12 Dr Fred Bove

I do not think that you would expect to find any changes in the X-rays of the shoulders of a person with decompression sickness. We think that somehow there is a vascular involvement of the long bones, although that is not an explanation of the pain. A bone scan would be a much more useful piece of information because it would tell you whether there was any alteration of the blood flow in the region of the pain. The X-ray obviously would not change for many months. So if you are concerned you might do a bone scan to see whether there are ischaemic areas.

Dr John McKee

The X-rays were taken 300 miles from a centre with scan equipment, but more to reassure the diver than me and partly to help decide whether to spend about \$100,000 transferring him to Sydney for treatment. Very frequently one gets these divers with various symptoms, and the transfer cost, by helicopter or by Hercules, is now pretty tremendous.

Dr Fred Bove

Just one more comment. When I was at Philadelphia, we treated a case of limb bends in a commercial diver working in the Atlantic Ocean who, by the time he got to the chamber had lost all his symptoms. We still treated him in the chamber. I would suggest that where there is a chamber available you should still treat the symptom free patient with perhaps a table 5. There is a remote chance that you are preventing long term changes in the bone.

HOW COMMON IS DYSBARIC OSTEONECROSIS?

John Knight

Every diver has heard of dysbaric osteonecrosis. Very few are aware that the risk of developing dysbaric osteonecrosis is low if one follows normal air diving procedures.

Surveys of naval divers in the UK (1) and the USA (2) have shown a very low incidence of dysbaric osteonecrosis. This contrasts with earlier surveys of compressed air workers. The major difference between the two groups was that compressed air workers spent a working shift under pressure while the divers had much shorter periods under water. Many of the naval divers with bone changes had been involved in experimental diving such as testing new decompression tables and deep diving.

JAPANESE EXPERIENCES

In 1976 there were two papers from Japan published in English. Ohta and Matsunage (3) reported a three year survey (1966-1968) of the men of a village (Ohura) on the shore of the Ariake Sea off Northern Kyushu where there

TABLE 1

CLASSIFICATION OF DYSBARIC OSTEONECROSIS

JUXTA-ARTICULAR LESIONS

- A1 Dense areas with intact articular cortex
- A2 Spherical segmental opacities
- A3 Linear opacity
- A4 Structural failures
- a) Translucent subcortical band
- b) Collapse of articular cortex
- c) Sequestration of cortex
- A5 Secondary degenerative arthritis (osteoarthritis)

were about 400 divers (active or retired). The men dived to collect the expensive shell fish Atruria Japonica from November to March (winter). Except during bad weather they dived in 10m to 30m, while there was light, with a lunch break of an hour. This gave two four-hour dives each day. During the rest of the year they collected other shell fish from 30m to 60m, worked on salvage jobs from 20m to 70m or more, and did construction jobs in 10m to 30m. To quote Ohta and Matsumaga they "had used no modern technique of decompression". This probably explains the fact that three to five men died each year from accidents or decompression sickness. Decompression sickness was very common, so common as to be considered unavoidable. Their treatment was baths and booze.

Of the 301 divers who were X-rayed, 152 (50.51) had bone lesions. Of these, 44 men (29%) had juxta-articular lesions. Between them they had 54 juxta-articular lesions. The group had a high incidence of previous decompression sickness, but there was no significant relationship between the site of the bends pain and the lesions. The incidence of bone changes was higher in those who had been diving longer and in those who had gone deeper. The results of the first year's X-rays had been published in Japan in 1966.

The figures were quite horrifying. In the group with up to 4 years diving experience, 22% had bone changes, none of which were juxta-articular. The 5 to 9 year group had an incidence of 46%. The 10 to 14 year group had an incidence of 71%. After 15 years of diving the incidence was 74% and after 20 years it settled at 82%. The deepest depth figures were just as depressing. No lesions in those who had not exceeded 9m, but there were only 8 of them. The 10m to 19m group had a 20% incidence, luckily for them without juxta-articular lesions. Those who dived between 20m and 29m had a 46% incidence, while 30m to 39m was associated with an incidence of 53%. The 40m

to 49m range had an incidence of 64%, while over 50m it was 74%.

The survey had some failings. Only 43 divers were X-rayed on all three possible occasions. Only 144 were X-rayed twice or more. So the figures are probably an underestimate of the incidence.

The same year Amako, Kawashima, Torisu and Hayashi (4) published a paper which carried the same survey on till 1972. Since the survey started the divers had obtained a small chamber which was used for deck decompression. They had also abandoned the lunch break. This report covered 450 divers X-rayed of whom 123 had been observed for more than seven years.

Of the 450 divers, 268 had bone lesions (59.5%). 81 had juxta-articular lesions (18% of the total). 30% of those with lesions had the potentially disabling juxta-articular lesions.

An interesting finding was that the divers developed bone islands between X-rays. This led the authors to add a third category (C. Bone Islands) to the usually accepted classification of dysbaric osteonecrosis.

The incidence of dysbaric osteonecrosis increased from 46% in the 16-19 year olds, to 49% for the next decade, to 70% for divers in their 30's and to 75% in the forties group. Those over 50 only had a 60% incidence, presumably because of death and retirement. The associations between experience and depth and increased osteonecrosis were confirmed.

The most interesting findings were those about the 123 divers who were studied for seven years. In 1965, 36 had multiple lesions, 18 had solitary lesions, while 69 had no lesions. By 1972 there were 69 with multiple lesions and 27 with solitary lesions. Only 27 divers had no evidence of dysbaric osteonecrosis. During the six years of the survey, 79 divers had developed new lesions. 27 of them had new juxta-articular lesions, 25 had new head, neck and shaft lesions, while 13 had developed bone islands, which are not normally considered to be due to dysbaric osteonecrosis. To confuse matters, some of those who had lesions on the first survey did not develop new ones. In all, 44 divers came through the survey without developing lesions.

THE AUSTRALIAN SCENE

In 1976 Williams and Unsworth (5) reported on the X-rays of 110 divers, 31 from the RAN, 15 from the NSW Police, 8 marine biologists or scientific workers, 37 commercial divers and 19 sports divers. Three divers were said to have used other than compressed air. This suggests the use of helium, but helium is not specifically mentioned in the paper. Naval clearance divers use oxygen-nitrogen mixtures which differ in composition from compressed air. Abalone divers are commercial divers as they make their living from diving. But abalone divers are known to treat decompression tables rather casually, while employee

divers are usually more careful. Abalone divers are not mentioned in the paper. Progression of any lesions discovered could not be followed as only 30% of the participants had two sets of X-rays taken.

84 divers (72%) had no bone changes, 8 (7%) had juxtaarticular lesions. Again this is a high proportion (30%) of those with lesions. 18 (17%) had head, neck and shaft lesions. Presumably two divers had both sorts of lesions as their table gives 20 with B type lesions while simple mathematics gives 18.

They also found 18 other X-ray lesions involving joints. Such things as osteochondritis dissecans, myositis ossificans, deltoid bursitis, shoulder dislocation, arthritis after fracture, traumatic arthritis, tendon calcifications, capsular calcification and periosteal avulsion do not appear in other reports. This group appears to have had more than its fair share of serious trauma as 22 lesions were attributed to trauma among 110 men (20%).

When one considers the various groups of divers included in the survey, the RAN had 13 of 31 with lesions (42%), the NSW Police 5 out of 15 (33%), the commercial divers 6 out of 37 (16%), other full time divers 1 out of 8 (12.5%) [there were no head, neck and shaft lesions in this group] and the sports divers had 3 with lesions from 19 subjects (15.4%).

The incidence of juxta-articular lesions was RAN 1 in 31 (3.2%), Police 2 in 15 (13%), commercial divers 3 in 37 (8%), other fulltime divers 1 in 8 (12.5%) and sports divers 1 in 19 (5.2%).

The figures for the RAN divers are high because only those RAN divers who had suspicious X-rays were referred to Prince Henry Hospital. When the total RAN diver population is considered the incidence was much the same as in other navies. Harrison (1) found 18 definite lesions in 383 divers (4.7%) while a survey of 834 USN divers (2) produced 16 with lesions (1.7%).

This paper by William and Unsworth (5) was the first report of dysbaric osteonecrosis lesions in sports divers, and one of the three cases was a juxta-articular lesion

OPTIMISM IN THE 1970's

In 1977 Dr John D Davidson of the MRC Radiological panel visited Melbourne. He gave a lecture on dysbaric osteonecrosis. He quoted, from MRC sources, the findings from five tunnelling contracts between 1955 and 1966.

There had been a decompression sickness rate of 1.3% Type I and 0.12% Type II, but the incidence of dysbaric osteonecrosis had been 19.2% of the 1,694 tunnel workers. 334 men had definite lesions while another 229 (13.31%) had suspected lesions. 7 of these lesions (3.5%) were reclassified as definite lesions during follow up.

The lesions were distributed as in Table 2. 40% were juxtaarticular. This was a much higher percentage of potentially disabling lesions than in the Japanese fishermen. However, the Japanese had nearly three times the incidence of bone changes so were more at risk.

TABLE 2

DISTRIBUTION OF 820 DYSBARIC OSTEONECROSIS LESIONS IN 334 TUNNEL WORKERS

JUXTA-ARTICULAR	
Dense areas	21%
Increased calcification	9%
Dense lines	5%
Structural failure	5%
Osteoarthritis	5%
HEAD, NECK AND SHAFT	
Dense areas	20%
Increased calcification	40%

At that time the MRC had X-rays of 2,300 compressed air workers and of 2,516 divers. 383 tunnellers (17%) had bone changes, while only 60 divers (2.4%) were affected. Dividing the men into air divers and helium divers, on the basis of a maximum depth of more than 50m, the incidence was 0.4% in 804 air divers and 2.7% in 1,158 helium divers. Modern commercial diving seemed to have beaten the bogey.

THE NORTH SEA IN 1979

Last year David Elliott told us about the latest figures from the North Sea. In August 1981 The Lancet published a paper on the subject (6). It was a survey of 4,980 divers of whom 4,670 (93.8%) had no lesion. 106 had suspected lesions (2.1%) and 207 (4.21) had definite lesions. 700 men have been X-rayed 5 or more times and over 2,000 have had three or more sets of X-rays. Interestingly, 82 men were excluded from the survey because of the poor quality of the X-rays. Included in the normal group were those whose "only radiological abnormality was a bone island, a cyst or some other unimportant change".

 $62 \text{ men } (1.2\%) \text{ had a definite juxta-articular lesion. } 142 \text{ men } (2.8\%) \text{ had a definite head, neck or shaft lesion.} Again, those with juxta-articular lesions formed a large proportion (43\%) of those with lesions.}$

The anatomical distribution is shown in Figure 1. As some men had multiple lesions, there were 72 juxta-articular and 274 head, neck and shaft lesions to record. The numbers in brackets are those with damaged joints.

When discussing the factors associated with dysbaric osteonecrosis, the authors, by controlling the experience factor, were able to show that age, per se, is not associated with the lesions. However, diving experience (and therefore in general, age) is. Prevalence rose from 0.7% for those

TABLE 3

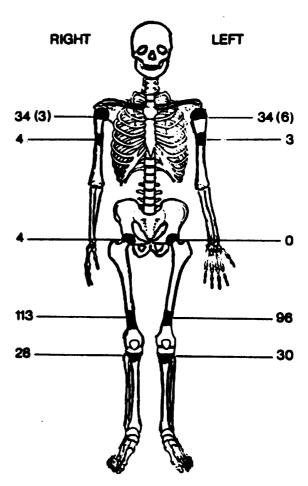
DYSBARIC OSTEONECROSIS IN 4980 COMMERCIAL DIVERS

LESION	DIVERS	%
None	4670	93.8
Suspected Head, Neck, Shaft	71	1.4
Definite Head, Neck, Shaft	142	2.8
Suspected Juxta-Articular	35	0.7
Definite Juxta-Articular		
Intact	53	1.1
With Damage to Joint	6	0.2
With Secondary Osteoarthritis	2	0.2
Surgically treated	1	0.2

with less than 4 years experience, to 2.2% between 4 and 8 years, 5.5% between 8 and 12 years and to 10.7% after 12 years of diving.

Fatter and heavier men were statistically more likely to develop lesions. So were men who had had decompression sickness. Of those who claimed never to have been bent (60%), 1.7% had developed lesions. But 10.7% of those who admitted having decompression sickness developed lesions. 8 of the 9 men who had damaged joints said that they had been bent.

FIGURE 1 SITES OF DYSBARIC OSTEONECROSIS IN 207 COMMERCIAL DIVERS



DIVING EXPERIENCE AND DYSBARIC OSTEONECROSIS

YEARS	NORMAL	LESIONS PREVALENCE
DIVING		

Less than 4	1262	9	0.7%
4 to 8	1300	29	2.2%
8 to 12	885	52	5.5%
12 or more	869	104	10.7%

TABLE 5

DECOMPRESSION SICKNESS AND DYSBARIC OSTEONECROSIS

DYSBARIC OSTEONECROSIS			
NO	YES	TOTAL	
2978	52	3030	
1228 4206	147 199	1375 4405	
	NO 2978 1228	NO YES 2978 52 1228 147	

Quite definitely, dysbaric osteonecrosis is related to the type of diving. Compressed air to 30m was lesion free. 31-50m gave 8 men lesions (0.8% of those exposed to this as a maximum depth). 51-100m gave 19 men lesions (1.6%), 101-200m gave 139 men lesions (8.1%) while 201m or more resulted in 30 men with lesions (15.8%). Saturation diving, with adequate records had been done by 1,725 men, of whom 143 had lesions. Again the incidence increased with depth.

The MRC report points out that femoral juxta-articular lesions are rare in divers and that only nine divers had damaged humeral heads making it impossible to be certain about the proportion of juxta-articular lesions that will progress to change. However, in 93 compressed air workers with juxta-articular lesions, nine went on to joint damage (9.7%). But one should include the 48 who had joint damage at the first survey which pushes the incidence

up to 40%. For the divers reported on, the figure cannot be less than 9 in 72 (12.5%).

TABLE 6

DYSBARIC OSTEONECROSIS AND MAXIMUM DEPTH

I MAXIMUM D %	DIVERS EPTH	DEFINITE IN METRES	LESIONS
Less than 30	317	0	0.0
31 to 50	1025	8	0.8
51 to 100	1171	19	1.6
101 to 200	1718	139	8.1
200 and deeper	190	30	15.8

The prevalence of bone lesions in the North Sea diving population has risen from 0.94% in 1975 to almost 5% in 1979. The incidence, proportion of new cases per year, has varied but remained below 1.27% of those at risk per year. There is a sharp rise in the incidence of dysbaric osteonecrosis after the sixth year of experience which is about the time most divers start helium and saturation diving.

1,725 saturation divers had 143 lesions (8.3%). Of these 27 were juxta-articular or damaged joints (1.5%) and 116 had head, neck and shaft lesions (6.7%). 3,255 men had never done saturation diving or were without sufficient data. Simple arithmetic gives us 38 men with juxta-articular lesions (1.2%) and 26 with shaft, head and neck lesions (0.8%) in the non-saturation group. This ignores multiple lesions in both groups. According to the number of lesions recorded up to 10 men had more than one juxta-articular lesion while 275 head, neck and shaft lesions were shared by 204 men.

DAMAGED JOINT

David Elliott told us last year that minor bends were very common in commercial diving and that the saturation depth is often 150 feet above working depth. These facts

TABLE 7

DYSBARIC OSTEONECROSIS AND SATURATION

MAXIMUM DEPTH SATURATION (m)	DIVERS	NORMAL	MOST RECENT DEFINITE HNS (%)	X-RAY RESULT DEFINITE JA (%)	DAMAGED JOINT
1 to 100	215	212	1 (0.5)	1	1
101 to 150	664	621	32 (4.8)	10 (1.5)	1
151 to 200	656	589	57 (8.7)	9 (1.4)	1
201 to 300	136	118	16 (11.8)	2 (1.5)	0
301 or deeper	54	42	10 (18.5)	2 (3.7)	0

could explain why the incidence of dysbaric osteonecrosis is higher now than it was in the middle of the 1970's.

ADVICE TO THE SPORTS DIVER

What can we make of this? Sports divers who do not go below 30m, and observe the tables properly are most unlikely to develop lesions.

Those who dive below 30m, to 50m, have approximately a 1% chance of developing lesions. On the basis of the MRC figures, which do not give the lesions associated with each depth, 36% of lesions will be juxta-articular, probably in the humerus. Recorded attacks of decompression sickness increase the incidence of bone lesions nearly five times. In Australia, many cases of decompression sickness are associated with rapid ascents.

Various surveys of both new and in use depth gauges have shown that a large proportion are inaccurate. Luckily many overestimate the depth. But many underestimate the depth and can lead the diver astray in his depth-time profile.

Careful diving, using a recently calibrated depth gauge, a waterproof watch, staying within the no-decompression limits and avoiding sudden unscheduled ascents and running out of air should allow the sports diver to enjoy his or her fun without risking any joints.

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THE SAFETY OF THE UNITED STATES NAVY DECOMPRESSION TABLES AND RECOMMENDATIONS FOR SPORTS DIVERS

Bruce Bassett

I will quickly review what Haldane found and what the United States Navy (USN) modifications to the original Haldane model were, as a result of several years of development of decompression tables. Then I will analyse the safety of the USN standard air decompression tables, leading on to recommendations for sport divers based on safety analysis.

Haldane basically had two observations. Testing his animals by exposing them to increasing depth, keeping them there long enough to attain equilibrium with the inert gas pressure, saturating them, and then decompressing them, he developed the concept that there was a critical supersaturation ratio. This is often expressed as a pressure ratio. One can always reduce the pressure by one half within the realm that he worked in, to pressures of 6 atmospheres absolute. However the important aspect is the driving force for inert gas to form bubbles. Therefore it is expressed as a ratio of the nitrogen pressure, trying to force gas out of solution, versus the barometric pressure trying to keep the gas in solution. That was the empirical observation. There was some critical supersaturation ratio and if he observed it, he did not get the animals in trouble with decompression sickness (DCS).

After looking at what factors were involved in inert gas uptake and elimination, he decided that handling all the variables was difficult. So he developed a mathematical model based on the half time equation. Haldane knew that there was an infinite number of curves that described the uptake and elimination of nitrogen in various regions of the body, depending on the combinations of fat and perfusion. Of course these rates may change with other variables such as cold and exercise. By choosing a number of time constants to put into the equation one can approximate to what is really happening in the body. That was his second concept.

The first decompression tables were built using the half times 5, 10, 20, 40 and 75 minutes, applied to the half time equation. As an example, if a diver went to a depth where the nitrogen pressure in compressed air was 4 atmospheres absolute for 40 minutes one can use this equation to calculate the amount of nitrogen that is taken up. A 5 minute half time after 40 minutes will have been exposed for 8 units of half time, so that tissue would be 100% saturated. It would have a nitrogen pressure of 4 atmospheres. A 10 minute tissue after 40 minutes would have been exposed to 4 units of half time so would be about 93.75% of 4 'atmospheres. The 20 minute tissue would be 75% saturated, it would hold 3 atmospheres of nitrogen pressure and so on. Using the empirical observation that a nitrogen pressure to barometric pressure ratio of 1.58 is safe, one can calculate the barometric pressure that the diver can safely ascend to. One multiplies the calculated nitrogen pressure by 1.58 which gives you the barometric.