RENAL TISSUE OXYGENATION FOLLOWING INDUCED HYPOTENSION IN DOGS


SUMMARY
Renal oxygenation was studied during induced hypotension in mongrel dogs, anaesthetized with 1–1.5% halothane in oxygen. Hypotension was induced with an infusion i.v. of sodium nitroprusside (SNP) 70 ± 17 μg kg⁻¹ min⁻¹ (mean ± SEM) or trimetaphan (TMP) 36 ± 16 μg kg⁻¹ min⁻¹, or by controlled arterial haemorrhage (45 ± 6 ml/kg of body weight). Mean arterial pressure (MAP), cortical (PcO₂) and medullary (PmO₂) tissue oxygen tensions, arterial (Pao₂), renal venous (PrvO₂), and urine (PuO₂) oxygen tensions were measured during the 40-min control, hypotension, and recovery periods. MAP was decreased to approximately 60% of the control value. PmO₂ decreased significantly (P<0.05) in all three groups while PcO₂ decreased significantly only in the haemorrhage group. Upon restoration of MAP to normal values, renal tissue oxygen tensions recovered in all groups, somewhat more rapidly in the SNP group. There were no significant differences in Pao₂, PrvO₂, and PuO₂ during control, hypotension and recovery periods in the three groups. Tissue oxygen tension values followed the changes in MAP, but were not hypoxic. It may be concluded that both SNP and TMP are hypotensive agents safe for the kidney.

The production of deliberate arterial hypotension during anaesthesia improves operating conditions and decreases blood loss (Enderby and Pelmore, 1951; Eckenhoff, 1978; Wildsmith et al., 1983). However, the advantages of this technique must be considered carefully in view of the sensitivity of renal function to hypotension and ischaemia. It is well established that renal function is dependent on the adequacy of renal perfusion pressure, a factor that is compromised in any procedure involving deliberate hypotension (Thurau, 1963; Koushanpour, 1976). Previous studies in man (Behnia, Siqueria and Brunner, 1978; Behnia et al., 1982) have demonstrated that endogenous creatinine clearance was significantly decreased during sodium nitroprusside (SNP) and trimetaphan camyslate (TMP)-induced hypotension, and that it returned to normal values within 1 h of the restoration of arterial pressure.

The present study was designed to assess the effects of deliberate hypotension on renal tissue oxygenation using SNP and TMP, two hypotensive agents used commonly in clinical practice. The results were compared with hypotension induced by controlled arterial haemorrhage, for which the mechanisms of action are comparatively well known (Selkurt, 1946; Dow and Fry, 1967; Bell and Harper, 1970).

MATERIALS AND METHODS

Experimental design. Fifteen mongrel dogs were allocated to one of three groups. The first group (five dogs) was made hypotensive by controlled arterial haemorrhage (45 ± 6 ml/kg of body weight, mean ± SEM), the second group (five dogs) was made hypotensive by continuous infusion i.v. of SNP at a rate of 70 ± 17 μg kg⁻¹ min⁻¹, and the third group (five dogs) was made hypotensive by infusion i.v. of TMP at a rate of 36 ± 16 μg kg⁻¹ min⁻¹. Mean arterial pressure (MAP) during hypotension was decreased to approximately 60% of the control value, and each animal was studied for three consecutive 40-min periods: control, hypotension and recovery.

Surgical procedures. Anaesthesia was induced with an infusion of thiopentone 8–10 mg kg⁻¹ i.v. The trachea was intubated without myoneural blockade, and anaesthesia was maintained with halothane in oxygen. Although the concentration of halothane varied from 1 to 1.5% in different animals, to maintain the same depth of anaesthesia and to yield MAP of about 100 mm Hg (70–110 mm Hg) during the control period, the halothane concentration remained essentially constant in a particular animal throughout the course of the experiment. Ventila-
tion was controlled, using a dual rate control Harvard Respirator pump (Model 607-D, Dover, MA). Arterial P\textsubscript{CO\textsubscript{2}} was maintained at 32 ± 1 mm Hg.

A 14-gauge Teflon catheter was placed in the abdominal aorta via a femoral artery and positioned at the level of the left renal artery (experimental kidney). This catheter was used to measure MAP via a Statham model P23d pressure transducer (Gould Inc., Oxnard, CA) and to collect arterial blood samples for gas analyses. In the haemorrhage group, the other femoral artery was catheterized for controlled arterial haemorrhage. A peripheral vein was cannulated also to allow administration of fluids and drugs. The rectal temperature was monitored and maintained around 37°C with an electric warming blanket. The animal was placed in the right lateral position and the left kidney was exposed through a flank incision. The left ureter was isolated and cannulated for urine collection and gas analysis. The left renal vein was catheterized with a 20-gauge Teflon catheter to permit sampling of renal venous blood. Two oxygen electrodes were implanted to a predetermined measured depth, one in the cortex 2 mm below the surface, and the other in the outer stripe of the outer medulla 6—7 mm below the surface where active solute transport is known to occur and oxygen consumption is expected to be maximum (Thaysen, Lassen and Munck, 1961; Torelli et al., 1966).

**Tissue oxygen measurement.** Cortical (P\textsubscript{CTO\textsubscript{2}}) and outer medullary (P\textsubscript{MTO\textsubscript{2}}) tissue oxygen tensions were measured polarographically using an electrode and measuring system kindly provided by International Biophysics Corporation (Irvine, CA). The electrode system consisted of a gold cathode embedded in a catheter and covered with a Hydrion membrane as described by Beran and colleagues (1978). A silver—silver chloride reference electrode was placed in muscle close to the kidney. The criteria for selection of electrodes were patterned after those of Schneiderman and Goldstick (1978). Before implantation, all oxygen electrodes were calibrated in 0.9% sodium chloride solution, at 37°C, saturated with gases of known oxygen tension (100% nitrogen, 12.3% oxygen and room air). In this manner, the current sensitivity, linearity, and response time of the oxygen electrodes were established, and only those electrodes which were linear and had 90% response times of less than 1 min were selected for implantation. The reliability of the oxygen electrodes was further verified using Cortill blood-gas control ampoules (Instrumentation Laboratories, Lexington, MA). All electrodes were calibrated before and after each experiment and found to yield practically the same results. Also, unlike those of Beran and colleagues (1978), our electrodes showed a negligible stirring artefact, indicating that the calibration was not changed when electrodes were placed in the tissues (Schneiderman and Goldstick, 1978). Furthermore, the stability of the electrodes was found to be satisfactory during the course of the experiments. To assess the ability of the implanted oxygen electrode to detect changes in arterial oxygen tension, at the end of each experiment Fi\textsubscript{O\textsubscript{2}} was changed from 99% to 21%, and back to 99%. Following the reduction in Fi\textsubscript{O\textsubscript{2}}, P\textsubscript{CO\textsubscript{2}} was decreased by approximately 40—45% within 60—90 s. However, P\textsubscript{MTO\textsubscript{2}} showed only 8—10% decrease in measured values. When the inspired oxygen was changed from 21% to 99%, both cortical and medullary tissue oxygen tensions increased and returned to previous values. Since the renal cortex has a higher blood flow than the medulla, these changes in tissue P\textsubscript{O\textsubscript{2}} might be expected.

Previous studies (Severinghaus et al., 1971; Bates, Feingold and Gold, 1975; Dent and Netter, 1976; McHugh, Epstein and Longnecker, 1979) have shown that halothane appears to increase polarographically measured oxygen tension. Since this artefact depends on the type of electrode, concentration of halothane, and possibly oxygen tension, we conducted an in vitro study to determine the magnitude of this artefact with the electrode and halothane concentrations used in the present study. We found that a halothane concentration of 1% resulted in an increase of 16.8 ± 6 (mean ± SD) mm Hg in polarographically measured oxygen tension at P\textsubscript{O\textsubscript{2}} 75 mm Hg. At P\textsubscript{O\textsubscript{2}} 150 mm Hg, the halothane artefact was 18.7 ± 6.5 mm Hg. The values of halothane artefact for 1.5% halothane concentration were about 1.5 times that found for 1% halothane concentration. Since the halothane concentration remained constant with time in each dog, the results of this in vitro study indicate that, in the range of oxygen tension and halothane concentration used in this study, the halothane artefact was essentially constant for a particular electrode in a particular animal. The average of halothane artefact for 1% halothane over the range of oxygen tensions of 75 and 150 mm Hg was 17.7 ± 6.2 mm Hg. This finding is consistent with that reported by Dent and Netter (1976), but is at variance with the results.
HYPOTENSION AND RENAL TISSUE OXYGENATION

TABLE I. Renal oxygenation in five dogs with haemorrhagic hypotension (mean values ± SEM; range in parentheses)

<table>
<thead>
<tr>
<th>Experimental periods</th>
<th>MAP (mm Hg)</th>
<th>Pao2 (kPa)</th>
<th>Prvo2 (kPa)</th>
<th>Pcto2 (kPa)</th>
<th>Pmto2 (kPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>102 ± 1</td>
<td>65.8 ± 3.1</td>
<td>11.2 ± 1.3</td>
<td>9.4 ± 0.5</td>
<td>22.0 ± 2.3</td>
</tr>
<tr>
<td></td>
<td>(100–105)</td>
<td>(31.9–69.8)</td>
<td>(8.0–15.7)</td>
<td>(8.0–11.3)</td>
<td>(10.9 ± 14.9)</td>
</tr>
<tr>
<td>Hypotension</td>
<td>62 ± 1</td>
<td>51.9 ± 6.5</td>
<td>9.2 ± 0.8</td>
<td>10.1 ± 1.2</td>
<td>10.4 ± 1.1</td>
</tr>
<tr>
<td></td>
<td>(60–68)</td>
<td>(29.9–66.5)</td>
<td>(7.1 ± 12.0)</td>
<td>(7.6 ± 14.0)</td>
<td>(6.1 ± 11.8)</td>
</tr>
<tr>
<td>Recovery</td>
<td>102 ± 6</td>
<td>59.7 ± 3.5</td>
<td>9.0 ± 0.8</td>
<td>9.0 ± 0.53</td>
<td>22.1 ± 1.9</td>
</tr>
<tr>
<td></td>
<td>(85–115)</td>
<td>(31.9–69.2)</td>
<td>(7.3–12.0)</td>
<td>(7.5–10.0)</td>
<td>(17.7–28.0)</td>
</tr>
</tbody>
</table>

40-min samples were averaged to represent the values for that period. Similar values from each dog in a given group were averaged and expressed as mean ± SEM.

Statistical analysis. Data were analysed for statistical significance using the paired, two-tailed t test between the values obtained for each time period (Snedecor, 1962). The values were considered significant if P values were less than 0.05. Any possible differences between the three groups were examined using analysis of variance on the measured control values, and on the changes in cortical and medullary P02.

RESULTS

Arterial haemorrhage. Table I presents a summary of the MAP, arterial oxygen tension (Pao2), renal venous oxygen tension (Prvo2), urine oxygen tension (Purvo2), renal cortex (Pcto2) and medullary tissue (Pmto2) oxygen tensions which were obtained from dogs made hypotensive by controlled arterial haemorrhage. Arterial haemorrhage resulted in an average decrease in MAP of 39%, which was returned to control value approximately 10 min after reinfusion of the lost blood. Renal Pcto2 and Pmto2 were reduced significantly (table II) during hypotension, but returned to control values upon restoration of the arterial pressure. There were no significant changes from control in Pao2, Prvo2, and Purvo2.

TABLE II. Comparison of the changes in arterial pressure, and changes in renal cortical and medullary oxygen tensions during the three different induced hypotensive procedures. Mean values ± SEM. By two-tailed t test: P < 0.05, **P < 0.01

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Number of dogs</th>
<th>MAP (mm Hg)</th>
<th>Pcto2 (kPa)</th>
<th>Pmto2 (kPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemorrhage</td>
<td>5</td>
<td>-39.9 ± 2.3**</td>
<td>-11.7 ± 2.2**</td>
<td>-4.50 ± 1.2*</td>
</tr>
<tr>
<td>SNP</td>
<td>5</td>
<td>-34.4 ± 4.3**</td>
<td>-5.6 ± 1.9*</td>
<td>-0.67 ± 0.47</td>
</tr>
<tr>
<td>TMP</td>
<td>5</td>
<td>-34.8 ± 5.3**</td>
<td>-10.7 ± 2.3**</td>
<td>-2.10 ± 1.2</td>
</tr>
</tbody>
</table>
and $PuO_2$ during the hypotension and recovery periods.

**Sodium nitroprusside infusion.** Table III presents a summary of the same variables in the five dogs receiving SNP. Shortly after the start of SNP administration, MAP decreased by an average of 36%. The MAP returned to control within 2–5 min of stopping the infusion. $PctO_2$ followed the changes in MAP and was reduced significantly (table III) during the period of hypotension. It returned to control somewhat faster than in the haemorrhage experiment. Unlike the haemorrhage group, there were no significant changes in $PmtO_2$ during SNP-induced hypotension (table III). There was no significant change from control in $PaO_2$, $PrvO_2$, and $PuO_2$ during the hypotension and recovery periods.

**Trimethaphan infusion.** Table IV presents a summary of the same variables in the five dogs made hypotensive by infusion of TMP i.v. After a short period of TMP administration, MAP decreased by an average of 37%, returning to control within about 15 min of stopping the infusion. Analogous to the haemorrhage and SNP experiments, changes in $PctO_2$ followed changes in MAP and TMP-induced hypotension caused a significant decrease in $PctO_2$ (table II). However, $PctO_2$ returned to control more slowly (15 min) than in the other groups. Unlike hypotension induced by haemorrhage, $PmtO_2$ was not changed significantly (table II) during TMP-induced hypotension. There were no significant changes from control in $PaO_2$, $PrvO_2$, and $PuO_2$ during the hypotension and recovery periods.

**Statistical analysis.** To verify that the three groups of dogs used in this study were in fact comparable, analysis of variance were calculated for the control values for all variables shown in tables I, III and IV. The results showed no statistical difference ($P > 0.35$) between each set of control values for the three groups, indicating that the dogs were all from the same population. Thus, any differences observed between the three types of induced hypotension cannot be the result of differences between the groups. Since analysis of variance on control values revealed no statistical difference between groups, we feel that the use of paired $t$ tests within each group is the most appropriate statistical test to ascertain the differences between the different experimental periods using each dog as its own control. Repeated-measure analysis of variance between

### Table III. Renal oxygenation in five dogs with sodium nitroprusside-induced hypotension. Mean values ± SEM (range in parentheses)

<table>
<thead>
<tr>
<th>Experimental periods</th>
<th>MAP (mm Hg)</th>
<th>$PaO_2$ (kPa)</th>
<th>$PrvO_2$ (kPa)</th>
<th>$PuO_2$ (kPa)</th>
<th>$PctO_2$ (kPa)</th>
<th>$PmtO_2$ (kPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>95 ± 7</td>
<td>51.9 ± 2.7</td>
<td>13.0 ± 1.3</td>
<td>10.5 ± 0.67</td>
<td>21.2 ± 2.5</td>
<td>12.2 ± 0.27</td>
</tr>
<tr>
<td></td>
<td>(80–110)</td>
<td>(45.9–61.2)</td>
<td>(10.6–17.3)</td>
<td>(8.7–12.4)</td>
<td>(13.3–27.7)</td>
<td>(7.7–17.3)</td>
</tr>
<tr>
<td>Hypotension</td>
<td>60 ± 3</td>
<td>48.4 ± 4.1</td>
<td>10.9 ± 0.67</td>
<td>11.3 ± 0.52</td>
<td>10.5 ± 1.6</td>
<td>10.1 ± 1.1</td>
</tr>
<tr>
<td></td>
<td>(53–70)</td>
<td>(41.2–59.9)</td>
<td>(9.4–13.3)</td>
<td>(10.0–12.4)</td>
<td>(6.3–15.7)</td>
<td>(6.4–12.4)</td>
</tr>
<tr>
<td>Recovery</td>
<td>89 ± 5</td>
<td>51.2 ± 4.7</td>
<td>10.0 ± 0.93</td>
<td>9.8 ± 0.4</td>
<td>19.2 ± 3.9</td>
<td>10.9 ± 1.5</td>
</tr>
<tr>
<td></td>
<td>(72–100)</td>
<td>(35.3–63.2)</td>
<td>(8.0–12.6)</td>
<td>(8.7 ± 10.6)</td>
<td>(15.0–21.8)</td>
<td>(7.1–16.0)</td>
</tr>
</tbody>
</table>

### Table IV. Renal oxygenation in five dogs with trimethaphan-induced hypotension. Mean values ± SEM (range in parentheses)

<table>
<thead>
<tr>
<th>Experimental periods</th>
<th>MAP (mm Hg)</th>
<th>$PaO_2$ (kPa)</th>
<th>$PrvO_2$ (kPa)</th>
<th>$PuO_2$ (kPa)</th>
<th>$PctO_2$ (kPa)</th>
<th>$PmtO_2$ (kPa)</th>
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<td>(7.1–16.0)</td>
</tr>
</tbody>
</table>
experimental periods for each dog indicated that the within variance was minimal compared with the variance between periods because the measured values during each of the three periods remained essentially constant. Furthermore, since $P$ values shown in table II suggest that the various types of induced hypotension produced different effects, we performed analysis of variance to test this for the observed changes in cortical and medullary $PO_2$. The results showed that the three types of induced hypotension had similar effects on cortical $PO_2$ ($F=0.47, P=0.64$) and on medullary $PO_2$ ($F=0.76, P=0.49$).

**DISCUSSION**

The purpose of this study was to compare renal tissue oxygenation during hypotension induced by three methods namely, infusion of SNP i.v., infusion of TMP i.v., and controlled arterial haemorrhage. A decrease in mean arterial pressure of around 40% resulted in a significant decrease in $Pc_{O_2}$ in all three groups (table II), and a significant decrease in $Pm_{O_2}$ in the haemorrhagic group, but insignificant changes within the SNP and TMP groups. On the other hand, analysis of variance showed that the three types of induced hypotension had similar effects. This apparent difference between the two statistical analyses is attributable to the small number of animals in each group and the inherent variability in the experimental measurements. Upon restoration of arterial pressure to normal, the pattern of recovery of renal tissue oxygenation was different in the three groups. We observed that the SNP group recovered somewhat faster than the haemorrhage group, and that the TMP group recovered somewhat more slowly than the haemorrhage group, probably because of the ganglionic blocking effect of TMP.

Depressed renal function following haemorrhagic hypotension has been well documented. Previous studies have established clearly the autoregulation of renal blood flow in response to changes in renal artery perfusion pressure (Thurau, 1963), while others have suggested that, during acute haemorrhage, autoregulation of renal blood flow may be absent (Selkurt, 1946; Dow and Fry, 1967). Since haemorrhage is known to produce marked stimulation of sympathetic activity, it has been suggested that the absence of blood flow autoregulation during haemorrhagic hypotension might be in part a result of sympathetic vasoconstriction of the renal vessels. This suggestion has been verified by Bell and Harper (1970). It is conceivable that decreases in renal blood flow secondary to hypotension might mediate the significant reduction in $Pm_{O_2}$ in the haemorrhagic group, possibly as a result of increases in renal vascular resistance secondary to haemorrhage-induced stimulation of the sympathetic and renin-angiotensin systems. During both SNP- and TMP-induced hypotension we found no change in either $Pm_{O_2}$ or $Pc_{O_2}$. This finding is in agreement with clinical observations in man (Behnia et al., 1982) which showed that urine $PO_2$ was not changed and that kidney oxygenation was not compromised. However, the present findings fail to support our previous suggestion in man (Behnia, Siqueria and Brunner, 1978) that urine $PO_2$ is a possible indicator of medullary tissue $PO_2$.

SNP is a short-acting, direct vasodilator, and has no known direct effects on myocardial contractility, and the central or autonomic nervous systems (Tinker and Michenfelder, 1976). Discontinuation of SNP infusion would therefore be expected to result in immediate and complete recovery, as seen in the present study. Moreover, although the infusion of SNP has been shown to stimulate renin release in normotensive man when mean arterial pressure is decreased to less than 70 mm Hg, renal vein renin returns to normal values within 15–30 min of stopping the SNP infusion, and of restoration of arterial pressure (Tinker and Michenfelder, 1976). In this regard, our findings are consistent with the known pharmacological properties of SNP.

Slower recovery of $Pc_{O_2}$ following cessation of TMP infusion may be explained by the following mechanisms. Administration of TMP causes a decrease in systemic arterial pressure, a decrease in splanchnic blood flow and increased intrarenal vascular resistance (Moyer, McConn and Morris, 1955). The increase in splanchnic resistance may be a result of the stimulation of the renin–angiotensin system secondary to the TMP-induced hypotension. We would expect that, with the passage of time, renal function would return to normal when the circulating renin–angiotensin has returned to normal values. Another possible explanation for the observed delay in the recovery of renal haemodynamics following cessation of TMP infusion might be the selective action of TMP on the splanchnic blood flow, or the possibility of histamine release following TMP-induced hypotension in dogs.

A major aim of the present study was to assess...
renal tissue oxygenation as an index of renal function, during induced hypotension, by means of measuring tissue, renal arterial, venous and urine oxygen tensions. Continuous measurements of tissue oxygen tension in both cortex and medulla provided a direct index of the viability of renal function during induced hypotension. As seen in tables I–IV, changes in tissue oxygen tension provide some information about the availability of oxygen during hypotension and recovery in the three groups.

In conclusion, the haemodynamic effects of induced hypotension in the three groups were similar, although the patterns of recovery were somewhat different. Despite this, direct measurement of tissue oxygen tension showed no evidence of marked renal hypoxia which could be detrimental to renal function. On this basis, we feel that both SNP and TMP are safe agents for inducing hypotension in clinical practice, although SNP might be considered a more desirable agent because of somewhat more rapid return of tissue oxygenation toward normal after its discontinuation. However, the risk of toxicity from the release of cyanide with administration of large doses of SNP should be kept in mind.

ACKNOWLEDGEMENTS

The authors thank Ms Nancy Murphy and Mr Tulip Shah for their excellent technical assistance, and Mr John F. O’Riordan for assistance with statistical analysis. We also thank Ayerst Laboratories for their generous gift of halothane and desired agent because of somewhat more rapid measurements caused by halothane.

REFERENCES


OXYGENATION DU TISSU RENAL APRES UNE HYPOTENSION PROVOQUEE CHEZ LE CHIEN

RESUME

Nous avons eudit l'oxygenation re nale au cours de l'hypotension provoquee chez des chiens anesthesies avec l 1 -1,5 % d'halothane dans l'oxygene. L'hypotension etait induite par une perfusion i.v. de NPS (70 ±17 µg kg⁻¹ min⁻¹, moyenne ± SEM) ou de TMP (36 ±16 µg kg⁻¹ min⁻¹) ou par une hémorragie arterielle controlee (45 ±16 ml kg⁻¹ de poids corporel). La pression arterielle moyenne (PAM), les pressions partialles tissulaires en oxygene corticale (Pco₂), et medullaires (Pco₂), les pressions partialles en oxygene arterielle (Pao₂), dans la veine re nale (Pvo₂) et urinaire (Pvo₂) ont ete mesurees au cours d'une periode controle de 40 min, des periodes d'hypotension et d'reveil. La PAM diminuait jusqu'a environ 60% de la valeur controle. La Pco₂ diminuait significativement (P<0.05) dans les trois groupes alors...
que la $P_{\text{mtO}}$ ne diminuait significativement que dans le groupe “hémorragie”. Lorsque la PAM revenait aux valeurs contrôle, les pressions partielles tissulaires rénales d’oxygène redevenaient normales dans tous les groupes plutôt plus rapidement dans le groupe NPS. Il n’y avait pas de différences significatives de $P_{\text{O}}$, $P_{\text{T}}$ et $P_{\text{U}}$ aux différentes périodes contrôle, hypotension et réveil dans les trois groupes. Les valeurs des pressions partielles tissulaires d’oxygène variaient dans le même sens que la PAM sans être hypoxiques, ce qui nous fait penser que le NPS et le TMP sont tous deux des agents hypotenseurs sans danger pour le rein.

NERENGewebe-Oxygenierung nach induzierter Hypotension beim Hund

ZUSAMMENFASSUNG

Bei Bastardhunden, die mit 1-1,5% Halothan in Sauerstoff narkotisiert wurden, wurde in induzierter Hypotension die Nierengewebsexzision untersucht. Die Hypotension wurde mit intravenöser Infusion von SNP (70 ± 17$\mu$g$kg^{-1}min^{-1}$ Mittelwert ± SEM) oder TMP (36 ± 16$\mu$g$kg^{-1}min^{-1}$) oder kontrolliertem arteriellem Aderlaß (45 ± 6 ml$kg^{-1}$) eingeleitet. Während 40 Minuten Kontrolle, Hypotension und Erholungsphase wurden der mittlere arterielle Druck (MAP), kortikale ($P_{\text{cto}}$) und medulläre ($P_{\text{mtO}}$) Gewebesauerstoffspannungen, arterielle ($P_{\text{aO}}$), Nierenvenen- ($P_{\text{vO}}$) und Urin- ($P_{\text{uO}}$) Sauerstoffspannung gemessen. MAP war auf annähernd 60% des Ausgangswertes reduziert. Bei allen drei Gruppen fiel $P_{\text{cto}}$ signifikant ($P<0,01$), während $P_{\text{mtO}}$ nur bei der Aderlaß-Gruppe signifikant absank. Nach Wiederanstieg von MAP auf Normalwerte erholte sich die Sauerstoffspannung im Nierengewebe in allen Gruppen, bei der SNP-Gruppe etwas schneller. In der Kontrollphase, Hypotension und Erholungsphase gab es zwischen allen drei Gruppen keine signifikanten Unterschiede in $P_{\text{cto}}$, $P_{\text{vO}}$ und $P_{\text{uO}}$. Die Werte für Gewebesauerstoffspannung folgten den Veränderungen des Blutdrucks, aber waren nicht hypoxisch. SNP und TMP scheinen also für die Niere ungefährliche hypotensive Substanzen zu sein.

OXIGENACION DEL TEJIDO RENAL DESPUES DE HIPOTENSION INDUCIDA EN PERROS

SUMARIO

Se estudió la oxigenación renal durante la hipotensión inducida en perros cruzados anestesiados con halotano al 1-1,5% en oxígeno. Se indujo la hipotensión mediante una infusión i.v. de 70 ± 17$\mu$g$kg^{-1}min^{-1}$ (promedio ± SEM) de SNP o 36 ± 16$\mu$g$kg^{-1}min^{-1}$ de TMP o mediante hemorragia arterial controlada (45 ± 6 ml/kg de peso corporal). La presión arterial media (MAP), las tensiones de oxígeno del tejido cortical ($P_{\text{cto}}$), del tejido medular ($P_{\text{mtO}}$), las tensiones de oxígeno arterial ($P_{\text{aO}}$), de la vena renal ($P_{\text{vO}}$) y de la orina ($P_{\text{uO}}$) fueron medidas durante un periodo de control de 40 min, durante la hipotensión y los periodos de recuperación. La MAP disminuyó hasta aproximadamente un 60% del valor de control. El $P_{\text{cto}}$ bajo de manera significante ($P<0,05$) en los tres grupos mientras que el $P_{\text{mtO}}$ disminuyó sensiblemente sólo en el grupo de hemorragia. Después de restaurar la MAP a valores normales, las tensiones de oxígeno del tejido renal se recuperaron en los tres grupos, algo más rápido en el grupo con SNP. Hubo diferencias significativas en el $P_{\text{cto}}$, el $P_{\text{vO}}$ y el $P_{\text{uO}}$ durante el periodo de control, de hipotensión y de recuperación en los tres grupos. Los valores de la tensión de oxígeno de los tejidos siguieron los cambios de la MAP, pero no fueron hipoxicos, lo que nos hace llegar a la conclusion que tanto el SNP como el TMP constituyen agentes hipotensores sin riesgo para los riñones.