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FACIAL ECCHYMOSIS FOLLOWING A DIVE WITH THE U.S. NAVY MK I DIVER'S MASK.

B. L. HART

Naval Medical Research
and Development Command
Bethesda, Maryland 20814-5044

Department of the Navy
Naval Medical Command
Washington, D.C. 20372-5210

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O. P. DAILY, CAPT, MSC, USN
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A diver presented with facial ecchymosis in the distribution of his mask following a dive to 43 fsw using the U.S. Navy MK I mask. There was no apparent problem during the dive. He had taken two or three aspirin tablets 1 week prior to the dive. Hematologic studies were notable for a bleeding time of 8 min and abnormal platelet aggregation. After 2 weeks free of medication, the subject's bleeding time was 5 min and platelet aggregation studies were normal. Differential diagnosis of skin lesions following diving is discussed. Face mask squeeze, rare with the MK I mask, appears in this case to be related to abnormal platelet function caused by previous aspirin ingestion.

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Skin lesions can occur during or after diving from several causes, including barotrauma and decompression sickness. An unusual case of facial injury consequent to diving is presented here.

CASE REPORT

A 34 year old, male, U.S. Navy diver in good health made a dive in a hyperbaric research chamber. Wearing the US Navy MK I Mod 0 diver's mask, he descended the ladder into 9 ft of water. The chamber was pressurized at a rate of 75 fsw/min, with a pause of several seconds to switch the breathing gas to a 90% N_2/10% O_2 mix at 21 fsw. Final pressure for the diver was 43 fsw. The diver had the mask adjusted comfortably, with the dial-a-breath just backed off from free-flow. He experienced no pain or difficulty during descent, reporting that the rig breathed easily and that it was a very comfortable dive. The diver ventilated the mask immediately after reaching depth but does not remember ventilating at all during descent. Moderate work was performed during 1 h at depth. Ascent was made at 60 fsw/min, with a brief pause at 30 fsw for switching the breathing gas back to air. Immediately after leaving the chambers, the diver was noted to have an area of redness around the eyes and forehead.

On postdive physical examination the diver was found to have well-demarcated, nontender, periorbital ecchymosis (Fig. 1). There was no facial edema. The outline coincided perfectly with the interior of the mask and border of the oronasal mask (Fig. 2). There was no conjunctival hemorrhage. Ecchymosis was also seen superior to the border of the mask, extending to the scalp. The diver felt well, and the examination was otherwise unremarkable. Another subject who had made the same dive had no problems. The ecchymosis faded over the next several days.

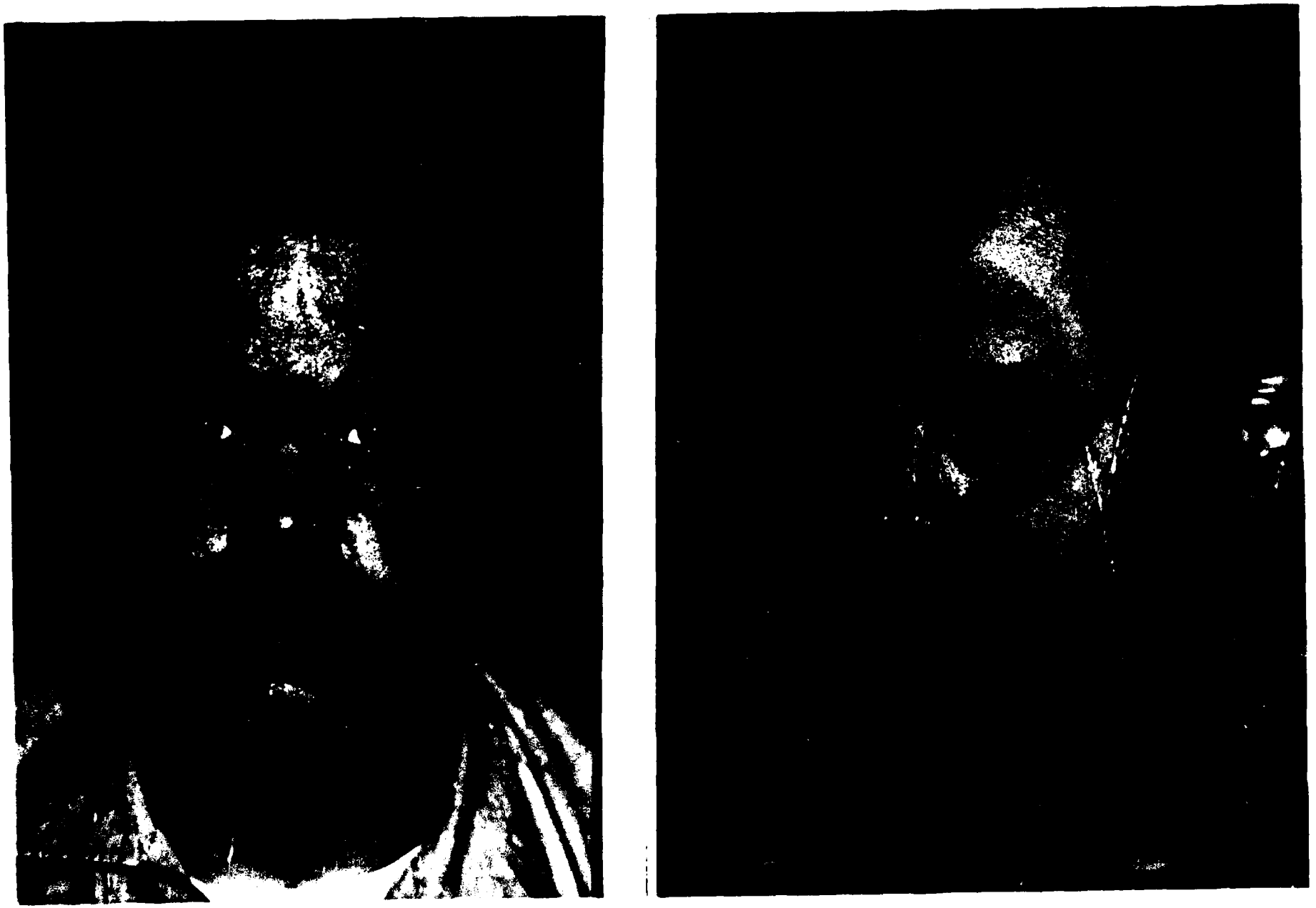

Figure 1

Figure 2

Figure 1. Postdive ecchymosis over the forehead and cheeks.

Figure 2. U.S. Navy MK I diver's mask, interior view, with and without neoprene seals. Note the similarity to the pattern of facial ecchymosis in Fig. 1.

Past medical history included major trauma to one foot, a tonsillectomy, wisdom tooth extraction, and a mandibular reduction, all of which healed without unusual bleeding or complications. There was no family history of bleeding disorders. The subject had taken several medications one week prior to this dive for a toothache, including two or three regular aspirin tablets. He had taken no medications within 24 h of the dive. The diver had a history of migraine headaches.

The mask was carefully examined. No problems were found, and other divers used the same mask without incident in following days.

HEMATOLOGIC STUDIES

Hematologic studies were done the following day. Bleeding time was 8 min (normal 2.5-9.5 min). Prothrombin time (PT) was 10.6 sec, and partial thromboplastin time (PTT) was 31.9 sec (normal ranges 9.6-13.2 sec and 25.0-39.0 sec, respectively). Platelet count was 230,700. A complete blood count revealed each of the following to be within the normal range for the testing laboratory: WBC 10,500; Hgb 15.5; Hct 47.6; MCV 93.4; MCH 30.4; MCHC 32.6.

Platelet aggregation was also measured (Fig. 3). Aggregation was intermediate in response to 40 µm ADP, 40 µm epinephrine, 2 µgm/ml collagen, and 1 µm arachidonic acid. Ristocetin (1.5 mg/ml) led to normal aggregation. These aggregation studies were also done the same day on blood from a control subject. The same reagents, at equal or lower concentrations, caused normal aggregation in the control.

These studies were repeated 2 wk later, during which time the diver had taken no medication. Bleeding time was 5 min. PT was 11.0 sec, and PTT was 31.9 sec. Aggregation, with reagents at the same concentrations as before, was normal.

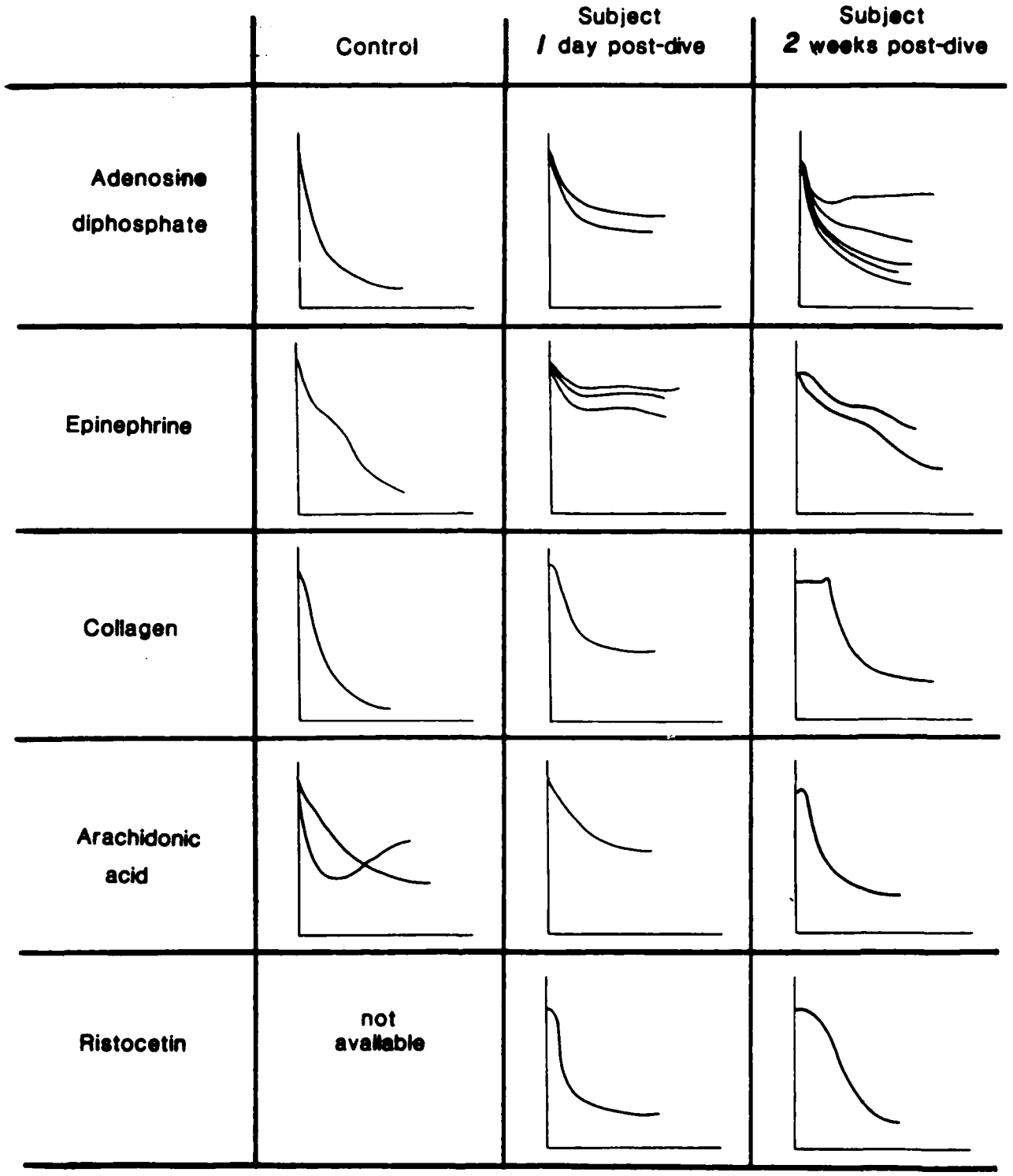

Figure 3. Platelet aggregation studies. Time is on the X axis, and the Y axis represents decreasing optical transmission. Aggregation in response to ADP, epinephrine, collagen, and arachidonic acid is impaired one day post-dive and normal two weeks later. Control studies were done on plasma from a normal volunteer one day post-dive.

DISCUSSION

The pattern of facial ecchymosis observed in this diver is typical of a face mask squeeze, in which a negative pressure develops within a mask during descent. Face mask squeeze is unusual in the Mark I and similar masks because the gas supply is within the mask and provides gas at ambient pressure. It is possible that in this case the oronasal mask fit so tightly that the space outside it could not equalize, thereby developing a negative pressure during descent. The absence of subconjunctival hemorrhage, however, argues against this explanation. An early report of squeeze mentions the typical "engorged and inflamed" conjunctivae (Liddell, 1842).

The abnormalities of platelet function are probably due to the aspirin taken a week before. Aspirin irreversably inhibits platelet cyclooxygenase, and platelet aggregation and bleeding time are disturbed even in normal individuals for 4-7 days after a single dose (Holmsen, 1976; Schrier, 1984; Weiss, 1975). The abnormalities of platelet aggregation, their spontaneous return to normal values when aspirin was withheld, and the normal coagulation studies and platelet count are consistent with an effect of the aspirin taken a week earlier. It is possible that this acquired platelet dysfunction may have contributed to the striking clinical response to a relatively minor degree of trauma.

Could the subject's presumed vasomotor instability, manifested by occasional migraine headaches, have been a factor? Some studies of decompression sickness in aviators have reported a high incidence of spontaneous clinical migraine in subjects who developed neurologic symptoms of decompressions sickness (Ferris, 1951; Masland, 1943; Fraser, 1943). No reports of an association between clinical migraine and dermatologic problems in diving could be found in the literature.

Could the decompression have been a factor? The presence of the lesion immediately upon surfacing argues against this. Although cutaneous hemorrhage can occur with skin decompression sickness, the lesions usually blanch to pressure, are more irregular in configuration than in this case, and are often accompanied by pruritis.

In summary, this man's injury appears to be a typical face mask squeeze, but it is notable for the lack of circumstances normally leading to such a condition. Aspirin ingestion is the most probable predisposing factor.

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