SURGICAL APPLICATIONS

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^{123}I-MIBG SPECT SCINTIGRAPHIC EVIDENCE OF CARDIAC DENERVATION AFTER TRANSMYOCARDIAL LASER REVASCULARIZATION

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Transmyocardial laser revascularization (TMLR) is an invasive therapy used to treat patients with severe refractory angina pectoris in whom standard treatment is insufficient or not possible. Randomized trials have reported a clear clinical benefit (relief of angina and improvement of QOL) of CO_2 or Holmium:YAG TMLR compared to maximal medical treatment. Furthermore, initial results of a prospective randomized trial also show a reduction in angina and improvement of QOL after XeCl excimer TMLR (Huikeshoven et al., this issue of Lasers Surg Med). The aim of the study described here was to investigate cardiac denervation as a working mechanism of stand-alone TMLR using ^{123}I-MIBG SPECT scintigraphy (MIBG and noradrenalin have similar molecular structures and both utilize the same uptake and storage mechanisms in sympathetic nerve endings) in combination with an algorithm for quantification of SPECT (Germano et al., J Nuclear Med 2000;41:712-9).
In 8 patients, TMLR was performed using a Ho:YAG (n = 3) or XeCl excimer laser (n = 5). In all patients angina was reduced by $\geq$2 classes (classification of the NYHA) at 3 to 12 months follow up, QOL was significantly improved and ^{123}I-MIBG SPECT scintigraphy showed decreased uptake up to 16 months follow up, indicating sympathetic myocardial denervation ($P = 0.00002$). Average summed defect scores were 13.4 ± 3.9 pre-operative vs. 23.9 ± 4.3 post-operatively. Pre- and post operative myocardial perfusion scintigraphy did not differ significantly. Our results indicate that relief of angina may be explained by destruction of nociceptors or cardiac neural pathways, resulting in a change in the perception of anginal pain after TMLR.

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BECK II OPERATION REVISITED: RETROGRADE PERFUSION OF THE ISCHEMIC MYOCARDIUM COMBINED WITH TRANSMYOCARDIAL LASER REVASCULARIZATION

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Background: Beck introduced the concept of relieving myocardial ischemia by perfusion through the coronary venous system in 1951. Angina was relieved, but there was high morbidity and mortality due to myocardial dysfunction and stiffness. The purpose of our study was to re-examine this early procedure.
Methods: Retrograde left ventricular perfusion through the proximally ligated coronary vein, combined with left ventricular laser channels (TMLR) was studied. There were three groups of animals. Group I: (n = 6) was the control. The left circumflex coronary artery (LCxA) was ligated, and graft inserted to the left circumflex coronary vein (LCxV). Group II: (n = 6) underwent LCxA ligation, LCxV graft, followed by TMLR. Group III: (n = 6) LV TMLR was performed, followed by LCxA ligation and LCxV graft. All procedures were performed through a left thoracotomy. Coronary venous pressure (VP) was monitored before intervention and throughout the procedures. Infarction was evaluated by histology studies.
Results: Group I: VP equaled arterial systolic pressure following LCxV. The LV became distended, stiff, and dysfunctional. Group II: VP rose to systolic levels following LCxV and fell to diastolic pressure following insertion of laser channels. Group III: VP did not rise. Histology indicated no infarction.
Conclusions: Retrograde perfusion can protect the myocardium from ischemia. The laser channels produce shunting between the arterioles and venuoles and the LV cavity, preventing edema and dysfunction. Combined retrograde perfusion and LV laser channels may be an option for patients with end stage coronary artery disease when conventional means of treatment fail. Further studies are necessary.

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IN THE WHOLE HEART PREPARATION THE NUMBER OF TRANSMYOCARDIAL LASER CHANNELS DEFINES MYOCARDIAL FUNCTION

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Transmyocardial revascularization (TMR) has been shown to relieve symptomatic ischemia. However, heart failure has been noted to be a major potential complication. This study evaluated the effect of TMR on myocardial function in relationship to number of channels.
Under general anesthesia, whole hearts (1.6 ± 0.3 g) were removed from 14 rats (7 control, 7 TMR) and placed in oxygenated physiologic buffered solution (PBS) with 200 mg/dl glucose at 34°C. The hearts were then perfused with PBS in a Langendorff setup. After 1 hr of perfusion, laser irradiation was performed using a Ho:YAG (3 Hz, 280 mJ/pulse) laser delivered to the epicardial surface of the left ventricle via a 600 μm core fiber. Channels were made while lasing and advancing the optical fiber through the full thickness of the left ventricle. Initially a series of 4 channels (5×) were made and this was followed by another series of 10 channels (3×) each separated by 10 min intervals. After each set of TMR channels, the maximal force of myocardial contractility was measured with increasing pre-loads from 0 to 8 g. Compared to control, the maximum force of myocardial contractility after TMR significantly decreased by 22% after 30 channels (5.4 ± 1.2 vs. 4.2 ± 1.3 g; $p < 0.05$) and by 35% after 50 channels (5.0 ± 1.5 vs. 3.4 ± 1.0 g; $p < 0.02$). This study demonstrated that myocardial dysfunction following TMR is dependent on the number of channels. In this rat whole heart preparation, more than 30 channels resulted in significant decrease of myocardial contractility.