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Influence of Thigh Tourniquet Release on Cardiovascular Parameters While Operated Limb in Elevated Position

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Abstract

Background: Release of pneumatic tourniquet causes hypotension, tachycardia and compensatory increase in cardiac index.

Objectives: This study was conducted to assess the effect of tourniquet release on cardiovascular parameters while operated leg in 20 degrees up position.

Design: Prospective randomized comparative study.

Setting: University hospital.

Methods: This study included 60 patients, ASA I scheduled for meniscal surgery through knee arthroscopy under general anesthesia. Patients were divided into two groups: group C (n=30) in which tourniquet was deflated with leg in neutral position while group S (n=30) tourniquet was released with leg in 20 degrees up position. Cardiovascular parameters displayed by ccNexfin monitor eg cardiac output (COP), cardiac index (CI), stroke volume (SV), mean arterial pressure (MAP), systemic vascular resistance (SVR), heart rate (HR), as well as arterial blood gas measurements. End tidal carbon dioxide, respiratory rate, end tidal sevoflurane and bispectral index values were also recorded before and after tourniquet deflation.

Results: There was significant decrease in MAP and SVR at all intervals post deflation in group C compared to group S whereas COP, CI, SV and HR were significantly increased in group C post deflation compared to group S. But there were no significant differences between groups regarding metabolic and respiratory parameters.

Conclusion: Tourniquet deflation while leg in elevated position caused attenuation in both the decrease in MAP, SVR and the compensatory increase in CI, COP and SV.

Limitations: This study is limited by its small sample size.

Keywords: Thigh tourniquet; Knee arthroscopy; Orthopedic surgery

Introduction

Applying pneumatic tourniquet for limb procedures would provide bloodless field thus minimizes blood loss and risk of blood transfusion with facilitation of surgical procedures and expedition of procedures. Pneumatic tourniquet has several complications among which acute hemodynamic changes have been described [1].

Hemodynamic changes associated with tourniquet deflation include; hypotension, tachycardia and increase in cardiac index [2]. These effects may be insignificant for healthy patients but, risky for patients with compromised cardiovascular system and the geriatric population.

For the high risk patients, minimizing those acute hemodynamic changes and the requirement of vasoactive agents with tourniquet release will be valuable [3,4]. ccNexfin monitor (ccNexfin-BMEYE, Amesterdam, Netherland) is a non-invasive monitor that provides real-time, beat-to-beat and continuous measurements of cardiac output (COP), cardiac index (CI), stroke volume (SV), systemic vascular resistance (SVR), mean arterial pressure (MAP) and heart rate (HR) by a simple finger cuff sensor.

ccNexfin monitor proved to be an effective hemodynamic monitor in patients with reduced arterial pulsations as patients on continuous flow left ventricular assisted devices [5]. Also, ccNexfin is validated for non-invasive continues blood pressure monitoring in pregnant population [6].

The aim of this study was to assess the effect of release of thigh tourniquet while operated limb in 20 degrees up position on hemodynamics and cardiac parameters measured by ccNexfin monitor.

Patients and Methods

Patients

After approval of the ethical committee and informed consent was obtained, 60 patients, aged 18-50 years, classified as ASA (American society of Anesthesiologists) physical status I, with no major cardiovascular and respiratory problems, underwent elective knee arthroscopy for meniscal surgery that involved the use of pneumatic tourniquet were enrolled in this prospective randomized study.

Patients with peripheral neuropathy, DVT in the limb, Reynaud's disease, and peripheral vascular disease were excluded from the study. Patients were premedicated with midazolam 0.03 mg/kg IV 10 minutes before pushing patients to operating theatre. Patients were put in supine position on operating theatre table (MAQUET, alpha maxx, Germany) which has the option of unilateral up-down leg positioning.

Before induction of general anesthesia, routine monitoring in form of electrocardiogram, non-dominant arm for non-invasive blood pressure and pulse oximetry (Cardio Cap II, Datex Omeda, Helsinki, Finland) whereas ccNexfin monitor (ccNexfin, BMEYE, Amesterdam, Netherland) was applied to the patient's dominant hand. After entering the patient's data (age, gender, weight in kg, height in cm) into the ccNexfin monitor, the finger cuff sensor was applied to the middle finger and a heart reference system was applied by connecting the wrapped ring finger cuff to the pericardial region (stuck to the patient's gown over the heart) (Figure 1).

This heart reference system ensures actual values by compensating for any gravitational hydrostatic effect during any body movements. A small wire fixing unit was wrapped around the patient's wrist. The ccNexfin monitor displayed continuous HR, arterial blood pressure (systolic, diastolic, and MAP), oxygen saturation, COP (liters/min), CI ($L/min/m^2$), SV (ml/beat) and SVR ($dyn.S/cm^5$) (Figure 2) [7,8].

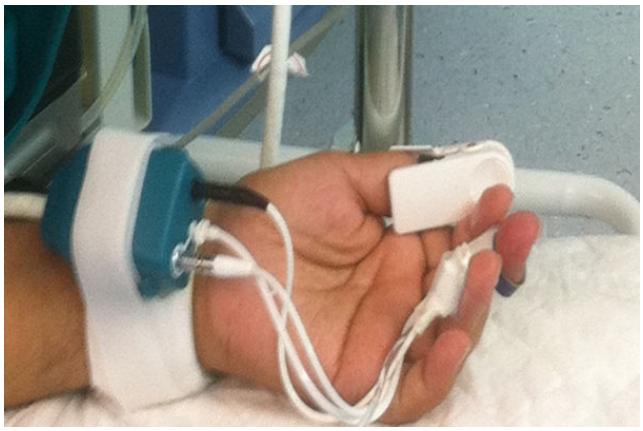

Figure 1: ccNexfin application.

Anesthesia

General anesthesia was induced in all patients with remifentanil 2 mcg/kg IV followed by bolus dose of propofol 2.5 mg/kg IV and laryngeal mask (LMA) size 3 or 4 was applied during propofol induced apnea. If the LMA failed to be inserted, malpositioned or resulted in ineffective ventilation, it was removed and patient's trachea was intubated with endotracheal tube and patient excluded from the study. Analgesia was maintained by remifentanil infusion at a rate of 0.1 mcg/kg/min while anesthesia was maintained by sevoflurane 1.5-2 vol % in nitrous oxide/oxygen mixture (50%/50%) with fresh gas flow 8 liters per minute. Sevoflurane was titrated to keep bispectral index (BIS) values between 40-60 (BIS, BISA-2000 Monitor R, Aspect Medical Systems, Leiden, and Netherland).

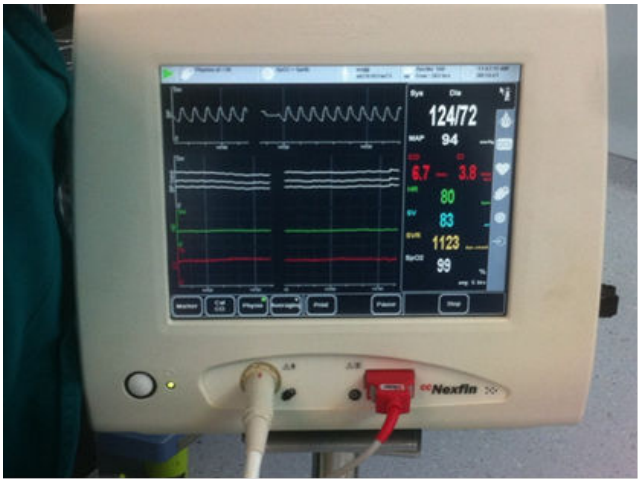

Figure 2: Cardiac parameters displayed by ccNexfin monitor.

Patients were manually ventilated till spontaneous respiration resumed. Radial artery cannula (20 G, ARROW, USA) was inserted in the non-dominant hand for repeated arterial blood gas (ABG) samplings. Each blood sample (1 ml volume) was introduced in Cobas b analyzer 221, (Roche OMNIS, Germany) for measurements of pH, PO_2, PCO_2, HCO_3, K and lactate. Fluid replacement was by lactated ringer infusion at a rate of 6 ml/kg/hr and body temperature was maintained above 35.5C° by covering the patient's chest and upper limbs with air blanket (Bair Hugger, Arizant Health Care Inc., USA). The operated limb was exsanguinated with Esmarch bandage then thigh pneumatic tourniquet was elevated to 350 mmHg or according to the surgeon's discretion.

At end of surgery and before release of tourniquet, patients were stratified into two equal groups (n=30 each) by using the sealed envelope method. Group C (control group) in which tourniquet was deflated with the operated limb in neutral position. While in group S (study group), the tourniquet was deflated with operated limb in 20 degrees up position and kept elevated for 15 minutes which was the study time.

Assessment parameters

Cardiovascular parameters displayed by ccNexfin monitor such as MAP, HR, COP, CI, SV, SVR, as well as ABG measurements (pH, PCO_2, PO_2, HCO_3, K, lactate), end tidal carbon dioxide (ET CO_2), respiratory rate (RR), BIS values, temperature and end tidal sevoflurane (ET sevoflurane) were recorded at the following intervals: T0 (10 minutes after induction of general anesthesia and before tourniquet inflation), Ti5 (5 minutes after tourniquet inflation, Tb (time just before tourniquet release), Td1, Td3, Td5, Td10, Td15 (1, 3, 5, 10 and 15 minutes respectively after tourniquet deflation).

Statistics

Power of the study

Sample size was calculated using Medcalc, with α=0.05, and 95% a confident interval (CI) and the power of study is 80% and clinically relevant difference in mean arterial blood pressure of 15 mmHg a total of 60 patients divided into two equal groups was found to be sufficient to conduct the study.

Data analysis

Statistical analysis was carried out with the Statistical Package for Social Sciences (SPSS) 15 software. The Student t-test was used for the comparison of continuous variables between the study and control groups and paired t-test used to compare variables within the same group. The data was expressed as mean values and standard deviations, or numbers and percentages, as appropriate. P-value of <0.05 was considered to be statistically significant. Chi-Square (χ^2) was used for comparison of non-parametric data as gender.

Results

There was no significant difference between the two groups in demographic data and operative details (Table 1). Most of the patients were males. In all cases, there was a bloodless field without use of atropine or ephedrine for bradycardia or hypotensive episodes.

	Group C (n=30)	Group S (n=30)
Age (yrs)	28 ± 2	29 ± 2
Gender (Male/Female)	(26/4)	(27/3)
Weight (kg)	(79 ± 5)	(80 ± 5)
Height (cm)	169 ± 10	167 ± 10
Tourniquet time (min)	48 ± 6	50 ± 4
Surgical time (min)	42 ± 5	40 ± 5
Anesthesia time (min)	82 ± 7	80 ± 9
Base line Hb (gm%)	12.7 ± 0.5	13.1 ± 0.4

Table 1: Values are means ± SD. Gender is in number.

With tourniquet inflation, MAP and SVR were significantly increased (P<0.05) at Ti5 and Tb compared to T0 while, with deflation there was significant decrease (P<0.05) in both MAP and SVR at all-time intervals in two groups, but group (C) showed significant decrease (P<0.05) in MAP and SVR compared to group (S) at all-time intervals post deflation.

In group (C) MAP was lower at Td1, Td3, Td5, Td10 and Td15 to a mean values 15%, 14%, 13%, 5% and 3% respectively than that of T0.

While in group (S) MAP was lower at Td1 and Td3 to the mean values 4% and 2% than that of T0 respectively. Whereas MAP was equal at Td5, Td10 and T15 in comparison to T0 (Figure 3).

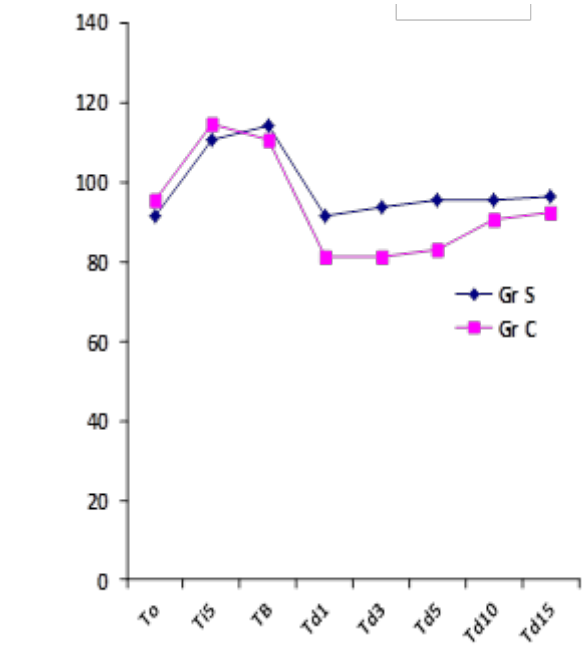

Figure 3: Mean arterial.

In group (C) SVR was lower at Td1, Td3, Td5, Td10 and Td15 to the mean values 30%, 29.2%, 28.5%, 22.8% and 15.3% respectively than that of T0 while in group (S), SVR was lower at Td1, Td3, Td5, Td10 and Td15 to the mean values 11.9%, 11.2%,9.1%, 7.72% and 5.33% respectively compared to T0 (Figure 4).

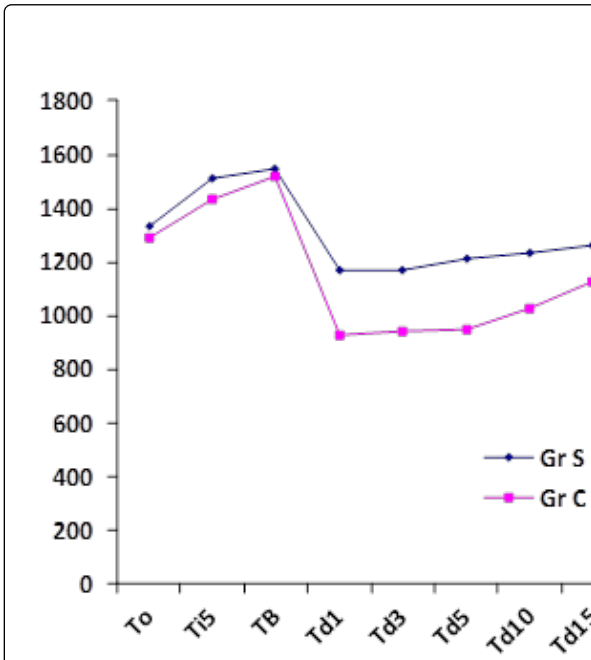

Figure 4: Systemic Vascular Resistance.

HR in group (C), increased significantly at Td1, Td3, Td5, Td10 and Td15 to the mean values 17.7%, 14.25%, 10.6%, 3.4% and 2% respectively compared to T0 whereas in group (S), HR increased to the mean values 2.3%, 1.1% and 1% at Td1, Td3 and Td5 respectively compared toT0 and it was almost the same values at Td10 and Td15 compared to T0. But HR was significantly increased (P<0.05) in group (C) compared to group (S) at all corresponding time intervals (Figure 5).

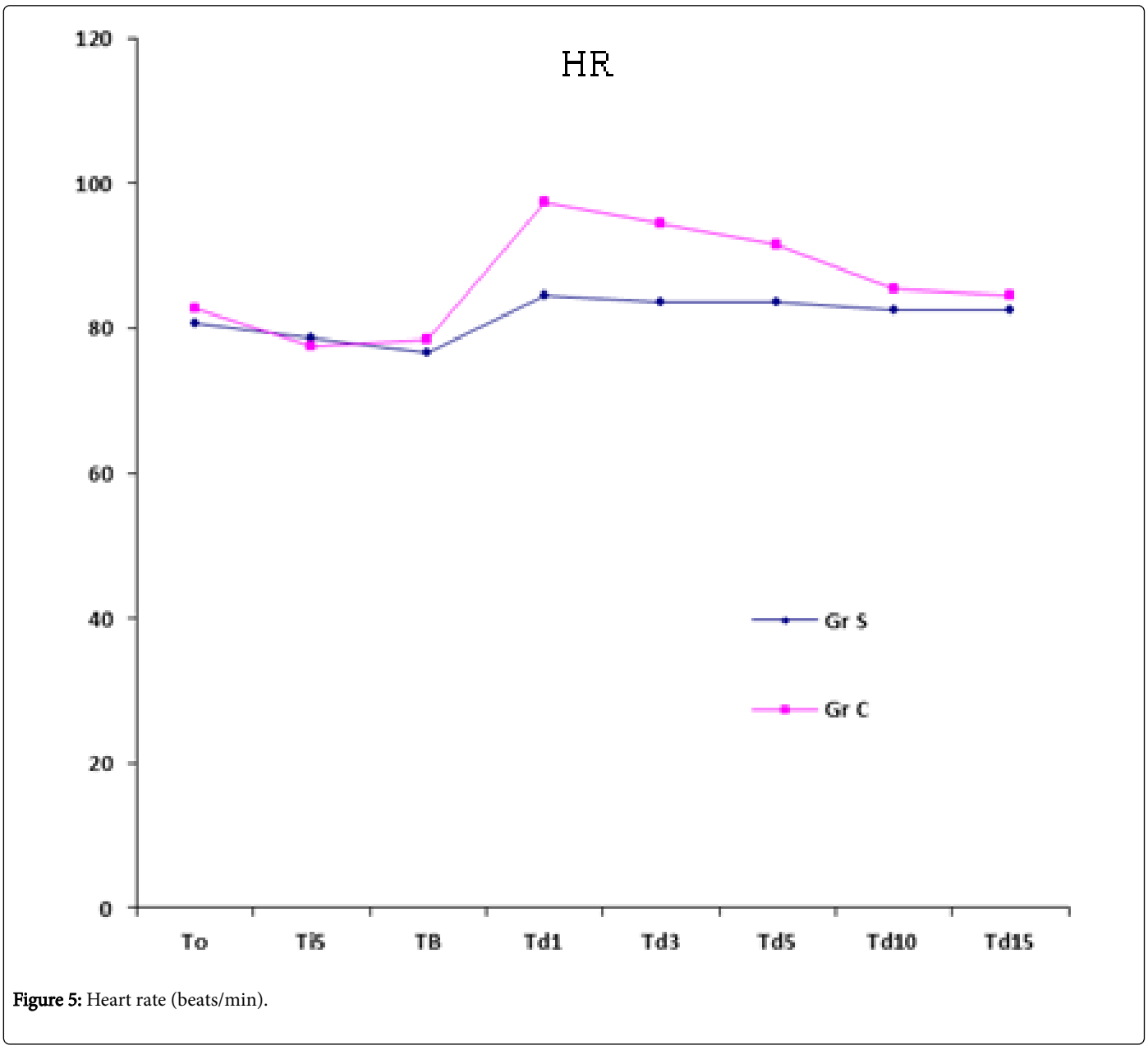

Figure 5: Heart rate (beats/min).

As regards SV in group (C), it increased significantly (P<0.05) at Td1, Td3, Td5, Td10 and Td15 to the mean values 35.4%, 34.8, 31%, 25.6% and 19% respectively compared to T0 while in group (S), SV increased with 14.8%, 13.2%, 12.8%, 11.7% and 11.5% at Td1, Td3,Td5, Td10 and Td15 respectively compared to T0. But SV was significantly increased (P<0.05) in group (C) compared to group (S) at all corresponding time intervals (Figure 6).

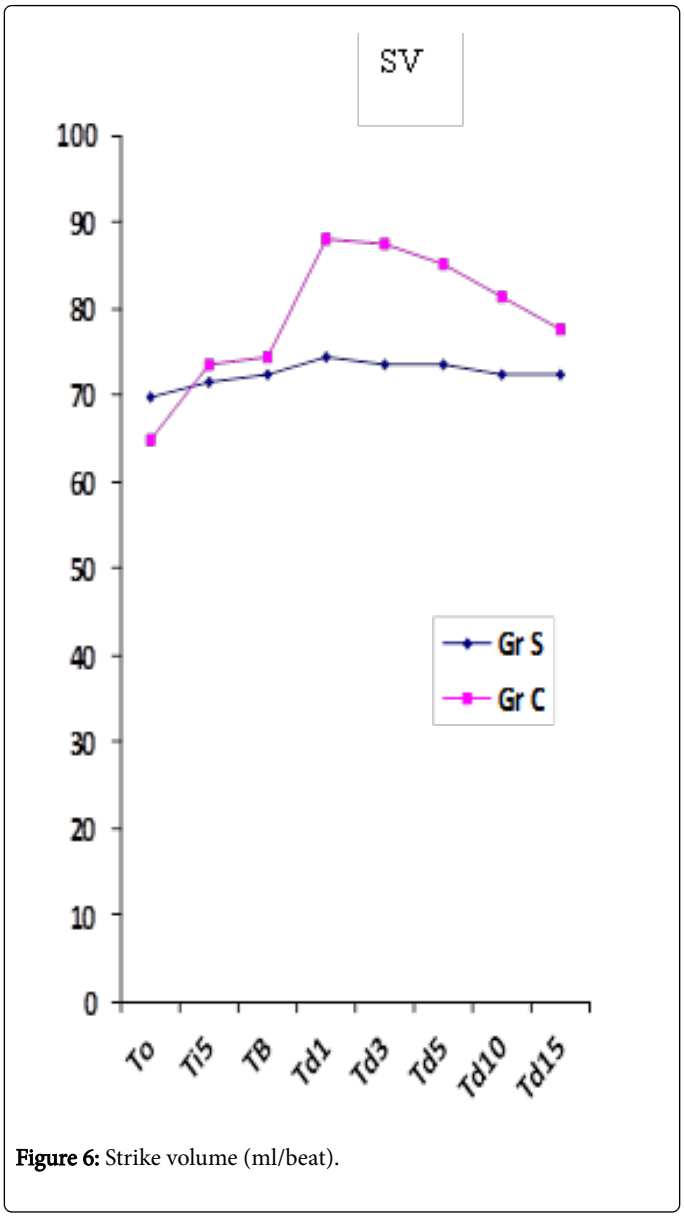

Figure 6: Strike volume (ml/beat).

On the other hand, COP was increased significantly in group (C) at Td1, Td3, Td5, Td10 and Td15 to the mean values 31.1%, 30%, 29.5%, 26% and 17.2% respectively compared to T0. Moreover COP increased in group (S) with the following mean values 7.8%, 7.5%, 7.3%, 3.3% and 1.2% at Td1, Td3,Td5, Td10 and Td15 respectively compared to T0. But COP was significantly increased (P<0.05) in group (C) compared to group (S) at all corresponding time intervals (Figure 7).

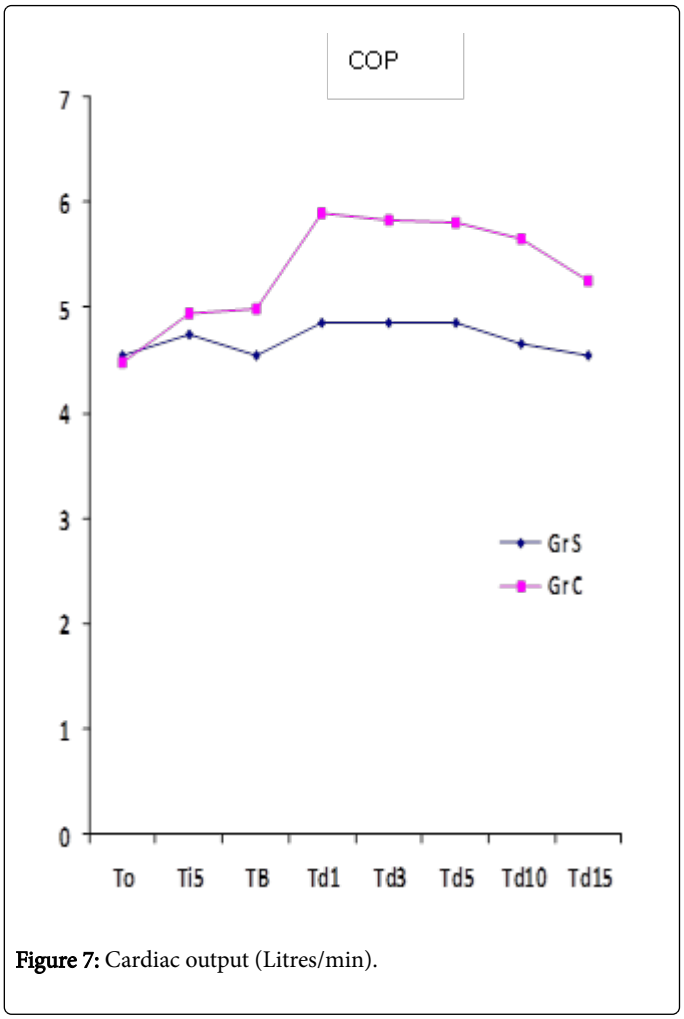

Figure 7: Cardiac output (Litres/min).

Also, CI increased significantly (P<0.05) in group (c) at Td1, Td3 ,Td5, Td10 and Td15 to the mean values 39.4%, 38.6%, 34.1%, 29.3% and 24.7% respectively compared to T0 while CI increased in group (S) with mean values 23.3%, 22.1%, 20.3%, 14% and 9.4% at Td1, Td3, Td5, Td10 and Td15 respectively compared to T0. But CI was significantly increased (P<0.05) in group (C) compared to group (S) at all corresponding time intervals. In both groups, pH was significantly decreased while, lactate, $PaCO_2$, $ETCO_2$, RR and K level were significantly increased at all intervals post deflation compared to T0 but, between group comparison, there was no significant difference in these metabolic and respiratory measurements (Table 2). There was no significant difference either in intra-group or inter-group comparisons as regards HCO_3, PaO_2, temperature, BIS values and ET sevoflurane.

		T0	Ti 5	Tb	Td 1	Td 3	Td 5	Td 10	Td 15
PH	Gr C	7.44 ± 0.008	7.43 ± 0.007	7.43± 0.006	7.31 ± 0.005*	7.30 ± 0.005*	7.31 ± 0.006*	7.34 ± 0.005*	7.34 ± 0.004*
	Gr S	7.43 ± 0.007	7.43 ± 0.008	7.44 ± 0.007	7.30 ± 0.004*	7.31 ± 0.005*	7.32 ± 0.005*	7.33 ± 0.005*	7.35 ± 0.004*
PCO_2	Gr C	36 ± 0.83	33.46 ± 0.5	33.43± 0.4	48.9 ± 0.82*	47 ± 0.78*	45.5 ± 0.5*	41 ± 0.81*	40.9 ± 0.77*
	Gr S	36 ± 0.78	33.66 ± 0.47	33.55 ± 0.6	48.1 ± 0.75*	46 ± 0.66*	44.5 ± 0.4*	40 ± 0.73*	40.2 ± 0.69*

Lact	Gr C	0.84 ± 0.05	0.85 ± 0.06	0.86 ± 0.06	2.34 ± 0.11*	2.33 ± 0.13*	2.35 ± 0.11*	2.35 ± 0.11*	2.34 ± 0.12*
	Gr S	0.85 ± 0.06	0.87 ± 0.04	0.88 ± 0.05	2.28 ± 0.10*	2.30 ± 0.12*	2.31 ± 0.10*	2.31 ± 0.10*	2.32 ± 0.11*
K	Gr C	4.05 ± 0.05	4.08 ± 0.06	4.04 ± 0.05	4.74 ± 0.05*	4.75 ± 0.06*	4.7 ± 0.05*	4.55 ± 0.06*	4.53 ± 0.05*
	Gr S	4.0 ± 0.07	4.03 ± 0.08	4.03 ± 0.07	4.70 ± 0.06*	4.73 ± 0.08*	4.67 ± 0.08*	4.5 ± 0.07*	4.49 ± 0.06*
$ETCO_2$	Gr C	31.5 ± 0.5	30.43 ± 0.6	30.5 ± 0.6	45.9 ± 0.8*	45.9 ± 0.75*	43.5 ± 0.5*	39.5 ± 0.6*	37.5 ± 0.7*
	GrS	32 ± 0.6	31.4 ± 0.5	31 ± 0.6	44.8 ± 0.7*	44 ± 0.8*	43 ± 0.6*	38.9 ± 0.5*	36.5 ± 0.6*
RR	Gr C	11.73 ± 0.44	11.53 ± 0.5	11.56 ± 0.5	18.46 ± 0.5*	18.46 ± 0.7*	17.5 ± 0.6*	15.5 ± 0.7*	14.6 ± 0.6*
	Gr S	11.6 ± 0.49	11.66 ± 0.47	11.6 ± 0.46	18.2 ± 0.6*	18.1 ± 0.5*	17.1 ± 0.6*	15.2 ± 0.5*	14.3 ± 0.8*

PCO_2: Partial Pressure of Carbon dioxide (mmHg); lactate (mmoL/L); K: Potassium (meq/l); Figure 7: Cardiac output (Litres/min)

$ETCO_2$: End tidal carbon dioxide (mmHg), RR (respiratory rate/min); Values are mean ± SD. *=P<0.05 when comparing values at all-time intervals to T0 in same group. There was no significant difference during between groups comparison of values at all-time intervals.

Table 2: Intra operative Respiratory and Metabolic Parameters.

Discussion

The aim of this study was to assess the effect of tourniquet release on hemodynamics and cardiac parameters while the operated limb in 20 degrees up position compared to tourniquet release while leg being at neutral position. Using pulmonary artery catheter; an invasive method, or a pulsed doppler echocardiography; an expensive tool, in a clinically free patients undergoing elective lower limb orthopedic procedure is not indicated. We used ccNexfin monitor for non-invasive cardiac parameters measurement. ccNexfin monitor has been validated for continuous blood pressure monitoring and cardiac parameters monitoring in critically ill patients [9,10].

With tourniquet inflation, there was a significant increase in MAP in both groups. Tourniquet inflation under general anesthesia causes increase in MAP which could be explained by possible three mechanisms: first, abrupt increase in SVR secondary to the reduction in limb vascular bed. Second, shifting of blood towards the central venous side as a result of limb exsanguination before tourniquet inflation, and lastly because of pain perception caused by tourniquet compression and limb ischemia [11]. In the current study, pain factor might be excluded as a cause of increased MAP 5 min (Ti5) post inflation due to absence of increase in HR, which is an indicator of pain, induced sympathetic activation. Also, it was not the timing of tourniquet pain which usually starts 30-45 min post inflation [12].

There was a significant increase in COP, CI, and SV in both groups at Ti5 compared to T0. Limb Exsanguination followed by tourniquet inflation will drain blood from the lower limb to the right side of the heart then to the left ventricle thus increasing left ventricular preload and according to the Frank-Starling curve; the SV will be increased [13]. As COP=SV × HR [14], the raised SV measured by ccNexfin subsequently increased the COP and CI. At time of tourniquet deflation, abrupt decrease in MAP occurs with intensity and duration dependent on the type of anesthesia provided [15]. Group C had a significant decrease in MAP and SVR at all intervals post deflation compared to baseline values (Tb and T0). This came in accordance with other studies done under general anesthesia [16]. Inkyung Song et al. showed significant decrease in MAP, CO and SV during 2 to 12, 4 to 6 and 2 to 6-min period after tourniquet deflation [17]. In group C, after tourniquet release, there was a significant increase in the SV, COP, and CI at all intervals compared to baseline values. This could be explained by a compensatory mechanism in which an increase in cardiac inotropism leads to an increase in SV with subsequent increase in COP and CI to maintain normal Blood Pressure [18]. This state of concomitant decrease of MAP and increase in cardiac work might expose patients with poor cardiac reserve to risk of cardiac morbidity. Parmet et al. study showed a slight increase in CI without a change in MAP or SVRI after tourniquet release under general anesthesia [19]. Parmet et al. study differs from ours as their patients were geriatrics with cardiovascular diseases and all of the patients were transfused with one unit of packed RBCs just before tourniquet deflation. In group S, MAP and SVR were decreased at all intervals compared to base line values, but MAP and SVR were higher than that of group C at all intervals. Also, in group S, the SV, COP and CI were increased at all intervals compared to base line values but in comparison to group C, it was lower at all intervals. Tourniquet inflation causes ischemia to the wall of arteries, veins and capillaries with accumulation of ischemic vasodilator metabolites produced by anaerobic metabolism and lipid peroxidation rendering the blood vessels poorly reactive and dilated [20-22]. Releasing tourniquet will lead to abrupt fall in SVR with pooling of blood in to the dilated limb vasculature, to the extent that the lower limb would show immediate 10% increase in its girth [23]. Tourniquet deflation with the operated leg being elevated might cause an increase in venous return aided by gravitational forces leading to increase in filling volumes of the ventricles and stretching the cardiac myocytes thereby, increasing the preload with subsequent increase in force of ventricular contraction thus increasing SV and COP [24]. Massimo et al. study [25] showed that after 1 min of passive leg elevation in patients with normal right ventricular function (pre induction RV ejection fraction >45%) resulted in significant decrease in HR compared to leg in neutral position. There was significant increase in right ventricular end diastolic and systolic index with significant increase in both systolic systemic arterial/right ventricular pressure gradient and diastolic systemic arterial/right ventricular pressure gradient without significant increase in CI. This was different from our study which showed post deflation increase in CI in group S. The difference in results could be explained that their patients underwent myocardial revascularization for ischemic heart disease with 6 of 10 participants and 3 of 10 participants were on beta blockers and calcium channel blockers respectively who would affect cardiac contractility.

$ETCO_2$ significantly increased with concomitant increase in RR in both groups, which persisted for 15 min post tourniquet release with no difference between both groups. Other studies [26,27], have demonstrated significant increase in $ETCO_2$ up to 7 and 10 min post tourniquet deflation. The patients' ventilation was controlled with a fixed minute volume pre and post tourniquet release. They didn't mention the settings of minute volume given which might be high enough to wash CO_2 in a shorter time. Chawla et al. study showed an increase in $ETCO_2$ with return to pre deflation values within 5 min which might be attributed to limiting limb tourniquet application to only 30 min [28].

In the current study there was significant increase in lactate level and decrease in pH in both groups post deflation up to 15 min. which came in accordance with results of Iwama et al. study [29] regarding persistent high lactate level up to 15 min, but the difference in their duration of reduced pH values which lasted only for 5 min. This might be attributed to normalized $PaCO_2$ level at 5 min post deflation leaving only the metabolic element which was high lactate. Whereas in ours, 2 elements were persistently high up to 15 min, the metabolic and the respiratory elements (Lactate, $PaCO_2$) which were sufficient to reduce pH values.

In conclusion, thigh tourniquet deflation with operated leg in elevated position caused attenuation in the decrease of MAP and SVR as well as the compensatory increase in COP, CI and SV compared to tourniquet deflation with the operated leg in neutral position. Furthermore, it had no effect on the respiratory and metabolic changes.

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