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Response to "Editorial Commentary: The Anterolateral Ligament: The Emperor's New Clothes?"

I read with interest and a certain amazement "Editorial Commentary: The Anterolateral Ligament: The Emperor's New Clothes?" by Dr. Andy Williams[1]

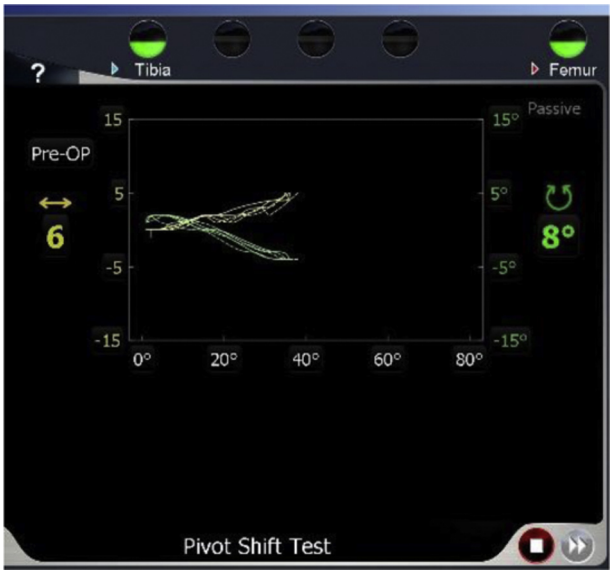

Fig 1. Diagram of the pivot shift test in the intact knee condition. The yellow line corresponds to anteroposterior translation; the green line to rotation.

on our article[2] "The Effect of Sequential Tearing of the Anterior Cruciate and Anterolateral Ligament on Anterior Translation and the Pivot-Shift Phenomenon: A Cadaveric Study Using Navigation." I wish to respond to some distinct and important elements of this commentary.

First of all, let me explain that our interest in the role of the secondary restraints of the anterior cruciate ligament (ACL) in the pathogenesis of rotational instability of the knee dates back to the late 1970s, and my first article on this topic was published in 1982.[3] Since that time, extra-articular tenodesis has been routinely performed following indications as recently published.[4-7] In comparison, Dr. Williams, whose "long interest" in this subject is attested to by his 2011 article,[8] can be considered the "new kid on the block." Moreover, as a full professor of Orthopaedics at University of Rome La Sapienza and chairman of the Orthopaedic Unit at Sant'Andrea University Hospital in Rome, I do not accept any lectures about scientific "rigor" and "due diligence." In actual fact, our biomechanical article recently published in *Arthroscopy* is only the last of a series of similar works from our group, the first one published in 2012,[9] 1 year before the article by Claes et al.,[10] in which the anterolateral ligament (ALL) was identified and described.

Turning to the content of the commentary, Dr. Williams reports as an example of lack of scientific rigor our citation of the work by Parsons et al.[11] on the importance of the ALL in controlling tibial internal rotation, missing that according to their findings, the ALL would be effective at knee flexion of $\geq 30°$. Dr. Williams reminds us that because "the devil is in the details," the entire manuscript should be read, not only the abstract, concluding that because the pivot shift (PS) occurs between 20° and 30° of knee flexion, according to the findings reported by Parsons et al.[11] The ALL is largely irrelevant to ACL injury and reconstruction. In our study, as shown in the videos and graphics, the PS appeared between 0° and 40° in the normal knees and in the ACL+ALL-injured knees (Figs 1 and 2), thus conferring a significant role to the ALL. Dr. Williams' comment is even more surprising given the comment by Parsons et al.[11] that "we can speculate that a positive PS sign in an ACL-intact knee could possibly be explained by unrecognized damage to the ALL." They actually identified this in all but 1 (83%) of the knees in their study. This raises 2 questions: how can unrecognized damage to a structure irrelevant or nonexistent produce a PS, and why was this irrelevant or nonexistent structure isolated by Parsons et al. in all but 1 knee? Therefore, the part of the commentary related to our possible misinterpretation of the work by Parsons et al. seems inappropriate.

Dr. Williams also suggested reading the article by Kittl et al.,[12] who identified the iliotibial band (ITB) as the most important restraint to internal rotation. I want to clarify that in all our studies,[13-16] we used hip-to-toe specimens in which the ITB was completely intact from proximal to distal insertion and was just incised along its fibers to approach the anterolateral capsule; this scenario is clearly more reliable than studies performed with above-the-knee cut specimens, without muscles and bony attachments. Moreover, the use of a navigation system allowed us to perform a real PS test on entire lower limbs and not a simulated PS with a 6DOF robot.

Furthermore, Dr. Williams questions the identification of the ALL in our article, suggesting that we did not know what we were cutting to create an ALL lesion. I would like to clarify that we just produced in the laboratory a kind of lesion that we know well and have found many times when exploring patients with an acute ACL tear.[17] Moreover, thanks to our long series of hundreds of patients operated on for apparently isolated acute ACL reconstruction with exploration of the lateral compartment since 1979,[7,17] we have never found a macroscopic tear of the ITB or its detachment from the tibial plateau. The same experience allowed us to perform several explorations in patients with a Segond fracture detectable on preoperative x-ray[18]; again, in all cases, the ITB was intact and the bony avulsion was found at the level of the ALL insertion site (Figs 3 and 4). Again, the question of Dr. Williams, "I am not sure which anatomic structure pulls off the Segond fragment," can receive a scientific response, and a published illustration[19] can be useful (Fig 5).

Finally, Dr. Williams extensively criticizes ALL reconstructions and extra-articular tenodesis, even though our study was only a time zero biomechanical