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Hyperventilation after tourniquet deflation prevents an increase in cerebral blood flow velocity

Purpose: In this study we examined whether normocapnia maintained by hyperventilation after lower limb tourniquet deflation prevents an increase in cerebral blood flow velocity.

Methods: Thirteen patients, undergoing elective orthopedic surgery, requiring a pneumatic tourniquet around the lower extremity, were divided into two groups. In group 1, ventilation was controlled at tidal volume of 10 mL·kg^{-1} and respiratory rate of eight per minute after tourniquet release. In group 2, ventilation was controlled to maintain $P_{ET}CO_2$ between 30 and 35 mmHg after tourniquet release. Arterial blood pressure, heart rate, peak and mean middle cerebral artery (MCA) flow velocity, and arterial blood gas were measured every minute for ten minutes after tourniquet release. The MCA blood flow velocity was measured using Transcranial Doppler ultrasonography (TCD).

Results: In group 1, the maximum peak MCA flow velocity was 53 ± 6 cm·sec^{-1} (50 % ± 6% increase compared with pre- release value), and achieved 3 ± 0.4 min after tourniquet release. In group 2, there was no increase either in mean or peak MCA velocity after tourniquet release.

Conclusions: Normocapnia maintained by hyperventilation after tourniquet deflation prevents an increase in cerebral blood flow velocity.

Objectif : Vérifier si la normocapnie maintenue par l'hyperventilation après le dégonflage d'un garrot autour du membre inférieur empêche l'augmentation de la vitesse du flux sanguin cérébral.

Méthode : Treize patients, admis pour une chirurgie orthopédique nécessitant un garrot pneumatique autour du membre inférieur, ont été répartis en deux groupes. Après le relâchement du garrot, on note que : dans le Groupe 1, la ventilation était maintenue au volume courant de 10 ml·kg^{-1} et la fréquence respiratoire à huit par minute ; dans le Groupe 2, la ventilation était contrôlée pour maintenir la $P_{ET}CO_2$ entre 30 et 35 mmHg ; la tension artérielle, la fréquence cardiaque, la vitesse moyenne et maximale du flux de l'artère cérébrale moyenne (ACM) et les gaz du sang artériel étaient mesurés à chaque minute pendant dix minutes. La vitesse du flux de l'ACM a été mesurée par échographie-Doppler transcrânienne (DTC).

Résultats : Dans le Groupe 1, la vitesse maximale du flux de l'ACM a été de 53 ± 6 cm·sec^{-1} (50 % ± 6 % d'augmentation en comparaison des valeurs précédant la libération du garrot), et a été atteinte 3 ± 0,4 min après le garrot. Dans le Groupe 2, il n'y a pas eu d'augmentation de la vitesse moyenne ou maximale du flux de l'ACM après le garrot.

Conclusion : La normocapnie maintenue par l'hyperventilation après le dégonflage du garrot peut empêcher une augmentation de la vitesse du flux sanguin cérébral.

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PNEUMATIC tourniquets are often used around the extremities during orthopedic surgery to obtain a bloodless surgical field. Ischemic metabolites released after tourniquet deflation provoke several physiological alterations.[1] Decreases in arterial pH and $PaCO_2$ and increases in lactate, potassium, $PaCO_2$ and $P_{ET}CO_2$ immediately after tourniquet deflation have been reported.[1-3]

Hirst *et al.* reported that a transient increase in cerebral blood flow (CBF) occurred after tourniquet deflation.[3] This transient increase in CBF may have a deteriorating effect in patients with head-injury.[4-5]

Considering the high correlation between CBF and $PaCO_2$ tension,[3] the increase in CBF might be prevented by maintaining normocapnia after tourniquet release.

The purpose of this study was to assess whether normocapnia maintained by hyperventilation after tourniquet deflation can prevent the increase in cerebral blood velocity after tourniquet release.

Materials and methods

After obtaining approval of the ethics committee of our institution and written, informed consent, 13 patients, undergoing elective orthopedic surgery requiring the use of a tourniquet on the lower extremity, were studied. No patients had pulmonary, renal, or hepatic disease. It was also confirmed that no patient suffered from neurological disease or cerebral vascular disorders by preoperative brain computed tomography and ultrasonography.

In the first study, the time-course of the changes in MCA flow velocity and $PaCO_2$ were examined in five patients (group 1) without changing ventilation rate.

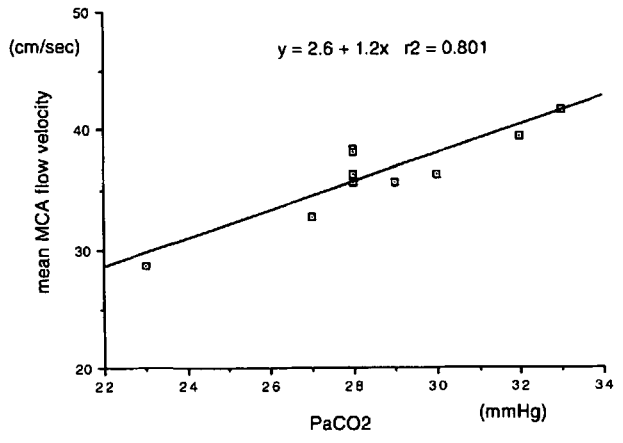

FIGURE 1 A representative plot between $PaCO_2$ and mean MCA flow velocity after tourniquet deflation. A close positive linear relationship is identified.

In the second study, the effects of normocapnia maintained by hyperventilation after tourniquet release were studied in the other eight patients (group 2).

All patients received 1 $mg \cdot kg^{-1}$ diazepam *po* an hour before anesthesia. Three electrodes were placed for electrocardiography (Hewlett Packard, Andover, MA). The left radial artery was cannulated with a 22-gauge indwelling catheter to monitor arterial blood pressure. Anesthesia was induced with 2 $mg \cdot kg^{-1}$ propofol, and 5 $\mu g \cdot kg^{-1}$ fentanyl, and 0.1 $mg \cdot kg^{-1}$ vecuronium. After tracheal intubation, ventilation was controlled by a mechanical ventilator with oxygen 50 % and N_2 50 %. The $P_{ET}CO_2$ was maintained between 30 and 35 mmHg with a tidal volume of 10 $mL \cdot kg^{-1}$ and respiratory rate of 8 bpm: $P_{ET}CO_2$ was monitored continuously by capnogram (Hewlett Packard). Anesthesia depth was maintained stable by infusion of propofol at 6-8 $mg \cdot kg^{-1} \cdot hr^{-1}$.

Transcranial Doppler ultrasonography

Flow velocity at the middle cerebral artery (MCA) was measured as previously described.[6] A probe (TC2-64; EME Co. Ltd., Uberlingen, Germany), using a 2 MHZ ultrasonic wave, was adjusted to detect MCA flow from the right temporal side. The Doppler signals were obtained through the right temporal window at a depth of 40-45 mm from the surface. The signal quality was determined by the characteristic high pitch sound and by the wave form of the sonogram display.

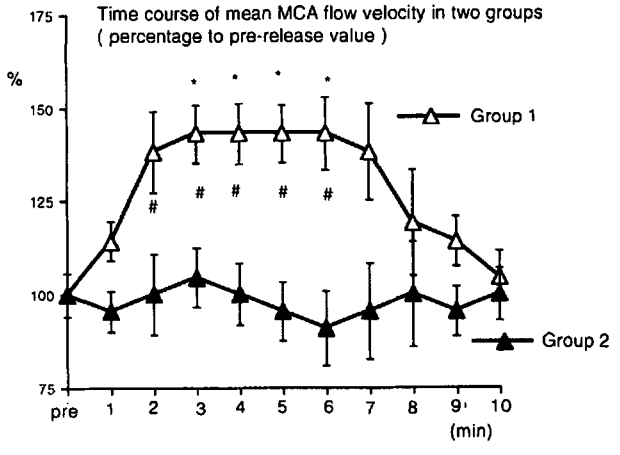

FIGURE 2 Time course of mean MCA flow velocity after tourniquet release (percentage to pre-release value).
* $P < 0.05$ compared with pre-release value.
† $P < 0.05$ compared with Group 2.

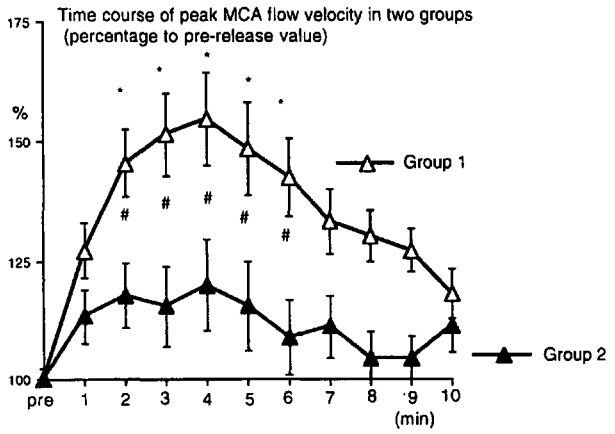

FIGURE 3 Time course of peak MCA flow velocity after tourniquet release (percentage to pre-release value).
* $P < 0.05$ compared with pre-release value.
† $P < 0.05$ compared with Group 2.

Study protocol

The extremity was exsanguinated with an Esmarch bandage and the pneumatic tourniquet was inflated to a pressure of 450 mmHg. Lactated Ringer's solution was infused at 5 $mL \cdot kg^{-1} \cdot hr^{-1}$. Arterial blood pressure, heart rate, peak and mean MCA flow velocity and arterial blood gas analysis were measured every minute for ten minute after the tourniquet release. Arterial blood was analyzed using a Stat Profile UltimaR (NOVA Biomedical Co., Boston, MA).

Group 1:Ventilation was controlled with a tidal volume of 10 $mL \cdot kg^{-1}$ and respiratory rate at 8 bpm throughout the study. In Group 2 ventilation was controlled with a tidal volume of 10 $mL \cdot kg^{-1}$ and respiratory rate 8 bpm prior to the tourniquet release. After tourniquet release, $P_{ET}CO_2$ was maintained between 30 and 35 mmHg by increasing the respiratory rate.

Data analysis

All data are expressed as means ± SEM. The results were analyzed by one-way repeated measure ANOVA, and post-hoc comparisons were assessed by Scheffe's test. Statistical significance was set at $P < 0.05$. Linear regression analysis was used for comparing changing in MCA blood flow velocity with changes in $P_{ET}CO_2$.

Results

No differences in demographic data were observed (Table I). Linear regression showed positive correlation between mean MCA flow velocity and $P_{ET}CO_2$ in all five patients ($0.86 \geq r^2 \geq 0.77$) (Figure 1). Table II (a) and (b), and Table III show time course of physiological variables of two groups. In group 1, the maximum $PaCO_2$ was 45 ± 0.8 mmHg (an increase of 27 % ± 5% compared with the pre-release value), and occurred 2 ± 0.4 min after tourniquet release. The maximum peak MCA flow velocity was 53 ± 6 $cm \cdot sec^{-1}$ (an increase of 50 ± 6% compared with the pre-release value), and occurred 3 ± 0.4 min after tourniquet release. Mean MCA flow velocity and peak MCA flow velocity were higher than the pre-release value from three to six minutes and from two to six minutes after tourniquet release, respectively. Mean arterial pressure (MAP) was lower than the pre-release value from one to seven minutes after release. Plasma lactate concentration was increased after the release and was stable for at least 10 min.

TABLE I Demographic data of the two groups

	group 1	*group 2*
Height (cm)	157 ± 4	158 ± 4
Weight (kg)	54.2 ± 2.5	56 ± 3.0
Age (yr)	53 ± 4	56 ± 5
Sex (M/F)	3:2	5:3
Tourniquet time (min)	67 ± 12	62 ± 10
Operative time (min)	81 ± 19	91 ± 16
Anesthetic time (min)	109 ± 21	111 ± 17

Value are means ± SEM

In group 2, there was no increase in either mean or peak MCA velocity after tourniquet release. Mean arterial pressure (MAP) was decreased and plasma lactate concentration was increased after tourniquet release, as in group 1.

Discussion

We prevented the increase in cerebral blood flow velocity by maintaining normocapnia immediately after tourniquet release. Flow velocity at MCA was measured by transcranial Doppler sonography. This is not a direct measurement of CBF. However, there are many reports describing the validity of MCA flow velocity as an index of CBF.[7] Thus, we considered that the alterations in MCA flow observed in this study reflect changes in CBF.

Our study showed that the increase in $PaCO_2$ lasted for six minutes after tourniquet release which was consistent with previous reports by Lynn *et al.* and Bourke *et al.*[2,8] In group 2 patients, hyperventilation for six minutes after tourniquet deflation prevented the increase in $PaCO_2$ and the increase of MCA flow. The value of peak MCA flow velocity in our study was smaller than that reported by Hirst *et al.*[3] This discrepancy may be attributable to the difference in anes-

TABLE II (a) The time course of physiological variables in Group 1

	pre-deflation	*time from the tourniquet deflation*				
time (min)		1	2	3	4	5
MAP(mmHg)	111 ± 4	88 ± 2*	90 ± 2*	92 ± 3*	92 ± 3*	92 ± 2*
HR(bpm)	75 ± 9	84 ± 8	80 ± 9	76 ± 10	72 ± 11	75 ± 9
$P_{ET}CO_2$ (mmHg)	31 ± 1	37 ± 1*	37 ± 1*	36 ± 1*	36 ± 1*	35 ± 1*
pH	7.446 ± 0.03	7.414 ± 0.03*	7.383 ± 0.02*	7.396 ± 0.03*	7.397 ± 0.03*	7.411 ± 0.03*
$PaCO_2$ (mmHg)	36 ± 3	42 ± 2*	45 ± 1*	42 ± 2*	43 ± 2*	42 ± 2*
PaO_2 (mmHg)	260 ± 12	238 ± 12*	259 ± 14	253 ± 13	264 ± 8	260 ± 11
lactate (mmol·L^{-1})	1.0 ± 0.1	1.8 ± 0.2*	2.1 ± 0.2*	2.0 ± 0.3*	2.1 ± 0.3*	2.0 ± 0.4*
Potassium (mEq·L^{-1})	3.8 ± 0.2	4.1 ± 0.2	4.2 ± 0.2*	4.0 ± 0.2	4.0 ± 0.2	3.9 ± 0.2
MCA flow velocity						
mean (cm·sec^{-1})	21 ± 3.0	24 ± 3.3	29 ± 3.7	30 ± 3.2*	30 ± 3.1	30 ± 4.3*
peak (cm·sec^{-1})	42 ± 3.6	42 ± 5.3	48 ± 7.3*	50 ± 8.0*	51 ± 8.2*	49 ± 8.0*
		time from the tourniquet deflation				
time (min)		6	7	8	9	10
MAP(mmHg)		94 ± 2*	95 ± 2*	101 ± 3	102 ± 3	105 ± 3
HR(bpm)		82 ± 7	76 ± 9	75 ± 9	74 ± 9	77 ± 9
$P_{ET}CO_2$ (mmHg)		34 ± 1	34 ± 1	34 ± 1	32 ± 1	32 ± 1
pH		7.413 ± 0.03*	7.414 ± 0.03*	7.425 ± 0.03	7.425± 0.03	7.428 ± 0.03
$PaCO_2$ (mmHg)		39 ± 2	40 ± 1	37 ± 2	37 ± 2	37 ± 2
PaO_2 (mmHg)		262 ± 12	258 ± 14	262 ± 14	264 ± 8	257 ± 13
lactate (mmol·L^{-1})		2.0 ± 0.3*	2.0 ± 0.4*	1.9 ± 0.3*	2.0 ± 0.4*	2.1 ± 0.5*
Potassium (mEq·L^{-1})		3.8 ± 0.2	3.8 ± 0.2	3.8 ± 0.2	3.9 ± 0.3	3.9 ± 0.3
MCA flow velocity						
mean (cm·sec^{-1})		30 ± 3.8*	29 ± 3.7	25 ± 2.7	24 ± 2.7	22 ± 1.9
peak (cm·sec^{-1})		47 ± 8.1*	44 ± 7.7	43 ± 7.5	42 ± 6.7	39 ± 7.0

(b) Peak MCA velocity and $PaCO_2$ after touniquet. release

Variables	*Mean ± SEM*
baseline $PaC0_2$ (mmHg)	36 ± 2
maximum$PaCO_2$ (mmHg)	45 ± 1
time to maximum $PaCO_2$ (min)	2 ± 0.4
maximum increase in $PaC0_2$ (%)	27 ± 5
baseline peak MCA velocity (cm·sec^{-1})	33 ± 3
maximum peak MCA velocity (cm·sec^{-1})	53 ± 6
time to maximum peak MCA velocity (min)	3 ± 0.4
maximum increase in peak MCA velocity (%)	50 ± 6

Value are means ± SEM *$P < 0.05$ compared with pre-deflation value

MAP: mean arterial pressure, HR:heartrate, $P_{ET}CO_2$:end-tidal CO_2

thetic method. Strebel *et al.* reported that inhalational anesthetics and propofol had different action on cerebral circulation.[9]

In group 1 subjects, there was a close relationship between the CO_2 accumulation in arterial blood and the acceleration of MCA flow after tourniquet release. The increase in $PaCO_2$ was compatible with the report by Bourke *et al.*, in which they documented that respiratory rate in spontaneously breathing patients increased after tourniquet release to compensate CO_2 accumulation.[8] Hirst *et al.* also reported that $PaCO_2$ and MCA flow velocity increased simultaneously after tourniquet release[3] but they did not demonstrate that maintaining normocapnia prevented the increase in MCA flow velocity. On the other hand, several reports documented that another factor may be responsible for the increase in CBF after tourniquet release. Laptook *et al.* in an animal study reported that increased blood lactate concentration induced an increase in CBF independently of changes in blood pH.[10] In our group 2 patients, the increase in CBF could be prevented by maintaining normocapnia immediately after tourniquet release. In this study, blood lactate concentration increased after tourniquet release to a value twice to pre-release value. Also, hyperventilation could prevent the respiratory acidosis, but not the metabolic acidosis. The metabolic acidosis persisted for more than 10 min after tourniquet release in both groups 1 and 2. Accordingly, our results demonstrated that the increase of $PaCO_2$ is likely to be the main cause of MCA flow increase after tourniquet release and that the other metabolic factors have no or minor roles in the increase of MCA velocity.

The transient increase in CBF after tourniquet release may have a detrimental effect in patients with

TABLE III The time course of physiological variables in Group 2

	pre-deflation	*time from the tourniquet deflation*				
time (min)		1	2	3	4	5
MAP (mmHg)	110 ± 7	89 ± 5*	89 ± 4*	87 ± 5*	88 ± 5*	88 ± 4*
HR (bepm)	87 ± 7	88 ± 4	84 ± 4	81 ± 3	80 ± 3	79 ± 4
$P_{ET}C0_2$ (mmHg)	33 ± 1	33 ± 1	33 ± 1	32 ± 1	32 ± 1	32 ± 1
pH	7.456 ± 0.01	7.414 ± 0.02*	7.406 ± 0.02*	7.411 ± 0.02*	7.414 ± 0.02*	7.417 ± 0.02*
$PaC0_2$ (mmHg)	35 ± 2	38 ± 3	39 ± 2	38 ± 2	38 ± 2	37 ± 2
$Pa0_2$ (mmHg)	203 ± 27	198 ± 24	223 ± 23	220 ± 22	227 ± 20	224 ± 20
lactate (mmol·L^{-1})	1.0 ± 0.1	1.8 ± 0.2*	2.0 ± 0.2*	2.2 ± 0.2*	2.2 ± 0.2*	2.1 ± 0.2*
Potassium (mEg·L^{-1})	3.6 ± 0.1	3.9 ± 0.1	3.9 ± 0.1	3.9 ± 0.1	3.8 ± 0.1	3.7 ± 0.1
MCA flow velocity						
mean (cm·sec^{-1})	22 ± 1.7	21 ± 1.9	22 ± 2.4	23 ± 3.7	22 ± 3.7	21 ± 3.6
peak (cm·sec^{-1})	45 ± 2.3	51 ± 5.9	53 ± 6.9	52 ± 8.6	54 ± 9.7	52 ± 9.6
		time from the tourniquet deflation				
time (min)		6	7	8	9	10
MAP (mmHg)		91 ± 5*	90 ± 5*	93 ± 6*	92 ± 6*	93 ± 5*
HR (bepm)		79 ± 5	79 ± 4	80 ± 5	78 ± 5	77 ± 5
$P_{ET}C0_2$ (mmHg)		32 ± 1	32 ± 1	32 ± 1	32 ± 1	32 ± 1
pH		7.423 ± 0.02*	7.425 ± 0.02*	7.425 ± 0.01*	7.427 ± 0.03	7.428 ± 0.03
$PaC0_2$ (mmHg)		37 ± 2	37 ± 2	36 ± 2	36 ± 2	36 ± 1
$Pa0_2$ (mmHg)		231 ± 23	224 ± 19	226 ± 21	224 ± 22	221 ± 26
lactate (mmol·L^{-1})		2.0 ± 0.2*	2.1 ± 0.2*	2.0 ± 0.2*	2.0 ± 0.2*	1.9 ± 0.3*
Potassium (mEg·L^{-1})		3.8 ± 0.1	3.8 ± 0.1	3.7 ± 0.1	3.7 ± 0.1	3.7 ± 0.1
MCA flow velocity						
mean (cm·sec^{-1})		20 ± 3.0	21 ± 2.6	22 ± 2.9	21 ± 2.3	22 ± 2.0
peak (cm·sec^{-1})		49 ± 8.1	50 ± 6.8	47 ± 5.4	47 ± 4.5	50 ± 5.4

Value are means ± SEM *$P < 0.05$ compared with pre-deflation value
MAP: mean arterial pressure, HR:heartrate, $P_{ET}CO_2$:end-tidal CO_2

cerebral complications, such as head injury. For example, Conatry *et al.* and Eldridge *et al.* reported that the increase in CBF could induce serious elevation of intracranial pressure in patients with head injury.[4,5] Therefore, it may be important to prevent an increase in CBF after tourniquet release in these patients. However, we did not examine patients with head trauma in this study. Thus, it is not clear whether patients with intracranial damage, who have already received hyperventilation to prevent the increase in intracranial pressure, show an increase in CBF after tourniquet release. Patients with severe head injury might respond differently to the tourniquet release. Further study is necessary to clarify the altered cerebrovascular reactivity after head trauma.

In conclusion, maintaining normocapnia by hyperventilation after tourniquet deflation prevents the increase in CBF velocity.

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