and not systemic, as occurs in cases following trauma or surgery. It is likely that death was from drowning when he found himself unable to ascent due to excess weights and his air supply became difficult to breathe.

Comments

This tragedy occurred as the final result of a series of sins of omission, each one individually minor and non fatal in nature. Nobody did anything terribly incorrect but neither did anyone remember Murphy's Law. Those involved were trained and intelligent and well intentioned but they failed to check that matters were as they appeared to be. The initial mistake was the issue of an Advanced Diver certification to divers of such limited experience, and a failure to convey to them their continued status as grossly inexperienced divers. It was this failure which made the tragedy possible.

Next came the communication breakdown, totally correct but incomplete information being provided with the request by their instructor to another person concerning their status as divers. Their possession of the correct documentary authority to confirm their "advanced" status led to an omission of what would have been an automatic, checking of their experience, had this been a dive shop organised boat dive. Their having an unjustified belief in their diving skills (as contrasted with their undoubted knowledge) led the others on the dive trip to forget to enquire concerning their diving abilities. All such factors were in place before the dive commenced.

Such was their confidence that the two divers brushed aside comments suggesting that they were overweighted for the proposed dive, forgetting their book-learning concerning depth related loss of wet suit buoyancy. Their confident management of their equipment and talk of wreck dives made easy the very natural decision of the other divers to take their usual dive partners rather than partner the visitors, the good visibility making this appear to be a safe and simple dive.

Failure to locate the anchor when the time for ascent drew near led them to expend precious air in their search for it, so they were close to a critical low-air state when making their decision to ascend. It was here that a fault which they had acquired during training produced their final joint error in that when they commenced their ascent the victim was below and therefore out of sight of his buddy. The final actions of the victim cannot be known but he may have found his air less readily available and his buoyancy vest apparently failing to fill when the inflation button was pushed, and forgotten there was the option of dropping his weight belt.

The final item in this catalogue of misunderstandings and procedural errors was the autopsy report, although this is more a matter of conjecture than established facts. Certainly a vigorous dive to 43 metres would result in enough air being dissolved in the tissues to require subsequent elimination of excess gas after returning to the surface. This can occur via the lungs in the living but occurs in the tissues where death has prevented the circulation from assisting this task.

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THE FLYING BENDS

A review of altitude decompression sickness with case reports, from hypobaric chamber operation at RAAF Base, Point Cook.

Marcus W. Skinner

Introduction

The Royal Australian Air Force (RAAF) Institute of Aviation Medicine has conducted hypobaric chamber training (Fig. 1) at the RAAF Base at Point Cook, Victoria, since 1962. All initial entry trainee aircrew (pilots, navigators, engineers and loadmasters) of the RAAF, Royal Australian Navy, Army and Air Traffic Control trainees undergo high altitude (hypobaric pressure) training. Experienced military pilots undergo refresher training at intervals of three years. The hypobaric chamber at Point Cook is also used for other members of the Australian Defence Force, overseas defence members and for civilians who require experience in the pressure changes of high altitude, including private pilots, glider pilots, balloonists and Nepal trekkers.

Air Force members who undergo very high altitude decompression to 13,500 m (45,000 ft) with predenitrogenisation include RAAF pilots and RAAF medical officers. Members undergo hypobaric experience training to prepare them for a rapid decompression, simulating the loss of cabin pressure in a military aircraft at high altitude. The effects of hypoxia and pressure breathing are also experienced in the chamber.

For the inexperienced a rapid loss of cabin pressure when at high altitude can be a frightening experience as has been clearly demonstrated in recent civilian aircraft accidents. The sudden exposure to rapid lowering of pressure is usually accompanied by loud noise, rapid drop in ambient temperature and sudden appearance of fog, all combined with rapid gas expansion within body cavities, giving rise to typical rapid pressure change symptoms such as ear pain and discomfort, abdominal distension, belching and flatus.

This article presents a review of hypobaric decompression sickness and illustrates this with some case reports.

FIGURE 1. Aircrew trainees undergoing hypobaric simulation at RAAF Point Cook.

History

In the year 1783 near Lyon man realised his dream of ascending to the heavens by means of a balloon, but it was not until 1934 at the Army Air Services Aero Medical Laboratory at Wright Field, Dayton, Ohio, that theoretical and practical investigations into many new aspects of Aviation Medicine occurred. It was at this time that research into the new entity, labelled by Armstrong "aeroembolism" or dysbarism (now called altitude decompression sickness), was commenced. Notably, this was over 100 years after Robert Boyle reported his experiments on the effects of pressure changes on experimental animals.

Decompression sickness (DCS) resulting from exposure to altitude is similar to that occurring after decompression from a high pressure environment, as in diving or caisson work. The effects of diving and caisson work exposure have been clearly recognised and studied for over 100 years (Triger in 1841 noted cases of decompression sickness in caisson workers) but only in the past 45 years has altitude decompression become important with flights into significantly hypobaric environments.

In the 1930s Armstrong[1] first demonstrated the vaporistion of body fluids at 19,000 m (63,000 ft) (now called Armstrong's line) and was the first to point to the dangers of decompression in flight. In the studies of Armstrong and Heim[2] on the effect of flight on the middle ear, where humans were systematically exposed to simulated altitudes in a decompression chamber, they demonstrated the fact that exposure to high altitude caused symptoms similar to those of caisson disease. Armstrong pointed out that the basic physical mechanisms were the same, whether a subject "ascended from four atmospheres to one or from one atmosphere to 0.25 atmosphere[4]".

Other countries were slow to follow the American lead in Aviation Medicine research. In 1939 the Royal Air Force Physiology Laboratories were only housed in a hut at Hendon and prior to World War II the Luftwaffe had only just commenced research into medical aspects of high altitude flight in hypobaric chambers using human subjects.

In actual flight operation in World War II[3], even with rates of ascent of 910 m (3,000 ft) min as in the P-51 Mustang and Griffon-engined Spitfire, actual symptoms of altitude

decompression sickness were rare. (This was probably due to the common practice of washing out nitrogen by pre-breathing 100% oxygen on the ground prior to flight.) Most commonly DCS was observed in bomber crews working in cold depressurised areas under physical stress.

Prior to 1959, over 17,000 cases of altitude decompression sickness were reported in numerous publications. Of these, 743 were reported as serious, including 17 fatalities.

Over the next two decades the incidence of altitude DCS decreased with increasing awareness of the condition, improved treatment regimes and facilities. In 1963 Downey[5] showed that bubbles produced in vitro in human serum cleared when compression to greater than sea level pressured occurred.

In 1969 Fryer published an extensive monograph on the various aspects of altitude DCS[6]. Between 1975 and 1985 90 cases of altitude DCS were reported in the Air Force Safety Journal (USAF)[7]. The altitude decompression sickness mishap rate was quoted in the range of 0.18-0.38 incidents per 100,000 flying hours, with trainer and cargo aircraft having greatest incidence. Importantly 68% of cases occurred between 5,500 m and 7,600 m (18,000 to 25,000 ft). The last reported fatality due to altitude DCS was in 1988 and involved a 51 year old USAF pilot[8].

Advances in technology have enabled the development of systems capable of transporting man into increasingly more hostile pressure environments, both hypobaric and hyperbaric. However, the understanding of physiological consequences of this exposure was poorly understood and the development of practical life support systems and treatment of patients exposed to these hostile pressures lagged behind the scientific progress. These consequences are now much more predictable and effective, safe advice can be given to individuals who wish to partake in diving and flying environments.

What is altitude decompression sickness and when does it occur?

Altitude decompression sickness is a well recognised consequence of exposure to hypobaric conditions in aircraft and hypobaric chambers. The same physical principles apply to hyperbaric conditions although the precise mechanism has never been unequivocally determined in either. It is clear that as the ambient pressure falls bubble formation occurs in the gas saturated body tissues. Saturation is due to the relatively poor solubility of nitrogen in blood so that the rate of fall of the partial pressure of nitrogen in the tissues on ascent to altitude lags behind that of the ambient pressure, in exactly the same way as ascent from depth in diving.

The mechanism involved in both altitude and diving decompression sickness are identical[9]. Studies on the factors influencing bubble formation show that significant differential pressures are required for bubbles to form spontaneously. It is not the aim here to summarise all the theoretical evidence, suffice to say that the tendency for bubbles to form is greater as the difference between the two pressures increases. Some nucleus, such as vessel irregularity, appears to be needed around which bubbles form.

The main factors[10] that influence the incidence of altitude decompression sickness, including scuba diving, are considered below. Interestingly Balladin[11] clearly showed venous gas bubbles in humans at altitudes of 910 m (3,000 ft) three hours after a no stage decompression dive to 50 ft.

Altitude Exposure

The threshold altitude has been reported as 5,500 m (18,000 ft)[12], but may be as low as 3,000 m (10,000 ft)[13]. Evidence at the USAF School of Aviation Medicine at Brooks Air Force Base in Texas indicates that bubble formation in body fluids may occur at this lower level, although these bubbles may not always be symptomatic. A study by Malconian[14] illustrated that altitude decompression sickness occurs at relatively low altitudes with repeated exposure to 4,500 m (15,000 ft). With increasing altitude above 5,500 m (18,000 ft) the incidence increases.

Rate of Ascent

The rate at which altitude is achieved is important. Contrary to earlier expectation the concept of explosive decompression sickness ,as might be expected when ejecting from an aircraft pressurised to 2,100 m (7,000 ft) cabin altitude to an environment at 13,600 m (45,000 ft), has been difficult to demonstrate experimentally below 19,000 m (63,000 ft) (Armstrong's Line[15]). A greater physical risk is hypoxia and loss of consciousness in 12-15 seconds[10]. The risk of barotrauma is also high[16]. Exposure to environmental pressure less than the vapour pressure of water at body temperature, higher than 19,100 m (63,000 ft), results in immediate and complete anoxia and ebullism (the boiling and outgassing of body fluids)[17]. Re-exposure, repetitive non-pressurised ascents to 7,600 m (25,000 ft), have been shown[4] in USAF studies to predispose aircrew to DCS. The decision by aircrew to remain at an altitude in excess of 5,500 m (18,000 ft) for mission requirements following depressurisation led to 68% of all USAF altitude DCS incidents. Many factors that influence the incidence of diving decompression sickness also correlate with the hypobaric environment.

Sex

Studies on female astronauts called upon the participate in extra-vehicular activities and exposed to hypobaric suit pressures clearly established a higher incidence of altitude DCS in females. The female:male ratio of altitude DCS was 3:1.

Age and Body Build

Early clinical analysis of thousands of altitude chamber decompressions during World War II revealed that relative susceptibility to altitude DCS increased by 9 fold between the ages of 18 and 28 years.

Exercise

It is well established that exercise at altitude increases the incidence and severity for altitude DCS. The effect of heavy exercise is equivalent to an increase in the altitude of exposure of 1,500 m (5,000 ft).

Previous Injury

No convincing evidence exists to associate previous injury with a higher incidence of altitude decompression sickness on theoretical grounds, but altitude DCS is seen more commonly in previously injured limbs.

Alcohol

The after effects of alcohol ingestion increases the susceptibility to altitude DCS.

Preflight Denitrogenisation

Preflight inhalation of 100% oxygen decreases the incidence of bends in proportion to the time of denitrogenisation. 30 minutes of breathing 100% oxygen will provide a significant degree of protection.

Flying following scuba diving

With many diving holiday packages now offered people fly to their dream diving destination, dive intensively and then fly home. Many are naively unaware of the dangers they are taking by extending their diving to the limit of their holiday.

Flying after diving can predispose to decompression sickness unless there has been sufficient time (surface interval) to allow excess nitrogen to diffuse out of the tissues. When the ambient pressure is reduced even further by climbing to altitude, bubbles may form.

Decompression sickness has been described during flight when scuba diving had taken place before departure[20].

Studies indicate that silent venous gas bubbles form at low altitudes. This has been confirmed by the intravascular presence of bubbles at 900 m to 3,000 m (3,000 to 10,000 ft) cabin altitude with ordinary no-decompression dives preceding altitude exposure by three hours[11]. It was noted that bubbles appeared within minutes of flight. This phenomenon was also seen when flying 24 hours after diving, but at a cabin altitude of 7,600 m (25,000 ft). A causal relationship between these Doppler (ultrasound) intravenous bubbles and the development of symptoms has yet to be established.

There is a small risk of decompression sickness after diving not followed by flying, even if the decompression tables are obeyed accurately. There is also a very small risk that silent stationary bubbles, which are just too small to cause symptoms at surface pressure, will do so with decompression to low altitudes. Cases of DCS have been shown to worsen during low-level helicopter transport[21], although in the main helicopter transport is safe.

Edmonds et al[22] advise that flight in an aircraft at cabin altitudes of 1,500 to 3,000 m be only conducted at least two hours after a no-stop (no-decompression) dive and 24 hours after a dive needing decompression stops.

In 1982 the British Medical Advisory Committee adopted safety guidelines for flying after diving. They recommended that for a no-decompression dive, with total time under pressure of less than one hour, the required time before flight to cabin altitude of 600 m (2,000 ft) minimum of two hours and to 2,450 m (8,000 ft) a minimum of four hours. All other compressed air dives required 12 hours before flight. Military aircrew who dive are restricted from flying duties for 24 hours.

Aeromedical evacuation of patients with decompression sickness

Movement of a patient with decompression sickness sometimes poses problems when the hyperbaric treatment facility is located at a significant distance from site of injury. Most aircraft are pressurised to 1,500 to 2,450 m (5,000 to 8,000 ft) cabin altitude and therefore flight will increase the size of bubbles.

Dully[22] showed that complacency and lack of rapid treatment for decompression sickness can result in severe complications, and that for long distance travel, movement by air is most appropriate although not without danger. If bubbles are causing pain then as they enlarge symptoms will worsen. Cases of decompression sickness are therefore best transported by aircraft at sea level pressure. The C-130 Hercules operated by the RAAF is capable of maintaining sea level pressurisation at relatively high altitude (5,800 m) (19,000 ft) and is therefore an ideal aircraft for this purpose when transportable chambers are unavailable.

For relatively short flights and for areas which do not have pressurised fixed-wing aircraft, the helicopter offers an excellent alternative. A study by Reddick[21] shows that movement of patients with decompression sickness by low-level helicopter flight is both safe and effective, especially when a pressurised aircraft is neither available nor practical.

Altitude Decompression Sickness from Hypobaric Operations

Hypobaric chamber exposures have proved to be a very safe and cost effective way to introduce flyers to the physiological limitations of unpressurised flight and the correct use of life support equipment. Deaths are rare, however fatal case reports[8,24,25] clearly demonstrate the rapidity with which seemingly mild symptoms can progress.

In the US Army, hypobaric chamber operations over a 63 month period showed the overall incidence rate for decompression sickness was 1.38 per 1000 exposures. The rate for technicians monitoring these was 6.16 per 1000 exposures and the rate for students was 0.64 per 1000 exposures[26]. The reason for this substantial difference is complex but the technicians have repeated exposure, are generally older and go to higher altitude.

All Australian defence force members who undergo hypobaric instruction and suffer, either during or after an actual decompression, untoward symptoms have a Decompression Chamber Physiological Incident report completed. This aims to develop improved control and treatment of chamber incidents, to monitor aeromedical training procedures and to evaluate individual recovery.

Since 1984 a total of six cases of altitude decompression sickness have been recorded from hypobaric chamber runs at Point Cook giving an incidence similar to that of the US Army hypobaric chamber operations.

Basic Flight Profiles

There are three basic profiles carried out in RAAF hypobaric chambers:

A The type A profile is designed to provide a rapid decompression from 2,450 m (8,000 ft) to 7,600 m (25,000 ft) to allow students to experience hypoxia at 7,600 m (25,000 ft) and to familiarise them with the use of oxygen equipment.

B The type B profile is designed to demonstrate the problems of vision at night and in particular, the effect of hypoxia, with decompression to 4,500 m (15,000 ft) for 35 minutes to allow for dark adaption.

C The type C profile is designed to provide rapid decompression from 7,600 m (25,000 ft) to 13,600 m (45,000 ft) and allow students to experience pressure breathing at 13,600 m (45,000 ft) (for 30 seconds), then hypoxia symptoms at 7,600 m (25,000 ft) and "free fall" from 7,600 m (25,000 ft) to 3,000 m (10,000 ft) using the emergency oxygen cylinder.

Case Reports

CASE 1
Onset of joint pain at altitude and persisting after descent.

A 33 year old RAAF member was undergoing initial decompression training. He was decompressed to 7,600 m (25,000 ft) and after seven minutes at this altitude he developed pains in the right elbow which increased in severity. Simultaneously right shoulder pain was noticed. On return to sea level pressure he complained of increasing pain in his right arm. A tentative diagnosis of joint DCS was made and he was put on 100% oxygen, rested, given fluids and transferred by road to a hyperbaric chamber for therapy. After 30 minutes on 100% oxygen his pain had almost gone but when oxygen was ceased during casualty assessment, prior to hyperbaric treatment, his symptoms returned to full. He was compressed on oxygen to 18 metres of seawater for five hours and his symptoms completely resolved. The significant predisposing factors in this incident were a mild injury to his right elbow one week prior to "decompression" and that he had flown by an HS-748 aircraft, along with other members from another RAAF Base, in the morning prior to chamber run. The duration of the flight was 0.7 hours, peak cabin altitude of only 300 m (1,000 ft) and there was no recent diving.

CASE 2
Joint pains and skin symptoms two hours after chamber flight.

A 37 old RAAF pilot who assisted in the running of the hypobaric chamber underwent a 7,600 m (25,000 ft) standard A run decompression. He completed it with a minor degree of apprehension due to ear pain on descent, but sustained no otic barotrauma. Two hours after finishing the decompression run he developed abnormal skin sensations over his forehead and back along with marked temporomandibular joint pain. Symptoms were only partially relieved by 100% oxygen. The patient was transferred to a hyperbaric facility and was treated on RN table 62 with rapid and full resolution of all symptoms. The significant predisposing factor was that the member forgot to undertake prebreathing 100% oxygen before recompression.

CASE 3
Joint pain on descent from altitude.

A 23 year old Army pilot undertook a type A hypobaric chamber run. He remained at 7,600 m (25,000 ft) for 15 minutes and on descent at 4,100 m (13,500 ft) he complained of left elbow pain. There were no predisposing factors. With 100% oxygen at ground level, the pain ceased. A hyperbaric specialist was consulted but because local recompression was not available and as the patient's state was satisfactory, conservative management was undertaken, with full resolution of symptoms.

CASE 4
Joint pains left elbow.

A 23 year old RAAF pilot undertook a type A hypobaric run to 7,600 m (25,000 ft). After eleven minutes when descent to 4,100 m (13,500 ft) was commenced, the member complained of left elbow pain. The "flight" was

aborted and the member placed on 100% oxygen with rapid resolution of his symptoms. There was no recurrence of pain. The member had not been diving and had no other significant predisposing factors.

CASE 5
Possible neurological decompression sickness.

A 31 year old chamber attendant participated in a standard A run to 7,600 m (25,000 ft) without incident. After the decompression run he developed slurred speech and right C8 dermatome dysaesthesia. He was confused, with blurring of his vision. He was urgently transferred to a hyperbaric unit, where with hyperbaric treatment, symptoms resolved completely. The only predisposing factor was that he had been jogging the evening prior to the decompression.

CASE 6
Neurological and joint decompression sickness.

The patient, a 41 year old Naval officer, underwent a type A flight without incident. 25 minutes after the flight he noticed right shoulder pain and this persisted until he fell asleep on the evening of the flight. It was not present the following morning. He did not make his symptoms known at this time, although the pre-flight brief clearly requested immediate notification of any symptoms. He underwent a second hypobaric run to 4,600 m (15,000 feet) and seven minutes into this he complained of marked tingling and pain in the right shoulder. The run was terminated and he was placed on 100% oxygen. Within 30 minutes he had no further symptoms but shortly after removal of his oxygen mask his symptoms returned and he developed slurred speech. It was at this stage the previous day's symptoms were admitted. He was put back on 100% oxygen and evacuated by a C-130 Hercules aircraft, pressurised to sea level, to a hyperbaric unit where with treatment his symptoms completely resolved without sequelae.

Discussion

The clinical manifestations of altitude DCS are varied. Table 1[27] presents the relative incidence of symptoms of altitude DCS.

The uniformly prompt response to 100% oxygen and hyperbaric therapy in all of the cases presented indicates that these patients were correctly diagnosed as suffering from decompression sickness.

The most common manifestation observed in the cases from hypobaric operations at RAAF Point Cook were joint and limb pain. In all these cases local pressure by means of a tight bandage or pneumatic cuff relieved the pain.

The USAF student exposures in hypobaric chambers show that joint pain symptoms alone predominated in 60% of treated cases with or without delayed onset.

TABLE 1

RELATIVE INCIDENCE OF SYMPTOMS OF ALTITUDE DECOMPRESSION SICKNESS

Symptom	Incidence (%) 8,500 m (28,000 ft) for 2 hours	11,200 m (37,000 ft) for 2 hours
Joint and limb pain	73.9	56.5
Respiratory disturbances	4.5	6.5
Skin disturbances	7.0	1.6
Visual disturbances	2.0	4.8
Neurological disturbances	1.0	.0
Collapse	9.0	25.8
Miscellaneous	2.5	4.8

Itching, tingling (the creeps) and formication often occur at altitude and are usually transient and only rarely progress to more serious manifestations. More severe skin manifestations of altitude DCS are possibly due to embolism[27].

Respiratory disturbances, the chokes, are an uncommon manifestation of altitude DCS but if the exposure to altitude is maintained the chokes almost invariably progress to collapse and death. The patient is pale, restless, peripherally shutdown but clammy with increasing bradycardia and hypotension. The patient then may lose consciousness. Fortunately it is rare.

Unlike divers, aviators rarely experience spinal cord manifestations of neurological decompression sickness, although cases 5 and 6 both appear to have developed neurological decompression sickness. Paralysis, paraesthesia and fits occur but no disturbance of smell or taste has been reported. Labyrinthine involvement is very rare.

The confusing and varied picture of patients with neurological decompression sickness has been readily mistaken for hysteria or hyperventilation by the uninitiated and should only be made when decompression sickness is excluded.

Aseptic bone necrosis seen in deep sea divers and abalone divers is almost non-existent in altitude decompression sickness. This disorder has not been reported in USAF hypobaric chamber attendants over a 20 year period[26].

Treatment

In nearly all cases of altitude DCS recovery is rapid as descent is carried out to low altitude but the definitive treatment of altitude decompression sickness involves immediate recompression in exactly the same way as for diving decompression sickness. It is not within the scope of this article to present the treatment regimes provided. RAAF Medical Officers seek advice from hyperbaric medicine specialists when a case of altitude decompression sickness is suspected of requiring treatment after immediate supportive therapy is commenced.

Conclusion

The effect of hypobaric chamber flights is analogous to returning to the surface after surface supplied scuba diving and carries the risk of decompression sickness. The cabin of an airliner can be considered a hypobaric chamber and therefore divers returning by air increase their risk of developing decompression sickness if they have been pushing the limits of their tables. Medical practitioners need to be aware that altitude-induced decompression sickness, although well described in military aviation medicine, can occur in civilians and its onset may be significantly delayed. It is essential that the condition is recognised by a careful history and clinical examination and immediate arrangements made for urgent transfer to a hyperbaric unit.

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SOLO DIVER

Bob Halstead

As an active instructor for 18 years I have observed the buddy system in operation on thousands of dives. This also means that I have seen the buddy system fail on thousands of dives. I think that the idea of two divers sharing a dive and caring for each other is a wonderful idea but in practice it is an almost impossible achievement. We know what should happen, but how many times have you seen buddies that are incompatible, either through ability or interest, or where one is dependant on the other, or where the only sign of buddy activity is at the surface under the direction of the dive master, underwater the divers go their own way or are so far apart they are virtually alone? How many dives have you seen where the buddies have spent the dive looking for each other, yes and alternately coming to the surface (the most hazardous place to be)? How many dives have you seen spoiled because of the buddy system, and how many divers are put off diving because of the buddy system, either because they cannot find a buddy or they think about what the fact of the buddy system tells us about diving? Are we still "braving the deep", is it really dangerous to dive alone?

I used to think I could do something about this and teach people how to buddy dive. It is a bit like marriage guidance. "Now Jane when you saw Jim signal that he was out of air and going to ascend, why did you chase off after the whale shark that was swimming past? What would a good buddy have done? Yes, I know you had plenty of air, but...".

Now I have more than a sneaking suspicion that some of you would have abandoned Jim too, for that swim with the whale shark, for the lobster you have just spotted, for the photo that is just a moment away, sometimes just for the fact that you have still got half a tank of air left and do not want to come up yet. I say this with some authority since for the past two years I have been operating our liveaboard dive boat, "Telita", and entertaining some of the world's most adventurous and experienced divers. To many, if not most, of these divers, the buddy system is a myth. OK, I admit it, after thousands of dives escorting students on training dives, I just love to dive by myself. Some of my most memorable and joyful dives have been with my lifetime buddy, and fellow instructor, my wife Dinah. Sharing underwater adventures together is something that makes our love stronger and our marriage more fulfilling, nevertheless we both enjoy the occasional dip by ourselves. What I am saying is that buddy diving, like marriage, does not work for everyone all the time. People can, will and do solo dive, but are they trained for it?

Instructor organisations have a choice, they can condemn solo diving, and by doing so ignore what I believe to be a distinct trend in diving. Even a recent Skindiver editorial (famous for its conservative views) mentioned a solo diver being "with" someone in the boat. Or they can take a pioneering view and determine under what conditions solo diving could be accepted as a "safe" activity. I believe that for some people in certain conditions solo diving is a safe diving activity in the same way that I believe that some people will never be safe diving no matter how good the conditions, or their buddies, are. I find it easy to accept that it is safer for an instructor to dive by himself or herself than to be leading two students on an early dive.

There is something else here as well that is not so obvious. Teaching the buddy system teaches dependence. I know it should not, but it does. We call that negative incidental learning, and it is something that we are all warned about at Instructor Training Courses. Because so many of our training exercises involve the buddy, we install in the student the subconscious reasoning that they do not have to be as proficient as all that because they will always have their