8. HEPATIC ARTERY EMBOLIZATION FOR LIVER CANCER

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INTRODUCTION
It is well known that the liver has a dual blood supply from the portal vein and hepatic artery. Approximately 75% of blood flow to the liver parenchyma is from the portal vein and 25% is from the hepatic artery; 50% of oxygenation is derived from the portal vein and hepatic artery each. However, most primary and secondary neoplasms of the liver receive the blood supply exclusively (more than 90%) from the hepatic artery [1,2].

Disruption of arterial supply to the tumors to create tumor ischemia and arrest tumor growth was initially proposed by Markowitz in 1952 [3]. Several experimental and clinical studies demonstrated the immediate effectiveness of devascularization. Gelin and others, using Xenon blood flow study, demonstrated a 90% decrease in tumor blood flow and a 35–40% decrease in hepatic parenchymal blood flow after hepatic artery ligation [4]. Mori and associates reported a selective necrosis of the tumors without damage to the normal liver parenchyma after ligation of the hepatic artery in a patient with metastatic gastric carcinoma [5]. In experimental hepatic tumors, Nilsson and others demonstrated that hepatic artery ligation produced selective tumor necrosis and resulted in prolonged survival [6]. However, in early clinical trials, hepatic artery ligation for the treatment of hepatic neoplasms did not satisfactorily improve or prolong patient survival [7–11]. This