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BLOOD FLOW TO THE KNEE JOINT OF THE DOG. EFFECT OF HEATING, COOLING AND ADRENALINE

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Little is known of the physiology of the blood flow to the joints, mainly through lack of a suitable animal preparation for study. Attempts have been made to estimate the joint blood flow indirectly in man by the measurement of intra-articular temperatures (Horvath & Hollander, 1949), and by the application of the plethysmograph to a knee segment (Bonney, Hughes & Janus, 1952).

METHOD

Nine adult dogs anaesthetized with pentobarbitone sodium (50 mg/kg) were used. The main source of the blood supply to the knee joint of the dog is the articular branch of the femoral artery, corresponding to the musculo-articular branch of the descending genicular artery of the human. This vessel arises from the femoral artery just before it passes under cover of the semimembranosus muscle and descends along the posterior border of the vastus medialis muscle, supplying one or two branches to it, and finally enters the medial aspect of the knee joint (Fig. 1).

Measurement of blood flow. One hindlimb was skinned, the skin being removed down to a point below the knee joint. After all skin vessels had been tied off, the skin was replaced and the joint kept at rectal temperature by surrounding it with pads of lint soaked in warm 0·9 % (w/v) saline. The femoral artery and its branches were exposed and ligatures tied at point (*a*) (Fig. 1) on the femoral artery, at (*b*) the origin of the saphenous artery, and on the small muscular branches (*c*). The afferent and efferent leads of a flow meter were inserted into the femoral artery proximal to the origin of the saphenous artery. Heparin (1000 units/kg) was then given intravenously and flow established through the bubble flow meter (Bruner, 1948). Blood flow was measured by timing with a stop-watch the transit of a small air bubble between fixed points, enclosing a known volume, on the flowmeter tubing.

In this preparation almost all the blood flowing through the meter reaches articular structures only. This was checked in all experiments when, just before the dog was killed, the dyestuff Monastral Fast Blue (BNVS Paste, I.C.I.) was injected into the flowing blood at its point of entry into the meter. Dissection then showed that most of the dye was located in the blood vessels in the synovial membrane; lesser amounts were found in the capsular ligament, accessory ligaments, and the extremities of the bones. A very small amount of dye was sometimes found in the insertion of the rectus femoris muscle to the patella.

Heating of the joint was accomplished by radiant heat, by the application of packs soaked in hot (65° C) saline, or by circulating water at 60° C through a tube of thin rubber dam wrapped round the joint. In one dog short-wave diathermy was employed. The joint temperature was lowered by packing crushed ice around the external surface.

Injections of adrenaline were made into the distal tube of the flowmeter.

Blood pressure was recorded from the carotid artery using a mercury manometer, and flows were measured only during times when the blood pressure remained constant.

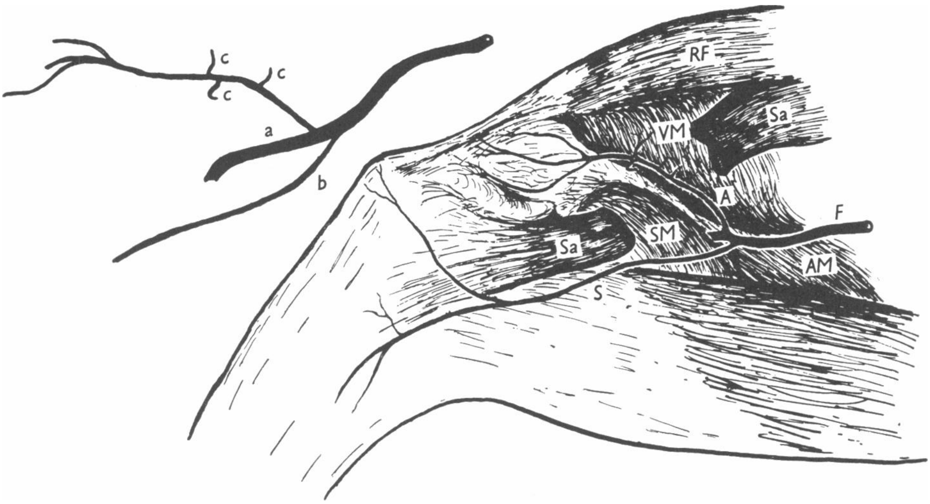

Fig. 1. Dissection of medial aspect of right knee-joint of a dog with a portion of sartorius removed. The inset shows the femoral artery and its main branches. *F*, femoral artery; *S*, saphenous artery; *A*, articular branch of femoral artery; *RF*, rectus femoris; *VM*, vastus medialis; *Sa*, sartorius; *SM*, semimembranosus; *AM*, adductor magnus. Ligatures are tied at *a*, *b* and *c* to isolate joint blood flow.

RESULTS

Resting blood flow. Table 1 shows the rate of blood flow to the knee joint structures of the anaesthetized dog (1·5–7 ml./min). These figures are taken from dogs showing a steady level of flow, maintained for longer than 30 min, with the knee-joint temperature at its normal value.

Effect of heating. When the temperature of the joint was increased the blood flow increased (Fig. 2). The temperature must be markedly raised to obtain a measurable increase in flow, changes of less than 10° C having little effect. Even with a high external temperature (60–65° C) the percentage change in flow was small compared with the values obtained for cooling (Table 1). On removing the heat source the flow returned to the control level.

Effect of cooling. Rapid cooling of the joint by ice packs caused a considerable diminution of flow (Fig. 3, Table 1). The flow decreased steadily to about half the initial value and thereafter remained constant. However, in some experiments the flow fell, rose, and fell again; a type of behaviour also described for skin blood vessels in response to low temperatures (Lewis, 1929). After removing the ice packs the flow returned slowly to the control level, partly owing to the delay in returning to normal temperature; but this delayed return also occurred when the joint was quickly restored to its resting temperature.

Adrenaline injected intra-arterially close to the knee joint in doses of 0·1 μg produced a marked vasoconstriction of short duration. Doses of 0·2 μg and above increased this effect and caused the flow to stop completely for a short time (Fig. 4).

TABLE 1. The blood flow to the knee joint of the anaesthetized dog. Resting values and percentage changes in response to heating and cooling the joint

Dog no.	Wt. (kg)	Mean resting flow (ml./min)	Maximum percentage change in flow: Heating 60° C.	Maximum percentage change in flow: Cooling 0° C
4	9·5	1·5	+57	−62
5	31	3·9	+40	−55
6	13	7·0	+50	—
7	25	3·5	+20	−60
8	17	5·0	+22	−55
9	22	2·5	+15	−50

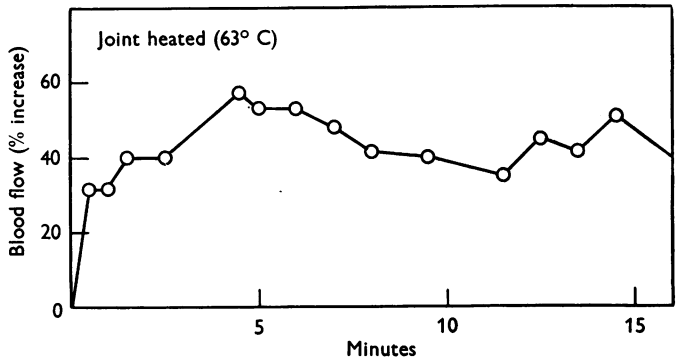

Fig. 2. Percentage increase in blood flow throughout a period of heating the knee-joint to 63° C.

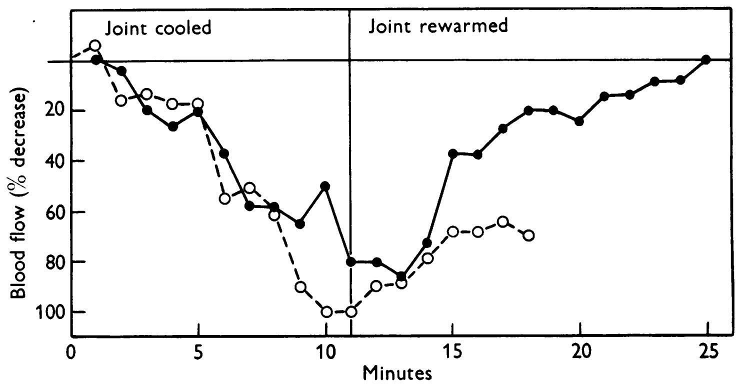

Fig. 3. Two experiments on the same dog to show percentage decrease in blood flow when the joint was cooled with ice packs and subsequently rewarmed to 39° C.

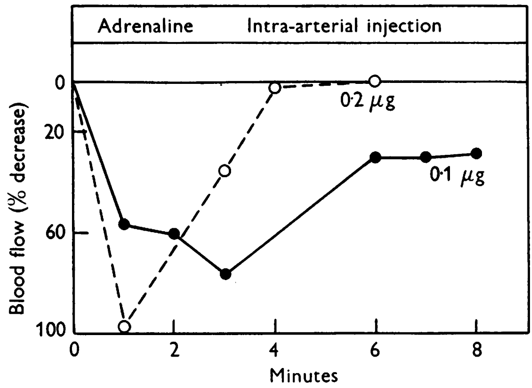

Fig. 4. Percentage decrease in joint blood flow produced by close intra-arterial injection of adrenaline in doses of 0·1 and 0·2 μg.

DISCUSSION

The results described above show that, at least in regard to response to temperature change and to intra-arterial adrenaline, the joint blood vessels behave like those of the skin. The relatively high rate of blood flow suggests that the articular tissues are possibly not as inert in their metabolic processes as is generally supposed, and it would be interesting to know the relationship between this high flow and the rate of secretion of the synovial fluid.

The behaviour of the joint circulation when the external temperature of the joint is increased was studied by Horvath & Hollander (1949), whose work implies that the blood vessels here dilate in response to cold and constrict to heat. They found that the application of a hot pack to the knee joint in man caused a fall in the intra-articular temperature, while cold packs raised this temperature. This is not supported by the results of Hunter & Whillans (1951), using the same technique, since they observed that the joint temperature fell when their subject was exposed to cold, and this they attributed to a reflex superficial vasodilatation resulting in a short period of excessive heat loss. According to our results, however, the decrease in the intra-articular temperature would be due to the constriction of the joint vessels in response to cooling. We have not observed joint blood vessels behave in the way supposed by Horvath & Hollander. In their plethysmographic study of flow in a knee segment Bonney *et al.* (1952) found that cooling the segment enclosed in the plethysmograph resulted in a fall in blood flow and heating gave the reverse effect. Because they found a similar decrease in flow when the circulation to the skin of the segment was suppressed by adrenaline iontophoresis, and further found that after this procedure cooling no longer decreased the blood flow, Bonney *et al.* suggested that there might be different reactions to cooling by articular and the superficial vessels. The direct measurement of flow in our preparation does not support this view.

The use of the knee segment in the plethysmograph as a method for measuring the joint blood flow is open to the obvious disadvantage that considerable amounts of other tissues are enclosed. Bonney *et al.* give the figure of 54 % by volume as tissue other than skin and muscle, but of this only 15 % of the total volume is true joint tissue. The results we have obtained with adrenaline throw some doubt on the accuracy of the method of Bonney *et al.* The direct determination of the flow response to intra-arterial adrenaline shows a simple vasoconstriction of the joint vessels. The plethysmographic method used on the knee segment showed a dilatation as the response to intravenous injection of 5 μg of adrenaline. This result of Bonney *et al.* disagrees with the present result, and we believe that the increase in flow which they observed may have been in the muscle enclosed in their plethysmograph. It is significant that the response they found to adrenaline shows a pattern which is typical of muscle and is not found elsewhere (Allen, Barcroft & Edholm, 1946). For these reasons it would appear that the use of the whole knee mass is not satisfactory for the determination of joint blood flow.

The knee joint of the dog prepared in the way described above has been found to provide a simple and satisfactory preparation for the study of the blood flow to articular structures, and offers a method for study of a relatively little known part of physiology.

SUMMARY

1. A preparation is described for the direct measurement of blood flow to the knee joint of the dog.

2. The resting level of flow to the knee joint varied in six dogs from 1·5–7 ml./min.

3. A rise in the temperature of the joint caused an increase in blood flow, and a fall in temperature a decrease in flow.

4. Adrenaline administered intra-arterially caused a constriction of joint blood vessels.

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