LIVER DAMAGE AND DEXTROSE TOLERANCE IN SEVERE BURNS

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INFORMATION concerning damage to the liver in patients with severe burns has been based in the past, largely, on histologic studies of postmortem material.[1] In the present study, objective evidence of hepatic damage was obtained during life by the use of liver function tests. Three patients with second and third degree burns of 15 to 25 per cent of the body surface were studied. Details of the case histories have been published elsewhere.[2] Disturbances in the fluid balance of these patients were corrected within 48 hours after the burn by the employment of plasma transfusions; and at no time did the patients show marked symptoms of secondary shock. Cases 5 and 9 recovered while Case 8 died with a hemolytic streptococcal septicemia, on the eighth day.

Bilirubinemia, bromsulphalein retention, hippuric acid output, plasma prothrombin concentration and dextrose tolerance were determined in the three cases at frequent intervals. The results are presented in Charts 1, 2 and 3. The hyperbilirubinemia, during the first 48 hours, may have been caused either by a rapid breakdown of red cells injured by heat in the burned area, or by failure of the liver to excrete bilirubin at a normal rate. An abnormal bromsulphalein retention was observed between the fourth and fifteenth days of each case. During this period, the capacity of the liver for producing glycine and forming hippuric acid became impaired to a variable extent in Cases 8 and 9, an observation which confirms the report of Boyce and McFetridge.[3] Moderate reductions in the plasma prothrombin concentration were observed in each case. As none of the other causes of hypoprothrombinemia were present, this finding may be regarded as presumptive evidence of hepatic damage.

The dextrose tolerance curve is the result of several factors, of which the functional capacity of the liver is one. Curves obtained on these patients show marked deviations from the normal. Although the curves might be regarded as evidence for impairment of the hepatic mechanism for glycogenesis, other factors, hormonal and dietary, must be evaluated before an accurate interpretation can be made.

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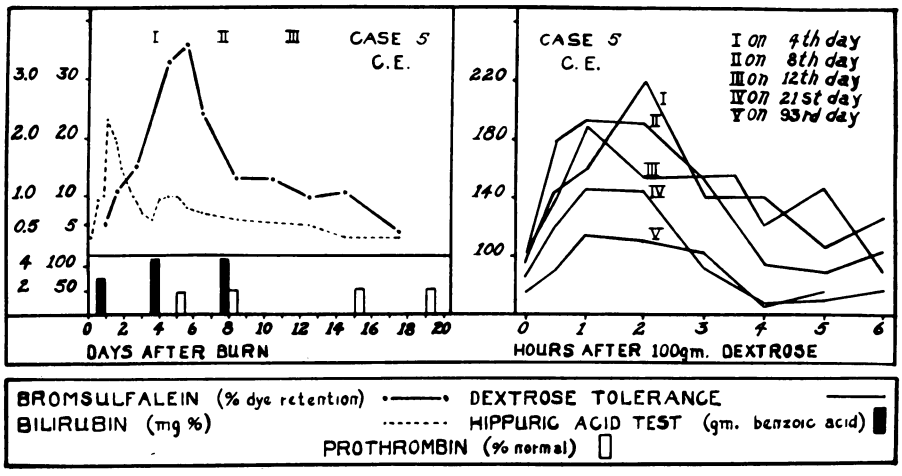

CHART 1, Case 5.

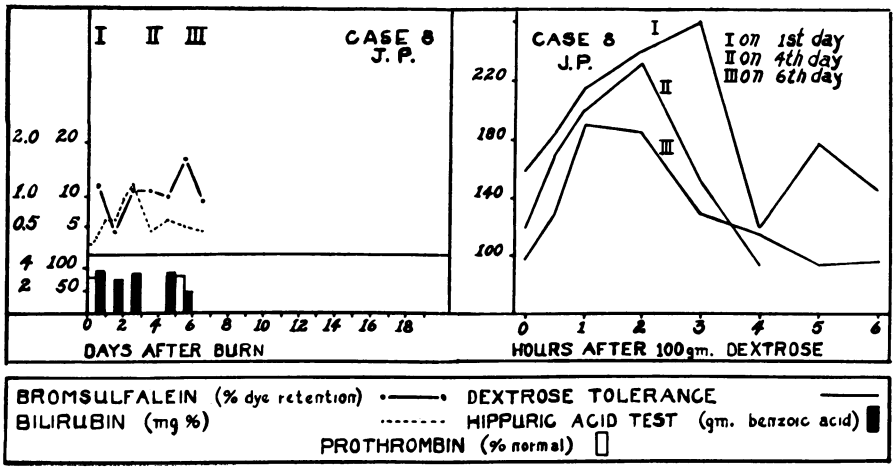

CHART 2, Case 8.

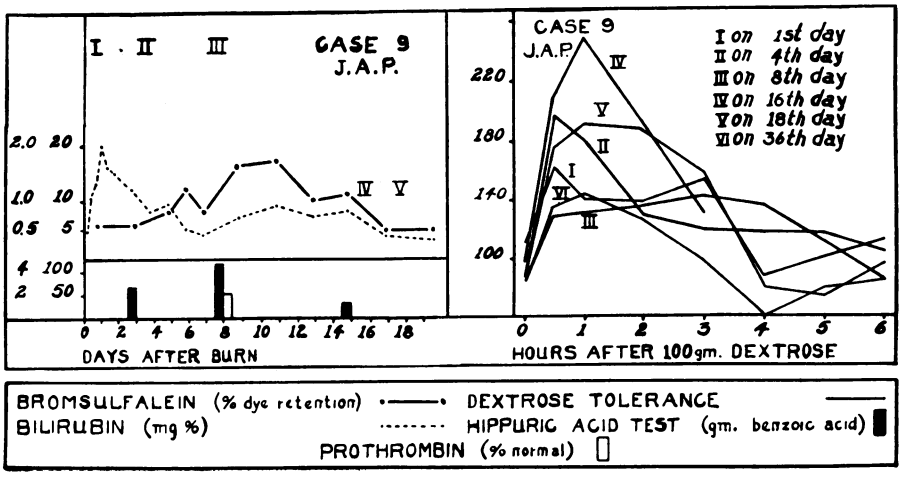

CHART 3, Case 9.

CONCLUSIONS

The effect of severe burns on bilirubinemia, bromsulphalein retention, hippuric acid formation, prothrombinemia, and dextrose tolerance were studied in three patients. The changes found in bromsulphalein retention, hippuric acid formation, and prothrombinemia indicate the presence of hepatic damage, especially during the period from the third to the tenth day following the injury. The changes in the bilirubin and dextrose tolerance are also suggestive of liver injury. Whether the cause of the disturbance in the liver is a toxin from the burned area, anoxia associated with capillary stasis, infection, or a combination of these factors cannot be stated at the present time.

REFERENCES

[1] Wilson, W. C., MacGregor, A. R., and Stewart, C. P.: The Clinical Course and Pathology of Burns and Scalds under Modern Methods of Treatment. Brit. Jour. Surg., **25**, 826, 1938.

[2] Elkinton, J. R., Wolff, W. A., and Lee, W. E.: Plasma Transfusion in the Treatment of the Fluid Shift in Severe Burns. ANNALS OF SURGERY, **112**, 150, July, 1940.

[3] Boyce, F. F., and McFetridge, E. M.: Studies of Hepatic Function by Quick Hippuric Acid Test; Various Surgical States. Arch. Surg., **37**, 443, 1938.

ERRATUM

In the article by Dr. Charles H. Watt, "A Modified Spur-Crushing Clamp and Its Use," appearing in the ANNALS OF SURGERY, **111**, 1076-1083, June, 1940, the legend for Figure 1 should read "The author's modification of the Stetten spur-crusher. The original Stetten clamp does not have the blade."

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