Abstracts

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Intra-operative colon mucosal oxygen saturation during aortic surgery

Lee ES, Bass A, Arko FR, et al. J Surg Research 2006;136:19-24.

Conclusion: A measurement of colonic mucosal oxygen saturation correlates with the stoppage and restoration of aortic flow during aortic surgery.

Summary: Colon ischemia complicating aortic reconstruction has a high mortality rate. The authors developed a method of measuring colonic mucosal ischemia during aortic surgery that correlates with interoperative events. A spectrophotometer probe is inserted into a patient's rectum before aortic reconstruction. The device allows continuous measurement of colonic mucosal oxygen saturation (CMOS). It was tested in 25 patients undergoing aortic reconstruction. Buccal mucosal oxygen saturation, mean arterial pressure, heart rate, pulse oximetry, and arrest and restoration of flow in the aorta were also recorded during each operative procedure.

An endovascular aneurysm repair (EVAR) was performed in 20 patients and an open repair was performed in five patients. During EVAR, CMOS decreased from a baseline of 56% ± 8% to 26% ± 17% during the period of infrarenal aortic balloon occlusion ($P < .0001$). During open repair, CMOS also decreased from 56% ± 9% to 15% ± 19% during clamping of the infrarenal aorta and iliac arteries ($P < .0001$). With restoration of aortic flow in both open procedures and EVAR, CMOS returned to baseline values (56.5% ± 10%, $P = .81$ compared with baseline). The time for CMOS to recover after restoration of aortic flow was 6.4 ± 3.3 minutes.

Comment: The technique offers a potential method of monitoring colonic perfusion intraoperatively. Obviously, a number of details still need to be determined, such as what is the lowest level of colonic mucosal oxygen saturation that will sustain colonic viability. In addition, the potential ability of this technique to monitor for postoperative colonic ischemia seems like an interesting line of inquiry.

Clinical syndromes and clinical outcome in patients with pulmonary embolism: Findings from the RIETE Registry

Lobo JL, Zorrilla V, Aizpuru F, et al. Chest 2006;130:1817-22.

Conclusion: Patients with pulmonary embolism (PE) who suffer pulmonary infarction as a manifestation of their PE have a better initial and long-term prognosis than patients with a PE that presents as dyspnea or circulatory collapse.

Summary: Information in this study was derived from the Registro Informatizado de la EnfermedadTromboEmbolica (RIETE) Registry of consecutive patients with acute venous thromboembolism (VTE). This is a multi-center observational registry that gathers and analyses data of clinical outcomes and treatment patterns in patients with symptomatic, objectively confirmed, acute VTE (deep venous thrombosis or PE). The current study focused on patients enrolled in the registry with PE who did not have pre-existing cardiac or pulmonary disease. Patients were classified into three clinical syndromes: pulmonary infarction, dyspnea, or circulatory collapse. Each group was analyzed with respect to clinical characteristics, laboratory findings, and 3-month outcomes.

There were 3391 patients (82% of the RIETE Registry) who presented with acute, symptomatic, objectively confirmed PE and who did not have chronic lung disease or heart failure. Of these patients, 1709 (50%) had pulmonary infarction, 1083 (32%) had isolated dyspnea, and 599 (18%) presented with circulatory collapsed. Of the entire group, 149 (4.4%) died during the first 15 days after presentation, and 2.5% of the patients with pulmonary infarction died between day 16 and day 90. Of those with isolated dyspnea, 6.2% died (odds ratio [OR], 2.6; 95% confidence interval [CI] 1.7 to 3.8) and 6.5% with circulatory collapse died (OR 2.7, 95% CI, 1.7 4.2). Thirty-one patients had recurrent PE. Fourteen of these patients had initially presented with pulmonary infarction, and five died (36%). Ten of the patients had initially presented with dyspnea and five died (50%). All seven patients with recurrent PE who initially presented with circulatory collapse died of their second PE.

Comment: The data make sense. Patients with pulmonary infarction usually present with pleuritic chest pain or hemoptysis reflecting peripheral emboli. Those with dyspnea and circulatory collapse have more central and larger emboli and thus have more extensive vascular occlusion. It is important to note that, no matter the initial presentation, recurrent PE ≤90 days is a highly fatal event.

Preoperative MDCT evaluation of the artery of Adamkiewicz and its origin

Takase K, Akasaka J, Sawamura Y, et al. J Comput Assit Tomogr 2006;30: 716-22.

Conclusion: Multidetector computed tomography (MDCT) allows identification of the entire length of the artery of Adamkiewicz and its origin in patients with thoracoabdominal aneurysms.

Summary: The artery of Adamkiewicz supplies the lower one third of the spinal cord and can arise from a posterior branch of an intercostal or lumbar artery. Identification of the artery of Adamkiewicz can be crucial in planning which, if any, intercostal vessels should be reimplanted or preserved during thoracoabdominal aneurysm repair. Proper identification and preservation of the artery of Adamkiewicz may reduce neurologic complications of thoracoabdominal aneurysm repair. This applies to both open and endovascular repair.

In this study, the authors evaluated the capability of an eight-slice multidetector CT scanner with 1-mm columniation, along with a rapid injection protocol, to evaluate the continuity of the artery of Adamkiewicz and its parent artery. Ten patients with thoracoabdominal vascular disease underwent MDCT, and in nine patients, the artery of Adamkiewicz was clearly visualized. The entire length of the intercostal or lumbar arteries leading to the artery of Adamkiewicz could be traced in eight patients. Three patients had occlusion of the parent artery of the artery of Adamkiewicz.

Comment: It makes sense that identification of a patent artery of Adamkiewicz and its parent vessel may be important in planning thoracoabdominal aneurysm repair. This may allow targeted preservation or reattachment of specific intercostal or lumbar vessels. It may also be equally important to know which patients have occluded parent vessels to the artery of Adamkiewicz. Perhaps in such cases, reattachment or preservation of intercostal lumbar vessels is not necessary in thoracoabdominal aneurysm repair. Equally possible, however, is that reattachment of additional vessels to preserve collateral flow will be required.

The pathogenesis of venous thromboembolism: Evidence for multiple interrelated causes

Brouwer JL, Veeger NJ, Kluin-Nelemans HC, et al. Ann Intern Med 2006;145:807-15.

Conclusion: Multiple genetic and environmental risk factors are additive in contributing to the risk of venous thromboembolism (VTE).

Summary: The authors sought to assess the effects of multiple thrombophilic defects and risk factors with respect to the absolute risk for VTE. This was a retrospective family cohort study conducted in a single university hospital in the Netherlands. Study subjects were 468 relatives of 91 probands with symptomatic hereditary deficiencies of protein C, protein S, or antithrombin. Relatives were tested for 10 thrombophilic defects in addition to the index deficiency. They were also assessed for exogenous risk factors such as immobilization, trauma, surgery, oral contraceptives, and pregnancy. Annual incidence and relative risk for VTE were compared in subjects with deficiencies of protein C, protein S, or antithrombin and in nondeficient relatives.

The annual incidence of VTE per 100 patient years in relatives with 0, 1, 2, or more additional thrombophilic defects or deficiencies were 1.16 (95% confidence interval [CI], 0.6 to 2.3), 1.75 (95% CI, 1.17 to 2.53), and 2.64 (95% CI, 1.67 to 3.96). The relative risk in nondeficient relatives was 0.06 (95% CI, 0.002 to 0.33) per 100 patient years. Adjusted relative risks were 16.3 (95% CI, 2.0 to 131.0), 50.3 (95% CI, 6.5 to 390), and 103 (95% CI, 12.5 to 843). In proband relatives with deficiencies of protein C, protein S, or antithrombin, 38% with no additional defects, 57% with one additional defect, and 81% with two or more additional defects had VTE by the age of 65 years. In comparison, only 5% of nondeficient relatives had VTE at 65 years ($P < .001$). In relatives with deficiencies of protein S, protein C, or antithrombin, and additional thrombophilic deficiencies or defects, exogenous risk factors further increased the risk of VTE from 1.2% to 2.5% per year (relative risk, 2.1; 95% CI, 1.1 to 4.2).

Comment: The basic message is simple. In individuals who already have a thrombophilic defect (protein C, protein S, or antithrombin deficiencies in this study), the risk of VTE increases further with additional thrombophilic defects or environmental risk factors. This, of course, is not the first time such an observation has been made. The magnitude of increased risk for relatives of probands with symptomatic protein C, protein S, or antithrombin deficiency is, however, sufficiently great that one wonders if prophylactic anticoagulation may be indicated in such patients.

Familial cervical artery dissections: Clinical, morphologic and genetic studies

Martin JJ, Hausser I, Lyrer P, et al. Stroke 2006;37:2924-9.