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CO-EXISTENCE OF LIPID AND GAS EMBOLI IN
EXPERIMENTAL DECOMPRESSION SICKNESS

A.T.K. Cockett, M.D., S.M. Pauley, M.D., J.C. Saunders, M.D.
and F.M. Hirose, M.D.

FACILITY FORM 602

N69-36741 (ACCESSION NUMBER)	
9 (PAGES)	1 (THRU)
CR-105751 (NASA CR OR TRX OR AD NUMBER)	04 (CODE)
	04 (CATEGORY)

From the Departments of Surgery/Urology, Harbor General Hospital, Torrance,
California, 90509 and UCLA School of Medicine, Los Angeles and the
Division of Urology, University of Rochester School of Medicine,
Rochester, New York.

Supported in part by Contract NRO-0014-66-CO 295 (NR 102-669) between
the Office of Naval Research, Department of the Navy and Harbor General
Hospital, and a grant from the National Aeronautics and Space

Administration ~~NG 237-62~~ NGA-05-007-003

UCLA California

A traditional concept of pure nitrogenous bubble embolization has arisen as the etiologic factor in decompression sickness. Quite properly recompression has been employed as the means of treating this syndrome. The results of treatment were usually dramatic and effective.

In 1961, we emphasized the importance of hemoconcentration in altitude type bends cases and suggested the extent of the plasma deficit based on standard clinical data.¹ Subsequent studies by our group employing mongrel dogs previously splenectomized documented the extent of this plasma loss.² Dextran (LMW) replacement was effective in treating shock and maintaining an effective circulating blood volume.^{3,4} Recompression was deliberately withheld.

These early findings suggested to us that the syndrome of decompression sickness was more complex than the simple bubble theory. LMW dextran reverses blood viscosity caused by fibrinogen by formation of a dextran-fibrinogen complex. This role is in addition to its colloidal, expansive properties. Dextran also affects hemostasis; it apparently alters the red blood cell surface by increasing its negativity thus encouraging repulsion of red blood cells.

A chance finding subsequently reported by our group seemed to clarify our understanding of this syndrome. We reported bone marrow emboli in pulmonary arterioles following experimental decompression sickness,⁵ and suggested that a number of emboli in the lungs, kidney and liver were of a

lipid nature. This new wide based approach was amply supported by our results in treating humans with decompression sickness. Dextran infusion begun immediately after diagnosis improved the patient's shocklike state and shortened the recompression schedule.

Recently we have re-sectioned lung areas known to be involved by emboli following experimental decompression. These pulmonary areas were obtained by biopsy or postmortem examination. Pulmonary localization in 14 dogs was made possible after the development and refinement of radioisotopic pulmonary scanning techniques.⁶

The purpose of this study is to report the results of tissue sectioning of lungs in animals overcompressed to 165 feet, maintained at depth for 60 minutes, and then decompressed to surface. Oil-red-O stains were made of treated and untreated animals. Treatment consisted of LMW dextran or intravenous heparin.

MATERIALS AND METHODS

Thirty-three mongrel dogs were employed in this study. All animals were overcompressed to 165 feet (73.5 PSIG) for 60 minutes. We have previously described our protocol.

Five animals unexposed to the chamber were sacrificed to provide baseline data for routine pulmonary fat stains.

Fourteen animals were treated by dextran following decompression. These animals underwent lung biopsy 48 hours after the chamber procedure and immediately following a second lung scan.

A third group of 14 animals was overcompressed in the usual fashion. After chamber removal five animals served as exposed but untreated controls while ten animals received IV heparin 3 hours after chamber removal. The control animals expired. Fat stains were made of lung tissue. Biopsies provided the basis for fat stains in the heparin treated group.

RESULTS

Unexposed Control Animals (5 dogs)

Fat globules may be seen occasionally in lungs of animals previously fasted for 24 hours. The fat globules are difficult to locate and may be seen in areas containing the smaller blood vessels near the bronchial cartilages.

Exposed Untreated Animals (5 dogs)

Numerous lipid emboli are easily seen on routine sections (Figures 1 and 2). The emboli appear most frequently in areas where hemorrhage and pulmonary edema are present.

Exposed and Treated Animals (24 dogs)

Fourteen dogs were treated by dextran alone. Ten animals were

treated by heparin alone (Table 1). Evidence of hemorrhage and pulmonary edema existing in pulmonary areas diagnosed by radioisotopic lung scanning is seen (Figure 3). Fat emboli are also seen in biopsied areas 48 hours after decompression (Figure 4).

DISCUSSION

Philp et al.⁷ and Barthelemy⁸ have reported the benefits of heparin as a lipemic clearing agent in decompression sickness. Survival rates were significantly improved with heparin. We recently confirmed the benefits of heparin in experimental decompression sickness.⁹ Lipid embolization would appear to be significant since recompression was not employed, and heparin was protective.

While gaseous embolization is undoubtedly a major factor in the genesis of decompression sickness, our experience with dextran would suggest the more important role of lipid emboli. Lipid content is also reduced after dextran treatment. A resemblance exists between traumatic fatty embolization and decompression sickness. Striking similarities when comparing both clinical syndromes are as follows:

- 1) A latent period develops before the onset of symptoms.
- 2) Symptomatology are identical in both. They include pulmonary edema, hypoxia, shock and central nervous system signs.

3) Petechiae in patients with decompression sickness follow skin patterns described in fat embolism.

4) Therapeutic regimens are similar.

SUMMARY AND CONCLUSIONS

1) Lipid embolization would appear to play a major role in the genesis of decompression sickness.

2) Evidence for the co-existence of lipid emboli and gaseous emboli is presented.

3) Heparin or dextran-effective lipemic clearing agents--are beneficial in treating experimental decompression sickness.

4) A combined therapeutic approach--recompression and dextran should be employed in treating human decompression sickness. Heparin may serve as a substitute in selected instances.

LEGENDS

Figure 1. Photomicrograph demonstrating fat emboli in small capillaries in pulmonary region (100X)

Figure 2. Fat embolus (oil-red-O) in capillary in lung (125X)

Figure 3. Hemorrhage and pulmonary edema in lung of dog exposed to 165 feet for 60 minutes (60X)

Figure 4. Fat emboli (oil-red-O) in small vessels surrounding bronchioles (100X)

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