

*Undersea & Hyperbaric Medicine, Vol. 21, No. 4, 1994*

## **Unexplained muscle swelling in divers**

**C. G. DAUGHERTY**

*Occupational Medicine Department, Neuroscience Center, Austin Diagnostic Clinic, Austin, Texas*

Daugherty CG. Unexplained muscle swelling in divers. *Undersea Hyperbaric Med* 1994; 21(4):425-429.—Muscle swelling in divers may represent a previously unreported form of decompression sickness. It shows marked, brawny swelling of skeletal muscle which clinically most suggests muscular lymphedema in its woody texture to palpation and unresponsiveness to recompression. Four episodes of unexplained muscle swelling in commercial divers are presented, none apparently resulting in muscular injury.

*decompression sickness, muscle swelling, lymphatic bends*

Although decompression sickness symptoms are commonly attributed to the joints and central nervous system, unusual cases of DCS such as localized extremity swelling have been described. Bert (1) included muscle pain in a list of symptoms of DCS. In a lengthy article in 1909, Keays (2) described almost 3,700 cases of compressed air illness in divers and tunnel workers in which several of the cases had localized extremity swelling. In 1945, Harvey (3) mentioned possible mechanisms of bubble formation in experimentally traumatized muscle but did not report actual cases of bends in uninjured muscle. None of these papers specifically describe or name DCS in muscle and, in more recent literature, there is no mention of this. Therefore, diving cases suggesting a possible form of DCS involving muscle tissue should be of clinical interest.

I present four cases of localized muscle swelling in commercial divers which occurred between 1977 and 1992. Three also had common symptoms of DCS, one had only muscle swelling. I do not know the cause of this swelling but two clinical characteristics suggest it could be muscular lymphedema.

### **CASE REPORTS**

#### **Case 1**

On 8 June 1977, a commercial diver finished "walking pipe" (inspecting a pipeline) in the Gulf of Mexico in 77.7 meters of seawater on mixed gas (CGD, unpublished). Sur-

face decompression (Sur-D—the diver completes certain water stops then is quickly brought to the surface, completing his decompression in a deck chamber) was uneventful until the chamber portion, when he noticed swelling of his right calf without pain, sensory changes, weakness, or other apparent abnormality. This was not thought significant, and decompression was completed. On deck, swelling was verified by measuring both calves with string. After radio discussion with the employer's medical consultant, he was recompressed on a U.S. Navy (USN) table 5 with no apparent change, then was held at the 30-ft stop while another physician flew to the site.

This evaluation revealed right calf circumference of 38.7 cm vs. 35.6 cm on the left. The skin on the right was normal to appearance and palpation and was not thickened or discolored. The underlying muscle was of very firm and regular texture with no palpable crepitus, no tenderness to palpation nor pain from forced dorsiflexion of the ankle or vigorous ankle movement, nor any edema of the foot or ankle. There was no sensory abnormality of the extremity nor any calf weakness. The balance of the physical exam was normal; the table 5 was completed without incident.

Because a prior episode in this diver had been suspected of deep vein thrombosis, he was given heparin and dexamethasone i.v. and placed on bed rest overnight. The next morning the right calf circumference was 39.4 cm with no other change. The diver was flown to a hospital where venography showed no abnormality of the deep vessels. On discharge the following day, the right calf measured 40.0 cm. A xeroradiograph of the calf 2 days later was also normal. By 21 July 1977, right calf circumference was 38.1 cm. The patient had been doing sports and strenuous activities without difficulty and was returned to diving without a specific diagnosis. By 28 September, both calves were 36.8 cm.

This diver stated he had the swelling in both calves 2 yr previously. Records from this incident showed he was evaluated for possible thrombophlebitis and pulmonary embolism, with negative results. He was told the problem did not seem decompression-related, but he was not given a diagnosis and soon returned to diving.

## Case 2

On 29 June 1978, a commercial diver was evaluated for a peculiar incident one week earlier (CGD, unpublished). On 22 June 1978, he made a dive on mixed gas to 84.7 msw to inspect pipeline valves. Decompression was uneventful; after exiting the chamber he noticed pain in both knees and was treated on a USN table 5 with total relief of pain. Shortly after this treatment, the diver noted painless swelling of his left calf. There was no apparent weakness or sensory abnormality; the most noticeable sensation was that of muscular engorgement, which he compared to having done "100 toe-ups."

The swelling persisted 2 days later, even after another uneventful dive and decompression; on Day 3 the diver received a USN table 6 with no apparent improvement. After the table 6 the diver immersed his foot and leg in ice water, which immediately reduced the swelling. The following day (26 June) he repeated this and the remaining swelling disappeared and did not recur.

Examination on 29 June revealed no abnormality. Both calves and ankles were of equal circumference and there was no tenderness, weakness, sensory changes, or visible abnormalities. Because of the strong resemblance to case 1 the diver and his supervisor were questioned closely but ultimately no diagnosis was made and he was allowed to continue diving.

### Case 3

On 11 January 1986, a commercial diver completed a mixed gas dive to 85.9 msw to perform inspection of a pipeline tie-in (CGD, unpublished). Decompression on a Sur-D schedule was uneventful until he reached the surface where, climbing the ladder, he noted weakness in his arms and shoulders. On deck there was a sudden, impact-like pain in both shoulders, so severe he had trouble removing his boots. The diver reported the shoulder pain, but not the weakness, and his supervisor interpreted this as type I DCS.

He was compressed to 15.2 msw (routine decompression depth). This reduced the shoulder pain to an ache, but the diver then noticed marked swelling of his left biceps and triceps. Upon learning this, the supervisor instituted a USN table 5 and compressed the diver to 18.3 msw. At this depth the remaining shoulder pain disappeared, but the arm swelling remained; the arm muscles were hard, felt "flexed," and were slightly sore to touch. The overlying skin appeared normal. One hour after completing the table 5, the shoulder pain recurred and worsened, new pain developed in the left lower extremity from the hip down and mild shooting pains from the left elbow to the wrist. The diver felt irritable and "antisocial." He was re-treated on a USN table 6.

All discomfort improved within 10 min, and by the end of the table all symptoms were gone except soreness in the posterior shoulder, slight tenderness in left triceps, and the unchanged muscular swelling. It was now the morning of 12 January 1986. The diver slept 4 h and felt generally well with only the swelling of his left arm. Examination on 14 January revealed brawny, firm swelling of the left biceps and triceps, minimal tenderness to palpation, and normal overlying skin. The left arm measured 34.5 cm and the right 32.25 cm (right hand dominant). There were no neurologic or general physical abnormalities. The diver was given table 5 four additional times during the next 2 days, without change; the muscular swelling gradually subsided. By 22 January 1986, the left arm measured 32 cm, and muscle texture was normal. A vague ache persisted for a few weeks, although the diver could work and lift weights without difficulty. On 22 February 1986, evaluation by a neurologist was negative and the aching had cleared. He was permitted to return to diving.

### Case 4

On 28 May 1992, a 36-yr-old commercial diver made a mixed-gas dive to 82.3 m for 22 min to burn steel; the dive was uneventful (CGD, unpublished). Near the end of the chamber portion of his Sur-D schedule, he developed pain in both wrists and the right knee, ankle, and shoulder. He exited the chamber briefly to urinate, and during this interval he noted the rapid onset of very firm swelling in his right deltoid muscle; the joint pains became markedly worse and there was paresthesia along the posterior right arm.

During an extended USN table 6 the joint pains and paresthesia cleared but the swelling increased, spreading to the trapezius muscle, right arm, and proximal ulnar forearm. After 6 h sleep, he received a US Navy table 5 with no change in the swelling. Repeated neurologic exams were normal after disappearance of the paresthesia. On 29 May a physician noted tense swelling of the deltoid, non-tender, causing difficulty in abducting the arm past 100°; there were no cutaneous abnormalities. By 2 June 1992, the swelling had completely subsided, and the rest of his follow-up evaluation was likewise normal; he returned to work on 15 June.

## DISCUSSION

Since marked muscle swelling is unlikely to escape detection, it must be unusual in clinical diving medicine and is not discussed in current standard references. In the early literature (1), Bert listed muscle pain as a symptom of DCS, and Bouchard wrote of "painful muscular swelling" in caisson workers, which responded to recompression. From the information given, these early cases do not seem to resemble the present ones.

In this century, publications from 1909 and 1945 mention possible muscle involvement in diving cases. The first, by Keays (2), presents data on 3,692 cases of compressed air illness, most with pain in various sites but no objective findings. Nine of these patients also had "local swelling": joint swelling, cutaneous and subcutaneous lymphedema, and subcutaneous emphysema of the extremities. Although Keays stated that pain can be caused by ". . . free gas beneath a tight fascia . . .," none of his cases, as described, showed muscular swelling.

In 1945, Harvey (3) lectured on the general topics of decompression sickness and bubble formation in blood and tissues. Part of the lecture centered around bubble formation in severely traumatized cat muscle after extreme altitude decompression. He also mentioned human x-ray studies in altitude chambers which showed ". . . air masses in joint cavities, in popliteal fat and in the fascia between muscles." There is no mention of DCS in untraumatized muscle or of muscle swelling after routine decompression.

The two notable clinical characteristics in these cases are the brawny, indurated swelling, clinically like lymphedema, and the failure of this swelling to respond to recompression. Localized edema in diving from bubble formation in lymph tissue has been studied (4-6), although Arturson and Grotte (6) described only "peripheral edema"; lymphedema in divers is usually discussed as it affects the skin (4, 5, 7, 8). It would probably require histologic study to determine whether the muscle swelling reported here is indeed due to lymphedema.

Failure of the swelling to respond to pressure is curious and surprising because, in diving, that which is caused by inadequate decompression usually responds to prompt recompression. However, this is typical of the cutaneous lymphedema sometimes seen in divers where the local burning, itching, and discoloration typically respond but the lymphedema itself does not, usually lasting a few days or more, regardless of treatment (7, 9, 10).

Arturson and Grotte (6) clearly showed bubbles in lymph vessels and glands and found no evidence that they and the edema were due to alterations in the blood/lymph barrier. They concluded: "Gas bubbles in lymphatic vessels and glands seem to cause a significant obstruction to lymph flow and must be an important factor in edema formation." However, an obstructing bubble should resolve with prompt recompression. Clinically, lymphedema might not clear as quickly as other types of edema (because lymph movement is relatively sluggish) but one suspects it should take less than days or weeks if all that is needed is to remove an offending bubble.

Although the swelling in these cases was clearly beneath the skin (except perhaps the forearm of case 4), these two points still seem to justify a clinical speculation that these cases may represent lymphatic DCS of muscle. Both lymphedema of skin and these four cases, spread over 17 yr of clinical diving medical practice, have been very uncommon occurrences. The only likely alternative cause would be trauma, but there is no information suggesting this. The divers and supervisors considered trauma (having never heard of postdive muscle swelling) but there was no work task or accident during the dives that would cause such sudden and noticeable swelling. It seems unlikely such an event would be overlooked or quickly forgotten; indeed, the swelling usually occurred at rest. Besides

having an obvious cause, trauma sufficient to produce rapid swelling of this magnitude should also cause definite pain and inflammation. In these cases, pain was usually absent and there was no heat or redness, not in keeping with traumatic muscular swelling.

One tantalizing detail is the witnessed improvement in case 2 from ice water immersion, after no response to pressure both in a chamber and in water. The latter would seem to eliminate hydrostatic pressure as a cure, leaving only temperature as a possible factor. Because 8 and 14 yr passed between this case and cases 3 and 4, respectively, this item was forgotten until those records were retrieved to write this paper; if future cases occur, ice water will be tried again.

In summary, localized muscle swelling in divers seems to be a rare event that is easily noticed. The swelling in these cases was benign, although there is an unpublished case of a diver with a compartment syndrome due to unexplained swelling of the extensor carpi ulnaris (C. Fife, personal communication, 1990). Where there is also obvious DCS of other tissues, as with three of these cases, treatment should be directed toward the major symptoms elsewhere; the muscle swelling does not respond to recompression (although perhaps to ice water) and subsides spontaneously over time without evidence of lasting harm. Where isolated swelling occurs without signs of DCS elsewhere, I recommend at least a USN table 5 to protect the diver from the physician's ignorance. Further information on the etiology of this swelling probably requires the opportunity to perform a muscle biopsy in a future case.

#### REFERENCES

1. Bert, P. *La pression barometrique*. Paris, Masson, 1878. Translated by M. Hitchcock and F. Hitchcock. Columbus, OH: College Book Co, 1943.
2. Keays F. *Compressed air illness with a report of 3692 cases*. Ithaca, NY: Department of Medical Publications, Cornell University, 1909:1-55.
3. Harvey E. Decompression sickness and bubble formation in blood and tissue. *Bull NY Acad Med* 1934, October:529, 534.
4. Elliott DH, Hallenbeck JM. The pathophysiology of decompression sickness. In: Bennett PB, Elliott DH, eds. *The physiology and medicine of diving and compressed air work*, 2d ed. Baltimore, MD: Williams & Wilkins, 1975:438.
5. Hallenbeck JM, Andersen JC. Pathogenesis of the decompression disorders. In: Bennett PB, Elliott DH, eds. *The physiology and medicine of diving*, 3d ed. San Pedro, CA: Best Publishing, 1982:435.
6. Arturson G, Grotte G. Mechanism of edema formation in experimental decompression sickness. *Aerosp Med* 1971; 42:58-61.
7. Elliott DH, Moon RE. Manifestations of the decompression disorders. In: Bennett PB, Elliott DH, eds. *The physiology and medicine of diving*, 4th ed. London: WB Saunders, 1993:488-489.
8. Edmonds C, Lowry C, Pennefather J. *Diving and subaquatic medicine*, 3d ed. Oxford: Butterworth-Heinemann, 1992:172-173.
9. Daugherty CG. *Field guide for the diver-medic*. Austin, TX: Coastal Aquatics Publications, 1992:93.
10. Daugherty CG. Comments on lymphatic bends. *Triage* 1988; 3:12.

