

**HEPATIC ARTERY AND PORTAL VEIN CONTRIBUTIONS TO  
EXTRACTION OF RADIocolloid BY CANINE LIVER**

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Recognition that intrahepatic tumors receive blood principally from the hepatic artery (1) raised questions as to whether both the hepatic artery and portal vein contribute blood to liver cells for extraction of radioactive colloids. We have approached this question by using  $^{99m}$ Tc-sulfur colloid in dogs and by selectively ligating the hepatic artery or portal vein.

**METHODS**

Adult mongrel dogs weighing 20–25 kg were anesthetized with intravenous sodium pentobarbital. The abdomen was opened through a midline incision and

the portahepatis exposed. Nonocclusive ligatures were placed around the hepatic artery of two animals; the portal vein was encircled in similar fashion in another two animals.

Each dog acted as its own control. The dogs were placed supine beneath a gamma-ray camera (coupled to a Nuclear Data 50/50 computer and magnetic-tape unit). One hundred microcuries of  $^{99m}$ Tc-sulfur colloid were injected into a peripheral vein and counts obtained over the liver as a function of time. After a constant counting rate had been reached, the value (background counts) was noted. The hepatic artery or portal vein was occluded by ligation of the vessel and an additional 500  $\mu$ Ci of  $^{99m}$ Tc-sulfur colloid were injected into the peripheral vein. Data were again accumulated with the gamma-ray camera and computer.

Analysis was carried out by noting the highest count ( $C_\infty$ ) over the liver, and the counts at each time (C) period (10-sec interval). Data were plotted according to the assumption that

$$C = C_\infty (1 - e^{-\lambda t}) \quad (1)$$

$$\ln \left( 1 - \frac{C}{C_\infty} \right) = -\lambda t \quad (2)$$

in which  $\lambda$  was the rate constant for radiocolloid accumulation. Controls had shown that two successive injections of radiocolloid in dogs did not significantly affect the rate constant. Hence any alterations can be attributed to the vessel ligation. For each rate constant, a half-time was calculated.

**RESULTS**

A typical result is shown in Fig. 1. A control  $T_{1/2}$  of 60 sec in this dog increased to 149 sec after portal vein ligation. A summary of the four dogs is shown in Table 1. Since the portal vein carries the

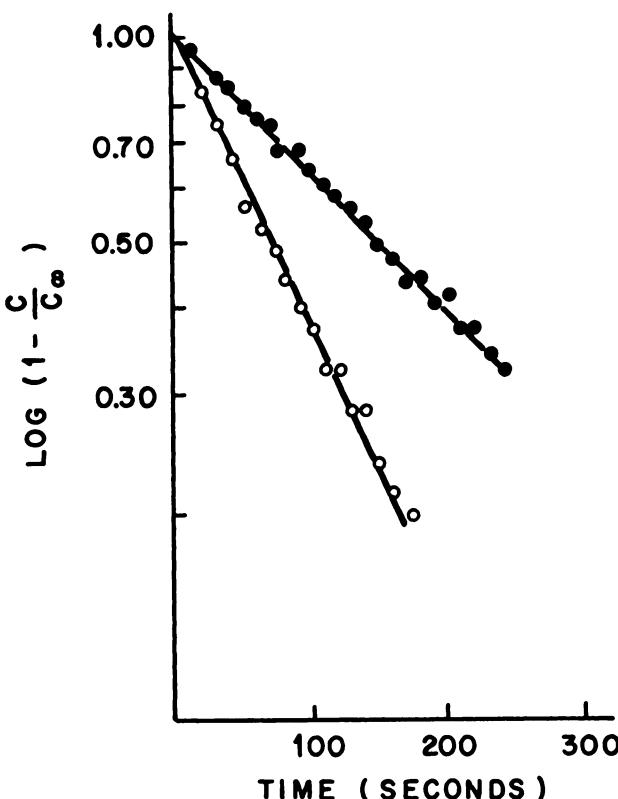


FIG. 1. Lower line represents data in dog during control period of  $^{99m}$ Tc-sulfur colloid accumulation ( $T_{1/2} = 60$  sec). Upper line is result after portal vein ligation ( $T_{1/2} = 149$  sec).

Received Oct. 28, 1971; revision accepted Jan. 25, 1972.  
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**TABLE 1. DATA ON HALF-TIMES FOR HEPATIC  
 $^{99m}\text{Tc}$ -SULFUR COLLOID ACCUMULATION  
IN DOGS\***

	Control $T_{1/2}$ (sec)	Ligation $T_{1/2}$ (sec)	Control ligation
Portal vein ligation	70	162	
	60	149	
Mean:	65	156	2.4
Hepatic artery ligation	73	92	
	61	90	
Mean:	67	91	1.4

\* The half-times were calculated from the rate constant in Eq. 2.

bulk of blood flow to the liver, it is perhaps not surprising that ligation of the vein resulted in a marked decrease in the rate of hepatic accumulation of the radiocolloid. However, hepatic artery ligation also resulted in a distinct (but lesser) decrease in the rate of uptake of the colloid. After hepatic artery ligation, the extraction efficiency of reticuloendothelial cells may be reduced because of decreased flow or because of cellular ischemia (that is, alteration of

either blood flow or extraction efficiency could yield diminished accumulation of radiocolloid). The experiments described here, which show a decrease in the rate of accumulation of radiocolloid after hepatic artery ligation, do not distinguish between these two possibilities. It is clear, however, that both the hepatic artery and portal vein can deliver radiocolloid to the liver for extraction by the reticuloendothelial system.

#### SUMMARY

In dogs, by having each act as its own control and ligating one or the other of the vessels, it was shown that both the hepatic artery and portal vein contribute blood for the extraction of  $^{99m}\text{Tc}$ -sulfur colloid.

#### ACKNOWLEDGMENT

This work was supported by USPHS CA 06519 and by ET-44B from the American Cancer Society.

#### REFERENCE

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