Pulmonary Vasomotor Responses to Epinephrine and Norepinephrine in the Cat

Influence of the Sympathetic Nervous System

By Helen X. Duke, M.B., Ch.B., Ph.D., and R. D. Stedeford, M.A., B.M., B.Ch.

Epinephrine has been shown to have pulmonary vasomotor effects in isolated perfused cats' lungs,1,2 the most usual effect is constriction, although dilatation can occur (further references are given by Daly3). In anesthetized cats, epinephrine causes an increase of pulmonary arterial pressure,4 but no measurements of pulmonary vascular resistance are possible, since the cardiac output was not measured. In anesthetized dogs the resistance to perfusion of a single lung lobe is increased by epinephrine.5 In man, the response to epinephrine or norepinephrine is more variable.6 The discrepancy between the results of various workers may be, at least in part, due to difference in activity of sympathetic vasomotor nerves during the experiments.

Previous work has usually been concerned with the responses of the pulmonary vascular bed to a single dose of epinephrine. In the present series of experiments, the effects of a constant intravenous infusion of epinephrine or norepinephrine on the pulmonary vascular resistance were compared either before or after bilateral stellectomy and removal of the upper thoracic sympathetic chains. It has been reported recently that the effects of a constant infusion of norepinephrine, into the right atrium of anesthetized dogs, are potentiated by section of the spinal cord and vagotomy. Some of the present results already have been reported briefly.

Methods

Cats of either sex (2.0 to 4.5 Kg.) were anesthetized with chloralose (0.1 Gm./Kg. intraperitoneally). They were placed on their backs on a warmed operating table. The trachea was cannulated, and a catheter (“Portex” polythene, no. 52) was passed up the left femoral vein, until its tip lay in the right atrium at the level of the fourth costal cartilage. The position was checked post mortem. The systemic blood pressure was recorded from the right femoral artery with a mercury manometer, and the left femoral artery was cannulated for collection of arterial blood samples. Arterial pressure in the pulmonary circulation was measured with a manometer filled with 0.9 per cent NaCl solution and recorded with a small capacity tambour. Recordings of the pulmonary arterial pressure (P.A.p.) were made by 1 of 2 methods: (A) A catheter was introduced into the pulmonary artery from the right external jugular vein. This catheter (“Portex” polythene, no. 52) was bent into a circle at the distal end (approximately 2 cm. in diameter) in a plane perpendicular to the rest of the catheter, the tip pointing in a counterclockwise direction, as seen from above. It was introduced into the right ventricle on an introducer slightly curved at the end. When the tip was in the ventricle, a pulsatile stream of blood emerged between the wall of the catheter and the introducer. The introducer was withdrawn then, and the catheter connected to the manometer. Counterclockwise rotation of the catheter, while passing it further in, normally led to the tip being passed into the pulmonary artery. When this occurred, the mean arterial pressure rose approximately 5 cm. saline solution. The position of the tip of the catheter was always checked post mortem and was usually found 2 to 20 mm. beyond the pulmonary valves. (B) The chest was opened down the midline of the sternum and held widely retracted. The animal was artificially ventilated by a Starling “Ideal” pump (20 strokes/min.). In a few experiments, constant peak positive pressure was used and the tidal air overflow volume recorded, by the method of Konzett & Rössler. A cannula was placed into the pulmonary arterial branch to the right upper lung lobe, and the right upper lobe was tied just distal to the cannula. In some of the earlier experiments, right ventricular pressure (R.V.p.) was recorded from a catheter in the right ventricle instead of the P.A.p.
Left atrial pressure (L.A.p.) was recorded from a needle (external diameter 1.25 mm. British Standard 18 gage, length 20 cm.), the tip of which was placed in the left atrium through the left bronchus,13 under direct vision through a bronchoscope. In experiments in which the chest was opened, the L.A.p. was recorded from a cannula in the left atrium. In both types of recordings, the L.A.p. was measured with a manometer filled with 0.9 per cent NaCl solution and recorded with a tambour. The zeros of the pulmonary arterial and left atrial manometers were arranged to lie approximately 1 cm. posterior to the mitral valve.

Oxygen consumption (over periods of 3 to 5 minutes) was measured using a closed circuit, a recording spirometer, and a carbon dioxide absorber. When artificial ventilation was used, the recording spirometer, and a carbon dioxide absorber. Known volumes of oxygen were added to the circuit over timed intervals, to keep the volume of gas in the circuit approximately constant. The oxygen consumption could be read from the spirometer tracing at the end of the experiment and corrected for the volume of oxygen added. Each recorded consumption was obtained from the mean of 2 periods of measurement, the results usually agreeing within 5 per cent.

Determinations of the cardiac output were made using the direct Fick principle. In the interval between 2 measurements of oxygen consumption, blood samples (approximately 5 ml. in 15 to 30 seconds) were taken successively from the femoral artery and right atrium. Blood oxygen contents were measured in duplicate with a Van Slyke manometric apparatus.

Pulmonary vascular resistance (P.V.R.) was calculated as P.A.p. (or R.V.p.) - L.A.p. in cm. saline solution/cardiac output in ml. per minute. The accuracy of the figure for P.V.R., obtained in this way, is mainly governed by the estimation of cardiac output. If the venous blood is incompletely mixed in the right atrium, then right atrial samples will not be representative of mixed venous blood. Simultaneous blood samples were taken from the right atrium and right ventricle in 6 tests in 3 experiments. The mean difference in oxygen content between these samples was 0.015 vol. per cent and the maximum difference was 0.64 vol. per cent. Assuming an arteriovenous oxygen difference of 7.0 vol. per cent, an oxygen consumption of 20 ml./min., and the maximal difference in blood oxygen content, there is a maximum error in the cardiac output of 17.5 per cent. Allowing for errors of measurement of blood oxygen content, vascular pressures, and oxygen consumption, the maximum possible cumulative error in the calculation of P.V.R. is 35 per cent. It is, however, unlikely that the maximum variation in one direction would occur in all the factors concerned in one observation, and in the other direction for another observation in the same experiment. In the experiments now reported, the P.V.R. was said to be changed "significantly" when the change was more than 35 per cent of the initial value. Since the P.V.R. could either rise or fall with time during an experiment, all test procedures were bracketed between 2 control observations.

Continuous infusions of drugs were made into the right femoral vein using a motor-driven calibrated syringe. The drugs were administered for 5 to 10 minutes before taking measurements of oxygen consumption and vascular pressures. The exact interval of time between starting the infusion and taking measurements was judged by the stability of the pressure records. Final measurements were made when, after stopping the infusion, the pressures were judged to be stable.

The drugs used were: epinephrine hydrochloride (Parke-Davis); norepinephrine (Laevophed, Bayer); 5-hydroxytryptamine creatinine sulphate (May & Baker); hexamethonium iodide (Hexitidine, Allen & Hanbury). The concentrations of the drug solutions were such that 0.25 to 0.5 ml. was infused per minute. The animals were heparinized (Liquemin, Roche, 1000 I.U./Kg.) at the end of the preliminary operations. The volume of blood, withdrawn as blood samples, was immediately replaced by blood from a donor animal or by dextran (Intradex, Glaxo). Electrical stimulation of the cardiac branches from the stellate ganglia was performed using a Palmer (London) "Students" stimulator. The stellate ganglia and their branches were exposed, usually before the first control observation in each experiment, by incising the parietal pleura bilaterally at the posterior limit of the first intercostal space. The parameters of stimulation varied between 3 and 35 volts, 0.62 and 1.4 msec. duration, frequency 1 to 30 per second in different experiments. Stimulation was continued using self retaining electrodes for as long as was necessary to complete the observations. Current spread to adjacent structures was minimized as much as was possible by arrangement of the electrodes. The stellate ganglia were removed by cutting their cephalic connections and avulsing them together with the upper 4 to 5 ganglia of the thoracic sympathetic chain.

Results

Epinephrine

Constant intravenous infusion of this amine (2 to 4 μg./min.) into preparations with an
Effect of a constant intravenous infusion of epinephrine (2 to 4 \(\mu\)g/min.) on the pulmonary vascular resistance. In this and the succeeding figures the anesthetic was administered at approximately 10:35 A.M. Pulmonary vascular resistance was calculated as:

\[
P.A.p. \text{ or R.V.p.} - L.A.p. \text{ cm. 0.9\% NaCl soln.}
\]

Cardiac output ml/min.

Points obtained during intravenous administration of epinephrine are marked \(\Delta\). Four separate experiments.

Intact sympathetic nervous system was without effect on the P.V.R., showing a constant decrease or a constant increase with time in different experiments (fig. 1).

Four experiments were performed in which the animal was artificially ventilated with the chest open, the right ventricular pressure was recorded from a catheter, and the left atrial pressure from a cannula. Epinephrine caused an increase in systemic blood pressure of 20 to 25 mm Hg, a rise of P.A.p. of 1 to 2 cm saline solution, and a fall of L.A.p. of about 0.1 cm. The cardiac output was increased in 2 of the 3 tests. No significant or consistent change in P.V.R. occurred.

Epinephrine after Acute Stellectomy

Seven tests were performed in 6 experiments in which epinephrine (2 to 4 \(\mu\)g/min.) was infused after bilateral removal of the stellate and upper 4 to 5 thoracic sympathetic ganglia. The chest was held widely open, and artificial ventilation was used; in 2 experiments, this was at constant peak positive pressure. Pressures in the pulmonary artery or right ventricle and left atrium were recorded from cannulae. The systemic blood pressure was increased by 5 to 50 mm Hg as the result of the infusion in 6 of the tests, and was unchanged in one. In 4 of the tests the cardiac output was increased by epinephrine (table 1) and in 3 it decreased. In every test, the P.A.p.

### Table 1

**Epinephrine: After Stellectomy**

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<td>30.0</td>
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</table>

*Observations during intravenous injection of epinephrine.
†Right ventricular pressure (R.V.p.).
‡Pulmonary arterial pressure (P.A.p.) recorded from branch of pulmonary artery (P.A.).
§C.O.=Cardiac output ml/min.
∥P.V.R.=Pulmonary vascular resistance.
or R.V.p. was increased, but the L.A.p. changes were small and not consistent. There was a significant and reversible increase of P.V.R. in 3 of the 7 tests (fig. 2) and in 1 test the P.V.R. rose during the infusion, but no final control reading could be made. The 2 experiments not obtaining an increase in P.V.R. (experiments 4 and 6, table 1) were those in which the highest initial P.V.R. was found. There was no apparent relationship between the changes in cardiac output and the changes in P.V.R.

Neglecting the results of the experiment (no. 2, table 1) in which no final control reading was obtained after stopping the infusion of epinephrine, the difference between the effects of epinephrine on the P.V.R. of the intact or sympathectomized animal is statistically significant ($\chi^2 = 4.5, p < 0.05$).

Some experiments were also performed, using hexamethonium iodide to block transmission in the sympathetic ganglia, in an attempt to avoid the surgical trauma involved in opening the chest. Five tests were made in 4 experiments after intravenous administration of this blocking drug (5 to 19 mg./Kg. in divided doses) to animals respiring naturally. Although hexamethonium lowered the blood pressure, the pressor response to bilateral occlusion of the common carotid arteries was not abolished, and infusion of epinephrine only caused slight and inconsistent changes in P.V.R.

**Stellectomy**

In 6 experiments, the P.V.R. was calculated before and 10 to 20 minutes after bilateral removal of the stellate ganglia and upper parts of the thoracic sympathetic chain. Ordinates and abscissae as in figure 1. The points obtained during intravenous administration of epinephrine are marked A. Three separate experiments. The figures by each point show the cardiac output (ml./min.).

In opening the chest. Five tests were made in 4 experiments after intravenous administration of this blocking drug (5 to 19 mg./Kg. in divided doses) to animals respiring naturally. Although hexamethonium lowered the blood pressure, the pressor response to bilateral occlusion of the common carotid arteries was not abolished, and infusion of epinephrine only caused slight and inconsistent changes in P.V.R.

**Stellectomy**

In 6 experiments, the P.V.R. was calculated before and 10 to 20 minutes after bilateral removal of the stellate ganglia and upper parts of the thoracic sympathetic chain. The chest was opened, and artificial ventilation was used. In 5 experiments (table 2), a fall of P.V.R. was produced by stellectomy in 3 of which the fall was significant (see fig. 3). The decrease in P.V.R. accompanied a fall in cardiac output. The P.V.R. was unchanged in 1 experiment (experiment 2, table 2). In the experiments in which stellectomy produced a fall in opening the chest. Five tests were made in 4 experiments after intravenous administration of this blocking drug (5 to 19 mg./Kg. in divided doses) to animals respiring naturally. Although hexamethonium lowered the blood pressure, the pressor response to bilateral occlusion of the common carotid arteries was not abolished, and infusion of epinephrine only caused slight and inconsistent changes in P.V.R.
Figure 3
Effect of bilateral removal of the stellate ganglia and the upper thoracic sympathetic chains of the pulmonary vascular resistance. Ordinate and abscissa and numbers as in figure 1. The time of stellectomy is indicated. Three separate experiments.

in P.V.R., there was usually a subsequent increase of resistance, but no increase in cardiac output.

Stellate Stimulation

Electrical stimulation of the cardiac branches from both stellate ganglia produced a significant and reversible increase of P.V.R. in 2 out of 4 tests in 4 experiments (table 3, fig. 4). These preparations were artificially ventilated with the chest held widely open. In 2 of 3 tests with constant peak positive pressure ventilation, the tidal air overflow was slightly decreased, indicating a decreased resistance to inflation of the lungs. In 2 tests stellate stimulation caused a small but reversible decrease in P.V.R. The P.V.R. was also probably decreased in 1 or 2 other experiments not in the table, in which the P.A.p. decreased during the stimulation, but the condition of the preparation did not permit readings to be taken.

Norepinephrine

In 2 experiments norepinephrine (2 or 4 µg./min.) was infused during normal respiration. There was no change in systemic blood pressure, but the P.V.R. was increased in 1 experiment, although unchanged in the other (fig. 5, table 4).

Norepinephrine after Stellectomy

In each of 2 experiments (table 5), the P.V.R. was reversibly increased by the infusion of norepinephrine (2 or 4 µg./min.). In one test, the increase was a significant one (fig. 5). There was no marked change in systemic blood pressure in either experiment. These preparations were artificially ventilated.

A summary of the results of the various test procedures is given in table 6, from which it can be seen that infusion of epinephrine after stellectomy often caused a reversible increase in P.V.R., whereas the same dose before stellectomy was without effect on the P.V.R. Stellectomy decreased the P.V.R. Significant increases in P.V.R. were also obtained by infusion of norepinephrine. Electrical stimulation of the cardiac branches from the stellate ganglia produced a significant increase of P.V.R. in 2 tests and a more doubtful, but reversible decrease of P.V.R. in 2 other tests.

Table 3

<table>
<thead>
<tr>
<th>Experiment no.</th>
<th>R.V.P.</th>
<th>L.A.p</th>
<th>C.O.†</th>
<th>P.V.R.†</th>
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<td>17.0</td>
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<td>0.033</td>
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*Observations made during stimulation of cardiac branches from both stellate ganglia.
†Headings as in table 1.

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PULMONARY VASCULAR RESPONSES

In the present series of experiments large doses of epinephrine by constant intravenous infusion were found to be without effect on the pulmonary vascular resistance in the cat anesthetized with chloralose. This was an unexpected finding because, as has been already indicated, in cat's isolated lungs epinephrine has well marked pulmonary vasmotor effects. The dose of epinephrine given was approximately 7 to 14 times that found by Polkow & von Euler in the adrenal venous blood of cats under chloralose anesthesia. After acute bilateral pulmonary sympathectomy, the same dose of epinephrine caused a significant increase of pulmonary vascular resistance in 3 out of 6 experiments.

Changes in pulmonary vascular resistance most probably reflect changes in the caliber of the pulmonary vascular bed between the points of measurement of pressure. The pulmonary blood vessels can probably change in caliber either actively, due to nervous, humoral or other effects, or passively due to changes in left atrial, intrathoracic, or intra-alveolar pressures. The effects of changes in the cardiac output and of variations in the rate of exchange of blood between the bronchial and pulmonary circulations must also be considered. The left atrial pressure was measured in this series of experiments, and the observed changes in its value could not be held responsible for the changes in pulmonary arterial pressure. The influence of changes in the cardiac output has been studied in isolated perfused cat lungs. The effects of changes in the rate of pulmonary arterial inflow are minimal at physiological flow ranges, so that if these results can be transferred to the different experimental conditions of the present experiments.
Table 4

<table>
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<th>Experiment no.</th>
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<th>R.V.R.†</th>
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*Observations during infusion of norepinephrine.
†Headings as in table 1.

Table 5

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<th>L.A.p</th>
<th>CO.†</th>
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*Observations during infusion of norepinephrine.
†Headings as in table 1.

series, it would not be expected that the marked changes in P.V.R. now found could be the result of variations in cardiac output. The changes in P.V.R. now reported also did not occur with consistent changes in cardiac output.

The conclusion is, therefore, that after acute sympathectomy epinephrine probably does cause active constriction of at least some part of the pulmonary vascular bed. The effects of norepinephrine have not been studied extensively, but the results of administration of this drug support those of epinephrine.

Stellectomy produced an initial fall in P.V.R., which later tended to increase. The reason for this increase has not been investigated, but it may be associated with intrinsic changes in the lungs, such as atelectasis or extrinsic changes, such as alterations in the rate of secretion of either epinephrine or norepinephrine.

Electrical stimulation of the stellate ganglia produced a significant increase of P.V.R. in 2 tests and a more doubtful decrease of P.V.R. in 2 other tests. This latter result is not surprising since Tribe found both pulmonary vasodilation and vasoconstriction on stimulating thoracic sympathetic nerves and the stellate ganglia in isolated perfused cat lungs. It has also been pointed out that epinephrine occasionally produces a decrease of P.V.R. in the isolated perfused cat’s lungs.

The difference in the effects of a constant intravenous infusion of epinephrine in cats with an intact central nervous system and in those in which an acute sympathectomy has been performed remains to be explained. The results now described could be attributed to a tonic pulmonary sympathetic vasoconstrictor tone, whereas in the intact animal, epinephrine is unable to produce vasoconstriction in blood vessels already constricted. Removal of this constrictor tone, by stellectomy, would unmask the vasoconstrictor effect of epinephrine. This vasoconstrictor tone may normally exist, or it could have been present because of the conditions of these experiments.

Another alternative explanation of these findings is that there is a reflex pulmonary vasodilatation in the cat, due to stimulation of the pressoreceptors, similar to that found by Daly & Daly in the dog. Thus, in conditions in which a rise of systemic blood pressure occurs in the cat with an intact sympathetic nervous system, such as during epinephrine infusion, the pulmonary blood vessels would be reflexly dilated. Thus, measurable changes of P.V.R. would not be found. Stellectomy abolished this reflex pulmonary vasodilatation in the dog. The presence of such a reflex in cats has however been disputed.

The part played by changes in bronchial caliber or lung elasticity on the pulmonary vascular resistance has not been investigated adequately in the present series of experiments, and further work is being undertaken on this point, as well as on the influence of the pressoreceptors on the present results.

The present results may play some part in elucidating the hitherto unexplained findings.
PULMONARY VASCULAR RESPONSES

that occur during exercise. Sympathetic nerves to the lungs, studied under controlled conditions in the anesthetized dog, appear to be predominantly constrictor. Whereas, in normal intact man exercise of varying degrees, in conditions in which epinephrine secretion would be expected to be increased, is without effect on the P.V.R., or has inconsistent and minimal effects.10 In other experiments in normal man exercising in the upright position, a fall in P.V.R. has been observed accompanying an increase of systemic B.P. and a fall in total peripheral resistance.17,18 These findings could be reconciled with the present results in that, in changes of epinephrine secretion do not, in fact, cause changes in P.V.R. when the sympathetic system is intact, because either the pulmonary vessels are tonically constricted or when the systemic B.P. rises a reflex pulmonary vasodilation occurs. The reflex pulmonary vasodilation might mask any direct constrictor action, due to nervous or hormonal mechanisms, as well as any passive effects on the pulmonary circulation due to changes in cardiac output.

Summary

Constant intravenous infusion of epinephrine has no effect on the pulmonary vascular resistance (P.V.R.) of the cat under chloralose anesthesia, either when the chest is opened and artificial ventilation is used, or when the animal is respiring naturally. The same dose of epinephrine given after removal of the stellate ganglia and upper 4 to 5 thoracic sympathetic ganglia, can cause an increase of P.V.R. Removal of such ganglia causes a decrease of P.V.R. Electrical stimulation of the cardiac branches from both stellate ganglia causes either an increase or a decrease of P.V.R. Increases of P.V.R. have been produced by intravenous infusion of norepinephrine.

Acknowledgment

Some of the apparatus used in these experiments was bought with a grant (to H.N.D.) from the Central Research Fund, London University. Thanks are due to Mrs. Marion Nixon who gave skilled technical assistance.

Circulation Research, Volume VIII, May 1960

Table 6

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<th>Decrease</th>
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</table>

*The numbers refer to the number of tests that were performed.

Summario in Interlingua

Le constante infusion intravenoso de epinephrina ha nullo effecto super le resistoutia pulmono-vascular (R.P.V.) de catos in anesthesia per chloralose, tanto quando le torax es aperte e ventilation artificial es usate como etiam quando le animal respira naturalmente. Le administration del mesme dose de epinephrina post ablation del ganglionos stellate e del ganglionos sympathetic superior thoracic 4 a 5 es capace a causar un augmento de R.P.V. Le ablation de ille ganglionos sin le administration de epinephrina resulta in un reduction de R.P.V. Le stimulation del branca cardiaca ab ambe ganglionos stellate causar un augmento o un reduction del R.P.V. Augmentos de R.P.V. essent producita per le infusion intravenoso de norepinephrina.

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Pulmonary Vasomotor Responses to Epinephrine and Norepinephrine in the Cat: Influence of the Sympathetic Nervous System

HELEN N. DUKE and R. D. STEDEFORD

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