

Adrenal Medullary Secretion in Tourniquet Shock

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After tourniquet (4 hours) of the left femoral region of the dog, secretion rate of catecholamines in adrenal venous blood was studied by fluorometric method and the following conclusions were obtained.

1) Continuous tourniquet alone did not cause increase of adrenal medullary secretion. However, when the 4 hours' tourniquet was followed by reopening of blood circulation, secretion rate of adrenal medullary catecholamines gradually increased and blood pressure decreased. Since increase of secretion of adrenal medullary catecholamines was not at all seen after splanchnicotomy, increase of secretion of adrenal medullary catecholamines derives from the adrenal medullary secretory nerve center.

2) When release of tourniquet was immediately followed by high pressure oxygen therapy with 3 ATA · O₂ for 45 minutes, secretion of adrenal catecholamines did not increase nor did blood pressure decrease. The effect of high pressure oxygen therapy was examined in view of acid-base balance and this was considered due to the fact that advancement to acidosis was temporarily compensated by the decrease of PCO₂ and by the increase of pH value, and occurrence of shock was restrained. Accordingly, acidosis may be one of the important factors for the occurrence of tourniquet shock.

INTRODUCTION

In 1941, BYWATERS⁶⁾ first clearly recognized the "crush syndrome" and adequately described during the bombings of London in Ward War II. Individuals whose limbs were pinned and compressed by falling debris for several hours, followed by release of the compression, developed a syndrome of shock, myohemoglobinuria, and subsequent anuria, uremia, and death.

Since the shock produced by crush injuries was thought to be based on a mechanism of tourniquet shock, the tourniquet shock was studied in animal experiments for the elucidation of its complicated causes. It

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was experienced clinically that reopening of blood circulation is followed by typical hypotension in the cases of coarctation of the aorta and obstruction of the abdominal aorta. This phenomenon has been called declamping shock or declamping hypotension.

BAUE et al.¹⁴⁾ reported that the occurrence of such hypotensive state may be due to a temporary pooling of blood accompanying the decrease of vasomotor tone after reopening of blood circulation in the ischemic area of the lower extremities. In the ischemic area, metabolic acidosis is caused by the accumulation of anaerobic metabolites such as lactic acid and this acidosis intensifies the shock. However, when interruption of blood circulation is of short period, such a hypotensive phenomenon is temporary.

Generally, the occurrence of tourniquet shock is considered to be related to metabolic acidosis¹⁴⁾, hyperkalemia¹⁹⁾, loss of body fluid¹⁵⁾, production of active polypeptide⁸⁾, unknown toxic substance²⁰⁾, or development of acute renal insufficiency^{17,22)}. Recently, in hemorrhagic shock and endotoxin shock, marked excessive tension of the sympathetic nervous system has been observed by means of measuring catecholamines in the adrenal venous blood^{18,16,9)}.

The author produced tourniquet shock in dogs by declamping the clamped lower extremities, measured the changes of blood pressure and secretion of adrenal medullary hormones (adrenaline and noradrenaline) by drawing adrenal venous blood, and discussed the role of the sympathetic nervous system. Further, the author attempted high pressure oxygen therapy for tourniquet shock, which proved to be effective in the aspects of adrenal medullary hormones and acid-base balance.

MATERIALS AND METHODS

Adult mongrel dogs weighing 7 to 16 kg underwent 24 hours' fasting prior to the experiments.

General anesthesia was performed by intravenous injection of sodium 5-ethyl-5-(1-methylbutyl) barbiturate (Nembutal) 25 mg per kg body weight. Left lateral abdominal region was incised and the adrenal venous blood was collected by exposing the left adrenal vein extraperitoneally and inserting a small glass cannula. The arterial pressure was recorded by directly setting a mercurial manometer on the right brachial artery.

Tourniquet shock was produced by femoral clamping method. First, the medial side of the left thigh was incised, and the femoral artery and vein were exposed and were ligated by the use of hemostats. Subsequently the thigh except for the femoral artery and vein was firmly clamped with a steel wire measuring 1 mm in diameter and then the entire blood circulation except that in the femur was completely

interrupted.

Declamping of the thigh was made by simultaneous release of the steel wire and hemostats, and the resumption of blood circulation was confirmed macroscopically. The hyperbaric chamber was designed specially for the dog with the interior dimensions of 450 mm in diameter and 1000 mm in length. Each experiment was performed with 3 ATA · O₂ (ATA: atmosphere absolute) for 45 minutes by pressurization during the first 15 minutes and decompressing during the last 7 minutes.

A. Estimation of Catecholamines

For the estimation of adrenal catecholamines, 2 ml of adrenal venous blood was collected at one hour intervals and catecholamines in serum were estimated by the method of EULER et al¹¹⁾. Farrand Photoelectric Fluorometer (Model A-3) and Micro-Ammeter (CAT. No. 124351) were used.

Animals were divided into four groups as follows.

Group 1 : Four dogs were assigned to this group. The changes of blood pressure and adrenal catecholamines were measured at one hour intervals with the lapse of 7 hours after tourniquet of the left thigh.

Group 2 : Using six dogs, the left thigh was clamped for 4 hours and then released. Blood pressure was measured 3 hours after release of tourniquet. Adrenal venous blood was collected before tourniquet, before release, and 30, 60, 120 and 180 minutes after release. In 3 animals, transection of the femoral nerve was performed in the region of the exposed femoral artery and vein, and of the sciatic nerve upon incision of the gluteal region for the purpose of eliminating stimulation to the central nerve system, particularly algesthesia, from the region of tourniquet.

Group 3 : In four dogs, tourniquet and release of the thigh were performed after carrying out homolateral splanchnicotomy, and catecholamines were examined in the same manner as in Group 2.

Group 4: In five dogs, tourniquet was released after 4 hours. Immediately after the release, each animal was placed in the high pressure oxygen chamber and was given high pressure oxygen with 3 ATA · O₂ for 45 minutes. Adrenal venous blood was collected before tourniquet, before release and 60, 120 and 180 minutes after release.

B. Measurement of pH, PCO₂ and Base Excess

In the following three groups, arterial blood was collected through the cannula inserted into the right femoral artery, and pH, PCO₂ and base excess of the arterial blood were calculated from Siggard-Andersen curve nomogram by ASTRUP method^{1,2)}.

Group 5: In 8 dogs, the same operation as in Group 1 was performed

and arterial blood was collected for measurement of pH, PCO₂ and base excess before tourniquet and 4, 5, 6 and 7 hours after tourniquet.

Group 6: In 8 dogs, the same operation as in Group 2 was performed and arterial blood was collected before tourniquet, before release and 60, 120 and 180 minutes after release.

Group 7: In 6 dogs, the same operation as in Group 4 was performed and arterial blood was collected at the same intervals as in Group 4.

RESULTS

(Changes of adrenal catecholamines and mean arterial blood pressure)

Group 1: The changes of secretion rate of adrenaline and noradrenaline in adrenal venous blood collected at an hour's intervals and of mean arterial blood pressure are shown in Table 1.

Table. 1: Changes of the adrenal medullary secretion and mean arterial pressure after tourniquet (group 1, 4 dogs)

| | | before | | 1 hr. | 2 hr. | 3 hr. | 4 hr. | 5 hr. | 6 hr. | 7 hr. |
|------|-------|---------|------------|---------|---------|---------|---------|---------|---------|---------|
| Ad. | M. | 0.005 | Tourniquet | 0.010 | 0.015 | 0.005 | 0.0075 | 0.0075 | 0.010 | 0.0175 |
| | ±S.E. | ±0.0029 | | ±0.0058 | ±0.0119 | ±0.0029 | ±0.0048 | ±0.0025 | ±0.0041 | ±0.0111 |
| Nor. | M. | 0.0025 | | 0.005 | 0.010 | 0.005 | 0.005 | 0.0075 | 0.010 | 0.005 |
| | ±S.E. | ±0.0014 | | ±0.0029 | ±0.0058 | ±0.0029 | ±0.0029 | ±0.0048 | ±0.0000 | ±0.0029 |
| B.P. | M. | 128.8 | | 127.5 | 118.8 | 114.3 | 111.3 | 115.0 | 118.8 | 118.8 |
| | ±S.E. | ±5.2 | | ±7.5 | ±6.0 | ±6.7 | ±5.6 | ±6.4 | ±6.6 | ±8.3 |

Ad. : Adrenaline ($\mu\text{g}/\text{kg}/\text{min.}$)

Nor. : Noradrenaline ($\mu\text{g}/\text{kg}/\text{min.}$)

B.P. : mean arterial blood pressure (mmHg)

M±S.E. : mean value ± standard error

The mean arterial blood pressure before tourniquet being 128.8 ± 5.2 mmHg and the mean value 7 hours after tourniquet being 118.8 ± 8.3 mmHg, there was no significant change in value with the lapse of time.

The basic secretion rate of adrenaline before tourniquet being 0.005 ± 0.0029 $\mu\text{g}/\text{kg}/\text{min.}$ and the maximum value being 0.0175 ± 0.0111 $\mu\text{g}/\text{kg}/\text{min.}$ at the interval of 7 hours, there was no significant difference nor was any specific trend in secretion rate.

The basic secretion rate of noradrenaline being 0.0025 ± 0.0014 $\mu\text{g}/\text{kg}/\text{min.}$ and the maximum secretion rate being 0.01 ± 0.0058 $\mu\text{g}/\text{kg}/\text{min.}$ and 0.01 ± 0.00 $\mu\text{g}/\text{kg}/\text{min.}$ after 2 and 6 hours, respectively, significant increase was noted after 6 hours but it was unrelated to the change of blood pressure.

Table. 2: Changes of the adrenal medullary secretion and mean arterial pressure after release of tourniquet (group 2, 6 dogs)

| | | before | | 4 hr. | | 0.5 hr. | 1 hr. | 2 hr. | 3 hr. |
|------|-------------|-------------------|------------|-------------------|---------|-------------------|-------------------|-------------------|-------------------|
| Ad. | M. ±S.E. | 0.010 ±0.0052 | Tourniquet | 0.0183 ±0.0079 | Release | 0.0383 ±0.0142 | 0.0533 ±0.0148 | 0.1383 ±0.0322 | 0.1520 ±0.0329 |
| Nor. | M. ±S.E. | 0.0067 ±0.0033 | | 0.0167 ±0.0061 | | 0.005 ±0.0022 | 0.0283 ±0.0162 | 0.030 ±0.0089 | 0.036 ±0.0093 |
| B.P. | M. ±S.E. | 140.8 ±8.9 | | 125.0 ±9.1 | | 95.8 ±5.8 | 90.3 ±4.1 | 70.3 ±4.7 | 72.0 ±8.5 |

Ad. : Adrenaline ($\mu\text{g}/\text{kg}/\text{min.}$)Nor. : Noradrenaline ($\mu\text{g}/\text{kg}/\text{min.}$)

B.P. : mean arterial blood pressure (mmHg)

M±S.E. : mean value ± standard error

Group 2: The experimental results for Group 2 are shown in Table 2. Mean arterial blood pressure decreased in all cases with the lapse of time after release, the value 120 minutes after release being 56% as compared with that before release. The values before tourniquet, before release, 30, 60, 120 and 180 minutes after release were 140.8 ± 8.9 mmHg, 125 ± 9.1 mmHg, 95.8 ± 5.8 mmHg, 90.3 ± 4.1 mmHg, 70.3 ± 4.7 mmHg and 72 ± 8.5 mmHg, respectively. As compared with the values before tourniquet and release, the values 30, 60, 120 and 180 minutes after release showed decrease with significant difference.

The secretion rate of adrenal catecholamines generally tended to increase with the lapse of time. Adrenaline increased more rapidly than noradrenaline. The secretion rate of adrenaline 120 and 180 minutes after release being 0.1383 ± 0.0322 $\mu\text{g}/\text{kg}/\text{min.}$ and 0.152 ± 0.0329 $\mu\text{g}/\text{kg}/\text{min.}$, the increase from 0.0183 ± 0.0079 $\mu\text{g}/\text{kg}/\text{min.}$ before release showed statistically significant difference ($P < 0.01$). On the other hand, the secretion rate of noradrenaline showed a trend of slight increase which was not significant. The findings in this Group 2 were decrease of blood pressure down to 50% and marked increase of secretion rate of adrenal catecholamine, particularly of adrenaline.

In 3 animals, neurotomy of the homolateral sciatic nerve and femoral nerve was performed prior to tourniquet. However, there was no evident difference in the degree of increased secretion of adrenal catecholamines and change of blood pressure between the neurotomy group and non-neurotomy group.

Group 3: The changes of mean arterial blood pressure showed a trend similar to those in Group 2. The values before release and 30, 60, 120 and 180 minutes after release being 123.3 ± 7.8 mmHg, 93.8 ± 14.8 mmHg, 95.0 ± 11.4 mmHg, 93.3 ± 12.5 mmHg and 82.0 ± 12.7 mmHg respectively, the values 60, 120 and 180 minutes after release showed

evidently significant decrease as compared with the value before release. In contrast, the increase of secretion rate of adrenal catecholamines, both adrenaline and noradrenaline, was so minimal as to be hardly measurable (Table 3).

Table. 3 : Changes of the adrenal medullary secretion and mean arterial pressure after splanchnicotomy (group 3, 4 dogs)

| | | before | | 4 hr. | | 0.5 hr. | 1 hr. | 2 hr. | 3 hr. |
|------|-------------|-------------------|------------------------------------|---------------|---------|---------------|---------------|-------------------|---------------|
| Ad. | M. ±S.E. | 0.0025 ±0.0025 | Tourniquet + Splanchnicotomy | 0 | Release | 0 | 0 | 0.0025 ±0.0025 | 0 |
| Nor. | M. ±S.E. | 0 | | 0 | | 0 | 0 | 0 | 0 |
| B.P. | M. ±S.E. | 123.8 ±3.8 | | 123.3 ±7.8 | | 93.8 ±14.8 | 95.0 ±11.4 | 93.3 ±12.5 | 82.0 ±12.7 |

Ad. : Adrenaline ($\mu\text{g}/\text{kg}/\text{min.}$)

Nor. : Noradrenaline ($\mu\text{g}/\text{kg}/\text{min.}$)

B.P. : mean arterial blood pressure (mmHg)

M±S.E. : mean value ± standard error

Group 4: The results for Group 4 are shown in Table 4. Mean arterial blood pressure after release showed no significant change except for the trend of slight increase one hour after release. As to adrenal catecholamines, adrenaline increased slightly 180 minutes after release but there was no significant difference. In comparison with Group 2, decrease of blood pressure and increase of catecholamine secretion in this group were negligible.

Table. 4 : Changes of the adrenal medullary secretion and mean arterial pressure before and after OHP. (group 4, 5 dogs)

| | | before | | 4 hr. | | 1 hr. | 2 hr. | 3 hr. |
|------|-------------|------------------|------------|------------------|--|------------------|------------------|------------------|
| Ad. | M. ±S.E. | 0.004 ±0.0024 | Tourniquet | 0.010 ±0.0032 | OHP after Release (3 ATA.O ₂ .) (45min) | 0.012 ±0.0038 | 0.010 ±0.0045 | 0.032 ±0.0111 |
| Nor. | M. ±S.E. | 0 | | 0.004 ±0.0024 | | 0.006 ±0.004 | 0.010 ±0.0045 | 0.014 ±0.004 |
| B.P. | M. ±S.E. | 120.2 ±6.6 | | 116.0 ±8.3 | | 125.8 ±16.6 | 118.4 ±11.9 | 112.0 ±8.6 |

Ad. : Adrenaline ($\mu\text{g}/\text{kg}/\text{min.}$)

Nor. : Noradrenaline ($\mu\text{g}/\text{kg}/\text{min.}$)

B.P. : mean arterial blood pressure (mmHg)

M±S.E. : mean value ± standard error

OHP. : oxygen at high pressure

(Changes of PCO₂, base excess and pH)

Group 5: Table 5 shows the changes of PCO₂, BE and pH before and

after tourniquet. The respective values being 35.67~42.04 mmHg, -5.23~-5.80 mEq/L and 7.259~7.357, there were no significant changes throughout the experimental course.

Table. 5: Changes of the arterial pCO₂, pH and base excess after tourniquet (group 5, 9 dogs)

| | | before | | 4 hr. | 5 hr. | 6 hr. | 7 hr. |
|------------------|-----------|-------------|------------|-------------|-------------|-------------|-------------|
| pCO ₂ | M. ±S. E. | 42.04±3.67 | Tourniquet | 37.86±1.28 | 35.67±2.18 | 37.66±2.35 | 41.58±3.20 |
| B. E. | M. ±S. E. | -5.23±0.94 | | -5.63±1.04 | -5.74±1.23 | -5.80±1.49 | -5.78±1.76 |
| pH. | M. ±S. E. | 7.295±0.023 | | 7.357±0.019 | 7.324±0.025 | 7.304±0.017 | 7.336±0.026 |

pCO₂ : mmHg

B. E. : Base Excess (mEq/L)

M±S.E. : mean value ± standard error

Transient increase of PCO₂ and decrease of BE and pH were generally noted before tourniquet but this might have been due to intravenous anesthesia and fixing of the animals at right lateral position.

Group 6: The changes of PCO₂, BE and pH before and after release of tourniquet are shown in Table 6. PCO₂ which was 40.83±3.44 mmHg before release somewhat increased to 47.30±4.50 mmHg 3 hours after release but the increase was not significant.

Table. 6: Changes of the arterial pCO₂, pH and base excess after release of tourniquet (group 6, 8 dogs)

| | | before | | 4 hr. | | 1 hr. | 2 hr. | 3 hr. |
|------------------|----------|-------------|-----------------|-------------|---------|-------------|-------------|-------------|
| pCO ₂ | M. ±S.E. | 38.19±2.56 | Tourni- quet | 40.83±3.44 | Release | 46.13±6.09 | 47.23±7.32 | 47.30±4.50 |
| B. E. | M. ±S.E. | -6.13±1.16 | | -5.54±0.88 | | -9.04±1.41 | -8.54±1.38 | -10.46±1.33 |
| pH. | M. ±S.E. | 7.311±0.028 | | 7.319±0.021 | | 7.235±0.024 | 7.251±0.026 | 7.226±0.026 |

pCO₂ : mmHg

B. E. : Base Excess (mEq/L)

M±S.E. : mean value ± standard error

Base excess which was -5.54±0.88 mEq/L before release decreased immediately after release and reached the value of -9.04±1.41 mEq/L after one hour, -8.54±1.38 mEq/L after 2 hours and -10.46±1.33 mEq/L after 3 hours, showing progressive and significant decrease.

The value of pH being 7.319±0.021 before release gradually decre-

ased to 7.235 ± 0.024 after one hour, 7.251 ± 0.026 after 2 hours and 7.226 ± 0.026 after 3 hours. As compared with the value before release, the values one and 2 hours after release showed significant decrease ($P < 0.05$) and the value 3 hours after release showed remarkable decrease ($P < 0.01$).

In view of the changes of pH, PCO_2 and BE after release, acidosis seems to be advanced mostly due to metabolic factors.

Group 7: The results of the high pressure oxygen therapy are summarized in Table 7.

Table 7: Changes of the arterial pCO_2 , pH and base excess before and after OHP. (group 7, 6 dogs)

| | | before | | 4 hr. | | 1 hr. | 2 hr. | 3 hr. |
|---------|---------------|-------------------|-----------------|-------------------|-------------------------|-------------------|-------------------|-------------------|
| pCO_2 | M. \pm S.E. | 42.82 ± 2.17 | Tourni- quet | 38.95 ± 3.27 | OHP after Release | 32.73 ± 4.67 | 28.25 ± 2.51 | 26.90 ± 1.50 |
| B.E. | M. \pm S.E. | -6.03 ± 1.13 | | -5.13 ± 0.59 | | -6.83 ± 1.46 | -7.53 ± 1.29 | -9.52 ± 1.51 |
| pH. | M. \pm S.E. | 7.299 ± 0.021 | | 7.350 ± 0.018 | | 7.371 ± 0.031 | 7.365 ± 0.027 | 7.354 ± 0.027 |

pCO_2 : mmHg

B. E. : Base Excess (mEq/L)

M \pm S.E. : mean value \pm standard error

OHP. : oxygen at high pressure

PCO_2 which was 38.95 ± 3.27 mmHg before release gradually decreased after release reaching 32.73 ± 4.67 mmHg after one hour, 28.25 ± 2.51 mmHg after 2 hours and 26.90 ± 1.50 mmHg after 3 hours, showing progressive decrease with significant difference ($P < 0.05$).

Base excess which was -5.13 ± 0.59 mEq/L before release decreased after release reaching -6.83 ± 1.46 mEq/L after one hour, -7.53 ± 1.29 mEq/L after 2 hours and -9.52 ± 1.51 mEq/L after 3 hours. As compared with the value before release, those after 2 and 3 hours showed decrease with evidently significant difference.

The value of pH which was 7.350 ± 0.018 before release showed no significant change after release.

DISCUSSION

As has been reported in hemorrhagic shock¹⁸⁾ and endotoxin shock⁹⁾, it was demonstrated by the experiment of Group 2 that, in tourniquet shock also, decrease of blood pressure and hypersecretion of adrenal catecholamines were present after release of tourniquet. The increase of secretion was remarkable particularly with adrenaline rather than

noradrenaline.

WALKER et al.²³⁾ observed in dogs that adrenal medullary secretion increased by tissue trauma or pain of the femur. Secretion of adrenal catecholamines in Group 2 showed no evident difference between the dogs with neurotomy of the femoral and sciatic nerves and the non-neurotomy group.

In order to investigate whether the increase of adrenal medullary secretion after the release of tourniquet as observed in the present experiment was induced by way of direct stimulation of the adrenal gland by humoral factor or by way of nervous impulse from the adrenal medullary secretory center, the author resected the greater and lesser splanchnic nerves of dogs under tourniquet. In these animals (Group 3), the change of mean arterial blood pressure was almost the same as that in Group 2. Though the blood pressure gradually decreased and approached the shock level, increase of secretion of adrenal catecholamines never occurred (Table 3). The same result was reported by SUGIHARA²¹⁾ in our department. Increase of secretion of adrenal catecholamines accompanied by shock after release of experimental strangulation ileus disappeared after splanchnicotomy on the homolateral side.

Similarly, EGDAHL⁹⁾ reported that increase of secretion of adrenal catecholamines after intravenous injection of voluminous endotoxin in dogs disappeared after transection of the spinal cord of C-7. It was clarified that increase of secretion rate of adrenal catecholamines after release of tourniquet in animals without splanchnicotomy derives from the excitement of the adrenal medullary secretory nerve center.

Moreover, acidosis has been seen commonly in secondary shock and is said to be important, particularly in tourniquet shock^{14,4)}. In order to study the relation between the development of acidosis and adrenal medullary secretion, the author measured PCO_2 , pH and base excess in the arterial blood. Group 5, the control group, showed no definite changes in acid-base balance but Group 6 after release of tourniquet showed increase of PCO_2 and decrease of pH and BE, development of acidosis mostly due to metabolic factors (Table 6), and increase of adrenal medullary secretion (Table 2).

HIGASHI¹³⁾ reported that in metabolic acidosis produced by infusion of HCl in the dog. Adrenal medullary secretion rate increased progressively in parallel with the development of metabolic acidosis. A statistically significant increase in adrenal medullary secretion was observed at a pH level of 7.00-7.20. Interruption of the splanchnic nerves abolished an increase in adrenal medullary secretion at a blood pH level above 6.80, but did not at a pH level below 6.70. At the pH level below 6.60 the secretion rates of the denervated gland did not differ significantly from those of the innervated one.

In the present experiments, the increase of secretion at this blood pH level disappeared also upon splanchnicotomy (Table 3).

For the treatment of tourniquet shock, therapies similar to those for acute renal insufficiency such as blood dialysis with dialyzer and peritoneal perfusion have been devised²²⁾ in addition to the common therapy for hemorrhagic shock and bacterial shock. However, there has been no report that these therapies are definitively effective. For hemorrhagic shock and bacterial shock, influence of high pressure oxygen has been studied and reported to be effective^{3,5,7,10,12)}.

For the purpose of studying whether or not such a favorable effect is expected in tourniquet shock, the author performed high pressure oxygen therapy with 3ATA · O₂ for 45 minutes immediately after release of tourniquet. As the result, intensive acidosis and increased secretion of adrenal catecholamines were not observed (Table 4) nor did the animals fall into shock at least for 3 hours after release of tourniquet. However, decrease of BE which is a metabolic factor could not be prevented (Table 7).

It may not be appropriate to conclude directly from the results of the present experiment that acidosis is the cause of tourniquet shock. However, MEHL et al.¹⁴⁾ observed onset of intensive metabolic acidosis in the dog after release of tourniquet and concluded that the therapy with THAM (tromethamine) was quite effective.

In the present study also, high pressure oxygen therapy was effective, though temporarily, to prevent the development of acidosis as well as to restrain the increase of secretion of adrenal catecholamines.

These evidences suggest that metabolic and respiratory acidosis plays an important role in the development of tourniquet shock.

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