scopic bilateral upper dorsal sympathectomy in two patients in whom hyperhidrosis recurred after CT-guided blocks. Before this procedure, one to three blocks had been done, and the patients underwent surgery 1 to 4 months after the last block had been performed. Unusual problems were encountered.

1. Thick adhesions between the lung and parietal pleura were present over the upper sympathetic ganglia. These adhesions prompted us to revert to an open supraclavicular access in one of the two cases. This was the only patient in whom we failed to perform the sympathectomy by the thoracoscopic approach.

2. After separating the lung adhesions in the other patient, the parietal pleura was no longer transparent, and the ganglia could not be visually recognized. It was therefore necessary to identify the costovertebral junction and open the pleura accordingly.

3. Dissection of the sympathetic trunk was difficult because edema and fibrosis surrounded the ganglia. These findings and the temporary relief from hyperhidrosis after the phenol blocks indicate that the location of the injections was correct. The histologic examinations of the resected specimens showed within the scar tissue sympathetic ganglia with preserved autonomic cells. This finding suggests that phenol blocks, although properly located, do not necessarily destroy the ganglia. It also explains the high recurrence rate of palmar hyperhidrosis after CT-guided percutaneous phenol sympathicolysis. When the thoracoscopic approach is chosen after failed phenol sympathicolysis, conversion into an open procedure should be anticipated.

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24/41/69325

Does laminated intraluminal thrombus within abdominal aortic aneurysm cause anoxia of the aortic wall?

To the Editors:

The role that laminated intraluminal thrombus (ILT) has in abdominal aortic aneurysm (AAA) pathogenesis and rupture is unknown. Wolf et al.[1] concluded that the probability of rupture is increased in aneurysms with larger-volume ILT. This finding was based on measurement of ILT dimensions and AAA expansion rates from serial computed tomography scans, and may possibly be due to an accelerated degradation of the aortic wall that is facilitated by the presence of the thrombus. We hypothesize that one means by which this accelerated degradation occurs is the ILT serving as a barrier to normal physiologic oxygen diffusion from the lumen to the inner layers of the aortic wall. Because the vasa vasora provide O_2 to only the adventitia and outer media of the aorta, the intima and inner media rely on luminal diffusion for oxygen supply.[2] As a result of the ILT, we suggest that the aortic intima and media become anoxic. We performed a precursory assessment of this hypothesis by an order-of-magnitude analysis of the oxygen diffusion through the ILT layer based on Fick's law, and this was compared to the oxygen consumption rate required for normal aorta.

The O_2 consumption rate of smooth muscle has been reported to be 0.021 μmol/min/g.[3] In the normal human aorta the estimated weight of smooth muscle cells (SMC) per cm^2 of luminal surface area is 0.20 g.[4] As an illustrative example, consider a typically sized AAA with inner wall surface area of 80 cm^2. This aneurysm would contain $0.20 \times 80 = 16$ g of SMC. Hence the SMC alone contained in the AAA requires $16 \times 0.021 = 0.34$ μmol/min of O_2 for normal respiration, and the total requirements for all cellular components would be even greater.

The oxygen flowrate from lumen to AAA wall (Fig. 1) depends on the effective permeability of the ILT to O_2, which is given by the product of the solubility (α) and diffusivity (D) of O_2 within the ILT material. As an estimate of the amount of O_2 available at the AAA wall due to diffusion from the lumen and through the ILT, we use the Fick's law relation

$$VO_2 = \frac{\alpha \cdot D \cdot A \cdot \Delta PO_2}{\delta}$$

Here VO_2 is the oxygen diffusional flow rate, and ΔPO_2 is the drop in oxygen tension over a layer of ILT with thickness δ and surface area available for diffusion A. The ILT is principally a stagnant red-cell suspension entangled within a platelet and protein mesh. Studies have indicated that the effective permeability of red blood cell suspensions to O_2 is always comparable to or less than that of the suspending or continuous phase (i.e., plasma).[5] This is so because high O_2 solubility within red cell hemoglobin is offset by reduced diffusion within red cells and tortuous diffusion around cells. The presence of protein and platelets in the red-cell mesh would further diminish O_2 permeability of the ILT. Thus, a reasonable upper-bound estimate of the O_2 permeability within the ILT is the permeability of O_2 in water or plasma at physiologic temperature[5]:

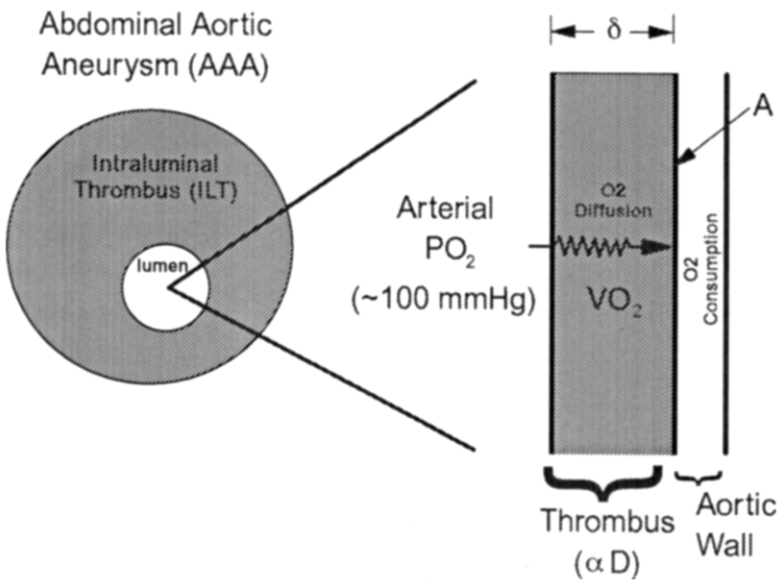

Fig. 1. Oxygen flow rate from lumen to AAA wall.

αD = (0.024 ml $O_2/cm^3/760$ mmHg) × (2.5×10^{-5} cm^2/s) = 7.9×10^{-10} ml $O_2/cm/mmHg/s$. The drop in oxygen tension across the ILT can be no greater than arterial blood PO_2 (100 mm Hg). Typical ILT thickness in AAA is 1 cm or more. For our example, A = 80 cm^2. With these values, the VO_2 through the ILT from the lumen to the aortic wall is calculated as 6.32×10^{-6} ml O_2/s. Using the gas law conversion of 2.243×10^{-2} ml $O_2/\mu mol$, this amounts to 1.69×10^{-2} μmol/min. This represents an O_2 flowrate 20 times less than that required for normal respiration of the SMC in AAA (1.69×10^{-2} μmol/min versus 0.34 μmol/min). The order-of-magnitude analysis presented here indicates that ILT may offer a significant barrier to O_2 transport to the AAA wall. Although our analysis was idealized for simplicity, parameters were chosen to estimate a maximal diffusive flow through the ILT, and none of our choices could likely account for the 20-fold difference between the estimated maximum available oxygen and that needed for normal respiration by the SMC alone in the AAA. We believe that poor diffusion of O_2 through the ILT within AAA causes anoxia, followed by necrosis and diminished resistance by the aortic wall to physiologic distending pressures. This hypothesized effect of ILT may potentially be important in the understanding of the natural history of AAA and we are investigating this further.

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24/41/69501

Regarding "Endovascular arterial intervention: Expression of concern"

To the Editors:

Doctor John M. Porter's observations in his commentary (J VASC SURG 1995;21:995-97) hit the mark. My only disappointment is that he did not specifically address the most pervasive problem: uncontrolled coronary artery interventions performed by invasive cardiologists who self-refer their patients.

As with peripheral endovascular interventions, there is sparce scientific evidence that coronary angioplasty or stent procedures have any long-term benefit, especially as compared with coronary bypass. The same specialists who