Changes in myocardial performance induced by pancuronium and gallamine in hypercapnic and hypocapnic dogs

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SUMMARY
Changes in myocardial performance after administration of gallamine 1.5 mg kg⁻¹ and pancuronium 0.1 mg kg⁻¹ were investigated in hypercapnic (P_{aCO_2} = 7.08 kPa) and hypocapnic (P_{aCO_2} = 2.74 kPa) dogs anaesthetized with thiopentone, nitrous oxide and halothane. Administration of pancuronium during hypocapnia caused a decrease of 25% in dP/dt max (corrected for changes in preload, afterload and heart rate). This change was not seen during hypercapnia, probably because of the associated sympathetic stimulation. By contrast, gallamine was without effect on dP/dt max in both groups. The increase in heart rate and cardiac output caused by the atropine-like action of these relaxant drugs differed in the hypercapnic and hypocapnic group of dogs, with the more pronounced response in the latter group. The duration of the chronotropic changes was the same in both groups.

Studies of the circulatory changes after gallamine and pancuronium are usually made in the normocapnic or slightly hypocapnic state. The haemodynamic responses to changes in blood carbon dioxide concentration might modify those resulting from the drugs, just as changes in acid-base state might affect the haemodynamic action of gallamine and pancuronium by changing their pharmacological properties or the reactions at the receptor sites.

The aim of this study was to investigate the changes in myocardial performance in hypercapnic and hypocapnic experimental animals with regard to both the degree and the duration of alteration after administration of gallamine and pancuronium.

METHODS AND MATERIALS
Twenty healthy dogs (mean weight 20.9 (SEM 2.1) kg) were studied.

Anaesthesia
Premedication consisted of morphine 1 mg kg⁻¹ i.m. Forty-five minutes later, thiopentone was given slowly i.v. until the eyelid reflexes disappeared (mean dose 15.1 mg kg⁻¹, SEM 0.5 mg kg⁻¹). An endotracheal tube was inserted and anaesthesia was maintained with halothane 1% in a mixture of 25% oxygen in nitrous oxide. Mechanical ventilation was provided with an Elema-Schonander Servoventilator 900 set to provide an expiratory minute volume of approximately 400 ml kg⁻¹, and a frequency of 20 b.p.m. The respiratory cycle was divided into three phases in the following proportion 25:10:65 for inspiration, inspiratory pause and expiration respectively. A constant inspiratory flow rate was used. The inspired oxygen concentration was controlled using an OHIO 200 oxygen monitor and the end-tidal carbon dioxide concentration using an infra-red analyser (Beckman medical gas analyser LB-2), sampled continuously from the proximal end of the tracheal tube. A heated humidifier was placed in the inspiratory limb of the ventilator tubing. A thermistor was placed in the oesophagus for continuous monitoring of temperature (Ellab M4-TEM), which was maintained in the range 36.0–37.0 °C (mean 36.8 °C, SEM 0.2 °C) using an insulating blanket and infra-red radiation lamp placed above the exposed parts of the dog.

Cardiovascular measurements
The c.c.g. was recorded. Lignocaine 2% was used to infiltrate the inguinal region before dissection of the vessel. Catheters for measuring aortic and central venous pressure were passed through the right femoral artery and vein and positioned in the aortic arch and just outside the right atrium respectively, and were connected to Elema–Schönander EMT 34 pressure transducers. A micro-tip pressure transducer catheter (Millar PC-350A, Millar Instruments, U.S.A.) was positioned in the left ventricle by retrograde catheterization of the left femoral artery. The left ventricular pressure signal was passed through a differentiator (accuracy 1% up to 75 Hz) to give the first derivative
(dP/dt). Through the left femoral vein, a 110-cm triple lumen thermodilution catheter (EDSLAB model 93–122–7 F) was passed into the pulmonary artery under fluoroscopic control; it was connected to an integrating computer, which allowed electronic integration of the temperature–time curve (Hewlett Packard 130 cardiac output computer). The exact position of the catheter was confirmed by injection of radiological contrast medium. The central venous catheter was used as the injection route for myoneural blocking drugs and for the cold saline used in the thermodilution technique for measuring cardiac output.

The e.c.g., pressure curves and first derivative of the left ventricular pressure were all recorded on an eight-channel ink-jet recorder (Elema-Schönander EM 81). The mean aortic and central venous pressure were computed electronically.

Biochemical measurements
Arterial blood was analysed at each stage of the experiment; PaO₂, PaCO₂ and pH were measured using a Radiometer ABL 1. Corrections were applied for temperature differences, using a Radiometer blood-gas calculator. The same calculator was used to determine base excess from PaCO₂, pH and haemoglobin concentration, which in turn was determined by a cyanmethaemoglobin technique.

Computations
Cardiac output was determined by the thermodilution method (Branthwaite and Bradley, 1968; Forrester et al., 1972) and was calculated from the values of initial blood temperature, initial injectate temperature and the integral of the blood temperature–time curve by using a program for the Hewlett-Packard 9821A desk computer. Stroke volume was calculated from cardiac output and R-R intervals from the e.c.g. Stroke volume and cardiac output were expressed as "normalized" values (ml kg⁻¹ body weight). Systemic vascular resistance was calculated using the formula:

\[
\text{mean aortic pressure} \times 80 \times 10^{-4} = \text{SVR (kPa.s cm}^{-3})
\]

where aortic pressure is mm Hg and cardiac output litre min⁻¹.

The maximum rate of increase of left ventricular pressure (dP/dt max) is known to be affected by changes in preload (left ventricular end diastolic pressure (LVEDP)), afterload (systolic pressure) and heart rate, and by changes in the inotropic state (Wallace, Skinner and Mitchell, 1963; Mason et al., 1972).

In a recent study in our laboratory (Videbæk, 1976) the statistical interrelationship between indices derived from ventricular pressure measurements on the one hand and heart rate, LVEDP and arterial systolic pressure on the other, was investigated. The design of the experiment was similar to the present study, although a barbiturate was used as the anaesthetic. By means of multivariate statistical analysis, a regression was found between dP/dt max and the haemodynamic parameters mentioned above:

\[
\log dP/dt \ max = -0.7604 \log RR \\
+ 0.1124 \log \text{LVEDP} \\
+ 0.2446 \log \text{systolic pressure} \\
+ 11.0384
\]

All partial regression coefficients were statistically different from zero (P<0.01 for all). By using this equation it is possible to compensate for changes in afterload and preload during the experiment and to evaluate the changes in dP/dt max induced by alteration of heart rate. In this paper the term dP/dt max corr is used for dP/dt max, when this figure is corrected for changes in LVEDP, systolic pressure and heart rate following the above regression.

Experimental protocol
The dogs were allocated randomly to one of four groups (hypocapnia-pancuronium, hypercapnia-pancuronium, hypocapnia-gallamine, hypercapnia-gallamine), each group consisting of five animals. In all groups, the volume of ventilation was set to achieve a "primary" arterial carbon dioxide tension of approximately 2.66 kPa. Blood-gas analysis was performed as soon as the arterial catheter was positioned, but always after at least 30 min of constant ventilation. In the hypocapnic groups this ventilation was continued. In the hypercapnic groups carbon dioxide was added to the inspiratory mixture in order to achieve PaCO₂ of approximately 7.32 kPa. When this was reached, 30 min was allowed to enable the cardiovascular state to stabilize. To avoid the depressant effects of extreme metabolic acidosis, the base deficit was adjusted so that it did not exceed a value of 5 mmol litre⁻¹ by infusion of small volumes of sodium bicarbonate 8.4%. The relaxant drugs were administered at about 2 h after induction of anaesthesia (mean 127 min, SEM 6 min); pancuronium was given in a dose of 0.1 mg kg⁻¹, gallamine in a dose of 1.5 mg kg⁻¹ body weight.
Control measurements of myocardial performance were obtained just before administration of the drugs, and at 1, 2, 3, 5, 10, 15, 20, 25 and 30 and at each 10 min thereafter. Records were taken with ventilation stopped (airway pressure 0 cm H2O; lung volume at functional residual capacity). Determinations of cardiac output were made in duplicate at each stage. The reaction to a second dose of relaxant was recorded 140 min after administration of the first dose.

**Statistical analysis**

The measured myocardial performance variables were compared with control measurements and the deviations expressed as per cent, taking controls as 100%. In each group the method of paired comparisons was used to describe the differences (two-tailed test). From one group to another the Student's t test for unpaired data (two-tailed) was used to evaluate the differences. P<0.05 was taken as a statistically significant difference.

**RESULTS**

**Control**

The differences in blood-gases and acid–base state between the hypercapnic and hypocapnic groups are shown in table I.

**Table I. Values for arterial blood-gases, acid–base state, haemoglobin and tidal volumes (mean values and SEM)**

<table>
<thead>
<tr>
<th></th>
<th>Hypocapnic group (n = 10)</th>
<th>Hypocapnic group (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO2 (kPa)</td>
<td>17.69 ± 0.53</td>
<td>17.96 ± 0.40</td>
</tr>
<tr>
<td>PaCO2 (kPa)</td>
<td>2.74 ± 0.08</td>
<td>7.08 ± 0.16**</td>
</tr>
<tr>
<td>pH</td>
<td>7.459 ± 0.01</td>
<td>7.257 ± 0.006</td>
</tr>
<tr>
<td>Base excess (mmol litre⁻¹)</td>
<td>-2.6 ± 0.4</td>
<td>-4.4 ± 0.4**</td>
</tr>
<tr>
<td>Haemoglobin (g litre⁻¹)</td>
<td>125 ± 5</td>
<td>135 ± 6</td>
</tr>
<tr>
<td>V/T expir. (ml kg⁻¹)</td>
<td>20.8 ± 0.6</td>
<td>19.2 ± 0.7</td>
</tr>
<tr>
<td>Primary PaCO2 (kPa)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.63 ± 0.09 (n.s.)</td>
<td></td>
</tr>
</tbody>
</table>

Significance for two-tailed Student's t test: ** P<0.01; n.s. = not statistically different from the value of PaCO2, in the hypocapnic groups.

Table II shows haemodynamic variables in the two different basal conditions. There were statistically significant differences in central venous pressure, LVEDP and systemic vascular resistance (SVR) between the two groups, but not in heart rate, mean aortic pressure, normalized cardiac output, normalized stroke volume and dP/dt max, although the values were greater in the hypercapnic group.

**Table II. Haemodynamic variables (mean values and SEM)**

<table>
<thead>
<tr>
<th></th>
<th>Hypocapnic group (n = 10)</th>
<th>Hypocapnic group (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beat min⁻¹)</td>
<td>92 ± 7</td>
<td>106 ± 8</td>
</tr>
<tr>
<td>Mean aortic pressure (mm Hg)</td>
<td>111 ± 6</td>
<td>116 ± 7</td>
</tr>
<tr>
<td>Central venous pressure (mm Hg)</td>
<td>6 ± 0.5</td>
<td>8 ± 1.0*</td>
</tr>
<tr>
<td>Left ventricular end diastolic pressure (LVEDP) (mm Hg)</td>
<td>6.7 ± 1.1</td>
<td>11.1 ± 1.0**</td>
</tr>
<tr>
<td>Normalized stroke volume (ml kg⁻¹)</td>
<td>111 ± 13</td>
<td>147 ± 12†</td>
</tr>
<tr>
<td>Normalized cardiac output (Q/kg = ml. kg min⁻¹)</td>
<td>1.19 ± 0.11</td>
<td>1.41 ± 0.10</td>
</tr>
<tr>
<td>Systemic vascular resistance (kPa.s cm⁻³)</td>
<td>0.521 ± 0.062</td>
<td>0.277 ± 0.024*</td>
</tr>
<tr>
<td>dP/dt max (mm Hg s⁻¹)</td>
<td>1773 ± 181</td>
<td>2277 ± 174</td>
</tr>
</tbody>
</table>

Significance for two-tailed Student's t test: * P<0.05; ** P<0.01; † P= 0.05.

**Gallamine**

The administration of gallamine significantly increased heart rate from control value for the first 60 min in both groups (P<0.05) (fig. 1), but with a slightly more pronounced response in the hypocapnic group. Normalized cardiac output increased to values which differed significantly from control for the first 30 min (P<0.05). The difference in cardiac output between the hypocapnic and hypercapnic group was statistically significant only in the 1st and 5th min (P<0.05). The observations for stroke volume follow the same pattern, but are not significantly different from the control values.

A small, although not statistically significant, decrease in LVEDP was recorded just before the reduction in cardiac output in the hypocapnic group at the 3rd min. In the hypercapnic group a decrease in LVEDP during the experiments was recorded (from 11 to 8 mm Hg, P<0.05). Mean aortic pressure, central venous pressure, systemic vascular resistance and dP/dt max were unaltered during the investigations. The corrected and uncorrected values for dP/dt max are given in table III. There was no difference in the duration of the cardiovascular changes between the two groups. The second dose evoked similar, although smaller, changes, of shorter duration.

**Pancuronium**

A statistically significant increase in heart rate from control values was recorded for the first 20 min in
### TABLE III. Gallamine: uncorrected and corrected dP/dt max values (mean and SEM) (mm Hg s⁻¹)

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Uncorrected</th>
<th>Corrected</th>
<th>Uncorrected</th>
<th>Corrected</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1601 ± 224</td>
<td>1524 ± 330</td>
<td>2268 ± 330</td>
<td>2268 ± 330</td>
</tr>
<tr>
<td>1</td>
<td>1815 ± 293</td>
<td>1524 ± 255</td>
<td>2480 ± 400</td>
<td>2253 ± 380</td>
</tr>
<tr>
<td>2</td>
<td>1796 ± 284</td>
<td>1587 ± 237</td>
<td>2342 ± 345</td>
<td>2158 ± 359</td>
</tr>
<tr>
<td>3</td>
<td>1810 ± 299</td>
<td>1551 ± 270</td>
<td>2342 ± 332</td>
<td>2097 ± 324</td>
</tr>
<tr>
<td>5</td>
<td>1839 ± 298</td>
<td>1514 ± 262</td>
<td>2327 ± 298</td>
<td>2075 ± 287</td>
</tr>
<tr>
<td>10</td>
<td>1815 ± 306</td>
<td>1521 ± 271</td>
<td>2420 ± 333</td>
<td>2092 ± 302</td>
</tr>
<tr>
<td>15</td>
<td>1790 ± 335</td>
<td>1522 ± 291</td>
<td>2420 ± 324</td>
<td>2152 ± 315</td>
</tr>
<tr>
<td>20</td>
<td>1846 ± 347</td>
<td>1573 ± 300</td>
<td>2516 ± 358</td>
<td>2211 ± 326</td>
</tr>
<tr>
<td>25</td>
<td>1869 ± 360</td>
<td>1602 ± 314</td>
<td>2470 ± 335</td>
<td>2186 ± 310</td>
</tr>
<tr>
<td>30</td>
<td>1873 ± 354</td>
<td>1637 ± 309</td>
<td>2574 ± 372</td>
<td>2260 ± 320</td>
</tr>
</tbody>
</table>

**Fig. 1.** Changes in heart rate, normalized cardiac output, (Q/kg), dP/dt max corr and systemic vascular resistance (SVR) after gallamine 1.5 mg kg⁻¹. Mean values and SEM.

**Fig. 2.** Changes in heart rate, normalized cardiac output, (Q/kg), dP/dt max corr and systemic vascular resistance (SVR) after pancuronium 0.1 mg kg⁻¹. Mean values and SEM.

Both groups (P<0.05) (fig. 2), but with a more pronounced response in the hypocapnic group. The difference between the two groups was statistically significant only at 2 min after the injection (P<0.05).

The normalized cardiac output changed in a manner similar to that of the control, but the difference was significant only in the first 10 min (P<0.05). No difference in cardiac output between the groups could be detected.

Normalized stroke volume and mean aortic pressure were unchanged during the experiments. Central venous pressure and LVEDP did not change in the hypocapnic dogs, but a reduction from the control value was recorded in the hypercapnic dogs (LVEDP...
from 11 mm Hg to 8 mm Hg, *P* < 0.05; central venous pressure from 10 mm Hg to 7 mm Hg, *P* < 0.05).

Systemic vascular resistance decreased from the control value in both groups, but especially in the hypocapnic group, where the reduction was found to be statistically significant for the first 10 min (*P* < 0.05). No significant difference was found between the two groups.

A significant reduction from the control value was recorded in respect of dP/dt max corrected in the hypocapnic group (*P* < 0.05), lasting from the 3rd to the 25th min. The values in the hypcapnic group were unaltered during the investigation. The uncorrected and corrected values for dP/dt max are listed in Table IV. The duration of the cardiovascular changes was the same in the two groups. Administration of a second dose was followed by less pronounced changes of shorter duration, but with the same trends.

<table>
<thead>
<tr>
<th></th>
<th>Hypocapnia (n = 5)</th>
<th>Hypercapnia (n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time Uncorrected dP/dt max</td>
<td>Corrected dP/dt max</td>
<td>Uncorrected dP/dt max</td>
</tr>
<tr>
<td>0 1944 ± 287</td>
<td>1944 ± 287</td>
<td>2276 ± 159</td>
</tr>
<tr>
<td>1 2418 ± 620</td>
<td>1754 ± 425</td>
<td>2830 ± 216</td>
</tr>
<tr>
<td>2 2195 ± 445</td>
<td>1646 ± 353</td>
<td>2710 ± 226</td>
</tr>
<tr>
<td>3 1878 ± 256</td>
<td>1464 ± 239</td>
<td>2517 ± 148</td>
</tr>
<tr>
<td>5 1862 ± 252</td>
<td>1460 ± 193</td>
<td>2535 ± 250</td>
</tr>
<tr>
<td>10 1827 ± 244</td>
<td>1465 ± 161</td>
<td>2522 ± 282</td>
</tr>
<tr>
<td>15 1794 ± 268</td>
<td>1590 ± 237</td>
<td>2494 ± 293</td>
</tr>
<tr>
<td>20 1808 ± 263</td>
<td>1575 ± 171</td>
<td>2509 ± 250</td>
</tr>
<tr>
<td>25 1807 ± 283</td>
<td>1672 ± 200</td>
<td>2565 ± 311</td>
</tr>
<tr>
<td>30 1828 ± 331</td>
<td>1769 ± 322</td>
<td>2463 ± 266</td>
</tr>
</tbody>
</table>

**DISCUSSION**

**Carbon dioxide**

Changes in arterial carbon dioxide tension are followed by changes in sympatho-adrenal activity. This response is evoked by stimulation or inhibition of centres in the hypothalamic region and is mediated through changes in catecholamine secretion (Sechzer et al., 1960; Cross and Silver, 1962). Accumulation of carbon dioxide induces an increase in cardiac output, heart rate and indices of myocardial contractility, while systemic vascular resistance decreases. Carbon dioxide washout leads to changes in the opposite direction (Price 1960; Cullen and Eger 1974). Cardiovascular changes evoked by carbon dioxide accumulation during anaesthesia are modified by different anaesthetics to a variable extent (Price et al., 1959). Haemodynamic responses to hypocapnia and hypercapnia during halothane anaesthesia have been determined by various investigators (Theye, Mild and Michenfelder, 1966; Prys-Roberts et al., 1967; Föex and Prys-Roberts, 1975). Changes occurred in cardiac output (augmented by hypercapnia) and systemic vascular resistance (augmented by hypocapnia) without modification of the contractile state of myocardium and without significant changes in heart rate, filling pressures or aortic pressure. Changes in stroke volume were found to parallel the changes in cardiac output. The factors governing alteration in cardiac output were changes in afterload and sympathetic activity (Föex and Prys-Roberts, 1975).

Our results confirm these findings with one exception. The increased filling pressures in hypercapnia indicate that changes in preload contributed to the increase in cardiac output.

**Atropine-like action**

Cardiovascular changes elicited by pancuronium and gallamine have been studied by many investigators. The use of these drugs in man is often followed by an increase in heart rate leading to an increase in cardiac output, although modified by the chosen anaesthetic and premedicant. Small increases in arterial pressure accompanied by decreases in central venous pressure occur. Stroke volume varies to only a minor degree. Systemic vascular resistance decreases after the injection of gallamine but not after pancuronium (Smith and Whitcher, 1967; Kennedy and Farman, 1968; Kelman and Kennedy, 1971; Coleman et al., 1972; Stoeling, 1972, 1973). In experiments using isolated organ perfusion, both pancuronium and gallamine produced a blockade of the muscarinic receptors of the heart, resulting in an increase in heart rate (Rathburn and Hamilton, 1970; Saxena and Bonta, 1970; Goat and Feldman, 1972; Duke, Fung and Gardner, 1975).

The increase in cardiac output after the injection of pancuronium and gallamine in our study was a result of changes in heart rate, as no alterations in stroke volume occurred. The small and inconsistent differences in percentage increase in heart rate and cardiac output between the hypocapnic and hypercapnic groups can be explained by the differences in heart rate in the control state. It has been shown that a negative correlation exists between heart rate before administration of pancuronium and maximum
increase in heart rate, and that previous administration of atropine inhibits the tachycardia seen after gallamine and pancuronium (Coleman et al., 1972; Reitan, Fraser and Eisele, 1973; Miller et al., 1975). The changes in systemic vascular resistance are parallel to the changes in cardiac output. As ganglionic transmission in the sympathetic nervous system is affected only slightly after pancuronium and gallamine (Buckett et al., 1968; Hughes and Chapple, 1976), it might be concluded that the changes in resistance were induced reflexly and secondary to the increase in cardiac output.

**Gallamine and the inotropic state**

Brown and Crout (1970) found that administration of gallamine increased the contractile force of guinea-pig and cat atria in vitro; similarly, they found increases in contractile force in the anaesthetized open-chest cat. Previous administration of propranolol inhibited these responses. It was suggested, therefore, that gallamine released noradrenaline from adrenergic nerve endings in the heart. In contrast to this, a study in man during open heart surgery using a Walton–Brodie strain-gauge arch failed to demonstrate any change in myocardial contractile force associated with administration of gallamine (Loh, 1970). However, a study using rabbit atrial strips and cat papillary muscle failed to reveal any change in contractile force after exposure to pancuronium (Duke, Fung and Gartner, 1975).

The results of blood catecholamine determination after pancuronium are conflicting. In one study, a significant increase in the concentration of both adrenaline and noradrenaline in plasma was recorded after pancuronium during halothane anaesthesia (Aurel, Cardan and Domokos, 1973). In other investigations no alterations were demonstrated during either nitrous oxide–oxygen–fentanyl or thiamyl-nitrous oxide–oxygen anaesthesia (Takki and Tamisto, 1973; Zsigmond et al., 1974).

Our results show that administration of pancuronium is followed by a 25% decrease in dP/dt max corr, when the drug is administered during hypcapnia, whereas no changes are detectable during hypercapnia. This negative inotropic action is seldom, if ever, recognized in clinical practice, because of the compensating effect of the positive chronotropic action of the drug. In our study, no increase in filling pressure or alteration in stroke volume was encountered during hypcapnia. Furthermore, it is demonstrated that the uncorrected dP/dt max values changed only to a minor extent from the control values. However, the possibility exists that administration of pancuronium to the hyperventilated patient with conduction disturbances (A–V block), treatment with β-blockers or cardiac compensation might disclose this effect and induce signs of pump failure, but further human studies are needed to elucidate this point. The difference seen between the hypercapnic and hypcapnic groups can probably be explained by the supportive action of the increased sympathetic activity which exists in the hypercapnic state.

**Acid–base state**

It is well known that changes in acid–base state affect the neuromuscular blocking action of gallamine (prolonged block associated with alkalosis) and pancuronium (acidosis reducing rate of recovery) (Baraka, 1967; Walts, Lebowitz and Dillon, 1967; Katz and Seed, 1970). This difference in duration of action has been related to changes in drug–protein binding and to changes in cholinesterase activity. Altered ionization of the relaxant molecule can be ruled out as an explanation of the different effects because the pKa values of both drugs are greater than 13 (Baraka, 1967; Dann, 1971). We found no differences in the duration of the cardiovascular changes elicited by either drug in the hypercapnic or hypcapnic state, although the possibility remains that separating the pH values by more than 0.29 units might have revealed some disparity, but this seems to be a question of academic interest only.
Second dose
Changes evoked by a second dose of relaxant are predictably of lesser degree and duration, and this was confirmed in our study. Although the control state before administering the second dose resembled the state just before administration of the first dose, the cardiac receptors must be partially occupied, resulting in only minor changes in the cardiovascular variables.

ACKNOWLEDGEMENTS

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REFERENCES


**VARIATIONS DES PERFORMANCES MYOCARDIAQUES PROVOKEES PAR LE PANCURONIUM ET LA GALLAMINE SUR DES CHIENS HYPERCAPNIQUES ET HYPOCAPNIQUES**

**RESUME**

On a etudie les variations des performances myocardiaques aprés administration de gallamine (1,5 mg/kg) et de pancuronium (0,1 mg/kg) à des chiens hypercapniques (Paco₂ = 7,08 kPa) et hypocapniques (Paco₂ = 2,74 kPa) anesthesiés à l’aide de thiopentone, de protoxyde d’azote et d’halothane. L’administration de pancuronium pendant l’hypocapnie a provoqué une diminution de 25% de la dP/dt max (corrigée pour variations dans le pré-charge ment, le postcharge ment et la fréquence cardiaque). Cette variation n’a pas été constatée pendant l’hypercapnie, probablement en raison de la stimulation sympathique qui lui est associée. Par contre, la gallamine est restée sans effet sur la dP/dt maxima le dans les deux groupes. L’augmentation de la fréquence et du débit cardiaques causée par l’action semblable à celle de l’atropine de ces deux agents décontracturants a différé dans les deux groupes de chiens (hypercapniques et hypocapniques), la réaction la plus prononcée se produisant dans ce dernier groupe. La durée des variations chronotropiques a été la même dans les deux groupes.