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Does Diabetes Reduce Myocardial Efficiency?

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The Question

Does diabetes reduce efficiency (the ratio of mechanical work output to metabolic energy input) of the heart, and of ventricular tissues (trabeculae)?

Results

Work, and change of enthalpy were measured and efficiency calculated, as functions of afterload (Figs.5 and 6). Twitch duration was prolonged in the diabetic preparations (Fig. 7). The diabetic hearts were incapable of generating high pressure. At high afterloads, their work outputs were reduced, and consequently, efficiencies were lower (Fig. 8). But, for diabetic trabeculae, their work outputs and efficiencies were unaltered (Fig. 9).

Methods

We measured the mechano-energetics of isolated rat hearts: Control and Streptozotocin-induced Diabetic (Fig. 1), using the Working-heart Rig (Fig. 2). We then dissected left-ventricular trabeculae (Fig. 3), and measured their mechano-energetics using the Work-loop Calorimeter (Fig. 4).

Fig. 1. Photograph of a fully-instrumented rat heart

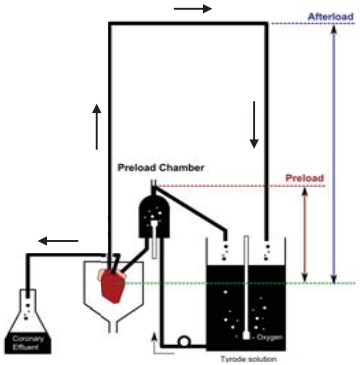

Fig. 2. Schematic (not to scale) of the Working-heart Rig.

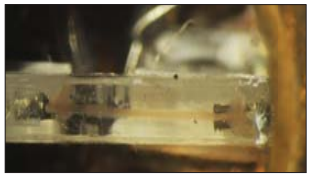

Fig. 3. Photograph (top view) of a trabecula, mounted in the Work-loop Calorimeter.

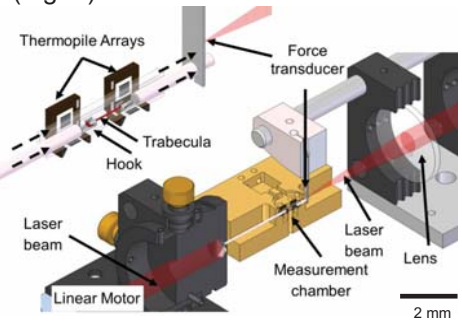

Fig. 4. Schematic, cut-away view, of the Work-loop Calorimeter.

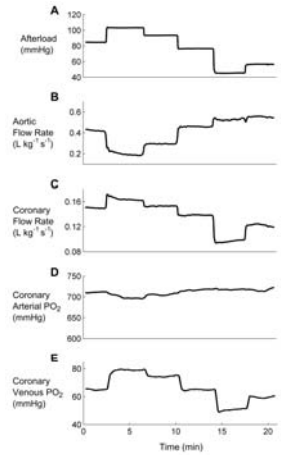

Fig. 5. Representative traces obtained from working-heart experiments.

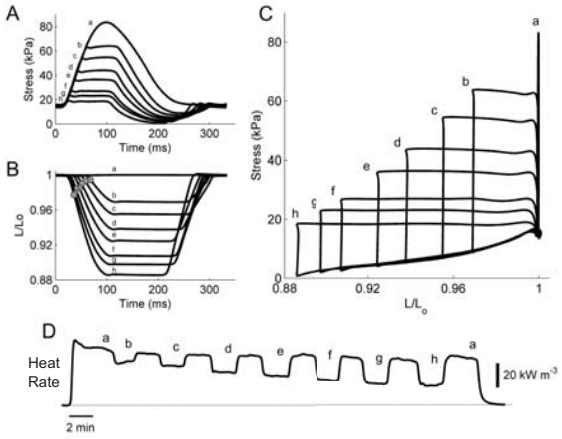

Fig. 6. Representative traces obtained from trabeculae experiments.

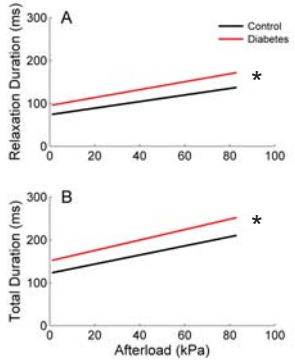

Fig. 7. *Significant prolongation of the twitch of diabetic trabeculae compared with control trabeculae.

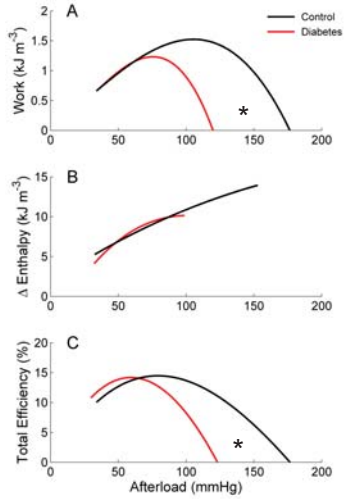

Fig. 8. Work, change of enthalpy , and total efficiency as functions of afterload from n = 16 diabetic hearts and n = 17 control hearts. *Significantly lower peak afterloads achieved by the diabetic hearts than the control hearts.

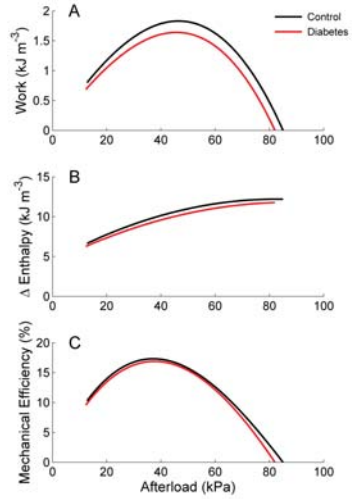

Fig. 9. Work, change of enthalpy, and mechanical efficiency as functions of afterload from n = 12 diabetic trabeculae and n = 15 control trabeculae.

Conclusions

Diabetes-induced contractile dysfunction at high afterloads <u>at the organ level</u> is due to prolongation of the twitch, which restricts diastolic filling, thereby compromising systolic ejection. Nevertheless, diabetes has <u>no effect</u> on the **<u>peak efficiency</u>** of either the heart or its isolated trabeculae.

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