INTRODUCTION:
Quadriceps weakness is a common consequence of knee joint injury and disease. Arthrogenic muscle inhibition (AMI), a neurological decline in muscle activation, leads to this quadriceps weakness and hinders rehabilitation by preventing gains in strength resulting in persistent functional and biomechanical deficiencies, the possibility for re-injury, and potentially placing patients at risk for chronic degenerative joint conditions. In light of this information, it seems critical to gain an understanding of the factors that contribute to AMI so that efforts to target and remove it can be implemented.

Joint injury is accompanied by numerous sequelae (i.e., pain, swelling, tissue damage, inflammation) making it difficult to ascertain, which ultimately leads to neurological muscle dysfunction. While effusion can result in AMI, the effects of pain are less understood despite the fact that most clinicians attribute AMI to pain. Employing techniques that introduce knee pain without accompanying injury may aid us in grasping its role in eliciting AMI. Therefore, the primary purpose of this study was to determine if knee pain alone would result in declines in quadriceps activation and strength. A secondary purpose was to determine if the magnitude of AMI would be greater when pain was provided along with a knee joint effusion. We hypothesized that pain alone would result in quadriceps inhibition and that the magnitude of inhibition would be greatest when effusion and pain were present simultaneously.

METHODS:
Fourteen healthy subjects (8 males and 6 females; aged 23.6 ± 4.8 years) had quadriceps strength and activation quantified under four randomized conditions: 1) normal knee, 2) effused knee, 3) painful knee, and 4) effused and painful knee. Prior to testing, approval for the research was gained through the Medical School Institutional Review Board of the University of Michigan and written informed consent for all subjects was obtained. Injections of 0.3 mL of 5% hypertonic saline were placed into the infrapatellar fat pad and utilized to induce pain, while 60 mL of hypotonic saline were placed into the knee joint capsule and used to induce effusion. During the effusion and pain condition both types of saline were injected. Quadriceps strength was assessed during the performance of a knee extension maximal voluntary isometric contraction (MVIC) with the knee flexed to 90°. Three contractions were completed with two minutes of rest provided between each trial. The average peak torque determined from the three MVIC trials was utilized to represent quadriceps strength. Quadriceps activation was determined using the burst superimposition technique, whereby a train of supramaximal electrical stimuli (100 ms train, 600 µs pulse duration, 100 pulses/second, 130 V) were delivered on top of the maximal isometric knee extension contractions. To quantify quadriceps activation, a measure of AMI, the central activation ratio (CAR) was computed by dividing the peak torque generated prior to the delivery of the electrical stimulus by the peak torque generated as result of the electrical stimulus. The CAR was calculated for each of the three trials and averaged for analysis. A 1 x 4 repeated measures ANOVA was utilized to compare quadriceps strength and activation across the four conditions. Bonferroni multiple comparison procedures were employed to make post hoc comparisons and an alpha level of P ≤ 0.05 was deemed significant for all tests.

RESULTS:
Significant differences between conditions were noted for both quadriceps activation (P = 0.001; Figure 1) and strength (P = 0.001; Figure 2.) The CAR was highest under the normal knee condition and differed from the three experimental knee conditions (effusion: P = 0.01; pain: P = 0.03; effusion + pain: P = 0.02). Similarly, the quadriceps MVIC differed from the other three knee conditions (effusion: P = 0.04; pain: P = 0.01; effusion + pain: P = 0.009), being greatest under the normal knee condition. No significant differences were noted between the three experimental knee conditions for either quadriceps strength or activation (P > 0.05).

DISCUSSION:
Our findings showed that knee joint pain produced quadriceps arthrogenic muscle inhibition. Considering that pain accompanies numerous knee joint injuries and conditions, the prevalence of AMI with joint trauma is likely high. The magnitude of inhibition resulting from pain was not large (5.7%), which agreed with data suggesting that pain contributes to a small, but significant portion of the AMI present after total knee arthroplasty. Notably, the relation between pain and AMI may be mediated by the severity of the pain experienced, but that connection requires future study.

Contrary to our hypothesis, the interaction of pain and effusion did not result in statistically different magnitudes of AMI (Pain only 5.7% vs. Pain + Effusion: 10%) suggesting that there was not an additive effect of the two stimuli. As injury severity influences the degree of AMI, we expected more noxious stimuli provided to the knee would increase quadriceps AMI. Although the pain and effusion were artificially-induced and may account for lack of significance, the degree of pain and the size of the effusion were of clinically-relevant quantities (60 mL joint effusion and 5 out of 10 on a visual analog scale), and thus we suggest our findings have meaning for clinical populations suffering from knee injury. Based on our results, we concluded that clinicians whose goal is to reduce AMI and improve muscle strength should employ interventions that target the removal of both pain and effusion.

REFERENCES: