LETTERS TO THE EDITOR

Absence of circulating PGI$_2$ response to bubble-provoking decompression

Bubble formation following decompression can damage vascular endothelium (1). A mechanical trauma to human blood vessels in vivo causes local release of anti-aggregatory prostacyclin (PGI$_2$) into the circulation (2, 3). Decompression is associated with abnormalities of platelet functions (4) and we investigated the possibility that this is caused by PGI$_2$ release into circulating blood ocasioned by the physical stimulus of intravascular bubble formation.

Three healthy men aged 25, 28, and 29, who were participating in a diving program, were studied. They had taken no medicines for 2 wk before the study and consented to giving 4 samples of venous blood. These were obtained from an antecubital vein immediately before, immediately after, 1 h, and 6 h after a simulated dive in a compression chamber. The pressure was increased over 3 min to 2.8 bar absolute (simulated depth 18 m). It was maintained for 75 min, before returning to atmospheric pressure over 1 min. Bubble detection was carried out using a 2 MHz probe; scoring was according to the established criteria of Spencer et al. + +, +++, and bubbles were detected over the precordium and inferior vena cava in 2 of the 3 subjects, 30–70 min after decompression. The white cell count in each subject rose after 6 h decompression 5.0 to 7.0, 5.1 to 9.1, and 4.0 to 7.0. This compression-decompression profile has been extensively studied by us and been found to reliably produce bubble formation in most subjects and minor symptoms in a few; in this instance, 1 subject reported joint pains postdive requiring treatment several hours after the exposure. PGI$_2$ was determined as its stable hydrolysis product 6-oxo-prostaglandin (PG)F$_{6}$ in using a highly sensitive and specific method based on gas chromatography-negative ion chemical ionization mass spectrometry (5). Concentrations of 6-oxo-PGF$_{6}$ in plasma from all subjects at all 3 times were at or below the detection limit of the assay (1 pg/ml, based on 5 ml of plasma).

We conclude that decompression sufficient to cause bubbles in the circulation, a rise in peripheral white blood count, and symptoms in 1 of 3 subjects did not increase circulating PGI$_2$ to levels where effects on platelets would be anticipated. We hope that the brief publication of this negative result will be of assistance to other workers contemplating research in this area.

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Long-term health consequences of diving accidents

An epidemiologic study which can tie together data on military diver health-performance history with information on previous or subsequent diving-related accidents (e.g., 1, 2) has potential value in assessing the adequacy of physical standards for diving as well as measures for preventing such accidents from occurring in the first place.

A recent report in Undersea Biomedical Research (3) addresses the hypothesis that there may be a relationship between a case of decompression sickness (DCS) in a U.S. Navy diver and subsequent hospitalization or medical board (medical-administrative evaluation for continuance on active duty) for whatever reason and perhaps many years later. The retrospective cohort study design (following a group of persons from a documented point of time in the past until a later event or outcome) that was used is a valuable epidemiologic method. However, important questions are still left unanswered in this report.

Many factors not examined and unrelated to diving may have independently affected the probability of the hospitalization or medical board occurring in the intervening years since DCS. These include total years and types of Navy diving done; history of recreational diving; overall health status, as perhaps measured by periodic multiphasic testing; patterns of physical exercise and cardiopulmonary fitness; sick call utilization behavior and for what illnesses; smoking and alcohol consumption; sociologic factors, e.g., divorce, death in the family; and degree of risk-taking behavior in general.

Additionally, after DCS occurred, other types of Navy duties could have provided different worksite exposures—physical exertion, liquid or gaseous chemicals, psychologic stresses—and, again, could have independently caused the hospitalizable illness or medical board.

Examination of these data, if they are available to the author, could lend more credence to the reported results and hypothesized association between DCS and later health risks.

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