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clinic the values lay within normal limits. The lesions tended to run a chronic course, and neither local therapy nor, in the case of diabetics, general management tended to speed their healing. In some cases the lesions slowly receded and left depressed scars.

Two ideas regarding the pathogenesis of this condition have been considered: (1) that which assumes the occurrence of a primary vascular injury, possibly by circulating toxins, with secondary thrombosis, necrosis, and fat imbibition and (2) that which assumes a local lipid disturbance in the skin, based on a general disturbance in fat metabolism. Neither hypothesis has proved satisfactory in the light of studies made both here and elsewhere. Therefore, the pathogenesis of this condition remains obscure.

BANTI SYNDROME (FIBROCONGESTIVE SPLENOMEGALY)

DEFINITION, CLASSIFICATION AND PATHOGENESIS

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The Banti syndrome is still an unsolved problem. The question of cardinal importance is whether the splenomegaly is primary or secondary to portal or hepatic changes.

Banti¹ stated the belief that the trouble lay primarily in the spleen and that the portal and hepatic changes were dependent on the splenic ones. The idea that the splenomegaly was the consequence of circulatory disturbances was put forward by Dock and Warthin² and further developed with the description of the thrombophlebitic splenomegaly.³ This theory occupied a position of definite advantage when it was recognized that the structure of Banti splenomegaly was chiefly congestive⁴ and that

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