U.S. AIR FORCE EXPERIENCE IN HYPOBARIC OSTEONECROSIS

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Hypobaric osteonecrosis has indeed occurred following military flight. Hodgson *et al.* (1968) presented this history from the files of Wilford Hall USAF Hospital:

> A 45-year-old B-29 crew member in combat was subjected to two episodes of rapid decompression to 35,000 and 45,000 foot altitudes within one week in 1953. On both occasions the aircraft was required to remain at altitude for an hour or more. He experienced pain . . . in several joints . . . and his symptoms required several days to resolve each time. After the second episode, he never was fully free of discomfort in the left hip joint. . . . In 1962, he was medically retired with a diagnosis of "aseptic necrosis of the left hip due to caisson [*sic*] disease. . . ."

By 1965, this man's hip required surgical repair, and the femoral head was replaced with a prosthesis. See Hodgson *et al.* (1968), Fig. 4.

From RAF experience the following case history was recorded by D. I. Fryer (1969): In about 1955, after 20 years of unpressurized aircraft flying, a 43-year-old photo-survey pilot suddenly suffered joint pains at 27,000 feet for a period of 6 hours. After he landed, he was confined to bed for 4 days suffering from severe pain in all his extremities. Between 1963 and 1967 he experienced 2 similar episodes, and on 6 other flights he had moderately severe shoulder and elbow bends. In May 1967 he experienced a sharp pain in his left shoulder associated with a "thunk" sound. Radiographs showed an impending "separation of a fragment from the head of the humerus." Six weeks later a jarring within this joint was distinctly heard and felt. The fragment had indeed separated.

Aside from J. H. Allan's (1945) claim of "calcific deposits and aseptic necrosis as predisposing factors in bends pain," these two cases apparently are the only specific accounts of disabling hypobaric osteonecrosis in the literature. They can be compared with 18 subatmospheric decompression sickness (DCS) fatalities (Fryer, 1969) in the USAF, RAF, RCAF, and USN from 1943 to 1958 (none since) and with 46 severe DCS cases successfully treated by compression, chiefly to 2.8 atm, between 1941 and the present time (as compiled by author J.C.D.). In both above-referenced cases, note that joint pain had persisted after landing, that there had been several such episodes, and that bone damage was found several years later.

Three reports (Ratnoff, 1943; Berry and Hekhuis, 1960; Hodgson *et al.*, 1968) exist dealing with roentgenography of bones and joints in men who in their regular duties were often exposed to simulated altitudes. The earliest (Ratnoff, 1943) concerned 21 military men who had undergone repeated exposures of 4 to 2 hours, or less, to altitudes of 35,000 and 40,000 feet. X-ray films of both hip joints were examined. "In none of the subjects were the roentgenographic changes described in caisson workers noted. Lesions of doubtful significance were present in two subjects, neither of whom had a history of bends."

In 1958, 623 USAF altitude-chamber men were X-rayed; the results on 579 of them were described by Berry and Hekhuis in 1960. Of this number, anterior-posterior views of the humeri, radii, ulnae, femorae, tibiae, and fibulae revealed not a single lesion that might be attributable to pressure changes. In the interim between 1958 and a follow-up study conducted in 1966 (Hodgson *et al.*, 1968), the X-rays of the additional 44 men were examined. An immature lesion was found in the midshaft of one right femur (see Fig. 2 of that study).

In their 1966 follow-up study, Hodgson *et al.* (1968) were unable to obtain X-rays on 459 of the entire sample. Although 291 of the original 623 men were still enlisted in the Air Force in December 1965 and 279 were in locations at which they could be reached, only 164 responded to the request for X-rays. In this second survey, it was found that the immature lesion mentioned

above had become "a mature intramedullary infarct" (see Fig. 1 of that study). It was 4 cm long and filled the entire thickness of the medulla. This man had a history of 6 attacks of bends.

In the 1966 study, as well, an additional case of aseptic bone necrosis was found in a man who had a history of 5 episodes of bends (see Hodgson *et al.*, Fig. 3). The lesion, 1 cm in diameter, was a round radiolucent area in the neck of the left humerus, just distal to the epiphysis. The lesion was "surrounded by a rim of increased bone density, and contained a few flecks of calcification. The 1958 X-rays are cut off just below the area of this lesion, so its onset cannot be documented."

Only 44 of the 164 respondents in the 1966 study had no history of DCS. Hence the *apparent* incidence of bony lesions may have been 2/120 among the altitude-chamber men who had suffered various mild forms of DCS, chiefly bends. To recapitulate, from 1958 to 1966 a lesion in one man had progressed to a mature medullary infarct of the femur. A lesion in a second man may or may not have been present earlier, but it was distinctly affecting the neck of the left humerus in 1966.

Even given the obvious deficiencies of the cited studies, it is possible to guess at the overall incidence of hypobaric osteonecrosis among men abruptly exposed to simulated high altitudes. From 1943 to 1966 the number was perhaps $(2+2)/(21+164)$, or 2.2% at the most. More likely the number could be as low as $(0+2)/(21+623)$, or 0.31%. Only the 2 fliers whose histories were given above have been disabled by hypobaric osteonecrosis; both of them had often suffered persistent postflight pain. They were fortunately not among the 18 known fatalities of subatmospheric DCS. On the other hand, they were unfortunately not among the 46 who were successfully treated from 1941 to date for severe reaction to hypobaric exposure.

In sum, disabling hypobaric osteonecrosis had indeed occurred — twice, surely. Initial X-ray films of 623 altitude-chamber men showed low incidences of bony lesions; it is difficult for obvious reasons to check an entire sample of that size 8 years later. Beginning in 1966 many USAF altitude-chamber operators have also been exposed regularly to 3 and 6 atm and are decompressed in exact accordance with U.S. Navy tables used in treatment of DCS. The 1966 USAF study was therefore biased toward a "last chance" attempt to find osteonecrosis in these operators before they began working in diving chambers as well. Because of this bias, only 2 cases of comparatively evident lesions were uncovered. Three cases of bone islands were excluded, plus one of endosteal scalloping, which could have yielded 6 cases, all told, out of 164 respondents in the 1966 study, had we chosen to use more liberal interpretative criteria at the time.

Of more importance, none of the original 623 men apparently suffered from or reported "postflight" symptoms of severe DCS. It is therefore felt that only those who have been afflicted with severe postflight reactions should be studied now and in the future. In other words, after treatment of such reactions by means of oxygen breathing at 2.8 atm, those individuals should be clinically observed in subsequent years.

The sequelae of severe DCS affecting the bones could thus be contrasted to the total of 46 incidences following subatmospheric exposure, recorded between 1941 and January 1972, that were successfully treated. Finally, it should be clear that the currently accepted treatment of altitude DCS persisting at ground level may prevent development of hypobaric osteonecrosis.

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DISCUSSION 2

Dr. ELLIOTT: Osteonecrosis is not just a question of X-ray changes. Some real problems are generated for the afflicted individual. I should like to cite a quotation from Alnor (1963), which may be of interest to Dr. Fairchild and his associates: "Of the 65 men kept under observation for more than 10 years in the German study, only 22 of them remained free of radiological evidence of lesions. Of the 43 with lesions, 17 had symptoms, and 7 were totally unable to work." So, for the men concerned, it is indeed a very real problem.

Dr. KINDWALL: I should like to ask Dr. Beckman what method of selection was used in picking the 27 divers of the Gulf of Mexico survey. Was it slanted toward men who indeed had symptoms? Was there anything other than pure random choice involved?

Dr. BECKMAN: There was no selection whatsoever.

Dr. HARRISON: I should like to congratulate Dr. Fagan on his survey and to reinforce one or two points that he made. He commented on how very subtle some of these early lesions are, and how very difficult it is to recognize them. He also stressed that the lesions are frequently multiple. His point is well taken, I think, that the frogleg position is rather an improvement over the British straight A-P X-ray of the hips.

Dr. Fagan also demonstrated something that has concerned many of us — *i.e.*, the association of these vague densities with what we now recognize as lesions. By "vague densities" I mean an area that one cannot with certainty identify as a bone island or accept as evidence of aseptic bone necrosis. Experience seems to indicate that these densities are certainly much more common in divers than in a comparable nondiving population.

Dr. Fagan also made the very relevant point that when one begins a roentgenographic survey and makes the initial readings, one does not want to know anything at all about the divers. The survey must begin as a blind reading. In later evaluations of the lesions, of course, some knowledge is helpful.

Dr. JONES: In our opinion there is a very definite association between metaphyseal lesions and irregular osteosclerotic lesions in epiphyseal regions. I think serial evaluation of the multiple features previously conceived of as "bone islands" may actually show that they are focal areas of necrosis.

Dr. WORKMAN: We have done 152 bone surveys on our own divers and on applicants seeking employment as divers; we have also done 10 planograms (tomograms) of these men. We made the routine films suggested by England's Medical Research Council.

Ten planograms were done of suspicious areas that we could not otherwise define. Ten of the 152 men studied had lesions of the head of the humerus. One was bilateral, with compaction of subchondral bone and disruption of cartilage. Three of these happened to be company divers and seven were diver applicants. Planograms were repeated on two subjects one year later; there was no perceptible change in the lesions in the mid-head of the humerus. As Dr. Fagan has shown, the lesions were radiolucent, sclerotic areas, with disruption or irregularity of the trabeculae. We found that planograms are extremely helpful in defining these things — much more so, I think, than the best cone spots we could get.

We refused 35 applicants for employment because of multiple bone lesions in which early stages of aseptic necrosis could not be excluded, or because of other hypertrophic joint changes that we felt were risky in active divers. Several of our older divers have been active for 10, 15, or perhaps 25 years; I am impressed that most have no lesions that we considered positive for osteonecrosis.

Dr. WALDER: We have done a survey of 230 professional civilian divers, men sent to us by an organization called the Construction Industry Research and Information Association. They were thus unselected by us. To date we have found 3 definite and 12 suspect positive lesions in the group.

Dr. ELLIOTT: That low incidence is very encouraging. I think we should remember that similar results have been reported in other surveys. One I would like to mention was reported by Graczyk (1970), who examined 67 Polish divers, all of whom had 10 or more years' diving experience. He found lesions in only 5 men, a 7% incidence. So there appear to be groups of divers in whom the incidence is less than that found in the majority of surveys. But the criteria of diagnosis in the Polish survey may not have been the same as those used by the Medical Research Council or the same as those used by the German authors.

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