Effect of thoracic limb tourniquet ischemia on local and systemic acid-base blood gases in donkey

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Received: 13 November 1987

ABSTRACT

Effect of thoracic limb tourniquet ischemia of 90 min duration was investigated in 6 adult donkeys of either sex. Studies were also conducted up to 30 min after tourniquet release. No alterations in PCO₂, PO₂, pH, oxygen saturation and HCO₂ were noted in systemic arterial circulation up to 90 min of ischemia. However, tourniquet ischemia resulted in severe local venous acidemia with a significant increase in PCO₂ and a nonsignificant fall in PO₂ and oxygen saturation. The release of tourniquet after 90 min caused increase in local limb venous pH with an appreciable fall in PCO₂. Limb venous PO₂ and oxygen saturation increased significantly after tourniquet release indicating poor oxygen exchange and utilization up to 30 min.

Tourniquets used routinely for bloodless surgical procedures of distal limbs in domestic animals have been reported to be associated with complications like paralysis (Rudge 1974), rupture of muscles (Butler and Back 1958), tissue damage and metabolic changes (Provan et al. 1966, Strock and Majno 1969). The effects of tourniquet application on acid-base and blood gases are reported in cattle and buffaloes (Singh et al. 1982, Peshin et al. 1985), but such detailed studies in equidae are lacking. The present investigation was undertaken to observe the systemic and local effects of thoracic limb tourniquet application on acid-base and blood gases in donkey.

MATERIALS AND METHODS

Healthy adult donkeys (6) of either sex were used. The technique of tourniquet application and design of the experiment was same as detailed by Singh *et al.* (1982). The animals were premedicated with intramuscular injection of acepromazine hydrochloride (0.1 mg/kg body

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weight) and restrained in lateral recumbency. The carotid artery and local limb vein (radial vein) were exteriorized under local infiltration anaesthesia. Arterial and local limb venous blood samples were collected after allowing stability time of 30 min to establish normal base values. Rubber tourniquet was then applied above the elbow joint and the samples collected at 15, 30, 60, and 90 min. After 90 min, tourniquet was released and blood samples were collected at 5, 10, 20 and 30 min.

Samples for blood gas analysis were collected anaerobically in heparinized plastic syringes and were analysed for pH, PCO₂, PO₂, HCO₃ and oxygen saturation with a blood gas analyzer (pH/blood gas analyzer system 1303, USA) at 37°C.

Statistical analysis of data was done with student's t-test at 5% level of significance.

RESULTS

Tourniquet ischemia of 90 min duration caused no appreciable systemic alterations in acid-base and blood gases. However, there was a fall in local limb venous pH (pHL), which was significant

at 15 min onwards (Table 1). There was also significant increase in local venous PCO₂ (PLCO₂) at 30, 60 and 90 min. The HCO₃ concentration of the limb vein decreased nonsignificantly. The limb vein PO₂ (PLO₂) and oxygen saturation were significantly decreased throughout the

period of tourniquet application.

The release of tourniquet after 90 min caused increase in both systemic arterial and local limb venous pH. These were significant at 20 and 30 min for pHa and pHL, respectively, after the release of the tourniquet. The PLCO₂ values decreased appreciably even below the base values and failed to return towards normaly up to the end of observation. The tourniquet release resulted in marked increase in PLO₂ and oxygen saturation. The increase in PLO₂ was significant only up to 30 min after tourniquet release. The PaO₂ and oxygen saturation remained unaffected.

DISCUSSION

The arrest of circulation of an extremity is clearly unphysiological, and physiobiochemical changes during such period are bound to occur. Similar studies in adult cattle demonstrated significant increase in local and systemic pH, PLO₂ and oxygen saturation after release of tourniquet, which failed to return to base values even up to 150 min (Singh et al.

1982).

The local venous acidosis during tourniquet ischemia is mainly attributed to increase in PLCO₂ (Scott et al. 1979, Peshin et al. 1985). In the present study, the decrease in pHL was significant from 15 min onwards, while PLCO2 increased significantly from 30 min. The local venous acidosis, thus could be attributed to both significant increase in PLCO2 and nonsignificant decrease in LHCOs. However, the magnitude of fall in limb venous blood pH and rise in PCO₂ was less than reported earlier (Scott et al. 1979, Peshin et al. 1985). This difference may be ascribed to either difference in duration of tourniquet application or in species of the animals.

During tourniquet application, the

local limb venous PO₂ and oxygen saturation decreased significantly. The fall in PLO₂ was of much higher magnitude than reported in cattle (Singh et al. 1981, 1982) and buffaloes (Peshin et al. 1985). At low PO₂ values, increased capillary permeability to fluid and proteins has been reported (Webb 1965) and this could be one of the possible causes of production of oedema after tourniquet application (Wilgis 1971).

This study failed to show any remarkable alteration in systemic acid-base and blood gases during tourniquet application. It appeared that all the changes are limited to the affected limb and no systemic response is elicited. This agrees with the previous reports of Singh et al.

(1982) and Peshin et al. (1985).

The release of tourniquet did not result in complete readjustment of acidbase and blood gases parameters. Local venous pH and PCO2 showed a progressive increase and decrease up to 30 min of observation. The PLO2 and oxygen saturation also remained higher than the base values. This is contrary to the observations made earlier, where all the parameters have been reported to return near base values within 10 to 20 min of tourniquet release (Wilgis 1971, Scott et al. 1979). However, our findings agree with the observations of Singh et al. (1982). In addition to the local effects, systemic changes were also seen, the systemic arterial pH increased progressively after the release of tourniquet. This appeared to be of metabolic origin since PaCO₂ was not affected.

The increase in pHL after tourniquet release was mainly due to fall in PLCO₂, but a slight increase in HCO₃ also appeared to be a contributary factor. This agrees with the findings of Singh et al. (1982). However, the increase in systemic arterial and local venous pH was of lesser degree in our animals than in cattle (Singh et al. 1982), but was of same magnitude as in buffaloes (Peshin et al. 1985). This could be so because our observations were limited only up to 30 min after the release of tourniquet against abservations of 150 min in cattle

Table 1. Local and systemic acid-base and blood gas changes in donkey with limb tourniquet ischemia of 90 min duration (mean values ± SE)

	LH CO ₃	22.4 ±1.01	19.7 ±1.66	19.4 ±1.39	19.1 ±1.21	19.2 ±1.57	21.4 ±1.28	23.4 ±1.10	24.1 ±1.35	23.0 ±1.18
	ан соз	21.3 ±0.97	22.7 ±1.06	22.5 ±1.24	21.5 ±1.58	21.3 ±1.20	21.9 ±1.31	23.1 ±1.20	24.4 ±1.34	23.3 ±1.02
	SL O	76.9 ±2.59	28.1* ±5.43	21.2* ±3.27	14.8* ±4.19	12.7* ±3.51	91.7* ±1.73	91.6* ±1.20	90.5* ±1.13	89.6* ±1.82
	Sa O ₂	95.0 ±0.52	92.3 ±1.66	93.8 ±0.97	94.6 ±1.02	94.5 ±1.21	92.6 ±2.24	92.7 ±1.08	94.1 ±1.02	93.5 ±1.35
	PI O ₃ mm Hg	49.6 ±3.16	21.5* ±1.13	13.3* ±1.31	8.4* ±1.55	5.9* ±1.65	68.6* ±4.47	64.8* ±2.37	59.6 ±2.69	56.4 ±1.89
	Pa O ₂ mm Hg	83.4 ±2.83	77.8 ±1.74	80.9 ±1.90	81.9 ±3.63	82.4 ±6.01	82.2 ±3.12	78.5 ±2.88	76.8 ±2.76	81.4 ±3.77
	PI CO ₂ mm Hg	42.7 ±1.56	48.7 ±2.26	55·1* ±2.26	60.3* ±2.86	68.5* ±2.37	39.8 ±1.37	39.3 ±1.86	38.5	36.9 ±0.83
	Pa CO ₃ mm Hg	37.2 ± 0.58	38.5 ± 1.40	36.6 ± 1.11	36.7 ± 1.14	36.3 ± 1.25	37.7 ± 0.93	37.4 ± 1.08	35.7 ± 0.89	38.2 ± 0.88
	DHL	7.328 ±0.022	2.208* ±0.019	7.151* ±0.055	7.122* ±0.053	7.096* ±0.065	7.362. ±0.039	7.391	7.410	7.413* ±0.034
	рНа	7.358 ±0.009	7.386 ±0.010	7.404 ±0.019	7.403 ±0.020	7.396 ±0.015	7.399 ±0.015	7.409 ±0.018	7.414* ±0.018	7.401 ±0.016
	Stage of observation	Baseline	T 15	T 30	T 60	T 90	TR 5	TR 10	TR 20	TR 30

*, Values differ significantly (P < 0.05), when compared with base values. T, Time intervals; TR, time intervals after release of tourniquet.

Poor oxygen exchange in affected limb after tourniquet release recorded in the present animals, though agrees with the previous reports (Wilgis 1971, Singh et al. 1982. Peshin et al. 1985) was of lesser magnitude. The poor oxygenation of tissues under such state has been ascribed to little oxygen diffusion across the capillary bed (Manaberger et al. 1966). The increase in local vein oxygen saturation could possibly also be due to defective oxygen consumption by the temporarily damaged cells. The PLCO2 after tourniquet release also remained low and it can be presumed that the tissues respiration of the limb was affected.

The normal tissue oxygen exchange did not return up to 30 min after the release of tourniquet in our study. Therefore, tourniquet application for a duration more than 30 min should be used only in unavoidable circumstances. However, further controlled studies are needed to observe the time taken for return of all the parameters to base level after tourniquet release.

ACKNOWLEDGEMENTS

We thank the Dean, College Veterinary Medicine and Chairman, Department of Surgery and Obstetrics for providing necessary facilities. We also thank the Director, Saddam Hospital, Mosul for permitting to analyse blood samples and Miss M S AL-Shawany and Miss S Y AL-Bazaz for actual analysis.

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