Association of Injury Energy Level and Neurovascular Injury Following Knee Dislocation

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Objective: To describe the associations between mechanism of injury energy level and neurovascular injury (NVI) following knee dislocation (KD) using a large representative sample of trauma patients and to examine risk factors within these groups.

Design: Retrospective cohort study.

Setting: Trauma centers participating in the American College of Surgeons National Trauma Data Bank.

Participants: Adult patients with KD without lower extremity fracture.

Intervention: Patients were grouped as ultra-low, low, or high-energy based on injury mechanism. Univariate/multivariate analyses assessed associations of energy level with NVI and of patient characteristics with NVI within energy-level groups.

Main Outcome Measurements: Rate of nerve and blood vessel injury.

Results: One hundred twenty-four patients with KD were identified; 181 sustained ultra-low-energy mechanisms, 275 low-energy, and 868 high-energy. Nerve injury occurred in 6% of ultra-low-energy injuries, 7% in low-energy, and 3% in high-energy ($P = 0.03$). Vessel injury occurred in 21% of ultra-low-energy injuries, 17% in low-energy, and 13% in high-energy ($P = 0.01$). On multivariate analyses, obesity was associated with nerve injury in the ultra-low-energy group (OR 4.9; 95% CI 1.0–24.0) but not with other energy levels. Obesity was also associated with vessel injury in the ultra-low-energy group (OR 4.0; 95% CI 1.6–9.7). Smoking, hypertension, and diabetes were not associated with NVI.

Conclusions: NVI following KD is more common after lower energy-level mechanisms. Obesity is associated with NVI in lower energy-level mechanisms. Physicians should be vigilant in screening for NVI in the setting of KD even with seemingly benign mechanisms of injury, especially in patients with obesity.

Key Words: knee dislocation, neurovascular injury, mechanism energy

Level of Evidence: Prognostic Level III. See Instructions for Authors for a complete description of levels of evidence.

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INTRODUCTION

Knee dislocation (KD) is a potentially catastrophic orthopaedic injury, which can result in limb-threatening neurovascular injury (NVI). The incidence of this uncommon injury, reported in 3 of 100,000 persons per year,[1] which may be underestimated because of spontaneous reduction before presentation—estimated to occur in up to 50% of cases,[2,3]– may result in the KD being unrecognized. The rate of concomitant NVI with KD varies widely, with vascular injury reported in 5%–65% of cases[2–13] and neurological injury reported in 4.5%–40% of cases.[8–10,12,14,15] The injury itself also represents a heterogeneous entity, as it can present after a range of significantly different mechanisms, and with or without associated fracture.

Keating summarized several epidemiological studies of KD and described 3 main mechanisms of injury related to motor vehicle accidents, sports, and falls. These were classified as high-energy, low-energy, and ultra-low-energy, respectively. The high-energy motor vehicle–related accidents accounted for approximately one-half of cases, low-energy sports injuries for one-third of cases, and ultra-low-energy falls constituting a minority, 12% of cases.[6] However, it should be noted that reports of KD following falls are increasing, especially in the obese population.[16]

Previous studies investigating risk factors for NVI following KD have found obesity to be associated with ultra-low-energy mechanisms, although it is not possible to conclude whether this is due to the mechanism or obesity itself.[2,16–19] Natsuhara et al[20] reported, in a large database study, male sex also to be associated with increased risk of vascular injury, although injury mechanism was not considered and this association may reflect other factors.

The most effective evaluation and management of knee dislocations remains controversial. A better understanding of the risk factors associated with NVI could be helpful in guiding risk stratification and screening for NVI in patients with KD to avoid missed injuries and limb loss. Historically, limb loss has been reported to occur in up to 15% of patients

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with a KD and in up to 86% of patients with vascular injury in whom intervention is delayed.[4,21] Previous studies have been limited by either small sample sizes in single-institution reviews or an inability to assess the mechanism in larger studies because administrative databases often do not record the mechanism of injury.

This study had 2 objectives. The first was to describe the association between the energy level of injury and NVI following KD without fracture using a large representative sample of trauma patients. We hypothesized that higher energy levels of injury would be associated with increased incidence of NVI. Secondarily, this study sought to investigate potential risk factors for NVI within the energy-level groups. We anticipated that, within the ultra-low-energy group, obesity would be a risk factor for NVI.

METHODS

The American College of Surgeons National Trauma Data Bank ACS NTDB is the largest aggregation of United States and Canadian traumatic injury data assembled, representing a convenience sample. The NTDB was queried from 2010 to 2012 to identify patients who sustained a knee dislocation using *International Classification of Diseases, Ninth Revision (ICD-9)* codes (836.50-54, 836.59, 836.60-64, and 836.69). Patients were excluded if a concomitant lower extremity fracture was present (see ICD-9 codes listed in **Appendix**, **Supplemental Digital Content 1**, http://links.lww.com/JOT/A441), as these represent potentially confounding injuries. Patients younger than 18 years of age and older than 64 years were excluded, as were patients with a Glasgow Coma Scale score of less than 13 because these patients may not have been able to adequately participate in a physical examination to allow for proper clinical screening of NVI.

Demographic and clinical variables were evaluated, including sex, age, race, obesity (defined as body mass index ≥30), smoking status, hypertension, and diabetes. Demographic and comorbid condition codes in the NTDB were used to define these variables. Specific injury diagnosis codes in the NTDB were used to assess concomitant nerve and vascular injuries. To characterize patients as having sustained a neurologic injury, *ICD-9* codes 355.3-4, 956.2-5, and 956.8-9 were used. To characterize patients as having a vascular injury, *ICD-9* codes 904.8 and 904.40-42 were used. Procedure codes were analyzed to assess what interventions were performed on these patients. Additional *ICD-9* procedure codes were used to identify patients who underwent closed reduction of the knee, arteriography, fasciotomy, application of external fixator, vessel repair, and nerve repair (see **Appendix**, **Supplemental Digital Content 2**, http://links.lww.com/JOT/A442).

Patients were stratified based on the energy level of injury. The injury mechanism for each patient was classified as either high-energy, low-energy, or ultra-low-energy (see **Appendix**, **Supplemental Digital Content 3**, http://links.lww.com/JOT/A443). Using descriptive statistics, demographic and clinical characteristics were summarized based on these energy level groups. Next, the incidence of NVI and the number of associated procedures performed were calculated for each energy-level group. Univariate analyses were performed using the χ^2 test. Covariates of interest were included in the development of multivariate logistic regression models to adjust for confounders. P values less than 0.05 were considered statistically significant. Odds ratios (ORs) and 95% confidence intervals were reported for covariates included in the multivariate logistic regression analysis.

This study received no external funding. Given the deidentified nature of the American College of Surgeons NTDB, it was exempt from approval by our institutional review board.

RESULTS

In total, 1324 patients with a KD and no lower extremity fracture were identified who met the inclusion criteria. Of patients sustaining an ultra-low-energy injury, 64% were females; of patients sustaining a high-energy injury, 76% of patients were males ($P < 0.001$, Table 1). High-energy–level injuries accounted for 65% of cases, low-energy 21%, and ultra-low energy 14%. Most patients who sustained knee dislocations were 18–34 years of age, comprising 42% of the ultra-low-energy group, 52% of the low-energy group, and 52% of the high-energy group ($P < 0.001$). There were significantly more patients with obesity in the ultra-low-energy group (27%) compared with the low-energy and high-energy groups (13% and 9%, respectively, $P < 0.001$).

Across all 1324 patients, the number of vascular and nerve injuries were 198 (15%) and 57 (4%), respectively. The rate of vascular injury varied across mechanisms of energy groups and was present in 21% of ultra-low-energy, 17% of low-energy, and 13% of high-energy mechanisms ($P = 0.01$). The rate of nerve injury also varied across energy-level groups and was present in 6% of ultra-low-energy, 7% of low-energy, and 3% of high-energy mechanisms ($P = 0.03$, Table 2). The rates of external fixation, arteriography, vessel repair, and nerve repair did not vary significantly across groups.

For high-energy injuries, males had a higher incidence of nerve and vascular injury than did females (OR 9.1 and 2.1, respectively, Table 3). Obesity was associated with both nerve and vascular injury in the ultra-low-energy group (OR 5.5 and 4.6, respectively) and with vascular injury in the low-energy group (OR 3.8). Smoking, hypertension, age, and diabetes were not strongly associated with NVI in any energy-level group.

On multivariate analysis adjusting for age, sex, smoking status, obesity, hypertension, and diabetes, among patients who sustained a lower energy knee dislocation, obesity was associated with nerve injury in the ultra-low-energy group (OR 4.9) and with vascular injury in the ultra-low and low-energy groups (OR 4.0 and 3.4, respectively, Table 4). Males had a higher risk of nerve injury, regardless of the mechanism of energy. Male sex was also associated with a higher risk of vascular injury in the high-energy group (OR 2.1). Neither smoking status, hypertension, nor diabetes were associated with an increased risk of vascular or nerve injury on multivariate analysis.

TABLE 1. Demographic and Clinical Characteristics by Injury Mechanism Energy of Adult Patients With Knee Dislocation, No Lower Extremity Fracture, and GCS Greater than or Equal to 13 in the ACS NTDB From 2010 to 2012

Characteristic	Ultra-low, N = 181, N (%)	Low, N = 275, N (%)	High, N = 868, N (%)	*P*
Sex*				
Female	116 (64)	103 (38)	212 (24)	<0.001
Male	65 (36)	171 (62)	656 (76)	
Age				
18–34	76 (42)	143 (52)	447 (52)	0.20
35–49	63 (35)	81 (29)	261 (30)	
50–64	42 (23)	51 (19)	160 (18)	
Race				
White-NH	80 (44)	153 (56)	539 (62)	<0.001
Black-NH	74 (41)	84 (31)	153 (18)	
Any Hispanic	16 (9)	26 (9)	109 (13)	
Other	11 (6)	12 (4)	67 (8)	
Obesity				
No	133 (73)	239 (87)	789 (91)	<0.001
Yes	48 (27)	36 (13)	79 (9)	
Smoking				
No	153 (85)	237 (86)	725 (84)	0.57
Yes	28 (15)	38 (14)	143 (16)	
Hypertension				
No	134 (74)	228 (83)	738 (85)	0.002
Yes	47 (26)	47 (17)	130 (15)	
Diabetes				
No	156 (86)	253 (92)	819 (94)	<0.001
Yes	25 (14)	22 (8)	49 (6)	

*One subject with sex not reported.
GCS, Glasgow Coma Scale.

DISCUSSION

This study found that NVI is more common following ultra-low and low-energy–level mechanisms of injury. In these lower energy knee dislocations, obesity was associated with increased risk of neurovascular injury.

The overall rate of vascular injury was found to be 15%. Although this may be similar to some previous studies in the literature, previously reported rates have varied significantly. Gray et al[22] reviewed 18 case series including a total of 934 knee dislocations and found an average rate of 26% for popliteal artery injury, ranging between 7% and 100% by study. The most recent of these, a case series of 138 dislocations by Stannard et al,[23] reported the lowest rate of 7%. Others report rates of KD–associated vascular injury ranging from 3% to 43%.[4,9,20,24–31] The heterogeneity of these historical results may be related to sample size limitations, as KD is itself an uncommon injury, with most of these studies tending toward smaller series of 60 cases or fewer. The injury severity described in these series may also be subject to selection bias toward more severe injuries. As well, the recognition of vascular injury may be subject to institution-specific protocols and biases. This study used a large-scale database for its sample, allowing for a large and geographically diverse data set, in an effort to overcome these obstacles. Of the 198 dislocations with associated vascular injuries, vessel repair was documented in 143; it is possible that for those patients who did not undergo repair, distal or collateral perfusion was sufficient enough, or the injury minor enough, to defer vascular intervention.

This study found an overall rate of nerve injury of 4%. This is lower than the average rate of 20% common peroneal nerve palsy found in a recent review by Keating,[6] in which rates of nerve palsy were reported ranging from 4.5% to 40%. Variable rates of nerve injury associated with knee dislocation have also been reported by others, ranging from 2.3% to 43%.[9,14,25,26,29–37] This wide range in incidence may reflect variation in screening and also definition of nerve injuries across studies. Nerve injury presents along a spectrum of severity; minor neurologic deficit in the absence of concern for loss of nerve continuity would likely represent a neuropraxia, which may not warrant further investigation or alterations in management beyond expectant observation and may or may not have led clinicians to officially diagnose a nerve injury. The possibility of some of these nerve injuries being, in truth, nonoperatively managed neuropraxis may also explain the relative infrequency of nerve repair procedures being attempted (7 repairs of 57 documented nerve injuries). Because of this study's reliance on the accurate and complete coding of relevant diagnoses, as well as the possibility of delayed or missed injuries, the overall true incidence of nerve injury may have however been underestimated. Given the above, the incidence of KD-related nerve injuries warrants further investigation.

Energy level was found to be significantly associated with variations in the incidence of neurovascular injury. Specifically, lower energy mechanisms were associated with a higher incidence of neurovascular injury. Some previous studies have attempted to stratify knee dislocations based on different mechanisms of injury; however, the association between mechanism energy level and neurovascular injury had not been explored.[6,17,19] This association of NVI with lower energy mechanisms reported above may in part reflect a selection bias, as lower energy KD patients may be more likely to present for evaluation electively because of experiencing a complication of the KD, whereas higher energy injuries are more likely to routinely present for evaluation. Therefore, a higher proportion of lower energy KD patients may be found to have NVI. This may also explain the slight predilection toward high-energy level injuries in our data set, which accounted for 65% of our sample. Low-energy and ultra-low-energy–level injuries accounted for 21% and 14% of knee dislocations, respectively. The aforementioned review by Keating[6] reports similar historical rates, but fewer high-energy level injuries. This also may be a reflection of the selection bias of our data set, which is based on trauma center data registries. It is also possible that lower energy mechanisms are associated with the given host factors that allow for less tolerance of transient neurovascular stretch about the dislocation, increasing the likelihood of NVI. These interesting and somewhat counter-intuitive findings warrant further investigation. However, it underscores the importance of remaining vigilant for neurovascular injury during the initial evaluation and screening of patients with suspected KD, even in the setting of minor inciting trauma.

TABLE 2. Incidence by Injury Mechanism Energy of Vascular or Neurologic Injury and Associated Procedures in Adult Patients With Knee Dislocation, No Lower Extremity Fracture, and GCS Greater than or Equal to 13 in the ACS NTDB From 2010 to 2012

Characteristic	Total, N = 1324, N (%)	Ultra-low, N = 181, N (%)	Low, N = 275, N (%)	High, N = 868, N (%)	*P*
Nerve injury	57 (4)	11 (6)	18 (7)	28 (3)	0.03
Vascular injury	198 (15)	38 (21)	48 (17)	112 (13)	0.01
Arteriography	201 (15)	25 (14)	48 (17)	128 (15)	0.47
External fixator	188 (14)	21 (11)	30 (11)	137 (16)	0.07
Nerve repair	7 (<1)	2 (1)	3 (1)	2 (<1)	0.12
Vessel repair	143 (11)	24 (13)	33 (12)	86 (10)	0.32
Fasciotomy	143 (11)	21 (12)	30 (11)	92 (11)	0.92
Closed reduction	509 (38)	96 (53)	119 (43)	294 (34)	<0.001

GCS, Glasgow Coma Scale.

Obesity was also found to be associated with an increased rate of NVI in lower energy knee dislocations. Previous studies have reported association between ultra-low-energy injuries overall with obesity.[16–19] Carr et al[16] describe ultra-low-velocity injuries most commonly resulting in anterior dislocation, which has been associated with higher risk of NVI because of rupture of the posterior capsule and subsequent stress transferred to the tethered neurovascular structures in the popliteal fossa. It has been proposed that, with increasing body mass index, the knee joint experiences more stress and is subsequently more prone to injury because of instability of the tibiofemoral joint.[19,38] The greater physical mass of the patient also increases the energy of the energies and stress associated with the injury, more so than the initial mechanism may suggest. The more substantial soft-tissue envelope around the knee has also been postulated to mask deformity,[16] potentially leading to delays in diagnosis and increased risk of more severe complications. These findings have been suggested by others, who also report on knee dislocations occurring in the patients with morbid obesity.[37,39] Of note, obesity had no significant association with NVI in high-energy mechanisms, indicating that, as the energy of injury mechanism increases, obesity becomes less predictive of concomitant NVI.

After multivariate analysis, male sex was associated with increased rates of associated nerve injury, regardless of whether the KD was a result of low-energy or high-energy mechanism.

TABLE 3. Univariate Associations Between Patient Characteristics and Neurovascular Injury by Mechanism Energy

	Nerve injury (OR, 95% CI*)			Vascular injury (OR, 95% CI*)		
Risk Factor	Ultra-low	Low	High	Ultra-low	Low	High
Sex						
Female	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Male	1.5 (0.4–5.2)	2.2 (0.7–6.9)	9.1 (1.2–67.1)	0.9 (0.4–1.9)	0.6 (0.3–1.1)	2.1 (1.2–3.7)
Age in years						
18–34	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
35–49	1.0 (0.2–3.7)	1.0 (0.4–2.7)	1.0 (0.4–2.4)	2.3 (1.0–5.2)	0.8 (0.4–1.6)	1.0 (0.6–1.5)
50–64	0.7 (0.1–3.8)	0.3 (0.0–1.9)	0.7 (0.2–2.3)	1.1 (0.4–3.0)	0.6 (0.3–1.6)	1.4 (0.8–2.3)
Race						
White-NH	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Black-NH	0.5 (0.1–2.2)	6.2 (1.9–19.8)	0.4 (0.1–1.6)	1.1 (0.5–2.4)	0.9 (0.4–1.8)	0.8 (0.5–1.4)
Any Hispanic	0.8 (0.1–7.3)	3.1 (0.5–17.8)	1.3 (0.5–3.6)	0.6 (0.1–2.8)	1.8 (0.7–4.7)	0.8 (0.4–1.6)
Obesity						
No	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Yes	5.5 (1.5–19.8)	1.4 (0.4–4.9)	0.8 (0.2–3.3)	4.6 (2.1–9.7)	3.8 (1.8–8.2)	1.2 (0.6–2.3)
Smoking						
No	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Yes	2.2 (0.5–8.8)	1.9 (0.6–6.0)	1.7 (0.7–4.1)	2.0 (0.8–4.9)	1.9 (0.8–4.1)	1.4 (0.8–2.3)
Hypertension						
No	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Yes	2.5 (0.7–8.8)	0.3 (0.0–2.1)	0.4 (0.1–1.8)	2.3 (1.1–4.8)	1.3 (0.6–2.9)	1.3 (0.8–2.2)
Diabetes						
No	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Yes	2.5 (0.6–10.2)	1.5 (0.3–6.9)	0.6 (0.1–4.6)	1.2 (0.5–3.3)	0.7 (0.2–2.6)	0.9 (0.4–2.3)

*ORs and 95% C.I. Derived From Logistic Regression Models Specific for Injury, Risk Factor, and Mechanism Energy.

TABLE 4. Multivariate Associations Between Patient Characteristics and Neurovascular Injury by Mechanism Energy

	Nerve injury (OR, 95% CI*)			Vascular injury (OR, 95% CI*)		
Risk Factor	**Ultra-low**	**Low**	**High**	**Ultra-low**	**Low**	**High**
Sex						
Female	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Male	2.6 (0.6–12.5)	3.8 (1.0–14.5)	7.3 (0.9–54.8)	1.2 (0.5–2.9)	0.6 (0.3–1.4)	2.1 (1.2–3.7)
Age in years						
18–34	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
35–49	0.7 (0.1–4.3)	1.4 (0.4–4.4)	1.1 (0.5–2.8)	2.8 (1.0–7.5)	0.7 (0.3–1.5)	0.9 (0.6–1.6)
50–64	0.3 (0.0–3.1)	0.3 (0.0–4.3)	1.0 (0.3–3.2)	1.3 (0.4–4.5)	0.5 (0.2–1.6)	1.3 (0.7–2.3)
Race						
White-NH	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Black-NH	0.4 (0.1–2.1)	6.4 (1.9–21.6)	0.4 (0.1–1.8)	1.2 (0.5–2.8)	0.7 (0.3–1.5)	0.9 (0.5–1.6)
Any Hispanic	1.4 (0.1–15.6)	2.3 (0.4–13.8)	1.3 (0.5–3.6)	0.6 (0.1–3.5)	2.2 (0.8–6.0)	0.9 (0.5–1.7)
Obesity						
No	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Yes	4.9 (1.0–24.0)	2.6 (0.5–12.9)	1.1 (0.2–5.0)	4.0 (1.6–9.7)	3.4 (1.4–8.3)	1.2 (0.6–2.4)
Smoking						
No	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Yes	1.1 (0.2–7.6)	2.4 (0.7–8.6)	1.6 (0.7–4.0)	1.7 (0.6–5.3)	1.8 (0.7–4.3)	1.3 (0.8–2.2)
Hypertension						
No	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Yes	3.1 (0.5–19.5)	0.2 (0.0–2.9)	0.5 (0.1–2.3)	1.5 (0.5–4.3)	1.5 (0.6–4.0)	1.2 (0.6–2.2)
Diabetes						
No	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Yes	1.5 (0.2–9.3)	4.0 (0.4–36.5)	1.2 (0.1–11.3)	0.7 (0.2–2.3)	0.7 (0.2–2.9)	0.7 (0.2–1.9)

*ORs and 95% CI derived from logistic regression models specific for injury and mechanism energy.

Males who sustained a high-energy KD also had higher incidence of vascular injury. Historic data have reported an increased incidence of knee dislocation overall in males compared with females of 2.5:1. This may be due to the traditionally held notion of knee dislocations as a high-energy injury and the classic association of males more commonly involved in higher energy trauma. However, more recent studies have recognized that KD may occur via lower energy mechanisms, especially, as noted above, in patients with obesity.[16] The increased incidence of NVI in males may be due to residual selection, with males tending toward higher mechanism energy than females, even within the same mechanism energy group.

Of note, neither age, smoking status, hypertension, nor diabetes were associated with nerve or vascular injury, regardless of the mechanism energy. However, it should be noted that patients older than 64 years of age were excluded from analysis. It is possible that older patients could have sustained a greater number of NVIs. The rates of 5 different procedures were identified: external fixator placement, arteriography, fasciotomy, vessel repair, and nerve repair. The incidence of none of these procedures differed significantly based on mechanism energy.

One of the strengths of this study was the use of the NTDB for collection of injury mechanism data. Previous studies on knee dislocations, including database studies, were not able to incorporate this information because such data were not reported in their data sets. This additionally allowed for analysis of a significantly larger sample population than previous studies. Second to this study, which includes 1324 cases of knee dislocation, the next largest study that examined the rate of neurovascular injury was by Werner et al[37] and included 215 cases. Because KD is a relatively infrequent diagnosis, most reports have small samples and combine low-energy and high-energy injuries in their analyses.[17] The use of large administrative databases in some recent studies have permitted for larger sample sizes; however, as an inherent limitation to their data set, these studies did not contain detailed information regarding the mechanism of injury and were therefore unable to investigate the mechanism of energy as a risk factor or modifying factor for NVI.[40,41]

There were some limitations to this study. To improve homogeneity among the study population, we excluded patients with a concomitant lower extremity fracture. Because of the constraints of the *ICD-9* coding system, the laterality of the lower extremity fracture was unknown, so patients with a fracture in the lower extremity contralateral to the knee dislocation were excluded. In addition, the NTDB is a convenience sample with an inherent selection bias toward more severe injuries because it includes patients presenting to participating trauma centers. It is possible that our study may have underreported the number of nerve injuries due to reporting methods. It is further possible that many nerve injuries were not reported because a diagnosis was missed in light of more significant injuries distracting the clinician. Moreover, the lower rate of 4% could be due to reporting omissions of a known nerve injury. Another limitation is the inability to know the laterality

of the neurovascular injury because previously mentioned *ICD-9* coding does not include this level of description. Bilateral knee dislocations, which are uncommon but have been reported in up to 4% of cases,[6] may likewise be misinterpreted by the data set. In addition, our sample set may have failed to capture dislocations that underwent spontaneous reduction before initial presentation, which as noted previously has been reported to occur in up to half of the cases. This may preclude accurate or timely diagnosis of the dislocation and is an unavoidable limitation. We report a rate of closed reduction of 38%, from which one may infer that these patients remained dislocated on presentation. The remainder may have reduced spontaneously or en route to the emergency department. As well, some may characterize only ligamentous injuries without frank dislocation (eg, combined anterior cruciate ligament/medial collateral ligament injuries) as knee dislocation equivalent entities and code them as such. Although this study did not purposefully include those patients, we were unable to distinguish them from true dislocations, given the diagnosis-reliant limitations of the data set. Furthermore, as with other database studies, reporting errors are possible.

In conclusion, knee dislocation is associated with neurovascular injury, regardless of the mechanism of energy, and a high degree of attentiveness is required during initial evaluation and management. This study suggests that obesity is an independent risk factor for NVI, in particular with lower energy dislocations. Because of this increased risk, such injuries should be treated as emergencies to minimize the risk of potentially limb-threatening complications.

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