of divers and thedives that are done each year, the fatality rate seems to be very low. We can never completely guarantee our safety during a dive, but we can certainly minimise the chances of a problem by ensuring we are healthy, fit and prepared enough to dive, that we have suitable and serviceable equipment, that we are adequately trained and experienced to do the particular dive, and that we use our common sense, and do not push the limits of safety.

The above article has been slightly edited from the form in which it will appear in a future issue of "Sportdiving in Australia and the South Pacific" and is reprinted here by permission of the author and of the publisher.

We look forward to publishing a lawyer's view of these inquests.

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ASTHMA AND DIVING.
Some Observations and Thoughts*

Carl Edmonds

People keep asking me for reprints of papers that I wrote on asthma years ago, in such literary papers as the SPUMS Journal, Pressure Newsletter, Undercurrent, etc. I do not keep copies of the past. In the hope of deflecting further requests, I present here selected excerpts from the 3rd edition of Diving and Subaquatic Medicine, which is in press and which will be distributed by the publisher, Butterworths, possibly in 1991-2. Requests for complimentary copies should be sent to them, not to me.

Asthma and diving deaths.

Only 1 % of American divers have a history of asthma, as judged from the 1988 DAN diver survey.[1] The figure is probably less in Australia, where medical questionnaires and examinations are required before diving. However at least 9 % of the deaths in Australian and New Zealand recreational divers[2] were in asthmatics and in at least 8% it was a major contributing factor.

* Edited excerpts from *Diving and Subaquatic Medicine,* 3rd Edition Butterworths (in press)

Most of these deaths were in clinically mild asthmatics who are otherwise physically fit young men.

The possible trigger factors for asthma provocation in scuba diving are;

1 Exertion (from overweighting, equipment drag, swimming against tides etc.),

2 Inhalation of cold, dry air (adiabatic expansion of dehumidified compressed air),

3 Hypertonic saline inhalation (bubbling or leaking regulators),

4 Breathing against a resistance (increased gas density, regulator problems, low air supply).

Many of these stresses are used clinically to initiate asthma as diagnostic provocation tests, and so the problems with this disorder are understandable. In a number of cases the diver was returning to obtain a salbutamol (Ventolin) spray; in others it had been used immediately before the dive.

Asthmatics, even more than others, had multiple contributions to death. The relative frequency of a compromised air supply, salt water aspiration, panic and fatigue, prior to drowning, was evident from the statistics

Asthma and diving accidents

Most experienced diving medical physicians are appalled at the though to asthmatics diving. This attitude stems from a number of places. They include:

1 Involvement with asthmatics who died;

2 The catastrophic histories from those who survived;

3 Differential diagnostic difficulties with asthmatics who have near-drowning and possibly also pulmonary barotrauma;

4 Therapeutic complexities in these cases, both regarding depth and oxygen exposures, and drug complications;

5 Training from their teachers, whose experience often

TABLE 1

ASTHMA DEATHS FROM 100 RECREATIONAL DIVING FATALITIES IN AUSTRALIA AND NEW ZEALAND

Autopsy cause of death	
drowning	7
pulmonary barotrauma	2
Other medical contributions	
salt water aspiration	5
fatigue and/or panic	5
Technique problems	
Compromised air supply	6

is based on earlier times, when there was no diver medical selection.

Most of us have seen too many asthmatics who, because of a commendable desire to achieve normality, are affronted by advice that they should not scuba dive. They tend to be very fit, somewhat macho, males with a type A personality. They, correctly, claim that their asthma is mild, easily correctable, and not a restriction in other sports. In fact, any clinically severe asthmatic is most unlikely to ever intend to dive.

Often they extrapolate their techniques of pre-exercise medication to diving, without realising that this will increase the likelihood of the sudden death syndrome, which had now become one of the commonest causes of recreational diving deaths. In many cases the diver was returning to obtain a salbutamol (Ventolin) spray, when the accident or death occurred, in others it had been used immediately before the dive. Some believed that the spray overcomes all airway restriction, not realising that the pharmacological effect is patchy and competes with the provoking factors.

The asthma provocation factors in scuba diving are mentioned above. Especially serious incidents tend to occur when more than one factor is present. These provoking factors, which lead to dyspnoea, fatigue, difficulties with the regulator or gas supply, panic emergency ascents, salt water aspiration, near-drowning or drowning, pulmonary barotrauma, etc, are rarely seen with free diving or recreational swimming, except in extreme circumstances.

Case Reports

These were selected from our own files, except where otherwise stated, to illustrate the variety of presentations. They are not from the ANZ death statistics referred to above.

Case 1

A, aged 25, was a very fit, mildly asthmatic, sportsman. He had been diving for four months when he went to 18 m for 20 minutes. Without an obvious reason, he performed a rapid ascent, developing dyspnoea and confusion on the surface and left sided paralysis within a few minutes. He was taken by helicopter to the Royal Australian Navy recompression chamber (RANRCC). He was initially compressed to 18 metres on oxygen, but as he did not regain consciousness he was then taken to 50 m.

After a three-day vigil, in which the patient was subjected to various procedures in an attempt to surface him, he died, still under pressure.

During that time he was treated conscientiously for his asthma, which was evident on auscultation, and for CAGE. He was given steroids and anticonvulsants (for his repeated epileptic episodes), measures to counter possible cerebral and pulmonary oedema, and to maintain his electrolyte and pH levels.

The autopsy revealed mild cerebral oedema, congestion of the meningeal vessels and ischaemic cell damage in the right frontal lobe and the the right thalamus. There was a tear on the posterior section of the upper lobe of the right lung, with intra-alveolar haemorrhages and rupture of alveolar septae. The lung basal membranes were thickened and muscles showed hypertrophy, consistent with asthma.

Provisional diagnosis:

Asthma, pulmonary barotrauma, CAGE.

From the Royal Australian Navy School of Underwater Medicine

Case 2

B, aged 33, had been a qualified diver for four years, despite being a known, but very mild, asthmatic. He was classified as fit by a doctor who alleged experience in diving medicine. The doctor also gave a script for salbutamol, and advised him to take it prior to diving. He followed this advice. He even had a pocket included in his wet suit to hold the inhaler.

He descended to 9 m for 20 minutes, then did an ascent to get his bearings. On returning to his companion, he appeared distressed and then made a further rapid ascent to the surface. There he appeared to be confused and removed the regulator from his mouth. He inhaled some sea water and then lost consciousness and went into a convulsion.

He was rescued by his companion, and within 30 minutes reached the RANRCC, by helicopter. He was comatose with brain stem spasms and with a very inadequate air entry, bilaterally. He was compressed to 18 m on oxygen.

Despite endotracheal intubation and 100% oxygen, at 18 metres, with positive pressure respiration, the PaO_2 level remained at 50-70 Torr. The $PaCO_2$ levels were usually above 100 Torr and the pH remained below 7.0.

Mainly because of the death of an almost identical asthmatic diver, just previously, after a descent to a much greater depth, it was decided to surface this patient over a period of approximately five hours, while attempting to maintain as high an oxygen partial pressure as possible.

The problem was in the combination of diagnoses, including cerebral gas embolism (the initial incident), asthma

(as detected by the significant bronchospasm) and drowning (caused during the attempted surfacing and rescue of the patient).

Going deeper, to overcome the effects of the air embolism, would be complicated by prejudicing his PaO_2 level. The greater depth and increased density of the gases would probably interfere with adequate ventilation, CO_2 exchange and cause acidosis.

Aminophylline could cause arterialisation of pulmonary air emboli. The coincidental hypothermia (33-35 °C) was not considered a definite problem, and might even be advantageous, if it was not for the effect sympathomimetics, required for the asthma, could have on cardiac arrhythmias. Steroids were given for the rather indefinite, but multiple reasons, as given above (asthma, cerebral damage, drowning, etc.).

Initially the chest X-ray verified gross pulmonary oedema, consistent with the combined effects of asthma and drowning. Subsequent chest X-rays revealed a persistent right lower lobe opacity, clearing up over the next month.

With attention to the respiratory status, the brain damage, fluid and electrolyte status, the patient gradually improved over the next few weeks and he regained consciousness. The result was a severely brain-damaged young man continually incapacitated by myoclonic spasms, which were almost certainly post-hypoxic but possibly contributed to by CAGE. There was a residual dysarthria, a left hemiparaesis, an ataxic gait and myoclonic jerks. The EEG was consistent with hypoxia and the CT scan was normal.

Provisional Diagnosis.

Asthma, CAGE, near-drowning.

Case 3

C, aged 43, was a very experienced diver who previously had asthma as a child, and who still had high pitched rhonchi on auscultation during hyperventilation.

A very eminent respiratory physician informed him that his lungs had quite adequate function for scuba diving. This advice was refuted by members of the Diving Medical Centre but academic brilliance won out.

Whilst exploring a wreck, at a depth of 27 to 18 m, he suddenly became aware, as he floated up over the deck, of a pain in the left side of his chest. He then attempted to ascend. The pain became worse as he ascended. He slowed down and the pain decreased. He took over half an hour to reach the surface. During this time there was a continual pain in the chest, aggravated if he tried to ascend rapidly.

With extreme courage, and commendable control over his breathing gas consumption, despite the terrifying circumstances, he did reach the surface, although in great discomfort. He was then given oxygen and transferred to hospital. The clinical and X-ray evidence verified the presumptive diagnosis of left pneumothorax, and a thoracentesis was performed.

He returned to the respiratory physician, to be reassured that it was unlikely to happen again. The Diving Medical Centre physicians, assured him that not only would it happen again but that, with the lung damage and the treatment received, it was more likely to happen again and that it should not have been allowed to happen in the first place. He decided, this time, to take our advice.

Provisional Diagnosis.

Asthma, pneumothorax with minimal provocation.

Case 4

D, aged 20, had been certified fit to dive despite an asthma history. Prior to the dive there were no symptoms, but he still took a salbutamol inhalation.

In his first deep water dive, after spending 8 minutes at 30 m, he took 23 minutes to reach 15 m. A burning pain in his chest then caused him to make a rapid ascent. He was pulled out semi-conscious and apnoeic. He had four grand mal seizures and was given oxygen on examination. There were no neurological defects other than disorientation, After 6 hours, during which time he had another three seizures, he was recompressed to 18 m on oxygen and treated with anticonvulsants. There was no evidence of a pneumothorax, and he was eventually treated on an air table at 50 m having continued to convulse while on O_2 at 18 m. He survived, but has subsequently stopped scuba diving.

Provisional Diagnosis:

Asthma, pulmonary barotrauma, CAGE,

Summarized from SPUMS Journal, reported by of Dr. David Clinton-Baker.

Case 5

E, age 23, was a very fit and courageous athlete, who had mild asthma and was advised against scuba diving. Unfortunately his father, who was a professor of medicine, succumbed to family pressure and signed a “fit for diving” certificate.

This patient suffered two episodes of a very similar nature. In neither case had he had any evidence of active asthma prior to the dive, and in the second episode he had actually taken a salbutamol spray before the dive. These dives were in a similar area to depths less than 10 m. After 20 to 30 minutes he had developed dyspnoea and attempted to return to shore. On the first occasion he had informed his buddy that he was returning to shore to get another salbutamol

TABLE 2
ASTHMA ASSESSMENT PROTOCOL

History of asthma over the last five years.	FAIL
Use of bronchodilators over the last five years.	FAIL
High pitched respiratory rhonchi or other respiratory abnormalities.	FAIL
High pitched expiratory rhonchi, on hyperventilation.	FAIL
High pitched expiratory rhonchi on hyperventilation ten minutes after a 5min @ 900 KPM/min exercise stress.	FAIL
FEV1/VC of <75 % of predicted value.	FAIL
Expiratory flow rates of < 60% of predicted value. (Basic spirometry; FEF 25,50, 75, MMEF etc.)	FAIL
Asthma provocation producing >10% reduction of expiratory flow rates FEV 1 or PEFR) after both conventional histamine and hypertonic saline provocation, preferably while breathing dehumidified cold air.	FAIL

If all the above are negative or clear, limited diving may be permitted to a maximum depth of 18 metres without any free ascent practice.

spray but he appeared to panic and inhaled sea water. He was then rescued in a comatose state and eventually recovered after helicopter transfer to a major hospital.

The second episode was of a very similar nature, except that he did not recover. The autopsy revealed evidence of drowning, with mild asthma.

Provisional Diagnosis:
Asthma, panic, near drowning and drowning.

Case 6

F, age 20, was a fit young diver who carried out 30 scuba dives to a maximum of 39 m, without incident, before being certified as fit for diving by an experienced diving physician. There was a past history of asthma for which he had used steroid inhalers. On examination there was no evidence of bronchospasm and the FEV1/VC was $3.9/4.5_1$, without bronchodilators. The chest X-ray was normal. He was advised that he would be medically fit to dive providing he was free of asthma and that he taken an inhalation of Berotec prior to each dive.

While undertaking in-water rescue and resuscitation exercises, to a maximum of 4.5 m he developed dyspnoea on the surface. He informed the instructor that he was suffering from asthma and was towed 30 m back to shore. By then he was cyanosed with wheezing on inspiration and expiration. He then lost consciousness and required expired air resuscitation (by two experienced internists). He suffered a grand mal seizure and then gradually improved following oxygen inhalation. He responded to treatment of his asthma, over the next few days, using aminophylline.

There was no evidence of CAGE, and the seizure was considered to be due to cerebral hypoxia. Later a history of an asthmatic episode four days previously was elicited. It was presumed that the asthma was triggered by the aspiration of sea water, exertion and cold exposure.

Provisional Diagnosis:
Asthma, near drowning

Case 7

"I have extremely mild asthma, which manifests perhaps once every three years for a brief time during a respiratory tract infection. As I did not encounter any asthmatic symptoms during strenuous high altitude mountaineering I thought it would be reasonable to try scuba diving. I learnt to dive in a warm shallow swimming pool and experienced no difficulties during this or my first sea dive. During my first deep sea dive, however, I had an extremely severe and sudden attack of bronchospasm at a depth of 30 m. I barely made it to the surface, where my obvious distress and lack of speech caused my partner to inflate my life jacket, thus compromising my respiration further. It was a frightening experience and I have not dived since."

J .J .Martindale. Summarised from a letter in reply, in the BMJ.

Provisional Diagnosis
Asthma, panic.

Protocol for assessing potential asthmatics

The protocol in Table 2 has been of value to us for the assessment of diving candidates with a history of asthma and a normal chest X-ray. If all the findings are negative or

clear, limited diving may be permitted to a maximum depth of 18 metres without any free ascent practice.

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A NEW CLASSIFICATION FOR THE DECOMPRESSION ILLNESSES.
Report on a workshop held at the Institute of Naval Medicine, Alverstoke, United Kingdom, October 1990

Des Gorman

Recently the Royal Navy Institute of Naval Medicine (INM) sponsored a 2 day workshop at the Institute to develop a new classification for the decompression illnesses. This was prompted by an attempt at organising a multi-centre trial of lignocaine in cerebral arterial gas embolism (CAGE), which foundered when diagnostic criteria for CAGE could not be agreed. Funding for the workshop was provided by INM and 35 delegates were invited and attended; including the author, Carl Edmonds, David Elliott, James Francis, Tom Shields, Ed Thalmann, Ed Flynn, Drew Dutka, Ramsay Pearson, Lindsay Symon, David Dennison, Richard Moon, Maurice Cross, Ian Calder, Hans Ornahagen and Yehuda Melamed. The proceedings will be published by the Undersea and Hyperbaric Medicine Society.

The existing classification.

Before the Workshop the decompression illnesses were conventionally divided into CAGE and decompression sickness (DCS). DCS was further divided in types I (mild) and II (serious) in a system proposed over 30 years ago for caisson work.[1] The workshop participants agreed that although sudden loss of consciousness in a scuba diving candidate on surfacing in a swimming pool was almost certainly CAGE and that left knee pain in a saturation diver developing 6 hours after reaching the surface was similarly certain to be DCS, between these two extremes differentiation was often impossible.

Furthermore it was agreed that:

a CAGE can present before reaching the surface;

b almost all cases of cerebral DCS have symptoms within 20 minutes of surfacing;

c many cases of cerebral DCS were likely to be due to arterialisation of venous bubbles and hence that DCS often initiated CAGE;

d arterial emboli could either precipitate DCS or occur concurrently with DCS (the so-called type III DCS [2]);

e most cases of CAGE did not have any evidence of lung damage;

f in submarine escapees de-novo formation of bubbles in arteries could not be completely excluded; and,

g while most cases of CAGE showed some spontaneous recovery many were static or progressive.

Many delegates reported that attempts at retrospective analysis of case histories had resulted in very low concordance between observers in the diagnosis of either