ANNALS OF SURGERY
Vol. 230, No. 6, 826–830

To the Editor:

Drs. Azakie, McElhinney, Higashima, Messina, and Stoney have described the reconstruction of the innominate artery for symptomatic atherosclerotic occlusive disease in 94 patients.[1] Their preferred approach, a transsternal endarterectomy of the innominate artery, was used in 68 patients; a transcervical approach for the endarterectomy was used in 4 patients, and an aortoinnominate bypass was used in 22 patients. There were three (3.2%) perioperative deaths, and severe postoperative events occurred in eight (8.5%) patients, of whom four (4.3%) experienced a stroke, two (2.1%) had a myocardial infarction, and two (2.1%) had a transient ischemic attack. Prior cerebrovascular procedures had been performed in 17 patients. A number of these patients had significant risk factors: tobacco smoking (82%), hypertension (49%), coronary artery disease (17%), hypercholesterolemia (16%), and diabetes mellitus (11%).

While we believe that an endarterectomy is a suitable procedure for innominate artery occlusions in most younger, healthy patients, its use in high-risk patients must be questioned. Patients who also have very advanced age (>70 years), severe coronary artery disease, extensive involvement of other extracerebral arteries, or severe pulmonary diseases would benefit from procedures that avoid a sternotomy. We have used an axilloaxillary bypass for reconstruction of occluded innominate arteries in five patients who were at a significantly high risk for complications.[2] A subcutaneous, gently U-shaped tunnel is made over the sternum, from axillary artery to axillary artery. Either an 8- or 10-mm Gortex graft is then passed through the tunnel, and end-to-side anastomoses are made between the graft and the proximal portion of the axillary arteries to lessen the risk of injury to the brachial plexus and tension on the graft. A carotid endarterectomy can also be done if significant carotid stenosis is present. The technique has been previously described.[2]

The results of 300 axilloaxillary bypasses indicates that there is only rare perioperative mortality.[2–8] The procedure is very safe and avoids manipulation of the innominate artery and the risk of embolization of residual plaque. Our 10-year primary patency rate for 39 axilloaxillary bypasses for innominate/subclavian occlusive disease is 88%, and the secondary patency rate is 91%.[2] We have had one perioperative transient ischemic attack but no mortality. Complete relief from symptoms has occurred in more than 85% of our patients. Others have had similar results. When lesions exist in the innominate/subclavian and carotid arteries, a simultaneous carotid endarterectomy and axilloaxillary bypass will improve cerebral blood flow, and our 10-year secondary patency rate is 93%.

High-risk patients and those with other life-threatening diseases, such as cancer, would be ideal candidates for the axilloaxillary bypass, and transsternal innominate endarterectomy should be reserved for young, healthy patients.

JOHN B. CHANG, MD
THEODORE A. STEIN, PHD
Long Island Vascular Center
Roslyn, New York

References

1. Azakie A, McElhinney DB, Higashima R, et al. Innominate artery reconstruction: over 3 decades of experience. Ann Surg 1998; 228:402–410.
2. Chang JB, Stein TA, Liu JP, Dunn ME. Long-term results with axillo-axillary bypass grafts for symptomatic subclavian artery insufficiency. J Vasc Surg 1997; 25:173–178.
3. Mozersky DJ, Sumner DS, Barnes RW, et al. The hemodynamics of the axillary-axillary bypass. Surg Gynecol Obstet 1972; 135:925–929.
4. Posner MP, Riles TS, Ramirez AA, et al. Axilloaxillary bypass for symptomatic stenosis of the artery. Am J Surg 1983; 145:644–646.
5. Myers WO, Lawton BR, Ray F, et al. Axillo-axillary bypass for subclavian steal syndrome. Arch Surg 1979; 114:394–399.
6. Lowell RC, Mills JL. Critical evaluation of axilloaxillary artery bypass for surgical management of symptomatic subclavian and innominate artery occlusive disease. Cardiovasc Surg 1993; 1:530–535.
7. Mingoli A, Feldhaus RJ, Farina C, et al. Comparative results of cartoid-subclavian bypass and axillo-axillary bypass in patients with symptomatic subclavian disease. Eur J Vasc Surg 1992; 6:26–30.
8. Owens LV, Tinsley Jr. EA, Criado E, et al. Extrathoracic reconstruction of arterial occlusive disease involving the supraaortic trunks. J Vasc Surg 1995; 22:217–222.

March 30, 1999

Author Reply:

Dr. Chang acknowledges the value of innominate endarterectomy for the treatment of innominate atherosclerosis in younger, healthy patients (age and health not defined). He questions its use in high-risk patients (>70 years, with severe coronary disease, other extracranial disease, or severe pulmonary disease), and he recommends these patients have a procedure that avoids a sternotomy, such as axilloaxillary bypass. Dr. Chang cites the transsternal operative mortality of 3.2% and severe postoperative events, including stroke (4.3%), transient ischemic attack (2.1%), and myocardial infarction (2.1%), among the 94 patients who underwent innominate reconstruction. In fact, only one death has occurred since 1966, and no deaths have occurred in the last 70 consecutive reconstructions. Among the six postoperative neurologic events, only one failed to resolve by hospital discharge. Thus, the mortality and neurologic morbidity in patients undergoing median sternotomy and innominate reconstruction was extremely low (1%) in the majority of treated patients.

The pattern of occlusive innominate atherosclerosis produced hemispheric retinal or right upper extremity embolization in more than half of the patients reported, and required removal or exclusion of the embolic source in these patients. We chose transsternal innominate endarterectomy or bypass believing that axilloaxillary grafting was ineffective treatment for this disease pattern.

Finally, Dr. Chang points to the safety of axilloaxillary bypass and cites only a rare operative mortality, which is also true for transsternal innominate operations in the last three decades. The

axilloaxillary bypass produced 10-year primary patency rate of 88%, as contrasted with 100% for innominate endarterectomy, and 85% for innominate bypass.

In conclusion, transsternal endarterectomy or bypass for the treatment of innominate atherosclerosis is safe, effective, and durable. In the rare patient unsuited for transsternal repair, endovascular angioplasty and stenting are attractive options, to be considered along with axilloaxillary bypassing, as Dr. Chang recommends.

RONALD J. STONEY, MD
Division of Vascular Surgery
University of California San Francisco
San Francisco, California

August 26, 1997

To the Editor:

The article by Farrell et al, "Pancreatic resection combined with intraoperative radiation therapy for pancreatic cancer,"[1] may be overly optimistic about its conclusions. They are to be congratulated on their minuscule complication rate. However, I believe they have mainly demonstrated the *safety* of intraoperative radiation therapy, not its efficacy.

Using the Rocky Mountain Cancer Center data for comparable survival statistics may be misleading. The patients in this reference group all underwent pancreaticoduodenectomies, whereas only 71.5% of the study group had Whipple resections. The two patients with distal pancreatectomies may have had more localized or contained disease. With such a small number overall, this difference may significantly influence median or 5-year survival. A comparison by stage would further clarify this question, and a comparison by cell type would answer the related issue raised by the authors in their comments on survival. In addition, the patients in the study group received both intraoperative radiation therapy (IORT) and external beam radiation. Postoperative adjuvant radiotherapy, with or without 5-fluorouracil, has already been demonstrated to enhance local control, and therefore might prolong median survival with no impact on long-term results. How can the authors be certain that the effects demonstrated in their series are due to the benefit of IORT alone?

Further amplification of the differences between the study group and the controls would answer several other questions. Did any of the controls with malignancy receive adjuvant therapy, especially external beam radiation? Was adjuvant therapy related to complications in the control group? What were the survival curves for pancreatic and nonpancreatic carcinomas? And does IORT really protect against anastomotic leak or stricture?

As the authors indicate, their survival curve approaches the known statistic for survival at 5 years. It is unclear that their study group is large enough, or free enough from confounding factors, to declare IORT beneficial. I agree that this modality may be used without adding significant morbidity based on their data, but they have not demonstrated that it can prolong median survival when used in comparable populations. Their conclusion that IORT is at least as good as any other therapy may also be interpreted to read that we have yet to discover a uniformly useful adjunct to surgery for pancreatic cancer.

CHRISTOPHER M. HUIRAS, MD, FACS
Department of Surgery
Franciscan Skemp Medical Center
Mayo Health System
La Crosse, Wisconsin

Reference

1. Farrell TJ, Barbot DJ, Rosato FE. Pancreatic resection combined with intraoperative radiation therapy for pancreatic cancer. Ann Surg 1997; 226:66–69.

February 8, 1999

Author Reply:

Dr. Huiras correctly points out that our comparison of patients undergoing pancreatic resection with intraoperative radiation therapy as well as postoperative adjuvant radiation therapy and chemotherapy might not be a comparable group to match against the Rocky Mountain Cancer Center Data, because the latter group were all pancreaticoduodenectomies, while only 71.5% of our study group had Whipple resections. On the other hand, one would guess that, if anything, our addition of distal pancreatectomies, for what were body/tail lesions, would be a disadvantage for our group in a comparison because in general, body/tail lesions have a poorer outlook that those in the head because they are discovered even later.

I certainly agree with Dr. Huiras that what we have mainly demonstrated in our article was the safety of the intraoperative therapy rather than its efficacy in terms of median or long-term survival. The many other questions he raises, about the survival curves for pancreatic cancers and whether or not intraoperative radiation therapy protects against anastomotic leak or stricture, are not questions we could answer from the initial small sample.

We are now just completing an analysis of more than 40 patients who underwent exclusively Whipple pancreaticoduodenectomies and although it is not a prospective randomized controlled study, we will—in comparison with other national samples such as the SEER group and our own historical controls—be able to make certain conclusions about the likelihood of the effect of this treatment plan on median and long-term survival. We certainly hope to have answers to the other questions Dr. Huiras raised in our larger sample as well. The manuscript is presently in preparation and will be submitted first to *Annals of Surgery* for review.

FRANCIS E. ROSATO, MD
Department of Surgery
Jefferson Medical College
Thomas Jefferson University
Philadelphia, Pennsylvania

August 4, 1998

To the Editor:

We read with interest the article by Branum et al,[1] in which the authors investigated postoperative and long-term results in 50

patients after surgery for necrotizing pancreatitis. The reported mortality of 12% is impressively low and represents an excellent result. However, the incidence rates of endocrine (40%) and exocrine (23%) insufficiencies 40 months after surgery seem high and justify a few remarks.

1. The detection of exocrine and endocrine insufficiencies is heavily influenced by the sensitivity of diagnostic tests applied. Oral glucose tolerance tests, serum insulin or c-peptide levels and pancreatic exocrine function tests as the secretin-cholecystokinin test yield much higher rates of impaired function than fasting serum glucose levels or the clinical evidence of steatorrhea. The authors did not indicate by which methods pancreatic insufficiencies were diagnosed.
2. The development of exocrine or endocrine insufficiencies after surgery depends on the amount of functioning pancreatic tissue preserved, and is determined by the following factors:
   a. Pancreatic function before surgery: In the reported series, 20% of patients presented with diabetes and 12% with exocrine insufficiency before treatment indicating a history of previous bouts of acute or chronic pancreatitis leading to parenchymal destruction;
   b. Amount of necrosis and tissues debrided: In a study we found significantly higher rates of diabetes in patients in whom partial pancreatic resections had to be performed due to the extension of necrosis compared to patients after debridement only[2];
   c. Length of the interval between surgery and follow-up: Doepel and associates[3] demonstrated increasing rates of diabetes depending on duration of follow-up (50% after 6 years); and
   d. Rate of recurrent attacks of pancreatitis mainly due to ethanol intake leading to further destruction of pancreatic tissue.

According to data presented by Branum et al,[1] Broome et al,[4] and our own experience,[2,5] however, exocrine and endocrine insufficiencies after surgical treatment of necrotizing pancreatitis may be easily treated in the great majority of patients and do not adversely affect the good long-term results.

Stephan Kriwanek, MD
Michael Gschwantler, MD
Christian Armbruster, MD
Department of Surgery
Rudolfstiftung Hospital
Vienna, Austria

References

1. Branum G, Galloway J, Hirchowitz W, et al. Pancreatic necrosis results of necrosectomy, packing, and ultimate closure over drains. Ann Surg 1998;227:870–877.
2. Kriwanek S, Armbruster C, Dittrich K, et al. Long-term results after surgery for acute necrotizing pancreatitis. Chirurgie 1996;67:244–248.
3. Doepel M, Eriksson J, Halme L, et al. Good long-term results in patients surviving severe acute pancreatitis. Br J Surg 1993;80:1583–1585.
4. Broome AH, Eisen GM, Harland RC, et al. Quality of life after treatment for pancreatitis. Ann Surg 1996;223:665–672.
5. Kriwanek S, Armbruster C, Dittrich K, et al. Long-term outcome after open treatment of severe intra-abdominal infection and pancreatic necrosis. Arch Surg 1998;1233:140–144.

To the Editor:

We read with interest the article of Sugiyama and Atomi.[1] Because our department has a large experience with intraductal papillary mucinous tumors (IPMT), the paper prompted several comments.

The authors report the high rate of malignant transformation, and conclude that surgery should be always indicated. Nevertheless, we have to qualify the opinion that IPMT would have a favorable prognosis, as stated in the conclusion of the abstract. Indeed, our experience, and others', does not confirm this so-called favorable prognosis, and demonstrates that survival after surgery is closely dependent on the presence of invasive carcinoma.[2–5] The recent increase of our already published experience confirmed our previous conclusions: in a series of 31 operated patients, the 5-year survival is 94% when only dysplasia is present (17 patients, no tumor-related death), compared with 19% in case of microinvasive or invasive carcinoma (14 patients). Furthermore, the preoperative diagnosis of malignancy is very difficult to assess, especially because of the absence of reliable markers of malignant transformation. When malignant transformation becomes evident at morphologic investigations (computed tomography [CT], magnetic resonance imaging [MRI], endoscopic retrograde cholangiopancreatography [ERCP]), it is frequently too late to offer the patient the chance to be cured. Although benign IPMT obviously has a slow growing rate, it can turn into a very aggressive tumor when malignant transformation occurs. Surgical resection is therefore indicated before that clinical evolution or investigations suggest malignant transformation.

Intraductal papillary mucinous tumors can be considered a disease of the entire pancreatic ductal system, and the need to perform a total pancreatectomy is reported in some series, often based on intraoperative frozen sections revealing involvement of the pancreatic margin by the tumor.[1,3,6] While it is absolutely necessary to exclude malignant infiltration of the surgical margin, the importance of the presence of microscopic dysplasia lesions at the pancreatic margin should be questioned. Indeed, in our experience on 23 operated patients alive after 6 months of follow-up, 13 (57%) demonstrated dysplasia of the resection margin upon microscopy of the surgical specimen. Total pancreatectomy was never performed. All of these patients have been regularly followed and controlled by CT, MRI, or ERCP. Only one patient experienced a recurrence with a macroscopic IPMT on the remaining pancreas, 66 months after a caudal pancreatectomy.[2] In cases of benign IPMT, recurrence after surgery is rare and occurs late, regardless of the presence or absence of microscopic dysplasia on the remaining pancreas. Considering the well-known side effects of total pancreatectomy, we advocate restricting the resection to the pancreas involved by macroscopic disease. The role of frozen sections could be limited in ruling out malignancy at the surgical margin. Pancreatectomy should not be extended when only microscopic dysplasia is found at intraoperative frozen sections of the pancreatic margin. Furthermore, when surgery is performed early in the evolution of the disease, the risk of malignancy is small, and limited resections, as segmental or caudal pancreatectomies, are often possible.

When dealing with IPMT, the guideline should be early surgery,

before the appearance of any signs of malignant transformation, thus allowing limited but complete resection of all the macroscopic disease.

JEAN VAN DE STADT, MD
JEAN CLOSSET, MD
MICHEL GELIN, MD
Department of Digestive Surgery
Erasme Hospital
Free University of Brussels
Brussels, Belgium

References

1. Sugiyama M, Atomi Y. Intraductal papillary mucinous tumors of the pancreas: imaging studies and treatment strategies. Ann Surg 1998; 228:685–691.
2. Azar C, Van de Stadt J, Rickaert F, et al. Intraductal papillary mucinous tumors of the pancreas: clinical and therapeutic issues in 32 patients. Gut 1996; 39:457–464.
3. Loftus EV Jr, Olivares-Pakzad BA, Batts KP, et al. Intraductal papillary-mucinous tumors of the pancreas: clinicopathologic features, outcome, and nomenclature. Members of the Pancreas Clinic and Pancreatic Surgeons of Mayo Clinic. Gastroenterology 1996; 110:1909–1918.
4. Traverso WL, Peralta EA, Ryan JA Jr, Kozarek RA. Intraductal neoplasms of the pancreas. Am J Surg 1998; 175:426–432.
5. Barbe L, Ponsot P, Vilgrain V, et al. Intraductal papillary mucinous tumors of the pancreas: clinical and morphological aspects in 30 patients. Gastroenterol Clin Biol 1997; 21:278–286.
6. Rivera JA, Fernandez-del Castillo C, Pins M, et al. Pancreatic mucinous ductal ectasia and intraductal papillary neoplasms: a single clinicopathologic entity. Ann Surg 1997; 225:637–646.

January 25, 1999

To the Editor:

We read the article "Neurovascular compression in the thoracic outlet syndrome" and the accompanying discussion.[1] The authors and participants discuss one of the most controversial subjects in clinical medicine without any mention of the serious questions raised in the neurologic and rehabilitation literature about the diagnosis and surgical treatment of thoracic outlet syndrome (TOS).[2–5] For an article to stand on scientific grounds, the authors must confront challenges from opposing schools of thought. Too often, we believe, surgical articles on TOS assert the benefits of surgery without informing the reader that there are opposing positions about the diagnostic methods used and the means of determining the success of TOS surgery.

We are troubled that the authors describe the electrodiagnostic methods they use to detect the type of TOS they operate on so frequently without mentioning that their procedure(s) have been challenged in the literature, including the editorial pages of the *New England Journal of Medicine*.[6–9]

Moreover, we are concerned by the absence of any discussion on the use of scientific tools (e.g., randomized controlled studies, independent evaluations) in assessing surgical success. In a recent article "Surgery and the randomized controlled trial,"[10] Solomon and McLeod note that "Surgeons have been criticized for a lack of adequate scientific assessment of new and old techniques and technology." Because this statement unfortunately applies to TOS surgery, we urge an important caveat for the reader: be careful in accepting operative success rates when the assessing group is not totally independent of the surgical team.[11] We are aware of only one series in the literature where TOS was evaluated by an independent team.[12] They described its success as "dismal."

In regard to controversial disorders, we respectfully suggest that the authors and the editorial reviewers have an obligation to their readers to point out that there are opposing views concerning both the methods of diagnosis used and the surgical treatments employed.

MICHAEL CHERINGTON, MD
Neurology Section
Centura Health
Denver, Colorado
ASA J. WILBOURN, MD
Department of Neurology
Cleveland Clinic
Cleveland, Ohio

References

1. Urschel HC, Razzuk MA. Neurovascular compression in thoracic outlet. Ann Surg 1998; 228:609–617.
2. Nelson DA. Thoracic outlet and dysfunction of the temporomandibular joint: proved pathology or pseudosyndromes? Perspect Biol Med 1990; 33:567–576.
3. Lindgren KA. Conservative treatment of thoracic outlet syndrome: a 2-year follow-up. Arch Phys Med Rehab 1997; 78:373–378.
4. Wilbourn AJ. The thoracic outlet syndrome is overdiagnosed. Arch Neurol 1990; 47:228–230.
5. Cherington M. A conservative point of view of the thoracic outlet syndrome. Am J Surg 1989; 158:394–395.
6. Urschel HC, Razzuk MA. Management of the thoracic-outlet syndrome. N Engl J Med 1972; 286:1140–1143.
7. Cherington M. Ulnar conduction velocity in thoracic-outlet syndrome. N Engl J Med 1976; 294:1185.
8. Wilbourn AJ, Lederman RJ. Evidence for conduction delay in thoracic outlet syndrome is challenged. N Engl J Med 1984; 310:1052–1053.
9. Relman AS. Responsibilities of authorship: where does the buck stop? N Engl J Med 1984; 310:1048–1049.
10. Solomon MJ, McLeod RS. Surgery and the randomised controlled trial: past, present, and future. Med J Austral 1998; 169:380–383.
11. Johnson AG, Dixon JM. Removing bias in surgical trials. Br Med J 1997; 314:916–917.
12. Franklin G, Fulton-Kehoe D, Smith-Weller T. Outcome of surgery for thoracic outlet syndrome in Washington worker's compensation, 1986–1991. Neurol 1994; 44(suppl 2):A389.

April 1, 1999

Authors' Reply:

Drs. Cherington and Wilbourn surprise us by questioning the benefits of surgery as reported and suggesting that conservative management was not employed.

All of the patients with predominantly nerve compression were treated conservatively for at least 3 months, many for years—far too long prior to considering surgical relief. Many of these patients were retrieved from psychiatric clinics, after worsening of their

clinical manifestations as showing neurologic deficit in the hand or peripheral embolization. Many had been treated for years by neurologists who were unaware of objective methods of diagnosis in TOS management, or who had misdiagnosed them with some other "nontreatable" neurologic problem.

All patients included were evaluated by at least two other physicians and most by myriad clinicians including neurologists, physiatrists, cardiologists, and psychiatrists. No patient with nerve compression was operated without a complete trial of conservative management, which had failed as ascertained by independent observers.

Most patients evaluated in the clinic and hospital were successfully treated by conservative management. Less than 10% of all patients diagnosed as TOS required surgical intervention.

The electrodiagnostic tests employed were varied and included conduction velocities and F-wave studies at Baylor. These have been carefully evaluated and have clearly demonstrated their objective value. Anyone who has had difficulty performing the conduction velocities across the thoracic outlet and has taken the trouble to spend time with the originators has been very comfortable with the reliability and the reproducibility of test results. Neither Dr. Cherington nor Dr. Wilbourn, who have been previously publicly invited to visit, have taken advantage of the opportunity.

The suggestion that the surgical procedure may be evaluated in a randomized controlled study, such as would be applied to a drug, is naive in the present day environment of litigation. All patients had objective independent evaluations totally separate from the decision to undergo surgery. All patients were independently evaluated postoperatively about the type of result achieved.

The multitude of practicing physicians and patients using this protocol have found objective improvement and are well satisfied with the results of this approach.

HAROLD C. URSCHEL, JR., MD
MARUF A. RAZZUK, MD
Dallas, Texas