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OBSERVATIONS ON LONG BONE MEDULLARY PRESSURE IN RELATION TO MEAN ARTERIAL BLOOD PRESSURE IN THE ANAESTHETIZED DOG

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To study the influence of variations in mean arterial pressures (MAP) on long bone medullary pressures, seven anaesthetized dogs were investigated. The medullary pressures were measured in the epiphyseal, the metaphyseal and the diaphyseal regions and remained rather constant when MAP was above 80 mmHg. Below this level of MAP a statistically significant ($P < 0.01$) reduction of the medullary pressures was seen. Comparing the mean medullary pressures obtained with ranges of MAP of 81–100 mmHg (the control medullary pressures) and of 61–80 mmHg, the greatest decline was seen in the epiphyseal and the diaphyseal regions, from 25.2 mmHg to 8.1 mmHg and from 26.7 mmHg to 8.3 mmHg, respectively. The corresponding decrease in the metaphyseal region was from 18.9 mmHg to 10.9 mmHg. The mean values of intraosseous pressure measured by our technique were between 20–30 mmHg and this is in accordance with measurements in normal humans found by other authors.

Key words: aetiology; blood supply; bone; femur head; osteoarthritis; physiology; regional blood flow

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Long bone medullary pressure is an indirect expression of the medullary blood flow. A low medullary pressure means a low blood flow and a high medullary pressure is related to an increased blood flow or an intraosseous congestion of blood due to obstruction of the venous outflow (Shaw 1963, Azuma 1964, Shim 1968). The influence of venous obstruction on the medullary pressure in long bones has been described under both normal and pathological conditions (Stein et al. 1957, Keck & Kelly 1965, Arnoldi et al. 1972).

The purpose of this study was to investigate the influence of variations in the arterial blood pressure on the epiphyseal, metaphyseal and diaphyseal medullary pressures in long bones in the anaesthetized dog.

MATERIAL AND METHODS

Seven mongrel dogs, weight 29–42 kg, and all above 2 years of age were investigated. Anaesthesia, technique and some of the monitoring equipment have previously been described (Eriksen et al. 1979). For continuous measurements of arterial blood pressure a catheter was placed in the left brachial artery. Central venous pressure was measured in the superior caval vein via a catheter introduced into the left brachial vein. Recording of arterial and central venous pressures were done using an Ellab M5-BCM amplifier and a Statham P 23Db transducer.

The medullary pressures were measured through metal cannulas (diameter 2 mm) placed with their tips centrally in the bone marrow of the left femoral epiphysis, the right femoral metaphysis and the right humeral diaphysis. To penetrate the bone cortex, drilling was necessary and a 1.8 mm drill was used for this purpose. No

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leakage of medullary blood was seen when the metal cannulas were placed in the bones. The correct positioning of the cannulas was confirmed by X-ray. Simonsen & Weel AE 840 transducers and Simonsen & Weel BAP 001 amplifiers were used for the continuous recording of the medullary pressures.

Using different levels of halothane anaesthesia and different intravenous loading with saline, variations in the arterial blood pressure were accomplished. Equilibration periods of at least 10 minutes during which the mean arterial pressure (MAP) remained constant were used before sampling of data. Registration of the medullary pressure in epiphyseal, metaphyseal and diaphyseal regions were performed at different levels of MAP. During the study the arterial oxygen tension and the arterial carbon dioxide tension were maintained at normal levels and monitored using a continuous blood gas analyser (Henningsen 1968).

All medullary pressures given are medullary perfusion pressures, i.e. the measured medullary pressure minus the central venous pressure. This correction has been made because of the obvious influence of venous stasis on the magnitude of medullary pressures in long bones (Shaw 1963, Keck & Kelly 1965).

In presenting our results various ranges of MAP have been chosen: < 61 mmHg, 61–80 mmHg, 81–100 mmHg, 101–120 mmHg, and >120 mmHg. Within these ranges both the mean values ± s.e. mean of MAP and the mean values ± s.e. mean of the corresponding medullary pressures in the epiphyseal, the metaphyseal, and the diaphyseal regions have been calculated.

The Mann-Whitney rank sum test for unpaired data has been used for the statistical analysis of the results obtained. The level of significance for differences was chosen as $P < 0.05$.

RESULTS

Table 1 summarizes the results of our study and Figures 1, 2 and 3 show the epiphyseal, metaphyseal and diaphyseal medullary pressure variations in relation to the different ranges of MAP. Within the normal range of MAP (81–100 mmHg) the corresponding medullary pressures were the control values.

Looking at the ranges of MAP lying above 80 mmHg medullary pressures within the respective regions were rather constant with mean epiphyseal medullary pressures of 25.2–30.7 mmHg, mean metaphyseal pressures of 18.9–20.5 mmHg, and mean diaphyseal pressures of 24.3–26.7 mmHg. However, with a range of MAP of

Table 1. Long bone medullary pressures ($P_{epiphysis}$, $P_{metaphysis}$, and $P_{diaphysis}$) in seven anaesthetized dogs in relation to different values of the mean arterial blood pressure (MAP). Mean values ± s.e. mean of MAP and mean values ± s.e. mean of the corresponding medullary pressures within the ranges have been calculated. The control medullary pressures are the mean medullary pressures within the normal range of MAP (81–100 mmHg)

Number of observations	Mean arterial pressure (MAP) (mmHg) Range	Mean ± s.e. mean within the range	Medullary pressures (P) (mmHg) $P_{epiphysis}$ Mean ± s.e. mean	$P_{metaphysis}$ Mean ± s.e. mean	$P_{diaphysis}$ Mean ± s.e. mean
14	61	47.1 ± 3.9	5.9 ± 1.0 ^{xxx}	8.3 ± 1.0 ^{xxx}	8.1 ± 1.2 ^{xxx}
14	61–80	72.9 ± 1.3	8.1 ± 1.8 $^{xxx}_{+}$	10.9 ± 1.0 ^{xxx}	8.3 ± 1.5 ^{xxx}
18	81–100 (normal range)	95.3 ± 1.2	25.2 ± 3.5 (control medullary pressures)	18.9 ± 2.0	26.7 ± 3.0
29	101–120	112.9 ± 0.9	30.7 ± 2.6 $_{+++}$	20.5 ± 1.5	25.7 ± 1.7 $_{+}$
17	120	135.3 ± 3.9	23.2 ± 3.5	17.5 ± 1.5	24.3 ± 2.9

Within the respective medullary regions $_{xxx}$: $P < 0.01$ indicates statistical significance of differences from control medullary pressures.

Within the respective ranges of MAP $_{+}$: $P < 0.005$ and $_{+++}$: $P < 0.01$ indicate statistical significance of difference of metaphyseal medullary pressure from epiphyseal or diaphyseal medullary pressure.

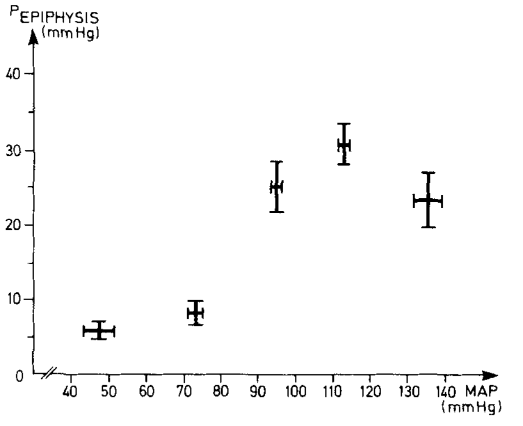

Figure 1. Long bone epiphyseal medullary pressures ($P_{epiphysis}$) in relation to the mean arterial blood pressure (MAP). Mean values ± s.e. mean of the epiphyseal medullary pressures obtained within different ranges of MAP (<61, 61–80, 81–100, 101–120, and >120 mmHg) are shown in relation to the mean values ± s.e. mean of MAP within these ranges.

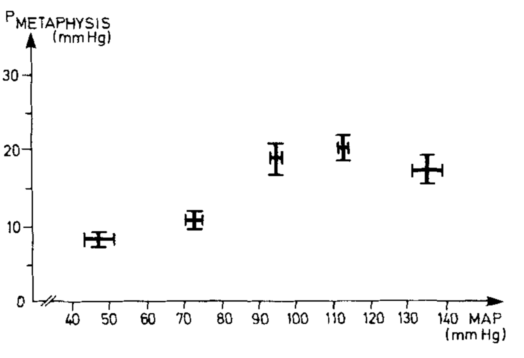

Figure 2. Long bone metaphyseal medullary pressures ($P_{metaphysis}$) in relation to the mean arterial blood pressure (MAP). Mean values ± s.e. mean of the metaphyseal medullary pressures obtained within different ranges of MAP (<61, 61–80, 81–100, 101–120, and >120 mmHg) are shown in relation to the mean values ± s.e. mean of MAP within these ranges.

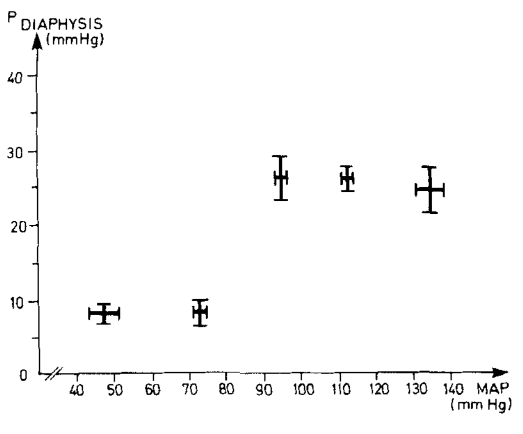

Figure 3. Long bone diaphyseal medullary pressures ($P_{diaphysis}$) in relation to the mean arterial blood pressure (MAP). Mean values ± s.e. mean of the diaphyseal medullary pressures obtained within different ranges of MAP (<61, 61–80, 81–100, 101–120, and >120 mmHg) are shown in relation to the mean values ± s.e. mean of MAP within these ranges.

61–80 mmHg a significant ($P < 0.01$) reduction of the medullary pressures in all three regions was seen. Comparing the mean medullary pressures with a range of MAP of 81–100 mmHg the greatest decline was seen in the epiphyseal and the diaphyseal regions, from 25.2 mmHg to 8.1 mmHg and from 26.7 mmHg to 8.3 mmHg, respectively. The corresponding decrease in the metaphyseal region was from 18.9 mmHg to 10.9 mmHg.

When MAP was between 101 and 120 mmHg the mean metaphyseal medullary pressure (20.5 mmHg) was significantly lower than both the mean epiphyseal medullary pressure (30.7 mmHg) and the mean diaphyseal medullary pressure (25.7 mmHg). By contrast, with a range of MAP of 61–80 mmHg the mean metaphyseal medullary pressure (10.9 mmHg) was significantly higher than the mean epiphyseal medullary pressure (8.1 mmHg). Apart from this no significant differences were observed between mean epiphyseal, metaphyseal or diaphyseal pressures within the various ranges of MAP.

DISCUSSION

From this study performed on anaesthetized dogs it seems that long bone medullary pressures exhibit a great constancy and are independent of arterial blood pressures as long as the MAP remains above 81 mmHg. Below an MAP of 81 mmHg a significant lowering of the medullary pressures is seen. This relation could be demonstrated in the epiphyseal, the metaphyseal and the diaphyseal regions.

The medullary pressures found at levels of MAP above 81 mmHg correlate well with medullary pressures obtained from the proximal femur in humans (Arlet et al. 1972).

The pressures measured by means of metal cannulas introduced into the bone marrow are probably in their origin a mixture of pressures, and provoked by the pulsations in small arteries, arterioles and capillaries, due to the fact that these structures are surrounded by a stiff, non-elastic, cortical bone (Hawk & Shim 1970). For normal function of medullary tissues to be maintained during various physiologic conditions, one might expect the presence of some sort of regulatory mechanism, which within wide limits of MAP would secure a stable and constant intramedullary pressure. If no such regulatory mechanism were present, increasing arterial pressures might result in increasing medullary pressures and hereby gradually a severe compression of the medullary tissue.

As mentioned earlier long bone medullary pressures give an indirect expression of medullary blood flow rate (Azuma 1964, Shim 1968). In most tissues, muscles above all, the blood flow rate is controlled in proportion to the need for nutrition, oxygen in particular (Stainsby 1973). The regulation of the blood flow rate seems to occur locally by a direct influence of oxygen (Guyton et al. 1972). This local blood flow regulatory mechanism is rather sensitive to external factors, but during normal resting conditions is probably sufficient to maintain stable pressure relationships within the bone marrow. However, during variations in the arterial blood pressure the local regulatory mechanism might be insufficient to a degree of 15 per cent (Guyton et al. 1972). Drinker & Drinker (1916) have shown that the arterioles of the bone marrow are supplied with sympathetic nerve fibres with a vasoconstrictor function. In most tissues the autonomic nervous system is not involved in local blood flow regulation during normal function of circulation, but provides an increased effectiveness of control under stress conditions such as exercise or haemorrhage (Granger & Guyton 1969). A specific function of the sympathetic vasoconstrictor nerve fibres supplying the medullary arterioles has till now not been described, but perhaps they are involved in an active regulatory mechanism which prevents a high medullary pressure during increases in arterial blood pressure.

The significant lowering of the medullary pressures seen at mean arterial pressures below 81 mmHg is probably the result of the body's attempt to maintain sufficient blood flow to the most vital organs, such as heart and brain, and caused by mobilization of both nervous and hormonal blood flow and blood pressure control mechanisms.

From our results it seems that a rather constant epiphyseal, metaphyseal, and diaphyseal medullary pressure is maintained at normal or above normal ranges of MAP. However, decreases of MAP to levels below 81 mmHg cause great pressure falls within the bone marrow of long bones. Probably low medullary pressures mean insufficient medullary blood flow and subsequently insufficient oxygenation and potential necrosis. Possibly the observations made in the present study may give a clue as to the cause of some orthopaedic conditions in which an "idiopathic" necrosis is involved.

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