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Common peroneal nerve palsy following total knee arthroplasty: prognostic factors and course of recovery.

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**Common Peroneal Nerve Palsy Following Total Knee Arthroplasty
Prognostic Factors and Course of Recovery**

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Abstract:

Common peroneal nerve palsy (CPNP) is a serious complication following total knee arthroplasty (TKA). There is little information regarding the clinical course and prognostic factors for recovery. Between January 2000 and December 2008, 44 patients (0.53%) developed CPNP following TKA and were matched to 100 control patients based on year of surgery, type of surgery and surgeon. Regression analysis was performed to identify prognostic factors for recovery. A significant difference was seen in CPNP patients who were on average younger (62.1 years) and had higher BMI (34.5 kg/m2) than those who did not have nerve palsy (67.5 years and 31.8 kg/m2, respectively). Only 37 patients with palsies could be followed, 32 (62.2%) had incomplete nerve palsy, twenty four (75%) of them fully recovered, while only 1 of patients with complete nerve palsy fully recovered. More severe initial injury was a negative prognostic factor for recovery of palsy (P b 0.03).

Keywords: Common peroneal nerve palsy; Total knee arthroplasty; prognosis; recovery

Introduction:

Common peroneal nerve palsy (CPNP) following total knee arthroplasty (TKA) is a relatively rare complication that can result in marked disability [1,2]. The common peroneal division of the sciatic nerve is the most frequently injured nerve because of its anatomical location, with a reported incidence ranging from 0.3% to 4% after TKA [3–6].

Although various etiological factors for CPNP exist, the cause of palsy may not be clearly identifiable in most cases [1,7]. Previous studies have identified risk factors for CPNP which include mechanical stretching of the nerve as a result of correction of valgus or flexion deformity during TKA, disruption of the blood supply to the nerve which may occur with prolonged tourniquet use, extensive blood loss and hypotension, and compression of the nerve due to hematoma formation or more frequently compression over the fibular head by an external agent such as the continuous passive motion (CPM) machine [1,2,5,6,8]. Additionally, patients with previous spinal pathology may be more sensitive to develop a nerve lesion at a distal location, due to any of the above mentioned mechanisms, described as the double crush phenomenon [8]. Postoperative epidural anesthesia, previous neuropathy and rheumatoid arthritis (RA) are also determined to be risk factors for CPNP in some studies [2,6,8,9].

Despite the presence of many studies evaluating the cause and incidence of CPNP, the natural course of the nerve palsy with regard to recovery is not well understood. This study was designed to elucidate the natural history of CPNP following TKA, specifically seeking to determine the time to recovery and the factors that influence the recovery of the nerve, in an attempt to identify modifiable factors that could enhance recovery.

Materials and Methods:

Between January 2000 and December 2008, 7405 consecutive primary and 898 revision TKAs were performed at our institution. After institutional review board approval, we retrospectively reviewed the TKA joint registry at our institution and identified CPNP in 44 patients (0.53%). There were 40 patients (90.9%) with nerve palsies after primary TKA and 4 patients (9.1%) with nerve palsies after revision TKA. Patients with CPNP were matched with 100 control patients without nerve palsy based on year of surgery, type of surgery (i.e. primary) and surgeon. The cohorts were then compared to identify potential risk factors for CPNP.

Demographics

Twenty five patients were female (56.8%) and 19 patients were male (43.2%). The mean age at the time of surgery was 62.1 years (range, 13.8–82.1 years). The average body mass index (BMI) was 34.5 kg/m2 (range, 13.8–59.7 kg/m2). The preoperative diagnosis included osteoarthritis in 34 patients (77.3%), osteonecrosis of the femoral condyle in 1 patient (2.3%), RA in 4 patients (9.1%), posttraumatic arthritis in 1 patient (2.3%), and revision surgery in 4 patients (9.1%)

Clinical and Radiographic Evaluation

All patients had completely normal motor (grade 5) and sensory nerve function preoperatively. Regional anesthesia was used in 39 patients (88.6%) and general anesthesia was used in 5 patients (11.4%). Indwelling epidural catheter was used for postoperative pain control in 38 patients (86.4%). Medial parapatellar approach was used in all patients. The average operative time was 138.9 min (range, 37.0–286.0 min), with a mode of 87 min. The average estimated blood loss was 108.8 mL (range, 50.0–300.0 mL).

Injuries were stratified as being complete or incomplete based on evaluation of the patient by a neurologist. Complete neurologic injury was defined as grade zero muscle strength with no motor function and loss of sensation in the dermatome distribution of the nerve; incomplete injuries demonstrated disruption of motor or sensory function but grade one or better muscle strength or presence of full or partial sensation along the dermatome distribution of the nerve [10,11].

We evaluated preoperative and postoperative standing anteroposterior and lateral radiographs of all patients. Anatomical alignment of knees was measured. Anatomical axis of 12° or more of valgus was considered as excessive valgus, between 6° and 12° of valgus was considered moderate valgus, and between 0° and 6° of valgus was considered as normal alignment. Any degree of varus in anatomical axis was considered as varus knee. We reviewed medical records of the patients including operative report, postoperative assessment notes and clinical follow-up to extract the relevant information for this study. Anesthesia records were used to determine type of anesthesia, operation time, and the time in which the tourniquet remained inflated.

Follow-Up

Patients were followed for a minimum of two years with a mean of 30.1 months (range, 2.1 to 9.8 years) or until full recovery of the nerve. For analysis of prognosis, only 37 (84.1%) patients who could be followed up were included even though all 44 patients were included for analysis of risk factors. Some patients were under a litigation process and could not be contacted, so were considered as lost to follow-up. Thirty seven (84.1%) of these patients were evaluated by a neurologist or surgeon at the time of diagnosis of palsy and on regular intervals thereafter. The mean duration of follow-up was 30.1 months. During the follow-up visits, each patient was

examined by their surgeon, and the extent of their neurologic recovery, functional status, and use of medications for neurologic symptoms were recorded.

The decision to perform magnetic resonance imaging (MRI), computed tomography (CT), or electromyography (EMG) was based on the clinical judgment of the attending surgeon and/or consulting neurologist. If the MRI was inconclusive, due to the effect of metal artifacts, ultrasound examination was performed to assess the course of the nerve for the presence of hematoma, suture strictures, or other external compressions.

The care protocol at our institution mandates that anyone with postoperative signs of CPNP be seen immediately and the constrictive dressing removed or loosened. The knee is also placed in flexion [1,5,12]. Epidural catheter is also discontinued and patients monitored closely. Patients with foot drop are placed in an ankle–foot orthotic.

Statistical Analysis

In order to evaluate risk factors for palsy, bivariate and multivariate logistic regression analysis (using 95% confidence intervals) were performed to assess age, BMI, history of previous knee surgery, history of diabetes mellitus (DM), RA, preoperative alignment and flexion contracture of the knee, and also procedure dependent factors including use of postoperative epidural catheters, use of CPM, and tourniquet time, as possible risk factors.

In order to identify prognostic factors for recovery, bivariate and multivariate logistic regression analyses were performed to assess gender, age, BMI, motor nerve involvement, time of onset, the extent of initial injury, DM and use of postoperative epidural catheter as possible prognostic factors. Patients with full recovery were compared with those who had partial recovery.

Categorical variables were described in frequencies indicated as percentages and were evaluated with Fisher's test. Non-categorical variables were assessed with t-test. Statistical significance was considered when P values were b 0.05 at a 95% confidence of interval. SPSS statistical software (Windows version 16.0, SPSS Inc. Chicago, IL.) was used for analysis.

Results:

Out of 44 patients with CPNP, 6 (13.6%) were diagnosed on the day of surgery, 8 patients (18.2%) on postoperative day (POD) 1, 19 patients (43.2%) on POD 2, 7 patients (15.9%) on POD 3, and 4 patients (9.1%) on POD 4 or later. The motor and sensory functions were both affected in 32 patients (72.7%), the motor function alone was affected in 10 patients (22.7%), and the sensory function was solely affected in 2 patients (4.5%). The nerve palsy was determined to be incomplete in 37 patients (84.1%) and complete in the remaining 7 patients (15.9%). In 26 patients (59.1%), neurology consultation was obtained. Based on the complete evaluation by neurologists or attending surgeons, cross sectional imaging (CT or MRI; 9 patients; 20.5%) and electromyography (8 patients; 18.2%) were obtained. The suspected etiology for the nerve palsy was compression in 6 patients which included direct compression by CPM machine in 5 (11.4%), and compression by hematoma in 1 patient (2.3%). The cause of nerve palsy was unknown in the remaining 38 patients (86.4%). Thirty-nine patients (88.6%) had regional anesthesia and 38 (97.4%) of those who received regional anesthesia had an epidural catheter placed for postoperative pain control. The epidural catheters were discontinued after diagnosis of nerve palsy.

Mean age of patients who had peroneal nerve palsy was 62.1 years, which was significantly lower than the control group with mean age of 67.5 years (P = 0.008; Table 1).

Mean BMI of patients with nerve palsy was also significantly higher at 34.5 kg/m2 compared to mean BMI of 31.8 kg/m2 in control group (P = 0.04). Of our 44 patients with peroneal nerve palsy, preoperative tibiofemoral angle was in excessive valgus (12° or more) in 6 patients (13.6%), moderate valgus (6°–12°) in 17 patients (38.6%), and varus in 21 patients (47.7%). Six patients (13.6%) had a flexion contracture of 20° or more. Although not statistically significant, the incidence of valgus tibiofemoral angle and flexion contracture was higher in the CPNP cohort compared to control patients group, (odds ratio = 1.94 and 2.72, respectively). A tourniquet, inflated to 250 mmHg was used in all patients. The mean tourniquet time was 73.9 min (range, 41–130 min), with a mode of 47 min in the CPNP cohort with only 2 patients (4.5%) having a total tourniquet time longer than 120 min. There was no difference in tourniquet time between the CPNP cohort and control patients. Four patients (9.1%) with CPNP had RA compared to 2 patients (4.5%) in the control group (P = 0.07). Similarly the incidence of DM was not different between the CPNP cohort (9/44) compared to control patients (11/100) (P = 0.18). Three patients (6.8%) in the CPNP cohort had a history of previous surgery, not including a prior arthroscopy or soft tissue reconstruction, which included high tibial osteotomy (1 patient), proximal tibia fracture fixation (1 patient), and distal femoral fracture fixation (1 patient) compared to only 1 patient who had high tibial osteotomy in the control group (P = 0.085).

The clinical course of 37 patients who could be followed up varied. Twenty-four of the 32 patients (75%) with incomplete palsy fully recovered (mean of 12.5 months, with a range from 6.7 to 27.7 months) while only 1 out of 5 patients (20%) with complete nerve palsies fully recovered (6.7 months), (Fig. 1).

Six months after onset of CPNP, 3/37 patients (8.1%) experienced complete neurologic recovery. Twelve months after onset of CPNP, 23/37 patients (62.2%) had maximal neurologic

recovery, with 14/37 (37.8%) having complete recovery. Two years after onset of CPNP, an additional 6/37 patients (16.2%) had maximal recovery, with 5/37 (13.5%) having complete recovery. Five out of the 37 patients (13.5%) required more than 2 years to have their maximal neurologic recovery with 3/37 (8.1%) having complete recovery.

The sole patient with follow-up that did not have motor involvement had a full recovery (100%), while out of the 36 remaining patients with motor involvement, 24 patients (66.7%) had full recovery. The average age of patients with full recovery was 62.5 years (range, 39.–80.6 years) while that of patients with partial recovery was 67.7 years (range, 40.7–82.1 years), (P = 0.266). The average BMI of patients with full recovery was 34.2 kg/m2 (range, 21.5–59.7 kg/m2) while that of patients with partial recovery was 37.7 kg/m2 (range, 25.3–49.6 kg/m2) (P = 0.557). Two patients out of the 25 patients with full recovery had DM, while 1/12 patients with partial recovery patients had DM. Twenty- one out of the 25 (84%) patients with full recovery had postoperative epidural catheter, while 11/12 (91.7%) patients with partial recovery had received postoperative epidural catheter (P = 1.0). One out of the 25 patients (4%) with full recovery had complete injury compared to four of 12 (33%) patients with partial recovery (Table 2). The bivariate and multivariate logistic regression analysis showed that only the state of initial injury was a significant factor influencing recovery. Patients with complete nerve injury had a lower chance of full recovery compared to patients with incomplete injury (P = 0.03; Table 3).

Discussion:

Total knee arthroplasty is one of the most commonly performed orthopaedic procedures with an expected increase over the coming years [13]. Common peroneal nerve palsy following TKA is a rare complication that has a poorly understood clinical course. In the present study, the

incidence of this injury was found to be 0.5%, which is consistent with the reported incidence of 0.3% to 4% [1–6]. The variability in the nerve palsy rate can likely be attributed to the different patient populations, indications for surgery, and vigor in diagnosis. Even though the reported incidence of CPNP is relatively low, there are a significant number of patients with subclinical palsies that go unrecognized. Our data show that the prevalence of clinically relevant CPNP may be similar to previous reports, but we realize that many patients may have had subtle nerve injuries that were not diagnosed.

In contrast to the tibial nerve, the peroneal nerve has few large funiculi with sparse connective tissue that restricts the elongation of the peroneal nerve. Therefore, the peroneal nerve is more easily predisposed to permanent neuronal damage [1,8]. During surgery, one can imagine that compression, traction, and ischemia may occur, resulting in nerve injuries. In fact, the reported cases of CPNP in the literature appear to be mostly related to numerous surgical and patient related risk factors such as preoperative deformity, RA, compression from hematomas, prolonged tourniquet inflation time, constrictive dressing, postoperative epidural analgesia, and previous neuropathy [2,6,14–18]. The stretch of the nerve and the surrounding soft tissues that occurs during correction of deformities such as excessive valgus and flexion contracture, is thought to result in compromised blood supply to the nerve and has been hypothesized as the cause of nerve palsy [19]. Severe damage has been shown to occur in the axon after 4% to 11% elongation of a human nerve and impaired microcirculation of a nerve can be seen after 8% elongation in the tibial nerve of rabbits [19,20]. It is thought that the stretching narrows both the extraneural and intraneural microvasculature and results in impaired blood flow [19,21]. However, not all studies including our own has shown preoperative valgus and flexion contracture to be significant risk factor for peroneal nerve palsy after TKA [6,15,22]. The use of

postoperative epidural catheter is shown in some studies to increase the risk of peroneal nerve palsy after TKA [2]. The anesthesia blocks pain perception, so the peroneal nerve might be physically compressed by bed rail or CPM machine. But, the relationship between postoperative epidural analgesia and peroneal nerve palsy was not supported in most studies [6,8]. Indwelling epidural catheter is used for postoperative pain control in all patients undergoing TKA at our institution. Thus, the reason for not being able to identify the use of epidural catheter as a risk for CPNP may relate to lack of adequate number of patients with epidural catheter in the entire cohort. Neuropathy, through double crush phenomenon, has been shown to be a risk factor of peroneal nerve palsy after total knee in some studies [2,8]. In a comprehensive study of 115 patients who were evaluated with electromyography, Upton et al confirmed that the susceptibility of peripheral nerves to injury is increased if there was a previous proximal lesion [16]. According to the literature DM is the most common cause of neuropathy. It causes a symmetrical, sensory, distal neuropathy. However, our study, as well as none of studies before, has shown DM to be a significant risk factor for peroneal nerve palsy following TKA [16]. In this study we found DM to be more common in palsy patients but no statistical significance was demonstrated (odds ratio = 1.8). However, most patients with mild to moderate diabetic neuropathy are asymptomatic and therefore likely unidentified. We did not have EMG and nerve conduction studies or a comprehensive sensory examination of all patients before operation, so the role of diabetic neuropathy as risk factor for CPNP is inconclusive in this study. Rheumatoid arthritis has been shown as a significant risk factor in some studies [6,9]. Patients with RA usually have knee flexion contracture and valgus, therefore the nerve palsy could be attributed to these risk factors. Knutson et al implied that the effect of RA on the development of peroneal nerve palsy was via indirect risk factors already discussed—the preoperative valgus deformity

and flexion contracture [9]. On the contrary, Schinsky et al found RA itself and not preoperative valgus deformity and flexion contracture to be the risk factor for peroneal nerve palsy [6]. Neuropathies that are associated with RA may also be a predisposing factor. In this study RA was more common in patients who developed CPNP, and had a statistical association to its occurrence (odds ratio = 3.2; P N 0.05). Most surgeons prefer to perform TKA using pneumatic tourniquets. Generally, up to 120 min of tourniquet inflation is considered safe [15,23]. Association of palsy with use of tourniquet after lower extremity operations has been shown [24–26]. The mechanism appears to be caused by both ischemia and mechanical deformation [25]. Although Horlocker et al, in a report of 8 nerve palsies in 361 TKA patients, found tourniquet time longer than 120 min a significant risk factor [8], most studies done to determine the risk factors for peroneal nerve palsy after TKA, did not find tourniquet time and pressure a significant risk factor.Wedid not find tourniquet time a significant risk factor for development of peroneal nerve palsy. Body mass index has not been shown in any studies to be a significant factor in peroneal nerve palsy after TKA. However, in this study we found BMI to be significantly higher in patients who had CPNP after TKA. This could be explained by more difficult exposure during operation in these patients which increases the risk of stretching thus injuring the nerve. Also, patients with higher BMI tend to put their hip in more external rotation while lying down. It might cause compression of peroneal nerve at the level of the fibular head either in bed or with the use of a CPM machine.

The current study was designed to evaluate the clinical course of patients with CPNP and identify prognostic factors for recovery. We found that CPNP resolves completely in two-thirds of patients by one year and recovery appears to continue up to two years. The main finding of our study was that the initial intensity of palsy (complete versus partial) was the most important

prognostic factor. This is in agreement with two previous studies that reported similar finding [1,2]. The proportion of patients who recovered in our study is slightly higher than reported rates in previous studies [1,2,5,6,15,27]. Asp and Rand found that, at a mean follow-up of 5.1 years, CPNP had fully recovered in 50% of patients [1]. Similarly in a study by Idusuyi et al, only 50% of patients experienced recovery of the nerve at a mean follow up of 3.9 years [2]. The reason for the higher rate of recovery of the nerve in our study may be multifactorial. The most important reason may be the fact that majority of the patients in our study were evaluated and monitored by a neurology team. The close monitoring and evaluation ensured that the cause for CPNP could be identified and reversed, if possible. For example, patients with hematoma formation underwent early evacuation of the hematoma that led to resolution of symptoms. The evaluation included the use of MRI, CT, or EMG based on the clinical judgment of the attending surgeon and/or consulting neurologist for instance in order to asses nerve entrapments in-between sutures. The neurology team also monitored recovery of the nerve closely with EMG, and placed patients on neurotropic agents such as gabapentin. A previous study has recommended exploration of the nerve in any patient not showing either clinical resolution or EMG improvement after 3 months [28]. We are of the opinion that unless a reversible cause for CPNP can be identified such as a hematoma, exploration of the nerve does not guarantee partial or complete recovery. Hence, with the exception of one patient with hematoma formation, none of the patients in our study underwent exploration of peroneal nerve. We do, however, have an aggressive protocol in place, which includes placement of the affected foot in an ankle–foot orthotic, and administer physical therapy involving range of motion exercises as well as electrical stimulation of the affected muscles to minimize disuse atrophy.

This study suffers some limitations: first, this is a single institutional study and the findings may not be generalizable to all patients with CPNP. We perform TKA with the patient in the supine position under regional anesthesia, using a medial parapatellar approach, and cemented components. Thus, some etiological and possibly prognostic factors may not have been detected in our "homogenous" patient population. Second, the retrospective nature of the study may introduce bias as a result of possible variation in recording clinical course and subsequent data collection. Finally, despite having a relatively large cohort of patients with CPNP, some factors influencing recovery may not have been identified using multivariate analysis due to small number of failures.

Despite the aforementioned limitations, the study included a cohort of patients who were followed closely by neurologists and orthopedic surgeons documenting the progress and recovery of the patients on regular intervals. This study reaffirms age and identifies BMI as risk factors for CPNP. Most importantly, we report that CPNP completely resolves in over three-fourths of patients with partial palsy and only one-fifth of patients with complete palsy. Degree of palsy (independent versus complete) was the sole independent predictor of recovery. Patients with higher BMI as well as patients with age greater than 65 years should be advised of a higher risk for developing CPNP.

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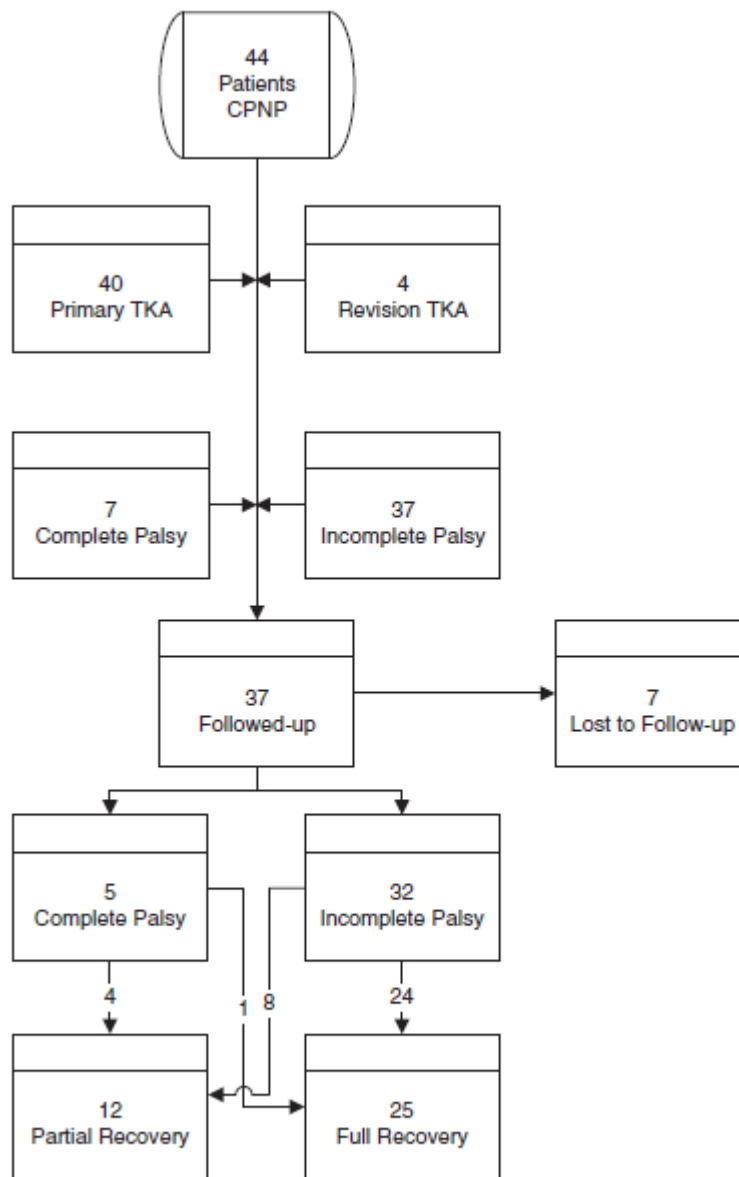
Fig. 1. CPNP cohort description throughout the study. Common peroneal nerve palsy = CPNP; Total knee arthroplasty = TKA.

Table 1
Risk Factors for CPNP.

Variables	CPNP Patients (N = 44)	Control Patients (N = 100)	P-Value
Age (average ± SD)	62.1 ± 13.1	67.5 ± 10.4	0.0008
BMI (kg/m^2 ± SD)	34.5 ± 8.8	31.8 ± 6.6	0.004
Valgus deformity (N)	6	7	0.15
Flexion contracture (N)	6	5	0.09
Previous knee surgery (N)	3	1	0.21
Duration of Tourniquet (min. ± SD)	73.9 ± 22.5	71.8 ± 26.3	0.54
RA (N)	4	2	0.07
DM (N)	9	11	0.18
Postoperative epidural catheter (N)	38	92	0.41

Common peroneal nerve palsy = CPNP; Number = N; Standard deviation = SD; Body mass index = BMI; Kilogram = kg; meter = m; Minutes = min; Rheumatoid arthritis = RA; Diabetes mellitus = DM.

Table 2
Results of the Initial State of Injury in Patients With Follow-Up.

Recovery	Full	Partial
Complete injury (N)	1	4
Incomplete injury (N)	24	8

Number = N.

Table 3
Multivariate Logistic Regression Analysis for Prognosis of Recovery.

	P-Value	Odds Ratio	95% Confidence Interval	
			Lower	Upper
Initial state of injury	0.03021	1.107988	1.01535166	1.20062434