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Reducing the Hypertensive Effects of the Prolonged Surgical Tourniquet Using a Dual-Cuff Strategy: A Prospective Randomized Controlled Trial

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ABSTRACT

We evaluated whether moving the "line of crush" from thigh to the calf before onset of tourniquet-mediated hypertension would prevent or diminish it. We also evaluated any change in pain or functional outcome. Twenty adult patients were recruited and randomly assigned to either control or intervention groups. Inclusion criteria: any willing participant >18 years old with foot and/or ankle pathology requiring an operation lasting >90 minutes. Exclusion criteria included contraindication to general anesthesia, peripheral neuropathy affecting lower limbs of any etiology, or chronic pain requiring regular opiate analgesia. The intervention group received a thigh tourniquet for 60 minutes, after which a calf tourniquet was inflated and the thigh tourniquet was deflated. The control group received only a thigh tourniquet throughout surgery. At 90 minutes, the control group had mean arterial pressure of 86.8 mmHg, compared with the intervention group at 76.3 mmHg ($p \leq .014$). At end of surgery, the difference had increased further (control 98.1 mmHg, intervention 78.3 mmHg ($p \leq .001$). Moving the line of crush during limb tourniquet application prevents development of the hypertensive response. For cases in which a prolonged tourniquet application is required, a dual-tourniquet technique will prevent intraoperative hypertension and may influence long-term pain and function.

Foot and ankle surgeons often request the application of a lower-limb tourniquet to create a bloodless field during surgery. However, there is a limit to how long a tourniquet can be applied. Although there is no absolute consensus, recommended practice for the lower limb is to keep the tourniquet time <2 hours because of the risk of ischemia and reperfusion injury (1–4). Prolonged tourniquet inflation is associated with macro- and microscopic pathological changes in muscle, nerve, and other tissue. In neural tissue, sustained pressure results in reversible neural conduction block, affecting both motor and sensory nerves (5). Sustained pressure on muscle results in deprivation of oxygen reserves and subsequent anaerobic metabolism and acidosis. In as little as 60 minutes of tourniquet application, ischemic damage occurs to muscle (6).

"Tourniquet pain" and "tourniquet-induced hypertension" are terms applied to phenomena that are exhibited by patients during sustained tourniquet application. Awake patients with no anesthesia report discomfort and a dull ache in the whole of the limb that worsens with time, typically becoming unbearable at ~20 minutes. Patients under general anesthesia do not normally exhibit physiological responses to sustained tourniquet application until 60 minutes, from which point there is a progressive elevation in blood pressure. In patients who are breathing spontaneously, there may be an accompanying elevation in minute ventilation. The hypertension appears to be linear over time and, from ~90 minutes, can require attempts to control or reduce it. There may be associated diaphoresis and pallor. Unfortunately, the blood pressure elevation is unresponsive to many drugs, including potent opioids (7–11). Aside from other concerns regarding tissue ischemia injury, these physiological changes limit the duration that a tourniquet can be applied, even in the presence of general anesthesia. The exact mechanism of tourniquet-induced hypertension is unknown, and certain occurrences are difficult to explain.

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Regardless of the exact mechanism of action by which the hypertension is generated, we theorize that the "line of crush" (tissue compressed by the inflated tourniquet) is the key trigger. If this line is moved before the onset of the hypertensive response, the hypertensive response could be prevented. The purpose of this study is to see whether moving the location of the tourniquet to a more distal location on the leg intraoperatively can reduce the mean arterial pressure (MAP) and subsequently the hypertensive response.

Patients and Methods

This study is a prospective randomized controlled trial, with a parallel group design. Full UK Research and Ethics Committee approval was obtained before starting the study.

The participants were recruited from the foot and ankle clinic of a trauma and orthopaedics department in a medium-sized district general hospital by a senior foot and ankle consultant surgeon (C.P.), who either performed or directly supervised all surgical procedures carried out in the trial. A total of 44 potential participants were seen during active recruitment. The study recruited between August 15, 2015, and April 22, 2016. Twenty-four patients were excluded based on criteria below, primarily through a requirement or intention for spinal anesthesia. Fig. 1 shows the Consolidated Standards of Reporting Trials study flow diagram.

Inclusion criteria were

- Diagnosed with foot and/or ankle pathology that requires an operative intervention lasting >90 minutes (e.g. hind foot fusion, Lapidus and Akin osteotomies, ligamentous reconstructive surgery);
- Age >18 years;
- Willing and able to give informed consent for participation in the study.

Participants were excluded if any of the following conditions applied:

- Contraindication to general anesthesia;
- Diabetic neuropathy affecting the lower limbs;
- Peripheral neuropathy affecting lower limbs of any etiology;
- Persistent pain of >3 months' duration requiring the use of regular (2×/day) opioid analgesia;
- Revision surgery;
- Lacking capacity to give consent.

Patients with comorbidities such as sickle cell disease, peripheral vascular disease, congestive heart failure or poor cardiac reserve, Raynaud's disease or other vasculitides, deep vein thrombosis, or thrombophlebitis do not meet our day surgery unit admission criteria, and therefore were not eligible for the study. This ensured that both groups comprised low-anesthetic-risk patients with no contraindications to receiving general anesthesia or lower-extremity regional block.

After informed consent, the participants were randomized using sealed envelopes (prepared by an independent team in a separate location) and assigned to control (single tourniquet) or intervention (double tourniquet) groups. The patients were blinded to this process, but the assessors were not. All patients received a standardized general anesthetic, which included a regional anesthetic technique of either a popliteal fossa block or an ankle block (Table) at the discretion of the anesthetist and according to institution guidelines. Induction was achieved with 10 to 15 μg/kg fentanyl and 1 to 2 mg/kg propofol. If intubation was to be achieved, atracurium 0.5 mg/kg was used. Maintenance anesthesia was achieved using ≥1 minimum alveolar concentration sevoflurane in oxygen/air mixture. Additional analgesia after tourniquet inflation consisted of paracetamol 1 g intravenously (i.v.), ketorolac 15 mg i.v., and aliquots of fentanyl, at the discretion of the anesthetist and if no contraindications were present. Ondansetron

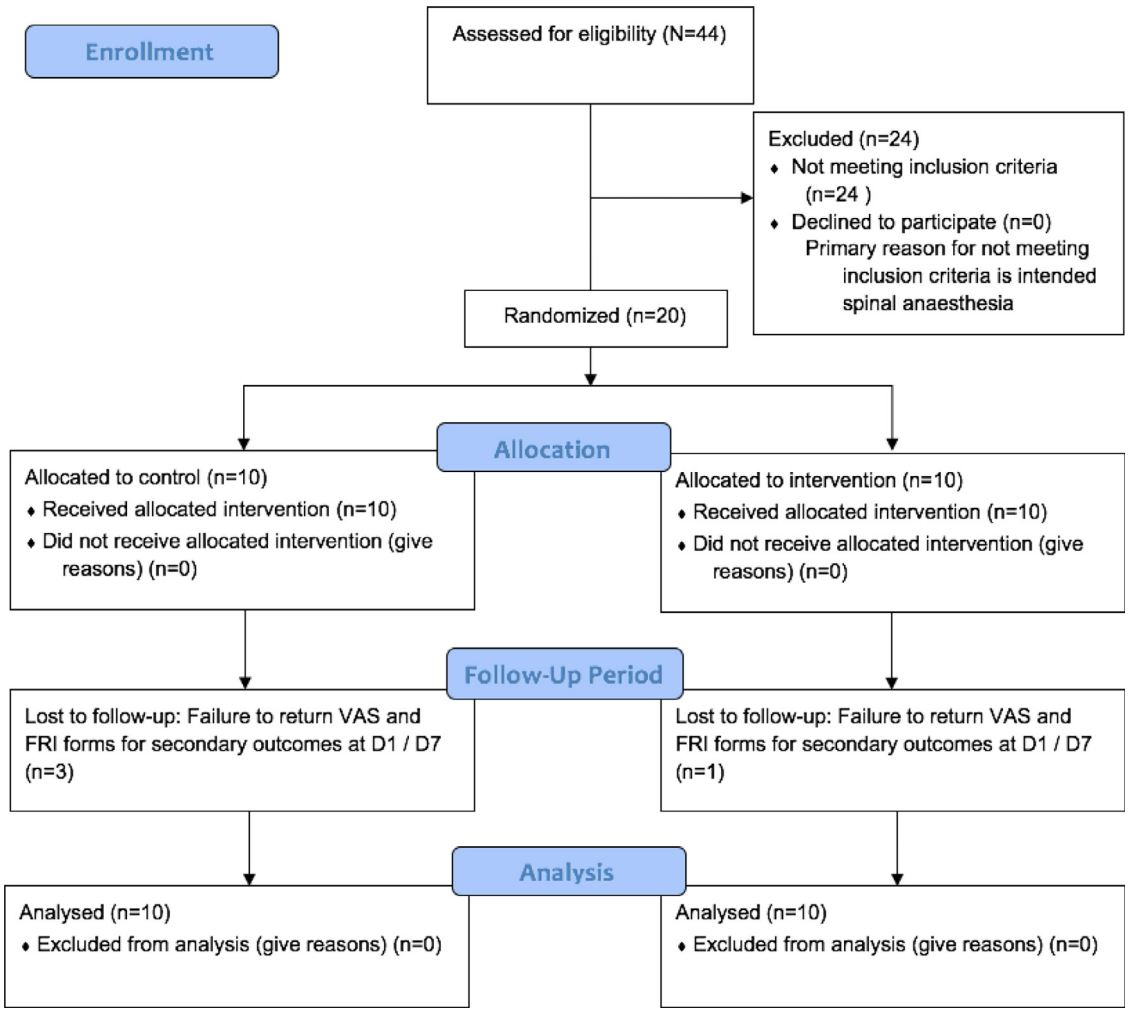

Figure 1. Consolidated Standards of Reporting Trials (CONSORT 2010) flow diagram outlining the study. Abbreviations: D, postoperative day; FRI, Functional Recovery Index; VAS, visual analog score.

Table
Demographic data

Characteristic	Control	Intervention	*p* Value
Age (yr)	47.1 (23 to 72)	55.1 (30 to 80)	≤.31
Gender (M:F)	4:6	2:8	
Height (cm)	166.8 (146 to 186)	166.7 (155 to 181)	≤.984
Weight (kg)	89.8 (58 to 120)	90.3 (62 to 132)	≤.961
Operation			
Ankle or hind foot fusion	2	5	
Tibialis posterior reconstruction	3	2	
Lapidus Akin osteotomy	2	1	
Ankle arthroscopy/lateral ligament complex repair	2	0	
Spring ligament reconstruction	0	1	
Lisfranc joint reconstruction	1	0	
Forefoot reconstruction	0	1	
Anesthetic regional block:			
Popliteal (successful)	8 (8)	10 (9)	
Ankle (successful)	2 (2)	0 (0)	
Mean total tourniquet time (min)	119.2 (97 to 142)	123.5 (90 to 155)	≤.633

Data are mean (range) or n. The control and intervention groups were equally matched for age, height, weight, and type of operation carried out. All patients received a lower-extremity block; all blocks were successful in the control group, and only 1 was unsuccessful in the intervention group, as evidenced by pain and no motor blockade.

4 mg i.v. was administered at the end of surgery for antiemesis. Physiological parameters were recorded preoperatively, after induction of general anesthesia, immediately before tourniquet inflation ($t0$), and at the following intervals after tourniquet inflation: 30, 60, and 90 ($t30$, $t60$, and $t90$), then every 5 minutes until end of surgery ($t\Omega$), defined as deflation of any remaining inflated tourniquet. In addition, parameters were recorded every 5 minutes after the deflation of any tourniquet for a minimum of 15 minutes.

In the control group, 2 tourniquets were applied, but only the above-knee tourniquet was inflated. Immediately before surgical skin preparation, the limb was exsanguinated with a Rhys-Davies exsanguinator (Anetic Aid, Portsmouth, UK), and the thigh tourniquet was inflated to 300 mmHg according to the surgeon and institution's usual practice.

In the intervention group, 2 tourniquets were applied before starting the procedure, around the thigh and high in the calf muscle bulk in anticipation of a gastrocnemius or tendo Achilles lengthening should it become necessary. Immediately before surgical skin preparation, the limb was exsanguinated with a Rhys-Davies exsanguinator (Anetic Aid), and the thigh tourniquet was inflated to 300 mmHg according to the surgeon and institution's usual practice. At 60 minutes, the calf tourniquet was also inflated to 300 mmHg, followed by release of the thigh tourniquet. This ensured continuity of a bloodless surgical field. In certain procedures such as a hind foot fusion, in which bony landmarks such as the tibial tuberosity are required for intraoperative referencing, our simple solution was to carefully cut a small hole in the drape overlying the landmark and cover it with a piece of Ioban (3M, Bracknell, UK) or similar antimicrobial-impregnated adhesive. Alternatively, a sterile calf tourniquet could be used, although we did not use such a device in our study (Fig. 2).

Recruitment ceased once the 20-participant threshold was achieved. All of the participants subsequently completed the study intervention. All surgery exceeded 90 minutes, so primary outcome data was able to be gathered on all participants. Four patients did not return their data for postoperative day 1 (D1) or D7 (3 control group, 1 intervention group); these patients have been included in analysis on an intention-to-treat basis.

Outcome Measures

The primary outcome measure was the MAP at 90 minutes ($t90$). Secondary outcome measures included

- MAP at baseline, postinduction and pretourniquet ($t0$), 30 and 60 minutes posttourniquet ($t30$ and $t60$), and at end of surgery ($t\Omega$);
- Heart rate (HR) at $t0$, $t30$, $t60$, $t90$, and $t\Omega$;

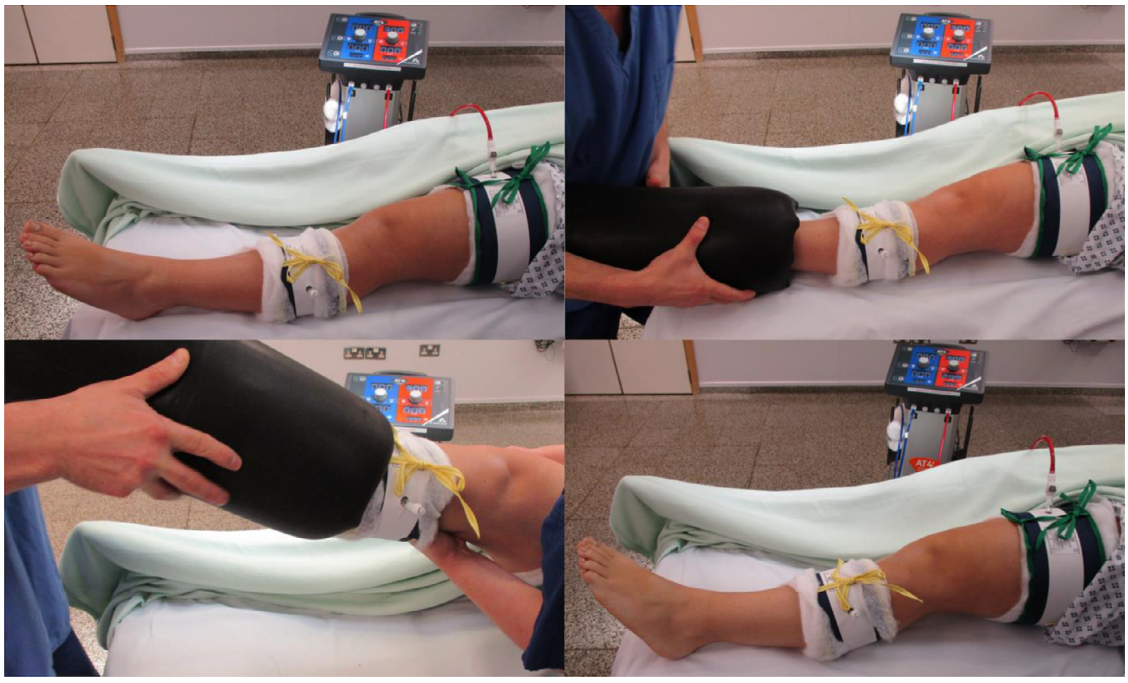

Figure 2. Demonstration of the location of the dual tourniquet sites and the ability to effectively exsanguinate over the calf tourniquet.

- Systolic blood pressure (SBP) at *t*0, *t*30, *t*60, *t*90, and *tΩ*;
- Postoperative pain using an 11-point visual analog scale (VAS) preoperatively (Dp), postoperative day of surgery (D0), D1, and D7;
- Function using the Functional Recovery Index (FRI) (5) at Dp, postoperative D0, D1, and D7.

Physiological parameters were measured immediately after induction of anesthesia and at 30-minute intervals while the tourniquet was inflated, until 60 minutes. They were then recorded every 5 minutes until the end of surgery or 15 minutes after deflation of the final tourniquet, whichever came last.

VAS and FRI scoring was carried out at 4 stages; preoperatively, postoperatively on D0 after recovery from anesthesia, on D1, and on D7. After discharge, participants were given the follow-up questionnaires and requested to complete and return them after completion at the respective time points.

Statistical Analysis

The power calculation was based on our previous observations and experience; we expected to demonstrate an elevation of MAP of ≥20 mmHg, compared with baseline in the control group. A power calculation performed by an independent statistician deemed that a study maximum population of 5 patients in each group would demonstrate an 80% probability of a difference between the 2 groups, if one were to exist. However, to reduce the likelihood of type 1 errors, to increase the relevance of any secondary outcome measures, and to account for any loss to follow-up, the sample size was doubled to 10 patients in each group.

MAP, SBP, and HR are continuous numerical values that are likely to be normally distributed; therefore the Student's *t* test was used for statistical analysis. Data obtained from the VAS scores were also analyzed using a *t* test. The FRI is an ordinal numerical parameter that is not normally distributed; therefore the Mann-Whitney *U* test was used to compare between groups, whereas the Friedman test (the nonparametric equivalent of a 1-way analysis of variance for comparing repeated measures) was used to analyze changes in scores during the postoperative period compared with baseline. Statistical significance was defined at the 5% ($p \leq .05$) level.

Funding

The study was funded entirely by the host organization, The Queen Elizabeth Hospital National Health Service (NHS) Foundation Trust, King's Lynn.

Results

Patient demographic data is presented in Table.

Primary Outcome Measure: MAP at 90 Minutes

At *t*90, the mean control group MAP was 87.8 mmHg compared with the mean intervention group MAP of 76.3 mmHg. This rise of the mean control group MAP above the mean intervention group MAP is statistically significant at $p \leq .014$, demonstrating a significant interaction effect and validating our primary outcome measure.

Secondary Outcome Measure 1: Change in MAP Between Groups at All Time Points

The control group MAP at *t*0 was 71.9 mmHg, rising to 74 mmHg at *t*60. The intervention group MAP at *t*0 was 74 mmHg, rising to 78.5 mmHg at *t*60. As such, from baseline to 60 minutes, there is no significant difference ($p \leq .31$) in MAP between the 2 groups (Fig. 3*A*).

Looking at change in MAP from *t*60 to *tΩ*, the control group MAP rises once again from 87.8 mmHg at *t*90 to 98.1 mmHg at *tΩ*. In contrast, the intervention group remains relatively stable between *t*60 and *tΩ*, with a MAP of 76.3 mmHg at *t*90 and 78.3 mmHg at *tΩ*. At each time point after *t*60, there is a clear change in MAP in the control group compared with the intervention group, with statistical significance at 90 minutes ($p \leq .014$) and at end of surgery ($p \leq .0012$). This demonstrates a significant interaction effect, as there is a lower absolute value in MAP at each time point within the intervention group compared with the control group (Fig. 3*A*).

The changes in MAP from baseline in the control group at *t*60, *t*90, and *tΩ* are 2.1, 15.9, and 26.2 mmHg, respectively, compared with the intervention group at 4.5, 2.3 and 4.3 mmHg, respectively. When we compare the change in MAP between the groups at any time point compared with baseline, this again demonstrates no significant difference at *t*60, $p \leq .47$; however, it demonstrates a significantly lower change in MAP in the intervention group at *t*90 ($p \leq .010$) and a profoundly significantly lower change in MAP at *tΩ* ($p \leq .00045$). This change in MAP further supports a significant interaction effect (Fig. 3*B*).

Secondary Outcome Measure 2: Change in SBP Between Groups at All Time Points

Analysis of SBP demonstrates a correlation similar to MAP, in terms of both absolute change in MAP (Fig. 4*A*) and change in MAP compared with baseline (Fig. 4*B*). Again, there was no significant change in absolute MAP between baseline and 60 minutes, with $p \leq .73$ and $p \leq .18$, respectively. However, when comparing the change in MAP relative to the baseline, a profoundly significant change after the intervention was introduced. At *t*60 versus baseline, $p \leq .35$; t90 versus baseline, $p \leq .025$; and end of surgery versus baseline, $p \leq .007$. Following the pattern previously demonstrated from the MAP data, the change in SBP over the course of the intervention is again different from the control; i.e., the rise in SBP above baseline is significantly lower in the intervention group than the control group, once again demonstrating a significant interaction effect.

Secondary Outcome Measure 3: Change in HR Between Groups at All Time Points

Analysis of the changing HR during the course of surgery does not mirror those of the MAP and SBP data initially. Our data suggest that there is no absolute significant difference at any time point (Fig. 5*A*).

Interestingly, when comparing the change in HR at each assessment stage relative to baseline, we found a significant difference between the group at end of surgery, where the rise in HR above baseline is significantly lower in the intervention group than control, $p \leq .048$. This perhaps demonstrates that of all the physiological parameters measured, HR is the last to decompensate (Fig. 5*B*).

Secondary Outcome Measure 4: Change in VAS Scores in the Postoperative Period

Our data analysis shows that there was no significant change in VAS at any of the 4 time points (preoperatively, D0, D1, or D7) comparing the intervention group to the control, either at rest or with movement. Although not of statistical significance, a trend did exist toward a reduced score in the intervention group compared with the control at each of the 4 assessment stages. It is important to note that the reduction in VAS score was not greater than the minimally clinically important difference, which is 30 points (12) (Fig. 6*A*,*B*).

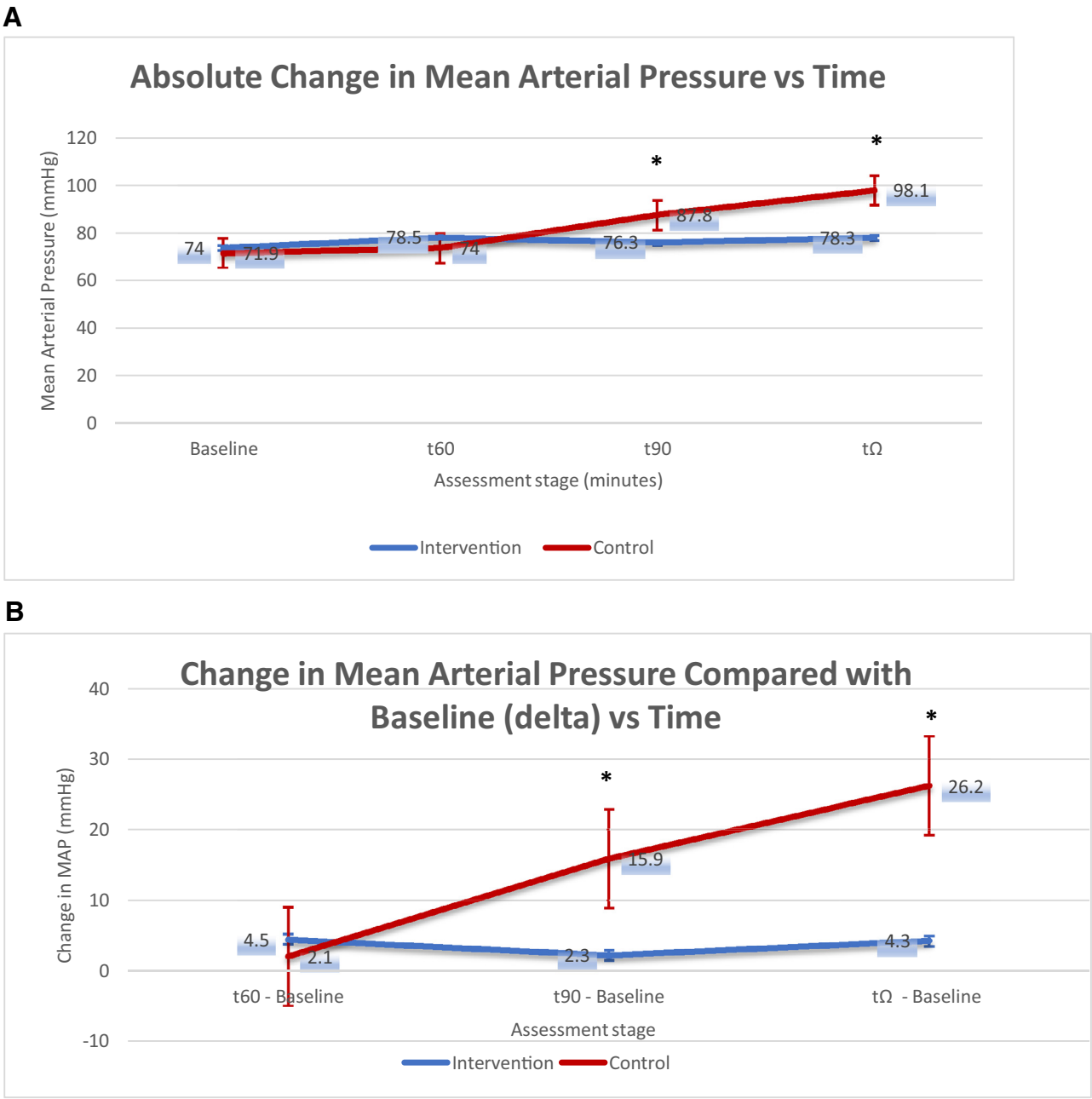

Figure 3. (*A*) Graph of the absolute change in MAP over time. Error bars denote SEM. *Statistically significant (90 min, $p \leq .014$; and end of surgery, $p \leq .0019$). (*B*) Graph of the change in MAP compared with baseline over time. Error bars denote SEM. *Statistically significant (*t*90 versus baseline, $p \leq .010$; and end of surgery versus baseline, $p \leq .00045$). Abbreviations: MAP, mean arterial pressure; SEM, standard error of the mean.

Secondary Outcome Measure 5: Change in FRI in the Postoperative Period

Analysis of the FRI data using the Mann-Whitney *U* test demonstrated a significantly lower score in intervention groups on D0, with the critical value of 1 and the lowest *U* statistic also being 1, producing $p \leq .05$. At D7, there was a trend toward significance in the intervention group, with a critical value of 12 and the lowest *U* statistic of 16. Because this data is nonparametric, the Friedman test was used to analyze the presence of 1-way variance in the repeated FRI scoring on the 4 postoperative assessment stages compared with baseline (preoperative scores) within the same group. No statistical significance was demonstrated between preoperative and D7 scores in either the control or intervention groups, with $p \leq .71$ and $p \leq .74$, respectively (Fig. 7).

Discussion

The outcomes of this study demonstrate that the dramatic physiological effects, notably the hypertensive and HR changes, associated with prolonged lower-limb tourniquet application can be averted by moving the line of crush before the onset of these changes, while still allowing a bloodless surgical field. There may be further benefits to such a technique, such as improved patient-rated outcome measures with regards to VAS scores and in the medium to longer term with regard to FRIs (13), but this study was not sufficiently powered to detect these effects.

Continuing use of lower-limb tourniquets for foot and ankle surgery is controversial, with some modern surgical techniques finding

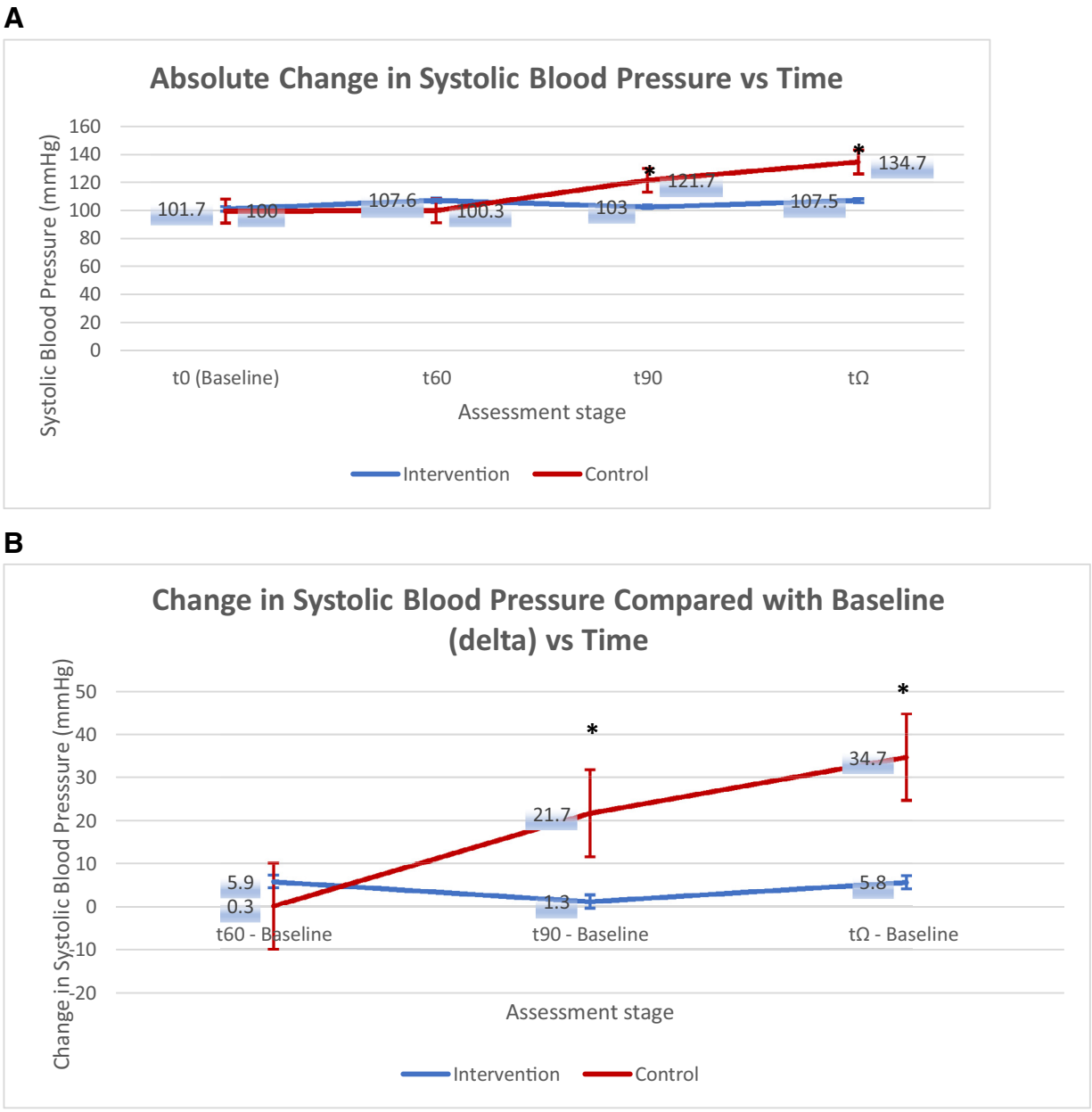

Figure 4. (*A*) Graph of the absolute change in MAP over time. Error bars denote SEM. *Statistically significant (90 min, $p \leq .026$; and end of surgery, $p \leq .011$). (*B*) Graph to illustrate the change in MAP compared with baseline over time. Error bars denote SEM. *Statistically significant (*t*90 versus baseline, $p \leq .024$; and end of surgery versus baseline, $p \leq .007$). Abbreviations: MAP, mean arterial pressure; SEM, standard error of the mean.

alternative ways to provide good operating conditions, such as performing knee arthroplasty without the use of a tourniquet (14). Although a recognized alternative, it is by no means ubiquitous in knee arthroplasty. Moreover, it is important to note that knee arthroplasty cannot be reasonably compared with foot and ankle surgery, which presents unique challenges. This is further supported by the use of limb tourniquets that are still standard practice across not only foot and ankle surgical practices, but also the upper-limb surgical community (15,16). In the context of persisting practice, techniques that minimize physiological disturbance, and which may reduce long-term complications, are valid and warranted.

Persistent crush injury to the muscles and nerves under a tourniquet has been clearly associated with adverse responses, including disruption of nerve conduction progressing to severe chronic pain neuroplasticity changes and profound histological evidence of muscle and nerve dysfunction (5,6,15–19). Crush injury to the skin and vasculature also results in significant damage and functional impairment (2,16,20–24). Indeed, it is even used in animals to produce a predictable chronic pain model on which to study chronic pain therapy (25). There can be little doubt that using a prolonged and severe single line of crush on the tissues of the lower limb results in adverse consequences, yet a requirement still persists for a bloodless surgical field to facilitate complex and developing mid- and fore-foot surgery.

Some of the early changes of significant altered neural conduction may be signified by a hypertensive response to prolonged tourniquet application. Some propose that altered conduction affects the large fibers first, but sustained afferent C-fiber input provides afferent nociceptive input to the body, resulting in a hypertensive response (19,26).

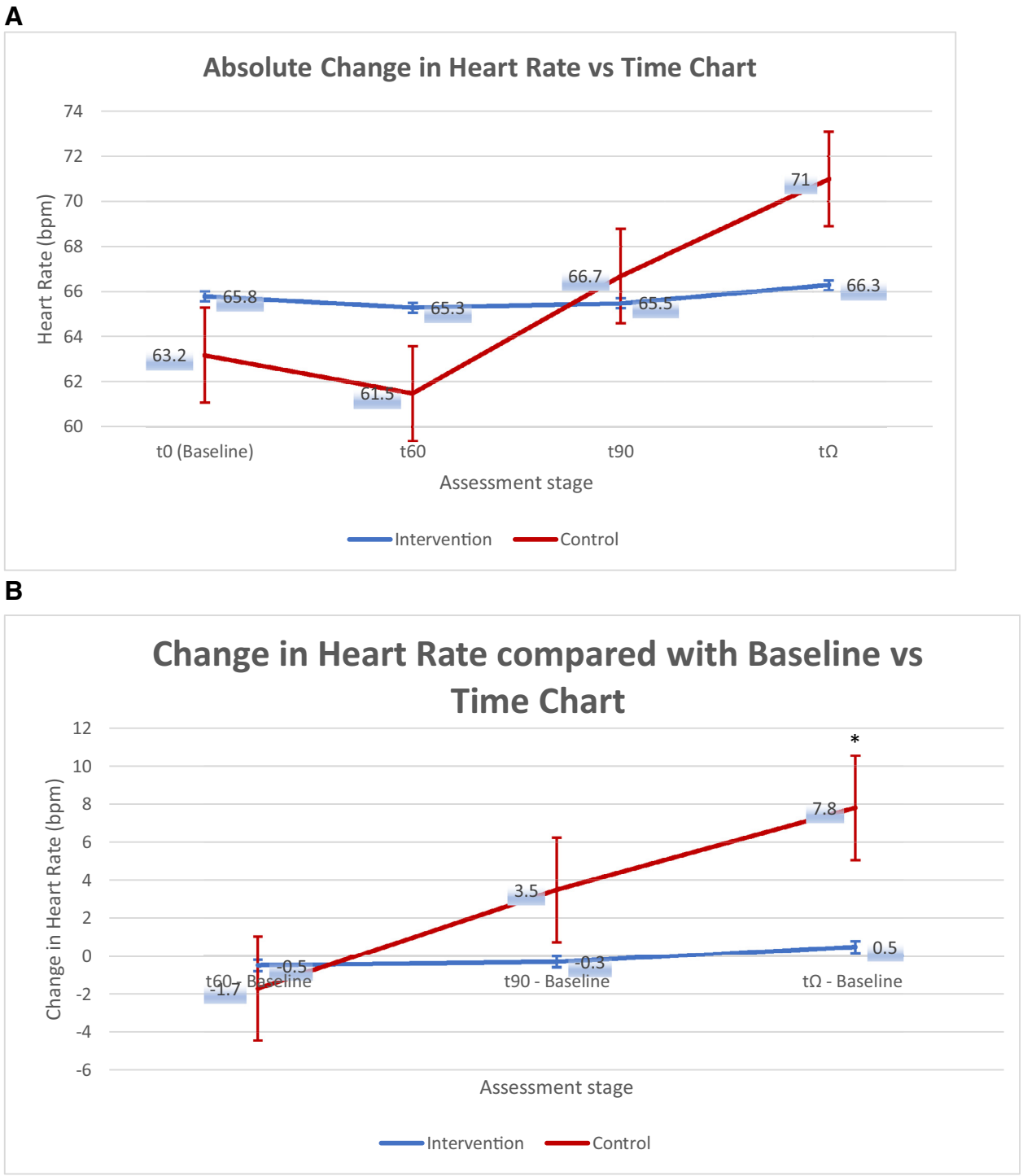

Figure 5. (*A*) Graph to illustrate the absolute change in heart rate compared over time with baseline. Error bars denote SEM. (*B*) Graph to illustrate the change in heart rate compared with baseline over time. Error bars denote SEM. *Statistically significant (end of surgery versus baseline, $p \leq .048$). Abbreviations: HR, heart rate; SEM, standard error of the mean.

Other suggested mechanisms include elevated systemic norepinephrine levels (11), with abnormal central activation of the sympathetic nervous system. Although this study did not focus on or target these mechanisms, one might expect a strategy of relocating the line of crush to prevent such a consequence for a significant amount of tissue of the lower limb.

There are other irregularities with a hypertensive response to prolonged tourniquet application that we believe have not been fully elucidated or explained. Why should a progressive linear hypertensive response to a crush injury occur after a predictable 60 minutes in almost every patient regardless of age, sex, muscle mass, etc.? If we consider this response to be driven by nociceptive pathways, why are opioids ineffective in delay or treatment? Other anesthetic agents such as ketamine have also been shown to have no significant effect on tourniquet-induced hypertension (27–30). Why is this response immediately and completely resolved within minutes of release of the

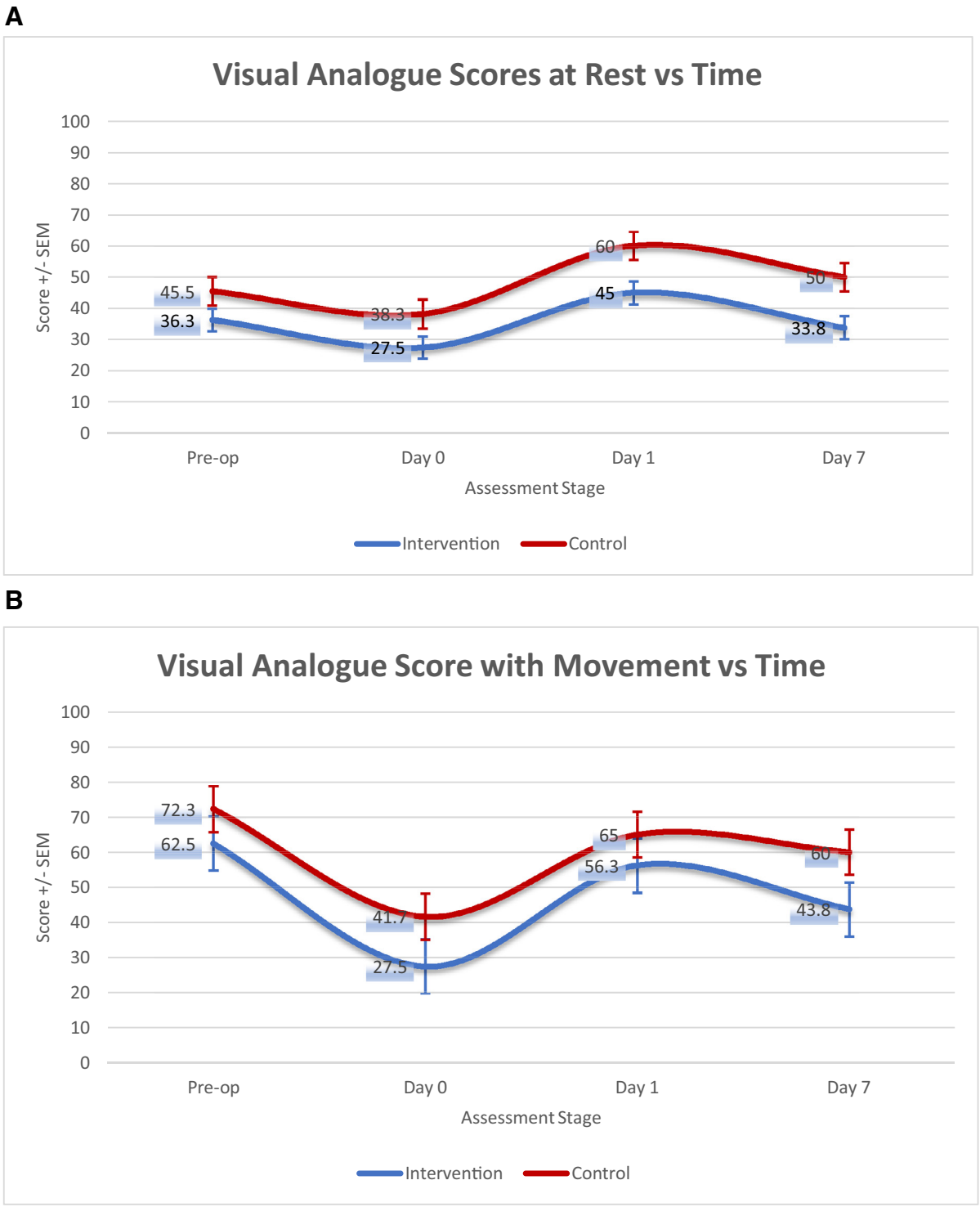

Figure 6. (*A*) VAS at rest over time. Error bars denote SEM. There is no statistical significant difference at any time point. (*B*) VAS with movement over time. Error bars denote SEM. There is no statistical significant difference at any time point. Abbreviations: SEM, standard error of the mean; VAS, visual analog score.

tourniquet? Why is there a progressive increase in minute ventilation in the spontaneously ventilated patient during such an event? Why does the HR not match the other physiologic responses to the same extent?

Regarding tourniquet use on any limb, as mentioned previously, there are no clear guidelines for orthopedic surgeons, with the exception of regularly updated publication in a nursing journal (1). The general consensus among orthopedic surgeons is that both minimal pressure and the least amount of time will decrease the risk of injury; however, tourniquet technique is ultimately the responsibility of the surgeon (23,24). With no clearly defined guidelines on tourniquet device application and safe duration of use (12,31), our double tourniquet technique offers a realistic, minimally expensive, and ubiquitous solution. It is common practice that lower-extremity tourniquets are frequently used for up to 2 hours (1,2). Ideally, interest and substantial

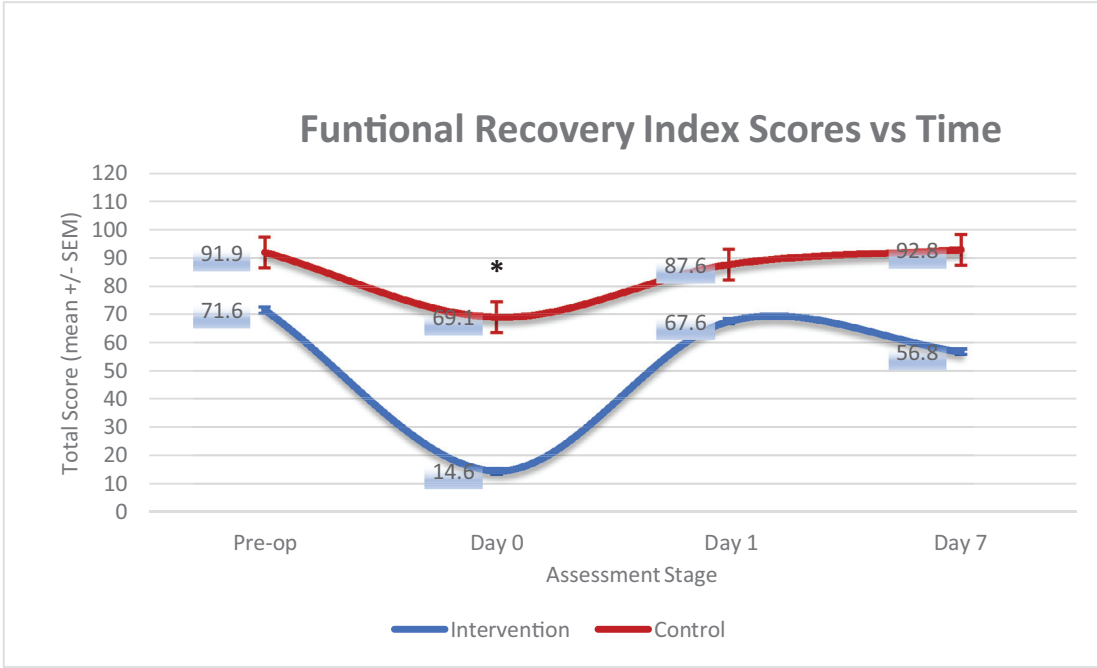

Figure 7. Functional Recovery Index score over time. Error bars denote standard error of the mean (SEM). Statistical significance is present at day 0 between the control and intervention groups ($p \leq .05$).

investment must be focused on fundamental research into the 2-hour "safe" tourniquet time limit, where a suitably equipped and resourced unit examines on a microscopic level the biochemical outcomes of end organ tissue oxygenation and muscle and neural function, which could ultimately put an end to what some might consider a necessary physiological insult. However, this was not the objective of our pragmatic study, which focuses on common practice in everyday National Health Service health economies, with their associated pressures and throughput.

By switching the tourniquet site from thigh to calf at 60 minutes, the patient is not subjected to the sustained compression effects and subsequent sequelae of local complications and systemic tourniquet-induced physiological derangements at a single site. Instead, the extremity benefits from partial reperfusion, and the line of crush is moved distally, all the while maintaining a bloodless surgical field. Such an ability to avoid physiologic consequences of prolonged tourniquet application has been previously demonstrated only in the upper limb using a double-cuff technique on awake patients, who subsequently tolerated up to 3 hours of surgery (32). Again, there is a counterargument that one could release the tourniquet at the 2-hour mark, then wait a period of time before reinflation. As far as we know, there is no evidence to suggest what this "safe period" is. In addition, once the limb has been reperfused, it cannot be as effectively exsanguinated a second time because of the challenges of maintaining a sterile field. We would like to make it abundantly clear that in using this technique of moving the location of the tourniquet to a more distal location on the lower limb intraoperatively, we adhered to the 2-hour safe period (1,2) with a total combined tourniquet inflation time of ~2 hours (Table). There should be no misconceptions: using our technique does not grant the surgeon an additional 2 hours of tourniquet use.

Finally, and possibly the least important reason, in the United Kingdom, National Health Service surgical lists are faced with increasing time pressures, where list efficiency is a priority in terms of patient throughput and achieving targets. There can be no doubt that patient safety is paramount. That is why we believe our method safely modifies existing practices with an anesthetically stable patient, with no derangement to physiological parameters, and without interruption to surgery. Moreover, we have also shown there is a definite improvement in the postoperative functional scores on the day of surgery after recovery from anesthesia, which can only benefit safe patient throughput in day-case surgery.

The limitations of this study include the small numbers in each cohort. However, statistically, we recruited double the number required for an 80% powered study to demonstrate a statistically significant difference between the 2 groups, if one were to exist. Given that the secondary outcomes relied heavily on ongoing patient participation in the first 7 days after their procedure, perhaps in retrospect, we should not have used postal questionnaires and instead have used a telephone or online survey so that loss to follow-up was minimal.

The study did not seek to address our underlying questions on the mechanism provoking the hypertensive response to persistent tourniquet application. However, we believe there are several further areas of research warranted to explore and understand this mechanism more fully, and that may result in not only greater understanding of the mechanism, but potentially also safer ways for patients to undergo surgery under tourniquet application, with less risk of significant functional sequelae.

This study moved a line of crush from a territory with normal/intact sensation to an area that has either ischemic diminished sensory afferent input or afferents that had been blocked by local anesthetic. Our initial remit was to work within the boundaries of normal clinical practice in assessing this pattern of relocation of tourniquet; thus, the results achieved here provide a readily usable solution to a regularly encountered clinical problem. However, we are unable to quantify the extent of the role of afferent neural blockade in negating the hypertensive response.

Finally, it could be argued that a third limb of the study should have been added, in which patients were randomized to a calf tourniquet–only group. During the creative conception of this project, the senior author (C.P.) felt that a calf tourniquet group would preclude such procedures as gastrocnemius lengthening or tendo Achilles lengthening, which are procedures in their own right but can also form part of prolonged foot and ankle reconstructive procedures. It is certainly an area for further research consideration, where the impact of tourniquet-induced hypertension is assessed using a calf tourniquet together with a

popliteal block. Although speculative at this stage, such research could perhaps direct us to using this potential elegant solution unless performing a case that would preclude one from using the calf tourniquet primarily, such as a gastrocnemius or tendo Achilles lengthening.

In conclusion, we have demonstrated an innovative method of ablating the hypertensive response to prolonged lower-limb tourniquet application while retaining the perceived surgical advantages of a bloodless operating field, which may have further implications for clinical practice and outcomes.

Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1053/j.jfas.2019.03.019.

References

1. Association of Perioperative Registered Nurses. Recommended practice for the use of the pneumatic tourniquet in the perioperative practice setting. AORN J 2007;86:640–655.
2. Odinsson A, Finsen V. Tourniquet use and its complications in Norway. J Bone Joint Surg Br 2006;88:1090–1092.
3. Derner R, Buckholz J. Surgical hemostasis by pneumatic ankle tourniquet during 3027 podiatric operations. JFAS 34:236–246.
4. Massey KA, Blakeslee C, Martin W, Pitkow HS. Pneumatic ankle tourniquets: physiological factors related to minimal arterial occlusion pressure. JFAS 38:256–263.
5. Han SE, Lin CS-Y, Boland RA, Bilston LE, Kiernan MC. Changes in human sensory axonal excitability induced by focal nerve compression. J Physiol 2010;588:1737–1745.
6. Murphy CG, Winter DC, Bouchier-Hayes DJ. Tourniquet injuries: pathogenesis and modalities for attenuation. Acta Orthop Belg 2005;71:635–645.
7. Kam PC, Kavanagh R, Young FF. The arterial tourniquet: pathophysiological consequences and anaesthetic implications. Anaesthesia 2001;56:534–545.
8. Noordin S, McEwen J, Kragh JF, Eisen A, Masri B. Surgical tourniquets in orthopaedics. J Bone Joint Surg Am 2009;91:2958–2967.
9. Kaufman RD, Walts LF. Tourniquet-induced hypertension. Br J Anaesth 1982;54:333–336.
10. Townsend HS, Goodman SB, Schurman DJ, Hack- el A, Brock-Utne JG. Tourniquet release: systemic and metabolic effects. Acta Anaesthesiol Scand 1996;40:1234–1237.
11. Crews JC, Sehlhorst CS. Response to maintenance of tourniquet inflation in a primate model. Reg Anesth 1991;18:195–198.
12. Lee JS, Hobden E, Steill IG, Wells GA. Clinically important change in the visual analog scale after adequate pain control. Acad Emerg Med 2003;10:1128–1130.
13. Wong J, Tong D, De Silva Y, Abrishami A, Chung F. Development of the functional recovery index for ambulatory surgery and anaesthesia. Anesthesiology 2009;100:596–602.
14. Smith TO, Hing CB. Is a tourniquet beneficial in total knee replacement surgery? A meta-analysis and systematic review. Knee 2010;17:141–147.
15. Younger AS, Kalla TP, McEwen JA, Inkpen K. Survey of tourniquet use in orthopaedic foot and ankle surgery. Foot Ankle Int 2005;26:208–217.
16. Drolet BC, Okhah Z, Phillips BZ, Christian BP, Akelman E, Katarincic J, Schmidt ST. Evidence for safe tourniquet use in 500 consecutive upper extremity procedures. Hand (NY) 2014;9:494–498.
17. Nitz AJ, Matulionis DH. Ultrastructural changes in rat peripheral nerve following pneumatic tourniquet compression. J Neurosurg 1982;57:660–666.
18. Crews JC, Cahall M, Behbehani MM. The neurophysiologic mechanisms of tourniquet pain; the activity of neurons in the rostroventral medulla in the rat. Anesthesiology 1994;81:730–736.
19. Crews JC, Cahall M. An investigation of the neurophysiologic mechanisms of tourniquet-related pain: change in spontaneous activity and receptive field size in spinal dorsal horn neurons. Reg Anesth Pain Med 1999;24:102–109.
20. MacIver MB, Tanelian DL. Activation of C fibers by metabolic perturbations associated with tourniquet ischaemia. Anesthesiology 1992;76:617–623.
21. Bridenbaugh PO, Hagenouw RRPM, Gielen MJM, Edstrom HH. Addition of glucose to bupivacaine in spinal anesthesia increases incidence of tourniquet pain. Anesth Analg 1986;65:1181–1185.
22. Brin YS, Feldman V, Ron Gal I, Markushevitch M, Regev A, Stern A. The sterile elastic exsanguination tourniquet vs. the pneumatic tourniquet for total knee arthroplasty. J Arthroplasty 2015;30:595–599.
23. Olivecrona C, Lapidus LJ, Benson L, Blomfeldt R. Tourniquet time affects postoperative complications after knee arthroplasty. Int Orthop 2013;37:827–832.
24. Estebe JP, Davies JM, Richebe P. The pneumatic tourniquet: mechanical, ischaemia-reperfusion and systemic effects. Eur J Anaesthesiol 2011;28:404–411.
25. Coderre TJ, Xanthos DN, Francis L, Bennett GJ. Chronic post-ischemia pain (CPIP): a novel animal model of complex regional pain syndrome-type I (CRPS-I; reflex sympathetic dystrophy) produced by prolonged hindpaw ischemia and reperfusion in the rat. Pain 2004;112:94–105.
26. Lowrie A, Jones MJ, Eastley RJ. Effect of a eutectic mixture of local anaesthetic agents (EMLA) on tourniquet pain in volunteers. Br J Anaesth 1989;63:751–753.
27. Satsumae T, Yamaguchi H, Sakaguchi T, Yamashita S, Yamamoto S, Kida H. Preoperative small-dose ketamine prevented tourniquet-induced arterial pressure increase in orthopedic patients under general anesthesia. Anesth Analg 2001 May;92:1286–1289.
28. Yamashita S, Yamaguchi H, Hisajima Y, Ijima K, Saito K, Chiba A, et al. Preoperative oral dextromethorphan attenuated tourniquet-induced arterial blood pressure and heart rate increases in knee cruciate ligament reconstruction patients under general anesthesia. Anesth Analg 2004;98:994–998.
29. Park JW, Jung YH, Baek CW, Kang H, Cha SM. Effects of low dose ketamine on tourniquet-induced haemodynamic responses during general anaesthesia. J Int Med Res 2007;35:600–608.
30. Ongaya J, Mung'ayi V, Sahrif T, Kabujugi J. A randomized controlled trial to assess the effect of a ketamine infusion on tourniquet hypertension during general anaesthesia in patients undergoing upper and lower limb surgery. Afr Health Sci 2017;17:122–132.
31. Crenshaw A. Surgical techniques and approaches. In: Canale ST, ed. *Campbell's Operative Orthopaedics*, 9th edition, Maryland Heights, MO: Mosby, 1998:29–142.
32. Neimkin RJ, Smith RJ. Double tourniquet with linked mercury manometers for hand surgery. J Hand Surg Am 1983;8:938–941.