We read with interest Junemann-Ramirez and colleagues’ article [1] on esophagogastric anastomotic leaks and their analysis of predictive factors, management and survival. We agree with the authors that esophagogastric anastomotic leaks are the most devastating complications after esophageal resection and that they carry high morbidity and mortality rates. The choice of surgical procedure has a lot to do with the ultimate outcome of the patient. We feel that the consequences of an anastomotic leak are far worse than with an intrathoracic anastomosis compared with a neck anastomosis. The Ivor Lewis esophagectomy has neither the advantages of a transhiatal esophagectomy (in avoiding a thoracotomy) nor does it have the advantages of a transthoracic (three-hole) total esophagectomy (where a neck anastomosis avoids the morbidity of mediastinitis in case of a leak). A three-hole esophagectomy also enables radical supracarinal mediastinal lymphadenectomy.

The authors’ conclusions that patients treated conservatively fared better than those who underwent surgical intervention could clearly be explained by a selection bias as it is likely that clinically stable patients would have been treated conservatively and sicker patients with mediastinitis would have been reoperated. We also fail to understand how a pyloric drainage procedure could reduce anastomotic leaks—decompression of the gastric tube could easily and effectively be achieved by nasogastric tube decompression. In our unit (we perform an average of 160 esophagectomies a year), we perform a three-hole total esophagectomy in all patients with a neck anastomosis, but do not perform a formal pyloric drainage procedure. None of the leaks we encountered could be explained by non-performance of a gastric drainage procedure. Most true anastomotic leaks in our unit are managed conservatively as the leak gets localized in the neck with no signs of mediastinitis. Gastric tube necrosis or ischemia, on the other hand, are managed aggressively by immediate surgical reoperation with disconnection of the anastomosis for reconstruction by a coloplasty as an interval surgery [2]. Needless to state, the latter group fare worse than the former.

References


Letter to the Editor

Atrial fibrillation after off-pump versus on-pump: Are we not missing a common pathophysiological pathway?

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As it could be expected, the retrospective clinical trial of Dr Enc et al. [1], aimed to investigate the difference in the postoperative incidence of atrial fibrillation (AF) between Off-pump and On-pump aorto-coronary grafting (ACG), is followed by a reply from Dr Raja [2] embellished with the scholarly statistical recalls. We agree with both authors in that the final answer will be brought about by a prospective randomised multi-centre trial, hence the intricate nature of various factors implicated in the occurrence of post-ACG atrial fibrillation makes its design difficult, if not possible, to be ruled by.

In addition to already recognised factors involved in, we would emphasise that the onset of new iatrogenic perfusion following ACG, deemed as aorto-coronary perfusion, can be a factor in the occurrence of AF as a unique pathophysiological pathway by inducing functional atrial ischemia. Although calling for being investigated, it is our belief that the incidence of postoperative AF would be the same in its entirety as the final product of both On-pump and Off-pump procedures is represented by aorto-coronary perfusion.

By opening the epicardial arteries that are functionally separated from systemic circulation, ACG sets up occurrence of systolic coronary perfusion disturbances, especially the onset of an early systolic retrograde flow into the graft, known classically as the competition flow, importance of which is inversely related to the degree of recipient artery stenosis [3]. As atrial perfusion occurs during the systole (the atrial diastole) [4], one can speculate that the latter could result in functional atrial mal-perfusion, playing as a substratum for AF. Yet, considering the increased sensitivity of the senile atrial myocardium to ischemia, the latter might elucidate why the age is recognised as the most powerful risk factor for postoperative AF.