Use of a tourniquet in patients with sickle-cell disease

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Fifteen patients, 13 male and two female, known to be carrying the sickle-cell gene (12 HbSS and 3 HbAS), who were undergoing operations requiring a bloodless field, were included in the study. Of the 12 with HbSS, seven had haemoglobin A_1 component of between 11 and 27%, three had fetal haemoglobin ranging from 5.7 to 29% and the remaining two had increased haemoglobin A_2 concentrations suggesting a beta non-thalassaemia combination. All had a tourniquet applied to the appropriate limb and were given general anaesthesia with moderate hyperventilation throughout the procedure. The tourniquet inflation time was 61.7 ± 27.5 min. The mean PaO_2 remained above 200 mmHg, mean $PaCO_2$ was less than 37 mmHg, and pH ranged between 7.40 and 7.45. There were no clinically important changes in BP or ECG. All patients made uneventful recoveries and none developed sickle-cell crises. It is suggested that it is safe to use tourniquet in patients with sickle-cell disease provided optimum acid-base status and oxygenation are maintained throughout the procedure.

Quinze patients dont 13 hommes et 2 femmes, connus porteurs du gène d'hématie falciforme (12 HbSS et 3 HbAS) ont été inclus dans l'étude. Ils subissaient des interventions nécessitant un champ exsangue. Parmi les 12 HbSS, sept avaient une HbA_1 entre 11 et 27%, trois avaient une Hb foetale entre 5,7 et 29% et deux autres avaient une HbA_2 accrue suggérant une combinaision β non thalassémique. Tous ont eu un garrot placé sur le membre approprié et ont eu une anesthésie générale avec une hyperventilation modérée pendant l'intervention. Le temps de garrot a été de 61,7 ± 27,5 min. La PaO_2 moyenne est restée au-dessus de 200 mm de Hg, la PCO_2 moyenne inférieure à 37 mmHg et le pH entre 7,40 et 7,45. Il n'y a pas eu de changement cliniquement important de la pression artérielle et de l'ECG. Tous les patients se sont réveillés sans problème, aucun n'a eu de crise d'hémolyse. On suggère qu'il est inoffensif d'utiliser un garrot en cas de thalassodrépanocytose pourvu que l'état acido-basique et l'oxygénation optimum soient maintenus pendant l'intervention.

Key words
BLOOD: sickle-cell disease;
COMPLICATIONS: sickle-cell disease;
EQUIPMENT: tourniquets.

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The use of a tourniquet to provide a bloodless field during surgery is generally discouraged in patients who carry the sickle-cell gene[1,2] because it may lead to circulatory stasis, acidosis, and hypoxaemia; the triad of clinical conditions known to induce sickling. However, the literature shows no clear contraindication to the use of a tourniquet in such patients.[3-5]

In the Eastern Province of Saudi Arabia 15–27% of the population carry the sickle-cell gene[6] with nearly 2% SS homozygotes.[7,8] Sickle-cell disease in this area is reported to have a relatively benign clinical course attributable to a high level of fetal haemoglobin. But it is increasingly recognised that there is much variability in the clinical expression of the disease in this region. Considerable morbidity, including extremely painful crises and osteomylitis,[9-12] has been observed. These and other surgical conditions of the limbs may sometimes require a bloodless field for operation.

Therefore, this study was undertaken in this environment in order to determine whether the use of a tourniquet is harmful to patients carrying the sickle-cell gene.

Methods

Patients carrying the sickle-cell gene who were undergoing surgery on a limb requiring a bloodless field were included in the study. They received premedication with diazepam 0.15 $mg \cdot kg^{-1}$ *po* one and half hours before operation. After establishing an *iv* line for hydration during operation, the patients were pre-oxygenated for two minutes before induction of anaesthesia. Intraoperative monitoring included ECG, blood pressure, pulse rate,

end-tidal carbon dioxide, and pulse oximetry. The induction sequence was as follows: fentanyl 2 $\mu g \cdot kg^{-1}$, atracurium 0.5 $mg \cdot kg^{-1}$, thiopentone 5 $mg \cdot kg^{-1}$, followed by tracheal intubation. Maintenance anaesthesia was with isoflurane 0.5–1%, nitrous oxide 50% in oxygen, supplements of fentanyl and atracurium, and controlled mechanical ventilation which was adjusted to produce $P_{ET}CO_2$ of 30–35 mmHg.

After settling the patient on mechanical ventilation, the radial artery was cannulated for arterial blood sampling. A bloodless field was established using an Esmarch bandage and the tourniquet inflated to 250 mmHg for the upper limb, and 300 mmHg for the lower limb. At the end of the operation, the inflation time was recorded before the tourniquet was deflated. Wound dressing and/or plaster of paris was applied and the residual neuromuscular blockade was reversed with atropine and neostigmine.

Four sets of arterial blood samples were obtained from all patients as follows: after induction of anaesthesia but immediately preceding tourniquet application (control), 30 min after tourniquet inflation, one minute and one hour after tourniquet deflation. A venous sample was obtained with every arterial blood sample to determine the irreversibly sickled-cell count (ISC), which was defined as the number of irreversibly sickled cells per hundred red blood cells. Clinical end-points of pain were used to determine thrombotic episodes.

Haemoglobin genotypes HbSS and HbAS were defined as erythrocyte haemoglobin S contents of more than 70% and less than 40% respectively.

Analysis of the data was done with the SPSS/PC+ statistical package. The means were compared using Student's t test for paired variables.

Results

There were 15 patients, 13 male and two female. The age range was from 12 to 35 yr with a mean of 21.3 yr (I 8.0). The lowest haemoglobin concentration was 7 $g \cdot dl^{-1}$ and the highest was 13 $g \cdot dl^{-1}$. The haemoglobin genotype distribution was as follows: 12 SS and 3 AS. Of the 12 HbSS cases, seven had haemoglobin A_1 component of between 11 and 27%, three fetal haemoglobin ranging from 5.7 to 29% and the remaining two had raised haemoglobin A_2 concentration suggesting a beta non-thalassaemia combination (Table I). All the 12 SS patients had suffered painful crises and some had received multiple blood transfusions in the past. One of them (patient #6) had had splenectomy. No clinically significant changes were observed in the arterial blood pressure and the ECG. The duration of tourniquet application ranged from 35 to 115 min (mean 61.7 ± 27.5).

Table II shows the perioperative changes in arterial blood gases and the irreversibly sickled-cell count. The mean PaO_2 remained above 200 mmHg, mean $PaCO_2$ was less than 37 mmHg and the pH ranged between 7.40 and 7.45. Minimal change occurred between the pre-tourniquet $PaCO_2$ and the $PaCO_2$ one minute after tourniquet deflation ($P < 0.05$). Minimal differences also occurred in base excess which were of no clinical importance. The mean irreversibly sickled-cell count did not change. When the data were analysed separately for the 12 SS patients, the findings were the same. None of the patients developed sickle-cell crises and all made uneventful recovery.

Discussion

The local effects of two-hour tourniquet ischaemia in a human limb include progressive hypoxaemia, hypercarbia, lactic acid accumulation and consequent decrease in pH.[12] Systemic effects after tourniquet deflation are due to the uptake of the local products of anaerobic metabolism from the recently ischaemic limb into the general circulation. *In vitro* studies of the effects of deoxygenation on the sickle-cell show that progressive decrease in oxygen tension from 70 mmHg to 25 mmHg provokes reciprocal increase in sickling and that sickling of 5% of red cells occurs at a PO_2 of 40 mmHg and 90% at PO_2 of 25 mmHg.[13] These studies suggest that intravascular sickling during the use of a tourniquet in sickle-cell patients is a theoretical possibility. However, some retrospective studies have shown that no harmful effects occurred after the use of a tourniquet in patients with sickle-cell disease.[3-5]

There are many experimental and clinical reports of the systemic effects of tourniquet release in patients with normal haemoglobins.[14-19] These suggest that the systemic effects are usually minimal, transient and of no clinical significance. Our results with the abnormal haemoglobin-S patients confirmed this observation and we found no changes in blood gas results, except in the $PaCO_2$ and base excess.

It has been suggested that since there may be an acute increase in $PaCO_2$ after tourniquet deflation, a period of hyperventilation would expedite the return of $PaCO_2$ and pH to normal levels.[20] The administration of sodium bicarbonate prior to tourniquet release has also been advocated.[15] In this study the patients were not given bicarbonate but their lungs were moderately hyperventilated until the end of anaesthesia. No clinically important changes were observed in the blood gases or in the irreversibly sickled-cell count which is an index of haemolysis in sickle cell disease.[21,22]

We suggest that it is the chronicity and persistence, rather than the acuteness of changes in PaO_2 and/or pH which precipitate the *in vivo* sickling process. Our find-

TABLE I Clinical variables in 15 sickle-cell patients requiring bloodless field for various operations

SN	Age	Sex	$Hb \cdot dl^{-1}$	Electrophoresis % S	A_1	A_2	F	Tourniquet duration (minutes)	Diagnosis	Operation
1	16	F	11.3	89*	11	0	0	50	Acute osteomyelitis tibia	Drainage and biopsy
2	17	M	8.4	71*	0	0	29‡	35	Acute osteomyelitis tibia	Decompression and drainage
3	14	M	9.6	73*	27	0	0	40	Acute osteomyelitis tibia	Drainage and biopsy
4	12	M	10.9	79*	21	0	0	65	Septic arthritis knee	Drainage
5	23	M	12.3	82*	16	2	0	95	Fracture dislocation ankle	Internal fixation
6	16	M	11.3	93*	0	7†	0	30	Acute osteomyelitis, tibia	Decompression
7	19	M	10.9	79*	21	0	0	105	Fracture radius and ulna	Internal fixation
8	18	M	10.7	35	63	2	0	55	Compound fracture, tibia	Debridement and POP
9	40	M	11.7	31	66	3	0	115	Fracture shaft lower tibia	Internal fixation
10	28	M	7.8	90*	0	4.3†	5.7‡	50	Acute osteomyelitis	Decompression and biopsy
11	30	M	13	38	59	3	0	45	Acute osteomyelitis	Decompression and biopsy
12	17	M	9	81*	0	1	18‡	45	Acute osteomyelitis	Decompression and biopsy
13	14	F	11	88*	22	0	0	40	Septic arthritis elbow	Drainage and biopsy
14	35	M	10.6	80*	0	0	20‡	105	Synovial hypertrophy knee	Synovectomy
15	20	M	11.6	76*	22	0	2	50	Septic arthritis knee	Drainage

*Patients with SS genotype.
†SS patients with beta non-thalassaemia combination.
‡SS patients with high fetal haemoglobin.

TABLE II Summary of changes in arterial blood gases and irreversibly sickled-cell count ($n = 15$)

Arterial blood gas	Pre-tourniquet	30 min after tourniquet application	1 min after release of tourniquet	1 hr in recovery room
pH	7.4 ± 0.04	7.44 ± 0.05	7.45 ± 0.05	7.42 ± 0.04
$PaCO_2$	36.3 ± 3.4*	33.7 ± 6.8	34.1 ± 3.8*	37.6 ± 6.1
HCO_3 mEq · L^{-1}	23 ± 1.3	22 ± 4.5	22 ± 4.1	23 ± 3.1
Base excess mEq · L^{-1}	0.36 ± 1.52	-0.06 ± 2.48	-0.3 ± 2.45	-0.75 ± 1.3
PaO_2 mmHg	225 ± 79	242 ± 68	211 ± 56	202 ± 57
O_2 saturation %	99.4 ± 0.8	99.6 ± 0.6	99.5 ± 0.4	99.4 ± 0.8
Irreversibly sickling count %	1.2 ± 1.0	2.5 ± 2.0	2.1 ± 1.2	1.8 ± 1.6

*$P < 0.05$.

ings confirm the experience of Stein and Urbaniak[3] and we conclude that the use of a tourniquet in sickle-cell patients is not associated with harmful effects provided that optimum acid-base status and oxygenation are maintained throughout anaesthesia.

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