Reports from the SPUMS SCIENTIFIC CONFERENCE, 1979

Sunday June 24th

Report by Dr Janene Mannerheim

Doctor Jefferson Davis commenced the SPUMS Conference for 1979 with an account of how a US Air Force Colonel became involved in Underwater medicine. Prior to 1959 the USAF had reported aver 1700 cases of altitude decompression sickness and of these 750 were serious (neurological). Jeff has researched these and reported them (Davis JC, Sheffield PJ, Schuknecht L, Heimbach RD, Dunn JM, Douglas G, and Anderson GK. Altitude Decompression Sickness: Hyperbaric Therapy Results in 145 Gases. Aviation, Space and Environmental Medicine August 1977: 722-730). He was treating aviators with decompression sickness and in Amsterdam became involved in hyperbaric oxygen therapy. Since then there has been an explosive increase in sports diving cases and he has treated over 100 sports divers with decompression sickness.

Decompression sickness can be seen as three separate diseases.

1. Gas separation (bubbling) requiring immediate treatment.
2. Blood/bubble interaction requiring complicated treatment.
3. Permanent tissue damage.

The terminology of decompression sickness needs clarifying. There are objections to classifying decompression sickness as Type I and Type II, eg. Limb bends is a tag which really means that at the same time as joint pain is occurring central bubbling can be picked up pre-cordially with a Doppler detector. Better terms could be found for aero-embolism and dysbarism as they are too confusing, meaning different things to different people.

Limb bends symptomatically can vary from mild to severe. Where are the bubbles? The gas probably separates in tendons and ligaments, causing a tedonitis. It has been shown that gas in the joint cavity is painless. Swelling of the hands can occur from lymphatic bubbles. Exercise aggravates the pain. Pressure eases it. A sphygmomanometer cuff inflated over the site of pain eases it, by squashing bubbles which have separated out.

Jeff presented slides of the School of Aerospace Medicine, Brooks Air Force Base, Texas. The hyperbaric chamber facility consisted of a large hypo/hyperbaric chamber used for treatment and research, and a small chamber. Both with depth capabilities of 225 feet. An unconscious patient can be slid in on a trolley.

Hyperbaric oxygen can be used to treat:

1. Decompression Sickness
2. Gas gangrene
3. Carbon Monoxide
4. Gas embolism
5. Refractory Osteomyelitis
6. Osteoradionecrosis
7. Soft tissue radionecrosis
8. Non-healing ischaemic soft tissue wounds
9. Slow bone healing after osteotomies
10. Burns

Decompression sickness which has been treated in his chamber includes:

1. Decompression sickness presenting with skin rash pruritis and mottling.

2. Presenting with fatigue, often a precursor (as are skin manifestations) of more serious symptom.

3. Chokes, presenting with dyspnoea, substernal pain, cyanosis and an irritable non-productive cough. McIver, Fife and Ikels have shown that its pathophysiology requires numerous central venous bubbles filtered in the lungs to form pulmonary gas emboli followed by a blood/bubble interation with a decrease in platelets and an increase in serotonin and in epinephrine (adrenaline for Australians).

4. Neurological decompression sickness. 15-25% of all cases (other than Pacific Island native divers) present as neurological decompression sickness. The clinical manifestations are confusing and there appears to be no pattern, which is understandable given the number of sites in the CNS where bubbles can lodge. Onset is gradual from within several minutes building up over approximately 6 hours. Never diagnose hysteria!

i. Spinal decompression sickness most commonly starts with low back pain - upper lumbar, lower thoracic level, radiating to the abdomen, then parasthesiae and weakness of the legs. It is followed by loss of the anal sphincter tone and bulbocavernosus reflex and finally urinary retention. What's that you say? The bulbocavernosus reflex. Put your finger in the patient's anus and with the other hand pull his penis. If the reflex is present the anal sphincter contracts and your finger is gripped. There was no information on how to elicit the reflex in females. Hallenback, Bove and Elliot's research on dogs supports the hypothesis that the following sequence of events occurs.

   a. Pulmonary Gas embolism
   b. Raised pulmonary arterial pressure
   c. Raised azygos vein pressure
   d. Obstruction of the epivertebral venous system causing stasis
   e. The stasis and bubbles lead to platelet aggregation and damage, thrombosis, congestion and haemorrhagic infacts on the white matter.

   Goats have been found to have congestive spinal infacts although they have only had symptoms of limb bends.

Case 39 years old, Male Oral Surgeon. One 72 cubic foot tank. Depth 110-160 feet. Ascended on reserve. He repeated the dive after a 30 minute surface interval. He developed abdominal pain, numbness and weakness of his legs. He recompressed himself on air at 20 feet. When he ascended he had only tingling of his right foot. Next day he dived to 140 feet, on ascent spent ten minutes at 70 feet and 15 minutes at 10 feet. He still had tingling of his right foot. Management: First do a neurological examination. Tremor of the hands found. He was compressed and decompressed on an extended USN Table 6, which gave complete recovery.

ii. Brain decompression sickness presents often with visual disturbances, blindness blurring of vision, etc. Also spotty motor and sensory loss, mental confusion, headache and convulsions. The mechanism possibly involves bubbles in the lipids.

Case A pilot pretended to have limb bends and was treated on a short oxygen table. He actually had visual defects but lied, as he was afraid that he would be barred from flying again. His visual defects persisted and he required treatment with an extended Table 6.

5. Labyrintine decompression sickness. This is a problem with saturation diving, though Jefferson Davis knows of two cases in sports divers. Clinically there is vertigo, tinnitus, nystagmus, nausea, vomiting, and hearing loss. The aetiology is little understood and there are several possible mechanisms hypothesising gas changes in the perilymph and the endolymph. Treatment requires prompt recompression on Table 6. A delay of 45-60 minutes gives poor results.

The differential diagnosis of the giddy diver

On Descent
1. Caloric
2. Barotrauma, ie. tympanic membrane or round window rupture

At Depth
1. Counter diffusion
2. HPNS

On Surfacing
1. Labyrintine decompression sickness which tends to occur up to one hour after the dive
2. Round window rupture which usually occurs during the dive.

The differential diagnosis of decompression sickness

Limb Bends	Injury
Chokes	Myocardial infarction
	Contaminated gas and oil mist.
	Pulmonary barotrauma
CNS	Head injury
	CVA
	Insulin dependent diabetic with hypoglycaemia
	Arterial gas embolism
Labyrintine	Round window rupture

Monday June 25th
Report by Dr Janene Mannerheim

Carl Edmonds' Underwater Oxygen Apparatus for treating decompression sickness in remote places was demonstrated in the pool.

Following this Jefferson Davis startled us with a case presentation of a diver who reached 240 feet on air, switched to oxygen for his decompression stop on ascent and then flew next day in a Boeing 747 pressurised to 5,000 ft. He later developed neurological decompression sickness. SPUMS members, known to dive and then fly, or dive and then climb active volcanoes, at least have not yet been caught at 240 feet on Scuba.

Despite treatment, 25% of those presenting with neurological decompression sickness are left with residual symptoms or signs. From a recent workshop in the US (to be published in September 1979) it was considered that immediate care, and care during transport are probably most important. It is important to choose the correct mode of transport, the type of chamber and then the appropriate treatment table. The sooner treatment with recompression is started the better. The longest delay involves getting a plane that can be pressurised to sea level (these include the Lear Jet, the Hercules C 130, Boeing 737 and Boeing 727) or a helicopter (if there are no mountains to be crossed).

Initially,

A. Give 100% oxygen by mask to wash out nitrogen at atmospheric pressure. Oxygen can be given for 20 hours before pulmonary toxicity occurs.
B. IV fluids. Low molecular weight Dextran has been advised, but Charles Wells, of Houston, Texas has shown that any fluid can be used. The aim is to avoid haemoconcentration and platelet aggregation.
C. Urinary catheter, to measure fluid output or to treat retention in paralytic cases.
D. Push oral fluids if intravenous is not available.
E. Steroids are controversial. Use Decadron 20 mg IV. Its maximum effect is reached in 12 hours.

Heparin before a dive can help to prevent decompression sickness, but heparin afterwards is not advisable because of the possibility of:

1. CNS bends with haemorrhage into the white matter.
2. Burst lung and its complications.

A one man chamber compressed with oxygen (eg. Vickers) is not advisable as you cannot do further neurological examinations, cannot extend the tables deeper and cannot cope with an emergency in the chamber. If using such a chamber for 30 minutes at 60 feet and a two hour ascent to the surface 1% of patients will develop pulmonary oxygen toxicity. It is mainly suitable for the therapy of burns or osteomyelitis.

The goal of hyperbaric oxygen therapy in the treatment of decompression sickness is to reduce the bubble size by compression to allow resolution of the bubble, to avoid its growth during ascent and to increase the oxygenation of the anoxic or damaged tissues.

USN Table 5 should be used only for limb bends, pain only type. It is important to do a neurological examination first as pain may mask CNS symptoms and signs. After about 30 minutes of 100% oxygen at pressure the capillaries shut down and tissue PO_2 drops. This can be prevented by switching to air for five minutes after 20 minutes on oxygen. While breathing air the patient can talk, drink fluids if there is no IV running, and a further neurological examination can be done.

USN Table 6 should be used when there is any one of the following:

1. Delay in reaching the chamber.
2. Any neurological manifestations.
3. Symptoms still present after 10 minutes on 100% oxygen at 60 feet.
4. Chokes.
5. Vasomotor instability.

With the prolonged use of 100% oxygen at 1 - 2 ATS it is difficult to differentiate between pulmonary oxygen toxicity and the chokes. If a regime of 20 minutes oxygen and five minutes air is used any pulmonary embarrassment can be assumed NOT to be due to pulmonary oxygen toxicity.

USN Table 6a should be used for air embolism. We were shown slides illustrating:

A. 92 cases of sports divers with decompression

sickness successfully treated with USN Tables 5 and 6.

B. 32 cases of decompression sickness. 8 of which were treated on air at 165 feet. Of these six were cured and 2 left with impairments. The remaining 24 were treated at 60 fsw on oxygen. 22 cleared with one or two treatments and two were permanently impaired.

C. The importance of treating even after extreme delay in reaching the chamber. Bubbles have been found up to 14 days later.

In a study of 25 air embolism cases, 8 occurred during swimming ascent training from 30 feet. A discussion on the advantages of the left lateral, head down, position took place. A slide showed the return of the cerebral circulation in a cat which was placed in the left lateral, head down, position following massive, visible, induced air embolism.

Tuesday 26th June

Report by Dr Janene Mannerheim

Excellent presentations were given by Jefferson Davis and Ian Unsworth on Hyperbaric Oxygen Therapy. Jeff commenced with the statement that chambers were not put to full use treating only decompression sickness (not enough cases) and should be used more often for hyperbaric oxygen therapy. His lecture dealt in detail with gas gangrene. It was noticed that most of the lay audience quietly departed as slides of clostridial myonecrosis and cellulitis were shown.

Inside the large double lock chamber used by Jeff, fire proofing facilities were illustrated, eg. spun-gloss blankets, lighting outside, no electrical connections, no spark sources, compressed air rather than electrically powered motors. Patients can sit or lie comfortably with on oxygen mask on. More than 9 patients can be treated at the some time. Modified aviation masks are used as they are lighter and more comfortable than others. Patients who cannot wear a mask are treated with a transparent hood which encloses the head. Intensive care of the very sick patient who may need general anaesthesia can be accomplished. The medical attendants breathe air and are decompressed breathing oxygen.

Outside the chamber the console includes gauges monitoring in ATA, PSI, mm Hg, and FSW.

Gas gangrene is caused by Clostridium perfringiens (ex Welchii). It requires ischaemic tissue to multiply. Under pressure the solubility of oxygen in plasma increases as the oxygen partial pressure increases, but there is very little increase in the carrying capacity of haemoglobin.

Patients can present with combinations of pain, tachycardia, skin bronzing, blebs, crepitus, myonecrosis, haemolysis, oliguria, obtunded sensorium, shock and seizures.

CASE A 19 year old male developed gas gangrene in one leg injured in a motor bike accident. Treatment required debridement of the wound, massive doses of penicillin, broad spectrum antibiotics, hyperbaric oxygen, tetanus prophylaxis, and whole blood replacement. He survived with good repair and skin graft.

According to Brummelkamp (1960) 100% oxygen can be used for 90 minutes at 3 ATA for three times in 24 hours or a maximum seven times in three days. The oxygen partial pressure reaches 2193 mm Hg. C perf stops producing toxin at a PO_2 of 250 mm Hg, and is killed at 1400 mm Hg. Anti-toxin should not be used.

Hitchcock reported the following survival rates in 1973:

Antibiotics alone	50% survival
Hyperbaric oxygen alone	No survivors
Surgery alone	No survivors
Hyperbaric oxygen &surgery	55% survivors
Hyperbaric oxygen and antibiotics	70% survivors
Hyperbaric oxygen and surgery and antibiotics	95% survivors

In Dr Davis' series the aetiology of gas gangrene was:

Trauma	68%
Elective Surgery	15%
Sports injuries	11%
Drug addicts	6%

Other case histories showed examples of a central necrotic zone with surrounding advancing zone, debrided wounds, X-rays of gas in the tissues, fasciotomies, liquified muscle, the importance of saving tissue in the injured hand.

Of all cases treated at Brooks AFB, Texas only one patient has developed pulmonary oxygen toxicity. Two cases of limb bends have occurred in the medical attendants.

Dr Ian Unsworth complemented Jefferson Davis' presentation on the use of hyperbaric oxygen therapy, showing the chambers at Prince Henry Hospital, Sydney. These are a large multiman chamber with three compartments, one of which is fitted with operating theatre facilities (with a depth limit of 80 Feet) and a smaller chamber attached to the large one. The small chamber has a depth limit of 350 feet of seawater. The control panels for the two chambers are side by side. Each compartment of the large chamber complex can be used as a separate chamber. The chamber complex has been involved in experimental work on pigs, treatment of a Red Setter and 300 fsw dives on heliox as well as treating humans.

Ian agreed with Jeff's list of indications for using hyperbaric oxygen therapy (detailed on page 5) and went on to discuss carbon monoxide poisoning and air embolism. He stressed the importance of recognising air embolism in general medicine, as out of 39 cases only one had been caused by diving.

Carbon Monoxide Poisoning

CO is found in coal gas and in natural gas (15%). Furnaces, kilns, wood fires, paper fires, and engine exhausts provide exogenous sources of CO. The most significant clinical criteria are loss of consciousness and finding the victim in a car, garage or gas oven. The so-called cherry red lips are not seen. The carboxy-haemoglobin bond is 250 times stronger than the oxy-haemoglobin bond. Toxicity is enhanced by hypoxia.

The half-life of CO in air is 250 minutes.
The half-life of CO in 100% oxygen at 1 ATA is 49 minutes.
The half-life of CO in 100% oxygen at 2.5 ATA is 9 minutes.

Statistics of CO poisoning in Australia are available with breakdown into male, female, age groups, accident and suicide.

Gas Gangrene

Between 1971 and 1977 58 cases have been treated at Prince Henry Hospital Hyperbaric Unit with a