Charcot neuroarthropathy (CN) is one of the many comorbidities that may develop secondary to diabetes. It is characterized by an evolutionarily progressive bone deformity and joint instability to subsequent chronic ulcerations. The prevalence and incidence of CN secondary to diabetic neuropathy ranges between 1% - 20%, respectively. One of the biggest challenges are non-healing ulcerations due to underlying bony deformity coupled with vascular insufficiency. The peroneus brevis muscle flap has previously shown sufficient results for treating large and small wound defects of the foot and ankle. Although the literature is vast, there is limited research on its efficacy with diabetic CN patients. The purpose of this paper is to present a case study using a rotational peroneus brevis flap for a chronic lateral ankle wound resulting from charcot neuroarthropathy.

Case Study
A 68 year old diabetic female with HTN, venous stasis and chronic pain was referred with a 2 month history of a right lateral ankle wound. Patient stated the wound was caused from chronic rubbing of her ankle brace. Patient had a History of poorly controlled diabetes with associated Charcot neuroarthropathy. Patient presented with a large lateral ankle wound with 3mm undermined, positive probe to bone, and superficial drainage. Patient underwent an MRI which highlighted the severe ankle deformity and instability of the ankle joint.

Literature Review
Charcot neuroarthropathy is a progressively destructive, severely debilitating disorder leading to structural damage, bone resorption and hypertrophic repair. The exact mechanism of neuroarthropathy remains uncertain, but the common assumption is that lack of proprioception and sensation leads to joint instability, ligamentous weakness, and subsequent deformity.4 Any disorder resulting in peripheral neuropathy including alcoholism, IV drug use, renal failure, congestive heart failure, malignancy, leprosy, spina bifida, and multiple sclerosis can cause charcot neuroarthropathy; however diabetes is the most common cause.5-7 A paper by Bienert et al. identified the most common trigger events as acute trauma, surgical procedures, infections, and vascular injuries. The most common areas affected are tarsal and tarsometatarsal joints which usually correlate to an increase in plantar pressures from an equinus contracture.7 Other common foot and ankle deformities subsequent to charcot neuroarthropathy include midfoot collapse leading to a ‘rocker bottom foot,’ ulcerations, significant dislocations of the midfoot and hindfoot, and instability of the ankle joint.8 Literature has shown muscle flaps are an excellent procedure for coverage of non-healing wounds. Muscle flaps have even been shown to effectively treat soft tissue defects with underlying osteomyelitis due to its vascularity and ability to deliver systemic antibiotics.6 The peroneus brevis muscle flap has been described for coverage of foot and ankle wounds with a variety of sizes.4 However, there is little research documenting their use for wound defects caused by the structural changes from charcot neuroarthropathy. Reports have shown this particular muscle flap provides reliability for soft tissue coverage as well as limited donor site morbidity and limited functional deficit.4 The peroneus brevis muscle flap can be transposed in either the proximal or distal fashion due to vascular supply. Perforators from the anterior tibial artery supply the proximally based flap while perforators from the peroneal artery supply the distally based flap.4,9,11

Ceran and colleagues documented successful results utilizing a proximally based peroneus brevis muscle flap for coverage of exposed hardware on the lateral malleolus.9 Both studies demonstrate the versatility of this distally based peroneus brevis muscle flap for coverage of heel, achilles, medial and lateral ankle wounds with successful and reliable results.9 There is currently no gold standard of treatment for CN; however, the ultimate goals are to avoid amputations and produce a stable, brace able, weight bearing plantigrade lower extremity that is free of infection with intact soft tissue.10 The common treatment options on treating the acute stage remains skin and soft tissue off loading.10 Alternative surgical treatment options include excisional debridement with internal fixation, external fixation anesthesia, or a combination.4,11 External fixation is the treatment of choice in patients with active infections, poor soft tissue coverage, history of DVT, and those noncompliant with non-weight bearing status.10

The patient was receiving IV antibiotics with routine hyperbaric oxygen therapy. Due to patient’s severe charcot deformity resulting in ankle varus and exposed fibula, further surgery was warranted. Patient received serial x-rays, noninvasive vascular studies and CT angiography. Patient underwent surgery including resection of distal fibula, achilles tenotomy, ankle fusion with application of multiple external fixator, peroneus brevis rotational muscle flap, application of hylon wound/round graft, and application of wound VAC. The patient continued with wound HBO therapy while in the postoperative period.

Conclusions
This case demonstrates a successful outcome for treating a non-healing wound due to chronic structural changes from charcot neuroarthropathy. The main goal was achieved for this patient who now has a braceable functional foot with an improvement of a chronic non-healing wound. However, it is imperative to stress continued education along with proper off loading during follow up visits to continue the correction of the deformity. This case can lead to further deformity and ulcerations. Further studies need to done to determine the long-term effectiveness of muscle flaps in diabetic charcot neuroarthropathic patients.

Analysis and Discussion
Wound closure while providing stability for complicated diabetic neuropathic patients remains a challenge. The balance lies between providing adequate reduction in the deformity along with sustainable wound coverage. To our knowledge, no other case studies have been presented utilizing a peroneus brevis muscle flap for closure in conjunction with an ankle fusion utilizing external fixation treating diabetic charcot neuroarthropathy. It should be noted that Capobianco et al. demonstrated positive functional outcomes utilizing a peroneus brevis muscle flap in conjunction with a staged medial column arthrodesis in a chronically infected hip in a patient with a chronic Charcot foot.6 There are many different modalities and products that may be utilized for off load. Options range from vacuum-assisted closure to negative pressure wound therapy to hyperbaric oxygen therapy. Despite the high complication rate of infection in diabetics, the peroneus brevis rotational flap, in this case provided excellent soft tissue coverage and wound closure to the lateral ankle defect.

Stability along with reduction of the deformity are crucial for the wound closure to be sustainable. There are numerous options that provide a more plantigrade foot including, exostectomy and arthrodesis with both internal and external fixation.12 Lowery et al. demonstrated that if instability is present along with recurrent ulcerations or pain, arthrodesis may provide a viable option despite a high rate of incomplete bony union. Our case demonstrates that arthrodesis for severe ankle deformity provided sufficient alignment and reduced the underlying bony burden to the lateral ankle.

References
3. Lowery N, Woods J, Armstrong D, Pfeifer A, Madan B. A paper based peroneus brevis muscle flap for coverage of heel, achilles exposed fibula, further surgery was warranted. Patient received serial x-rays, noninvasive vascular studies and CT angiography. Patient underwent surgery including resection of distal fibula, achilles tenotomy, ankle fusion with application of multiple external fixator, peroneus brevis rotational muscle flap, application of hylon wound/round graft, and application of wound VAC. The patient continued with wound HBO therapy while in the postoperative period.

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Figure 1
A. Interspace examination of ankle joint. B. Interspace extending to clearly penetrate bone. C. Internal iliac artery and iliac vein D. Demonstrating flap vessels. 
Figure 2
A. B. Mortise and lateral side of preoperative ankle. C. D. 2 week post operative 6 days. 
Figure 3
A. Timeline demonstrating initial surgery. B. Talar incision technique and steps. C. External fixator removed of 12 weeks. 
Figure 4
A. Poster operative dressing change B. External fixator removed of 12 week.