EFFECT OF XYLAZINE-KETAMINE ON ARTERIAL BLOOD PRESSURE, ARTERIAL BLOOD PH, BLOOD GASES, RECTAL TEMPERATURE, HEART AND RESPIRATORY RATES IN GOATS

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Abstract

The effects of xylazine-ketamine administration on arterial blood pressure, arterial blood pH, blood gases, rectal temperature, and heart and respiratory rates were recorded in 5 healthy female goats weighing 17-29 kg. Xylazine (0.2 mg/kg, i.m.) was administered 15 min prior to ketamine (10 mg/kg, i.v.). All baseline measurements were taken before the xylazine administration and were repeated at 5, 15, 30, 45, and 60 min intervals after induction of anaesthesia with ketamine. It was found that heart rate decreased at 15 to 60 min and rectal temperature decreased significantly at 30 to 60 min but respiratory rate did not change significantly. Mean arterial blood pressure declined significantly at 15 to 60 min after anaesthesia. PaO₂ did not change significantly but PaCO₂ values increased significantly at 5, 15, and 60 min. Values of pH decreased significantly at 5 and 15 min. According to this study, xylazine-ketamine combination is responsible for declined arterial blood pressure, bradycardia, increased PaCO₂, decreased pH and hypothermia in anaesthetized goats.

Key words: goats, anaesthesia, xylazine, ketamine, physiological functions.

Ketamine can be used for anaesthesia in sheep and goats without fear of convulsions. Muscle relaxation is poor, but is improved by sedatives such as diazepam or xylazine (8). Ketamine has been acclaimed on account of its safety and its favorable effects upon cardiovascular and respiratory systems and because of its sympathomimetic and antiarrhythmic properties, it is useful in poor-risk and hypovolaemic patients (13). Xylazine hydrochloride is a typical α₂-adrenoreceptor agonist of the non-opioid group, having analgesic, sedative and muscle relaxant effects and is used commonly in clinical practice (16). Sheep and goats are ideally suited to local anaesthetic techniques under sedation or manual restraint (21). Ketamine is widely applied in goat anaesthesia and can be used on its own, or in combination with xylazine or diazepam (8, 21), in order to minimize or eliminate some unpleasant effects such as high muscle tone, trembling, increase in body temperature, and in intraocular and arterial pressure (11, 12, 16-18, 21).

The aim of this study was to evaluate the effects of xylazine and ketamine administration on arterial blood pressure, blood gases, arterial pH, heart and respiratory rates and rectal temperature in goats.

Material and Methods

This study was carried out on 5 healthy female Iranian goats weighing 17-29 kg. The goats were placed in left lateral recumbency for measuring arterial blood pressures and collecting blood samples. Arterial catheters flushed with 2/1000 heparin solution were placed using local anaesthesia into the right carotid artery via a 5 cm skin incision. Xylazine (0.2 mg/kg i.m.) was injected 15 min prior to the administration of ketamine (10 mg/kg, i.v.). All baseline measurements were taken before the xylazine administration and were repeated at 5, 15, 30, 45 and 60 min intervals after induction of anaesthesia with ketamine. Heart and respiratory rates were recorded with a stethoscope and counting thorax respiratory movements/min. Rectal temperature was taken with a digital thermometer. Baseline heart and respiratory rates and rectal temperatures were measured prior to the placement of an arterial catheter under lidocaine induced local anaesthesia. The blood samples were kept on ice and blood gases (PaO₂, PaCO₂) and pH were measured by a blood gas and acid-base analyzer within 60 min. Paired-samples T test was used for the analysis of the data and P value of less than 0.05 was considered to be statistically significant.

Results

Serial changes of heart and respiratory rates, rectal temperature, arterial blood pressure, blood gases (PaO₂ and PaCO₂) and pH values are presented in Table 1.
Table 1
Mean physiological values determined in goats before and after treatment
with xylazine (0.2 mg/kg, i.m.) and ketamine (10 mg/kg, i.v.)

<table>
<thead>
<tr>
<th>Anaesthesia time (min)</th>
<th>Values</th>
<th>Baseline</th>
<th>5</th>
<th>15</th>
<th>30</th>
<th>45</th>
<th>60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathing rate (min⁻¹)</td>
<td></td>
<td>20.2± 2.86</td>
<td>22.4 ± 9.60</td>
<td>25 ± 15.22</td>
<td>22.6 ± 15.38</td>
<td>19 ± 10.90</td>
<td>17.1 ± 5.45</td>
</tr>
<tr>
<td>Heart rate (min⁻¹)</td>
<td></td>
<td>114.2 ± 7.09</td>
<td>84.8 ± 20.27</td>
<td>80 ± 17.49</td>
<td>76.6 ± 11.73</td>
<td>76 ± 13.26</td>
<td>74.4 ± 7.79</td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td></td>
<td>39.52 ± 0.35</td>
<td>39.38 ± 0.43</td>
<td>39.22 ± 0.46</td>
<td>38.96 ± 0.41</td>
<td>38.78 ± 0.49</td>
<td>38.48 ± 0.61</td>
</tr>
<tr>
<td>Arterial blood pressure (mmHg)</td>
<td>134.2 ± 6.83</td>
<td>105.2 ± 25.65</td>
<td>101.8 ± 14.72</td>
<td>101.8 ± 17.87</td>
<td>104.8 ± 15.04</td>
<td>112.4 ± 16.29</td>
<td></td>
</tr>
<tr>
<td>PaCO₂ (mmHg)</td>
<td></td>
<td>37.98 ± 3.21</td>
<td>57.48 ± 6.99</td>
<td>54.46 ± 1.47</td>
<td>50.82 ± 8.95</td>
<td>46.62 ± 7.00</td>
<td>45.64 ± 5.33</td>
</tr>
<tr>
<td>PaO₂ (mmHg)</td>
<td></td>
<td>105.54 ± 32.08</td>
<td>83.18 ± 47.50</td>
<td>84.88 ± 30.80</td>
<td>111.20 ± 23.97</td>
<td>98.84 ± 42.56</td>
<td>97.3 ± 31.85</td>
</tr>
<tr>
<td>pH</td>
<td></td>
<td>7.374 ± 0.043</td>
<td>7.266 ± 0.041</td>
<td>7.301 ± 0.042</td>
<td>7.345 ± 0.058</td>
<td>7.388 ± 0.059</td>
<td>7.404 ± 0.030</td>
</tr>
</tbody>
</table>

* P < 0.05; ** P < 0.01 compared to baseline; ± SD

Although there was no significant difference in respiratory rate, the mean heart rate decreased significantly at 15 to 60 min following ketamine administration (P < 0.05). Mean arterial blood pressure declined and the decline was highly significant at 15 to 60 min after anesthesia (P < 0.01). Mean PaO₂ did not change significantly but mean PaCO₂ values increased at 5, 15 and 60 min (P < 0.05) and the increase was highly significant (P < 0.01) at 5 and 15 min. Mean pH values decreased highly significantly at 5 and 15 min (P < 0.01). The anaesthetic combination decreased significantly the mean rectal temperature at 30 to 60 min and the decline was highly significant (P < 0.01) at 60 min.

Discussion

The purpose of anaesthesia is to provide reversible unconsciousness, amnesia, analgesia, and immobility with minimal risk to the patient. Anaesthetic drugs and adjuncts may, however, compromise patient homeostasis at unpredictable times and unpredictable ways (10).

Mild respiratory depression has been reported in ketamine administration and this is usually manifested by an increased rate which does not compensate for a decreased tidal volume. On the other hand, xylazine as a member of α₂-adrenoceptor agonist may produce tachypnoea (8). The breathing rate per second is of limited value without some reference to tidal volume and previous trends because normal rates can vary so widely. A change in breathing rate is, however, often a sensitive indicator to some physiologic changes (10). In this study, the anaesthetic regimen had no significant influence on respiratory rate, but Kul et al. (16) showed significant changes at 15, 30 and 60 min after xylazine-ketamine administration in dogs. The decline of respiratory rate in rabbits and cats had been showed previously by Borkowski et al. (3) and Verstegen et al. (22), respectively.

The results showed that mean heart rate decreased significantly at 15 to 60 min after xylazine-ketamine administration. The major side effects of α₂-adrenoreceptor agonists on the cardiovascular system may have contributed to the decreased heart rate in this anaesthetic regimen (8, 16). Although ketamine may increase the heart rate by the increased sympathetic activity and decreased vagal tone, xylazine overrides these effects by excitatory carotid baroreceptor reflex induced by hypotension and decreased sympathetic and increased vagal activity. Kul et al. (16) found a prolonged decrease in the heart rate to 120 min in their study with xylazine-ketamine administration in dogs and Moens and Fargetton (17) showed a 27% decrease in the heart rate at 45 min. The same results in cats had been obtained previously by Allen et al. (1).

The effects of xylazine on the arterial blood pressure depend on the relative effects of the central and peripheral stimulation. There is often an initial hypertensive phase, followed by a more prolonged period of arterial hypotension. The hypertensive phase is not always evident after intramuscular injection, possibly because of reduced peak blood concentrations of the drug (8). Ketamine causes a short-lived vasodepressor response that is followed by longer-lasting potent pressor response (2). The depressor phase of ketamine anaesthesia occurs within the first few minutes of induction and is due to a direct relaxation of vascular smooth muscles (2). There have been several proposed mechanisms for the secondary pressor response to ketamine, including direct central depression of baroreceptor activity, thereby reflexively increasing sympathetic tone and by direct inhibition of norepinephrine uptake at adrenergic nerve endings, producing peripheral sympathomimetic actions (23).

However, in the present study sympatholytic activity of xylazine was more effective than the effects of ketamine on arterial blood pressure, therefore mean arterial blood...
pressure declined highly significantly at 15 to 60 min (P < 0.01). Kul et al. (16) found that xylazine-ketamine induced a decline in arterial blood pressure lasting 120 min. Kitzman et al. (14) showed that peripheral vasopressor effects of ketamine are inhibited by xylazine when two agents are combined, and this finding supports the present results in the goats.

The analysis of carbon dioxide and oxygen in an arterial blood sample defines pulmonary function (9). Ketamine alone or in combination with xylazine has been shown to cause hypercarbia, acidosis, and a 10% or greater depression in the PaO2 (24). Both components of this anaesthetic combination have been shown to cause hypoventilation when used alone (20, 24). Grant and Upton (6) did not find any significant changes in arterial carbon dioxide tension after i.m. administration of xylazine (50 µg/kg) in conscious sheep but a slight degree of arterial hypoxaemia with a 10% reduction in arterial oxygen tension values at 30 min was seen in their study. In the present study, the anaesthetic combination produced hypercarbia and acidosis especially at 5 and 15 min after anaesthesia but PaO2 did not change significantly. Hypercarbia and acidosis due to ketamine-xylazine is in agreement with past reports in rabbits and mice given this anaesthetic (5, 20). A lack of changes in PaO2 levels, accompanied by a rapid increase in PaCO2 which is reported in our study causes a ventilation/perfusion mismatch and it was in accord with Kolata (15) with diazepam-ketamine i.v. administration in dogs. On the other hand, some researchers did not find any change in PaCO2 and PaO2 after administration of xylazine-ketamine or xylazine in cats and dogs, respectively (1, 7). Ruminants, especially sheep and goats, are highly sensitive to the effects of α2-adrenoceptor agonists requiring a reduction in dose of approximately 20 to 40 times when compared to species such as the cat, dog or horse (6). The difference in drug effects and drug sensitivity between species may be responsible to variations in the physiological responses.

On the basis of literature, both ketamine and xylazine may produce hypothermia (19). In our study significant decrease in rectal temperature was observed at 30 to 60 min after anaesthesia and the decrease at 60 min was highly significant. The decline in body temperature is in agreement with Bush et al. (4) when ketamine was used in non-human primates but Kul et al. (16) and Wyatt et al. (25) did not find any significant changes in body temperature after ketamine-xylazine administration in the dog and rabbit.

This study has shown that ketamine-xylazine administration in goats depresses the cardiovascular system and care must be taken in minimizing any depression which may result from anaesthesia or surgery. On the other hand, the decline in body temperature may affect other physiologic parameters, therefore monitoring of body temperature is essential for anaesthetized patient.

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References


