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Special article

Sudden death of non-cardiological origin during exercise. The dysbarism model☆

Muerte súbita de origen no cardiológico durante el ejercicio. El modelo del disbarismo

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Every now and then, it is important to review episodes of Sudden Death in the context of sports activity. In this sense, the recent article published by Sitges and Brugada fulfils that objective[1]. It may be an appropriate occasion to review other sudden death mechanisms during sport or professional physical exercise due to a non-cardiological origin.

Activities involving significant changes in the main environmental conditions – pressure, temperature and humidity – introduce compensating mechanisms of high complexity that may be, in themselves, the cause of an unexpected outcome. Compensating mechanisms of intrinsic origin, not necessarily from cardiological origin, also occur in mountain climbing, speleology, desert survival, and other extreme environmental exercises. More specifically yet, the dysbaric conditions experienced by aviators, astronauts, compressed air workers, and to a greater extent by divers, constitute a special model known as *Dysbarism*.[2] From a quantitative point of view, the most significant, modifications happen in SCUBA-Diving, as responsible of the greatest morbidity. However, this may be the cause of an inadequate risk assessment in each of these activities. The explanation lies in the fact that, while these accidents are generally fatal, in the case of underwater dysbaric disorders a whole set of preventive and therapeutic measures can lead to possibility of survival, following specific guidelines within the so-called *Diving Medicine*,[3] that permits a greater study of these mechanisms.

The incidence of serious accidents in SCUBA diving has sharply declined in recent years, and fortunately dysbarism figures are currently far from what was reported some decades ago.[4] However, in the Mediterranean countries, a new factor of high concern has emerged: the sudden death of middle or advanced age divers, after apparently normal dives, in which no obvious factor was detected as the responsible cause. These worrying phenomena are observed in both modalities, SCUBA and *Breath-Hold Diving* also incorrectly called Free-diving. The mechanisms that may explain these accidents do not have a cardiac problem as their main cause, but a heart failure is the final result of a previous chain of events.

Strictly speaking, no human form of life is really adapted to an aquatic environment. Only our distant relatives, the marine mammals, have achieved a permanent adaptation with a prolonged apnoea time, a large reserve of myoglobin, changes in lung volume and structures, and a significant slowing of their heart rate. This allows them regular and continuous immersion periods at great depths.

In some cultures, far from our own, the exploitation of marine resources has made the practice of an extreme form of Breath-hold diving a routine daily activity based on their own resources, and with overexploitation of the physiological limits.[5] But even in such cases we cannot correctly speak about a real mechanism that allows an acclimatization to the aquatic environment. Neither can be explained from the famous breath-hold divers with apnoea times up to 5 min and reaching more than 200 msw of depth. The human being is only able to develop, at most, a few aquatic survival procedures, which in no way can be considered as a form of adaptation, or even not acclimatization.

The characteristics of the aquatic environment limit underwater immersion to voluntary apnoea, during some few minutes. In SCUBA or semi-autonomous diving, the use of everyday more sophisticated respiratory devices, combines pressurizing of the respiratory media, formerly compressed air, with the use of synthetic breathing gas mixtures with variable oxygen percentage and an inert gas which is not necessarily nitrogen.[6] This allows a relatively prolonged stay under water maintaining a cardiorespiratory

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function in acceptable conditions for relatively long periods of time.

Under these conditions, human physiology faces a real challenge in maintaining acceptable hemodynamic conditions within a denser medium, anaerobic for humans, and subjected to a significantly higher pressure. The fact of breathing air – or other gas – at increased pressure causes by itself significant but silent ventilatory changes that the diver does not perceive as long as his/her respiratory dynamics are maintained in apparently normal conditions.

The SCUBA diver must exhale the air through a valve that close the respiratory circuit during inspiration, and open it at the beginning of expiration. The so-called *Diving-Regulator* allows the expert diver to maintain normal ventilatory dynamics, although reversing the phases of the respiratory cycle. Certainly, the diver makes an almost passive inspiration, while the expiration becomes a voluntary and forced movement. The rhythmic and voluntary sequencing of both phases is one of the requirements for the expert skill that allows an adequate and apparently physiological respiratory cycle.

The inexperienced Breath-hold diver ignores that his/her lungs are suffering an unavoidable mechanism of pressure-induced pulmonary collapse, that reduces up to 50% its volume at a depth of 10 msw, or at a quarter part when 30 msw are reached.[7] But this does not happen in SCUBA divers whose respiratory devices allow a correct rhythm, without changes of volume, always conditioned to breathing a respiratory mixture at the same environmental hydrostatic pressure. This mechanism is apparently physiological, and the expert diver does not appreciate any significant change.

However, at the end of their underwater activity, some divers experience a tiredness which is not proportional to the physical activity. This has been the reason for some surprises and alarm since the beginning of this activity. Some studies have shown that the energy discharge of a normal and shallow depth immersion, even with minimal physical activity, implies a metabolic response comparable to that of a long or medium intensity exercise.[8] The answer to this dilemma follows several mechanisms.

The pressurized air is proportionally denser, which conditions the amplitude of the thoracic movements and increases the respiratory work without the subject being aware of that.[9–11] The increase of thermal loss during immersion is about 25 times higher than in the air due to the greater activation of both thermic convection and conduction mechanisms.[12] Even in a tropical environment, where dives are performed at water temperatures in the surface close to 30 °C, a prolonged water immersion with poor isothermal protection will lead to the state of hypothermia. The survival time depends on water temperature and on the efficacy of the neurovegetative mechanisms of thermoregulation which maintain during a limited period of time the necessary homothermic life conditions.[13–15] Underwater breathing at high pressure adds another important respiratory thermal dissipation through evaporation, proportional to the density of the air, which is in direct relation to the increase of absolute pressure.

During the return to atmospheric normality after a high pressure respiratory incursion, either in SCUBA diving or another activity under pressure, the well-known *Decompression Sickness*, active but silent in most cases, may occur. During some hours after surfacing, the asymptomatic diver experiences a complex phenomenon of multifocal gas polymicroembolism that triggers a chain of hemodynamic and rheological disorders with hypovolemia, haemoconcentration, and compensated consumption coagulopathy, which in their smallest dimension, remains hidden.[16,17] Only under great dysbaric stress, do these mechanisms become important producing a real systemic disorder whose leading manifestation is the so-called *dysbaric shock*. In the case of pilots and astronauts, the same mechanisms of slow depressurisation occurs during the ascent phase, or abruptly if an *Explosive Decompression* happens due to a failure in the aircraft's pressurizing devices, which has been sometimes the cause of space tragedies.

But in Breath-hold diving, during the few minutes the diver is able to remain underwater, the main conditioning factor is hypoxemia, as well as progressive hypercapnia.[18] When the hydrostatic pressure is very high, the pulmonary collapse is almost total, and implosion can affect rigid intrathoracic structures. Until few years ago, this was considered as an insurmountable barrier that only aquatic mammals could surpass thanks to unknown mechanisms.

One of them is the flooding of the respiratory system by liquids coming from a vascular system that remains functioning with apparent hemodynamic normality under conditions of very high surrounding pressure. It is a kind of pulmonary by-pass, whose English name is widely used, without translation, by scientists of the whole world independently of their mother language. The so-called *Blood Shift*, unknown until recently, warrants the protection of respiratory structures subjected to extreme pressure, something that was considered unrealistic only a few years ago.[19] It was thought that these mechanisms were unique to aquatic mammals, but we currently do know that humans can also experience that, although only occasionally, unlike our ancestors, who use them continuously. Furthermore, in deepest Breath-hold diving, a transient splenic discharge increases the circulating blood volume in order to enhance the oxygen reserve.[20,21]

Under these conditions, the mechanism of returning to normality is more complex than its protective establishment. The reposition to their place of fluids that have transiently occupied an unusual space, only in order to keep intact the intrathoracic structures can cause, if not done properly, that part of the fluids remain in such areas not involved in respiratory function in normal atmospheric conditions.

All these measures suggest that the so-called Pulmonary Oedema of Immersion, considered as a strange phenomenon and rarely observed in human underwater activities, takes on a more important role than suspected.[22–26]

Episodes of sudden death following aquatic activities usually occur in middle or advanced age people, who dived under cold or even warm, but not hot, waters to which, for some reason, they have not managed to efficiently adapt. They had not suffered dyspnoea, or water inhalation, or tiredness, or excessive exercise, and their decompression procedure was normally followed. But from time to time, one of these divers manifest, after emersion, an unusual discomfort or general malaise, followed by a consciousness impairment and, in some cases, a quick onset of a critical emergency in front of his/her astonished fellow divers, ignorant of the tragedy that is about to happen. For this reason, emergency measures are usually not adopted until it is too late. Of course, the heart has a role on these mechanisms, but this occurs as a final result of a previous chain of hemodynamic disorders. The heart suffered the consequences, but it was not the cause.

Fortunately, and differently to what may be thought, research in diving medicine is deep, sophisticated, and varied. The goal is not only the interest in protecting the health of the divers, although, fortunately, institutions such as the DAN-EUROPE Foundation are devoted to this activity, carrying out a noble research in leisure diving.[27] But the powerful Oil Industry, which requires extraction at great depths with underwater human participation, is forced to develop sophisticated technology whose complexity exceeds by so far, the aerospace biomedical research. It is more difficult to have a human being working underwater at more than 500 msw,[28,29] than to keep an astronaut during extravehicular activities or a moon ride, since his/her space suit is a microhabitat that allows a close-to-normal physiology provided their devices work as expected.

Despite all the above, SCUBA diving is a safe activity, and the number of accidents in the aquatic environment is lower than those developed within other common activities considered to be healthy. In the past, insurance companies excluded underwater activities coverage because they considered diving too dangerous. But currently they realized their error, and this activity has been converted in one of their most lucrative preferential targets given its low accident rate. But these good news are counteracted by a high mortality and fatality. While the likelihood of a dysbaric accident is low, its severity can be important.

We have managed to reduce the number of dysbaric accidents to very low figures and, if they occur, the therapeutic facilities at our disposal can solve satisfactorily even the most serious cases.[30] We have the challenge now to make innocuous a pleasant activity such as SCUBA diving, in spite of the fact that it necessarily induces important biological survival mechanisms, that can be qualified as a kind of *Physiologically Pathological* status. That is to say, the subject whose physical activity takes place under dysbaric conditions, even in his/her utter ignorance, develops a series of survival mechanisms, which, if they were not the result of a physiological requirement to deal with adverse and imperative environmental conditions, they could be properly considered as a pathologic event in the terrestrial and atmospheric environment own of the habitat for which we have been conceived.

Conflict of interest

The author declares no conflict of interest.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.medcle.2017.02.024.

References

1. Sitges M, Brugada J. Muerte súbita en el deportista. Med Clin (Barc). 2016;147:540–2.
2. Desola Alà J. Enfermedades disbáricas. Disbarismos. Rev Clin Esp. 1995;195:741–3.
3. Desola Alà J. ¿Medicina subacuática? Med Clin (Barc). 1990;94:377–80.
4. Desola J, Bohe J, Garcia A, Abós R, Canela J. Prognostic factors of dysbaric disorders. Evidence based conclusions after a multivariant analysis of 554 cases. In: Cali-Corleo R, editor. Proceedings of the XXVI annual scientific meeting of the European Underwater and Baromedical Society (EUBS). 2000. p. 17–23.
5. Kohshi K, Wong RM, Abe H, Katoh T, Okudera T, Mano Y. Neurological manifestations in Japanese Ama divers. Undersea Hyperb Med. 2005;32:11–20.
6. Escrich E, Solanas M, Desola J. Fisiología de la respiración en ambientes especiales. In: Tresguerres JAF, editor. Fisiología humana. 3.ª ed. McGraw Hill; 2005. p. 663–72.
7. Desola Alà J. Trastornos en medio acuático y subacuático. In: Farreras P, Rozman C, editors. Medicina interna. 18.ª ed. Barcelona: Elsevier; 2016. p. 2508–11.
8. Desola J, Balague A. Gasometric, biochemical, and metabolic variations detected after SCUBA-diving. In: Van Liew HD. Amsterdam: Joint Meeting on Diving & Hyp Med; 1990. p. 87–8.
9. Peacher DF, Martina SD, Otteni CE, Wester TE, Potter JF, Moon RE. Immersion pulmonary edema and comorbidities: case series and updated review. Med Sci Sports Exerc. 2015;47:1128–34.
10. Earing CM, McKeon DJ, Kubis HP. Divers revisited: the ventilatory response to carbon dioxide in experienced SCUBA divers. Respir Med. 2014;108:758–65.
11. Boussuges A, Pontier JM, Schmid B, Dussault C. Paradoxical gas embolism after SCUBA diving: hemodynamic changes studied by echocardiography. Scand J Med Sci Sports. 2014;24:111–6.
12. Gerth WA. On diver thermal status and susceptibility to decompression sickness. Diving Hyperb Med. 2015;45:208.
13. Soteras I, Subirats E, Oliver R. Hipotermia accidental. Med Clin (Barc). 2011;137:171–7.
14. Shattock MJ, Tipton MJ. 'Autonomic conflict': a different way to die during cold water immersion. J Physiol. 2012;590 Pt 14:3219–30.
15. Iyer A, Rajkumar V, Sadasivan D, Bruce J, Gilfillan I. No one is dead until warm and dead. J Thorac Cardiovasc Surg. 2007;134:1042–3.
16. Desola Alà J. Accidentes de buceo (1). Enfermedad descompresiva. Med Clin (Barc). 1990;95:147–56.
17. Desola J. Enfermedad por descompresión. Jano. 2008:43–51.
18. Ferretti G, Costa M, Ferrigno M, Grassi B, Marconi C, Lundgren CE, et al. Alveolar gas composition and exchange during deep breath-hold diving and dry breath holds in elite divers. J Appl Physiol. 1991;70:794–802.
19. Ferrigno M, Lundgreen C. Breath-hold diving. In: Brubakk AO, Neuman TS, editors. Physiology and medicine of diving, vol. 153, 5th ed. Saunders, Elsevier; 2003. p. 180.
20. Schagatay E, Richardson MX, Lodin-Sundström A. Size matters: spleen and lung volumes predict performance in human apneic divers. Front Physiol. 2012;3:173.
21. Palada I, Eterovic D, Obad A, Bakovic D, Valic Z, Ivancev V, et al. Spleen and cardiovascular function during short apneas in divers. J Appl Physiol. 2007;103:1958–63.
22. Louge P, Coulange M, Beneton F, Gempp E, Le Pennetier O, Algoud M, et al. Pathophysiological and diagnostic implications of cardiac biomarkers and antidiuretic hormone release in distinguishing immersion pulmonary edema from decompression sickness. Medicine (Baltimore). 2016;95:e4060.
23. Gempp E, Louge P, Henckes A, Demaistre S, Heno P, Blatteau JE. Reversible myocardial dysfunction and clinical outcome in SCUBA divers with immersion pulmonary edema. Am J Cardiol. 2013;111:1655–9.
24. Edmonds C, Lippmann J, Lockley S, Wolfers D. SCUBA divers' pulmonary oedema: recurrences and fatalities. Diving Hyperb Med. 2012;42:40–4.
25. Fraser JA, Peacher DF, Freiberger JJ, Natoli MJ, Schinazi EA, Beck IV, et al. Risk factors for immersion pulmonary edema: hyperoxia does not attenuate pulmonary hypertension associated with cold water-immersed prone exercise at 4.7 ATA. J Appl Physiol. 2011;110:610–8.
26. Mahon RT. Exploring the depths of immersion pulmonary edema. J Appl Physiol. 2011;110:589–90.
27. Cialoni D, Pieri M, Balestra C, Marroni A. Flying after diving: in-flight echocardiography after a SCUBA diving week. Aviat Space Environ Med. 2014;85:993–8.
28. Lafay V, Barthelemy P, Comet B, Frances Y, Jammes Y. ECG changes during the experimental human dive HYDRA 10 (71 atm/7200 kPa). Undersea Hyperb Med. 1995;22:51–60.
29. Gardette B, Delauze HG. Les techniques d'intervention subaquatique: moyens, méthodes, recherches et perspectives. Bull Acad Natl Med. 1996;18:975–83.
30. Desola J. Celerius, profundius, periculosius. Med Clin (Barc). 1995;104:739–41.