

## Critical Review

# Epilepsy and Recreational Scuba Diving: An Absolute Contraindication or Can There Be Exceptions? A Call for Discussion

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**Summary:** Recreational scuba diving is a popular sport, and people with epilepsy often ask physicians whether they may engage in diving. Scuba diving is not, however, without risk for anyone; apart from the risk of drowning, the main physiological problems, caused by exposure to gases at depth, are decompression illness, oxygen toxicity, and nitrogen narcosis. In the United Kingdom, the Sport Diving Medical Committee advises that, to dive, someone with epilepsy must be seizure free and off medication for at least 5 years. The reasons for this are largely theoretical. We review the available evidence in the medical literature and diving websites. The risk of seizures recurring de-

creases with increasing time in remission, but the risk is never completely abolished. We suggest that people with epilepsy who wish to engage in diving, and the physicians who certify fitness to dive, should be provided with all the available evidence. Those who have been entirely seizure-free on stable antiepileptic drug therapy for at least 4 years, who are not taking sedative antiepileptic drugs and who are able to understand the risks, should then be able to consider diving to shallow depths, provided both they and their diving buddy have fully understood the risks. **Key Words:** Epilepsy—Recreational scuba diving—Seizure.

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Recreational scuba diving (using “self-contained underwater breathing apparatus”) is a popular sport and it is not uncommon for neurologists to be asked by their patients with epilepsy whether they may dive. People in the United Kingdom, taking antiepileptic drugs (AEDs) for epilepsy, whether or not experiencing seizures, have been banned from scuba diving (Hallenbeck, 1984; Millington, 1985; Sykes, 1994; Bove, 1996; Taylor et al., 2002). The UK Sport Diving Medical Committee considers that the requirements that someone with epilepsy must fulfill before being passed as able to dive are “5 years free from fits and off medication” (UK Sport Diving Medical Committee, 2004). Both the Professional Association of Diving Professionals and the National Association of Underwater Instructors regard a history of seizures as contraindications to diving (NAUI, 2005; PADI, 2006).

Scuba diving is a hazardous sport, as it requires mechanical aids to support respiration, and is performed under wa-

ter. Essentially, the diver carries a tank of high-pressure air (or suitable gas mixture), and this is delivered to the diver at ambient pressure by means of a regulator. The diver also carries a weight system and a buoyancy control device (BCD). Diving associations advise people to dive with a “buddy,” a diving partner who shares mutual responsibility for safety.

Apart from the hazards caused by the marine environment and the risk of drowning, there are three main physiological problems that may occur as a result of exposure to gases at depth. These are decompression sickness or illness (DCI), oxygen toxicity, and nitrogen narcosis. These problems can all occur in any diver; each therefore needs to assess the risk/benefit ratio before diving. Questions that need to be addressed include:

1. What are the physiological risks for anyone scuba diving?
2. Are these risks any greater for a person with a history of seizures?
3. Do AEDs themselves pose a risk for someone scuba diving?

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4. Are seizures likely to be induced by recreational scuba diving in people with epilepsy controlled by AEDs?
5. Is recreational diving by a person with controlled epilepsy likely to endanger the life of others?
6. What benefits may people with epilepsy derive from scuba diving?

A further physiological problem that could be considered is carbon dioxide toxicity. This is most likely to occur in divers using a closed or semiclosed circuit rebreathing apparatus (DeGorordo et al., 2003); most recreational scuba divers use open circuit apparatus. It may also occur when exercising at depth, or due to breath holding, neither of which is recommended for recreational divers. It will therefore not be considered further. Carbon monoxide poisoning may also affect any diver after a "bad fill," and vomiting from whatever cause could prove fatal.

We looked for evidence about diving and epileptic seizures within the medical literature and by accessing diving websites. A PubMed search of English language articles published between 1984 and 2006 was conducted using the following search terms: (1) epilepsy and diving; (2) seizures and diving; (3) diving fatalities; (4) diving injuries; (5) diving accidents; (6) diving morbidity; (7) diving mortality; (8) diving physiology; and (9) drugs and diving. We found a paucity of evidence on this subject. We also investigated the epidemiology of diving in people with epilepsy, to see whether any theoretical risks are borne out in practice.

### PHYSIOLOGICAL RISKS INHERENT TO RECREATIONAL SCUBA DIVING

#### Decompression sickness/illness

Decompression illness results from the reduction in the ambient pressure surrounding a body (Thalman, 2004). As the diver submerges, and the surrounding pressure increases, more nitrogen ( $N_2$ ) is forced into solution in the body tissues as the partial pressure of  $N_2$  increases (Barratt et al., 2002). The tissues become saturated and as  $N_2$  is inert it is removed only by diffusion or blood flow. When the pressure decreases as the diver later surfaces, the dissolved  $N_2$  returns to the gaseous state faster than the blood can remove it. Rapid ascent is considerably more dangerous than a controlled ascent, which allows more time for the  $N_2$  to be removed by the blood flow. The  $N_2$  not removed is liable to form bubbles in the musculoskeletal system (causing "the bends"), and in the marrow of long bones, although whether or not the bubble formation is the cause of the symptoms of DCI is not proved (Hamilton and Thalman, 2003). If  $N_2$  cannot escape through the airways, usually because of breath-holding, it can rupture into the pulmonary venous system [leading to cerebral arterial gas embolism (CAGE)], the perivascular sheaths or the pleural cavity (Barratt et al., 2002). Asthma and bronchitis can also facilitate CAGE by trapping gas in the

lungs (Newton, 2001) (although mediastinal emphysema or pneumothorax are more likely), as can patent foramen ovale and other causes of right to left shunt, (Madsen et al., 1994; DeGorordo et al., 2003) as bubbles are able to pass from the venous to the arterial circulation. CAGE accounts for almost one in four fatalities in recreational divers (Newton, 2001). Patients with CAGE may present with alteration of consciousness, seizures, (Newton, 2001) "stroke"-like symptoms, or paralysis (Bove, 1996). Excess nitrogen can remain for hours in the body, and, although the initial effects of bubbles are mechanical, there may also be secondary biochemical effects of activation of leukocytes, platelets, complement, and the clotting cascade (Barratt et al., 2002).

DCI can, to a large extent, be prevented by avoiding rapid ascent and by use of decompression tables or diving computers. It is, however, a probabilistic event, and could occur on any dive (Madsen et al., 1994). Other risk factors include deep/long dives, cold water, and hard exercise at depth (Thalman, 2004). DCI can occur even when no rules have been violated, and this is referred to as "undeserved" or "unexpected" DCI. It is said that DCI is "still a mystery in many ways" (Almon and Ugucioni, 1997). It used to be thought that it was possible to spend an unlimited amount of time at depths of 9 m or less (Barratt et al., 2002); while this is no longer held to be true, dive tables indicate that the risk of DCI at depths of less than 10 m is very small, provided the dive is not prolonged (Spira, 1999).

#### Oxygen toxicity

Hyperbaric oxygen ( $O_2$ ) exposure is known to cause CNS  $O_2$  toxicity, usually manifested by seizures (commonly focal, but generalized seizures may occur in 5–10% patients (Newton, 2001)); the mechanism is unknown (Chavko et al., 1998). It has been suggested that it may be due to changes induced in cerebral blood flow (Chavko et al., 1998) or to the formation of free radicals from molecular  $O_2$  during metabolism (Green and Leitch, 1985; Newton, 2001; DeGorordo et al., 2003). It is also known that GABA falls after exposure to increased pressures of  $O_2$  (Green and Leitch, 1985).

The risk of  $O_2$  toxicity increases with the pressure of  $O_2$  experienced and the duration of exposure (Emerson, 2002). It occurs more often at depths of 50 m or more when breathing compressed air (Green and Leitch, 1985), but may occur at 2 atmospheres pressure (approximately 10-m depth) or less when pure oxygen is being breathed (Spira, 1999). The overall incidence of  $O_2$  induced convulsions is extremely low (around 0.03% in a hyperbaric chamber) (Hampson and Atik, 2003).

#### Nitrogen narcosis

Nitrogen narcosis is the narcotic-like effect precipitated by increased partial pressure of inspired  $N_2$  at depth (Emerson, 2002). Although it can happen to any diver (Worf, 2002) and is not entirely predictable, it usually

occurs at deeper depths and with more rapid descent, (Emerson, 2002) and resolves with return to the shallows (Worf, 2002). The symptoms increase with depth, starting as light-headedness and euphoria from 30 m, through poor judgment and overconfidence, and hallucinations, to loss of consciousness at great depths (Royal Naval Medical Service, 2004). The behavioral disturbances manifested seem to occur regardless of which inert gas is breathed, and, although the mechanism is not yet elucidated, it is assumed to be similar to those of general anesthetics (Turle-Lorenzo et al., 1999). It may be, however, that nitrogen acts directly by potentiating GABA neurotransmission at the GABA<sub>A</sub> receptor, thus producing its narcotic effect (Abraini et al., 2003).

### ARE THESE RISKS INCREASED IN SOMEONE WITH CONTROLLED EPILEPSY?

#### Decompression sickness/illness

There is no published literature to support an association between DCI and epilepsy. Uncontrolled epilepsy could, however, predispose to the development of DCI if a seizure occurred while underwater, thus precipitating an acute ascent (Madsen et al., 1994). It is therefore unlikely that someone with fully controlled epilepsy would be more at risk of DCI than someone without epilepsy. It is possible, however, that CAGE could be precipitated by a fast ascent from as shallow as 10 m if breath holding during a convulsive seizure occurred.

#### Oxygen toxicity

It is important to try to establish whether people with controlled epilepsy are more likely to suffer from O<sub>2</sub> toxicity, and hence from its seizures, than those without epilepsy. Two case histories have been reported which may suggest that people with epilepsy are more likely to have such seizures in a hyperbaric chamber than those without epilepsy (Emerson and Ozer, 1998). One, an experienced diver, developed DCI after a "dive" in the chamber. On recompression with O<sub>2</sub> she had a generalized seizure preceded by a simple partial seizure, and afterwards an EEG was reported as being abnormal, showing generalized epilepsy. This was reported as "unmasking latent epilepsy." However, as the EEG changes were generalized, and the seizure was of partial onset, it is unclear whether the two were closely related. The second was a female having a therapeutic compression, who had a "grand mal" seizure. She had, however, been treated with high doses of pethidine, a metabolite of which may be proconvulsive. Thus it seems unlikely that either of these cases had an intrinsically altered seizure threshold which was affected by hyperbaric oxygen (and therefore unlikely that these case reports show that people with controlled epilepsy are more likely to suffer from O<sub>2</sub> toxicity and seizures than someone without). A recent case report describes partial seizures developing in a man treated in a hy-

perbaric chamber, but the patient had recently had surgery for frequent seizures secondary to radiation necrosis following treatment for an anaplastic astrocytoma (Doherty and Hampson, 2005). No other recent histories are available of people with known epilepsy being treated in a hyperbaric chamber. A study in the 1940s found no correlation between preexisting EEG abnormalities and time to convulsion in people exposed to high partial pressures of O<sub>2</sub>. Indeed, two patients with gross EEG abnormalities who were diagnosed with epilepsy had no increased tendency to convulse (quoted in Green and Leitch, 1985). One study is reported as showing hyperbaric O<sub>2</sub> treatment producing reduction of seizures in children with epilepsy, but the scientific nature of this study is questioned (quoted in Hardy et al., 2002).

#### Nitrogen narcosis

There is no reason to suspect that people with a history of seizures would be more likely to suffer from Nitrogen Narcosis than anyone without such a history.

### ARE THESE RISKS INCREASED IN SOMEONE TAKING AEDS?

#### Decompression illness

The secondary biochemical effects of DCI could potentially interact with AEDs. These effects are secondary to the formation of bubbles, however, and are therefore unlikely to take effect until the diver is safely on land. Anyone suffering from DCI should not dive again for one month (Emerson, 2002) by which time the biochemical effects should also have cleared. There is no other reason why DCI should preferentially affect anyone taking AEDs.

#### Oxygen toxicity

There is no reason to suppose that AEDs would increase the risk of O<sub>2</sub> toxicity. Indeed, in rats, carbamazepine has been shown to exhibit a dose-related protective effect against hyperoxic seizures (Reshef et al., 1991), so people with epilepsy controlled by carbamazepine could potentially also be protected. A case report of CAGE causing seizures in a hyperbaric environment showed that phenytoin also appeared to inhibit seizure activity (Weaver, 1983).

#### Nitrogen narcosis

Some drugs, particularly alcohol and sedatives, are thought to have an additive effect on N<sub>2</sub> narcosis (Royal Naval Medical Service, 2004). As stated above, the mechanism of N<sub>2</sub> narcosis is assumed to be similar to that of anesthesia with nitrous oxide. The British National Formulary (Joint Formulary Committee, 2004) does not list any AEDs as having interactions with nitrous oxide anesthetics, although it states that nitrous oxide produces an increased sedative effect when general anesthetics are given with anxiolytics and hypnotics. Clearly, if any particular

AED is shown to have a sedative effect, then it would be inappropriate for someone sedated by that AED to dive; this may be difficult to ascertain objectively.

It is possible that AEDs acting on the GABA<sub>A</sub> receptor (clonazepam, diazepam, phenobarbital, primidone, topiramate, valproic acid that act directly; tiagabine that inhibits the neuronal reuptake of GABA) may interact with high partial pressures of nitrogen. This could produce unexpected side effects, perhaps including nitrogen narcosis.

#### **OTHER RISKS DUE TO AEDS**

It has been argued that people with epilepsy will have impaired performance and reaction time due to AEDs (Millington, 1985); those affected in this way by their AEDs and those taking AEDs known to be sedative probably should not dive.

Biochemical and hematological changes occur in people who dive (Philp et al., 1975), and in people who develop DCI (Jacey et al., 1976). The significance of these changes on AEDs, or indeed on any other prescription or nonprescription drugs, is unknown.

#### **ARE SEIZURES LIKELY TO BE INDUCED BY SCUBA DIVING IN PEOPLE WITH EPILEPSY CONTROLLED BY AEDS?**

A large questionnaire study showed that approximately 50% of people with epilepsy report seizure-precipitating factors, such as emotional stress (21%), sleep deprivation (12%) and physical exercise (3%) (Nakken et al., 2005). Consequently several factors operating during a dive may be associated with an increased risk of seizures underwater, because they may alter the seizure threshold. These factors include the seizure-precipitating factors above, as well as hypothermia, hyperventilation, breathing oxygen under increased pressure (see above) (Millington, 1985), and changes in drug metabolism (Fountain and May, 2003). Many of these are only important during diver training.

Up to 30% people with epilepsy may experience stress as a seizure trigger, and some report that the stress of participating in sports may exacerbate seizures (Fountain and May, 2003). Aerobic exercise may occasionally cause seizures, but is more likely to reduce seizure frequency; those whose seizures are exacerbated by exercise generally recognize the association (Fountain and May, 2003). Interictal epileptiform activity has been shown to remain unchanged or decreased during or immediately after exercise, even in some people with exercise-associated seizures (Fountain and May, 2003). Hyperventilation performed at rest may trigger absence seizures and epileptiform activity on EEG, by producing hypocapnia and cerebral vasoconstriction. During exercise, however, hyperventilation is a compensatory response to avoid hypercapnia, is usually not associated with seizure exacer-

bation, and causes relative suppression of EEG abnormalities (Fountain and May, 2003). However, inexperienced divers tend to hyperventilate more than those who have dived more often, although this is often an indication that further training is required. Tiredness and lack of sleep have also long been recognized as potential seizure precipitants (Spector et al., 2000; Nakken et al., 2005). If any person with epilepsy knows that any of these factors are likely to lower their own seizure threshold, then clearly that person should not scuba dive.

Regular physical exercise induces hepatic microsomal enzymes, and the release of fatty acids into the blood stream may compete with protein binding, leading to a higher free fraction of AEDs with important protein binding, such as phenytoin, valproate, tiagabine, and benzodiazepines. Theoretically it could be advisable to adjust AED doses in patients engaged in regular exercise, but a prospective study found no effects of exercise on the blood levels or rate of metabolism, and no correlation between minor fluctuations in the blood levels and seizure occurrence (Fountain and May, 2003).

While the secondary effects of DCI include biochemical changes, they would be unlikely to provoke a seizure.

#### **IS DIVING BY A PERSON WITH CONTROLLED EPILEPSY LIKELY TO ENDANGER THE LIFE OF OTHERS?**

The person with completely controlled epilepsy may be able to decide that the benefits of diving outweigh the small risks of having a seizure. It is imperative, however, that the health and safety of the diving buddy be considered. If a diver has a seizure at depth, the buddy would need to reach the surface quickly, putting himself at risk of DCI (Emerson, 2002). One author quotes "several instances of divers having seizures under water who went onto drown, but in addition a diving buddy also drowned while trying to rescue the convulsing diver" (Bove, 1996), but it is not clear whether the divers who had seizures did so because of epilepsy or because of O<sub>2</sub> toxicity. An Australian study of 100 consecutive scuba diving fatalities found that in only 14% of the fatalities did a buddy remain with the person who died, although a further 20% only separated because of the problem causing the fatality (Edmonds and Walker, 1989).

#### **BENEFITS OF SCUBA DIVING FOR PEOPLE WITH EPILEPSY**

People with epilepsy tend to be less active, less physically fit and less likely to participate in sports than the general population and to have more psychological problems (Fountain and May, 2003). A study of a regular exercise program on a small number of people with epilepsy showed that behavioral outcomes were positively influenced by moderate exercise with no impact on seizure

frequency; 10 of 14 randomized to exercise were seizure-free before starting the study and remained so throughout the study (McAuley et al., 2001). It is important to realize, however, that this study involved a regular, frequent and supervised exercise regimen. People with epilepsy who wish to engage in recreational scuba diving should build up their exercise tolerance, while remaining seizure-free, before considering scuba diving.

### EPIDEMIOLOGY OF DIVING DEATHS IN PEOPLE WITH EPILEPSY

As diving is currently banned for people on AEDs for epilepsy, any epidemiological data will be biased, as the numbers of people with epilepsy who dive will necessarily be very small. People with medical conditions for which diving is contraindicated are, however, known to dive. One study sent anonymous questionnaires, which included a list of medical conditions, to members of 29 Australian diving clubs; 346 replies were received (Taylor et al., 2002). For each condition individuals completing the questionnaire could answer “never,” “in the past,” or “now and I dive with it.” Almost half the divers were overweight (a risk factor for DCI), and 8% had asthma or chronic obstructive airways disease (both of which could precipitate arterial gas embolism) in their history, with 3% with current symptoms. Although none had current seizures, 2 of 346 respondents had a history of epilepsy. Other authors note that divers have commented on how easy it is to avoid the detection of medical conditions during the diving medical examination (quoted in Taylor et al., 2002). Additionally, once medical certification is received, no further assessments are required to continue diving (Strauss and Borer, 2001). The survey of 100 consecutive scuba diving fatalities in Australia and New Zealand found that 9% of victims had been specifically advised by a diving medical expert or dive instructor that they were unfit for scuba diving (Edmonds and Walker, 1989). Altogether 25% had preexisting medical contraindications, including one person with epilepsy (which probably did not contribute to the death) and nine with asthma (which may have contributed to the death in eight cases).

In 2000 and 2001, the Divers Alert Network (DAN) received notification of 2,212 injured divers who were recompressed. Only eight patients were reported to have preexisting central nervous system disease (including seizures, migraine, or central nervous system injury). In the same period, DAN was able to obtain records of 168 diving fatalities in the United States and Canada. Only three of the deaths occurred in patients with a history of seizures (including one recently hospitalized for seizures); the presumed causes of death were “air embolism” (in a subject who ascended rapidly, and in whom no AEDs were found), “drowning” (in a new diver, who struggled at the surface) and “cardiac” (shortness of breath noted

on the swim back to shore, in a man with severe coronary artery disease and type I diabetes) (Divers Alert Network, 2002, 2003). The South Pacific Underwater Medical Society records show that, between 1972 and 1999 there were four fatalities in divers with a history of epilepsy (Douglas Walker, personal communication, 2006).

The British Sub-Aqua Club (BSAC) publishes annual reports of diving incidents (Cumming, 2005) in the interest of promoting diver safety. In the last 9 years 3,744 incidents have been recorded, including 161 fatalities. There is often very little information available on the incidents. In this nine-year period the reports of three deaths (of 161) have mentioned convulsions or “fits.” One was using a mixture of gases usually employed by technical divers and therefore probably not relevant to recreational scuba diving. One collapsed while on land, but was said to have had a fit while in the water. In the other, postmortem examination showed that he had suffered “something akin to an epileptic fit” while underwater. There is no data to determine whether or not any of these three people had a previous history of seizures. A fourth fatality was related to an oxygen toxicity convulsion.

Nonfatal incidents included 22 with mention of convulsions or fits. Eleven of these were probably oxygen toxicity seizures. Three had a previous history of seizures, but had not declared them. One was due to anaphylaxis, and in the other cases it is not possible to establish whether or not the person had a history of seizures. In total, therefore, 3 of 161 fatalities and up to 10 of 3,583 nonfatal incidents may possibly have been related to epilepsy. Thus there is very little reliable epidemiological evidence to suggest that a past history of seizures does, or does not, imply increased risk to recreational scuba divers.

### DIVING AND PEOPLE WITH EPILEPSY

There is little doubt that seizures under water are extremely dangerous, particularly if consciousness is lost. Even if consciousness is not lost, the regulator could be dislodged, and the subject drown (Fountain and May, 2003). The consequences of a seizure could potentially be reduced by using a full face mask, as used by police and technical divers (Hamilton and Thalmann, 2003), instead of the usual separate device kept in the mouth by clenching the teeth. A full-face mask would be more likely to remain on the face than the usual regulator if a seizure should occur.

A seizure involving only loss of awareness could pose problems, as maintenance of position in the water requires subtle changes to be made to the air in the BCD. If these changes are not made there is a risk of accelerated ascent or descent, causing problems with DCI and risking loss of contact with (and therefore rescue by) the dive buddy.

DCI should not otherwise occur more frequently in people with epilepsy, but the treatment may involve

hyperbaric oxygen. There appears to be little evidence that O<sub>2</sub> toxicity, either during the dive or during recompression, is more likely to occur in people with controlled seizures. It would seem prudent, however, that people with controlled seizures should not use Nitrox (an oxygen enriched mixture, including less N<sub>2</sub> than air and sometimes used to prevent DCI), in case their propensity to have hyperoxic seizures was increased.

## DISCUSSION

In 1985 the Professional Advisory Board of the Epilepsy Foundation of America wrote that they “strongly believe that persons with epilepsy whose seizures are controlled can and should lead full lives without any personal restrictions” and that they should therefore be allowed to dive, but not teach diving, as long as they are fully aware of the risks (Dreifuss, 1985). The flurry of letters in reply to this suggested that many people did not agree (Meckelburg, 1985; Millington, 1985). Twenty years later, the situation has not changed.

Risk taking is an integral part of life and people with epilepsy, like everybody else, should be allowed to make informed decisions about risk. In the United Kingdom people with epilepsy, including those on AEDs, are allowed to drive a motor vehicle provided they have been seizure free for a year. A seizure while driving at speed could potentially injure far more disinterested people than a seizure at depth, although the latter would most likely kill the diver and endanger the life of the diving buddy. People with epilepsy may decide that the risk is worth taking. The chance of seizure recurrence after a period of remission is difficult to quantify, and is dependent on several factors; a history of tonic-clonic seizures, the occurrence of further seizures after starting AED treatment and use of more than one AED all increased the risk of seizures in a randomized study comparing continued AED treatment with slow withdrawal in people who had been seizure free for at least 2 years (Medical Research Council Antiepileptic Drug Withdrawal Study Group, 1993). The risk of seizures recurring decreases with increasing time without seizures, but the risk is never completely abolished; people with epilepsy who wish to scuba dive must be aware of this. In the MRC AED withdrawal study, of 510 patients who had been seizure-free for at least 2 years and who were randomized to continue AED treatment, 22% had had seizures within 2 years, but the risk declined steadily thereafter (Medical Research Council Antiepileptic Drug Withdrawal Study Group, 1991). We suggest that until more information about the risk is available, an empirical approach would be to delay scuba diving in people on stable AED therapy until at least 4 years have elapsed since the last seizure. The chance of a seizure happening in a subject seizure free for 4 years by chance during a 60-min period under normal, nonstressful conditions

would be one in over forty thousand. Although this risk may be increased by an unknown amount in someone under stressful hyperbaric conditions, this could be compared with the risk of DCI occurring in any dive, which is said to be one in five to ten thousand (Newton, 2001).

Data on diving in people with epilepsy who are seizure free (whether or not on medication) are not available in the literature. Objections to diving by people who have been seizure-free for a long time are largely theoretical. The situation is similar to that which was the case for people with diabetes mellitus until the early 1990s. In 1991, however, in response to advances in diabetes care, the UK diving authorities changed their recommendations and allowed certain people with well-controlled diabetes to dive (UK Sport Diving Medical Committee, 2004). The Diving Diseases Research Centre conducted a preliminary open, crossover, controlled study in a hyperbaric chamber to examine the effects of diving on serum glucose levels (Edge et al., 1997), and is conducting a further study using questionnaires on divers with diabetes doing “real life” diving. Although it would not be advisable to try to make people with epilepsy have seizures in a hyperbaric chamber, it could theoretically be possible to try to elucidate some of the presumed affects of pressure on AEDs.

There is anecdotal evidence from many practitioners that people with epilepsy may go to areas of the world where diving regulations are less stringent in order to avoid the diving ban. This is clearly very bad practice, and fatalities are far more likely to occur in these conditions. The BSAC data also confirms that people with known seizures may deny having epilepsy to avoid a ban (Cumming, 2005).

It is not possible to rule out an increased risk in people with epilepsy who are seizure free on medication, and the current data do not allow a precise assessment of the magnitude of any risk. Therefore, we recommend caution when evaluating and advising a patient with controlled seizures who requests permission for scuba diving. For each individual, all risk factors for recurrence or for possible complications should be considered. It is important that the person is aware of all the possible risks, and capable of making the decision. No person should dive if sedated by their drugs, and perhaps diving should be limited to depths no greater than 10 m, as the risks of DCI are minimal at this depth unless breath holding occurs. It has been suggested that diving would be safer if the diving buddy was a fully qualified rescue diver, fully aware of the history of seizures. Anyone who has not been seizure-free for at least 4 years should be strongly advised not to dive. People with epilepsy are at higher risk if they dive than those without epilepsy; however, many people with epilepsy will dive regardless of rules and regulations, often concealing their condition. We suggest potential divers be given the best individualized advice available and that

they should be warned of the risks and dangers, so they can make an informed decision.

There may, of course, be legal implications for practitioners in agreeing that individual people with epilepsy may be able to consider recreational scuba diving. It is important that physicians should be aware of the legal implications, and should discuss these with their insurers, particularly in countries with a litigious environment. All discussions with patients who wish to dive should be carefully documented, and it may be appropriate to ask the patient to sign a statement that the risks have been discussed.

This is still a controversial issue, and more research is needed to evaluate the risks. The practitioner can only give the patient the best currently available information, and then the decision as to whether to dive has to be the result of a consensus between the person with controlled seizures, the diving buddy, the insurer, and the family.

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