

feasible, as the still-rare reports of valvular or coronary bypass operations demonstrate.

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Histologic Appearance of Transmyocardial Laser Channels After 4½ Weeks

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Preliminary results of clinical studies suggest that transmyocardial laser revascularization is an effective treatment for patients with chronic angina that cannot be treated by other means. The mechanism of this effect remains controversial. We present autopsy results from a patient obtained 4½ weeks after operation that show that the channels do not maintain patency. Further work is needed to determine the frequency of channel patency and its relation to clinical benefit.

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Transmyocardial laser revascularization (TMLR) is an experimental treatment for patients with angina that is refractory to medical therapy who are also unsuitable for treatment with coronary artery bypass grafting or angioplasty [1-3]. A high-energy laser is used to make ~1 mm channels through the myocardium into the left ventricular chamber. Although the epicardial region of the channel seals within a few moments after creation, it has been hypothesized that the remainder of the channel remains patent and that blood can flow from the left ventricular chamber to directly perfuse the myocardium, totally bypassing the epicardial vasculature [1, 4, 5].

Although results of clinical studies continue to suggest that this treatment is effective in relieving angina, data from animal studies have been mixed. Results of some

studies have suggested that channels remain patent and protect against infarction [5], but other studies have challenged these primary hypotheses [6, 7]. Although limitations of clinically available techniques have made it impossible to test whether blood can flow *through* TMLR channels in the hearts of treated patients, histologic sections obtained at the time of autopsy have been interpreted by previous investigators as demonstrating patent channels weeks to months after the procedure [1, 2].

In this case report, we present autopsy results obtained from a patient who died 4½ weeks after TMLR of complications unrelated to a failure of the treatment. The results show that the channels do not maintain patency in this patient.

The patient was a 71-year-old man with a history of diabetes, hypertension, and smoking. He suffered an inferior wall myocardial infarction 18 years ago (1977), after which he underwent three-vessel bypass grafting with vein grafts. He was free of angina for approximately 17½ years. Six months before referral to Columbia-Presbyterian Medical Center, he reported a crescendo pattern of exertional and rest angina despite gradual increase in medical therapy, which ultimately included atenolol, diltizem, isosorbide dinitrate, furosemide, and lisinopril. Cardiac catheterization revealed a patent sequential graft to the first diagonal branch of the left anterior descending artery and first obtuse marginal artery, an occluded graft to the posterior descending artery, a totally occluded proximal right coronary artery, a very small left anterior descending artery, a small and diffusely diseased second obtuse marginal artery, and a long posterior descending artery (wrapping around the apex) with diffuse disease that filled via collaterals from the left circumflex artery. The diffuse nature of the disease in both the posterior descending and second obtuse marginal arteries rendered the patient unsuitable for treatment with either coronary artery bypass grafting or angioplasty, and it was decided to treat the patient medically. However, he continued to have symptoms of angina and was referred for consideration of TMLR in January 1995. An exercise thallium test was performed. He exercised 6 minutes on a modified Bruce protocol to a peak heart rate of 125 beats/min; he stopped exercising because of shortness of breath, but denied chest pains. Comparison of rest and exercise thallium scans revealed a fixed posterior defect and reversible defects in the inferoapical, anteroapical, and lateral walls.

Transmyocardial laser revascularization was performed on February 7, 1995, with The Heart Laser (PLC Systems, Inc, Milford, MA), which consisted of applying a total of 25 laser shots (40 J/channel) over the inferior, apical, and anterolateral regions of the heart. Twenty-four of these were confirmed to penetrate into the ventricular cavity as evidenced by visualization of bubbles filling the left ventricle on the transesophageal echocardiogram. The patient did well after the operation and was discharged on the seventh postoperative day. However, he presented on postoperative day 24 after a bout of prolonged chest pain at rest. Cardiac catheterization was

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performed, which revealed a new critical stenosis in the previously patent saphenous vein graft. The lesion was dilated and a stent was placed with good result. A standard anticoagulation protocol was initiated. However, after the second warfarin dose (total of 20 mg), the prothrombin time became markedly elevated and remained elevated despite the discontinuation of heparin administration and, ultimately, the administration of fresh frozen plasma. The patient sustained a large, fatal intracerebral bleed. The family consented to an autopsy.

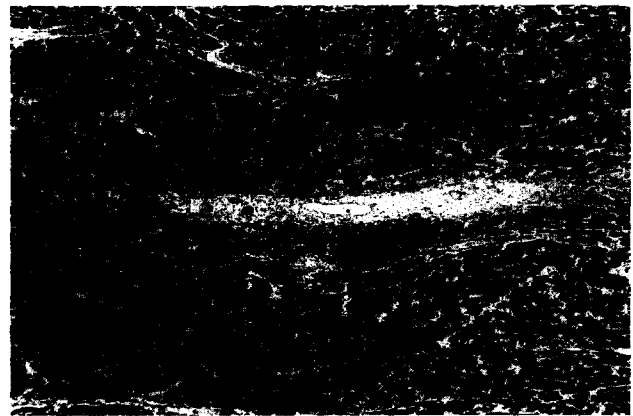
Numerous fibrous plaques were easily identified on the epicardial surface of the heart, which demarcated the sites at which laser channels had been created. Gross inspection of the endocardium also revealed fibrous plaques, presumably located at the original entry sites of the channels into the left ventricle, suggesting that any direct connection between the chamber and the myocardium was not patent. When the myocardium was cut from epicardium to endocardium in multiple transverse sections, channel regions were easily identified by eye as elliptical fibrous transmural scar extending from the epicardium to the endocardium. Microscopic examination was performed on nine of the channels; samples were chosen randomly from all portions of the treated regions (Fig 1). Each channel consisted of fibrous scar, some of which included thin-walled capillaries in the central area of the channel remnant (see Fig 1A). No channel showed any residual patent central passage at any level through the myocardial wall.

Comment

Preliminary clinical experience indicates that TMLR provides an average reduction of two angina classes (based on the Canadian Cardiovascular Society angina classification) in patients who are otherwise refractory to medical therapy and considered untreatable by bypass operation or angioplasty [3]. However, the mechanism of this effect is uncertain. Results of one nuclear scanning study [8] suggest that regional myocardial perfusion is improved in areas treated by TMLR 6 months, but not 3 months, after the procedure. This finding provides a clue that the mechanism of action may not be related to direct myocardial perfusion through channels because, if that were the case, the clinically observed immediate reduction in angina would be associated with an immediate and demonstrable improvement in flow.

Although only about a third of the original channels were subjected to microscopic examination and it is possible that some of the channels that were not examined could have been patent, most of the channels were identified during gross inspection and all had the same appearance; any patent channels with diameter of about 1 mm would have been identified.

Thus, in contrast to previous histologic specimens, which have been interpreted as suggesting that TMLR channels remain patent several months after the procedure [1, 2, 4], the present results indicated that TMLR channels did not maintain patency in the present patient. Further work is needed to determine the frequency of



A



B

Fig 1. Histologic specimens showing the channel region. The central area of the channel remnant demonstrates fibrous scar. Typically (A), there are numerous thin-walled capillaries and sinusoids seen within the region but no prominent patent central passage. In several sections examined, the fibrous region was devoid of such capillaries (B).

channel patency in a larger number of patients and its relation to observed clinical benefit. We propose that mechanisms other than blood flow through patent channels should be considered.

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INVITED COMMENTARY

Burkhoff and colleagues report their observations on the anatomic appearance of transmyocardial channels created with a high-energy CO₂ laser in 1 patient who died 4½ weeks after therapy. Transmyocardial revascularization (TMLR) with a high-energy CO₂ laser is a relatively new surgical technique currently restricted to the treatment of symptomatic angina patients whose illness is refractory to conventional treatment. We have performed this operation at the Texas Heart Institute since July 1993 and have used the laser as sole therapy in more than 25 patients.

Results of our preliminary studies [1, 2] indicated that the operation gives immediate, significant relief from anginal symptoms and that its positive subregional effects on myocardial perfusion are detectable by positron emission tomographic scan at 3, 6, and 12 months postoperatively. No change in regional or global myocardial systolic function was seen in this group. More significant, however, were the sustained relief from anginal symptoms and the improved treadmill time over a 12-month period. Our findings also suggested that the preoperative presence of congestive heart failure, unstable angina, or malignant arrhythmia was a strong predictor of poor postoperative outcome. Furthermore, we observed that patients who had patent saphenous vein grafts with poor distal run-off before the operation had a higher incidence of adverse events during the operation than patients who presented with occluded saphenous vein grafts and a coronary anatomy that included collateral arteries.

These preliminary observations were valuable in designing the patient selection criteria for the phase III randomized clinical trial, approved by the Food and Drug Administration, which is presently under way at more than 10 centers in the United States. The goal of the trial is to investigate the efficacy of TMLR in relieving angina. It is not a histologic study. The channels produced by TMLR may be directly (or indirectly) responsible for improved perfusion, but the exact mechanism by which perfusion is improved and symptoms are relieved is not clear. Positron emission tomographic studies have shown that TMLR improves relative subendocardial perfusion,

and histologic evidence of patent channels has been reported in two autopsy studies [3, 4], but whether the laser channels are responsible (directly or indirectly) for the improved perfusion is still unclear [1, 4]. Further histologic evidence of laser channels [3, 5] may offer proof for such a mechanism of perfusion. Negative findings, however, simply beg the issue.

A preoperative angiogram performed in the patient in this report had revealed an 18-year-old patent saphenous vein graft that protected the entire anterior and anterolateral myocardium. Twenty-four days after the TMLR procedure, a stenosis developed in this graft that required the placement of a stent. Aggressive coagulation therapy was initiated, which resulted in intracerebral bleeding and eventual death. We believe that careful patient selection is needed in these cases, not only to increase the chance of a good postoperative outcome but also to assess more accurately the efficacy and clinical value of the multicenter study.

Burkhoff and colleagues raise questions about the mechanism of improved blood flow after TMLR. Basic research addressing these questions may be of value; however, no conclusion can be drawn about the mechanism of TMLR on the basis of an observation in a single patient. The main issue, from a clinical standpoint, is whether TMLR can be shown to improve anginal symptoms and treadmill tolerance to a greater extent than medical therapy in controlled, randomized trials. Pathologic examinations cannot measure relative increases in blood flow; thallium and positron emission tomographic scans can, and they have demonstrated improvement in TMLR patients.

Penicillin was used successfully for more than 20 years before its mechanism of action was understood. The exact mechanism by which digitalis works is still debated. Fifty years ago Sam Levine defined the aims of medicine in treating heart disease: to prolong life, to diminish suffering, and to increase the physical and mental efficiency of the patient [6]. Medical science should attempt to understand how a therapy works, but the attempt should not jeopardize the pursuit of a beneficial therapy.

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Retained Sponge After Thoracotomy That Mimicked Aspergilloma

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A 63-year-old man, who had had operation for the treatment of pulmonary tuberculosis 40 years before the present disorder, was admitted to our hospital with massive hemoptysis. Radiologic examinations showed a mass shadow with a crescent air sign resembling aspergilloma. Operative exploration showed a well-encapsulated retained surgical sponge between the middle and lower lobes. A bronchial fistula was present in the lower lobe. The appearance of the crescent air sign was caused by drainage of exudative effusion around the retained sponge. Intrathoracic retained surgical sponges associated with bronchial fistula should be included in the differential diagnosis of patients who have mass shadows with crescent air signs but no evidence of *Aspergillus* infection, and who have a history of thoracotomy.

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Although there have been several reports of intraperitoneal retained surgical sponges [1, 2], intrathoracic sponges rarely have been reported [3]. We present a case of an intrathoracic retained surgical sponge, which had been left at the site of operation for pulmonary tuberculosis 40 years before resembled aspergilloma in both clinical symptoms and radiologic findings. We also reviewed four reported cases of intrathoracic retained surgical sponges that also showed clinical and radiologic findings resembling aspergilloma [4-6].

The patient was a 63-year-old man. He had received a thoracoplasty and segmentectomy of the right upper lobe for the treatment of pulmonary tuberculosis at another hospital in 1955. A chest roentgenogram in 1961 showed a mass shadow 10 × 6 cm in size in the middle lung field,

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but no further examination or treatment was performed because he had no symptoms. Since 1977, he had complained of hemosputum every 1 or 2 years. On March 7, 1995, he suddenly complained of massive hemoptysis and was admitted to Saiseikai Central Hospital.

Chest roentgenography, computed tomographic scan, and magnetic resonance imaging scan showed a thin-walled cavitory lesion consisting of a homogeneous mass and an air crescent, which resembled aspergilloma (Figs 1, 2). The size of the lesion was decreased to 7 × 5 cm compared with that in 1961. However, the computed tomography-guided biopsy did not show *Aspergillus*, and the serum anti-*Aspergillus* antibody levels were negative. Bronchoscopy showed a coagulum in the anterior basal bronchus of the right lower lobe, which was suspected to be the drainage bronchus. Laboratory examination showed low-grade inflammation, ie, 0.4 mg/dL of C-reactive protein, a blood sedimentation rate of 27 mm/h, and a white blood cell count of 5,900/μL. Bronchial angiography showed a remarkable increase in flow of the vessels to the capsule of the cavitory lesion.

Thoracotomy revealed a well-encapsulated retained surgical sponge, measuring 5 × 4 cm, between the middle and lower lobes. A bronchial fistula was present in the lower lobe. The lesion was opened, and a fenestra was made. After 2 months, the bronchial fistula was closed by suture, and the lesion was obliterated by intrathoracic transposition of the major pectoralis muscle flap. The postoperative course was satisfactory, and the patient was discharged from the hospital 1 month after the last operation.

On pathologic examination, the specimen consisted of a standard size surgical sponge containing fibrinoid necrotic material. Bacteriologic examination showed no infection, including tuberculosis and *Aspergillus*.

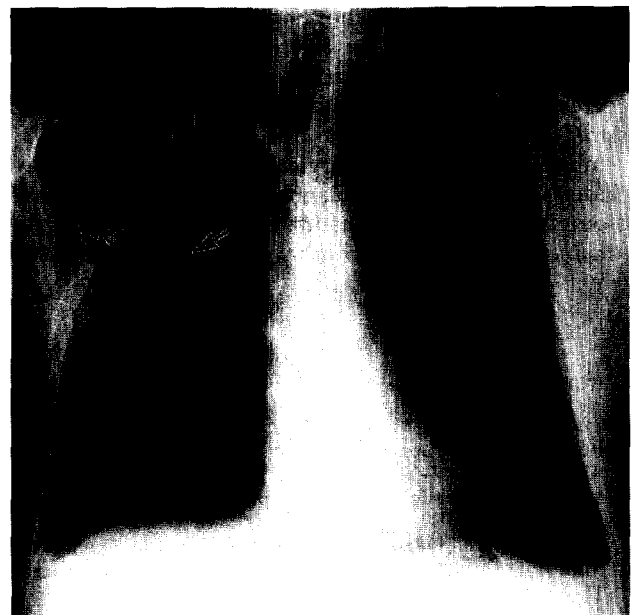


Fig 1. Chest roentgenogram. Arrows indicate the wall of the cavitory lesion.