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Discussion

Dr. Champ Lyons: It has been my privilege to follow some of the developments in these cases as Dr. Hubbell lives 100 miles from Birmingham and comes up regularly to assist us in the teaching program. I have also had the privilege of reviewing his paper.

To me it is extremely significant that hypoalbuminemia has resulted in these cases. I think perhaps the emphasis that has been given on the hypoalbuminemia by this paper is the most significant finding. Dr. Hubbard has stated that he would rather not do a fistula into an open portal vein because of this. I am sure he is probably right, but I would like to suggest that there may perhaps be another mechanism involved. The Scandinavian literature reported the development of hypoalbuminemia as the result of the Bilroth II anastomosis and its correction by the conversion of that anastomosis to a Bilroth I. I think Dr. Hawkins in some of his papers, I can't quite recall the exact ones, mentioned that as one reason for preferring the Bilroth I. Dr. Ravdin told me this morning that he had on two occasions found patients with Bilroth II's and hypoalbuminemia who had been cured by converting to a Bilroth I. The mechanism of a hypoalbuminemia produced in these patients may prove to be a very fertile field for investigation. I think it also raises a very obvious obligation on the part of those who are doing various end-to-side or side-to-side anastomoses for relief of portal hypertension to follow and study the albumin levels.

We happen to have followed with Dr. Longmire in a preference for side-to-side, and I was quite pleased to see that in a certain percentage of his patients there has been an actual rise in albumin. Perhaps careful attention to both eualbuminemia and hypoalbuminemia will afford a basis for the long-term comparison of end-to-side and side-to-side shunts.

Dr. William P. Longmire, Jr.: I would like to compliment Dr. Hubbard on his interesting presentation. We have been interested in this general subject and have reviewed the literature recently in an effort to evaluate cases of this type.

The problem of ammonia intoxication occurs under three conditions. In the first place, it occurs

in dogs with normal liver after portocaval anastomosis; it occurs in man when portocaval anastomosis is performed and the liver is normal, as in the cases described by McDermot and one case that Dr. Hubbard described this afternoon. Secondly, it occurs occasionally in patients with cirrhosis following all three types of portocaval anastomosis. Thirdly, it occurs in patients with severe cirrhosis without any type of anastomosis.

How can one fit all this problem together? It seems to us that the only way in which this can be logically explained is by postulating a compensating mechanism for the patient with a cirrhotic liver, whether it is increased arterial supply or something else, which develops gradually over a long time as the collateral circulation forms. A patient with a cirrhotic liver is better able to tolerate a sudden diversion of the portal stream into the systemic circulation than is the patient with a normal liver, as we see in our patients who have survived for more than five years without neurologic symptoms and without hypoprotenemia.

We also believe that the occurrence of neurological symptoms and ammonia intoxication in patients with a shunt, and patients with cirrhosis, depends entirely on the critical level of hepatic function. Patients with ammonia intoxication do not have this disturbance alone; they have other symptoms of severe hepatic dysfunction, and we feel that once the general function of the liver sinks below a critical level, these patients develop neurological changes or other warning signs of hepatic failure regradless of whether the portal blood passes through the liver or bypasses this organ through a portocaval shunt.

I should like to thank Dr. Hubbard again for his excellent presentation of these interesting cases. I believe this is a real contribution to our medical knowledge.

DR. T. BRANNON HUBBARD, JR. (closing): I would like to thank Dr. Lyons and Dr. Longmire for their kind remarks. Dr. Lyons' idea of reconstructing these patients after the method of a Bilroth I procedure is an interesting one and the results probably should be explored. However, the tuition in the school of experience is rather high and I for one cannot afford another semester in this particular course.

With regard to Dr. Longmire's discussion, I wonder if the fact that the cirrhotic gets along in many cases so well with an Eck fistula, whereas these three perfectly normal livers did not, might be related to the recent work of Bollman and associates. As you know, they have recently shown that if the portal vein of a dog is ligated in stages, thus resulting in a collateral circulation, then an Eck fistula is followed by a relatively normal existence without hepatic degeneration. This work was discussed in the manuscript but has not been mentioned today due to lack of time. Thank you very much.