Detecting Associations between Knee Rotational Laxity and Kinematics in a Healthy Population

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Abstract

Passive knee laxity, which is determined by the soft tissue and articular structures of the knee, allows for translation and rotation of the knee. While laxity increases following soft tissue injury, there is a lack of clinical evidence demonstrating a relationship between increased passive laxity and subsequent clinical problems such as osteoarthritis. However, there is increasing evidence that abnormal active rotational kinematics during walking are a potential risk factor for osteoarthritis (OA) initiation and progression. Establishing a relationship between passive knee laxity and active knee kinematics is therefore clinically important, but at present there is a paucity of information on this interaction between passive and active knee movement. The purpose of this study was to test for correlations between passive knee laxity and active knee rotational movement.

Active knee kinematics of 15 healthy subjects was evaluated during walking and jogging, and translational and rotational passive laxity was tested with the KT1000 and custom Knee Rotation Device (KRD), respectively.

The results showed that increased rotational laxity correlated with increased knee rotation during the stance phase of walking ($R^2 = 0.41$, $p = 0.03$), but this relationship was not significant during the stance phase of jogging ($R^2 = 0.19$, $p = 0.17$). There was no correlation between translational and rotational knee laxity ($R^2 = 0.06$, $p = 0.26$) or translational laxity and rotational kinematics ($R^2 = 0.03$, $p = 0.25$). These results suggest that the structures of the knee that determine passive rotational laxity influence rotational motion while walking more significantly than while jogging, possibly because of the stronger influence muscle activation has on knee rotation while jogging.

In conclusion, the correlation between rotational laxity and kinematics may explain why ACL deficient and reconstructed individuals, who display abnormal rotational knee motion during walking, develop early-onset osteoarthritis.

Recently, the medical and scientific communities have shown heightened interest in understanding knee laxity and its impact on the ambulatory motion of the knee. Knee laxity is influenced by the support structures of the knee, such as the ligaments, menisci, and muscle tendons [1-2]. Knee laxity is most commonly measured during two different motions at the knee: anterior-posterior translation and internal-external rotation [3]. Translational laxity can be measured clinically by the KT1000 device [4], but rotational laxity does not have widely adopted or useful clinical tools for measurement [5-7].

Accurately measuring rotational knee laxity is especially important because abnormal rotational laxity has been reported after anterior cruciate ligament (ACL) and meniscus tears [5, 8, 9]. The ACL functions to resist anterior translation and internal rotation of the knee [10], and the meniscus functions to disperse the weight of the body and reduce friction during movement in the knee [11]. Individuals with an ACL or meniscus tear are also at a high risk of developing early-onset osteoarthritis (OA) [12], a disease suggested to be caused by changes in the load-bearing regions of cartilage in the knee [13]. Given that abnormal rotational laxity follows these injuries, it is necessary to better understand the impact of rotational laxity in healthy knees on other knee functions and its role in altering knee loading patterns and OA development.

While studying laxity provides information on the passive translational and rotational motions of the knee, analyzing knee kinematics, provides greater insight into the active translational and rotational motions of the knee during weight-bearing activities such as walking or jogging. As with rotational laxity, significantly abnormal rotational kinematics have also been detected in individuals with meniscal tears, ACL tears, and ACL reconstructions [10, 14-16]. Kinematic data reveal the extent to which the knee rotates under load, which is relevant to understanding the initiation of OA because during altered rotation, the loading profiles of the cartilage in the knee can change. Cartilage, which has previously been conditioned for specific loading, begins to see altered loads for which it is not well adapted, which is believed to initiate the degenerative process [13, 17].

A previous study has shown that the translational laxity measured with the KT1000 can predict translational kinematics of ACL reconstructed knees [18]. However, despite many studies on knee rotational properties [1, 5-7, 16, 19-21], there remains a lack of knowledge on the relationship between knee rotational laxity and kinematics. Physicians believe that a more "vertical" ACL graft placement during reconstruction results in abnormal dynamic rotational motion [22], but there is no present clinical test to assess this relationship. A greater understanding of the relationship between passive rotational laxity and active kinematics may provide insight onto the risk factors for OA development.

A new device named the Knee Rotational Device (KRD) has recently been developed in the Stanford University Biomotion

Fig. 1. Subject with reflective PCT markers used for motion tracking with Qualysis.

Laboratory to measure passive rotational laxity for this purpose. The objective of this study was to assess correlations in healthy individuals between passive knee laxity and active kinematics by testing the following hypotheses: 1.) Increased rotational knee laxity is associated with increased knee rotation during walking and jogging; 2.) Increased anterior-posterior (AP) translational knee laxity is associated with increased knee rotation during walking; 3.) Increased AP translational knee laxity is associated with increased rotational knee laxity.

Materials and Methods

Fifteen healthy subjects were recruited based on the following selection criteria: aged between 18 and 40 years, BMI under 28, and no previous knee injury (avg 24 years old, 1.67 m, 62 kg). IRB-approved informed consent was obtained from each subject.

Data for active tibial rotation were collected according to a previously described protocol [23, 24]. Each subject was recorded performing three walking and jogging trials at a self-selected normal speed. The point-cluster technique, a set of 21 skin-based reflective markers, was employed to determine the amount of rotation of the tibia in relation to the femur during these trials (Fig. 1). A 9-camera optoelectronic motion capture system, Qualisys, was used to collect marker motion data. The subject walked and jogged over a force plate (Bertec, Columbus OH) embedded in the floor to allow for identification of the stance phase of walking. The stance phase of the walking cycle is defined as when the foot is in contact with the ground. Stance phase knee rotation from the three trials was averaged, resulting in one rotation value for each activity per knee per subject. The absolute side-to-side difference in knee rotation was calculated for the subject, with the left knee used as the reference. The motion of the knee was tracked by relating the motion of the marker clusters to anatomical coordinate systems based upon bony landmarks from the reference trials. This method of motion analysis has been previously described [10, 24, 25].

After completing the walking and jogging trials, each subject was tested with the KRD to measure passive rotational laxity while wearing the reflective markers used during the gait test (Fig. 2). The KRD consists of an orthopedic boot mounted to a stable circular platform that enables rotation in the axial plane. Each subject lied down in the supine position on a mattress with one foot strapped into an ankle brace within the orthopedic boot, which limited ankle rotation. To minimize coupled femoral rotations, the knee was positioned at 25 degrees of flexion, which was measured by a goniometer, and a large strap was placed across the subject's thigh (Fig. 3). Ankle and femoral rotation were limited to allow for measurements of only the rotation of the tibia relative to the femur. Two unloaded reference trials were recorded with each subject's knee in natural alignment, followed by two loaded trials when a constant torque of 3.5 N was applied once a 1.8 kg weight was attached to the device. The weight rotated the platform axially, resulting in the isolated rotation of the tibia (Fig. 3 and 4). This torque was determined to be safe based on results from previous studies with similar or larger applied torques [5, 20, 21]. Subjects were instructed to relax the leg muscles during the loaded trial to allow for passive tibial rotation.

Individual knee rotation values were calculated for the reference and loaded trails based on the orientation of the reflective marker clusters of the tibia with respect to the femur. The amount of knee rotation was averaged for each of the two trials, and then the average amount of rotation during the reference trial was subtracted from the average amount of rotation during the loaded trial. The absolute side-to-side difference in average rotation was calculated for each subject, with the left knee used as the reference. This value was used in the following equation to calculate rotational laxity, resulting in one rotational laxity value per subject.

$$Knee\ Rotational\ Laxity = \frac{Tibial\ Rotation\ (\text{deg})}{Applied\ Torque\ (N \cdot m)}$$

Immediately following rotational laxity testing, each subject was tested with the KT1000 device to measure translational knee laxity. In order to eliminate interpersonal variability with the device, one person conducted all KT1000 tests and measured maximum anterior translation. Absolute side-to-side differences

Fig. 2. Image of Knee Rotation Device (KRD). A weight of 1.83 kg applies a 3.51 N torque, rotating the platform axially.
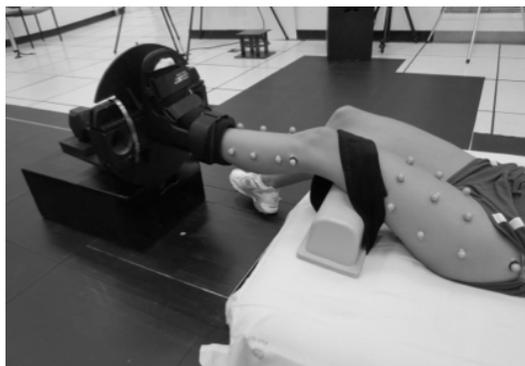

Fig. 3. Rotational laxity testing set-up with KRD. Subject lies with foot in orthopedic boot with the knee at 25 degrees of flexion. A black Velcro strap is placed over the thigh to prevent femoral rotation.

Fig. 4. Image of weight applied to KRD. Motion capture cameras detect changes in position of the reflective markers on the tibia and femur to calculate the knee rotation.
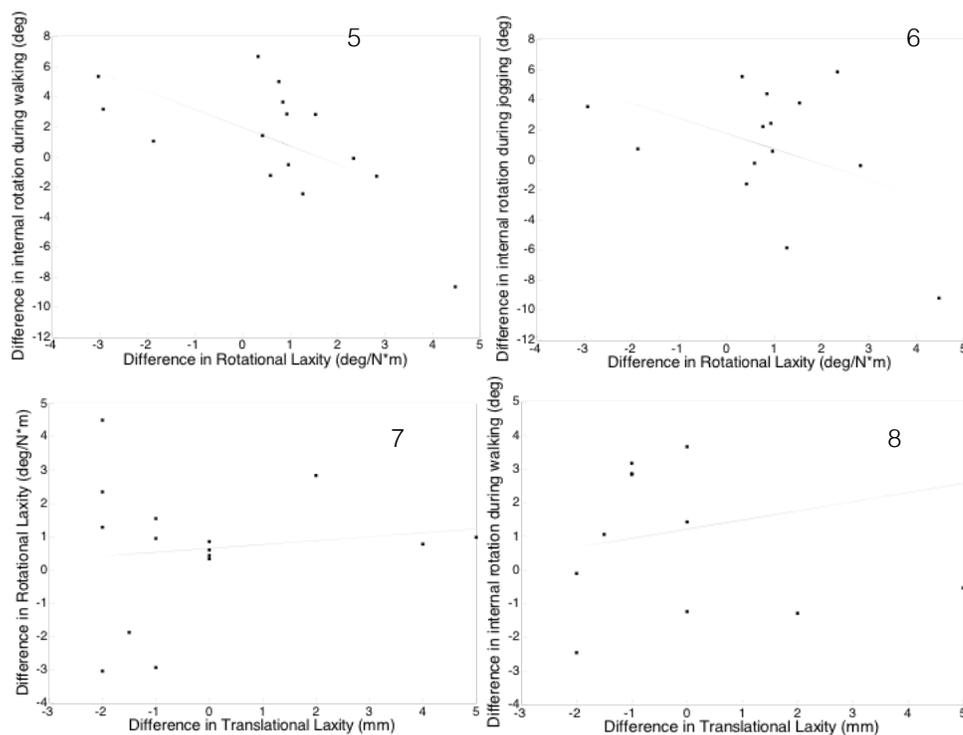
Fig. 5. Side-to-side difference in rotational laxity compared to side-to-side difference in average internal rotation of the knee during the stance phase of walking ($R^2 = 0.41$, p = 0.03).

Fig. 6. Side-to-side difference in rotational laxity compared to side-to-side difference in average internal rotation of the knee during the stance phase of jogging ($R^2 = 0.19$, p = 0.17).

Fig. 7. Side-to-side difference in translational laxity compared to side-to-side difference in rotational laxity ($R^2 = 0.06$, p = 0.26).

Fig. 8. Side-to-side difference in translational laxity compared to side-to-side difference in average internal rotation of the knee during the stance phase of walking ($R^2 = 0.03$, p = 0.25).

were calculated, with the left knee used as the reference, resulting in one maximum translational laxity value per subject.

Four linear regression statistical analyses were performed on the data sets. First was a comparison of side-to-side rotational knee laxity differences to the average amount of knee rotation during the stance phase of walking, followed by the same laxity difference compared to the average amount of knee rotation during the stance phase of jogging. Third, side-to-side differences in translational laxity were compared to side-to side differences in rotational laxity. Lastly, these translational laxity differences were compared to the average amount of knee rotation achieved during the stance phase of walking. Significance was determined with an alpha value set at 0.05.

Results

During the walking and jogging trials of the gait analysis, the subjects showed an average side-to-side difference in internal tibial rotation throughout stance phase of $1.19° \pm 3.82°$ and $0.85° \pm 4.25°$, respectively. The average side-to-side difference in rotational laxity measured by the KRD was $0.63° \pm 2.01°/Nm$, and the average side-to-side difference in maximum translation measured by the KT1000 was 0.1 mm.

After comparing the knee rotational laxity to knee rotation during the stance phase of walking, the linear regression showed a significant correlation between greater rotational knee laxity and greater rotation during gait ($R^2 = 0.41$, p = 0.03) (Fig. 5). However, the correlation between rotational knee laxity to rotation during the stance phase of jogging was not statistically significant ($R^2 = 0.19$, p = 0.17) (Fig. 6). The amount of knee translational laxity showed no significant correlation with the amount of rotational laxity ($R^2 = 0.06$, p = 0.26) (Fig. 7), nor was it associated with the amount of knee rotation during the stance phase of walking ($R^2 = 0.03$, p = 0.25) (Fig. 8).

Discussion

Correlation between rotational laxity and knee rotation during walking

The results supported the hypothesis that increased knee rotational laxity is correlated with increased knee rotation during the stance phase of walking, which suggests that the structures that determine passive laxity do influence knee rotational motion during walking.

This finding that passive rotational laxity influences dynamic rotations may explain why individuals with ACL ruptures or reconstructions display a 60-90% higher risk of developing OA in their injured knee than their contralateral knee [12, 27]. While the ACL functions to resist anterior translation and internal rotation of the knee, there is increasing evidence that abnormal knee rotational kinematics is the critical factor in the development of knee OA in this population [13]. It has been reported that reconstructed knees display abnormal rotation during walking relative to the contralateral knee [16], but passive rotational laxity has yet to be studied. It is possible that the increased abnormal laxity following injury leads to abnormal rotational kinematics in ACL reconstructed knees during walking, which may be a potential risk factor for osteoarthritis. Given that physicians report that ACL grafts are often placed more vertically than the native ACL during reconstruction, this more vertical orientation may be causing abnormal rotational laxity, which may lead to abnormal rotational kinematics and eventual OA development.

This correlation may also explain why women have a greater risk of developing early-onset osteoarthritis than males. Previous studies have shown that not only do women display greater rotational knee laxity than males [5, 20, 28], but are also 1.7 times more likely to develop early-onset OA [28]. A study of female knee cadavers reported that knees with OA had significantly greater laxity than knees without OA [29]. Based on similar results, several researchers have inferred that abnormal laxity leads to premature osteoarthritis [29, 30]. The results of this laxity

study establish that increased rotational laxity is associated with increased rotation at the knee during walking, which motivate further studies on gender differences in knee rotation during walking and rotational laxity to determine if these are risk factors for osteoarthritis in women.

Correlation between rotational laxity and knee rotation during jogging

Although increased rotational laxity was significantly correlated with increased rotation of the knee during the stance phase of walking, the hypothesis was not supported for the results of the jogging trials. There was a trend towards increased rotational laxity correlating with increased rotational kinematics during jogging, but it was not statistically significant. This weaker correlation during jogging is possibly due to greater muscle activation during jogging than during walking [31]. Increased muscle activation has been suggested to limit the amount of knee rotation [24, 32, 33], which would explain why the relationship between the passive and active motion is not as significant during jogging. Since the KRD only measures passive rotational laxity, it is understandable that rotational laxity is not correlated as significantly with active rotation during jogging than during walking.

Correlation between translational laxity and rotational laxity

Comparing the rotational and translational laxity results showed that the two laxity measurements are not associated with each other, suggesting that one laxity measurement should not be used to predict the other. Clinicians often evaluate general body laxity by bending the thumb back to the wrist or hyperextending the fifth finger, and relating the degree of laxity present within that joint to all joints [34]. However, our findings suggest that general laxity measurements are not sufficient in describing joint laxities in all directions of motion. This suggests that translational laxity measurements of the knee would not be sufficient in predicting rotational knee kinematics, and that independent rotational laxity testing would be necessary to make this prediction.

Correlation between translational laxity and knee rotation during walking

The fact that passive AP translation was not associated with the amount of knee rotation during walking is consistent with clinical studies that report restoration of normal translational knee laxity after surgery does not mitigate the risk of premature OA [19]. These results point to the need for an independent measure of passive rotational laxity in patients with ACL injury. The finding that the range of passive rotational laxity in a healthy population correlates with variation in dynamic rotation during walking suggests that the increased passive rotational laxity following ACL injury will be a good marker for abnormal dynamic rotation during walking since the range of rotational laxity will be greater.

Limitations

This study had several limitations in both the laxity and kinematics testing. Although the design of the KRD attempts to minimize ankle motion, it is uncertain the extent that a subject's ankle rotated within the device. To limit this error, we used verbal feedback from the subjects regarding their ankle motion during laxity testing and adjusted the ankle brace if they reported motion. The skin-based marker system for estimating knee kinematics may have also introduced error. The reflective markers reveal the motion of the skin and the soft tissue overlying the bone instead of the bones themselves, but the point cluster technique attempts to minimize this error [35], Other kinematics testing methods exist, such as cine-MRI, fluoroscopy, and high-speed radiography, but skin-marker-based motion analysis still remains the most feasible and effective way of tracking kinematics during overground walking.

Conclusion

This study demonstrated the potential to develop a clinical test to assess passive rotational laxity that can reflect dynamic function. This test could help evaluate the capacity of ACL reconstruction to restore dynamic rotation, and more importantly, the test could be used to avoid the risk of OA in the long term. Clinicians would benefit from the immediate feedback supplied by the meaningful measure of passive rotational laxity following ACL reconstruction. Future studies may include using this clinical test to determine whether small variations in ACL graft placement have an affect on active rotational kinematics that can be predicted by the rotational laxity measurements. It is possible that restoring natural rotational laxity with more native graft placement will restore normal rotational kinematics, which may reduce subsequent clinical problems following reconstruction such as OA.

References

1. Wang CJ and Walker PS. Rotatory laxity of the human knee joint. J Bone Joint Surg Am 1974; 56.1:161-170.
2. Markolf KL, Mensch JS, Anstutz HC. Stiffness and laxity of the knee—the contributions of the supporting structures. A quantitative in vitro study. J Bone Joint Surg Am 1976; 58.A:664-674.
3. Hsieh HH and Walker PS. Stabilizing mechanisms of the loaded and unloaded knee joint. J Bone Joint Surg Am 1976; 58.A:87-93.
4. Daniel DM et al. Instrumented measurement of anterior laxity of the knee. J Bone Joint Surg Am 1985; 67:720-726.
5. Haughom BD, Souza R, Schairer WW et al. Evaluating rotational kinematics of the knee in ACL-ruptured and healthy patients using 3.0 Tesla magnetic resonance imaging. Knee Surg Sports Traumatol Arthrosc 2011; 20.4:663-670.
6. Lorbach O, Wilmes P, Theisen D et al. Reliability testing of a new device to measure tibial rotation. Knee Surg Sports Traumatol Arthrosc 2009; 17:920-926.
7. Musahl V, Bell KM, Tsai AG et al. Development of a simple device for measurement of rotational knee laxity. Knee Surg Sports Traumatol Arthrosc 2007; 15.8:1009-1012.
8. Markolf KL, Kochan A, Amstutz HC. Measurement of knee stiffness and laxity in patients with documented absence of the anterior cruciate ligament. J Bone Joint Surg Am 1984; 66.A:242-252
9. Zhang LQ, Zeng K, Wang G et al. Dynamic and static properties of the human knee joint in axial rotation. IEEE/EMBS 1997; 19:1738-1741.
10. Andriacchi, T.P. and Dyrby, C.O. Interactions between kinematics and

loading during walking for the normal and ACL deficient knee. J Biomech 2005; 38:293-298.

11. Fithian DC, Kelly MA, Mow VC. Material properties and structure-function relationships in the menisci. Clin Orthop Relat Res 1990; 252:19-31.

12. Lohmander LS, Englund PM, Dahl LL et al. The long-term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis. Am J Sports Med 2007; 35.10:1756-1769.

13. Andriacchi TP, Mundermann A, Smith RL et al. A framework for the in vivo pathomechanics of Osteoarthritis at the knee. Ann Biomed Eng 2004; 32.3:447-457.

14. Netravali NA, Giori NJ, Andriacchi TP. Partial medial meniscectomy and rotational differences at the knee. J Biomech 2010; 43.15:2948-2953.

15. Zhang LQ, Shiavi RG, Limbird TJ et al. Six degrees-of-freedom kinematics of ACL deficient knees during locomotion—compensatory mechanism. Gait Posture 2003; 17.1:34-42.

16. Scanlan SF, Chaudhari AMW, Dyrby CO et al. Differences in tibial rotation during walking in ACL reconstructed and healthy contralateral knees. J Biomech 2010; 43:1817-1822.

17. Andriacchi TP, Koo S, Scanlan SF. Gait mechanics influence healthy cartilage morphology and osteoarthritis of the knee. J Bone Joint Surg Am 2009; 91.1:95-101.

18. Scanlan SF, Chaudhari AMJ, Blazek K et al. Sagittal ACL graft orientation influences passive and dynamic anterior tibial translation. Proceedings of ASB 2007; P4-14.

19. Tashman S, Collon D, Anderson K et al. Abnormal rotational knee motion during running after anterior cruciate ligament reconstruction. J Sports Med 2004; 32.4:975-983.

20. Mouton C, Seil R, Agnostinis H et al. Influence of individual characteristics on static rotational knee laxity using the Rotameter. Knee Surg Sports Traumatol Arthrosc 2012; 20:645-651.

21. Branch TP, Browne JE, Campbell JD et al. Rotational laxity greater in patients with contralateral anterior cruciate ligament injury than healthy volunteers. Knee Surg Sports Traumatol Arthrosc 2010; 18:1379-1384.

22. Brophy RH, Selby RM, Altchek DW. Anterior Cruciate Ligament Revision: Double-Bundle Augmentation of Primary Vertical Graft. Arthroscopy 2006. 22.6:683.e1-683.e5

23. Andriacchi TP, Alexander EJ, Toney MK et al. A point cluster method for in vivo motion analysis: applied to a study of knee kinematics. J Biomech Eng. 1998; 120:743-749.

24. Dyrby CO and Andriacchi TP. Secondary motions of the knee during weight bearing and non-weight bearing activities. Journal of Orthopaedic Research 2004; 22:794-800.

25. Andriacchi TP, Dyrby CO, Johnson TS. The use of functional analysis in evaluating knee kinematics. Clin Orthop 2003; 410:44-53.

26. Park HS, Wilson NA, Zhang LQ. Gender differences in passive knee biomechanical properties in tibial rotation. J Orthop Res 2008; 36.7:937-944.

27. Lohmander LS and Roos H. Knee ligament injury, surgery and osteoarthrosis. Truth or consequences? Acta Orthop Scand 1994; 65.6:605-609.

28. Felson DT, Zhang Y, Hannan MT. The incidence and natural history of knee osteoarthritis in the elderly, the framingham osteoarthritis study. Arthritis Rheum 2005; 38.10:1500-1505.

29. Graham GP and Fairclough JA. Early osteoarthritis in young sportsmen with severe anterolateral instability of the knee. Injury 1988; 7:200-207.

30. Scott D, Bird H, Wright V. Joint laxity leading to osteoarthrosis. J Rheumatol Rehabil 1979; 18:167-169.

31. Cappellini G, Ivanenko P, Poppele RE et al. Motor Patterns in Human Walking and Running. J Neurophysiol 2006; 95.6:3426-3437.

32. MacWilliams BA, Wilson DR, DesJardins JD et al. Hamstrings cocontraction reduces internal rotation, anterior translation, and anterior cruciate ligament load in weight-bearing flexion. J Orthop Res 1999; 17.6:817-822.

33. More RC, Karras BT, Neiman R et al. Hamstrings—an anterior cruciate ligament protagonist: an in vitro study. Am J Sports Med 1993; 21.2:231-237.

34. Myer GD, Ford KR, Paterno MV et al. The effects of generalized joint laxity on risk of anterior cruciate ligament injury in young female athletes. Am J Sports Med 2008; 36.6:1073-1080.

35. Alexander EJ and Andriacchi TP. Correcting for deformation in skin-based marker systems. J Biomech 2001; 34:355-361.

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