Correspondence
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PVT after splenectomy

I read with interest the article on the incidence of portal vein thrombosis (PVT) after laparoscopic splenectomy (*Can J Surg* 2005;48:352-4).[1] I agree with the authors' assumption that the frequency of PVT after laparoscopic splenectomy is just as high as that after open splenectomy. I believe that the laparoscopic procedure per se has no bearing on the occurrence of PVT, which is found mainly in patients who undergo splenectomy for benign and malignant hematologic conditions. I also believe that PVT is more likely to occur after splenectomy if the postsplenectomy platelet count is greater than 1000×10^9/L, although it may occur at lower counts as the authors have indicated.

In a review of 55 patients who underwent open splenectomy for various indications in our unit, only 2 (3.6%) had PVT; both had portal hypertension, and splenectomy was performed as part of an emergency devascularization procedure for bleeding esophageal varices.[2] PVT should be suspected in any patient who is not progressing smoothly in the early postoperative period after splenectomy and who complains of vague upper abdominal pain and has a low-grade fever in the absence of an apparent cause. The 2 patients who suffered PVT in the authors' study had idiopathic thrombocytopenia (ITP). In our series, none of the patients with ITP had PVT, and although we practise in an area where sickle cell disease is prevalent, none of them suffered PVT after splenectomy.[2] Also, although hematologic causes were the second commonest indication for splenectomy after trauma, PVT did not develop, casting doubt on the assumption that PVT is more common after splenectomy for hematologic conditions. My personal theory — which needs further substantiation — is that the frequency of splenic and portal vein thrombosis is increased when the splenic artery is first tied before proceeding with splenectomy. For the time being, we have to accept — as the authors implied — that there is no increased incidence of PVT after laparoscopic splenectomy.

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References

1. Harris W, Marcaccio M. Incidence of portal vein thrombosis after laparoscopic splenectomy. *Can J Surg* 2005;48:352-4.
2. Meshikhes AW, Mubarek MA, Abu-Alrahi AI, et al. The pattern of indications and complications of splenectomy in Eastern Saudi Arabia. *Saudi Med J* 2004;25:447-50.

(Drs. Harris and Marcaccio reply)

Thank you for your interest in our article. Your response does highlight a number of important issues. First, there is growing evidence that the prevalence of occult PVT is many-fold greater than that of clinically evident disease. Improvements in diagnostic imaging will likely continue to augment this discrepancy. Although it seems prudent that patients with occult PVT receive anticoagulants, it is unknown if anticoagulation improves patient outcomes. On the other hand, symptomatic patients obviously need to be investigated and treated promptly.

Although our study suggests that the rate of PVT after laparoscopic splenectomy is at least as high as after open splenectomy, Ikeda and associates[1] found in a recent study that the incidence of thrombosis after laparoscopic splenectomy was 55% — significantly higher than after open splenectomy. Although further research is needed, it remains possible that unique features of laparoscopic splenectomy (pneumoperitoneum, positioning, use of staplers) do modify the risk of thrombosis. But how operative factors contribute to the risk of thrombosis remains unknown.

Lastly, it is important to acknowledge that even if all patients are screened for PVT using the same imaging techniques and protocol, it seems evident that the rate of PVT will vary among series. It is well established that hematologic malignant conditions do carry an elevated risk of thrombosis. Hence, the composition of each series will influence the observed frequency of thrombosis. It is also likely that benign disease such as ITP, sickle cell disease and thalassemia each have unique risk profiles. Furthermore, it is always worth considering whether additional comorbidities specific to a patient population being served (i.e., protein C and S deficiencies, synchronous malignant conditions) are influencing observed outcomes.

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Reference

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Preoperative fasting guidelines

As a principal investigator in studies of a 2- to 3-hour fast for clear liquids in Calgary in the late 1980s, and a member of the American Society of Anesthesiologists' task force that developed evidence-based fasting guidelines, I wish to discuss the Canadian Association of General Surgeons (CAGS) review and commentary of preoperative fasting for adults.[1]

The authors state that the "classical surgical and anesthesia dictum has been that" healthy patients "should be kept NPO from midnight the night before operation." This is questionable if by "classical" they mean an established pattern of practice sanctioned by a body of literature. In 1883, Lister[2] recommended that patients should drink clear liquid about 2 hours before surgery, but there should be no solid matter in the stomach. For the next 80 years, textbook fasting guidelines were 2–3 hours for clear liquids and 4–6 hours for easily digestible solids. These were consistent with the known rapid gastric emptying of clear liquids and the slower digestion and emptying of solids. It was only in the 1960s that most American anesthesia textbooks changed, without new evidence, to NPO after midnight.

Our first Calgary study demonstrated