plasmatic influences on O_2 affinity. In solution the hemerythrin O_2 affinity is, however, distinctly higher (P_{50} near 7.5 mm at 15 °C, see figure, b). A similar influence of lysis has been observed in hemerythrin from the sipunculids *Dendrostomum* and *Phascolosoma*[14,15], suggesting that erythrocytic factors may depress the O_2 affinity of hemerythrins as do allosteric phosphate cofactors with hemoglobin in vertebrates.

In the absence of a Bohr effect and cooperative O_2 binding, *Priapulus* hemerythrin differs from that of brachiopods *(Lingula)*[16], but resembles sipunculid hemerythrins, which generally, however, have higher O_2 affinities[17,18]. The relatively low O_2 affinity, however, resembles that found in the annelid *Magelona* (P_{50} = 13 at 15 °C)[19].

The O_2 affinities suggest that *Priapulus* hemerythrin can only load O_2 when the ambient O_2 tension is high, temperature is low, and/or the uptake of O_2 is efficient, limiting the fall in O_2 tension across the respiratory surfaces. However, measurements of coelomic O_2 tensions of live animals exposed to water with atmospheric O_2 tensions for periods exceeding 1.5 h, demonstrate the occurrence of steep O_2 tension gradients across the body walls of *Priapulus*[7]. The present findings thus correlate neatly with the observed restriction of *Priapulus* in nature to waters of low temperature and high O_2 tension[4,5].

Our data suggest that *Priapulus* hemerythrin cannot play an important role as a continuous transporter of O_2 from the respiratory surfaces to the metabolizing tissues. The pigment is extravascular and moreover lacks a sigmoid, pH dependent O_2 equilibrium curve – factors which enhance O_2 unloading in acid tissues by vascular pigments. It is, however, probable that the pigment will play a role as a short-term store of O_2, tiding over infrequent bouts of activity associated with burrow ventilation, anal appendage contractions and coelomic fluid mixing[6,7]. O_2 uptake rates of 15–20 ml · kg^{-1} · h^{-1} at 10 °C suggest that the measured pigment-bound O_2 could sustain the animal's respiration for about 25 min in well aerated water[7].

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Baroreceptor reflex sensitivity after acute blood volume expansion in anesthetized dogs[1]

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Summary. Changes in heart rate resulting from mechanically induced changes in arterial blood pressure were used as a measure of baroreflex sensitivity. This sensitivity was shown to be decreased after volume expansion.

In conscious dogs, rapid blood volume expansion causes an increase in mean arterial pressure, pulse pressure and heart rate[2]. The heart rate response is known as the Bainbridge reflex[3]. Since baroreceptor reflex characteristics would predict a decreased heart rate in response to an increased arterial pressure, a modification of these reflex characteristics after volume expansion has been postulated. To determine the sensitivity of the baroreflex, Vatner et al.[2] measured the relationship between the cardiac pulse interval and the systolic arterial blood pressure following an i.v. injection of methoxamine in conscious dogs. The increase in cardiac pulse interval for a given increase in arterial pressure was reduced after volume expansion. The authors concluded that the baroreflex sensitivity was decreased. The same conclusion was reached by Stinnett et al.[4] in a study on anesthetized rabbits. These authors found that heart rate responses to left aortic nerve stimulation were reduced after volume loading.

It was the purpose of this research to use mechanically induced blood pressure changes to determine the sensitivity of the baroreceptor reflex before and after blood volume expansion.

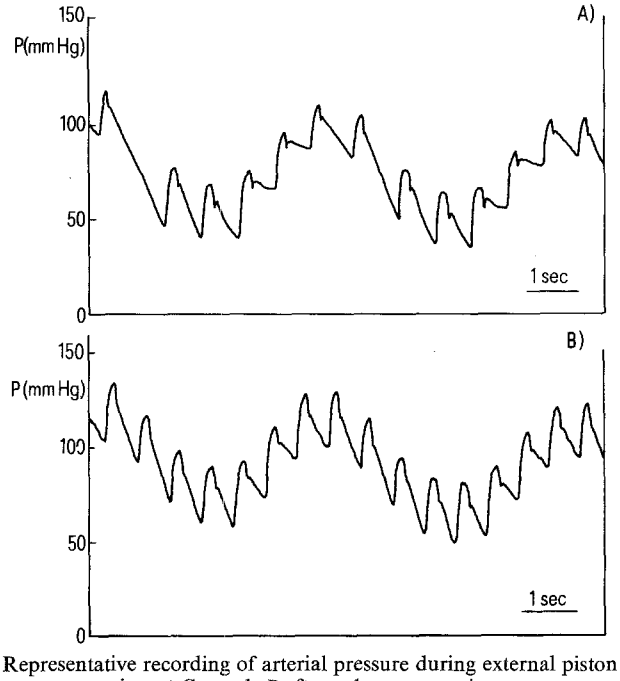
Methods. Experiments were performed on 6 mongrel dogs weighing from 18 to 25 kg. The dogs were premedicated with morphine sulfate (2 mg/kg) and anesthetized with sodium pentobarbital (20 mg/kg). Central venous pressure was monitored using a Statham (P23AC) manometer connected to the thoracic vena cava at the level of the right atrium by way of a cardiac catheter. Blood volume was determined in all animals at the onset of the experiment, using Evans Blue dye (T 1824). Aortic pressure was measured with a Pieper catheter-tip pressure gauge[5]. This gauge

SD of the normalized cardiac pulse intervals before and after volume expansion

Experiment	Control	Volume expansion
1	0.334	0.134
2	0.363	0.260
3	0.114	0.021
4	0.332	0.150
5	0.227	0.210
6	0.089	0.048

was inserted via the right femoral artery and positioned in the descending aorta at mid-heart level with the aid of a fluoroscope. Aortic pressure was digitized at a rate of 200 samples per sec and displayed on a digital oscilloscope (Nicolet Explorer III). Numerical values of time and pressure were available for each sample point. This oscilloscope was also capable of storing the data of each experimental run on magnetic disc for later analysis.

To test the sensitivity of the baroreflex, mean arterial blood pressure was fluctuated slowly around its mean. For this purpose, a sinusoidal piston pump was attached to the abdominal aorta by means of a cannula inserted into the left femoral artery. In operation, the cycle period was 5 sec and the stroke volume was adjusted to cause a 50 mmHg swing in mean arterial pressure. The increase in blood pressure caused a decrease in heart rate while the decrease in blood pressure caused an increase in heart rate (figure A). Because of the delay in the baroreflex the highest cardiac pulse interval (CPI) occurred after the peak in the arterial pressure. CPI's were measured during the pump cycle and normalized by dividing them by the CPI of the steady state prior to the pumping. As a measure of the baroreflex sensitivity the variability of all normalized CPI's for 1 pumping cycle was determined by calculating their SD.



Representative recording of arterial pressure during external piston pump operation. *A* Control; *B* after volume expansion.

In all animals, the effect of volume loading on the baroreflex sensitivity was evaluated by expanding the animal's blood volume by 30% with dextran 70 (Macrodex, Pharmacia Lab). The SD of the normalized CPI's obtained during pumping was again determined.

Results. The figure illustrates a representative experiment. Figure A depicts the effects of pumping on arterial pressure and heart rate under control conditions, while figure B shows the same variables after volume loading. The variability of the CPI's is significantly decreased after volume loading indicating a lower sensitivity of the baroreflex.

Volume expansion caused an average increase in central venous pressure of 4.0 ± 1.1 mm Hg and an average rise in mean arterial pressure of 19.2 ± 6.9 mm Hg. The heart rate increased by 6.8 ± 3.2 beats/min. The table presents the values of the SD of the normalized CPI's before and after volume expansion. The variability of the CPI's was lower after volume expansion in every experiment. Using a paired t-test it was shown that the decrease in this variability was significant at the 0.02 level.

Conclusion. The consistent and statistically significant decrease in the variability of the normalized CPI's after volume loading indicates that volume loading decreased the sensitivity of the heart rate response to blood pressure changes. It does not indicate the site of the reflex arch at which the changes occur. The results support the hypothesis proposed by Vatner et al.[2] and Stinnett et al.[4] that volume loading decreased the baroreflex sensitivity. However, in their experiments artifacts due to nerve stimulation or methoxamine effects could not be excluded. The purely mechanical stimulus used in the experiments presented here excludes these possible artifacts.

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The effect of caerulein on epithelial growth in the mouse gall bladder[1]

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Summary. Caerulein, a synthetic decapeptide, was injected into mice in order to study its effect on DNA-synthesis activity in the gall bladder epithelium. Histoautoradiography after the injection of labeled thymidine was used. Higher labeling indices were observed at 8, 12 and 24 h after caerulein injection. These data indicate that caerulein, apart from its cholecystokinetic effects, exerts a trophic effect on the gall bladder mucosa.

Several gastrointestinal polypeptide hormones have been shown to influence epithelial cell replication in the gastrointestinal mucosa or in the pancreas. Cholecystokinin (CCK-PZ) is known to promote DNA synthesis and growth in the pancreas[3], and to stimulate the secretion of glycoproteins in mouse gall bladder epithelium[4]. The decapeptide caerulein, which shows a striking resemblance to the C-terminal octapeptide of porcine CCK-PZ, has properties similar to CCK-PZ on the pancreas[5]. In the present study, caerulein was administered to mice in order to study its effect on DNA synthesis in the gall bladder mucosa, using a histoautoradiographic method.