult cases. As most series deal with navy divers who have only marginally exceeded decompression requirements, the value of the air tables was not adequately quantified. They seemed of more value to the milder Type I cases than the others.

   The introduction of standardised oxygen tables using 100% oxygen interspersed with air breathing (Table 5 and 6 in the US Navy Diving Manual) gave far more flexibility and improved results. They are able to be extended and interchanged with the air tables at certain depths - mainly 30 feet and 60 feet. These tables became popular because of the improved results and the economy in time, needing only 135 minutes and 285 minutes respectively. The physiological advantages are in the speed of bubble resolution and increased oxygenation of tissues in countering the pulmonary arteriovenous shunting effects. Disadvantages include a fire hazard, oxygen toxicity, and the

occasional intolerance of a distressed patient to oxygen, or a mask, and the less immediate reduction in bubble size, ie. to less than half the volume achieved with the 165 foot standard air tables. Although the pressure gradient of nitrogen from the intravascular bubble to the blood is increased with oxygen breathing at 60 feet, if the diver has previously dived in excess of 60 feet, then there could well be a positive pressure gradient from tissue extravascular bubble still in effect during the early phase of recompression.

Despite the above qualifications, the use of oxygen has received world wide acceptance as a supplement to the air tables.

C. *Reoompression to a depth which produces a clinically acceptable result* allows a more flexible approach. The gas mixture which is likely to result in the maximum safe inert gas and oxygen pressure gradients may then be administered. For example, if the diver requires recompression to a depth of 100 feet, a 50% oxygen mixture may be used, and the oxygen percentage may be increased on reaching the shallower depths. In these circumstances, the rate of decompression must initially be decided arbitrarily - eg. 1 foot ascent per 4 minutes using 100% oxygen, and increasing the time by one minute for every 10% decrease of oxygen in the inspired mixture. The percentage of oxygen to be used may be calculated on the depth of the patient.

When administering oxygen mixtures the inert gas will usually be nitrogen, but this should be avoided in three circumstances. The first is with depths in excess of 180 feet due to nitrogen narcosis. The second is when the subject has respiratory distress. Under this condition either 100% oxygen or an oxygen/helium mixture should be administered, depending on the depth. The third is during the “off oxygen” periods, when it is customary to delay the onset of pulmonary oxygen toxicity by intermittently administering a low oxygen gas for an arbitrary time, eg. 5 minutes each half hour. In the case of decompression sickness due to air diving, at these times 20% oxygen/80% helium may be given to the patient who is in a good clinical condition. This helium mixture could temporarily aggravate the clinical condition of a seriously ill patient.

Decompression is halted at any stage if symptoms recur or others develop. Further recompression may be needed until an acceptable clinical result is achieved. The rate of decompression can be varied according to the response in the individual cases, and this may need to be slowed by up to 50% for saturation dives.

The value of this flexible form of therapy is that it is orientated towards the individual patient, and that it does not limit the medical officer by the imposition of standard tables which may be wholly inadequate for the severity of the case. The disadvantage is that it can only be used by experienced medical personnel and that constant attention to the clinical state of the patient is required.

All three approaches have some validity and may be relevant in different circumstances.

An additional technique of considerable value when there is no significant time factor involved, and if the patient is severely affected, is the 12 hour or “overnight soak”. It may be of benefit for many reasons to halt all decompression for this prolonged time. Bubbles have a chance to resolve before Boyle’s Law comes into effect with decompression. Also, tissue supersaturation of gas will become equilibrated with the ambient pressures. Oxygen toxicity may be relieved. Last but not least, medical and chamber personnel will be able to regroup and reorganise

The administration of 100% oxygen during the short term transport of patients to recompression facilities is recommended for 3 reasons: to increase nitrogen bubble resolution, to improve oxygenation to ischaemic areas, and to reduce the hypoxic effects of arterio-venous shunts. For any recurrence of symptoms after compression therapy, the administration of 100% oxygen by mask also may prove beneficial. The post therapy intermittent use of 100% oxygen is likely to considerably reduce the requirement for further recompression treatment irrespective of which of the above regimes is applied.

Whenever oxygen is used at atmospheric or greater pressures, attention must be paid to the problems of respiratory oxygen toxicity. It is strongly suggested that the oxygen parameters should not exceed those likely to result in neurological or pulmonary toxicity (ie. pO2 of 2.0 ATA, and the vital capacity measurements should not be permitted to decrease by more than 20%).

Supportive Therapy.

Other forms of treatment have been used for decompression sickness. Heparin has been advocated because of its lipaemic clearing activity and its preventative effect on platelet clumping. It may be indicated in cases of disseminated intravascular coagulation which have no evidence of systemic infarctions and bleeding. Correction of coagulation defects seems a more logical approach to the problem of disseminated intravascular coagulation in decompression sickness. Low molecular weight dextran has also been used in these cases and may be indicated when the haematological changes become marked. Vasodilatation has been attempted by drugs and reflex stimulation. Mannitol has been used to reduce cerebral and spinal oedema, as has the administration of steroids. Dexamethisone 10 mgm IV and 4 mgm IM every 6 hours, is often used in neurological cases. It must be discontinued within 72 hours unless maintenance steroids are to be used. Hyperbaric oxygen therapy, repeated daily, may also be of value in these cases. Hypothermia has been proposed for persistent cerebral oedema. Hypovolaemic shock will require intravenous fluid replacement, with appropriate clinical monitoring. Symptomatic therapy is also required, eg. urinary catheterisation, administration of analgesics, etc.

It is essential to realise that general medical treatment is required during the recompression regimes. Patients should not be left unattended in recompression chambers, and especially whilst breathing increased oxygen concentrations. First aid and resuscitation techniques are often required as are accurate clinical assessments, and for these reasons it is desirable to have a trained medical attendant in the chamber. It may be necessary to consider the possibility of decompression sickness occurring in the attendants, especially when the patient is subjected to oxygen or oxygen-enriched mixtures. The decompression regimes are based on the gas mixtures being breathed by the patient, and not the air being breathed by the attendants.