grafts are rejected in 30 days, indicating an immunological memory. Hagfish only produce one type of serum antibody which is through to be a basic evolutionary 'building block', and which should be useful in the understanding of the evolution of the immune response.

As can be seen a great deal of the research being done at present will take many more years before fruition and possibly much of it will amount to nothing, but undoubtedly many breakthroughs will be made in this virtually untapped field of potential in the years to come.

CLINICAL CASE REPORT

HISTORY

SUNDAY 15TH APRIL (Sydney time) 1973

Dive 1.30pm The patient (a 29 year old Nauruan Islander) underwent a dive to approx. 250 ft (bottom depth) for 20 min. Reason for the dive was treating another diver for 'pain only' bends. No previous dives on Sunday. Dive to ?200 ft for ?10 minutes preceding day (details of depths and times difficult to ascertain). Shortly after leaving bottom the patient ran out of gas and surfaced immediately (? using emergency supply).

10 minutes after surfacing he noticed dizziness and vertigo (self rotating in environment) and inability to walk straight. There was also some 'numbness' of both lower legs.

One hour late rthe patient noted weakness of the right leg. This progressed over the next hour to a total paralysis of the right leg and a marked weakness of the left leg. He was then admitted to Nauru General Hospital.

Five hours after the dive he was noted to have severe abdominal pain, vomiting, paralysis of right leg, paresis of left leg with a total sensory loss of mid thigh level right leg and knee level left leg. He was given 100% O_2 by mask during which time the vertigo apparently settled and the leg anaesthesia largely resolved.

Arrangements were then made to transfer him to HMAS Penguin by Air Nauru (Fokker Fellowship).

MONDAY 16TH APRIL

4.00am On arrival at the School of Underwater Medicine at HMAS Penguin, he was noted to be conscious but drowsy and weak. Pulse rate: 100/min. BP 130/100. HS normal, chest clear. Abdomen soft but tender, non-distended and no bowel sounds noted. Bi-lateral grade I aural barotrauma. In-dwelling bladder catheter in situ. IV infusion into (R) leg had become blocked.

Positive neurological findings were :-

Right beating nystagmus - exaggerated on right lateral gaze, abolished on left lateral gaze.
Mild left upper limb inco-ordination.
Equal power, tone and reflexes in both upper limbs, but weak.
Complete paralysis of right lower limb with marked weakness of all muscle groups of left lower limb (could just flex knee and ankle).
Lower limb tone was equal, perhaps reduced, but reflexes were increased on the right side of ankle clonus. Plantar responses were flexor.
Sensation. Light touch and pin prick sensation intact but equivocal results were obtained with proprioception and two-point discrimination.

A diagnosis of Spinal Decompression Sickness (involving lumbar segments L2. L3-L4 region) was made. An IV infusion was recommenced and he was given Lasix 20mg and Decadron 8mg in infusion.

5.45 am He was placed in the recompression chamber and was given Stemetil 25mg IMI for nausea. At this stage the nystagmus was no longer present. After 10 minutes at 30 ft on 100% O_2 no change was noted except that lower limb reflexes were absent. No further change after 10 minutes at 60 ft.

He was changed to 40% O_2 and compressed to 120ft for 10 minutes - no change. After 10 minutes at 165ft on 40% O_2 the reflexes were noted to be brisk but there were no other changes. He was decompressed to 60ft at 10 ft/min and then decompressed according to Table 6B (RAN) - the long O_2 table.

7.00 am A Rheomacrodex infusion was commenced (500ml over 5 hours). No improvement was noted until 1.30pm when slight contraction of the right quadriceps was noted.

2.00 pm The chamber reached the surface. On examination, the patient was able to elevate his right knee 4-5" off the bed. Power had increased in the left leg. Reflexes were brisk with slight right ankle clonus. Abdominal tenderness was less. 100% O_2 by mask was continued 1 hour on, 15 minutes off until 8.00pm.

TUESDAY 17TH APRIL

An attempt at spontaneous voiding overnight was unsuccessful and he had to be re-catheterised. There was no change in his neurological status. He was still on IV fluids and Decadron.

10.00 am He was given hyperbaric O_2 for 60 minutes at 60ft with an ascent of 3 min/ft. Following this session there was a marked increase in the right quadriceps contraction and some slight hamstring contraction noted.

2.00 pm A further episode of OHP was given. Following this a further improvement in power in te right quadriceps and hamstrings was noted and for the first time, weak plantar flexion at the ankle. Power in the left leg had also improved. More detailed sensory testing

than previously revealed slight dullness of sensation of left leg below mid thigh. There was marked dullness (almost anaesthesia) of sacral and perianal region. Joint position sense was normal. The catheter was removed but again had to be replaced that evening.

WEDNESDAY 18TH APRIL

In view of the encouraging response to OHP on the preceding day a further session of hyperbaric oxygen was given. Assessment after surfacing showed a minimal paresis in the left leg and on the right leg, strong quadriceps and hamstring contraction, moderate plantar flexion and weak extension of the ankle and moderate plantar flexion of the toes. Sensation appeared intact in all areas. He was still unable to void, had not opened bowels and had slight lower abdominal tenderness. The chest was clear and there was no evidence of pulmonary O_2 toxicity.

He was then transferred to the Royal North Shore Hospital for intensive rehabilitation in the Spinal Unit.

The discharge summary from that hospital noted that "over the next six weeks he made a gradual improvement in muscle power in the lower limbs. The catheter was able to be removed from his bladder, and on discharge he had a residual volume of less than 100ml. He wa able to micturate by tapping above the pubis. His walking improved and on discharge he was able to walk safely with two sticks and could manage steps."

COMMENT

Once again the benefit of hyperbaric oxygen as opposed to recompression per se was demonstrated.

There was no improvement at all once down to 165ft, but after several hours in the chamber at 60ft or less on 100% O_2 there was some slight improvement.

Further improvements were noted after each session of hyperbaric oxygen.

The mechanism is obviously not just reduction of bubbles at this stage. The most logical answer is that the OHP overcomes ischaemia of tissues not already infarcted, but whose function is impaired, however other processes may be involved.

Several studies have shown, in animals whose spinal cords have been artificially injured, an elevation of the cord pO_2 under hyperbaric conditions. Some workers also mention a reduction in spinal oedema, although the mechanism of such is not clear. It may just be secondary to improved tissue oxygenation. The vasoconstrictive effect of OHP is said to have a beneficial effect in reducing cerebral oedema, although this has not been proven in spinal oedema.

Two main points arise from this case and that reported in the previous Newsletter. One is that a beneficial effect of recompression/ hyperbaric oxygen is seen even up to 50 hours after the onset of symptoms so that in serious cases an attempt to reach a chamber should always be made.

Secondly, the improvement seen with repeated sessions of OHP gives support to the concept that this may be a useful therapeutic modality in the management of traumatic spinal injuries as well.

The School of Underwater Medicine, in association with Royal North Shore Spinal Unit will investigate this concept further in 1974 using sheep with mechanically injured spinal cords.

SPUMS MEETINGS

i. NSW BRANCH MEETINGS - 14 JULY 1973

a. Venue for 1974 Conference

The general feeling present was that the conference should be held in Summer because of the warmer water temperature for diving. A weekend in late November 1973 was suggested as being suitable for the 1973 Annual General Meeting and it was decided to ask the Queensland members if they would care to organise such an event. February 1974 was tentatively suggested as a suitable time for the Diving Medicine Conference.

b. A suggestion that SPUMS communicate with the Minister for Customs and Excise in order to attempt to have the levy on imported compressed air inflatable life jackets diminished or waivered. It was pointed out that the SDAA in NSW had failed in its moves in this direction, and it was felt by all, that an approach from a professional medical body such as SPUMS may receive more attention, as this item of safety is considered an essential part of diving equipment for all divers.

c. Discussions (preliminary) concerning the design of a motif for letterheads and the Newsletter. Bob Thomas and Jim Hazel both produced some interesting designs. Opinions were divided and it was decided to press for a decision, based on submitted designs from any source, at the AGM.

d. Bob Thomas informed the meeting of initial discussions with MR Fred Hayes, Managing Director of Fred Hayes (Printers) Pty Ltd. General agreement had been reached concerning the printing of 150-200 copies of an improved Newsletter for a cost of approx. $120.00 per edition. This offer was particularly generous as it was essentially a non-profit contact. The Newsletter at this stage was envisaged as consisting of approx. 16 pages of quarto (bookshelf size) format. The Editor also repeated his request for more contribution by members for the Newsletter.

e. A letter to the Secretary from the Diving Retailers was read. This concerned the involvement of SPUMS in safety aspects of diving and included divers medical examinations. Advice was requested as to SPUMS attempts at rectifying the shortage of suitable qualified diving doctors.