

# Direct epicardial shock wave therapy improves left ventricular function in an experimental model of ischemic heart failure

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## Device and producing company:

CardioGold® CG050 (Cardiac Regeneration Technologies, Woodstock, USA, a subsidiary of Tissue Regeneration Technologies (TRT) / manufactured by MTS-Europe GmbH, Konstanz, Germany)

## Introduction:

Prognosis of ischemic heart failure is poor and therapeutic options are limited. Shock wave therapy (SWT) reportedly induces VEGF overexpression in ischemic skin flaps and ischemic myocardium. Here, we hypothesized that epicardial SWT improves ventricular function by enhancing angiogenesis in an experimental model of ischemic heart failure in rats.

**Methods:** Adult Sprague Dawley rats were subdivided into 3 groups: sham-operated (sham), infarcted myocardium with epicardial SWT (SWT group) and infarcted myocardium without epicardial SWT (control). Four weeks following myocardial infarction (MI), epicardial SWT (100 impulses at 0.38 mJ/m<sup>2</sup>) was applied directly to the infarcted region in the SWT-group, control animals were untreated. Cardiac function was evaluated using echocardiography before MI, 4 weeks after MI and 12 weeks after SWT. Angiogenesis was evaluated 12 weeks after treatment in serial sections stained with von Willebrand Factor antibody, which were digitalized and morphometried.

**Results:** As compared to sham group (50±4%), left ventricular function decreased in the SWT (21±9%, p<0.001) and control (18±4%, p<0.001) group 4 weeks after MI. Fourteen weeks after epicardial SWT, left ventricular function improved in the SWT-group as compared to 4 weeks after MI (37±8%, p=0.021) and as compared to the controls (21±4%, p<0.001). Quantitative histology revealed more vital cells (384±84 cells/field in SWT vs. 288±56 in controls, p=0.02) and enhanced angiogenesis (7.1±3.3 vessels/field in SWT vs. 3.2±1.8 in controls, p=0.016) in the SWT group.

**Conclusion:** Direct epicardial shock wave therapy improves left ventricular function and induces neoangiogenesis in an experimental model of ischemic heart failure in rats.