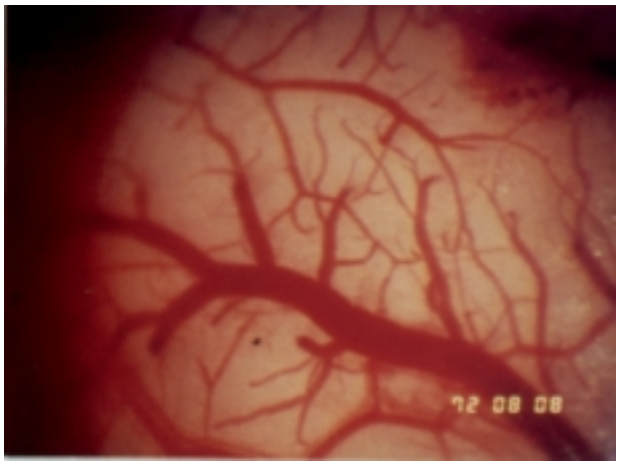

Figure 2A Photomicrograph (x500) of rabbit brain surface before arterial gas embolism.

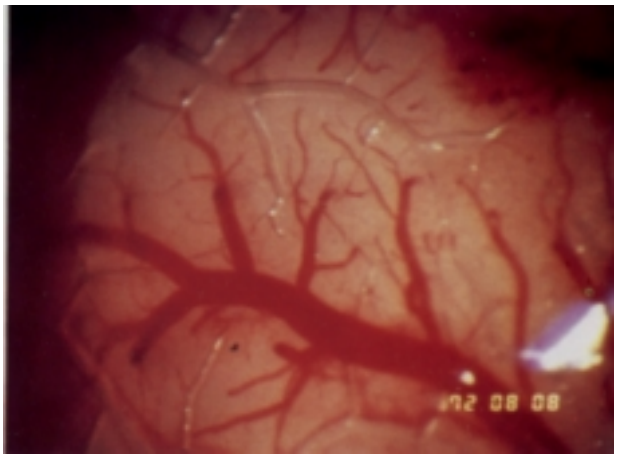

Figure 2B Photomicrograph (x500) of rabbit brain surface after arterial gas embolism.

SPUMS ANNUAL SCIENTIFIC MEETING 1987

SCOMBROID POISONING

John Knight

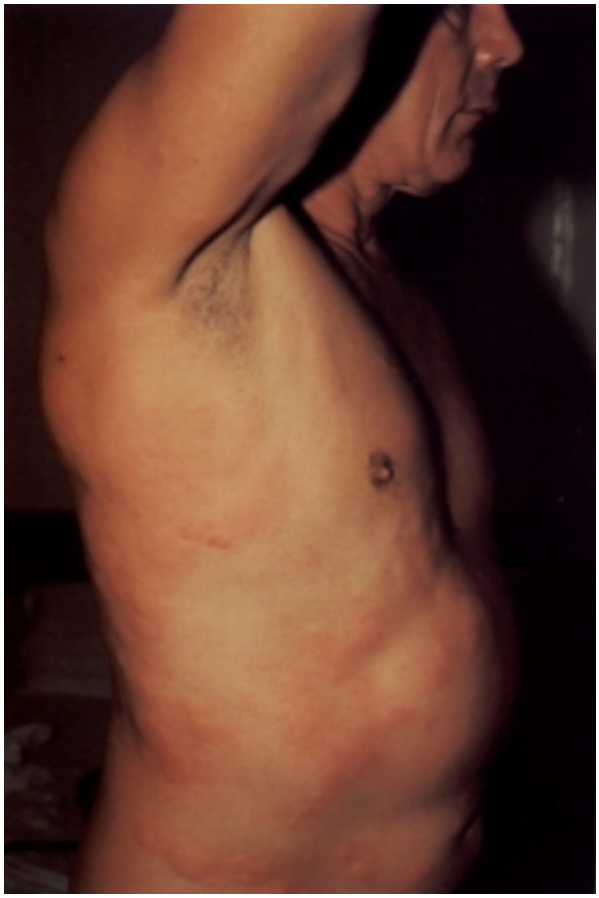

Figure 1. Side view of the author at 0100 on 25 April 1985.

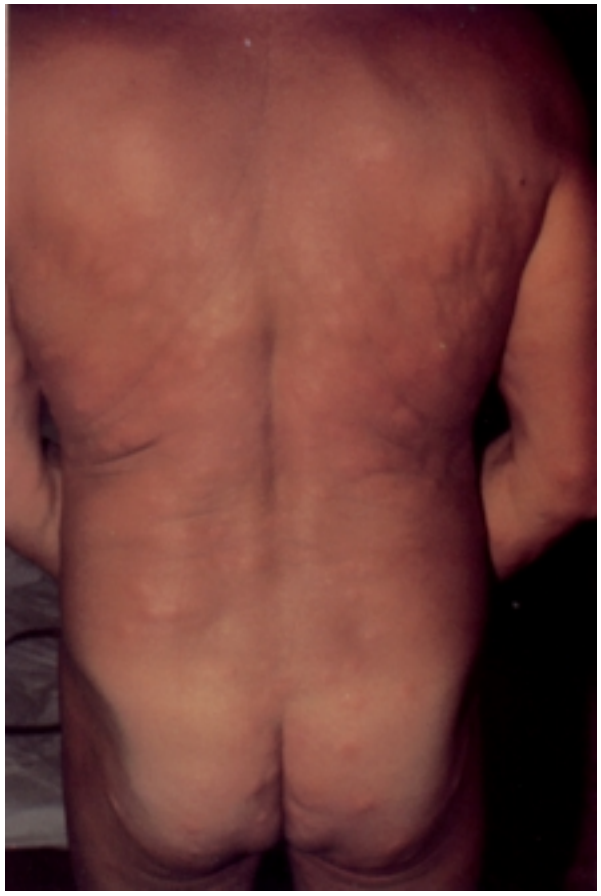

Figure 2. Back view of the author at 0100 on 27 April 1985.

The Scombroid fishes, which include mackerel and tuna, can cause a particularly dramatic and unpleasant form of poisoning. It is a series of histamine reactions. This paper is a personal recollection of what it does to the patient and the effects of treatment combined with a short review of what is known about it.

Case History

During the 1985 SPUMS meeting in the Maldives most members had their fill of the local fish and by the final night refused the barbecued fish. I had some about 2000 and was given a larger than usual helping. The fish tasted excellent. The rest of the evening was spent at the bar, leaving for my room about 2330. On the way I started to itch on my arms and body and thought that I had been bitten by mosquitoes. When I undressed I had a number of blotchy spots on my skin which itched. However it was not too difficult to get off to sleep.

About 0100 I woke itching and scratching. My face felt swollen and my mouth felt rather like the after effects of dental analgesia. However I could still smile but in repose I looked as if the end of the world was nigh. The rash had spread widely and obviously was not mosquito bites (Figures 1 and 2). It was then that the penny dropped. I had eaten some tuna and now had an obvious histamine rash. I had scombroid poisoning. I took a promethazine (Phenergan) tablet with two betamethasone (Celestone) tablets and managed to get back to sleep.

At 0500 when we had to get up I was covered in blotches. My face and body were both involved and I was itching madly. Carl Edmonds took some photographs of me at this stage. More promethazine and betamethasone made me feel a bit better but by the time we were waiting at the airport I was having difficulty standing. By the time we got to Singapore all I wanted to do was collapse into bed.

Next morning the "maps" were even more pronounced, promethazine was not controlling the itching and there were no more betamethasone tablets. At 1300 I still had a swollen face and many blotchy areas (Figure 3) so I sought local medical aid and was given more betamethasone tablets. I needed two every six hours to control the itching and stop the appearance of new blotches. After 24 hours on steroids my face was nearly normal and the blotches were beginning to disappear although there were plenty still visible (Figure 4). Over the next three days the dose was reduced to one betamethasone tablet every six hours and I had to stay on this dose for five days as symptoms recurred every time I reduced the dose by a tablet. Then I was able to reduce the dose by a tablet a day. When I had taken the last tablet, even though I had been on steroids for a fortnight, I still had some blotches.

Compared with ciguatera the course of the disease was short and, once adequate steroid treatment was started, painfree. The reversal of temperature sensation with ciguatera lasts for months and drinking alcohol can restart the symptoms. Mercifully neither occur with scombroid poisoning.

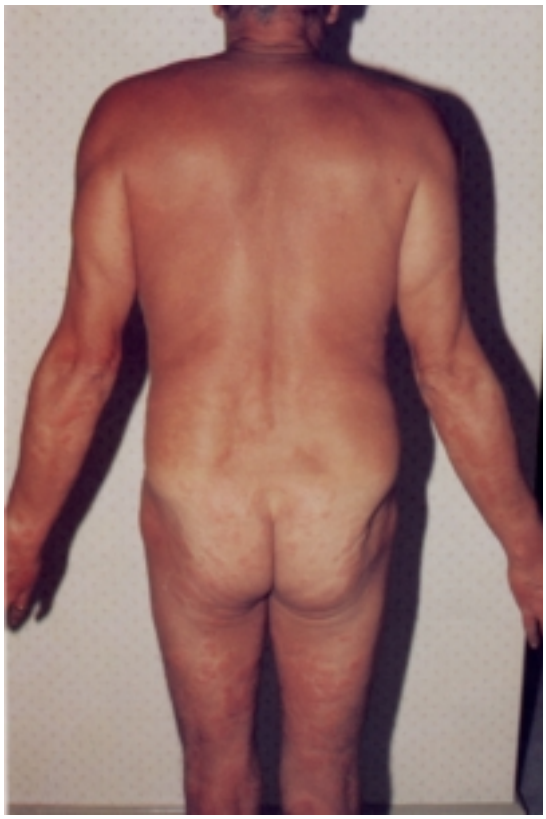

Figure 3. Back view of the author at 1300 on 26 April 1985.

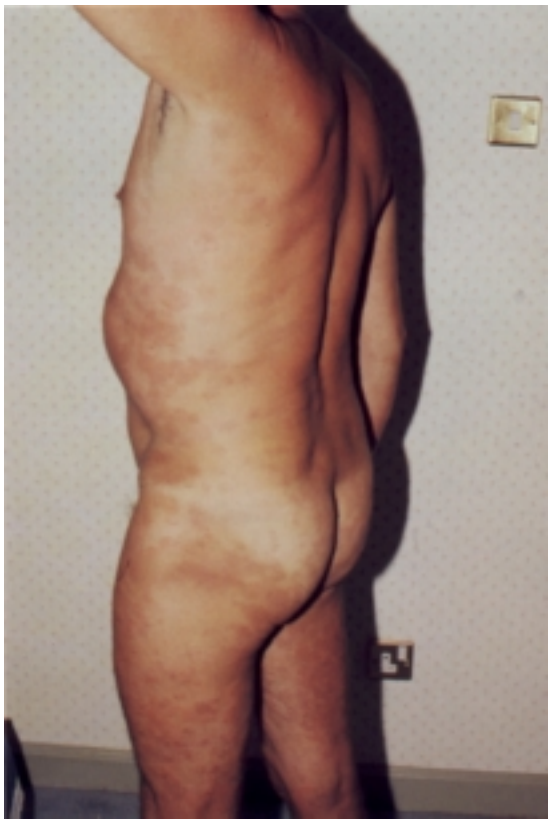

Figure 4. Side view of the author at 1400 on 27 April 1985.

Causation

Not much has been written recently about scombroid poisoning. I have been able to trace six papers or letters to the editor in journals since 1975[1-6] and there are a few words about it in "Australian Animal Toxins"[7] and "Diving and Subaquatic Medicine".[8] The most information I came across was published in 1961.[9] The relevant chapter was written by Kimata and dealt mostly with work done in Japan.

50 years ago Igarashi found large amounts of histamine in the muscle of the chub mackerel inspite of treatment with antibacterial solutions. He thought that the histamine was due to autolysis. Kimata was unable to reproduce these results. He considered that it was bacterial action which caused the histamine build up. The maximum histamine production, both in rate and amount occurred at 20°C in dark meat fishes, such as mackerel, horse mackerel, bonito and tuna, which were the only ones to produce histamine with bacterial spoilage. Oddly enough little histamine was produced at 37°C and less surprisingly none was produced when the fish were kept at 0°C. There was no evidence of a link between the freshness of the fish and histamine production.

The amino acid histidine appears to be necessary for the formation of histamine which is not only found in the muscle of fish. Kimata stated that histidine production was least around 17°C. It was three times as much at both 6°C and at 35°C.

It appears that the fish become poisonous when they are left at room temperature or exposed to the sun, for several hours. Kimata's work suggests that bacterial action turns the histidine in their muscles into histamine.

However it seems to me that other amines must also be formed as the effects of histamine are short lived. Anyone who has been stung by stinging nettles knows that symptoms seldom last for more than an hour or two. So histamine is probably not the only poison produced in the fish. Kawabata gave the name saurine to substances having the same action as histamine which he though were present with the histamine. Besides saurine a scombrotoxin, which also has histamine like effects, has been postulated. From the fact that my symptoms lasted for many days, I am of the opinion that there was either a histamine provoking poison present in my body or a long lasting, slowly broken down, congener of histamine producing the same effects.

It is obvious that the site of the histidine degradation and the size of the dose of poison influences the onset of the symptoms as a number of people ate the fish that night but only one had symptoms.

Confirmation of the diagnosis

If any of the fish is left when the patient develops symptoms the diagnosis can be confirmed by laboratory testing showing a high histamine content in the muscle. The critical histamine level to produce symptoms is 100 mg/ 100 g of fish muscle.

Histamine producing bacteria

Proteus morganii has been shown to produce both histamine and saurine when innoculated into raw fresh tuna flesh. This organism is thought to be the main cause of scombroid poisoning. Other histamine producing bacteria include *Salmonellae, Shigella dysenteriae, Clostridum perfringens, Escherichia coli* and *Aerobacter aerogenes*. The identifications date from between 1910 and 1939 and come from Europe as well as Japan.

Histamine producing bacteria were found on the surface of all freshly caught fish in the 1950s in Japan. The bacteria were presumed to come from the fishing gear, nets and boxes. This was in the days before nylon nets became common.

Conclusions

Anyone who has eaten fish of the tuna and mackerel family and then develops symptoms of histamine release has probably developed scombroid poisoning. Treatment with antihistamines is helpful in reducing the itch but probably will not influence the progress of the illness very much. Steroids in large doses are needed to control the symptoms and the rash.

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DECOMPRESSION METERS PHILOSOPHICAL AND OTHER OBJECTIONS

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The use of decompression meters (DCMs) is not new, and has involved a wide range of apparatus, from mechanical to electronic, and both diver-worn and remote. The Canadian Defence and Civil Institute of Environmental Medicine surface-based decompression computer represents one extreme of this development and has proved useful. However, the active marketing of a new range (not "new-generation" as is claimed) of diver-worn DCMs requires that the case against such devices be stated again.

Multi-level Diving

A major advantage claimed for DCMs is that they account for the multi-level nature of most recreational diving. Consequently, a DCM will "permit" a longer exposure to pressure, for a given multi-level dive, than that allowed by the traditional use of the same decompression schedule (which assumes that the entire exposure was at the maximum depth).

The number of cases of Decompression Sickness (DCS) presenting for treatment in Australia and New Zealand has increased since 1980 and has shown an alarming predominance of nervous symptom involvement. These episodes of neurological DCS often arise after dives that either were conducted in accordance with conventional tables (with and without fudging), or were within no-decompression limits (despite being multi-level).

Based on current treatment rates it is anticipated that in 1987 between 300 and 400 divers will be treated for DCS in Australasia. While this does not establish that the disease rate (eg. DCS/1000 diving hours) has increased, it is clear that the diving practice of the recreational diving community needs to become more conservative. This recommendation for safer diving is not consistent with the increased exposure possible with DCM-controlled multi-level diving.

Measurement of Exposure

While the marketing information released with each new batch of DCMs declares the arrival of a "new generation" of devices, this is simply not true. All devices that have been sold, and are about to be sold, measure depth and time, and not tissue nitrogen tensions. What does change with each new model is how the information is manipulated and presented. The expected body-tissue nitrogen tensions are calculated from this input, using one or more mathematical models. In general, these models are perfusion-based and do not account for the diffusion limits of intracellular fluid. Whatever the basis of calculation, it is important to understand that the kinetics of inert gas uptake and elimination have not been accurately described. Not surprisingly then, the accuracy of calculated tissue nitrogen tensions using these available mathematical models of decompression is quite poor.

This intrinsic inaccuracy of decompression models, and hence of DCMs, will remain until a DCM can directly measure an individual's tissue nitrogen tension (eg. using transcutaneous or implanted electrodes). Such a DCM would only then be a "new generation" device.

Electronic Reliability

An absolutely reliable electronic instrument has not and never will be built. Trials with all available DCMs have shown a real, although often small, failure rate (including total display loss). Obviously electronic diver-worn DCMs can never be used in isolation. Divers using DCMs should always carry and use a hard copy of suitable decompression tables.

Summary

Although DCMs are simple to use and account for multi-level diving, it is not possible to support or advocate total reliance on them. They may have a useful role in diving, but only in conjunction with a careful dive plan and concurrent use of a hard copy of decompression tables.

ASSESSMENT OF THE ORCA EDGE DIVE COMPUTER

Carl Edmonds and Tim Anderson

INTRODUCTION

The Royal Australian Navy School of Underwater Medicine first became interested in decompression meters used by divers during 1972. Many patients sought treatment for decompression sickness, following the use of the SOS decompression meter. A study of this meter showed that it indicated shorter decompression times than required by the US Navy decompression tables when used for repetitive dives, and for dives in excess of 60ft.[1] The Farrallon Multi-Tissue Decomputer was also studied[2] but was unacceptable because of its unreliability. The DECO-BRAIN suffered a similar fate when tested, approximately two years ago.

The senior author was involved in the treatment of a diver in 1986 who used an Orca EDGE for two dives to 87ft, after which she developed decompression sickness. It appeared that the meter had allowed a dive combination that would not be permitted by the US Navy tables. There were several possible explanations of this decompression incident: a chance occurrence because of the fallibility of the decompression tables, a misreading of the meter, a fault within the meter itself, or the meter programme permitted unsafe diving profiles.

It was against this background that it was decided to test the EDGE decompression meter's no-decompression repetitive dives and compare these with the established decompression tables.