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13. ABSTRACT

Mumps virus infection without parotitis was observed in one member of a four-man U.S. Navy diving team during a 15-day 600-foot chamber dive. As part of a biomedical program, blood samples were obtained periodically throughout the dive. This report details the clinical course of the disease, the biochemical changes that occurred, and the serum viral titer studies. There were no apparent differences between mumps in the hyperbaric environment and mumps at ambient pressure.

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Case of Mumps During Hyperbaric Exposure

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THE COURSE OF MOST viral diseases has been followed only after the onset of clinical symptomatology. A recent opportunity to observe clinical, biochemical, and viral antibody changes in a case of mumps virus infection without parotitis occurred when a subject under intensive biomedical monitoring developed mumps during hyperbaric exposure. Biochemical changes were apparent the day the chamber dive to a simulated depth of 600 feet of seawater began although clinical signs did not occur until the second day in the helium-oxygen atmosphere.

METHODS

The week prior to the hyperbaric exposure periodic fasting venous blood samples were obtained by venipuncture at 6:00 a.m. to provide baseline serum bio-

chemical monitoring. The analyses reported here were performed by standard laboratory methods. The chamber exposure was for a total of 15 days including 7 days at 600 feet and 8 days of decompression. A complete description of the dive profile and the results of biochemical determinations has been published.[1]

RESULTS AND DISCUSSION

Clinical Description: Immediately prior to descent, the subject underwent a physical examination that revealed no abnormalities. Twenty-four hours after reaching a pressure equivalent to 600 feet he developed malaise and fever which persisted for eight days. The second day of the dive he developed testicular tenderness which persisted throughout the remainder of the dive. At no time during the dive did he demonstrate any salivary gland signs or symptoms. Upon completion of the decompression the only finding was some residual testicular tenderness. It was noted that two of the diver's children developed classical mumps the day of onset of his symptoms.

Biochemical Observations: Figure 1 shows the changes that were observed in haptoglobin, creatine phosphokinase (CPK), lactic dehydrogenase (LDH), and amylase compared with viral titers, clinical symptoms, and the dive profile. The first noticeable change was an increase (3-fold) in serum amylase on the day the dive began. At this point, no changes in serum viral titers* were evident nor had the diver reported any symptoms. The next sample, obtained two days later as an acute phase sample, showed significant increases in LDH, CPK, and haptoglobin. Mumps viral titers had increased only two-fold, generally insufficient to establish diagnosis.

Of these serum constituents CPK appeared to be elevated in all the divers and is presumed to be associated

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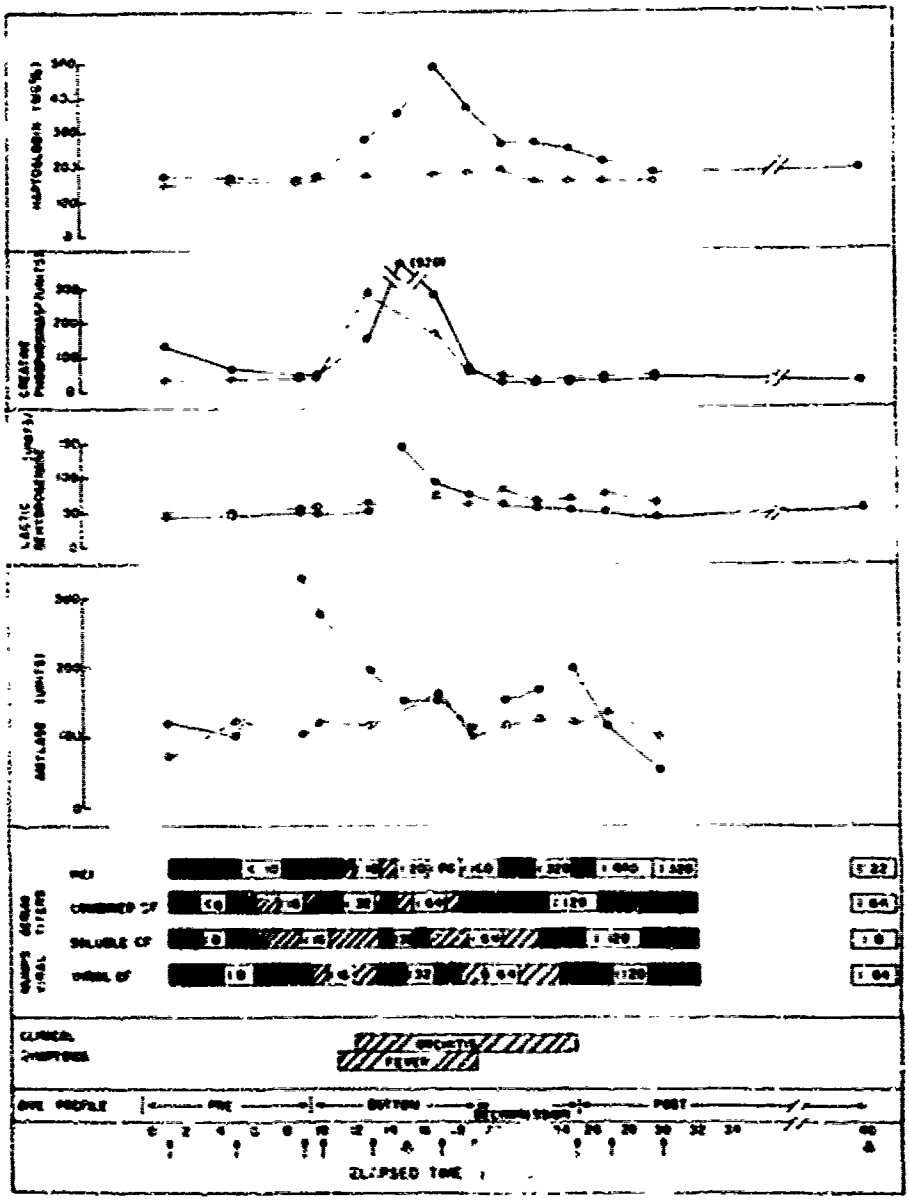

Fig. 1. Serum viral titers, biochemical changes and clinical symptoms of diver with the mumps in relation to the hyperbaric exposure. During the period indicating fever, the subject had morning spiking fevers and general malaise.

- ↑ indicates sample times for obtaining blood.
- Δ indicates an acute and a convalescent sample obtained on the diver with mumps.
- ● value of determination for subject who contracted mumps.
- ○ mean of seven other divers on this and an identical dive.
- HAI hemagglutination inhibition antibody.
- CF complement fixation antibody.

with the hyperbaric exposure. Although slight increases in LDH and haptoglobin were noted in the non-infected divers, the increases in the diver with the mumps were much greater. The elevated amylase level suggested salivary gland or pancreatic involvement, although no symptoms associated with the pancreas or salivary gland were apparent.

Mumps antibody titers were not significantly elevated until the eighth day of the dive. During decompression the three other divers had significant elevations of mumps hemagglutination inhibition antibody titer from <1:10 to 1:80, presumably due to the close contact with the subject. These three divers had a history of mumps in childhood and were presumed to be immune. This secondary increase in mumps antibody in the historically immune individuals was not as great as that observed in the diver with the mumps. On a subsequent identical dive there was no change in antibody titer in any of the four divers, suggesting that these antibody changes were not associated with hyperbaric exposure. Table I lists the viral antigens that were used in screening the divers' serum. The titers of other viral antibodies listed did not change in either of the two dives.

TABLE I. ANTIGENS USED IN SCREENING SERUM FROM DIVERS EXPOSED TO A HYPERBARIC ENVIRONMENT

Complement Fixation Antibody Test	
Parainfluenza	Cytomegalovirus
Mumps: soluble, viral, combined	Rubeola
Adenovirus group	Poliovirus 1, 2, 3
Herpes Simplex	Coxsackie B1, B2, B3, B4, B5, B6
Reovirus group	Influenza
Mycoplasma pneumoniae	Rubella
Respiratory syncytial	
Hemagglutination Inhibition Antibody Test	
Mumps	Rubella
	Influenza A,

CONCLUSIONS

Two important points are evident as a result of this observation, especially as man is subjected to more frequent and longer duration hyperbaric exposures. The first point is that there was no significant difference in the course of one infectious disease when the acute phase occurred under extremely altered environmental conditions. Secondly, the observed increase in mumps antibody titer demonstrated that immunoglobulin production, a normal response to an infectious agent, was apparently unimpaired when the subject was in a hyperbaric environment.

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