

1 **The Anterolateral Complex of the Knee: Results from the International ALC Consensus**
2 **Group Meeting**

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48 Abstract

49 The structure and function of the anterolateral complex (ALC) of the knee has created much
50 controversy since the 're-discovery' of the anterolateral ligament (ALL) and its proposed role
51 in aiding control of anterolateral rotatory laxity in the anterior cruciate ligament (ACL)
52 injured knee. A group of surgeons and researchers prominent in the field gathered to
53 produce consensus as to the anatomy and biomechanical properties of the ALC. The
54 evidence for and against utilization of ALC reconstruction was also discussed, generating a
55 number of consensus statements by following a modified Delphi process.

56 Key points include that the ALC consists of the superficial and deep aspects of the iliotibial
57 tract with its Kaplan fibre attachments on the distal femur, along with the ALL, a capsular
58 structure within the anterolateral capsule. A number of structures attach to the area of the
59 Second fracture and hence it is not clear which is responsible for this lesion. The ALC
60 functions to provide anterolateral rotatory stability as a secondary stabilizer to the ACL.
61 Whilst biomechanical studies have shown that these structures play an important role in
62 controlling stability at the time of ACL reconstruction, the optimal surgical procedure has
63 not yet been defined clinically. Concern remains that these procedures may cause
64 constraint of motion, yet no clinical studies have demonstrated an increased risk of
65 osteoarthritis development. Furthermore, clinical evidence is currently lacking to support
66 clear indications for lateral extra-articular procedures as an augmentation to ACL
67 reconstruction.

68 The resulting statements and scientific rationale aim to inform readers on the most current
69 thinking and identify areas of needed basic science and clinical research in order to help
70 improve patient outcomes following ACL injury and subsequent reconstruction.

71

72 **Introduction**

73 Since the 2013 publication by Claes et al. regarding the anatomy of the anterolateral
74 ligament (ALL)[7], there has been a great deal of controversy surrounding the presence of
75 the ALL, and its potential role in the control of anterolateral rotatory laxity of the knee
76 following anterior cruciate ligament (ACL) injury. Numerous anatomical and biomechanical
77 studies have followed, with conflicting results. While some studies have been promoting the
78 importance of the ALL[4, 7, 12, 27], others have been refuting it[15, 44, 57]. Journal
79 editorials have been written, some favouring[33] and others questioning the significance of
80 the ALL[37], and orthopaedic meetings are filled with varying opinions and interpretations
81 of the published data. Clinical studies have been published, with members of the
82 orthopaedic community developing new ways to address the 'rediscovered ligament', whilst
83 others have focused on the anterolateral soft tissues as a complex that may or may not
84 need to be addressed in the face of ACL injury[18].

85

86 With such controversy comes the need for clarity of thought, and a focus on those specific
87 areas where evidence is lacking. With good resources at hand, evidence should be utilized
88 to guide treatment paradigms; and where such evidence is lacking, the need for studies
89 investigating specific research questions should be identified. To this end, an international
90 consensus group was convened, with the task of producing a position statement on the
91 current evidence in terms of the anatomy and function of the anterolateral complex (ALC),
92 and the assessment and treatment of ALC injuries in association with an ACL injury.

93

94 Thirty-six international researchers and clinicians in the field were invited to join a meeting
95 to discuss the below points pertaining to the ALC and anterolateral rotatory laxity. The
96 group met in London, UK, in October 2017 with the specific aims of:

- 97 1. Developing a consensus in terms of the anatomical terminology utilized for
98 structures within the ALC.
- 99 2. Producing position statements as to the kinematic role of key structures in the knee,
100 pertaining specifically to anterolateral rotatory laxity and ACL deficiency.
- 101 3. Providing clinical guidance on when to utilize an anterolateral procedure in the ACL
102 deficient knee.

103

104 **Methods**

105 Thirty-six researchers and clinicians were initially contacted via email and asked to complete
106 an online survey compiled by the Chairs of the meeting (AG and CB). The questions posed
107 and collated responses may be found in the supplementary material. Based on the
108 responses of 33 participants, 22 statements were generated pertaining to the three main
109 aims of the meeting. A modified Delphi consensus discussion was then held during a one-
110 and-a-half-day meeting in London UK, attended in person by 27 individuals, with three
111 individuals providing prerecorded presentations and a further two calling in via
112 teleconference. Each structured session included a summary of the published literature, as
113 well as time in the cadaveric laboratory for dissections of the ALC and associated structures
114 and demonstration of reconstructive techniques. Following each structured session, a
115 consensus discussion was held, moderated by the two chairs of the meeting (AG & CB).
116 Each statement generated from the results of the survey was discussed and revised, until an
117 acceptable level of consensus was achieved. A majority of 80% was determined *a priori* as

118 being a satisfactory level of consensus. Opposing views were documented. Statements that
119 did not reach the required majority, or those that were felt to not be relevant were
120 discarded from the final paper (see supplementary material).

121

122 **Consensus Statements and Discussion**

123 Following discussion of the available evidence 13 statements were accepted and are
124 presented below. These are accompanied by a summary of the pertinent evidence and
125 rationale supporting each statement.

126

127 **Anatomy**

1. The ALL exists as a structure within the anterolateral complex.
2. The structures of the anterolateral complex, from superficial to deep, are:
 - Superficial IT band and iliopatellar band
 - Deep IT band incorporating
 - Kaplan fiber system
 - Supracondylar attachments
 - Proximal
 - Distal
 - Retrograde (Condylar) attachment continuous with the Capsulo-osseous layer of the IT band
 - ALL and capsule
3. The ALL is a capsular structure within Seebacher Layer 3[46] of the anterolateral capsule of the knee.
4. The ALL has variable gross morphology between individuals in terms of size and thickness.
5. The ALL predominantly attaches posterior and proximal to the lateral femoral epicondyle and the origin of the LCL, runs superficial to the LCL, and attaches on the tibia midway between the anterior border of the fibular head and the posterior border of Gerdy's Tubercle.
6. There is an attachment of the ALL to the lateral meniscus.

128 Numerous historical studies have investigated the structures on the anterolateral side of the
129 knee, from Segond's description of the eponymous fracture of the anterolateral tibia[47],
130 to Kaplan's original work in 1958 describing the layers and attachments of the iliotibial band
131 (ITB) to the femur[26], and then on to the paper by Terry et al., breaking down the lateral
132 fascia lata into its component parts[55]. It was Terry et al., in fact, who first described the
133 iliotibial tract as the 'true anterolateral ligament of the knee'. Further work by Lobenhoffer
134 et al. in 1987 documented the existence of a retrograde fibre tract, providing a static
135 stabilizer of the lateral side of the knee via its connection from the deep fibres of the IT tract
136 to the lateral tibial plateau[31]. In this article, they commented that this was the same
137 structure that Werner Müller had previously called the 'lig. Femoro-Tibiale laterale
138 anterius'[35].

139

140 Descriptions of the anterolateral complex anatomy are confused by overlapping
141 nomenclature. Vieira et al. are often attributed to being the first to describe the ALL[58],
142 although this was same name that Terry et al. used to describe the capsule-osseous layer of
143 the iliotibial tract. Vincent et al. further described a structure that was more anterior to the
144 lateral collateral ligament (LCL)[59], with Catherine et al. suggesting that the new ALL was in
145 fact the same structure that had previously been described by Hughston, namely the mid
146 third capsular ligament[4]. Following the initial description by Claes et al. in 2013, Dodds et
147 al.[12] and then Kennedy et al.[27] have provided the most distinct descriptions of this
148 structure that we now refer to as the ALL. Histologically, this structure has been
149 characterized by dense and well-organized connective tissue collagen bundles consistent
150 with ligamentous tissue[16]. Furthermore, it has been demonstrated that the ALL has

151 significantly different biomechanical properties to adjacent capsule and similar properties to
152 other capsular ligaments such as the inferior glenohumeral ligament[49].

153 Seebacher et al. described Layer 3 of the anterolateral capsule as splitting into a superficial
154 and deep lamina anterior to the LCL, and enveloping it[46]. Based on this information, the
155 group concluded that the ALL is a structure within Layer 3 of the anterolateral capsule, and
156 that the superficial lamina is the ALL with the deep lamina being the true capsule of the
157 knee at this level.

158

159 The present lack of consensus in terms of the nomenclature used to describe the various
160 structures of the ALC stems from a number of issues, including:

- 161 • Lack of clear photographs and corresponding diagrams in historical papers
- 162 • Description of anatomy on both embalmed and fresh specimens
- 163 • Differences in dissection technique that may introduce 'dissection artifact'

164

165 Following demonstration of a number of dissection protocols[4, 9, 29], the group was able
166 to identify and describe the key structures of the anterolateral complex, as illustrated in the
167 attached figures (Figures 1-7).

168

169 **Segond Fracture**

7. Multiple structures (ALL, deep ITB, and biceps aponeurosis) attach in the region of the Segond fracture and it remains unclear which may be responsible for this lesion

170

171 In regard to the Segond fracture, much debate ensued in regard to the cause of this bony
172 avulsion. Paul Segond originally described a ‘fibrous pearly band’ attached to the bony
173 avulsion that we now call the Segond fracture, which is pathognomonic of an ACL injury
174 [47]. Whilst there is little objective evidence as to the cause of this injury pattern, several
175 authors have demonstrated that the previous literature has probably underestimated the
176 incidence of this injury pattern. Specifically, Klos et al.[30] and Cavaignac et al.[5]
177 demonstrated that the incidence on ultrasound (30-50%) is higher than visualized with
178 either plain radiographs or MRI. More recent studies suggest that it is not only the ALL that
179 attaches in this region[6], but also the capsulo-osseous layer of the IT tract as well as an
180 expansion of the short head of biceps fascia[1].

181

182 **Biomechanics of the Anterolateral Structures**

8. The primary soft tissue stabilizer of coupled anterior translation and internal rotation near extension is the ACL. Secondary passive stabilizers include:
 - The ITB including the Kaplan fiber system
 - The lateral meniscus
 - The ALL and the anterolateral capsule

9. The ALL is an anisometric structure

183

184 A number of important cadaveric biomechanical studies have been published investigating
185 the kinematics of the knee following sectioning of the ACL and the anterolateral structures.
186 Spencer et al. demonstrated that sectioning of the ALL resulted in a statistically significant
187 increase in anterior translation and internal rotation after the ACL was sectioned during an
188 early-phase pivot shift[54]. Similar findings were also published by Rasmussen et al.[43],
189 clearly showing an increase in internal rotation following ALL sectioning using a 6-degree of

190 freedom robot. Sonnery-Cottet et al.[51] and Monaco et al.[34], both utilizing navigation,
191 demonstrated increased internal rotation laxity during a dynamic pivot shift test following
192 an ACL/ITB deficient and ACL/ALL deficient setting respectively.

193

194 Kittl et al. examined the effect of ALL sectioning, as well as division of the superficial and
195 deeper layers of the iliotibial tract[28]. Using a 6 degrees of freedom robot, they found the
196 ALL to have only a minor role in controlling internal rotation in the ACL deficient knee. The
197 IT tract, in particular the deep and capsulo-osseous layers, made a greater contribution to
198 internal rotation control at larger flexion angles, with the ACL having its greatest
199 contribution closer to extension.

200

201 Conversely, Guenther et al. examined the anterolateral capsule during anterior translation
202 and internal rotation by means of optical tracking analysis and strain mapping[15]. These
203 researchers observed the anterolateral capsule to behave more like a fibrous sheet rather
204 than a distinct ligamentous structure, disputing the existence of a discrete ALL. Thein et al.
205 published their findings in a serial sectioning study showing that the ALL only engaged in
206 load sharing beyond the physiological limits of the ACL[57]. As such they concluded that the
207 ALL was a secondary stabilizer to anterolateral translation only after loss of the ACL, rather
208 than a co-stabilizer.

209

210 Similar conclusions were made by Noyes's group, who further examined the role of the ALC
211 structures during a simulated pivot shift[21]. This was the first study to utilize a
212 combination of anterior translation, valgus and internal rotation. During this study, they
213 demonstrated that an isolated ALL sectioning in the ACL intact knee resulted in no increase

214 in anterior tibiofemoral compartment translation, concluding that the ALL does not function
215 as a primary restraint to the pivot shift [21]. In a further study, the same group observed
216 that sectioning of the ALL and the ITB in ACL deficient knees converted 71% of the
217 specimens to a grade 3 pivot shift as measured by composite tibiofemoral translations and
218 rotations[39]. In contrast, Inderhaug et al. demonstrated that when a combined ACL and
219 anterolateral injury exists, isolated ACL reconstruction fails to restore normal knee
220 kinematics. Specifically, Inderhaug et al. demonstrated that only combined ACL and lateral
221 extra-articular procedures (ALL reconstruction or lateral tenodesis) were able to restore
222 normal kinematics in this scenario[24].

223

224 The lateral meniscus also plays a role in the control of anterolateral rotation. Two studies
225 [32, 48] have both shown increased lateral compartment anterior translation and internal
226 rotation in the setting of lateral meniscus posterior root tears. The role of the ALL as a
227 peripheral anchor of the lateral meniscus has been questioned. Corbo et al. observed that
228 the infra-meniscal ALL fibers were significantly stiffer and stronger than the supra-meniscal
229 fibers[8]. The clinical significance of the infra-meniscal fibers is yet to be determined.

230

231 **Biomechanics of Lateral Extra-Articular Procedures**

10. Time zero biomechanical studies show lateral extra-articular procedures used as an augmentation to ACL reconstruction have the potential to over-constrain normal motion of the lateral compartment compared to the intact knee. The clinical significance of this is as yet unknown.

11. Causes of over-constraint of lateral extra-articular procedures may include:

- Fixation of the graft with the tibia in external rotation
- Over-tensioning of the graft

12. Despite concerns often being raised, to date the group is not aware of any

clinical evidence that lateral extra-articular procedures used as an augmentation to ACL reconstruction lead to accelerated progression of OA

232

233 A number of studies have now examined the biomechanics of ALC reconstruction, most of
234 them acknowledging the difficulties with extrapolating artificially created injury patterns
235 and laboratory results to the clinical scenario. Spencer et al. studied the effect on anterior
236 translation and internal rotation in an ACL deficient knee of both a Lemaire type lateral
237 extra-articular tenodesis (LET) compared with an ALL reconstruction as described by Claes et
238 al[54]. The ALL reconstruction had little effect on controlling rotation or translation;
239 however, we now know that the anatomical description that formed the basis of this
240 reconstruction was incorrect as the femoral graft position was anterior and distal to the
241 lateral epicondyle, not posterior and proximal. The LET produced a composite reduction of
242 rotation and translation with the latter reaching statistical significance.

243

244 Kittl et al. studied the length change patterns of ALC reconstructions based upon graft
245 attachment site [29]. The most isometric position was a proximal and posterior attachment
246 on the femur, attached distally to Gerdy's tubercle and with the graft passed deep to LCL.
247 They therefore concluded that a LET would be the most efficient form of reconstruction if
248 passed deep to the LCL.

249

250 Dodds et al. demonstrated that a femoral attachment posterior and proximal to the origin
251 of the LCL resulted in minimal length change during the flexion cycle[12]. Conversely, if
252 using the femoral attachment described by Claes et al.[7], a number of authors have shown
253 that the ALL lengthens with flexion, and as such would cause the ALL to tighten in higher

254 degrees of flexion [3, 29, 62]. From these studies, it is clear that if an ALL reconstruction is
255 to be of benefit in controlling the pivot shift, then an attachment posterior and proximal to
256 the LCL, and hence posterior to the center of rotation of the knee, should be chosen, so that
257 the ALL graft is tight near knee extension.

258

259 ALL reconstruction and LET have now been compared in ACL reconstructed knees.
260 Inderhaug observed that an LET graft tensioned at 20N and passed deep to the LCL was
261 effective at controlling rotation with minimal over constraint of internal rotation [25].

262 Both a modified Lemaire tenodesis and a modified Macintosh tenodesis, with a graft path
263 deep to the LCL, were found to restore intact knee kinematics in combination with an
264 anatomic ACL reconstruction. Furthermore, an ALL reconstruction based on previous
265 anatomic descriptions, was found to provide minimal effect on internal rotation of the knee.

266 In another study, the same authors demonstrated that by passing an LET graft deep to LCL,
267 the graft could be tensioned at a number of different flexion angles with no detrimental
268 effect[24]. The same study also demonstrated that the ALL reconstruction described by
269 Sonnery-Cottet et al. only controlled knee laxity when tensioned in full extension [24].

270 Studies by Schon et al. observed that an ALL reconstruction using a single graft tensioned
271 with 88N caused significant over constraint of internal rotation, no matter what angle of
272 fixation was used[45]. The high graft tension in this study has been questioned and may

273 explain the over-constraint observed, with later studies suggesting 20N to be the
274 optimal[25]. A further study by the same group compared their ALL reconstruction (based
275 on the anatomy described by Kennedy et al.[27]) to the modified Lemaire technique,
276 utilizing varying knee flexion and graft tension parameters at fixation. In this study, they

277 found that the Lemaire LET resulted in greater reduction in anterior translation and internal

278 rotation during a simulated pivot shift manoeuvre compared to the ALL reconstruction;
279 however, both reconstructions caused an element of over constraint [14].

280

281 Noyes et al. demonstrated that, at time zero in a knee with combined ACL and ALC injury, an
282 anatomically placed bone-patellar tendon-bone (BTB) ACL reconstruction secured in 25
283 degrees of knee flexion adequately controlled knee kinematics without the need for an
284 additional ALL reconstruction during a simulated pivot shift [38]. However, a residual
285 increase of 5-7 degrees of internal tibial rotation occurs with ALC injury at high flexion
286 angles, which is not controlled by ACL reconstruction. The clinical significance of this was
287 questioned as an indication for a concurrent LET procedure.

288

289 Similarly, Herbst et al. investigated the role of LET in both an isolated ACL injury and ACL
290 plus ALC injury[19]. These researchers concluded that the addition of an LET had no
291 additional benefit to knee stability in the isolated ACL deficient knee when an ACL
292 reconstruction was performed. However, the LET was required in the combined injury to
293 restore normal knee kinematics. The question raised by this work is whether an isolated
294 ACL injury is often seen, or if a concomitant ALC injury occurs at the time of ACL rupture.
295 Based on a number of other studies, it is clear that in a knee demonstrating a high-grade
296 laxity pattern, an isolated ACL injury is rarely seen. Instead, concomitant meniscus and
297 lateral soft tissue injuries are often observed, which may further support the need for an
298 anterolateral procedure in combination with an ACL reconstruction[36]. The prevalence of
299 concomitant anterolateral structure lesions in acute ACL injuries have been reported to vary
300 from 40% to 90% depending on the chosen method of detection.[5, 13, 17].

301

302 At present, it is not possible to ascertain which reconstruction technique is superior to
303 another, as the experimental set up and associated testing protocols differ between studies.
304 If using an LET type procedure, it is recommended to pass the graft deep to the LCL prior to
305 femoral fixation[24, 29]. Passing the graft deep to the LCL appears to provide a more
306 optimal direction of action throughout the flexion cycle, as well as providing a more
307 forgiving position of fixation, in terms of avoiding over constraint, as the LCL attachment
308 serves as a fulcrum. If instead performing a combined ACL and ALL reconstruction, the
309 technique described by Sonnery-Cottet, tensioned in full extension, would appear to
310 provide the most optimal ALL reconstruction kinematics[24].

311

312 Concerns relating to over-constraint of the lateral compartment remain an issue. Inderhaug
313 et al. have looked at lateral compartment contact pressures following LET[23]. They
314 demonstrated that a small increase in lateral compartment contact pressure was observed
315 after LET. However, the increased pressure was found to be insignificant compared with the
316 contact pressure seen in the lateral compartment during normal physiological loading [23].
317 The clinical importance of over constraint of internal rotation is currently unknown, but to
318 date there is no known evidence supporting lateral extra-articular procedures causing or
319 accelerating the development of osteoarthritis[11].

320

321 **Clinical Evidence for Augmentation of ACL Reconstruction with Lateral Extra-articular**

322 **Procedures**

- | |
|---|
| <p>13. Clinical evidence is currently lacking to support clear indications for lateral extra-articular procedures as an augmentation to ACL reconstruction. Appropriate indications may include:</p> <ul style="list-style-type: none">– Revision ACL |
|---|

- High Grade Pivot Shift
- Generalized ligamentous laxity/Genu recurvatum
- Young patients returning to pivoting activities

323

324 Lateral extra-articular tenodesis has a long clinical history. Having been the stand-alone
325 procedure of choice to address anterolateral knee laxity in the first half of the 20th Century
326 by Strickler, Lemaire and later Macintosh, it soon became apparent that intra-articular ACL
327 reconstruction would provide a better control of knee stability. Surgeons reported the
328 results of their lateral reconstruction, which was developed to aid in the control of
329 anterolateral rotatory stability, later to be added to intra-articular ACL reconstruction.
330 Lemaire, Losee, Andrews, Ellison and later versions of the Macintosh to name but a few
331 were reported in a variety of publications. Recent meta-analyses have shown that these
332 combined procedures performed well at reducing rotatory laxity, but no differences in
333 anterior translation nor patient-reported outcomes were observed[10, 20, 50].

334

335 Whilst remaining popular in Europe, the addition of an LET fell out of favour in North
336 America following publications from O'Brien et al. [40] and Anderson et al. [2]. The former
337 paper was a retrospective comparison of BTB ACL reconstruction with or without a lateral
338 tenodesis in 80 patients. Whilst there were significant methodological limitations of this
339 study, in particular its underpowered nature to elicit a difference in clinical outcome, the
340 lack of differences in outcome and the concern of over-constraint in these patients led to
341 the recommendation from an AOSSM consensus group to abandon the lateral-based
342 procedures[41]. A commentary from James Andrews in the AJSM following publication of
343 the O'Brien paper suggested that whilst good results can be achieved with an isolated BTB
344 ACL reconstruction, there are likely to be individuals who may still benefit from a lateral

345 procedure. The latter paper of Anderson compared three surgical techniques, concluding
346 that similar results could be found with either a hamstrings or patellar tendon autograft ACL
347 reconstruction, with a lateral tenodesis offering very little benefit. Of note, they cautioned
348 about the risk of over-constraint of internal rotation, and hence the concern for the
349 development of OA, although this was not specifically studied.

350

351 With recent studies showing a high failure rate in young patients [60], there is likely room
352 for improvement in ACL reconstruction methods. However, these failures cannot all be
353 attributed to the technique itself, as there are many reasons for ACL reconstruction failure.
354 These include poor neuromuscular rehabilitation, early return to sport and participation in
355 high risk pivoting sports. However, at the time of surgery, there are still many areas where
356 surgeons can influence outcome. Good surgical technique is paramount, including
357 avoidance of the technical error of improper graft placement. Failure to address meniscal
358 tears, concomitant soft tissue laxity patterns and issues of alignment may all contribute to a
359 higher risk of ACL failure.

360

361 Systematic reviews with meta-analyses of comparative studies [10, 20, 50] have all
362 demonstrated that the addition of a lateral based procedure to an ACL reconstruction
363 improves rotational laxity control, but has no impact on anterior translation nor patient
364 reported outcomes. Importantly, no studies have demonstrated an increased risk of
365 osteoarthritis with the addition of an LET. Zaffagnini et al. recently published the 20 year
366 outcomes of an over-the-top hamstring ACL reconstruction with a lateral tenodesis[61].
367 There was no generation of lateral compartment or patellofemoral OA associated with the
368 procedure. Similar results were found in a long term follow up of patients treated in

369 Lyon[42]. A more recent meta-analysis did not find any evidence of OA in the knee in 11
370 years of follow up, contrary to reports of isolated LET procedures which clearly showed an
371 increased prevalence of OA when the ACL was not addressed concomitantly[11].

372

373 At present, there is no high-level evidence to guide clinicians as to when a lateral based
374 procedure should be added to an ACL reconstruction. Historic studies have tended to
375 include 'all-comers', and were generally based upon small numbers of patients. Sub-group
376 analyses in meta-analyses have therefore not been possible due to the significant
377 heterogeneity of inclusion and exclusion criteria.

378

379 The more recent studies by Sonnery-Cottet et al. have demonstrated the potential benefit
380 of adding an ALL graft to a hamstring tendon ACL reconstruction. In 2015, two year
381 outcomes of 92 patients were reported demonstrating only a 1% re-rupture rate with only 7
382 patients having a grade one pivot shift[53]. This was followed in 2017 by a comparative
383 cohort study of 502 young patients engaging in pivoting sports, and therefore exposed to a
384 high risk of graft rupture, undergoing ACL reconstruction[52]. In the largest comparative
385 series of any type of extra-articular reconstruction to date, the data has demonstrated
386 significantly lower ACL graft rupture rates in the combined ACL and ALL group (4%) when
387 compared to isolated patellar tendon (16%) and hamstrings tendon autograft (10%) groups,
388 with a further study observing low complication rates[56].

389

390 In contrast, a recent study by Ibrahim et al. has shown minimal differences in the outcome
391 following addition of an ALL graft to a standard hamstrings autograft ACL
392 reconstruction[22]. However, this study utilized a non-anatomic ALL reconstruction

393 technique (femoral insertion proximal and anterior to LCL, instead of posterior and
394 proximal), was underpowered and did not select out patients who would be at a higher risk
395 of failure, such as young patients returning to pivoting sport or those with high grade laxity.

396

397 Based on the current evidence, the consensus group was unable to make definitive
398 recommendations as to when a lateral procedure should be added to an ACL reconstruction.

399

400 **Conclusions**

401 The 13 consensus statements generated from the ALC Consensus group are intended to
402 provide some clarity of anatomical nomenclature and a better understanding of pertinent
403 biomechanics associated with the ALC. Strategies to address persistent anterolateral
404 rotatory laxity and ACL reconstruction failure are warranted due to the high rates of graft
405 failure that we continue to see in young active individuals. There has been controversy over
406 the 're-emergence' of the ALL and associated anterolateral reconstructive procedures. It is,
407 however, evident from this consensus that there is still considerable clinical research to be
408 performed to determine the optimal scenarios for augmentation of a primary ACL
409 reconstruction with an anterolateral procedure in order to improve outcomes for patients.

410

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413 Nephew PLC to enable this meeting.

414

415

416

417 **Figure Legend**

418 **Figure 1.** Lateral structures of the right knee showing the superficial IT band, iliopatellar
419 band and the attachment to Gerdy's tubercle. The line of asterisks (*) represents the deep
420 IT band corresponding to the capsulo-osseous layer

421

422 **Figure 2.** The superficial ITB is reflected posteriorly, demonstrating the Kaplan fibre system.
423 The Proximal and distal (supracondylar) fibres are shown, continuing distally from the
424 intermuscular septum.

425

426 **Figure 3.** The retrograde (condylar) Kaplan fibres are shown to be continuous with the
427 capsulo-osseous layer of the ITB, as marked by the line of asterisks (*) attaching distally to
428 Gerdy's tubercle.

429

430 **Figure 4.** A) The FCL (*) is shown with the knee at 90°, neutral tibial rotation; B) An internal
431 tibial rotation torque is applied to the tibia demonstrating the ALL (#) tensioned across the
432 FCL, running from posterior and proximal to the lateral femoral epicondyle to a position
433 midway between the fibular head and Gerdy's tubercle.

434

435 **Figure 5.** The ALL is dissected free from the FCL, shown to be within layer 3 of Seebacher's
436 layers of the lateral retinaculum.

437

438 **Figure 6.** The close relationship of the ALL, FCL and popliteus tendon is demonstrated.

439

440 **Figure 7.** The relationship of the ALL and lateral meniscus is demonstrated, with the scissor
 441 demonstrating the course of the lateral inferior geniculate artery. Meniscofemoral and
 442 meniscotibial attachments of the ALL can be observed.

443

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