● Original Article

Safer Recreational Diving

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レクリエーションダイビングは比較的安全なスポーツで，すべての年代にますます人気を博しているものである。推計するとアメリカには200万人以上のダイバーがいることになろう。ダイバーズ・アラート・ネットワーク・アメリカ（DAN アメリカ）の収集した最新のデータ(1)では，1995年までに治療された減圧症の症例は1132件で，そのうち809件に関する報告がDANに送付されている。こうした症例中27.3%は痛みのみのⅠ型減圧症（DCS）で，64.9%はⅡ型（神経型）減圧症，7.8%が動脈ガス塞栓（AGE）であった（表1）。

死亡例が104名報告されている。しかしながら，この比較的少ない数字も，以下に検討するようにいくつかの要因を詳しく見てゆくことで，おそらく大幅に減少させることができよう。

Recreational diving is a relatively safe sport and is increasingly popular for all ages. Rough estimates indicate there may be more than 2 million sports divers in America. The latest data[1] collected by the Divers Alert Network America (DAN America) which is for 1995 indicated 1132 cases of decompression illness treated, of which 809 reporting forms were sent to DAN. Of these cases 27.3% were pain only Decompression Sickness (DCS) I, 64.9% DCS II (neurological) and 7.8% arterial gas embolism (AGE) (**Fig.1**). There were 104 fatalities reported. However, even this relatively low number can probably be significantly reduced by closer attention to a number of factors as discussed below.

Fitness to Dive

All divers must have adequate physical and cardiovascular reserves which enable them to respond to sudden exertion without premature exhaustion and probably panic, which is often the precursor to a too rapid ascent and an accident. Physical fitness is defined as physical endurance or aerobic capacity measured by the response in oxygen consumption to an increasing work load.

The problem of lack of fitness is important also to an aging population of divers with its emphasis on increased risk of cardiovascular disease. The 1995 data indicate that of the 104 fatalities, 60 were autopsied. Of these, no less than 20 included cardiovascular disease as the primary cause of death. It was the exertion that killed, not diving itself, and it could just as easily have occurred from some other sudden excessive exertion. Major risk factors for coronary artery disease include smoking, high blood cholesterol, male gender, hypertension, diabetes and a genetic predisposition. An older diver needs a healthy diet, not be overweight and engage in aerobic exercise three times per week. An electrocardiogram and exercise stress test is highly recommended to a level of 13 METS (metabolic equivalents) in divers aged 50 and over, although 10-11 METS may be sufficient (1 MET=3.5mL $kg^{-1}min^{-1}$). By comparison an average 6 METS equals heavy snow shove-

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Fig.1 Conventional Disease Diagnosis

Final Diagnosis	1995 Percent	1994 Percent	1993 Percent	1990-92 Percent	1987-89 Percent
DCS I	27.3	25.4	23.0	19.3	21.0
DCS II	64.9	64.8	67.4	68.3	63.0
AGE	7.8	9.7	9.6	12.4	16.0
TOTAL	100.0	100.0	100.0	100.0	100.0

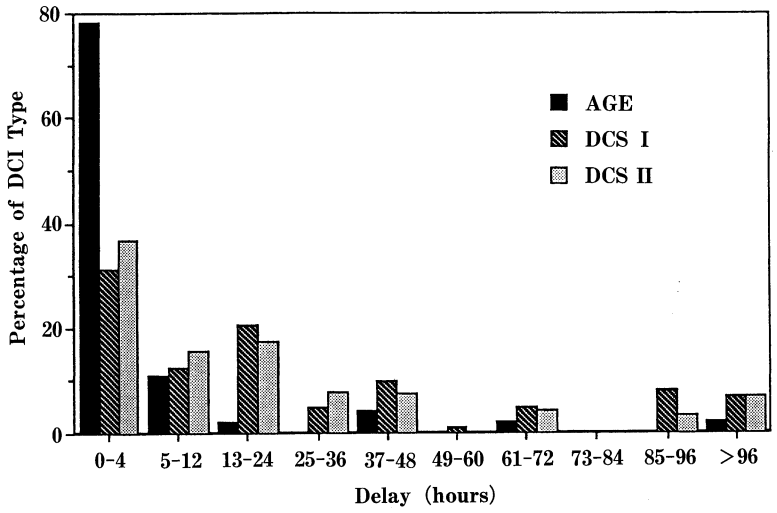

Fig.2 Delay from Onset of Symptoms to Calling for Assistance

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Training

Diving accident data recorded by DAN since 1987 has shown a trend for more accidents to occur in divers with less than 2 years experience or who have 20 or fewer lifetime dives. More recently this percentage has decreased and the number of advanced divers in accidents seems to be increasing. The reasons are unclear, other than perhaps better education and awareness of accident risks and advanced divers diving more deep dives even to over 130 ft. Unfortunately many new open water divers believe that as new certified divers they can dive down to 130 ft or 100 ft for repetitive dives when they should restrict their dives to 60 ft or less until more experienced. The extra problems of deep diving, currents, drift diving, cold water, wrecks, caves, etc. must be approached cautiously. Advanced diving requires further training for adequate safety, especially in an overweight, unfit and inexperienced diver.

One area of training that is sadly lacking is knowledge of decompression illness (DCI), signs and symptoms, and resulting delay by divers in calling for assistance and recompres-

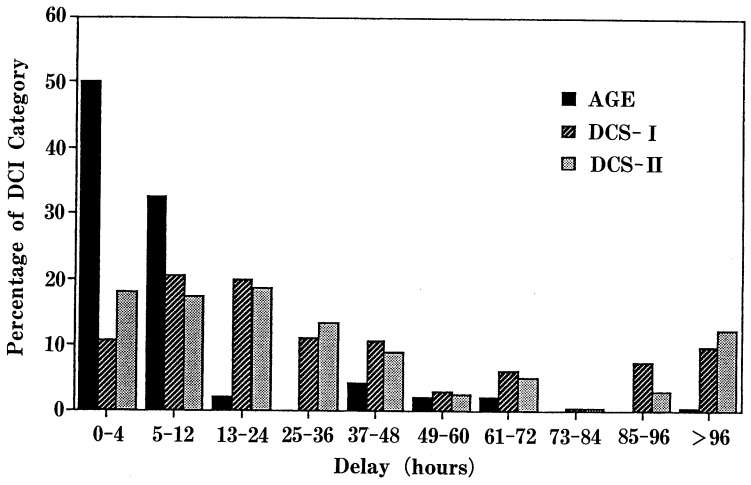

Fig.3 Delay from Onset of Symptoms to Recompression Therapy

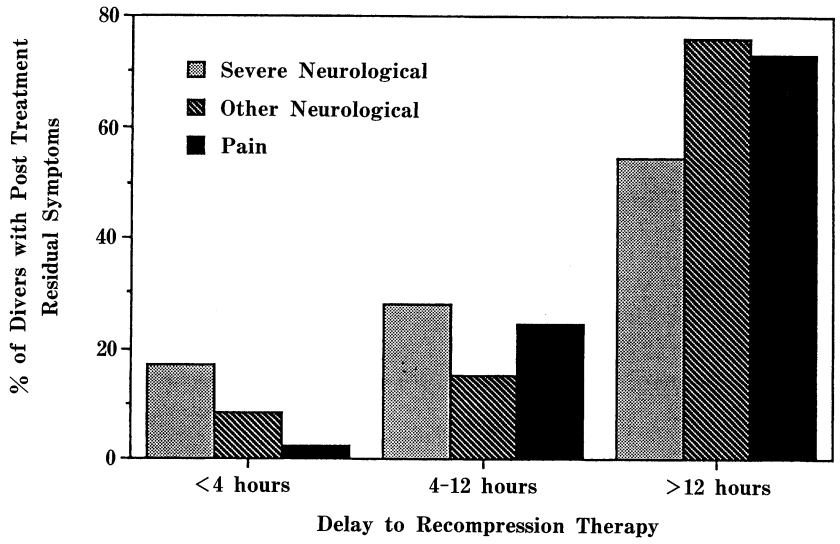

Fig.4 Percent Divers with Post-Recompression Residual Symptoms as a Function of Percentage of Divers with Pre-Recompression Symptoms and Delay to Recompression Therapy

sion. DAN accident data has consistently indicated that only some 35% of all DCS cases reported symptoms in the first 4 hrs and 7% had waited over 96 hours to seek assistance (**Fig.2**).

As a result, in 1995 only 50% of arterial gas embolism (AGE) cases received hyperbaric oxygen therapy within 4 hrs of symptom

onset and 83% within 12 hrs. Again, only 11% of DCS I (pain only) and 18% of DCS II (serious, neurological) were treated within 4 hrs, and 31% and 36% respectively within 12 hrs (**Fig.3**). Causes are due to diver denial, failure to recognize the symptoms of AGE or DCI, remote situations requiring long delays due to evacuation and waiting to see if the symptoms "will just go away". A serious consequence of such delay is incomplete resolution of symptoms (**Fig.4**). Divers need better training to recognize signs and symptoms of decompression illness. In 1995 the most frequent symptoms are shown in **Table 1**.

Table 1 Most Frequent Symptoms of DCI 1995

Pain	203
Numbness	129
Dizziness	44
Weakness	35
Headache	25
Nausea	25
Itching	21
Unconsnciousness	4
Paralysis	3
Other	186
	590

Ascent Rate

It is my firm belief that ascent rates are too fast and that reduction will result in a major reduction also in the incidence of decompression sickness.

Attempts to develop safe decompression tables have been ongoing since Haldane in 1906[2]. Today there are numerous tables attempting to do this including U.S. Navy, PADI DSAT, NAUI, Buehlmann (Swiss), DCIEM (Canadian), British Royal Navy and French Navy tables, etc.[3] plus very many dive computers algorythms. These are based on various models, the most common being Haldane's theories. No decompression bottom times for diving with these vary enormously; for example, at 100 ft from 25 mins to only 7 or 8 mins. All obviosly cannot be correct and inspite of these computers and tables, over 1,000 cases of decompression illness still occur among recreational divers worldwide every year.

Consideration of the DAN accident and mortality data indicate in general that the accidents occur proportionately across the distribution of divers by age, sex, certification, etc. and seemingly regardless of the table or computer used[1]. Some other common factor would seem to be involved.

Most of the table theories are based on Haldane's concepts that nitrogen at increased pressures enters "tissues" with specific tissue half-times e.g. 5, 10, 20, 40 and 75 mins. This purely mathematical concept relies on uptake and elimination to be basically the same (which is not the case as gas elimination is slower than uptake) and that there is a critical gas tension for a given tissue which should not be exceeded[3]. If so, such supersaturation will result in the formation of bubbles.

Over past decades, as table and computer developers tried to eradicate decompression sickness, the number of tissue half-times grew to as many as 16 for Buehlmann developed tables with tissue half-times from 4 to 635 minutes. This was because the hypothesis inferred bubbles causing DCS most likely occurred in the so-called slow tissues, where nitrogen uptake and elimination was slow and therefore was more likely to supersaturate on too fast an ascent. Thus, if a certain long tissue half-time failed, one of the alternatives was to add a still slower tissue half-time and recompute the table[4]. These decisions also were predicated on the fact that most bends in the U.S. Navy were pain only DCS I anyway. Only with the growth of recreational diving with its deep short dives has the domi-

Table 2 Suggested Rates of Ascent

1905	Haldane	10 m/min	30 ft/min
1056	U.S. Navy	18 m/min	60 ft/min
1957	Workman	18 m/min	60 ft/min
1968	Hempleman(UK)	15 m/min	50 ft/min
1966-76	Hills	12 m/min	40 ft/min
1975	Buehlmann	10 m/min	30 ft/min
1995	Bennett	10 m/min	30 ft/min
1995	U.S. Navy	10 m/min	30 ft/min
1995	Wong	3 m/min	10 ft/min

nance of neurological injury been observed as it is today.

Now the incidence of decompression illness and the distribution among recreational divers is also much the same around the world, showing predominantly neurological signs and symptoms. This applies regardless of the table variables plus others such as male, female, young, old, heavy, thin, cold or warm water, drift or swimming, etc. So why have we been unsuccessful in stopping decompression illness?

It would seem that obsession with Haldane and similar theories which were developed more for pain only bends has rather blinded table modelers. Tables and computers vary widely and do not seem the relevant factor in recreational diving. The one common factor for all sports divers that has received very little direct attention, is the rate of ascent (Bennett 5, 6) which has been 60 ft/min.

Too fast a rate of ascent may result in neurological injury in two ways. First, too rapid ascent rates may cause discrete ruptures of the lung tissue from subtle over-expansion causing arterial gas embolism with bubbles finding their way to the spinal cord and brain. Bubbles can also grow autochthonously, i.e. spontaneously in neurological tissues if there is sufficient supersaturation and cause decompression illness.

Most diving in past decades, based on U. S. Navy tables, recommended an ascent rate of 60 ft/min. However, most sports divers, in fact, have been noted to ascend at much faster rates, even as high as 140 to 200 ft/min for the last 10 ft, especially using modern buoyancy compensators[4].

The current use of most tables, etc. of 60 ft/min emanated from the U.S. and was purely empirical and based on the ease of use of 1 ft/sec. Haldane, however, from which such tables were developed, actually proposed rates twice as slow at 30 ft/min. Some variations in past ascent rates are shown in **Table 2**.

Let us reconsider what is actually going on with a Haldane model. The fastest tissue is blood; next is the highly perfused spinal cord and brain (**Table 3**). In fact, their perfusion is so fast that these "fast tissues" saturate very quickly, compared to connective tissues at the joints with their few blood vessels and which, therefore, will require very much more time to reach an equal saturated equilibrium.

Table 3 Tissue Half-Times for Nitrogen (From Edmonds et al. 1992)

Blood	Very short
Spinal cord	12.5 mins
Inner ear	146-238 min
Joints and bones	304-635 mins

Table 4 Air Series: Summary of Neurologic DCI Incidence, Functional Severity, and Skin DCI Severity

	Linear Group, $n=20$	Fast/Slow Group $n=20$	P Value
Neuro DCI cases	11	5	not applicable
Severe cases	9 (1 death)	1	0.01
Skin DCI<20%	13	6	0.057

From Bloome, J.R., Undersea and Hyperbaric Med. 23: 19-26, 1996.

Thus the "tissue" of concern in fast ascent is the highly saturated blood and spinal cord, not the joints and bones, which in the short deep dives of recreational diving will be no where near saturation.

Previous Animal and Human Investigations on Ascent Rates

There is some confirmation of these probable effects of ascent rates from both animal and human studies. First in animals, Lehner et al[7] exposed sheep for 30 minutes at 3.3-5.2 ats abs to 24 hrs at 2.2-2.9 atm abs using multiple exposures and surface intervals. After 528 dives there were 151 cases of DCS. Short deep dives had a lower overall incidence (16%) than shallow long dives (49%). However, there was a more frequent neurological DCS (Type II) with short deep exposures compared to long shallow ($P<0.001$).

Further, Smith and Stayton[8] reported that sheep with doppler cuffs implanted around the vena cavae had more venous bubbles during ascent at 60 ft/min than at 30 ft/min. Pollard et al.[9] stdied ascent rate and post dive exercise effects on DCS in rats and noted more DCS at 60 and 45 ft/min compared to 30 ft/min and earlier death at 60 ft/min compared to 30 ft/min.

In humans, Wong, in 1995[10], reported on pearl divers who made repetitive dives of up to 10 dives a day, often shallow to deep, or the deepest dive in the middle of the dives and multiday for as much as 8 consecutive days. They utilized doppler and modified by trial and error procedures to produce the least bubbles. Depths ranged from 11 m (35 ft) to 35 m (112 ft). By modifying the ascent rate to a slow 3 m/min (9.6 ft/min) and instituting a decompression stop at 9 m (28.8 ft) breathing

Table 5 Effect of Safety Stops on Venous Gas Emboli[12]

Means	Depth	Time	Bubbles
Safety Stop 20 ft/3 min	98 ft	28 min	9
No Stop	101 ft	28 min	21

oxygen, 30,095 dives were made in 1992 by 86 divers with only 4 cases of musculoskeletal bends. In 1993 there were 3 such cases and in 1994 none. Wong attributed this remarkable success to the slow rate of ascent of 3 m/min (9.6 ft/min) compared to 18 m/min (57.6 ft/min).

In 1996 Broome[11] reported a study in pigs comparing decompression from a 200 ft/24 min dive using a linear 20 ft/min to surface or a non linear fast ascent deep (60 ft/min to 110 ft), followed by a slow ascent shallow profile (12.9 ft/min) to the surface. Both ascents took 10 mins.

The results shown in **Table 4** indicate that the slower ascent at 12.9 ft/min from 110 ft had a significant effect in decreasing the incidence of neurologic decompression illness.

It is possible that the present more common use of 30 ft/min rather than 60 ft/min may still not be slow enough and we will need to try 10 ft/min as did Broome and Wong. Whatever is correct, it seems certain that ascents need to be slower and divers should be trained and educated accordingly.

Dive Procedures

There are some other techniques which improve safety. One is to ensure that the surface interval between dives is never less than 1 hour. Make no more than 3 dives per day for 6 or 7 days. Many liveaboards make as many as 6 but they are usually shallower and shallower so that the last dives are almost treatments for any bubbles present.

A day off diving in the middle of a week of diving is a good idea, too, to permit off gassing of nitrogen.

A DAN oxygen unit or similar device should be onboard or close by if diving from the shore and individuals trained in its use plus knowledge of who to call and how in an emergency. DAN has an international oxygen training program and specially designed equipment available.

A safety stop for 3-5 minutes at 15-20 ft is an excellent concept, too, and helps, also to slow the ascent.

Safety Stops

In recent years, safety stops were instituted at 15 ft for 3-5 mins in the hope of reducing the incidence of decompression illness and was viewed as similar to slowing the ascent. Uguccioni[12] used precordial doppler to measure bubbles during ascent in divers breathing nitrox (36% O_2). Two profiles were evaluated by 50 divers, one direct ascent, and one with a safety stop for 3 mins at 20 ft. The results at ascent rates of 60 ft/min showed significantly only 9 divers with bubbles who used the stop compared to 21 with no stop (**Table 5**).

In 1990 only 42% of the injured dives reported by DAN were making safety stops. By 1993 this had increased to 56% and is common today. However, while this may have been beneficial in reducing the incidence of

Table 6 DAN Accident Data for Divers Making Repetitive Dives[1)]

	Number of Divers		
Max. Depth	DCS I	DCS II	AGE
<60 ft depth	10	29	5
60-80 ft depth	25	78	7
81-101 ft depth	26	65	4
>101 ft depth	31	75	2

AGE from 15.5% in 1990 to 7.8% in 1995, there was little or no effect on the incidence of DCS. In fact, in the DAN database there are more cases of DCS I and II with a safety stop than without.

This is probably because with a rapid ascent of 60 ft/min, even from 60 ft, plus a 3 -5 min stop, the diver will be on the surface in only about 4 mins. Yet the tissue half-time for spinal cord is 12.5 min and a much slower ascent rate would give more needed time for gas elimination. Otherwise bubbles would probably have formed during the 1 min ascent and the stop is then of little value. Indeed such a stop may add more gas to the existing bubbles and make matters worse.

Effects of Depths

There is a tendency for many divers today to exceed the arbitrary depth limit for recreational diving of 130 ft. They should do this only with the knowledge that the risk of decompression sickness rises with depth and at depths as deep as 160 ft or more can be as high as 15% compared to the less than 1% for 60-80 ft dives.

An analysis of DCI incidents from the 1994 DAN database emphasizes that dives less than 60 ft are considerably lower in risk. **Table 6** shows in repetitive dives the number of cases in 1994 distributed by depth and severity.

Clearly diving deeper than 60 ft suggest there is much more risk. Interestingly, the other depths indicate an amazingly consistent number of accidents regardless of the depth and that the risk of DCI II is about 3 times that of pain only (DCI I) regardless of the depth. A baseline of how many divers who were not affected would help interpretation. This will need to wait to the results of the DAN Project Dive Safety which is trying to collect 1 million dive profiles and accident data by early in the next millennium.

In conclusion, it is clear that attention to the factors discussed could help to materially reduce the accidents and deaths occurring in recreational diving. In the final analysis, however, it is the diver who must be responsible for his or her own safety. Joining DAN is a positive step in this direction too.

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