

Portal hypertension: A comparative model of arterio-venous fistula

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Complications of portal hypertension remain perplexing physiologic phenomena in the understanding of shunt hemodynamics with multiple theories. Hyperdynamic circulation was also found in sepsis, chronic anemia and arterio-venous (A-V) fistula which relate to an increase in nitric oxide. We hypothesized that portosystemic collaterals may mimic an A-V fistula in which the high pressure portal blood connects with the lower pressure systemic venous circulation. Although these collaterals decompress the portal circulation, a number of secondary hemodynamic phenomena occur which increase portal blood flow and tend to counteract the portal hypotensive effect of the portosystemic shunt. The consequent increases in cardiac output and portal blood flow perfuse the compromised liver. As portal blood flow increases, collateral flow increases and is nearly totally shunted in the systemic circulation. This shunt may eventually introduce a vicious cycle of

hyperdynamic circulation into a compromised host. Ultimately, high-output cardiac failure occurs, leading to cirrhotic cardiomyopathy.

The A-V fistula model may help to identify potential sources of increased NO and norepinephrine production which remain undefined in PHT. Recognizing the existing *underfilling* and *overflow* theories of ascites accumulation, the A-V fistula model expands our existing knowledge of systemic (hepatopulmonary syndrome, cirrhotic cardiomyopathy), splanchnic (variceal formation) and renal hemodynamics (hepatorenal syndrome) of cirrhotic and non-cirrhotic PHT by a single concept. Based on this new application, we can: 1) provide explainable reasons why some of modalities of treatments yield better results eg. β -blockers, NO inhibitors, selective shunt, and TIPSS, 2) establish specific aims in order to study and treat PHT.

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