Pathology

Whales, sonar and decompression sickness

Arising from: Jepson, P. D. *et al. Nature* **425,** 575–576 (2003).

We do not yet know why whales occasionally strand after sonar has been deployed nearby, but such information is important for both naval undersea activities and the protection of marine mammals. Jepson *et al.* suggest that a peculiar gas-forming disease afflicting some stranded cetaceans could be a type of decompression sickness (DCS) resulting from exposure to mid-range sonar[1]. However, neither decompression theory nor observation support the existence of a naturally occurring DCS in whales that is characterized by encapsulated, gas-filled cavities in the liver. Although gas-bubble formation may be aggravated by acoustic energy, more rigorous investigation is needed before sonar can be firmly linked to bubble formation in whales.

On the basis of the available information, the DCS hypothesis of Jepson *et al.* contains two flaws. First, whales do not develop sufficient gas supersaturation in the tissues on ascent to cause extensive bubble formation in the liver. The gas available for supersaturation is limited to that present in the lungs at the onset of each held breath. During descent, the thorax is compressed[2], and the residual gas volume in the compliant lungs is forced, by Boyle's law contraction and alveolar collapse, into non-respiratory conducting airways, where it is sequestered from the circulation[3]. Not enough gas is taken up to produce bubbles, except possibly during multiple rapid dives to depths approaching that of the lung's closing volume[3].

Once nitrogen uptake is blocked by lung collapse, the partial pressure of nitrogen in the blood actually decreases for the rest of the dive as nitrogen is distributed to tissues[4]. However, nitrogen accumulation in the liver, intestines and other visceral organs is limited by the diving response, which directs arterial blood away from these organs to the brain and heart[5]. On ascent, bubbles leaving supersaturated tissues must enter the venous blood and return to the lungs. These bubbles cannot pass into the liver (except in the unlikely event that they are of portal origin) unless they bypass the lung, which serves as a bubble trap. Jepson *et al.* do not explain why DCS, if it did occur in the whales they investigated, should affect the liver disproportionately.

Second, large gas-filled cavities in the liver, many encapsulated in dense fibrous tissue, are inconsistent with the pathology of DCS in humans and other mammals in which the bones, joints, lungs and central nervous system are primarily affected. The liver is rarely involved, and never to the extent described by Jepson *et al.* in the cetaceans that they investigated, even when the body's gas burden is very large. Bubbles have been observed in hepatic sinusoids and in the portal vein, but large encapsulated bubbles have not been reported[6]. Liver lesions are equally unexpected in 'recurrent' DCS, in which chronic lesions are found only in the long bones and the central nervous system. It is unlikely that fibrotic hepatic lesions of the type described by Jepson *et al.*, which would require days or weeks to develop, could be caused by a brief exposure to sonar.

We agree with Jepson *et al.* that further investigation is needed, including an analysis of the composition of the gas in the bubbles. But we believe that identifying the cetacean gas disease with DCS is premature because its pathology not only differs from that underlying the syndrome in other mammals, but it also cannot be explained by any physiological mechanism related to diving.

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1. Jepson, P. D. *et al. Nature* **425,** 575–576 (2003).
2. Williams, T. M. *et al. Science* **288,** 133–136 (2000).
3. Houser, D. S., Howard, R. & Ridgway, S. *J. Theor. Biol.* **213,** 183–195 (2001).
4. Falke, K. J. *et al. Science* **229,** 556–558 (1985).
5. Kooyman, G. L. & Ponganis, P. J. *Annu. Rev. Physiol.* **60,** 19–32 (1998).
6. Francis, T. J. R. & Mitchell, S. J. in *Bennett and Elliott's Physiology and Medicine of Diving* (eds Brubakk, A. O. & Neuman, T. S.) 5th edn 530–556 (Saunders, Philadelphia, 2003).

Jepson et al. *reply* — We did not, as Piantadosi and Thalmann suggest[1], present our findings as conclusive evidence of decompression sickness (DCS). We stated neither that DCS occurs naturally in cetaceans, nor that exposure to active sonar increases its occurrence[2]. However, we restate that there is now a generally accepted link between some beaked-whale strandings and sonar use, and that lesions in some cetaceans demonstrate that *in vivo* bubble formation (embolus) can occur and persist.

Progressively increasing concentrations of nitrogen in cetacean tissues after repetitive diving have been studied empirically in bottlenose dolphins (*Tursiops truncatus*)[3] and higher levels are predicted for northern bottlenose whales (*Hyperoodon ampullatus*) on the basis of their rate of descent or ascent and depth of diving[4]. Nitrogen supersaturation could be further increased by an accelerated rate of ascent, possibly to a critical point where bubbles form.

Even if naturally occurring levels of nitrogen supersaturation in the tissues of diving cetaceans are normally insufficient to initiate bubble formation, a theoretical possibility remains that cetaceans with nitrogen-supersaturated tissues could experience bubble growth or formation as a result of intense acoustic exposure[4,5]. There was a clear spatial and temporal link with active naval sonar exposure in the case of the beaked whales in the Canary Islands, as well as in previously reported beaked-whale strandings[6,7].

The lesions in the Canary Island beaked whales and in the UK cases (mainly dolphins) differed. The beaked whales had acute, systemic and widely disseminated lesions consistent with, although not diagnostic of, DCS[8]. The large hepatic cavities found exclusively in the UK cases are atypical of DCS in humans and experimental animals. For logistical reasons, the central nervous system was examined in only two UK cases and the bones were not examined in any. We cannot therefore confirm or refute the presence of lesions consistent with gas embolism in bone or the central nervous system. However, large numbers of gas bubbles were seen in portal veins and sinusoids in the livers from all UK cases examined microscopically, consistent with DCS in humans[8].

As cetaceans differ from humans behaviourally (as obligate, repetitive breath-hold divers), physiologically (for example, in their diving reflex)[9] and anatomically (as in their retia mirabilia, large portal veins and diaphragmatic sphincters)[10,11], it may be too simplistic to assume that the distribution, severity and chronicity of lesions induced by gas emboli will be similar in both human divers and free-living cetaceans. Extensive sublethal bubble formation in human DCS is an acute medical emergency. Without medical intervention, a free-living cetacean suffering the same fate would continue diving for days, weeks or months afterwards unless death or stranding intervened.

Lesion pathogenesis in the stranded cetaceans[2] may ultimately be explained by bubble formation, possibly in response to either rapid decompression or acoustic exposure of nitrogen-supersaturated tissues[5]; however, it is not clear how marine mammals mitigate the accumulation of nitrogen gas while diving and defend themselves against nitrogen-bubble formation. These uncertainties do indeed argue for caution in interpreting the limited studies available.

A. Fernández*, M. Arbelo, R. Deaville, I. A. P. Patterson, P. Castro, J. R. Baker, E. Degollada, H. M. Ross, P. Herráez, A. M. Pocknell, E. Rodríguez, F. E. Howie, A. Espinosa, R. J. Reid, J. R. Jaber, V. Martin, A. A. Cunningham, P. D. Jepson
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1. Piantadosi, C. A. & Thalmann, E. D. *Nature* doi:10.1038/nature02527 (2004).
2. Jepson, P. D. *et al. Nature* **425,** 575–576 (2003).
3. Ridgway, S. H. & Howard, R. *Science* **206,** 1182–1183 (1979).
4. Houser, D. S., Howard, R. & Ridgway, S. H. *J. Theor. Biol.* **213,** 183–195 (2001).
5. Crum, L. A. & Mao, Y. *J. Acoust. Soc. Am.* **99,** 2898–2907 (1996).
6. *US Department of Commerce and US Navy Joint Interim Report* http://www.nmfs.noaa.gov/prot_res/overview/Interim_Bahamas_Report.pdf (2001).
7. Frantzis, A. *Nature* **392,** 29 (1998).
8. Francis, T. J. R. & Mitchell, S. J. in *Bennett and Elliott's Physiology and Medicine of Diving* (eds Brubakk, A. O. & Neuman, T. S.) 5th edn 530–556 (Saunders, Philadelphia, 2003).
9. Ridgway, S. H. in *Mammals of the Sea: Biology and Medicine* (ed. Ridgway, S. H.) 590–747 (Thomas, Springfield, Illinois, 1972).
10. Harrison, R. J. & Tomlinson, D. W. *Proc. Zool. Soc. Lond.* **126,** 205–233 (1956).
11. Simpson, J. G. & Gardener, M. B. in *Mammals of the Sea: Biology and Medicine* (ed. Ridgway, S. H.) 298–418 (Thomas, Springfield, Illinois, 1972).