by treating with cysteine. The results are given in Table 1, and again suggest that oxidation has a much

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Treatment _	Per cent. of initial activity measured at the same pH		
	Myosin 1  Measured at pH 9.2	Myosin 2	
		Measured at pH 9.0	Measured at pH 6.3
0.0075 per cent. H <sub>2</sub> O <sub>2</sub> for 15 min. at 22° C,	· . 5	14	49
H <sub>2</sub> O <sub>2</sub> as above followed by 0.02 M cysteine for 15 min. at 22° C.	32	49	58

greater effect upon the activity measured in the more alkaline range. When oxidation has taken place, restoration of the activity by reduction takes place to only a small extent for the acid range, but to a substantial extent for the alkaline range.

One might be tempted to look upon the differential effect of oxidation and reduction as evidence for two types of enzyme in the myosin preparations. However, we have been unable to obtain any evidence for fractionation by precipitation at high or low salt concentrations. The failure of attempts to separate the activity from myosin has also been reported by Bailev.<sup>6</sup> The possibility that a different type of functional group in the same molecule or micelle may be primarily responsible for enzymatic activity in each of the pH ranges also suggests itself. Most studies of this enzyme have been made in the pH ranges around 8.5 to 9.5. As the pH is reduced the activity declines and then rises again in the region between 7.5 and 6.5. Although the activity is only about half as great as that at the alkaline optimum, it is this pH which may be expected to be found in the intact muscle. The present studies indicate that the behavior of the enzyme may differ qualitatively as well as quantitatively in the two pH ranges. Whatever the ultimate explanation may be, these experiments do indicate the need for particular attention to pH effects in considering the in vivo action of muscle ATP-ase.

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## STUDIES ON THE PERIPHERAL BLOOD IN PATIENTS WITH THERMAL BURNS.

1. THROMBOCYTOPENIA1, 2, 3

THE blood platelets, counted by a direct method,4 were followed on 13 patients with thermal burns.

6 Kenneth Bailey, Biochem. Jour., 36: 121, 1942.

The patients ranged in age from 4 to 81 years; 5 were males and 8 females. The total area of body surface burned varied from 10 to 75 per cent. and in 9 of the 13 patients was 25 per cent. or more. Third-degree burn was present in all. In 9 cases 20 per cent. or more of the body surface was involved with a third-degree burn.

Surface treatment in most cases was petroleum jelly dressings, often with pressure dressings or plaster casts. One patient was treated with tannic acid and silver nitrate, and one with "triple dye."

Shock was present in 6 cases; in 2 it was slight and in 4 moderate. All the patients received citrated plasma. The administration of large amounts of citrated plasma to normal subjects did not cause a thrombocytopenia.

Hemoglobinemia was present for varying periods of time in all the 13 patients.

For purposes of analysis, platelet counts below 150,000 per cu. mm were considered decreased. In 10 patients the initial platelet count a few hours after burn was above this level. In 3 cases the first platelet count, obtained 9, 38 and 72 hours after the burn, was already below 150,000 per cu. mm.

No decrease in platelets was observed in one patient, who died 7 hours after the burn. He was a 75-yearold man with 15 per cent. of his body surface involved, of which 5 per cent. was a third-degree burn.

In 12 cases, a decreased platelet count was observed 7 to 57 hours after the burn. The lowest counts obtained occurred within 23 to 96 hours after injury and ranged from 9,000 to 96,000 per cu. mm. Six of these patients died within 4 days after the injury, and in these cases the thrombocytopenia persisted until death. In the remaining six, the platelets returned to a normal level in from 4 to 9 days. However, death occurred subsequently in all but one case. This patient is living and apparently completely recovered, fifteen months after injury.

Purpura was observed in 2 patients. In one case petechiae and ecchymoses were widespread.

The cause of the thrombocytopenia and its possible role in the complication of burns is at present under investigation.

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3 Read before the meeting of the Burns Committee, Washington, D. C., December 21, 1943. 4 F. J. Pohle, Am. Jour. Med. Sci., 197: 40-47, 1939.

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<sup>&</sup>lt;sup>2</sup> The work described in this paper was done in part under a contract recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Harvard University.