IPE, SDPE, PESD: What's in a name?

I was quite entertained to read the clashing commentaries by Dr. Mike Davis [1] and Dr. Carl Edmonds [2] regarding immersion pulmonary edema (IPE). Much of their disagreement centers on nomenclature for the condition and whether IPE warrants splitting into "scuba divers pulmonary edema" (SDPE) and "swimming-induced pulmonary edema" (SIPE).

In 1997, Richard Dunford and I published only the third description of the syndrome, reporting seven afflicted North American scuba divers [3]. We referred to it as "pulmonary edema of scuba divers" (PESD). Surprisingly, I have not been contacted for my opinion on this most important issue. If Drs. Davis and Edmonds do not want to use a name with historical context and no one can think of a more easily pronounceable acronym, I'm for IPE with tongue firmly in cheek.

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References

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- 3. Hampson NB, Dunford RG. Pulmonary edema of scuba divers. Undersea Hyperb Med 24(1):29-33.

TBI study questioned: Dr. Gottlieb

I was pleased, surprised and upset that a special issue of the (*UHM*) Journal (Vol 43, No 5, 2016) was devoted to one study concerning the role of HBO₂ in treating TBI.

The pleasure was to see this important subject finally beginning to be being dealt with seriously by the Society; surprised that an entire issue was devoted to this study when most of the published material really was not worthy of publication as separate and distinct papers but could have been dealt with more efficiently

by archiving them and then referencing them and using the space for other research or clinical studies; upset because of what I perceived to be the poor quality of science and medicine associated with this fabulous multimillion dollar opportunity of doing a randomized, double-blinded and sham-controlled study. The concept of doing such a study was a noble desideratum. Unfortunately, it was poorly designed and, from the description of what was done, may even have indicated a lack of knowledge of treating neurological disorders with hyperbaric oxygen (HBO₂) therapy.

Despite the great physical details obtained on the recruited subjects there was no clear statement as to what medications these subjects had been exposed that could have altered the outcome(s). For example, were any of the subjects ever given mefloquine, an antimalarial drug known to cause symptoms similar to PTSD? The symptoms can last for years after the patients stop taking the drug [1], and the associated adverse effects may not necessarily have been amenable to HBO₂ therapy: although not necessarily related, it should be noted that the adverse effects of mefloquine were not responsive to the usual PTSD treatments.

Two of the primary objectives of the study were:

- "to identify endpoints for future efficacy trials of potential treatments for post-concussive syndrome" and
- 2. "to explore potential associations between changes in brain function, anatomy and participant reported outcomes"

Yet, the design of the study and the techniques employed could never permit these aims to be realized. Despite the detailed functional testing there was no direct evidence of comparative before and after therapy changes in the injured brain areas. The authors were aware of this limitation and, therefore, they had to be aware that they could not meet their objective . . .

"Despite the prevalence of TBI, standard-of-care treatments for mTBI and post-concussive syndrome are lacking...One of the challenges of testing potential interventions in this population is the lack of a reliable and valid set of outcomes for measuring deficits and treatment effect in the mTBI population" (pg. 615).