Reid HA. Antivenom in Sea Snake poisoning. Lancet. 15 March 1975: 622-623.

Reid HA. Sea Snake Antivenom: Successful trial. Brit. Med. J. 1 Sept 1962: 576-579.

Reid HA. Diagnosis, prognosis and treatment of Sea Snake Bite. Lancet. 19 Aug 1961: 399-402.

Reid HA and Marsden AT. Pathology of Sea Snake poisoning. Brit. Med. J. 6 May 1961: 1290-1293.

Sitprija V, Sribhibhadh R and Benyajati C. Haemodialysis in poisoning by sea snake venom. Brit. Med. J. 24 July 1971: 218-219.

Sutherland S. Hydrophiilae. Sea snakes. Australian animal toxins. Oxford University Press. 1983: 159.

Tamiya N. Sea snake venoms and toxins. The Biology of Sea snakes ed. Dunson, 1975: 385-415.

Tamiya N and Maeda N. Neurotoxins of Australian Sea Snakes. Neurotoxins; Fundamentals and clinical advances. 1979: 95-101.

White J. Ophidian envenomation. A South Australian Perspective. Records of Adelaide Children's Hospital. 1980-81; 12(3): 311-421.

THE UNDERSEA MEDICAL SOCIETY MEETING JUNE 1983, ST JOVITE, CANADA

Jim Lloyd

The meeting was actually the eighth symposium on underwater physiology sponsored by the Undersea Medical Society (UMS). Although I am not a physiologist it happened that I was in Canada at the time.

The meeting was very interesting if a little bit high powered. They seemed to concentrate mostly on events occurring below 500 metres and that does not have much relevance to the Federal Police Diving School. The meeting was held at St Jovite in the Province of Quebec, La Belle Province I should call it as it is now a unilingual province. Luckily we were allowed to speak English at this particular conference. Some of the delegates from out of Canada, in fact most of them, were most surprised to find this venue was 80 km from Montreal and a $50.00 taxi fare from either of the airports. Most of us knew that Grey Rocks Inn was a well known ski resort. Being primarily a ski resort there was no air conditioning in the rooms so it was rather unfortunate that summer came early in Canada this year. The daytime temperatures were 35°C dropping to 22°C overnight. It was very hard to turn off the central heating in the rooms. However, there were plenty of distractions, golf, tennis, sailing, water skiing, horseback riding, you name it, they could provide it. There were the biggest mosquitoes not in captivity and an abundance of biting black flies.

The organisation went off very smoothly except for one little hiccup. No programme had been issued to anybody before they actually arrived on the spot and then we found that the scientific programme did not start until the second day. This was alright for those who were just there for a tax free holiday, but rather upsetting for those who had juggled very tight schedules to get there on time.

The meeting was entirely devoted to poster sessions. This was apparently a new departure for the UMS and they are not going to repeat it. The format of each session was a half hour review of the subject by an invited speaker, then an hour session in the poster room followed by a half hour discussion. Sixty four papers were presented in this way.

The only formal lectures were the introduction and the keynote session, and one formal lecture which was the Kronheim Memorial Lecture. Apart from this, the only relief from the posters was a film that Peter Bennett put on of the Atlantis Four Dive

The poster format was really rather annoying both to the delegates and to the presenters. The invited review at the beginning of each session was supposed to give an introduction to the posters themselves, so that everyone would be nicely primed by the time they got into the poster room. Unfortunately the quality of the reviews varied considerably from a mere paraphrase of the printed abstract, which themselves varied considerably in quality, to a half hour dissertation on the reviewer's own work, completely ignoring the other papers in the session. The poster room was not nearly big enough for the numbers present and the advantage went to tall delegates with hypermetropia. The shorter delegates like Dennis Walder and myself had problems.

The paper presenters also found themselves having to stand by their production and repeat the same speech over and over again to small groups of constantly changing people. There was a general agreement that they could have said all that they wanted to say much better in a ten minute lecturette, to the whole group. This would have got through the same amount of material in the same time. The discussion sessions were also pretty barren because those who really wanted to discuss a paper already had done so with the presenter in the poster room. The only thing that can be said in favour of this method of presentation is that it does tend to keep people awake.

There was an official welcome to Canada by a Canadian delegate, who read a message from the Governor General of Canada, who is a very keen diver and was unfortunately not able to attend, although he would have liked to be there. There were also a few words from the Lieutenant Governor of Quebec which were of course in French and an opening address by John Hallenbeck, the outgoing President of the

Society.

The keynote address was given by Lambertson and his title was "Adventures, Adventurers and Advances in Undersea Medicine". This was a witty review and history of the speciality.

The other formal lecture was the Kronheim Memorial Lecture, which came halfway through the proceedings, was a lot more interesting. This was by Professor Irwin Fridovich, Professor of Biochemistry at Duke University. He had the advantage of having an audience of physiologists rather than of biochemists. He was able to make himself very interesting, certainly as far as I was concerned, although I am not a physiologist and I am certainly not a biochemist. His subject was "The biology of oxygen radicals, their regularities and irregularities". This was quite fascinating. He took the view that oxygen is a toxic substance and that we live in spite of, and not because of, it. All aerobic organisms have had to evolve defensive mechanisms against oxygen in order to survive in the aerobic environment. The difficulty with oxygen arises not from the neutral oxygen molecule, but from the super-oxide molecule which is produced during metabolism. This, although not too unpleasant in itself, reacts with hydrogen to form reactive free radicals particularly peroxide and free hydroxyl. These are the toxic components. All the aerobic organisms which he had tested except for one, produced one or more superoxide dismutases. These are catalases which promote the decomposition of super oxides to neutral oxygen and water molecules which are quite safe. The one exception is a particular species of lactobacillus, but that does exactly the same thing without a catalase but by using a divalent inorganic manganese. His message was that every aerobic organism has some built-in defence against oxygen and that possibly the relevance to underwater medicine could be that by following this lead we could increase the resistance of organisms to oxygen toxicity and oxygen poisoning.

In an interesting aside, he mentioned that superoxide dismutase is obtained from separated microchondria very similar to those of most species of bacteria. This supports the evolutionary theory which says that mitochondria and multicellular organisms arose way back in evolutionary history by synergism between bacteria and the original unicellular organisms. He also mentioned that the dismutase enzyme is radioprotective and in fact is effective as a radioprotective agent even after irradiation. This is quite logical of course, because one of the mechanisms of radiation damage is the presence of free radicals.

His final speculation was that for prophylaxis against oxygen poisoning we should stimulate production of superoxide dismutases. There are possible ways of doing this. One example is that walnut trees sprayed with paraquat increase dismutase production by a factor of fifteen, which suggests that by investigating the other organic phosphates we may find an effective antidote against oxygen poisoning.

That again, I regard as logical, because there is also evidence that radiation damage is produced by organic phosphates, as shown by the combined effects of nerve gas and radiation damage, which some of you here will remember that I used to plug when I was lecturing in radiation biology at the RAN nuclear, biological and chemical warfare school.

I will just pick out a few papers for comment. None of the papers was provided in detail at the meeting and we will have to wait for the proceedings to be published to see the actual text.

Papers were grouped by topics. There was a paper on oxygen toxicity in closed circuit scuba divers, from the US Navy Experiment Diving Unit in Panama City. The military volunteers were subjected to a number of different profiles. The presence of oxygen toxicity was assessed by the incidence of convulsions or definite oxygen symptoms or probable oxygen symptoms. The conclusion was that 15 minutes at 40 feet is the maximum safe time before severe CNS toxicity is likely. However a diver exposed to 25 feet, for a longer period, can still make an excursion to 40 feets for almost as long as would be expected without the prior exposure. This information only applies to 100% oxygen closed circuit diving.

The other papers on this topic did not interest me much. Most reported essentially negative results.

The second session was on inert gas exchange, counter diffusion bubble formation. These were more interesting to someone who looks after divers. Macintosh et al of the Naval Submarine Medical Research Laboratory at Groton concluded that acclimatization to decompression stress may indeed exist but that definite evidence is lacking.

There was an interesting presentation by Lambertson, on the supersaturation isobaric inert gas counterdiffusion syndrome. He showed some horrible pictures of pigs which had been counter diffused at atmospheric pressure. This could be a useful experimental model by the generation of stable and readily produced gas embolism by counterdiffusion, which could save the need for experiments at pressure.

The Defence and Civil Institute of Environmental Medicine (DCIEM) in Toronto had a presentation on the conditions required for heterogenous nucleation in the physiological environment. This was a purely mathematical study, based on the crevice model. The conclusion was that there are three possible sites where nucleation bubbles could occur. These are the inner side of the microchondrial membrane, the ruffled border of osteoclasts and possibly the simple contact of cells such as red cell rouleaux in low shear flow. All these sites apparently having the correct contact angle, crevice angle and crevice diameter and if the surface tension is correct, they should be sites where nuclei could generate. Yount at the University of Hawaii has been creating bubbles by decompression of thin slices of agarose gelatin. He showed some nice pictures based on cross microscopy and trans-section election micrography.

Yount and Hoffman from the University of Hawaii had a presentation on decompression theory. They preferred the dynamic critical volume hypothesis of the available hypotheses. At one extreme is Haldane and at the other is Hills and the truth probably lies somewhere in between. As I believe in moderation in all things, I will go along with that.

A presentation from the Department of Pharmacology and Physical Chemistry in Oxford on the in vivo investigation of micronuclei in decompression sickness was fascinating. They had decompressed the common shrimp, which when suitably illuminated is translucent, and actually observed bubbles under the carapace. The main thrust of their experiment was to try and eliminate the nuclei by precompression to up to 400 atmospheres. They found that they did indeed eliminate the nuclei but that they all came back again after a three hour interval. They contrasted this rapid regeneration of bubbles with in vitro experiments where precompression to this extent has shown that liquids, with suitable precautions, remain free of nuclei for weeks afterwards. Obviously in vivo those precautions do not apply and so the bubbles come back. The pictures of bubbly shrimps were fascinating.

A presentation on "Scuba disease revisited" came from the Naval Medical Research Institute at Bethesda. They referred to Carl Edmonds' 1970 paper from the RAN on salt water aspiration. They compared that with experience in the USN in the '50's when there were a number of cases of "scuba disease" as they called it, including one death. They fixed the problem by regular cleaning and decontamination of scuba gear as they assumed it was due to pseudomonas infecting the equipment. Although there has been no case of scuba disease in the USN since then they concluded that "Scuba disease remains a potential health hazard nevertheless for recreational, military and commercial divers, especially those diving in warm and humid environments. Regular cleaning and decontamination of scuba regulators, hoses and mouth pieces is imperative. Medical personnel involved in the training and treatment of divers must be made aware of this disease." Carl Edmonds disagreed that salt water aspiration syndrome is the same problem as "Scuba disease".

An interesting presentation came from the Tokyo Medical and Dental University concerning decompression sickness in caisson workers digging a tunnel in Japan. They were monitored on the job by taking gelatin capsules with them under pressure and counting the number of bubbles formed. Each worker had six capsules in a little round pill box with a magnifying lens as the lid strapped onto his wrist. On coming up from each shift they would count the number of bubbles, divide by six and register the mean number. The conclusion was that in those showing a mean count of less than 10 bubbles (±7.5%) there was no decompression sickness while at 25 bubbles there was just over 3%. I have some free samples here if anyone wishes to check their bubbles after the dive tomorrow.

Still continuing on decompression sickness, a paper on "Fatal chokes in sheep" came from the University of Wisconsin. They had exposed sheep to pressure then given them a very short rest at the surface and then taken them up to 8,000 feet simulated altitude for fifteen minutes if they had not already got the chokes. If they did not have the chokes before they went up they had by the time they got to altitude. They were rather surprised that they had a high incidence o£ fatal chokes in profiles that differed very little from what people often do in practice. Their final paragraph reads "The occurrence of fatal chokes under conditions so little different from relatively benign exposures is alarming. The conditions are not very far removed from those encountered in flying after diving, diving at altitude or caisson and tunnel work at high altitudes. Except for one case reported there has been very little serious consideration of chokes as a potentially lethal complication of such pursuits. We suggest this should be taken more seriously in the future."

Another contribution from DCIEM in Toronto was "Bubble induced local hydrostatic pressure gradients, as a possible cause of dysbaric osteonecrosis". They came to the conclusion that local cell death and micro fracture of the bone matrix by the presence of bubbles within living bone tissue is a mechanism which appears to explain the aetiology of the disease. An important test of this mechanism would be to design decompression experiments on isolated osteocytes or isolated living bone, but this has not been done yet.

A paper on inner ear decompression sickness in the squirrel monkey, again from DCIEM, I found quite incomprehensible and I cannot even find any conclusions in the preprint.

A paper from the Naval Health Research Centre in San Diego was "Retrospective evaluation of recompression procedures within the US Navy.". This overview of past clinical records of cases concluded that the US Navy tables as they exist are quite satisfactory so long as they are correctly used. All the problems arose from people who modified the tables to suit their own ideas. What used to be the Royal Naval Physiological Laboratory and which is now The Admiralty Marine Technology Establishment (Physiological Laboratory) presented "The effect of presaturation on the maximum submarine escape depth of goats". What the practical application of that is, since one does not normally carry goats in submarines I am not quite sure. However, they also considered the implications of this to human research. The conclusion was that any research programme into human limitations of buoyant ascent from submarines following presaturation should commence at the maximum compartment pressure and the minimum escape depth, rather than the minimum compartment pressure and the maximum escape depth. They suggested that the amount of pre-pressure applied before making the escape is the critical thing for the survivability of a deep submarine escape.

A paper on ultrasonic bubble monitoring came from Oxford, the Department of Pharmacology and Physical Chemistry, who used a Doppler bubble counter applied to the knee and upper thigh.

There was another paper from Hawaii, from a Dr Lim, on experimental attempts to influence decompression thresholds in saturation dives in animals. This involved pre-exposures to high pressure for short periods to compress the nuclei. He came to the conclusion that over pressures, to the extent that were tested, did not reduce subsequent bubble formation.