

Investigating the effect of hyperglycemia on embryonic heart development using the Chick Embryo Model

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BACKGROUND

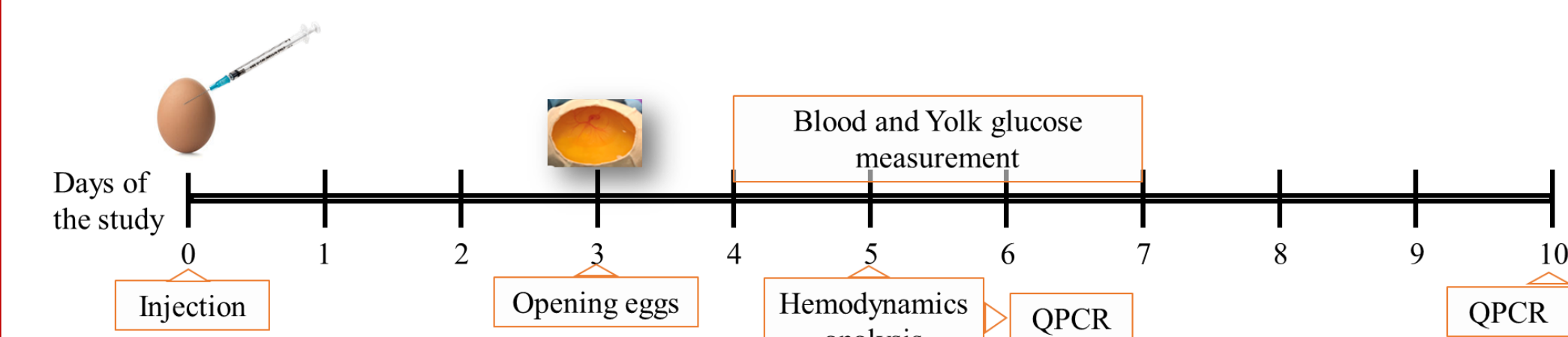
- Congenital heart disease (CHD) is the most common birth defect. Even with remarkable advances in care, it remains the leading cause of non-infectious death in infants.
- High blood sugar during pregnancy is associated with congenital heart defects
- Biological mechanisms responsible for these defects are yet to be identified

OBJECTIVE

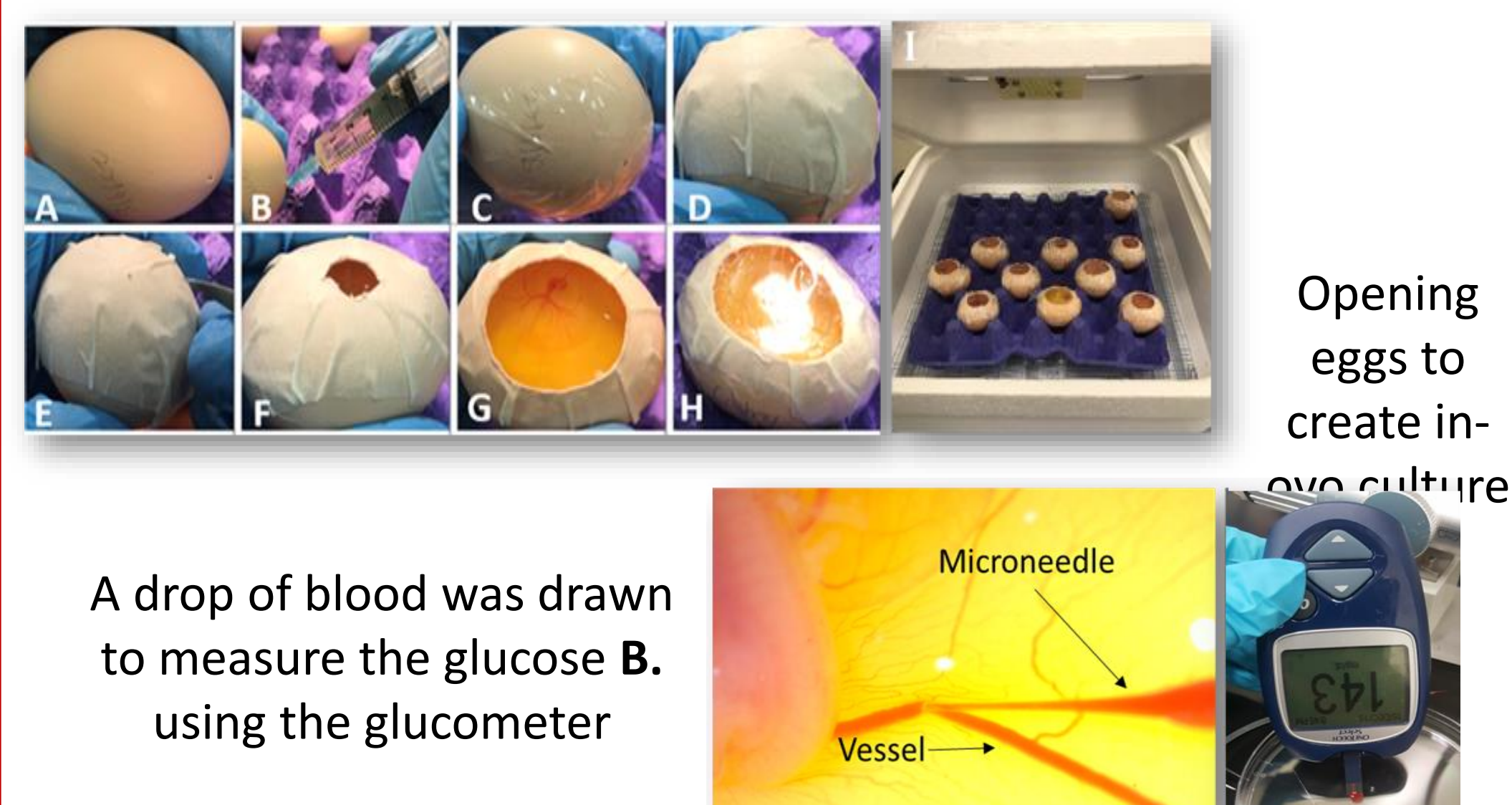
- Generate a hyperglycemic model in chick embryo.
- Study the changes related to hyperglycaemia in the developing heart.

METHODOLOGY

- Study time line summary showing groups, and end points where analysis was performed.

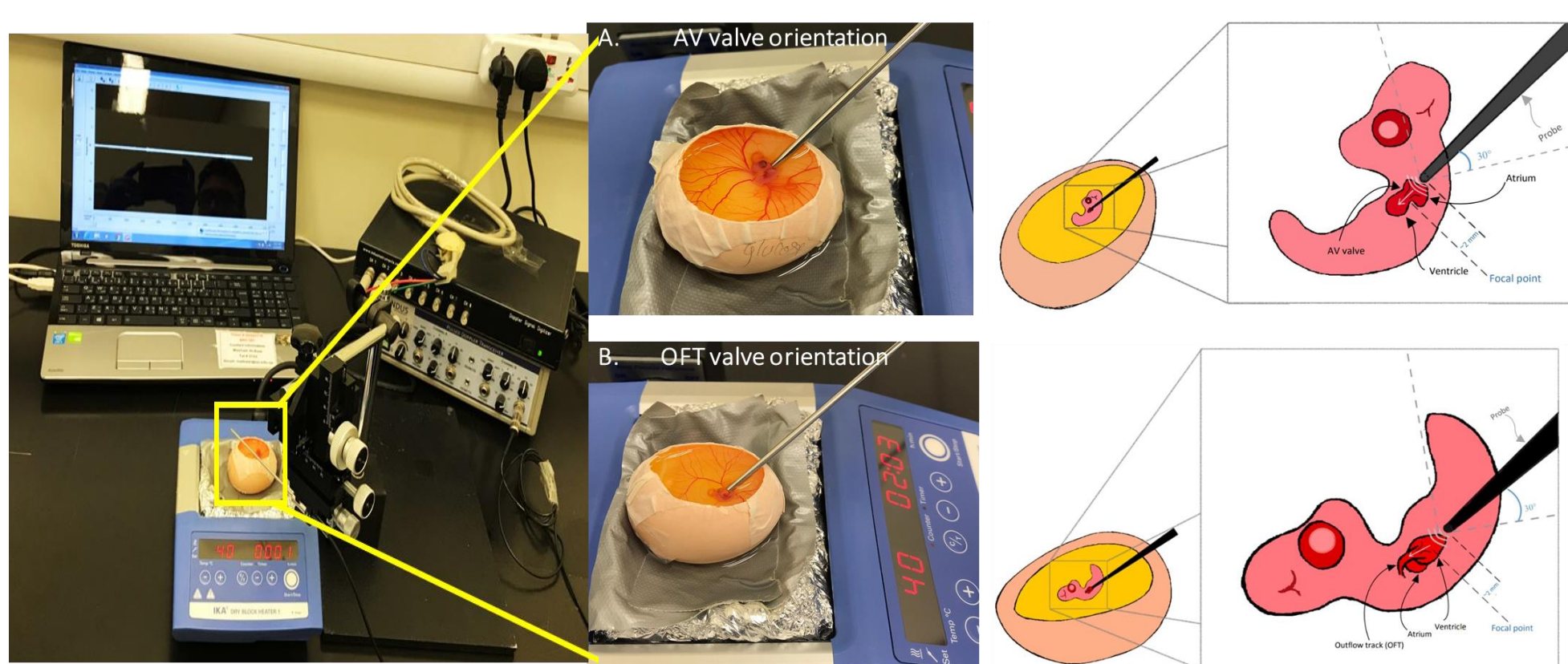


- Hyperglycemic model generation & glucose measurement



A drop of blood was drawn to measure the glucose B. using the glucometer

- Heart development and injury gene expression analysis via QPCR
- Echocardiography analysis.

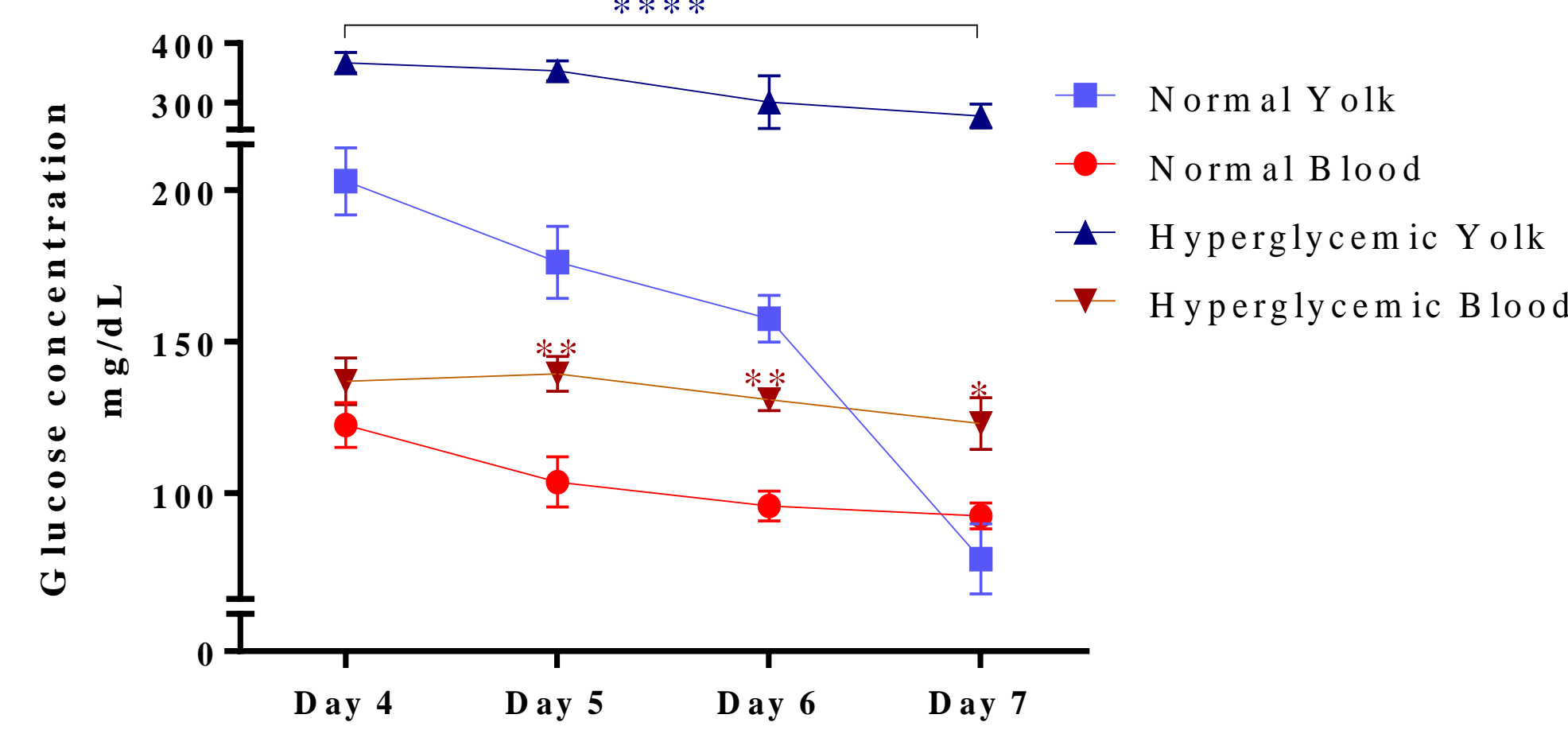


Echocardiography was done by removing the embryonic membrane, the in-ovo culture was placed on a dry heat block the probe was adjusted toward the heart as shown in B. to obtain the out-flow track (OFT) signal. The embryo was then flipped and oriented as in A. to obtain the atria ventricle (AV) flow.

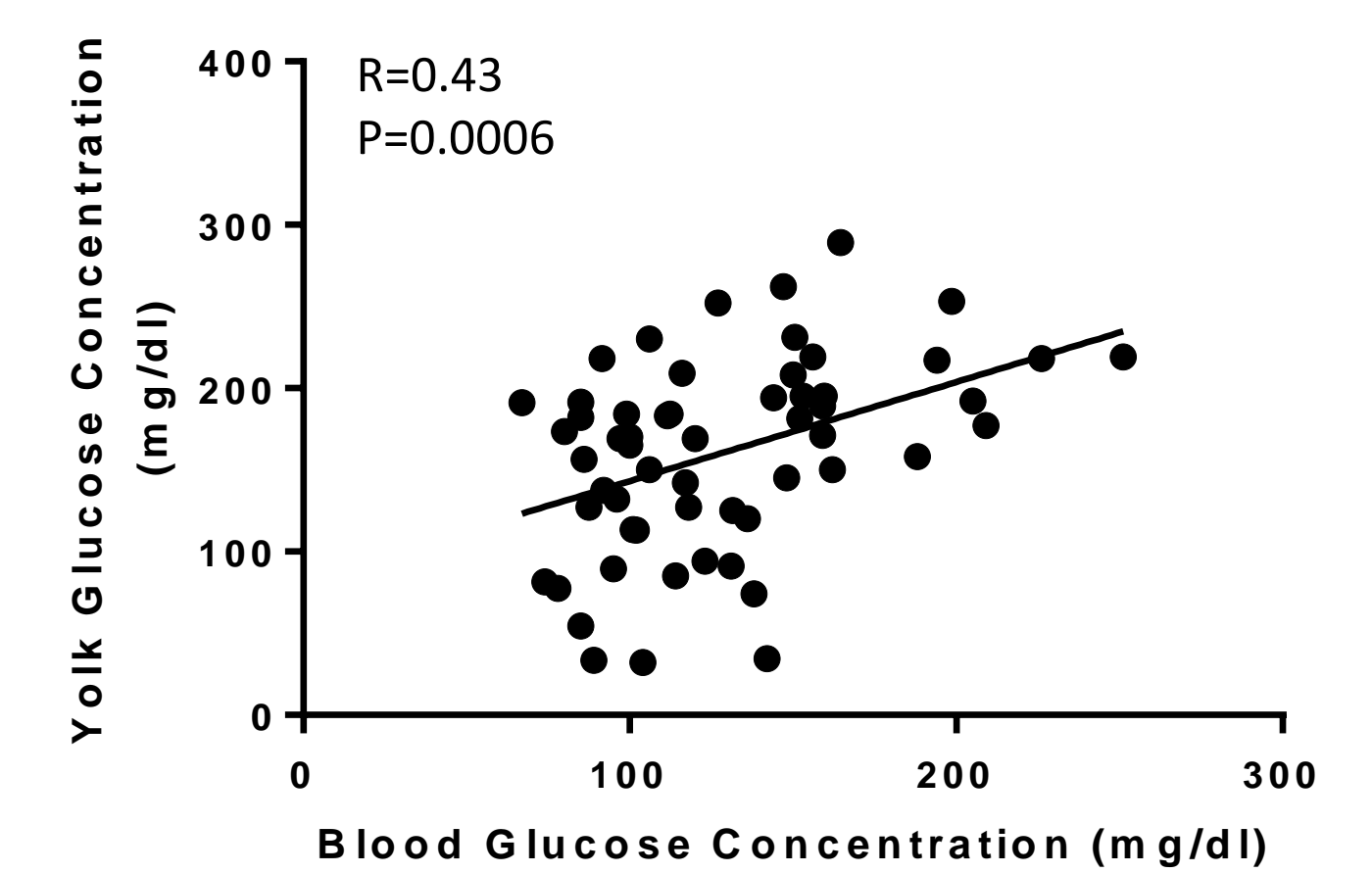
References

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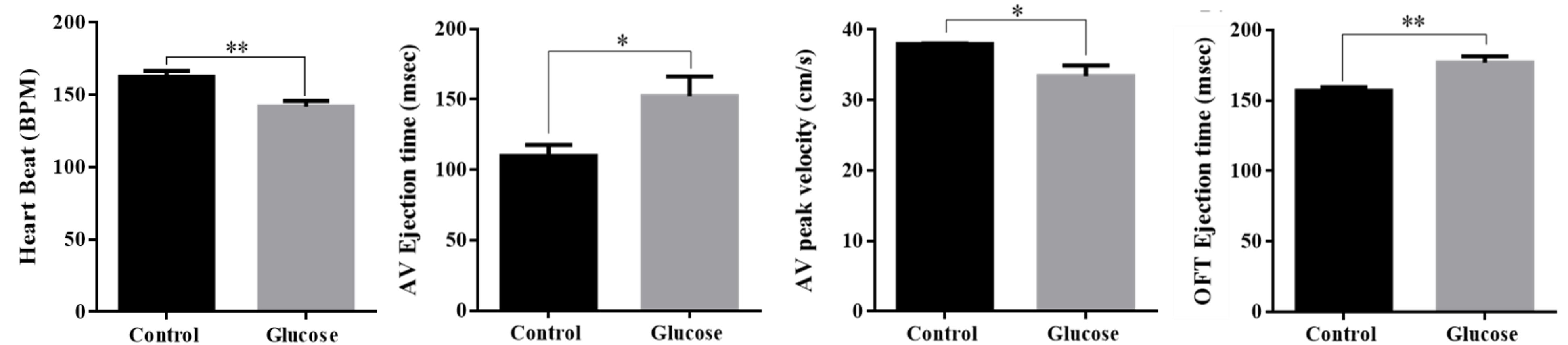
RESULT



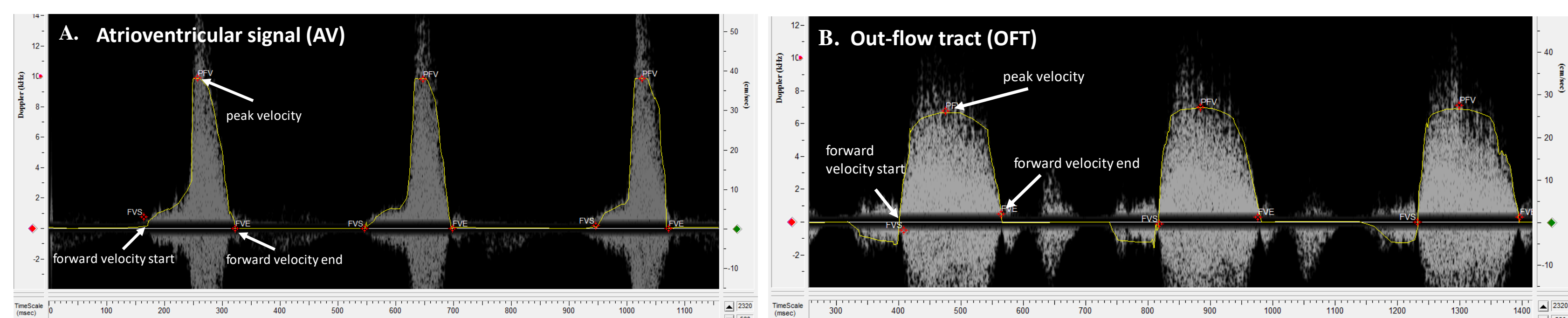
Increasing the yolk glucose levels by glucose injection, caused a significant increase in the blood glucose levels.



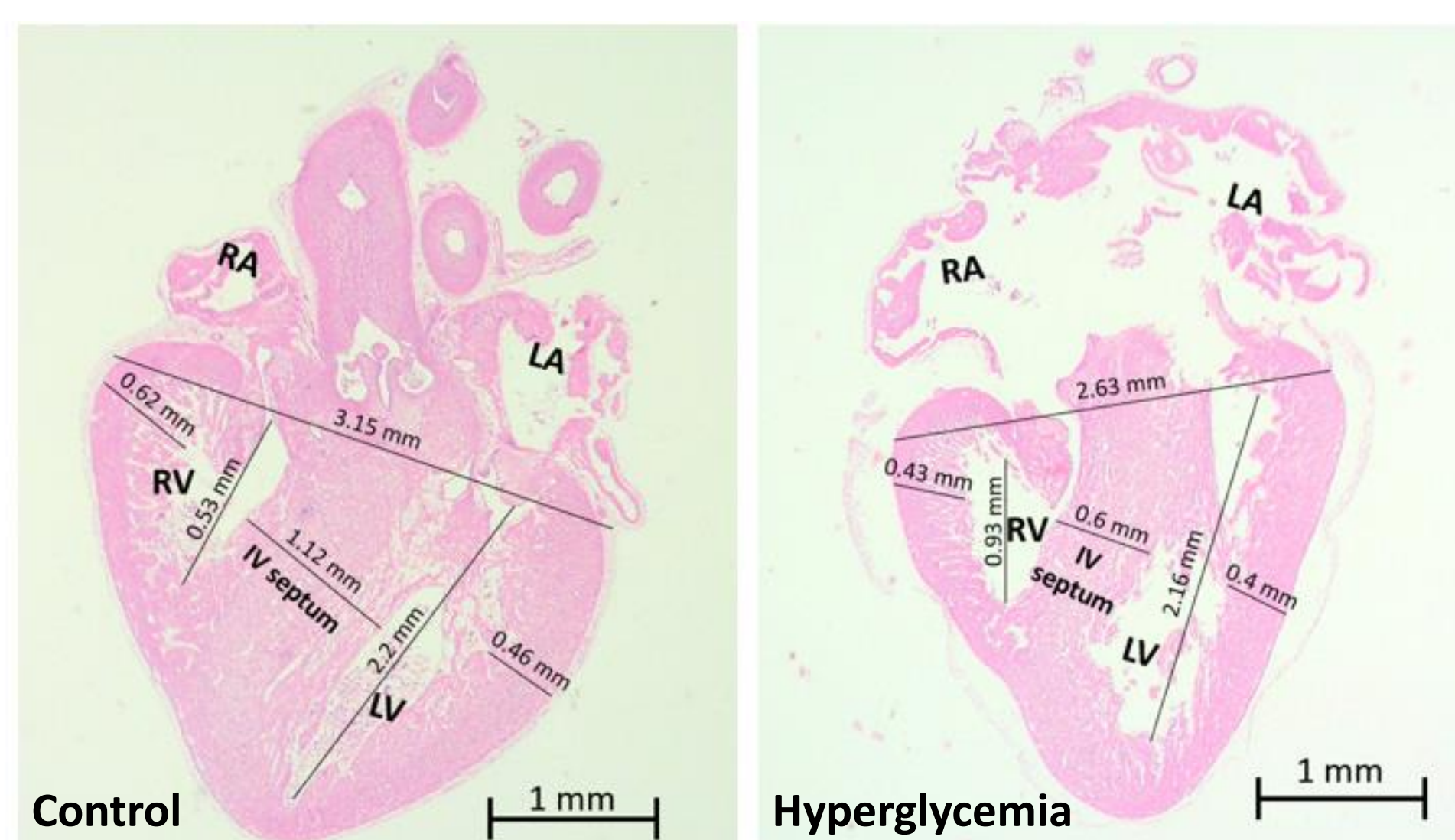
Pearson correlation showed that the Yolk and blood glucose concentration were positively correlated



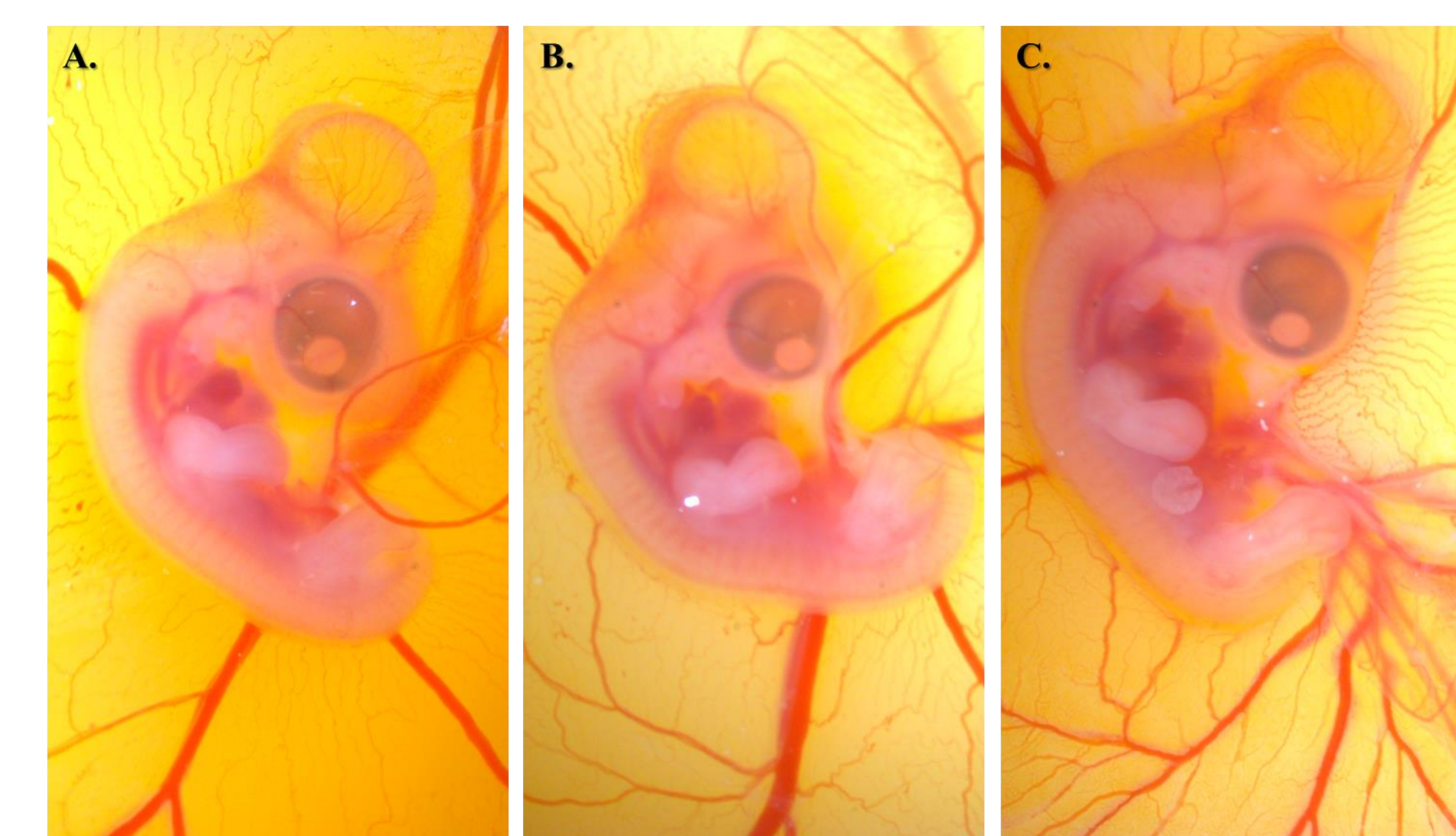
Hyperglycemia resulted in significantly decreasing chick embryo heart beat, increase in the anterior ventricular (AV) and outflow tract (OFT) ejection times & an increase in the AV peak velocity.



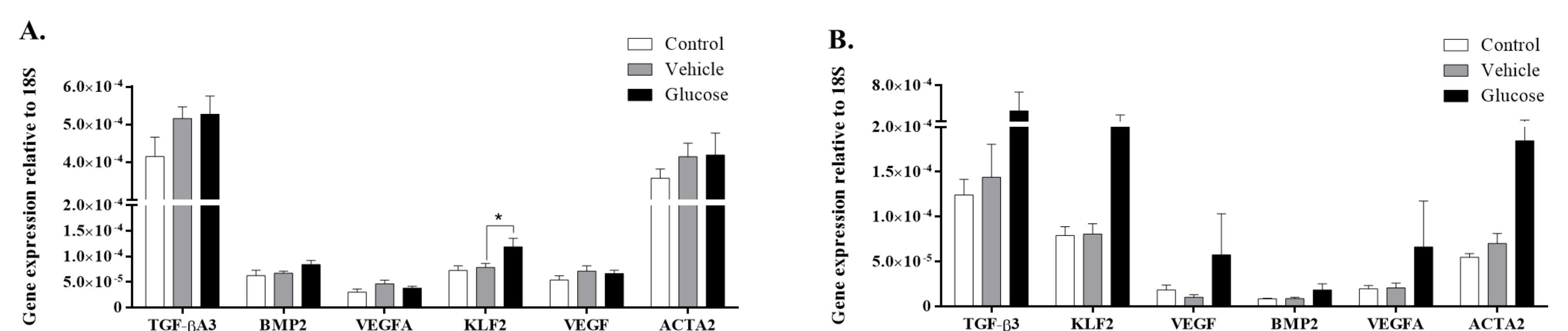
Analysis via Doppler Signal Processing Work Station.



Hyperglycemia lead to the decrease in ventricular and septum wall thinness, ventricular size, structure of the valves and over all heart size.



Chick embryo at day 5 of incubation. A representative image of A. Control, B. vehicle injected group and C. glucose injected group. An increase in the vascularization was observed in the hyperglycemic group.



Relative gene expression at A. day 5 and B. day 10. Hyperglycemia resulted is significantly elevating KLF2 (sheer stress marker) expression at day 5.

CONCLUSION

Using the chick embryo as a model, we showed that hyperglycemia effect the structural development of the heart which lead to the defect in the function of the heart valves as well as heart ventricles.