INTRODUCTION: Compartment syndrome occurs when increased intramuscular pressure (IMP) causes decreased vascular perfusion of a muscle compartment to a point no longer sufficient to maintain viability of the muscle and nervous tissue contained within the compartment. This is most frequently a result of bone fracture but can also be caused by extreme exertion, prolonged compression or peripheral vascular disease. If not corrected by emergency fasciotomy, Volkmann’s contracture ensues with muscle and nerve necrosis resulting in a replacement of muscle by fibrotic scar tissue and permanent damage to nerves traveling through the compartment.

Clinical signs pointing to compartment syndrome are myoneural pain with passive stretch, paresthesia, paresis, pink skin color, and presence of a distal pulse. However, the sensitivity of these signs for diagnosing compartment syndrome is reported to be as low as 13%. Critical early diagnosis of compartment syndrome is the measurement of IMP with a pressure level higher than normal (>30 mm Hg) with clinical symptoms or ≤30 to 40 mmHg below mean arterial pressure. Currently, IMP is invasively measured to evaluate potential compartment syndromes with the insertion of a slit catheter or similar pressure transducing apparatus directly into the muscle compartment(s) at risk. This method is accurate and reproducible, but invasive, painful and an infection risk. The development of an accurate, reproducible, and noninvasive technique for the measurement of IMP in suspected compartment syndromes would ease diagnosis for physicians and improve comfort for patients.

METHODS: COMPARTMENT SYNDROME MODEL: We utilized a thigh tourniquet to impede venous return and induce a transient increase in IMP. Normal volunteers (n=8) lay supine on a standard hospital table. A deflated tourniquet was applied to the left mid-thigh and the left leg was prepared in a sterile fashion. A small region was locally anesthetized with Lidocaine and slit catheters were inserted into the anterior compartment of the leg at the anesthesia site for direct IMP measurement. First, a baseline IMP recording was taken for at least two minutes. Then, tourniquet pressure was applied in a stepwise fashion from 40-100 mmHg in 20 mmHg increments with four minutes at each step. The tourniquet was then released and IMP recording continued for an additional two minutes.

PPLL TECHNOLOGY: The pulse phase-locked loop (PPLL) device is a low-power ultrasound instrument designed to detect and continuously monitor very small displacements (on the order of microns) between the ultrasound transducer (on the skin surface) and any sub-dermal tissue that is capable of reflecting the ultrasound pulse. The device transmits a 2 MHz tone burst acoustic pulse through a small (1 cm) ultrasound transducer coupled to the skin surface that is propagated through the skin and muscle tissue and reflects off underlying tissues back to the same transducer, which receives the signal. The PPLL is able to “lock on” to a specific reflection at a variable and specified depth within the tissue as measured by the acoustic path length. The PPLL compares the phase of the transmitted pulse with the received ultrasonic pulse from the lock point and uses a feed back loop to maintain a constant 90° phase difference by altering the frequency of each successive emitted pulse. By following the frequency shift in PPLL output, micrometer level changes in path length between the transducer and the lock point can be measured continuously. For this study, we chose the interosseous membrane between the tibia and fibula as our lock point so that the PPLL frequency shift would give us a continuous measurement of changes in diameter of the anterior muscle compartment. Each arterial pulsation that courses through a muscle compartment causes a transient expansion of this dimension as the muscle fascia accommodates and rebounds from the cardiac pressure pulse, generating a periodic and characteristic fascial displacement waveform that the PPLL is able to record in real time.

NONINVASIVE MEASUREMENT: For PPLL measurements, healthy volunteers (noninvasive group, n=6) lay supine on a standard hospital bed. Standard ultrasound gel was applied to the PPLL transducer and it was placed on the skin over the mid-anterior compartment. The ultrasound reflection spectrum was viewed on an oscilloscope and the PPLL lock point was set to the interosseous membrane reflection. The presence of a periodic fascial displacement waveform corresponding to arterial pulsation was confirmed. This waveform was recorded continuously and thigh tourniquet venous occlusion was performed as described above.

DATA ANALYSIS: The PPLL data were analyzed for amplitude and shape of the fascial displacement waveform. To allow mathematical characterization of fascial displacement waveform shape, Fast Fourier Transformation (FFT) was performed on 10-second waveform averages and the ratio of amplitudes of the fundamental harmonic (FH) to the second harmonic (H2) was obtained for each time point (FH/H2).

RESULTS: In the invasive group, stepwise increases in thigh cuff pressure caused a significant increase in invasively measured IMP, rising from a normal mean baseline pressure of 12.1 mmHg (SE=1.5) to a mean pressure of 27.4 mmHg (SE=2.4) at 100 mmHg of thigh cuff pressure (n=8, p<0.001). IMP quickly dropped after release of thigh cuff pressure, returning to within 3 mmHg of the baseline measurement within two minutes in all subjects. In the noninvasive group, PPLL FH/H2 was similarly raised by thigh cuff pressure from a normal mean baseline value of 1.12 (SE=0.07) to a mean FH/H2 of 1.85 (SE=0.18) at 100 mmHg of thigh cuff pressure (n=6, p=0.0193). FH/H2 in the noninvasive group returned to within 0.05 of baseline within two minutes following release of thigh cuff pressure. These data are graphically represented below:

DISCUSSION: Our study documents the viability of a novel technique for transiently increasing IMP in the lower leg of normal human subjects using a proximal thigh tourniquet. This provides a safe and easily performed mechanism for evaluating the efficacy of non-invasive measurement techniques for diagnosing increased IMP. We also show that the PPLL device is able to detect fascial displacement waveforms in the anterior compartment of the leg that correspond to arterial pulsation. These waveforms are characteristic in shape in the normal leg, and consistently change under conditions of elevated IMP with the waveforms becoming progressively less complex as IMP increases. FFT analysis of these waveforms yields a unitless number, the FH/H2, which is able to detect between normal and elevated IMP. Implementation of the PPLL device would revolutionize the manner by which to test IMP for diagnosing compartment syndromes. This test would be noninvasive, painless, and could be used continuously to provide more accurate data than individual samplings of pressure taken at random times. We are currently in the process of a clinical trial comparing IMP and FH/H2 measurements in patients who present to be evaluated for potential compartment syndromes. This will allow us to calculate a specific correlation between IMP and FH/H2 and give us the sensitivity and specificity of PPLL for detecting compartment syndromes in a clinical setting.