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REHABILITATION OF A PARALYSED DIVER

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Introduction

Since the 19th century, recompression has been utilised to treat decompression sickness (DCS). Since then tremendous work and research has been done to improve the efficacy of treatment. Decompression sickness has classically been divided into Type I and II, depending on site of involvement and presenting symptoms and signs. This clinical classification was presented as an attempt to differentiate serious from not so serious cases, and thus to facilitate therapy decisions and prognosis prediction.

The Diving and Hyperbaric Medical Centre of the Republic of Singapore Navy was established in 1971. From 1 April 1989, it has been reorganised and renamed the Naval Medicine and Research Centre (NMRC). In addition to providing therapeutic care to divers of the Singapore Armed Forces, its Diving Medicine Branch also caters to civilian divers of all categories.

In Singapore, sports diving has fast been gaining popularity and there is a growing population of sports divers. Another group of divers seen at NMRC are the commercial divers involved in offshore oil exploration around this region. However, it is the divers of the fishing vessels in the Indo-Pacific region that form the majority of decompression sickness cases seen at NMRC (Table 1).

NMRC has used a variety of recompression tables for the treatment of DCS. However, we have abandoned short oxygen tables and now only use long oxygen tables for the treatment of both type I and type II cases, as studies have shown that divers with so-called type I DCS can develop an overt neurological deficit over time.

Profile of a fisherman diver

In Singapore fisherman divers band together and contract themselves to owners of small fishing boats. The crew of each boat numbers from 6 to 12. Of these, 3 or 4 are fisherman divers. Fishing is usually done at the South China Sea or the Indian Ocean, depending on the monsoon season. These deep sea fishermen operate hundreds of miles from Singapore.

However, fisherman divers in Singapore are a dying breed. 10 years ago, there were about 3,000 of them. Now, only 300 are plying this trade still with the same primitive equipment. They usually come from a poor education background and receive no formal instruction in diving physiology nor in safety precautions.

TABLE 1

FISHERMAN DIVERS TREATED AT DHMC1972 TO 1988

	DS Type		
Year	**I**	**II**	**Total**
1972	0	3	3
1973	0	5	5
1974	0	4	4
1975	0	2	2
1976	0	3	3
1977	0	6	6
1978	0	5	5
1979	1	7	8
1980	0	6	6
1981	0	5	5
1982	0	3	3
1983	4	4	8
1984	1	6	7
1985	3	5	8
1986	0	0	0
1987	0	2	2
1988	2	1	3
Total	11	67	78

Today the situation is improving. Since the 70s, our Diving Medical Centre has been actively educating these divers in safety measures and avoidance of DCS. Now they know the various diving tables, ascend with decompression stops and the number of local DCS cases seen is dwindling. We are now seeing more fisherman divers with DCS from neighbouring countries such as Indonesia and Malaysia. During the period 1984 - 1988, one third of fishermen DCS cases seen were from Malaysia or Indonesia, compared with the period 1972 - 1983, when only 15% were from these 2 countries. (Table 2).

Diving characteristics

The job of a fisherman diver is typically as follows. He will first do a breathhold dive to survey the area for fishes. It is said that the "Taikong" (the chief of divers), has the sixth sense of "hearing" where the fish will be before the crew make their dives. Once a suitable site is found, he will descent with the aid of weight belts and surface supplied

TABLE 2

NATIONALITY OF THE FISHERMAN DCS CASES 1972 TO 1988

	Number	Percentage
Singaporean	62	79.5
Malaysian	9	11.5
Indonesian	7	9.0
Total	78	100.0

TABLE 3

THE MAJOR PRESENTING SYMPTOMS OF FISHERMAN DIVERS WITH DCS 1972 TO 1988

Presentation	Number	Percentage
Joint Pain	35	44.9
Weakness	52	66.7
Sensory Deficit	40	51.3
Bladder Involvement	21	26.9
Bowel Movement	19	24.4

breathing apparatus (SSBA) to plant the explosives. The divers may dive from 3 - 6 times a day, and each dive may take up to several hours. The depth of diving is down to 45 m (150 feet), but averages 24-30 m (80-100 feet). Diving is done using crude surface supply apparatus. Air is usually supplied by a compressor pump directly to the diver.

Not infrequently, the dives are interrupted due to compressor failure. As a result, the divers are required to ascent rapidly. Another occasion that they need to do this is when they are attacked by sharks or pirates attack the boat. Even in routine dives, no decompression stops are made as these divers are ignorant of diving physiology and safety precautions.

Characteristic presentation of a fisherman DCS case

Because of the high nitrogen load (due to repetitive dives) and rapid ascent rate (due to ignorance of decompression stops), the onset of symptoms usually occurs within 15 minutes of surfacing. They usually suffer from severe type II bends, frequently associated with loss of consciousness and paraplegia with loss of bladder and bowel functions (Table 3). Of the 78 fisherman divers with DCS seen at our centre, 18 (23%) had loss of consciousness at the site of the incident.

As most fishing boats operate hundreds of miles from Singapore, it will take them several days to reach our treatment centre. During this period, the fisherman divers usually attempt wet recompression during their journey back. However, no proper schedule is followed and frequently after alleviation of the initial symptoms, the patient will be pulled up so rapidly that there is a recurrence of symptoms and the diver becomes even worse off than his initial condition.

Associated with the paraplegia or paraparesis, there are various degree of sensory loss, bladder and bowel dysfunction, dehydration and bed sores due to poor nursing care. Because of the neurologic bladder with urinary retention, catheterisation is sometimes attempted by the fisherman, using non sterile techniques, including the use of wires or even the outer coating of a section of electrical wire. As a result, there is often urinary tract infection. Recently these fisherman divers have started to use sterile disposable catheters.

Effects of treatment

Despite the delay, which may be more than 10 days, recompression using oxygen tables is still the mainstay of treatment, together with correction of dehydration and treating any complications such as urinary tract infection, bed sores, etc. and then rehabilitation.

Unlike acute cases, where the injury is due mainly to the blockage of blood flow by nitrogen bubbles, in these delayed cases, the hypoxic state of the spinal cord is due mainly to complications caused by the bubbles. These include blockage of vessels by fibrin and platelet clots with damage to the walls of capillaries and small vessels leading to abnormal permeability. This allows leakage of plasma into the interstitial space and causes oedema, resulting in haemoconcentration and dehydration. All these will contribute to the hypoxic state. Thus in addition to recompression oxygen tables, rehydration therapy using crystalline fluids and plasma expanders, steroids and aspirin are commonly used to treat our delayed cases.

Table 4 shows the time lapse before recompression therapy was started. There were 4 cases which presented more than 15 days after the insult. These case histories appear below.

Case 1

Mr OKC, 47 year old Malaysian, Chinese, Male.
Date of incident: 16 October 1978
Place of incident: South China Sea
Dive profile:
Did 3 dives to 120 feet on the same day. Each dive lasted 60 minutes. No decompression stop.
Symptoms at site of incident:
Weakness left lower limb, loss of sensation of left

TABLE 4

THE DELAY (NO. OF DAYS) IN THE DECOMPRESSION TREATMENT 1972 TO 1988

Days of Delay	Treatments	Percentage
1 - 2	5	6.4
2 - 5	39	50.0
>5 - 10	21	26.9
>10 - 15	9	11.6
15 or more	4	5.1
Total	78	100.0

thigh, no loss of bladder or bowel function.
Date of first recompression treatment: 31 October 1978
Physical examination before treatment:
Power: 3-4/5 left lower limb
Sensation: Sensory level L3 left lower limb
Treatment:
IV Rheomacrodex in Saline
Dexamethasone Injection for 3 days
Recompression using Table 6B.

Result:
30 minutes after the start of recompression therapy, the patient reported improvement in sensation and power. By the end of one treatment, he had full power in both lower limbs, and normal sensation.

Follow-up:
He was found to have dysbaric osteonecrosis affecting both the humeral heads when long bone X-rays were done on 3 November 1978. He was advised against further diving, and was given a recommendation to help him obtain a hawker's licence. On 7 April 1979, he was awarded compensation for a 39% disability ($14,000). He was last seen on 6 December 1979, when he was still diving despite our advice.

Case 2
Mr TCK, 40 year old Singaporean, Chinese, Male.
Date of incident: 25 May 1979
Place of incident: South China Sea
Dive profile:
Did one dive to 120 feet. Lasted 120 mins. Slow ascent with no decompression stop.
Symptoms at site of incident:
Onset 15 minutes after ascent. Had loss of consciousness, and wet recompression was attempted.
Date of first recompression therapy: 11 June 1979
Signs and Symptoms before treatment:
Only complained of joint pain involving the left shoulder and left elbow. Power 5/5, sensation intact, good bowel and bladder control.
Treatment:
Recompression using RN Table 61.
Results:
Complete resolution of pain after one treatment with no residual symptoms and signs.
Follow-up:
Last seen 15 September 1980. Found to be well. However, dysbaric osteonecrosis was noted in the shaft of both femur and tibia, and also in the right femoral neck. He was advised against further diving, and a recommendation letter was given to assist him to obtain a licence to be a fishmonger in a market.

Case 3
Mr YCH, 39 year old Singaporean, Chinese, Male
Date of incident: 22 March 1983
Place of incident: South China Sea
Dive profile:
Did 2 dives to 84 feet on the same day. First dive lasted 30 minutes. Second dive lasted 75 minutes. Ascent with no decompression stop.
Symptoms at site of incident:
Had joint pain, loss of sensation and weakness of both lower limbs. Also had loss of bladder and bowel function. Attempted wet recompression and self catheterised with Foley's Catheter.
Date of first recompression treatment: 6 April 1983
Signs and symptoms before treatment:
Power 5/5. Sensory level L3-4, Constipation +, anal reflex +, catheterised, cremasteric reflex +.

Treatment:
Recompression table 62 (modified)
IV Rheomacrodex 10%.
IM dexamethasone
Results:
Power 5/5. Hypoaesthesia from both knees down. Able to pass urine. Anal reflex +.
Follow-up:
Last seen on 8 August 1983. Power 5/5. Sensation: Slight numbness along the posterior aspect of both legs. Otherwise well. He was advised to limit his diving to non-decompression dives.

Case 4
Mr LMK, 26 year old Malaysian, Chinese, Male
Date of incident: 25 May 1979
Place of incident: South China Sea
Dive profile:
Dived to 150 feet for 90 mins. Slow ascent with no decompression stop.
Symptoms at site of incident:
Immediately after ascent weakness both lower limbs, loss of sensation from the waist down. Able to pass urine and use bowels.

Date of first recompression treatment: 20 June 1979
Signs and symptoms before treatment:

Power 3-4/5 both lower limbs, sensory intact.
Treatment:
Recompression Table 62.
Results:
Power 4/5 both lower limbs, sensation intact.
Second Treatment: 21 June 1979 Table 62.
Results:
Power 4+/5 - 5/5, sensation intact.
Follow-up:
Last seen on 3 June 1983. He was well with no permanent disability. No evidence of osteonecrosis noted in X-rays.

The above 4 cases clearly indicate that even after prolonged delay, recompression therapy with appropriate adjuvants will improve DCS. It is thus our policy to treat all decompression sickness and to repeat therapy if necessary until there is no further improvement.

TABLE 5

RESULTS OF TREATMENT GIVEN TO FISHERMAN DIVERS WITH DCS 1972 TO 1988

Results of Treatment	Number	Percentage of Total
Complete Recovery	17	21.8
Almost Complete Recovery	22	28.2
Definite Improvement	29	37.2
No Definite Improvement	10	12.8
Clinically Deteriorated	0	0
Died	0	0
Total	78	100.0

Rehabilitation

As seen in Table 5, only 21.8% of those treated recovered completely from their DCS, although a further 28.2% had near complete recovery. So 50% of those treated have a significant disability, requiring some form of rehabilitation. The residual disabilities include loss of motor power, sensory loss, bowel and bladder dysfunction. With an effective programme, a good percentage of these disabilities can be minimised and overcome, and the patient can regain useful psychomotor and social skills.

In Singapore all cases of DCS who have residual disability after recompression therapy are further managed at the Department of Rehabilitation Medicine (DRM) at Tan Tock Seng Hospital. The majority of cases seen at the DRM are DCS Type II with spinal cord involvement. In a study conducted by Tan and Balachandran[1], there was a total of 15 cases of DCS with spinal involvement treated at DRM from 1973 to 1978 and referred to DRM. All these had bladder and bowel function involvement. Five also had bone complications. All were male Chinese, with ages ranging from 25 to 64. Out of the 15, two had weakness of all 4 limbs whilst the rest had weakness of both the lower limbs. The results of rehabilitation are shown in Tables 6, 7 and 8.

Team approach

For total management of a DCS patient with residual disability, a team of professionals is required. The NMRC and DRM have teamed up to provide the rehabilitation programme for divers. Each specialist may be called upon to assist from their respective units. This team consists of:

1 Specialist in diving medicine.
2 Specialist in physical rehabilitation medicine.
3 Physiotherapist.
4 Social worker.
5 Psychologist.
6 Urologist.
7 Orthopaedic surgeon.
8 Radiologist.
9 ENT surgeon.

Aims of Rehabilitation

The aims of rehabilitation after hyperbaric therapy are:

1 To achieve the highest level of useful motor power possible.
2 To help the patient to adjust psychologically to his limitations.
3 To achieve a catheter free state where the bladder is able to empty satisfactorily with voluntary control.
4 To enable normal bowel function.
5 To achieve functional independence and ultimately to return to the patient's former occupation.

Bladder Training

Unlike motor weakness and sensory deficit which improve considerably after recompression therapy, a significant percentage of cases of neurologic bladder do not improve even after repeated recompression. These people require a different mode of rehabilitation. Some require surgical intervention (transurethral resection of the sphincter), medication (Baclofen to improve micturition), and finally bladder training. The aim is to prevent overdistension, minimise local damage and infection, and obtain an adequately emptying bladder, with a low and sterile residual urine.

TABLE 6

SENSORY INVOLVEMENT (PAIN)

Case	Before Recompression Therapy	After Recompression Therapy	After Rehabilitation
KNS	Loss below T12	Hypoaesthesia below T12	Normal
WSK	Loss below T7	Hypoaesthesia below T12	Hypoaesthesia
ESL	Loss below T7	Hypoaesthesia below L5	Brown Sequard below C7
OTB	Loss below C7	Hypoaesthesia below T5	Hypoaesthesia below T5
KTK	Hypoaesthesia below T3	Hypoaesthesia below T3	Hypoaesthesia below L3
LPC	Hypoaesthesia below T5	Hypoaesthesia below T8	Normal
TKL	Hypoaesthesia below T10	Hypoaesthesia below T 10	Hypoaesthesia below T10 improved
SKS	Hypoaesthesia below T7	Hypoaesthesia below L2	Hypoaesthesia below T2 improved
QTH	Loss below T8	Loss below L3	Hypoaesthesia below L3
THS	Loss below T9	Hypoaesthesia below T10	Hypoaesthesia below L1
LKL	Loss below T5	Loss below T8	Normal
TLK	Loss below T8	Loss below T10	Loss below L5
NKS	Loss below T7	Loss below L2	Hypoaesthesia below L3
CYP	Loss below L1	Loss below L3	Hypoaesthesia below L3
QKH	Loss below T5	Loss below T10	Hypoaesthesia below L1

Bladder training is done at the DRM. On admission the patient is first subjected to a series of tests to get a bladder profile. The URODYN 5000, the latest urodynamic equipment acquired by the DRM, is used. This machine is able to provide information about the urinary flow rate, intravesical pressure, abdominal pressure, detrusor pressure, urethral pressure, water and carbon dioxide cystometry and electromyography.

With this information, we can then determine whether the patient has an upper motor neurone (UMN), lower motor neurone (LMN) or mixed type of bladder dysfunction. The capacity and efficiency of the bladder and the condition of the sphincters can also be shown. With these findings, the appropriate bladder training method can then be planned for the particular patient.

With a LMN lesion, the bladder tone tends to be flaccid. Emptying of the bladder can be assisted by cholinergics like carbachol, ubretid and pyridostigmine. In UMN lesions, the bladder tends to be unstable with a small capacity. This state can be improved by medications like oxybutynin to relax the tone. Dibenyline is also used to relax the internal sphincter. The capacity of the bladder can be gradually increased by hydrodilation, a process using water to distend the bladder. If the patient still finds difficulty in micturition after a bladder training programme with medication, he is again assessed by the urologist for possible surgical correction. A sphincterotomy can be done for those with high sphincter tone, and transurethral resection of the prostrate (TURP) done for patients with enlarged prostates.

Prior to 1987, an indwelling catheter was used. Now 4 hourly intermittent catheterisation is used. Every 4 hours a doctor will apply abdominal tapping and compression just before inserting the disposable catheter. The volume drained by the catheter is recorded. This method is continued until the residual volume is below 100 ml. Once this is achieved, the patient is taught to do tapping and compression every 4 hours.

During bladder training the patient is given restricted fluid (normally 2 litres between 6 am and 6 pm). Serum creatinine, serum urea and electrolytes are done on admission. Urine microscopy is done twice weekly and urine culture and sensitivity thrice weekly during the entire period. If possible, an annual intravenous pyelogram is also done to detect early outlet obstruction, including urinary stricture formation.

Results of Bladder Training

Although recompression therapy fails to restore fully the neurologic bladder in some of the cases, a proper bladder training programme can lead to a satisfactory functional

TABLE 7

BONE AND MUSCLE POWER INVOLVEMENT

Case	Before Decompression Therapy	After Decompression Therapy	After Rehabilitation	Dysbaric Ostenecrosis
KNS	Paraplegic: Right Grade 2 Left Grade 3	Grade 3 Grade 4	Normal	Right Femoral Head affected (2 years after injury)
WSK	Paraplegic: Grade 0 bilateral	Grade 2 bilateral	Grade 4 + bilateral	Nil
ESL	Tetraplegic: Grade 0 all limbs	Paraplegic Grade 4 Bilateral	Right Grade 4 + Left Grade 5	Nil
OTB	Tetraplegic: Grade 0 all limbs	Upper limbs Grade 3 + Lower limbs Grade 1 +	Upper limbs Grade 4 + Lower limbs - Grade 2-3	Nil
KTK	Paraplegic: Grade 0 bilateral	Grade 2 + bilateral	Right Grade 4 Left Grade 3	Nil
LPC	Paraplegic: Grade 0 bilateral	Right Grade 4 Left Grade 3	Grade 5 Grade 4	Nil
TKL	Paraplegic: Grade 0 bilateral	Right Grade 3 Left Grade 4	Grade 4 Grade 5	Nil
SKS	Paraplegic: Grade 0 bilateral	Right Grade 2 Left Grade 3	Grade 3 Grade 4	Both femora and tibiae and right humeral head and shaft (6 months after injury)
QTH	Paraplegic: Grade 0 bilateral	Right Grade 4 Left Grade 3	Grade 4 + Grade 4	Left Femoral Head affected (2 months after injury)
THS	Paraplegic: Right LeftGrade 2-3	Grade 2 Left Grade 5	Right Grade 4	Normal Nil
LKL	Paraplegic: Grade 2 bilateral	Right Grade 3 Left Grade 4	Grade 4 + Grade 5	Right Femoral Head affected (1 month after injury)
TLK	Paraplegic: Grade 0 bilateral	Right Grade 3 + Left Grade 0	Grade 4 + Grade 0	Right Femoral Head (2 months after injury)
NKS	Paraplegic: Grade 0 bilateral	Right Grade 2 + Left Grade 3 +	Grade 3 + Grade 4	Nil
CYP	Paraplegic: Grade 3 + bilateral	Grade 4 bilateral	Grade 4 + bilateral	Nil
QKH	Paraplegic: Grade 0 bilateral	Right Grade 4 Left Grade 3	Grade 5 Grade 4	Nil

TABLE 8

BLADDER INVOLVEMENT

Case	Before Recompression Therapy	After Recompression Therapy	After Bladder Training	Drugs Used and/or Surgical Intervention
KNS	Neurogenic Bladder with acute retention of urine	Straining required to initiate micturation	Normal function	Nil
WSK	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Nil
ESL	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
OTB	Neurogenic Bladder with acute retention	No improvement	Spontaneous Micturation	Transurethral resection of sphincter
KTK	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Nil
LPC	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Nil
TKL	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
SKS	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
QTH	Neurogenic Bladder with acute retention	No improvement	Micturation needs tapping and compression	Nil
THS	Neurogenic Bladder with acute retention	Straining required to initiate micturation	Straining required to initiate micturation	Nil
LKL	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Nil
TLK	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
NKS	Neurogenic Bladder with acute retention	No improvement	Micturation needs compression	Lioresal 5 mg tab tds
CYP	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
QKH	Neurogenic Bladder with acute retention	No improvement	Normal function	Nil

state. In the study done by Tan and Balachandran[1], the majority of cases with impaired bladder function after recompression therapy achieved near normal function after rehabilitation training.

Bowel Training

Those DCS cases with bowel dysfunction are first put on a high roughage diet and laxatives such as Senokot and Agarol to help defaecation. In addition, a Dulcolax suppository is given every third day. With this regime, most patients will be able to empty their bowels regularly. Other measures that may be taken are enema soap and water or digital evacuation of the rectum. With this regime, the bowel will be reconditioned to empty every third day. For resistant cases, the regime may be continued indefinitely, or until such time spontaneous defaecation is possible.

Physical Training

Fishermen treated for DCS who are left with significant residual motor weakness (usually spinal bends with paraparesis) are referred to DRM, whose doctors will assess them for suitability and potential.

The programme for physical rehabilitation starts with a baseline assessment of the motor power of various muscle group. There is a graduated exercise programme starting with non- weight bearing exercises, followed by partial weight bearing and finally full weight bearing.

Training is assisted with the use of various aids and appliances. For strengthening the upper limbs, free weights are used extensively. To strengthen finger grip, a ball of plasticine like material is used to provide resistance for finger exercises. In addition, spring exercises are done. The limbs are exercised against the recoil force of springs of various strengths.

For the lower limbs free weights and spring exercises are used for initial strengthening. When the lower limbs are strong enough to proceed to partial weight bearing exercises parallel bars, walking frames and walking sticks are used. Most of these require good upper limb strength. For paraplegics the main aim is to achieve good upper limb function by doing weight lifting. The patient is then taught wheel chair transfer by the occupational therapist. This is to enable them to move themselves from the wheelchair to the commode, bed, etc.

In addition, there is a pool for hydrotherapy at the DRM. This is ideal for patients with severe lower limb weakness as buoyancy will eliminates the effect of gravity, thus allowing the limbs, which are normally too weak to move against gravity, to move freely.

Social Adjustment

As noted before, about a quarter of the fishermen DCS cases suffer from dysbaric osteonecrosis. If the dysbaric osteonecrosis is severe or affects the articular surfaces, we advise them against further diving and assist them in seeking compensation and alternative suitable employment. The same is done for severe type II DCS with residual functional impairment, such as spinal bends with paraparesis or hypoaesthesia. They are assisted in their application for a hawker's licence, a job which is highly lucrative and sought after even by healthy persons.

However, there are some "hardcore" divers who continued to dive despite their disability. This is most commonly due to the fact that their livelihood depends on being a fisherman diver, and they cannot find an alternative job.

For those with permanent disability, we assess the degree of disability and assist them in obtaining their workman's compensation entitlement. In the rehabilitation centre, they are taught and trained in various self-care activities such as going to the toilet, eating, dressing, etc. This is done for all patients with severe physical disabilities and the aim is to achieve complete independence either with or without aids or appliances.

Somatosensory evoked potentials (SSEP)

SSEP involves the stimulation of a peripheral nerve either in the upper or lower limb and the recording of the evoked potential. It has been proven useful in the investigation of disorders affecting the nervous system. Demyelinating and degenerative disorders display abnormalities in SSEP and it can be used to provide information on the site of the lesion as well as a means to monitor the progress of the disease and the efficacy of therapeutic measures.

At our centre, we are now conducting a study to compare how well the SSEP recordings correlate with the clinical findings. So far we have managed to do a SSEP recording for 9 of our DCS cases after their recompression therapy. So far no pre-recompression recording has been done for any of the cases. The results obtained and the clinical findings are given in Table 9.

SSEP Results

NORMAL VALUES

Median Nerve

N1 : 16.5 - 20.9 ms

P1 : 20.8 - 28.4 ms

Posterior Tibia Nerve

N1 : 43.2 - 51.8 ms

P1 : 34.0 - 39.5 ms

TABLE 9

SSEP RESULTS

Case	Nerve tested	Result	Clinical Findings
KSL	Right Median Nerve	N1 19 ms P1 44 ms*	Hypoaesthesia right hand
	Right Posterior Tibia Nerve	N1 48 ms P1 35 ms	Normal
AFY	Left Median Nerve	N1 21 ms P1 28 ms	Normal
	Left Posterior Tibia Nerve	N1 51 ms P1 44 *	Normal
HCL	Left Median Nerve	N1 17 ms P1 49 ms*	Normal
	Left Posterior Tibia Nerve P1 36 ms	N1 52 ms	Hypoaesthesia T8 downwards
LE	Left Median Nerve	N1 18 ms P1 21 ms	Hypoaesthesia C5, 6, T1
	Left Posterior Tibia Nerve	N1 47 ms P1 37 ms	Normal
ZAK	Left Median Nerve P1 20 mns	N1 16 ms	Normal
	Right Median Nerve P1 20 ms	N1 16 ms	Normal
	Right Posterior Tibia Nerve	N1 49 ms P1 64 ms*	Hypoaesthesia L2 downwards
TCT	Left Median Nerve P1 26 ms	N1 18 ms	Normal
	Left Posterior Tibia Nerve	N1 41 ms P1 36 ms	Normal
AL	Right Median Nerve	N1 16 ms P1 20 ms	Normal
	Left Posterior Tibia Nerve	N1 45 ms P1 36 ms	Normal
AHG	Median Nerve	N1 19 ms P1 35 ms*	Numbness fingers
	Posterior Tibia Nerve	N1 70 ms* P1 60 ms*	Patchy numbness lower limbs
SGN	Left Median Nerve	N1 20 ms P1 30 ms*	Normal
	Right Median Nerve	N1 20 ms P1 31 ms*	Hypoaesthesia T1-2
	Left Posterior Tibial Nerve	N1 8 ms P1 39 ms	Normal
	Right Posterior Tibia Nerve	N1 6 ms P1 40 ms	Normal

* denotes prolonged

NORMAL SSEP RESPONSES

Group One	Normal Clinical Findings
AL	
TTC	

Group Two	Abnormal Clinical Findings
KSL	Loss of proprioception right lower limb
LE	Hypoaesthesia C5 6 T1
HCL	Loss of pin-prick T8 downwards

ABNORMAL SSEP RESPONSES

Group Three	Normal Clinical Findings
AFY	Prolonged P1 right PT nerve
HCL	Prolonged P1 left median nerve
SGN	Prolonged P1 left median nerve

Group Four	Abnormal Clinical Findings
KSL	Prolonged P1 right median nerve with hypoaesthesia R hand
ZAK	Prolonged P1 right PT nerve with hypoaesthesia L2 onwards
ASG	Prolonged P1 median nerve with numbness fingers Prolonged P1 PT nerve with numbness lower limbs
SGN	Prolonged P1 right median nerve with hypoaesthesia T1-2

Discussion

In the first group, normal SSEP responses correspond to normal clinical findings. As SSEP measures the electrophysiological integrity of the sensory pathways, it gives an indication that the recovery from decompression sickness is complete.

In the second group of patients where normal SSEP responses were obtained inspite of clinical neurological deficits, it lends support to the suggestion put forth by Giblin[2] and Halliday and Wakefield.[3] They proposed that the evoked responses depended on the integrity of the posterior column pathways but were unaffected by lesions of the spinothalamic tracts. The only anomaly in the group, LKS, may be explained by a study which suggested that some of the afferent impulses responsible for the cortical potential travelled by the spinothalamic tracts.

Group three results suggest that inspite of apparent full clinical recovery, residual damage had occurred secondary to decompression sickness. SSEP then, provides a sensitive means to detect subclinical damage. This result is supported by Palmer et al[4] where extensive morphological changes existed in the spinal cord of divers who had a history of decompression sickness but recovered fully. The possibility of recruitment for neighbouring neurones in such cases to provide compensation for the damaged one may explain the normal clinical findings in such divers.

In group four, prolonged SSEP responses were obtained in patients with sensory loss mostly to pin-prick. This differs from the conclusion drawn by Halliday and Wakefield.[3] Whether this is due to the different aetiologies of the two study groups, (decompression sickness vs non-diving conditions, e.g. prolapsed intervertebral disc, cervical spondylosis, cerebrovascular haemorrhage, etc.) is uncertain. There is the opportunity for a more extensive prospective study involving a much larger sample size.

The most important conclusion obtained thus far in our present study is that abnormal SSEP were obtained in patients who are apparently normal subjects. This has important implications for our young naval divers and brings to mind the question whether routine SSEP study should be conducted for them. The difficult problem of medico-legal compensation also needs to be clarified in cases where prolonged SSEP were to be obtained in apparently normal divers. The other question that needs to be addressed is whether one should allow divers who have apparent full clinical recovery after decompression sickness but abnormal SSEP recordings to continue diving. Whether one previous episode of decompression sickness predisposes the spinal cord to further insults remains to be solved.

Dysbaric osteonecrosis

Another complication of diving is dysbaric osteonecrosis. Although the majority of patients present with decompression sickness, 36 out of the 78 DCS patients were found to have dysbaric osteonecrosis by post-treatment long bone X-ray. The disability suffered by those affected varies from mild asymptomatic type B to severe type A requiring arthrodesis and arthroplasty to achieve a pain free or functional state.

Conclusion

Since the beginning of our work in 1972[5], fisherman divers have made up the majority of all DCS. Because of their typical late presentation they have contributed enormously to our experience in treating delayed cases. From the results we have obtained we are convinced of the potential benefit in treating cases even after a delay of more than 15 days. We have yet to establish the cut off time after which decompression therapy will have no beneficial effect.

On the other hand, it is also clear that there will be some cases where recompression therapy will not help. However, even so, a well planned and carried out rehabilitation programme can help to improve the patient's physical, social and mental state. It is because of this that we have adopted the team approach with the Department of Rehabili-

tation Medicine and various specialist departments of the Tan Tock Seng Hospital. We aim to give all the DCS patients the complete management that they deserve.

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TABLE 1

SOME ADAPTATIONS TO APNOEIC DIVING IN AQUATIC MAMMALS

Respiratory

Tolerance of thoracic squeeze

lung collapse
elastic chest wall
mechanically tough tracheo-bronchial tree

Protection from decompression sickness

lung collapse (loss of gas exchange)
Decreased sensitivity to hypoxia and hypercapnia
Increased ventilatory/gas exchange efficiency

Cardiovascular

Blood shift into thoracic cavity
Venous spincters and sinuses
Large spleen
Diving Reflex CVS components

Biochemical

Increased blood (haemoglobin) and tissue (myoglobin) oxygen stores
Diving Reflex Switch to anaerobic metabolism

Hypothermia/Insulation

Efficient locomotion

BREATH-HOLD DIVING

Michael Davis

Introduction

There is a beautiful photograph by Flip Nicklin in the December 1984 issue of the National Geographic magazine of a sperm whale sounding. This epitomises for me the wonder and majesty of the diving mammals, that diverse group of animals of widely varying structure, function, habitat and behaviour who grace our oceans (Table 1). This paper briefly reviews the physiology of these animals and aspects of human breathhold diving, whilst the bibliography provides a selection from the literature rather than an exhaustive list.

Asphyxia is a progressive process which begins at the moment that external gas exchange ceases. The cessation of respiration leads to hypoxia, hypercapnia and acidosis, the triad of asphyxia. The successful mammalian diver can postpone the inevitable functional collapse that follows the cessation of breathing by virtue of three main mechanisms:

1 Enhancement of the oxygen stores in the body
2 Acid buffering of the products of metabolism
3 Circulatory reduction and redistribution, leading to metabolic conservation.

Regulation of these responses is essentially identical to that governing the protective reaction to asphyxia in terrestrial animals, the observed differences being quantitative rather than qualitative.

Scientists have approached the study of these remarkable animals' adaptation to asphyxia both in the laboratory and in their natural environment. Although some laboratory work has been criticised, field and laboratory research are, in fact, complimentary, and not conflicting. "Exploring what an animal is capable of is not the same as asking how it normally behaves."