cellular exchange of potassium, for instance, transport of magnesium across the cell membrane is a relatively slow process.

Summary. Mg^{28} was injected intravenously into 11 pregnant rabbits between 28 and 30 days of gestation. Fetuses and placentas were removed at intervals ranging from 7 minutes to 26 hours. Maternal tissue uptake of Mg^{28} resembled that previously found in nonpregnant young adult rabbits, except that uptake in bone and muscle was slower. Concentration of Mg^{28} in the placenta rapidly rose above the maternal plasma level. Specific activity of magnesium in all fetal tissues studied reached a fairly constant value by 26 hours. Magnesium turnover in tissues of the fetus *in utero,* especially in bone and muscle, is considerably more rapid than that in the respective tissues of the mother.

Mg^{28} was supplied by Brookhaven Laboratory on allocation from U. S. Atomic Energy Comm.

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Systemic and Coronary Hemodynamic Effects of 2 Amino-4-Methyl Pyridine.* (26021)

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2 amino 4 methyl pyridine, also known as RA 1226, W 45, and Ascensil® is a sympathomimetic agent which has been reported to have a marked pressor effect in the experimental animal(1,2). At higher dosage levels (1-3 mg/kg) a moderate positive inotropic effect is reported(3). Furthermore the agent is reported to have a long period of action and no tachyphylaxis(2). The present study concerns the acute systemic and coronary hemodynamic effects of this agent.

Material and methods. The study was done in 10 mongrel dogs weighing between 19 and 26 kg. Anesthesia was secured by administration of 3 mg/kg of morphine sulphate subcutaneously followed one hour later by 0.25 ml/kg of a 50/50 mixture of Veterinary pentobarbital and Dial-Urethane.† During the ensuing 1 hour after administration of the anesthetic cardiac catheters were maneuvered fluoroscopically into the pulmonary artery, coronary sinus and the right atrium and Cournand needles were placed in the femoral arteries. Pressures were recorded by Statham Strain gages on the Gilson Macro-polygraph with means determined by electrical integration. PH was determined on whole blood with the Cambridge model R pH meter. Cardiac outputs were determined by the Fick principle simultaneously with the Hamilton indicator dilution method. Coronary blood flow was measured by the N_2O method. Blood gas analyses were done by the method of Van Slyke and Neill whereas expired air was analyzed by the method of Scholander. Formulae used for calculations are those used traditionally in measurements of coronary blood flow(4), ventricular work and vascular resistance were calculated by the usual formulae(5) without subtracting atrial pressure

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† Veterinary nembutal contains 60 mg pentobarbital /ml. Dial-Urethane was supplied by CIBA Pharm. Products, Summit, N. J., and contains Dial 100 mg/ml, Monoethylurea 400 mg/ml, and Urethane 400 mg/ml.

TABLE I. Hemodynamic Effects of Ascensil.

Parameter	Before ± SEM	After ± SEM	% change	p value <
Heart rate	88 ± 9.4	54 ± 8.5	−38.7	.001
Mean systemic art. pres.*	126 ± 5.3	149 ± 5.8	+18.3	.001
" pulmonary art. pres.*	12 ± 1.0	13 ± .9	+ 8.3	.3
" right atrial pressure*	1.6 ± .31	3.1 ± .52	+93.8	.001
Body O_2 consumption	116 ± 5.6	112 ± 6.4	− 3.4	.1
" R. Q.	.82± .02	.82± .02	—	—
Arterial O_2 content†	18.8 ± .5	18.2 ± .5	− 3.2	.2
A − VO_2 difference†	4.4 ± .2	6.7 ± .3	+52.3	.001
Art. − cor. sinus O_2 difference†	12.1 ± .9	12.3 ± .8	+ 1.7	.7
Cardiac output (l/min.)	2.7 ± .1	1.7 ± .1	−37.1	.001
Left ventricular work‡	4.7 ± .4	3.4 ± .2	−14.9	.01
Right " " ‡	.5 ± .06	.3 ± .04	−40.0	.01
Total periph. resist. (cg units)	3803 ±223	7329 ±274	+92.7	.001
" pulm. " (")	358 ± 33	642 ± 77	+79.3	.001
Coronary blood flow§	106 ± 7.2	78 ± 5.5	−26.4	.01
Cardiac O_2 consumption§	12.4 ± .6	9.4 ± .5	−24.2	.001
" R. Q.	.84± .01	.82± .03	− 2.4	.7
Coronary vascular resist. (units)	1.24± .08	1.99± .15	+60.5	.001
Index of efficiency‖	.38± .02	.37± .02	− 2.6	.3

* Expressed in mm Hg. † Expressed in ml/100 ml of blood. ‡ Expressed in kg meters /min. § Expressed in ml/100 g myocardium/min. ‖ kg meters of left ventricular work done/min. divided by ml of O_2 used/100 g of left ventricle/min.

since left atrial pressure was not determined. The drug was administered with a constant infusion apparatus at a rate of 4 mg/min starting 5 minutes before the second determination of cardiac output and continuing throughout measurement of both output and flow. One animal received slightly less drug because of technical difficulties with the infusion apparatus but the results were similar to the others and the data are included.

Results. Results are summarized in Table I. Heart rate was decreased by 38.7% ($p<0.001$) whereas mean arterial blood pressure was increased by 18.3% ($p<0.01$). Pulmonary arterial blood pressure was not increased significantly, but mid right atrial pressure increased 93.8% ($p<0.001$). Neither the minute volume of respiration, oxygen consumption, carbon dioxide excretion or body respiratory quotient changed significantly. Although arterial oxygen content did not change significantly there was a 20.7% decrease ($p<0.001$) in mixed venous oxygen content with a 52.3% increase ($p<0.001$) in the arterio-venous oxygen difference. Similarly the mixed venous carbon dioxide content did not change, but arterial carbon dioxide content decreased with a significant increase in the mixed venous-arterial carbon dioxide content (+51.5%, $p<0.001$). The coronary sinus blood oxygen content did not change, nor did the arterial coronary sinus oxygen difference. Neither femoral arterial nor coronary sinus blood pH changed significantly. Hemoglobin and hematocrit were identical in the first and second studies. Cardiac output determined by the Fick principle decreased 37% ($p<0.001$) and output as measured by the Hamilton indicator dilution method decreased the same amount. Left ventricular work decreased 14.9% ($p<0.01$) and right ventricular work decreased 40% ($p<0.01$). At the same time total peripheral resistance increased by 92.7% ($p<0.001$) and pulmonary vascular resistance increased 79.3% ($p<0.001$). Coronary blood flow decreased (−26.4%, $p<0.01$). Myocardial oxygen consumption per 100 g decreased by 24.2% ($p<0.001$), but cardiac respiratory quotient did not change. Coronary vascular resistance increased 60.5% ($p<0.001$) and the calculated index of efficiency remained unchanged.

Discussion. Previous investigation has shown that Ascensil® produces an increase in cardiac output in both compensated and decompensated rat heart-lung preparations(1). No chronotropic effect was shown, but a positive inotropic effect was demonstrated(1,2,3). Intravenous administration of this agent in the dog produced bradycardia and an increase in blood pressure presumed to be due to increased peripheral vascular resistance(2). No

tachyphylaxis occurred in contrast to many of the sympathomimetic series of drugs(2). Perfusion of the coronary vessels by the drug was associated with a variable response as determined by the Rein flow meter(3).

The present investigation has shown a remarkable increase in systemic, pulmonary, and coronary vascular resistance after administration of 2 amino-4-methyl pyridine. The bradycardia is presumed to be vagal secondary to increased peripheral vascular resistance and elevated systemic arterial pressure, since it can be blocked by atropine.

Contrary to the effects of some sympathomimetic agents no increase in body or cardiac oxygen consumption was produced by administration of 2 amino-4-methyl pyridine. The reduction in cardiac output was sufficient to decrease left ventricular minute work even though calculated stroke work and systemic arterial pressure were increased. In line with reduced cardiac work and slower cardiac rate, coronary blood flow and myocardial oxygen consumption decreased. That there was no significant change in cardiac efficiency, seems particularly significant in view of the decrease in cardiac output, since work against increased pressure is reported to be more costly to the myocardium than work induced by increased flow(6). This maintenance of efficiency is most probably attributable to the decrease in cardiac rate since it has been shown that cardiac rate is inversely related to efficiency(7).

Conclusion. 1. The systemic and coronary hemodynamic effects of Ascensil®, (2-amino-4-methyl pyridine) also known as RA 1226 and in the German literature as W 45, has been investigated in 10 mongrel dogs. 2. Its administration has been associated with a marked increase in peripheral, pulmonary and coronary vascular resistance accompanied by an increase in peripheral arterial and right atrial pressure. 3. Considerable reduction occurred in cardiac output, coronary blood flow and myocardial oxygen usage per 100 g per minute, subsequent to its administration. 4. Calculated cardiac efficiency, body oxygen consumption, total body respiratory quotient and cardiac respiratory quotients were unchanged.

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Glyceride Content of Human and Canine Red Blood Cells. (26022)

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During studies on clearance of intravenously administered fat emulsions, chemically direct measurements were made of the tri-, di-, and monoglyceride content of washed red blood cells. Because of some unusual and interesting observations, it was believed desirable to report the results of these studies.

Methods. Plasma was separated from the cells of heparinized whole blood samples in a 5°C refrigerated centrifuge. The cells were washed with an equal volume of 0.9% NaCl solution, recentrifuged at 2260 g for 30 min-