ACUTE DECOMPRESSION SICKNESS IN COMPRESSED AIR WORKERS EXPOSED TO PRESSURES BELOW 1 BAR IN THE SINGAPORE MASS RAPID TRANSIT PROJECT

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ABSTRACT

The Singapore Mass Rapid Transit (MRT) project started compressed air work in Oct 1984. Eleven km of underground tunnels out of 20 km were built using this method. Cases of decompression sickness (DCS) arising from compressed air work are rare with working pressures less than 1 bar gauge. However, there were 10 cases of DCS in the MRT project who were exposed to less than 1 bar pressure. The authors present their clinical features and attempt to explain the observations in relation to theories of bubble nuclei formation, gas loading and rate of decompression. The formation of bubble micronuclei are correlated with physical factors like heavy exertion, and the use of vibrating tools. The concept of extremely long tissue half-times in the absorption of nitrogen in the body is discussed as a contributory factor to the development of DCS under 1 bar.

Keywords: Decompression Sickness, Bubbles, Compressed air work.

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INTRODUCTION

Decompression Sickness (DCS) occurring at pressures of less than 1bar gauge or 14.7 psig is very unusual. A literature search revealed that probably only Behnke (1969) has ever reported instances of cases of DCS in less than 1 bar exposures (1). In his report, he recorded 9 cases of DCS occurring among compressed air workers (CAWs) exposed to less than 1 bar pressure at the Bay Area Rapid Transit (BART) project in California.

Personal communication from Kindwall has also revealed that some experienced physicians in the field are aware of, anecdotally, cases here and there but the authors are unable to trace any publications with this specific finding. Kindwall was also familiar with 4 cases of DCS occurring at less than 1 bar.

In the Singapore Mass Rapid Transit (MRT) project, 10 cases of DCS occurring at less than 1 bar were seen and all responded to recompression therapy with complete resolution of symptoms.

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DECOMPRESSION SICKNESS

Hazards associated with the use of compressed air in civil engineering have long been recognized. Triger (1841) described cases of decompression sickness (DCS) in CAWS in Chalonnes, France (2). In 1854, Pol and Watelle described the ill-effects of DCS or what is commonly known as 'bends' or caisson's disease in CAWS (3). They pointed out that the danger did not lie in entering a shaft containing compressed air nor in remaining there, but rapid decompression alone was responsible for the sickness. They reported pains in the limbs, cerebral and respiratory symptoms and the fact that some men died. They rightly recommended recompression as a therapeutic modality.

PATHOGENESIS

The rapid reduction in ambient pressure causes the dissolved nitrogen to form nitrogen bubbles in tissues. The exact mechanism of bubble formation, even after 100 years of research, is still unclear. Theories of bubble formation like de novo nucleation, supersaturation, tribonucleation and in vivo cavitation have been suggested as possible causative factors of the bubbles in decompression sickness.

The effects of bubbles in the circulation were comprehensively studied by Paul Bert, a leading French physiologist. Paul Bert (4) in a series of experiments with goats and other small animals established the role of nitrogen bubbles in DCS. He also demonstrated that once bubbles formed, they would decrease in size only gradually because the tension of the bubbles is only slightly more than the alveolar nitrogen tension. The dissipation of bubbles from tissues can only be effected by very slow diffusion of the nitrogen gas through tissue layers to the nearest blood vessels. He described bubbles

found in blood vessels of spinal cord centres in dogs even at the 4th day after the decompression incident.

Many other workers (5 – 7) also showed that gas bubbles arose both intravascularly and within tissues. Intravascular bubble formation can lead to embolisation and mechanical obstruction of blood vessels. This was the earliest proposed mechanism explaining the observed symptoms and the findings of ischaemic changes in the various organs. The fact that bubbles can be detected by histology, direct observation and doppler ultrasonography (8) indicate that nitrogen bubbles are the causative agents in DCS.

Further studies since the 1930's have found that aside from the physical effects of bubbles causing embolisation and obstruction of small blood vessels, bubble-blood biochemical interactions occur in-vivo. This phenomenon may account for some of the clinically observed symptoms like inflammation around joints, relapsed symptoms, and biochemical changes in the blood. Concurrent work done by Swindle in 1937 (9) and End in 1938 (10) showed sludging of red cells with the formation of emboli and petechial infarcts in spinal cord and brain in DCS. Subsequent work done by numerous researchers have shown that the bubbles produced changes in the blood and tissues with both morphological and metabolic consequences. These include alteration in platelet function, changes in the plasma levels of catecholamines, lipids, proteins, enzymes and the coagulability of blood. Leitch and Hallenbeck showed that the pathology may also be caused by arterial gas embolism leading to peripheral vascular obstruction by gas (11). Cord segments involved showed varying degrees of haemorrhage and occasionally vascular congestion. Microscopic petechiae were present in both the grey and white matter. These appearances were compatible with hypoxia or embolic episodes. Thorsen et al in 1987 (12) showed with the help of scanning electron microscopy, activation of human platelets by nitrogen micro bubbles.

PREVENTION OF DECOMPRESSION SICKNESS

In the Admiralty Report of 1908, Haldane conceptualised that bubbles would not be liberated in body tissues if the drop in ambient pressure did not exceed a ratio of 2:1 and that the variable time course of nitrogen uptake and subsequent elimination could be simulated by a family of discrete hypothetical half-time tissues. This "critical ratio" hypothesis led to the subsequent development of decompression schedules. Haldane believed therefore that exposure to the equivalent of 10 metres of sea water pressure or to pressures of less than 1 bar gauge were safe, as the 2:1 ratio was not exceeded (13).

Later decompression tables developed for compressed air work in England were based on Haldanian principles and were refinements of earlier tables. The Blackpool Tables were devised by Hempleman following the realisation that the previous British decompression procedures promulgated in 1958, resulted in a high DCS incidence in new starters, and especially for exposures exceeding 4 hours (14). There was also a high incidence of bone necrosis. Hempleman's proposed calculations for the safe exit of personnel from low pressures were based on 2 assumptions. The first assumption was that after spending a time t at a certain pressure P_1, if rapid decompression to a lower pressure P_2 is required, the permissible pressure ratio r should be a constant and equal to 2. The second assumption was that the shape of the pressure-time curve for the onset of DCS would be similar with the curve for the uptake of nitrogen by the whole body.

With the above assumptions, Hempleman was able to calculate the decompression rates for various exposure times and pressures. Ascent from 0.9 bar to the surface can be made directly as the rate does not exceed the curve for nitrogen elimination from the body.

THE SINGAPORE MASS RAPID TRANSIT PROJECT

The Singapore Mass Rapid Transit (MRT) project was Singapore's first extensive use of compressed air in tunnelling work. Compressed air was used by 6 contractors of the project to build about 11 km of the 20 km underground stretch of tunnels. Working pressures were relatively low when compared to other major compressed air tunnelling work elsewhere in the world (15-16), the maximum pressure being 2.35 bars (Table I). The MRT project also followed the CIRIA code of practice and utilised the Blackpool Tables for decompression of CAWs from the tunnels.

A total of 1737 CAWs were involved in this project. Compressed air was used over a total of 31 months. 188,538 man-decompressions were carried out. Over this period, there were 164 cases of acute decompression sickness (DCS) This gave an overall DCS incidence of 0.087% as reported in a separate paper by How et al in 1989 (17). 160 of these cases were Type I while the remaining 4 were Type II DCS (Table II).

The tunnel projects were completed in record time and because of the haste in completing the projects, long working hours (sometimes exceeding the limits of the Blackpool Tables) were employed. This might have resulted in some of the DCS cases.

It is notable that of the 160 cases of Type I DCS, 10 cases developed DCS following exposures to below 1 bar gauge pressures. This is a rare occurrence and this report examines these 10 unusual cases of DCS that occurred in the Singapore MRT project.

MATERIALS AND METHODS

The Diving and Hyperbaric Medical Centre (DHMC) of the Republic of Singapore Navy provided medical support for the compressed air tunnelling aspect of the MRT Project. Medical selection and routine health checks of compressed air workers (CAWs) were conducted at DHMC. Cases of decompression sickness were treated either on-site by the duty medical officer, or at DHMC in Sembawang Camp. A detailed clinical assessment form was used to ensure uniformity of reporting and subsequent data analysis.

FINDINGS

General

Eight cases of DCS following exposures to below 1 bar gauge pressures, occurred at Contract 109 while another two cases were seen in Contract 301. 7 of the cases occurred after exposure times of 12 or more hours (maximum 12 hours 22 mins), including the time utilized for decompression. The other 3 cases occurred after exposures of between 10 hours 45 mins and 11 hours 45 mins. At pressures less than 1 bar, CAWs were decompressed to the surface at not greater than 0.4 bars per minute.

Table I
Comparison Of Various Compressed Air Contracts

Contract	Period of Compressed Air Work (Mths)	Total No of CAWS*	Maximum Pressure (Bar Gauge)	No of Man Decompressions	No of Cases of DCS** (Overall)	DCS** Incidence Overall %
East River Tunnel N Y 1914 – 21	84	–	3.26	1360000	680	0.05
Howrah Bridge India 1938	6	509	2.72	12400	353	2.8
Lincoln Tunnel N Y 1955 – 56	18	704	2.31	138000	44	0.03
Dartford Tunnel 1957 – 59	24	12000	1.90	122000	685	0.56
Blackwall Tunnel 1960 – 64	44	1536	2.65	81000	863	1.1
Tyne Road Tunnel 1960 – 64	38	650	2.86	44800	711	1.6
Hong Kong Islandline 1982 – 85	36	3966	2.85	443430	2003	0.45
Singapore MRT 1984 – 1987	31	1737	2.35	188538	164	0.087

* CAWs = Compressed Air Workers
** DCS = Decompression Sickness

Table II
Incidence of DCS By Contract

Contract	Maximum Pressure (Bar Gauge)	Number of DCS			Number of Man Decompressions		Incidence of DCS					
							Overall %			Over 1 Bar %		
		Type I	Type II	Total	Total	Above 1 Bar	Type I	Type II	Total	Type I	Type II	Total
104	1.50	36	1	37	79363	39064	0.045	0.001	0.046	0.092	0.003	0.095
105	1.43	3	0	3	27976	937	0.011	0	0.011	0.320	0	0.320
107	1.60	31	0	31	8757	2679	0.354	0	0.354	1.157	0	1.157
108	1.95	26	1	27	19520	6666	0.133	0.005	0.138	0.390	0.015	0.405
109	1.50	28	2	30	38110	5550	0.073	0.005	0.078	0.505	0.036	0.541
301	2.35	36	0	36	14812	9163	0.243	0	0.243	0.393	0	0.393
Total	–	160	4	164	188538	64059	0.085	0.002	0.087	0.250	0.006	0.256

The lowest working pressure where DCS cases were reported, was 0.8 bar gauge while the highest was 0.95 bar. One was an assistant surveyor, another an assistant foreman, whilst the others were compressed air workers (Table III).

Table III
Decompression Sickness (DCS) at less than 1 bar pressures in relation to Race, Category of worker, Body fat, Age, Working pressure, and time of onset of symptoms

S/No	Case	Race	Contract	Type of Worker	% Body Fat	Age	Working Pressures (Bar Gauge)	Date of Incident	Exposure Time #	Time of Onset After Decompression	DCS Type
1	Mr S P	Thai	109	CAW	11	32	0.80	010285	12 hr	A few hours	1
2	Mr S K	Thai	109	Asst Surveyor	6	28	0.80	010285	10 hr	A few hours	1
3	Mr D C	Thai	109	Asst Foreman	20	39	0.80	080285	12 hr	A few hours	1
4	Mr S T	Thai	109	CAW	9.4	18	0.95	231285	12 hr	1 1/2 hours	1
5	Mr N	Thai	109	CAW	15	24	0.95	251285	11 hr 45 m	3 hours	1
6	Mr S B	Thai	109	CAW	11.3	31	0.95	291285	12 hr	1 1/2 hours	1
7	Mr U S	Thai	109	CAW	12	25	0.95	291285	12 hr	3 hours	1
8	Mr V A	Indian	109	CAW	15	29	0.75	160286	10 hr 55 m	4 1/2 hours	1
9	Mr KYK	Korean	301	CAW	19	31	0.90	210187	12 hr 22 m	6 hours	1
10	Mr CJC	Korean	301	CAW	14.2	35	0.94	160387	12 hr 22m	28 hours	1

including decompression time of not more than 10 minutes

ENVIRONMENTAL FACTORS

During the exposure periods shown in Table II and III, tunnel temperatures were between 22 and 34 degrees Centigrade (Chart 1). The relative humidities were between 70% to 96% (Chart 2).

Chart 1: The MRT project in Singapore
Temperature Chart

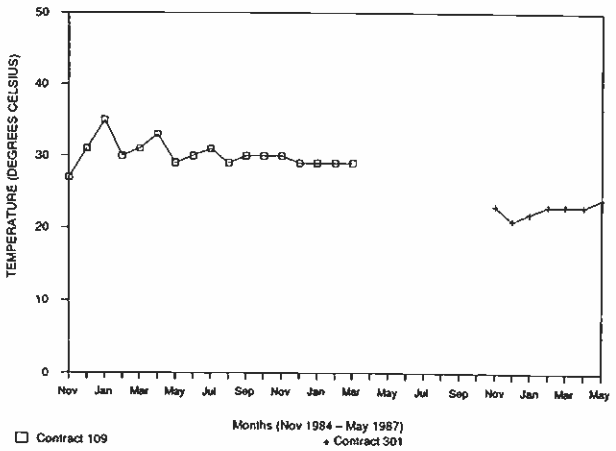
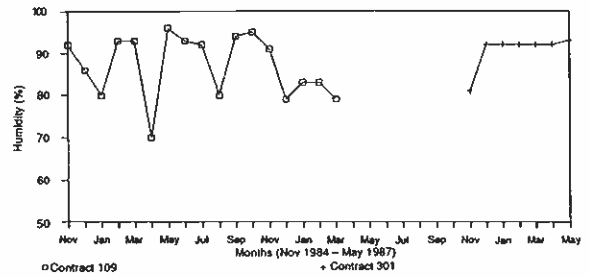


Chart 2: The MRT project of Singapore
Relative Humidity Chart



CLINICAL PRESENTATION

Symptomatology

Pain was the commonest presentation. It presented as deep joint pain with tenderness in 1 worker and associated with warmth in 2 workers. No swelling or rashes were seen. One worker had numbness around his joint (Table IV).

Table IV
Clinical presentation of 10 cases of Type 1 DCS

Symptomatology	No of Cases
Pain: Deep Pain	10
Constant	2
Radiation of Pain	0
Limitation of Movement	3
Tenderness of Joint	1
Joint Numbness	1
Lymphatic Swelling	0
Warmth Around Joints	2
Joint Effusion	0

Onset of symptoms

The majority (60%) of the cases presented with symptoms within 6 hours of decompression (Table III). 30% were not sure when the first symptoms came about but gave a history of experiencing them a "few hours" after decompression. The last case developed symptoms after 28 hours. A presumptive diagnosis of DCS was made in view of the characteristic symptoms. The patients were treated with recompression. The symptoms were relieved completely after 10 minutes of recompression, confirming the diagnosis.

Site

All cases were of the Type I variety with symptoms of joint pain. Monoarticular joint pain was seen in 30% of the cases. Polyarticular joint pain was seen in the other 70% of cases. 30% of the cases showed both upper limb and lower limb joint involvement while isolated upper limb joint involvement was seen in 20% of the cases.

The commonest joints affected were the knee joints in 80% of the cases (bilateral in 40% and unilateral in 40%). One case presented with multiple joint pain in both elbows, hips and knees (see Table V).

Treatment

All cases were treated with recompression therapy. Table 61 (oxygen table) was used in 50% of the cases. CIRIA 1 (air table) was used in 40% of the cases. CIRIA 1 and Table 62 was used in Case 9 when there was, initially, no relief of symptoms after using the CIRIA 1 table. Complete relief of pain were recorded in all 10 cases. (Table V).

Table V
Summary Table Of Symptomatology And Treatment Used

Case No	Affected Site								Treatment Table Used	Outcome of Treatment
	Shoulders		Elbow		Hips		Knees			
	UNI	BI	UNI	BI	UNI	BI	UNI	BI		
1								-/	61	Complete Cure
2						-/		-/	61	Complete Cure
3	-/					-/		-/	61	Complete Cure
4							-/		CIRIA 1	Complete Cure
5		-/							61	Complete Cure
6							-/		61	Complete Cure
7							-/		CIRIA 1	Complete Cure
8				-/		-/		-/	CIRIA 1	Complete Cure
9			-/				-/		CIRIA 1 and 62	Complete Cure
10				-/					CIRIA 1	Complete Cure

Note: UNI – UNILATERAL
BI – BILATERAL

DISCUSSION

The cases seen manifested characteristic symptoms of DCS and responded to treatment. The 10 cases represented a DCS incidence of 0.005% of man decompressions for all pressures and 0.008% of man decompressions below 1 bar.

None of the persons in this study was obese. The maximum percentage body fat was 20% and the minimum 6% (Table III). The average percentage body fat was 14.5%. Obese persons are known to be more susceptible to DCS than thin people (18). But there were only 27 obese persons with >24% body fat in the Singapore MRT Project out of 1737 persons. This accounted for the bias of thin persons developing DCS. In addition, obese persons were allowed shorter exposure times to compressed air work. This reduced the likelihood of any of them getting DCS.

In our study, the oldest worker was a 39-year old assistant foreman. With the exception of the other surveyor, the rest were CAWs. The youngest CAW affected was 18 years old (average age: 29.2 years). As the number of cases is small, we are unable to show any correlation of DCS with age, obesity and type of work performed. However, we note that the cases occurred at very long exposures and that the persons were involved in heavy work. The long hours probably allowed for almost complete tissue saturation with nitrogen, even with the very "slow" tissues (those tissues which take a very long time to become saturated with nitrogen).

Two cases not treated at DHMC are worthy of note. These 2 CAWs completed their work in Singapore at less than 1 bar tunnel pressure and were flying back to Bangkok the same evening, when they developed joint pains. The joint pains subsided when the plane finally landed in Bangkok. The symptoms were probably due to the low cabin pressure of the commercial jet which precipitated altitude DCS.

Interviews with various Korean workers also revealed that 3 of them had developed joint pains after such exposures but they did not report to their supervisors. Subsequent exposure to compressed air relieved their symptoms and they ignored their symptoms.

POSSIBLE MECHANISMS OF DCS IN LESS THAN 1 BAR

We are uncertain of the mechanisms which caused DCS in these 10 persons. We feel that the following mechanisms are likely to be involved.

In CAWs, the tendency for bubbles to form is governed by the principles of fluid mechanics. It has been shown that a large force is required to form bubbles in vitro, unless bubble nuclei are present. These forces may be due to tribonucleation, cavitation or even from spontaneous in vivo nuclear fission.

The CAWs were involved in heavy work involving lifting and the use of vibrating tools. This can cause the formation of micronuclei by the process of tribonucleation. Tribonucleation is induced in vivo when 2 closely opposed surfaces separated by fluid are forced apart. The negative forces or low pressures generated as a result of the separation of the 2 surfaces result in the formation of bubble nuclei.

Another mechanism where microbubbles may be formed is related to the increase in haemodynamics in CAWs performing heavy work. At the molecular level, fast moving fluid particles, by cavitation, generate sufficient negative forces behind the particles to cause the formation of microbubbles. Other mechanisms which may cause microbubble formation include a suggestion by Walder and Evans (1974) that spontaneous in-vivo nuclear fission may be the aetiology of gas micronuclei (19).

The CAW enters and exits from compressed air daily after spending long hours in the compressed air environment. This form of repetitive exposures greatly increases their predilection to DCS. Experience from repetitive dives in fisherman divers has shown that there is an increase in incidence of DCS in the second and subsequent dives. In the first exposure, small asymptomatic bubbles may have been formed. In the subsequent exposures, these asymptomatic small bubbles formed sites of further bubble growth, accounting for the increased incidence of DCS in subsequent dives.

Bubble micronuclei do not cause symptoms by themselves. The CAW must have enough gas loading in his tissues to enable the bubbles to grow. In addition, the rate of decompression must be great enough to overwhelm the CAW's circulatory capacity to transport the excess nitrogen from the tissues to the lungs.

Although the CAWs were decompressed according to the CIRIA recommendations, they had spent more than 8 hours in the compressed air tunnels. It is possible that there are very "slow tissues" that become totally saturated only after long exposure times. During decompression, the nitrogen is released, but because of the slow half-times, the rate of nitrogen elimination from the tissue is slower than the rate of decompression. This tipped the balance and could have resulted in bubble formation localised in that slow tissue. Tissue bubbles when formed, can cause physical distortion of the tissue planes and stretch nerve endings. This manifested as symptoms of pain and numbness seen in our CAWs.

Since the 19th century, Pol and Watelle suggested slow decompression to eliminate the problem of DCS in CAWs. Haldane (14) believed that bubbles were not liberated if the drop in ambient pressures did not exceed a ratio of 2:1. The variable time course of nitrogen uptake and subsequent elimination could be simulated by a family of discrete hypothetical half-time tissues. Haldane took tissues with half-times up to 75 minutes to develop his Decompression tables which were adopted by the British Admiralty. Subsequent development and improvements to Haldane's tables were later adopted for various compressed air tunnelling projects in England. In 1958, Hempleman devised the current compressed air tunnel tables which were incorporated into the CIRIA report.

The disadvantage of using the CIRIA procedures for decompression of our CAWs from less than 1 bar exposure was the fact that very long half-time tissues were not considered. Our workers worked between 8 to 12 hours at pressures approaching 1 bar gauge with 12 hours on the surface. Benhke had proposed that there are tissues with up to 120 min half-time which will require up to 14 hours to desaturate 99%. As a result, calculations for the decompression times for CAWS with exceedingly long exposures must take this into consideration. The interval between exposures must also be greater than 14 hours for these long half-time tissues. The CAWs therefore might have accumulated nitrogen due to the

long exposures and repetitive nature of their work. This is a possible reason why some of our CAWs developed DCS as they had worked longer than 10 hours within the tunnels and had spent less than 12 hours at the surface.

The US Navy's experience with long and deep exposures have also revealed deficiencies in assuming Haldane's 2:1 ratio for decompression. They found unacceptably high rates of DCS when assuming Haldane's theory of using a 2:1 decompression rate. A better proposal was made by Workman. He proposed that blood perfusion of the tissues (excluding tissue diffusion) is the chief factor affecting the rate of gas transport. In his calculations, Workman considered more tissues as well as slower tissues, some with half-times of 1000 minutes (20). The critical ratio varied at each depth for a particular tissue. He devised a linear scale of "M" values, showing the maximal allowable supersaturation for each hypothetical tissue at each depth for the whole range of decompression for Nitrogen and Helium Diving. These formed the basis for the derivation of the now adopted US Navy Tables.

The decompression tables of the US Navy and the tables promulgated in the CIRIA report may have in general prevented symptomatic bubbles from occurring in the CAWs. Brian Hills proposed the "Thermodynamic Model" of DCS (21). He believed that gas bubbles are formed during decompression with the US Navy tables and the decompression rate merely controlled the size of the bubbles. The primary event and the critical insult which produced the symptoms of DCS do not coincide. The primary event is the activation of one or more of a reservoir of nuclei normally present in tissue into growth and hence the inception of a stable gaseous phase. The inception of this gas phase occurred randomly.

In the CAWs, it is possible that the DCS in the limbs was caused by the local pressure differential resulting in the distortion of a nerve ending beyond its pain provoking threshold. This onset of limb DCS is more dependent upon the volume of gas separated from solution. As described earlier, the inception of gaseous phase in the limbs of the CAWs can be profuse and rapid due to the presence of micronuclei created by tribonucleation and cavitation in the joint. The tissue as a result can only withstand minimal supersaturation before "dumping" gas in excess of thermodynamic equilibrium into the gaseous phase.

The likelihood that DCS in the 10 CAWs was due to long exposures resulting in bubble formation is further supported by recent experiments. Studies by Eckenhoff (1989) with the doppler ultrasound (22), showed that 60% of persons had bubbles recorded over the precordium, although none developed DCS. This implied that for a very long and shallow exposure, there is definite evidence that bubble formation occurred. This evidence demonstrated the definite possibility of bubble formation even at low pressures.

The CAWs worked at high temperature and humidity for long durations. An additional contributing factor to be considered is dehydration. Dehydration reduces the circulating blood volume resulting in reduced amounts of nitrogen gas which may be carried by the blood. During decompression, the increased gas load at the tissue levels was not eliminated fast enough due to the reduced blood volume. This might have resulted in the buildup of bubbles in the tissues resulting in symptoms of DCS.

Age and obesity have been noted in factors that predispose workers in the development of DCS. Older workers tended to have less efficient cardiovascular systems which possibly result in a reduced capability in the clearance of the excess nitrogen bubbles. In Singapore, the majority of CAWs were less than 35 years old. Obese workers who had greater than 24% body fat were given shorter exposures to compressed air work. The 10 cases of DCS were neither old nor obese and are therefore unlikely to have been affected by these factors.

One of the patients gave a history of working from 8 am till about 8 pm in the tunnel and between 4 am to 7 am, he moonlighted as a newspaper vendor to supplement his income. This might have precipitated the development of DCS as prolonged exertion results in an increase in the number of bubble nuclei, and the sites where further bubble growth could occur.

Our experience has however shown that in 90% of the cases, DCS occurred when exposure was at or greater than 0.8 bar (Table III). Two cases, one at 0.94 bar (Case No: 10) and one at 0.9 bar (Case No: 9) exceeded the 12 hour limit of the CIRIA recommendations. A disadvantage of using the CIRIA regulations was that for exposures less than 1 bar, regardless of the exposure time, no decompression stops were required and the CAW can be decompressed direct to the surface. The assumption that DCS did not occur if the exposure pressure is less than 1 bar must be questioned in the light of the demonstration of bubbles detectable with the doppler at shallow exposures by Eckenhoff (20).

CONCLUSION

The 10 cases of DCS were young and healthy individuals of various ethnic groups. There was no obvious individual predilection to DCS. Various factors related to the nature of the work like heavy manual labour and repeated entries into the compressed air environment might have resulted in the formation of microbubbles through the process of tribonucleation and cavitation. The long exposure times and the rate of decompression caused the microbubbles to grow and produce symptoms in these 10 CAWs. Exactly why it happened in these 10 CAWs and not the other CAWs, we cannot be certain. We feel that a combination of long exposure times, heavy manual labour, repeat daily entries into the compressed air environment and inadequate time at atmospheric pressure led to the development of DCS.

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