Effect of Internal Mammary Arterial Mobilization on Sternal Blood Flow

D

carried out in a cadaveric situation and did not study the in vivo effects of surrounding intact peristeum and musculature, as well as intact intercostal vessels.

Bahn and colleagues (see page 878) have made an admirable attempt to study sternal blood flow in an in vivo situation after isolation of the left internal mammary artery. Although the number studied was small, the authors concluded rather convincingly that blood cell flux, as measured by laser Doppler flowmetry, seems to be unchanged after complete mobilization of the internal mammary artery. This, to my knowledge, is the first study to record these data in vivo.

The first question that must be asked is: "Is laser Doppler flowmetry adequate to obtain such data?" I believe the answer to that question is affirmative. Laser Doppler flowmetry has been utilized in both the laboratory setting and the clinical setting and found to be an excellent means to monitor blood cell flow in both cortical and cancellous bone. In cancellous bone, the depth of penetration of the helium neon red laser has been recorded up to 14 mm with a threshold thickness of recording of 2.7 mm. Laser Doppler flowmetry has also been shown to be useful in the experimental measurement of bone blood flow in sheep, rabbits, dogs and guinea pigs.

In our clinical experience (unpublished data), the laser Doppler flowmetry unit is used to measure the intact vascular status of free tissue transfers on almost a daily basis in complex head and neck reconstruction using many types of osseous and osseocutaneous transfers. We are convinced of its value and reproducibility in reading blood cell flux within bone.

I have never held the belief that complete isolation of the internal mammary artery completely devascularizes the sternum in an in vivo situation. If the peristeum is carefully left intact, as well as the costal insertions of the rectus abdominis and pectoralis muscles, I believe there is enough cross circulation intact from the thoracoacromial vascular leach of the pectoralis muscle, as well as reverse flow from the musclulophrenic and inferior epigastric system through the rectus abdominis muscle, to maintain viability of the osteocytes within the sternum. Why, then, does there seem to be an increased incidence of sternal infection when both IMAs are elevated? This is perhaps due to many factors—prolonged operative time or more complex medical problems with the patient. Perhaps the sternum maintains normal basal flow values; however, it may lack the inherent responsiveness of hyperemia and increased vascular flow needed to fight off a contaminating lode of bacteria. This is available to a sternum with an intact internal mammary artery, although this is certainly speculative.

I commend the authors for their study. I hope they will continue their work so that a larger patient
population can be observed. Perhaps correlation between patients who became infected and blood cell flux numbers would help us determine standardized values that may be of help to all who deal with these sternal infections in our day-to-day practices.

N. Bradly Meland, M.D.
Rochester, Minnesota

REFERENCES

2 Swiontkowski MF, Hagen K, Shack RB. Adjunctive use of laser Doppler flowmetry for debridement of osteomyelitis. Ortho Trauma 1989; 3:1
4 Swiontkowski MF, Tepic S, Perren SM, Moor R, Ganz R, Bahn BA. Laser Doppler flowmetry for bone blood flow measurement: Correlation with microsphere estimates and evaluation of the effect of intracapsular pressure on femoral head blood flow. Ortho Research 1986; 4:392

Recidivism Revisited
A Case for Steroids in Relapsing COPD?

Patients who “fail to do well,” (relapse promptly after treatment) may be considered guilty of contributing to their own ill health through non-compliance. At least one study has tried to prevent such recidivism in patients with reversible airflow obstruction by injecting a long-acting intramuscular corticosteroid at the time of the initial visit. In patients with COPD, the causes for relapse may be multiple, and include exacerbation of airflow obstruction which theoretically could respond to steroid administration. Proof of a causal role for reversible airflow obstruction in relapses of COPD is still difficult to obtain. Although hospitalized patients treated with steroids showed a greater responsiveness to bronchodilator therapy after 12 hours, no impact of steroid on length of hospital stay was reported and steroid administration to patients in an emergency department (ED) setting did not reduce admission rates.

The study by Murata et al in this issue (see page 845) suggests that steroid administration has a beneficial effect on relapse rate in patients with COPD who are prone to relapse (my emphasis). Although their study is retrospective, the criteria used to match visits in the same patient include the major therapeutic confounders, and the post-matching analysis of other variables which might be considered to influence outcome reveals no major difference. The reduction in relapse rates they report is of clinically important magnitude, and if confirmed in a prospective study would provide strong evidence for the use of steroids in patients with COPD who are prone to relapse.

I have emphasized the relapsing aspect for four reasons. First, this is a population of interest clinically, in that it represents 20 percent of patients with respiratory distress seen in the ED. Second, because the negative results of previous trials may have occurred because the “relapse prone” subgroup was eclipsed by the greater number of non “relapse prone” patients. Third, because the “relapse prone” population can be identified by history without recourse to further investigations (sputum or blood eosinophilia) which have been variably successful in identifying stable COPD patients who improve on steroids. Fourth, because although Murata et al state that there was no difference between their “relapse prone” and non “relapse prone” patients with respect to bronchodilator response and that they excluded patients with asthma, a 20 percent improvement in FEV1 was seen in both groups of patients. This suggests that assessment of such reversibility of airflow obstruction may be unimportant in deciding whether to give steroids or not, and that the key factor should be the “relapse prone” history.

From the beneficial effects of steroids on relapse rate in “relapse prone” COPD patients, and considering the possible beneficial properties of steroids in such decompensated COPD patients as discussed by Murata et al, it is likely that modulation of the inflammatory response process plays a key role. Viewed in this light, the therapeutic issues in management of the “relapse prone” COPD patient appear very similar to those relating to the treatment of acute bronchial asthma, with increasing agreement that steroids should be used early rather than late. The optimal steroid regimen is still unclear. Murata et al used a combination of intravenous and oral steroids. It is probably important, as they suggest, that steroids be continued for some days to allow reversal of the inflammatory response. The need for parenteral steroid administration in addition to oral is not proven, and should be studied in view of the very infrequent, but potentially severe acute myopathy reported with parenteral steroid administration. The potential role of inhaled steroid using the higher dose metered aerosols should also be explored.

If the findings in the study of Murata et al are substantiated, then the recidivist when a “relapse prone” patient relapses is likely to be not the patient, but the physician who did not prescribe steroids.