**Commentary: From the lab to clinical reality: Small step or giant leap?**

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Park and colleagues examined in an experimental fashion the biomechanics of neochordal length on papillary muscle forces. They used a complex but validated setup to study explanted mitral valves from healthy swine and measured the papillary muscle forces in relation to variable chordal lengths in the P2 area. They found that increased chordal length increased the papillary muscle forces. The same was observed when chordal lengths were shortened, but this happened to a lesser degree. These findings indicate the importance of determining the optimal chordal length when performing neochordal implantation during mitral valve repair.

The effort of the authors is to be applauded because the question addressed is absolutely clinically relevant. Any surgeon involved in mitral valve repair is confronted from time to time with hesitation regarding the optimal length of implanted neochords. This length is not only important to avoid recurrent leaflet prolapse or induced leaflet restriction, but also, according to the present study, has an influence on subsequent papillary muscle forces. Excessive papillary muscle forces are assumed to put a leaflet segment in jeopardy of recurrent chordal lengthening or rupture.

The outcomes of this study should ideally lead to relevant clinical consequences. It would be wonderful to have an intraoperative tool to measure papillary muscle forces and hence determine optimal chordal length. Such a standardized approach could probably lead to more universal excellent mitral valve repair outcomes. Indeed, mitral valve repair currently remains a bit of an art—and some artists are better than others. So the question remains: Are the outcomes of this study applicable to daily practice?

Like usual, reality is often more complex than an experimental setup simulates. At first, there seems to be quite some difference between the 8 samples in measuring the mean papillary muscle force. Indeed, the SE of the mean papillary muscle force ranged from 0.2 to 0.3 N for each measured neochord length condition, which relative to the differences between measurements, is a large SE. To me it seems that this large variation influences generalizability and standardization negatively. Besides, only P2 areas were studied and we remain unsure whether or not these findings are also applicable to other prolapsing areas such as P1, P3, or even anterior mitral leaflet prolapse.

When confronted with ruptured chordae, mitral valve surgeons observe lengthened chords adjacent to the ruptured ones. The present experimental setup was created by inducing P2 prolapse by cutting of chords, whereas the adjacent chords remained unchanged. This is quite a different situation. It remains doubtful whether or not the current findings are applicable to a different scenario and this will require further study.

Finally, most mitral valve repairs in clinical practice are supplemented with an annuloplasty to prevent recurrent annular dilation and potentially reduce subvalvular stress. This is another factor that may render interpretation of the current data difficult.

Nevertheless, the authors are to be congratulated on a nice experimental study. Although there are shortcomings (as always), they made an excellent effort to answer a
clinically relevant question. Although we may come close to simulating clinical conditions in an experimental setup, it seems we are not quite there yet. I encourage the authors to continue their research efforts!

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