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INVITED COMMENTARY

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Biomechanical imbalance of neochordal forces: the dark side of mitral valve prolapse repair

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Mitral valve prolapse (MVP) repair based on replacement of chordae tendineae with expanded polytetrafluoroethylene (ePTFE) neochordae is an established technique, which already proved able to provide stable MV function and excellent repair durability [1, 2]. According to the 'respect rather than resect' paradigm, neochordal implantation has allowed for repair of mitral valves (MVs) that otherwise would be replaced, preserving leaflet mobility and offering a potentially more physiologic-like MVP repair with respect to leaflet resection [3, 4]. Nonetheless, extensive experience and surgical skills are required to estimate the amount and the length of ePTFE sutures allowing for optimal neochordal tuning and post-procedural success [5, 6].

In this issue of the *European Journal of Cardio-Thoracic Surgery*, Zhu *et al.* [7] proposed an interesting experimental analysis aimed to systematically assess the impact of neochordae length changes on neochordal forces and MV haemodynamics. To this purpose, using a left heart simulator, posterior P2 prolapse was reproduced on porcine MVs by excising at least 2 native primary chordae; subsequently, 2 ePTFE neochordae were implanted with one tied to the corresponding native exact length and the other tied to a variable, i.e., sub-optimal, length.

Key finding of the study is that suboptimal setting of neochordae length leads to minor effects on leaflet coaptation (post-repair regurgitant fraction was comparable to the baseline condition), but to more tangible alterations in the forces transferred from the leaflets to the papillary muscles by the neochordae. Interestingly, these alterations are mild when considering the sum of the magnitudes of these forces (these alterations span from −14% to + 33% versus optimal configuration) but are major when considering the force transferred by the neochordae with suboptimal length (these alterations instead span from −85% to +131%). This evidence suggests that neochordae settings that allow for a globally physiologic-like load transfer through the subvalvular apparatus may not be sufficient to prevent imbalances in the distribution of such load transfer over multiple neochordae, which in turn can lead to unphysiological stress overload in the leaflet tissue at the insertion of the sub-optimally tuned neochord. Also, the data by Zhu *et al.* suggest that this local effect may be worse in case of overly short neochord, as evident from the occurrence of leaflet tethering, than in case of overly long neochord. Under a comparable setting of neochordae implantation, prior numerical finite element analysis highlighted that the tethering of the posterior scallop close to the overly short neochord is combined with the local exacerbation of mechanical stress [8]. Furthermore, a sub-optimal MVP repair may also impact on the surrounding tissues: for instance, it has been recently hypothesized that MVP-related altered papillary muscle forces could be a plausible trigger for localized fibrosis and scarring of the adjacent left ventricle myocardium [9].

Hence, these findings may have a practical implication, i.e., surgeons should focus especially on avoiding local tethering by any neochord when testing neochordae setting upon saline pressurization.

Yet, as usual when considering data obtained from *in vitro* or computational models, the interpretation of the findings by Zhu *et al.* should be cautious, bearing in mind the simplifications inherent to these modelling approaches. In this specific case, it may be worth mentioning two aspects.

First, all the tested neochordae configurations were characterized by a neochord with optimally tuned length, which could mitigate or even compensate for the effects of the suboptimal setting of the complementary neochord. However, if the errors in the tuning of neochordae length are due to the standard phenomenon of diastolic phase inversion when testing neochorde upon saline left ventricle pressurization [5, 6], it may be reasonable to expect that sub-optimally length tuning would affect both the implanted neochordae. In such a scenario, alterations in load transfer and possibly also in leaflet closed configuration may become more evident.

Second, the study systematically analysed only the implantation of 2 ePTFE sutures. However, the spectrum of neochordae configurations available to surgeons is much wider: for instance, more than 2 neochordae may be implanted, and pre-configured loops may be used. The conclusions suggested by the *in vitro* analysis by Zhu *et al*. may not apply to these other configurations, which could be less or more prone to imbalances in neochordal forces due to errors in the setting of the length of a single neochord.

In conclusion, the study confirms the relevant contribution of biomechanical analysis to the proper setting of neochordal lengths for MVP repair, highlighting that the sole MR reduction may be not enough to define an optimal MVP repair. To this purpose, on-going research and efforts based on both experimental and computational studies will be essential to advance our current understanding of the "dark" side of MVP biomechanics, which may provide evidence of the mechanistic determinants for optimal MVP repair.

REFERENCES

[1] David TE, David CM, Lafreniere-Roula M, Manlhiot C. Long-term outcomes of chordal replacement with expanded polytetrafluoroethylene sutures to repair mitral leaflet prolapse. J Thorac Cardiovasc Surg 2020; 160:385–94.e1.

[2] David TE, Armstrong S, McCrindle BW, Manlhiot C. Late outcomes of mitral valve repair for mitral regurgitation due to degenerative disease. Circulation 2013;127:1485–92.

[3] Perier P, Hohenberger W, Lakew F, Batz G, Urbanski P, Zacher M *et al*. Toward a new paradigm for the reconstruction of posterior leaflet prolapse: midterm results of the "respect rather than resect" approach. Ann Thorac Surg 2008;86:718–25.

[4] Del Forno B, Tavana K, Ruffo C, Carino D, Lapenna E, Ascione G *et al*. Neochordae implantation versus leaflet resection in mitral valve posterior leaflet prolapse and dilated left ventricle: a propensity score matching comparison with long-term follow-up. Eur J Cardiothorac Surg 2023;64: ezad274. doi: 10.1093/ejcts/ezad274.

[5] Park MH, Marin-Cuartas M, Imbrie-Moore AM, Wilkerson RJ, Pandya PK, Zhu Y *et al*. Biomechanical analysis of neochordal repair error from diastolic phase inversion of static left ventricular pressurization. JTCVS Tech 2022;12:54–64.

[6] Elde S, Woo YJ. Neochords: how long, how many, too many? JTCVS Tech 2023;22:59–64.

[7] Zhu Y, Lee SH, Venkatesh A, Wu CA, Stark CJ, Ethiraj S *et al*. Biomechanical engineering analysis of neochordae length's impact on chordal forces in mitral repair. Eur J Cardiothorac Surg 2024;ezae008. doi: 10.1093/ejcts/ezae008

[8] Sturla F, Votta E, Onorati F, Pechlivanidis K, Pappalardo OA, Gottin L et al Biomechanical drawbacks of different techniques of mitral neochordal implantation: when an apparently optimal repair can fail. J Thorac Cardiovasc Surg 2015;150:1303–12.e4.

[9] Park MH, Kampen A, Melnitchouk S, Wilkerson RJ, Nagata Y, Zhu Y et al Native and post-repair residual mitral valve prolapse increases forces exerted on the papillary muscles: a possible mechanism for localized fibrosis? Circ Cardiovasc Interv 2022;15:e011928.