Editorial

DCS or DCI? The difference and why it matters

There are few issues that generate as much confusion in diving medicine as the nomenclature of bubble-induced dysbaric disease. Prior to the late 1980s, the diagnosis 'decompression sickness' (DCS) was invoked for symptoms presumed to arise as a consequence of bubble formation from dissolved inert gas during or after decompression. These bubbles were known to form within tissues, and also to appear in the venous blood (presumably after forming in tissue capillaries). A second diagnosis, 'arterial gas embolism' (AGE) was invoked for symptoms presumed to arise when bubbles were introduced directly to the arterial circulation as a consequence of pulmonary barotrauma.[1]

This approach was predicated on an assumption that the underlying pathophysiology could usually be inferred from the nature and tempo of resulting symptoms. DCS was considered to exhibit a slower more progressive onset, symptoms were protean (including pain, rash, paraesthesias, subcutaneous swelling, and neurological symptoms), and the neurological manifestations were mainly attributable to spinal cord or inner ear involvement. In contrast, AGE was considered to exhibit a more precipitous onset (often immediately on surfacing), and the principal manifestation was stroke-like focal neurological impairment suggestive of cerebral involvement.

In 1989 an association between a large persistent ('patent') foramen ovale (PFO) and serious neurological DCS was independently reported by two groups,[2,3] and subsequently corroborated for neurological,[4–8] inner ear,[6,9] and cutaneous[10] DCS by multiple studies. The assumed pathophysiological role of a PFO in this setting was to allow bubbles formed from inert gas in the venous blood to avoid removal in the pulmonary circulation and to enter the arterial circulation. These bubbles could then pass to the microcirculation of vulnerable target tissues where inward diffusion of supersaturated inert gas from the surrounding tissue could cause them to grow.[11]

This emergence of 'arterialisation' of venous bubbles as an important vector of harm in some forms of DCS resulted in a challenge to the use of traditional 'DCS/AGE' terminology. It was suggested that very early onset of cerebral symptoms after diving could be explained not only by arterial bubbles introduced by pulmonary barotrauma, but also by venous bubbles crossing a PFO into the arterial circulation. Moreover, once venous bubbles had entered the arterial circulation they were then technically 'arterial gas emboli'; thus creating confusion with arterial gas emboli from pulmonary barotrauma. To many commentators, it made little sense to use diagnostic labels (DCS and AGE) that implied a particular pathophysiology when the two disorders might be difficult to tell apart, and had mechanistic processes in common.

An alternative approach derived at a UHMS workshop in 1991 was to shift from nomenclature that implied a particular pathophysiology, to a descriptive system that lumped both DCS and AGE together under the label "*decompression illness*" (DCI).[12] Using this system, terms to describe the organ system(s) involved and the progression of symptoms were applied. For example, a diver with worsening upper arm pain after a dive could be suffering 'progressive musculoskeletal DCI'; and a diver who lost consciousness immediately on surfacing but regained consciousness minutes later would be considered to be suffering 'remitting cerebral DCI'. Classifying cases in this manner made considerable sense at a clinical level, particularly given that there was an emerging consensus that manifestations of DCS and AGE that potentially overlapped did not require different approaches to recompression treatment.

This descriptive classification of bubble-induced dysbaric disease gained substantial traction in the community, though not always with a full appreciation by users of the intended nuances of its application. Indeed, it became increasingly common over time to see the terms DCS and DCI used interchangeably; for example, authors using the term DCI to specifically infer the consequences of bubble formation from dissolved gas. This highlights one of the shortcomings of the DCI terminology: it becomes confusing when discussing dysbaric disease at a theoretical or experimental level when the nature of the insult is known or there is a specific intent to discuss bubble formation either from dissolved gas or from pulmonary barotrauma.

The potential for confusion between mechanisms and manifestations of DCS and AGE as one of the principle drivers for adopting the DCI terminology deserves further discussion. It is tempting to suggest that if venous bubbles cross a PFO into the arterial blood then any resulting symptoms should be considered a manifestation of 'AGE'. However, there seems little sense in re-naming the primary pathophysiological event (DCS caused by bubble formation from inert gas) just because the bubbles have distributed elsewhere; especially using a name that commonly infers a completely different primary event (bubble formation from pulmonary barotrauma). Moreover, there are grounds for suggesting that these two processes may not be as difficult to distinguish as previously believed. Venous inert gas bubbles are small,[13] and of a similar size distribution to those used as bubble contrast during PFO testing.[14] Decades of experience in testing thousands of divers (and other patients) for PFO using bubble-contrast echocardiograpy have shown that even when strongly positive (that is, large showers of bubbles enter the arterial circulation), symptoms of any sort are

very rare. There are sporadic reports of evanescent visual or cerebral symptoms, but (to this author's knowledge) reports of the focal or multifocal cerebral infarctions that can be caused by large arterial bubbles introduced iatrogenically or by pulmonary barotrauma are lacking. One could argue that in the context of PFO testing the brain is not supersaturated with inert gas (which might cause small arterial bubbles to grow), but being such a 'fast tissue' nor is it likely to be after diving.[11] Thus, while sustained showers of small inert gas bubbles crossing a PFO after diving appeal as a plausible cause of transient visual symptoms or dysexecutive syndromes after diving, they are less likely to be the cause of dramatic stroke-like events occurring early after surfacing.

In the final edition of Bennett and Elliott it was suggested that one editorial approach to the terminology conundrum would be to utilise the traditional terminology (DCS and AGE) when referring specifically to the pathophysiology and manifestations of bubble formation from dissolved inert gas or pulmonary barotrauma respectively, and to utilise the descriptive (DCI) terminology in clinical discussions when a collective term is useful, or when discussing individual patients where there is either ambiguity about pathophysiology or no need to attempt a distinction.[1] Diving and Hyperbaric Medicine recommends a similar approach. The journal is reluctant to attempt to generate or apply hard 'rules' in relation to terminology of bubble-induced dysbaric disease, but we strongly discourage use of the term 'arterial gas emboli(ism)' to characterise venous inert gas bubbles that cross a right-to-left shunt such as a PFO. The pathophysiological consequences of bubble formation from dissolved inert gas should be regarded as decompression sickness (DCS). There is an expectation that authors are cognisant of the above issues and attempt to adopt terminology that reflects these considerations and best suits the circumstances of their manuscript.

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