Cardiovascular Performance of Alaska Sled Dogs during Exercise

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ABSTRACT
Radiotelemetry was used to study regional blood flow distribution in Alaska sled dogs during cross-country runs. Doppler ultrasonic flowmeter transducers were chronically implanted on the coronary, renal, and mesenteric arteries, terminal abdominal aorta, and ascending aorta or pulmonary artery, and a miniature blood pressure gauge was installed in the aorta or carotid artery. Flow and pressure data telemetered from dogs running on the trail were received and recorded remotely. The heart rate, 40 to 60/min in sleeping dogs, increased to 80 to 100/min when the dogs were ambulatory and to 100 to 150/min when the dogs were excited before a race. Heart rate accelerated to 300/min at the start of exercise and commonly remained at that level throughout prolonged runs. Aortic blood pressure averaged 130/90 mm Hg at rest, but the systolic pressure often exceeded 300 mm Hg when the dogs were running. A transient drop in mean pressure occurred at the onset of running, but mean pressure during sustained exercise was practically identical to that at rest. Flow in the terminal aorta increased 9 to 12 times and coronary flow 5 to 6 times, but mesenteric and renal flows were unchanged during violent, prolonged exercise. These findings contrast with diminished visceral flows recorded in exercising humans and suggest that compensatory redistribution of flow is not a significant reserve mechanism in these animals during exercise.

ADDITIONAL KEY WORDS flow distribution telemetry remote monitoring regional blood flow coronary flow cardiovascular reserve

Legendary feats of stamina and endurance have been attributed to teams of Alaska sled dogs. However, the performance of modern sled dog teams trained for competitive racing often surpasses even these standards. For example, in the 1966 North American Sled Dog Championships, a 12-dog team pulled a sled and driver 20.3 miles cross country in subzero weather in 77 minutes, and the elapsed time for three heats totalling 71 miles was 280 minutes, i.e., the entire team ran 71 consecutive sub 4-minute miles while under load. At the completion of such runs, the dogs show little evidence of fatigue and are often restless and willing to run even further. The remarkable exercise capacity of these animals implies that their cardiovascular reserve mechanisms are well developed. On this basis they are ideal models for investigation of adaptation to exercise.

During the past two winters we have studied the cardiovascular performance and regional distribution of blood flow in exercising Alaska sled dogs by a telemetry system developed especially for this purpose. The study was designed to test the hypotheses that blood flow to visceral beds is reduced during severe exercise and that the attendant redistribution of blood flow is an important mechanism in the cardiovascular adaptation to exercise. To test these hypotheses, devices for measuring and telemetering regional blood flows and blood pressures were chronically implanted in Alaska sled dogs. Changes in regional blood flows and blood pressures

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were then recorded while these dogs were pulling loaded sleds over cross-country trails during subzero temperatures.

Methods

These experiments were conducted on members of a sled dog team at the Arctic Aeromedical Research Laboratory in Fort Wainwright, Alaska. The team consisted of approximately 36 dogs which were worked regularly in shifts of 8 to 12 dogs on cross-country trails. The team was not of national championship caliber but performed creditably in local competitions.

Blood flowmeter and blood pressure transducers were chronically implanted in dogs that were regarded as representative of the team. All 18 dogs were subjected to laparotomy for installation of flow transducers on the left renal artery, the superior mesenteric artery, and the terminal abdominal aorta above its bifurcation. Eight dogs were also subjected to thoracotomy for implantation of flow transducers on the circumflex coronary artery and on the ascending aorta or pulmonary artery.

Blood velocity was measured with the Doppler Ultrasonic telemetry flowmeter. This instrument has an inherently stable, well-defined zero reference and provides an accurate measurement of phasic blood velocity. Previous studies have demonstrated that calibration of the flowmeter is linear both on the bench and in vivo (1-2). For these experiments, each transducer was calibrated in vivo against an external frequency standard over the range of 0 to 20 kHz, i.e., blood velocity 0 to 200 cm/sec. These calibrations were recorded on magnetic tape and were the basis for subsequent computations. The measured velocity may be converted to volume flow by simple arithmetic if the cross section of the vessel is fixed and its dimension known. In these experiments, the vessel wall was constrained by a close fitting, rigid transducer of known dimension that limited radial expansion and fixed the vessel caliber. Dimensions of the vessel lumen within the transducer were determined in some dogs by direct inspection of the cut vessel shortly after completion of the experiment. Similar measurements were made later in six dogs that were killed at intervals up to 19 months after the implants. We recognize that these static measurements are not a precise representation of the lumen diameter under dynamic conditions; for this reason, mean flow levels have been designated as calculated values. Since the demodulation technique used in these experiments eliminated the capability for direction sensitivity, measurements were limited to vessels in which reverse flow does not occur or constitutes a minimal fraction of the total.

Blood pressure was measured with miniature solid-state gauges implanted directly into the bloodstream (3). The frequency response of these gauges is flat to over 100 Hz, and they are linear over the range of 0 to 500 mm Hg. In six dogs the transducers were placed in the aortic arch; in the remainder, a transducer was inserted into the left carotid artery after surgical exposure 1 or more days before the study. The gauges were calibrated against a mercury manometer immediately before implantation and after removal and against a calibrated P23Db strain-gauge manometer while in vivo. All dogs were ambulatory within 12 hours after surgery; all were kept inside from 2 to 5 nights before being returned to their accustomed outdoor existence. Exercise runs were started after 1 week and were gradually increased in severity. After 6 weeks the performance of the animals subjected to surgery was indistinguishable from that of the control animals. Recordings were made during the seventh to tenth postoperative weeks.

During exercise runs the animals were fitted with a canvas backpack which had a saddle bag on either side for carrying the pressure-flow telemetry system (Fig. 1). An elastic bandage wrapped loosely around the animal's torso reduced motion of the saddle bags during running. A hole cut in the center of the pack allowed access to the lead wires as they emerged from the skin. For most applications the apparatus is powered by small self-contained mercury batteries. However, at the extremely low temperatures encountered (as low as $-40^\circ$C in these studies), these batteries failed rapidly. We therefore substituted a nickel-cadmium military aircraft battery carried in a warmed insulated box aboard the sled. Electrical power reached the dog by a cable running along the harness assembly.

For simultaneous telemetry of velocity and pressure, the output from each flowmeter and the pressure gauge modulated separate subcarrier oscillators. Signals from the subcarrier oscillators were summed, and this complex signal in turn modulated a 260-mHz oscillator-transmitter (4-5). The telemetered information was transmitted up to 3 miles and was received by a directional antenna erected on the roof of the Arctic Aeromedical Research Laboratory. The receiving station consisted of Nemms-Clarke 1302 B and DEI CP-20 telemetry receivers for demodulating the incoming signals and discriminators for recovering information from subcarrier oscillators. The demodulated FM signals representing velocity and pressure were recorded.

Made to our specifications by Konigsberg Instruments, Pasadena, California.
Top: One of the teams in action on a trail along the Chena River. Data were transmitted from the last dog in line, wearing a pack saddle. Lower left: Sled with box containing Ni-Cd battery powering the telemetry transmitter; the signal was radiated from the antenna mounted in front of the box. The backpack worn by the driver contains a 27-MHz transmitter for communication with the recording station. Lower right: One of the dogs with chronically implanted instruments used in this study. The dog is wearing a pack saddle containing electronic apparatus; an elastic wrap around the torso stabilized the packs.

For each exercise run, the team consisted of a leader and 5 to 12 dogs. Lengths of the runs varied from 1 mile to over 30 miles; the duration of the runs extended from a few minutes to over 2½ hours, and on several occasions, we tried to determine the dog's maximal endurance. Total weight of the sled, driver, and other equipment abroad was 169.6 kg, but the work of the animals with implanted instruments was sometimes further increased by adding extra weights, equivalent to 25% of the animal's body weight, to the saddle bag (Fig. 1).

The sample included recordings of both multiple and single velocities with and without simultaneously recorded blood pressure. We tried to obtain a sleeping or resting record in each animal before it was harnessed to a sled with the remainder of the team, since this latter activity was always accompanied by manifesta-
tions of excitement and anxiety. The dogs were then run at their maximal rate; to a large extent the pace was determined by the lead dog, which was always a control animal. On cross-country runs on the longer trails, stops were made only when necessitated by equipment failure; on the shorter trails, however, repetitive laps were required, and the dogs frequently slowed or stopped briefly at the completion of each lap.

**Results**

*Heart Rate.*—The heart rate in sleeping or resting sled dogs was typically 40 to 60/min, and a sinus arrhythmia was usually present. The rate increased to 80-100/min when the dog was alerted, stood up, or walked about. As soon as the dogs sensed that they were to run, they became excited, strained at their leashes, pawed the snow and ice, and frequently made false starts. Indeed, securing the sled and team with an ice anchor was necessary to prevent the team from running. During such excitement, the heart rate was usually 120 to 150/min. The onset of running, the rate typically accelerated immediately to over 300/min. Once a steady pace was established, the rate was usually maintained between 250 and 300/min; some dogs maintained rates around 300/min throughout an hour's run. When the dogs stopped running, the rate dropped sharply to about 150/min within the first minute and thereafter decreased more slowly as the resting level was approached.

*Blood Pressure.*—The resting aortic blood pressures in these dogs ranged between 150/100 mm Hg and 100/70 mm Hg and averaged 130/90 mm Hg; in those dogs in which pressure was measured by a gauge inserted into the carotid artery, the systolic peak was usually higher by 20 to 30 mm Hg. In 36 of 39 instances, the onset of exercise was accompanied by a transient decrease in mean blood pressure, usually 5 to 10 mm Hg, which recovered to control levels within 20 seconds after the onset. During exercise, the peak systolic pressure occasionally exceeded 300 mm Hg and the diastolic run-off was rapid, usually to a level below the resting control value, so that the pulse pressure was significantly increased. However, the mean blood pressure during sustained exercise was approximately that recorded at rest; in no case was mean blood pressure significantly increased during running.

![FIGURE 2](Changes in terminal abdominal aorta flow and in heart rate in a sled dog during the transition from rest to exercise.)
Regional Blood Flows.—Blood velocity was monitored in the terminal abdominal aorta and in the renal, mesenteric, and coronary arteries. In some instances, velocity was measured at two or more of these sites simultaneously with the blood pressure; but, for the most part, the recorded data represented a single velocity and pressure.

Terminal Aorta.—The increase in blood flow to skeletal muscle during exercise, which has previously been documented by numerous investigators, was repeatedly confirmed in these experiments. The changes in terminal abdominal aortic flow during the transition from sleep to exercise are exemplified in Figure 2. When the dog was sleeping, the peak flow velocity reached 75 cm/sec during systole but approached zero during diastole. However, when the dog was running, blood velocity in systole increased to about 140 cm/sec and remained well above zero throughout diastole. The net effect of these changes in flow velocity and the fivefold increase in heart rate is reflected in the mean flow which, in this case, was increased more than nine times over the sleeping level.

Coronary Dynamics.—The changes recorded in coronary dynamics during the transition from rest to exercise are exemplified in Figure 3. Peak circumflex coronary velocity increased from about 25 cm/sec to almost 75 cm/sec, and mean flow increased approximately fivefold. Phasic waveforms of coronary blood velocity recorded at rest and during exercise are shown in Figure 4.

Phasic flow typically fell to zero at the end of systole in the resting dog but was well above zero throughout the cardiac cycle in the running dog.

Changes in coronary hemodynamics during a prolonged run under load are shown in Figure 5; heart rate, blood pressure, and coronary flow typically increased as the dog made the transition from rest to exercise. The dog's heart rate remained near 300/min, and coronary blood flow increased threefold while the dog was running; sometimes greater increases occurred. On each of the several occasions when the team slowed or stopped briefly, both coronary flow and heart rate dropped sharply, and at the end of the run, coronary flow returned to the resting level within a few minutes. Mean blood pressure was somewhat more labile in this animal than in most, but was not significantly increased during exercise, so that the net response of the coronary bed was that of vasodilation.

Mesenteric Artery.—The level of mesenteric artery blood flow changed very little during exercise. A recording of mesenteric artery dynamics during a 36-minute run is reproduced in Figure 6. Although the pulse pressure increased to approximately 240 mm Hg immediately after each start, mean pressure showed no sustained increase. Although phasic flow varied slightly and transiently,
Phasic waveforms of circumflex coronary flow and carotid pressure in a sled dog at rest and during exercise.

Coronary dynamics during a run on the trail. Several telemetry drop-outs occurred near the end of this run.

the mean level of mesenteric flow during running was unchanged from that at rest or during recovery. Indeed, no single instance was recorded in which mesenteric flow decreased appreciably during exercise, despite prolonged runs which maintained the heart rate at 300/min for up to an hour.

Phasic waveforms of mesenteric artery velocity and blood pressure at rest and during exercise are shown in Figure 7. Inspect-
Mesenteric artery dynamics in a sled dog during a trail run of about 1 hour.

A comparison of phasic waveform of mesenteric artery flow and aortic blood pressure during rest and exercise.

This change in stroke flow was offset by the increase in heart rate, i.e., the changes in heart rate and stroke flow were approximately reciprocal.

Maximal Exercise and Renal Flow.—Occa-
occasionally during sled dog competitions or routine trail work, one or more of the dogs collapsed in his traces, apparently unable to run further. Figure 8 shows details of the changes in renal artery flow and heart rate in a dog that collapsed after carrying 4.08 kg of ballast (20% of its body weight) in a saddle bag during a run of 2½ hours. Mean renal artery flow, which averaged approximately 15 ml/kg/min when the dog was resting or standing (A), stabilized at about 12 ml/kg/min during running (B). After more than 2 hours of continuous running on the trail, the values for heart rate and mean flow were unchanged (C). At this point the dog was floundered, fell down in the snow, and refused to run further (arrow 1). Since the telemetered cardiovascular data contained no evidence that the animal was in dire straits, the dog was encouraged to resume running, but soon collapsed, again without obvious cause. Phasic waveforms of renal

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blood velocity and aortic blood pressure reproduced in Figure 9 show that renal stroke flow decreased when the dog was running.

**Discussion**

Increased muscle blood flow is a fundamental cardiovascular adaptation to exercise; in these dogs, flow through the terminal abdominal aorta was increased up to twelve times the sleeping value, whereas that through the circumflex coronary artery was increased approximately sixfold. Previous investigators have reported that skeletal muscle flow in man may be increased as much as twenty-fold whereas coronary flow has been reported to increase up to eightfold (6). Two sources are available to supply the extra quantity of blood required by exercising muscle, i.e., augmented total cardiac output and redistribution from regional beds. The quantitative aspects of the increase in cardiac output during exercise have been adequately documented and are in general well known (6). Alterations in the regional distribution of blood flow are also believed to contribute to the increase in skeletal muscle flow, but the actual patterns of redistribution are not well known and data quantitating diversion of blood flow from splanchnic areas to skeletal muscle beds during exercise are lacking. This mechanism is potentially capable of extending the exercise capacity significantly; if present, it represents an important cardiovascular reserve mechanism.

The concept of compensatory redistribution of flow during exercise is teleologically attractive, since the blood supply to both gut and kidney far exceeds their metabolic requirements. Both areas are richly endowed with nerve fibers and are known to respond to sympathetic influences, whether nervous or humoral, by vasoconstriction. Redistribution may quite reasonably then be predicted to result from the general increase in sympathetic activity which occurs with exercise, the extent being related to the general level of exercise.

Diminished visceral blood flow repeatedly been demonstrated in exercising human sub-

jests. Thus, renal blood flow, estimated by PAH clearance, and splanchnic blood flow, by BSP clearance, have been reported to be reduced during exercise, and in general, the reduction in flow is proportional to the exercise severity (6-8). However, evidence for reduced visceral flow is less convincing in exercise studies conducted on intact, unanesthetized animals with chronically implanted blood flowmeters. Herrick et al. (9) used chronically implanted thermostrohmuhrs to measure flow to the major peripheral beds of exercising dogs and reported no decrease in either renal flow or mesenteric flow, although iliac artery flow was greatly elevated. These studies were later criticized because of inadequacies of the device that measured flow (10). In 1962, Rushmer et al. (11) used pulsed ultrasonic flowmeters to record blood flow from the same sites in healthy exercising dogs; mean blood pressure and renal and mesenteric flows remained unchanged during exercise, whereas iliac flow increased several fold. However, the level of exercise (3 mph on a 12% grade) might have been an inadequate stress to invoke compensatory visceral vasoconstriction.

We were unable to demonstrate a significant reduction in either renal or mesenteric blood flow in exercising sled dogs, even during exhaustive exercise in which the animal eventually collapsed. This suggests that redistribution of flow was not a significant reserve mechanism and that the bulk of the increase in skeletal muscle flow was derived elsewhere, i.e., from an increased cardiac output.

We have previously shown that renal and mesenteric flows may be acutely interrupted when marine mammals or dogs are immersed under water (12, 13). This demonstration of the “diving response” was evidence that physiological mechanisms for regulating flow to these beds remained viable in spite of long-term chronic implantation of blood flowmeters around major vessels leading to these organs, i.e., that vasoconstrictor responses were not obscured by the choice of instrumentation. This procedure was also used in
the present experiments; transient immersion of a dog's nose under water invariably resulted in instantaneous reduction or even interruption of visceral flow, indicating that vasomotor influence on renal and mesenteric beds was present and that the Doppler device was capable of sensing such flow changes when present. In diving animals, the period of flow interruption is necessarily limited by the tolerance for immersion and is not long enough to inflict permanent tissue damage due to hypoxia. The same might be true in exercising humans, who are largely incapable of sustaining heavy work loads for more than a few minutes (e.g., the 4-minute mile). However, in animals capable of extending themselves severely for several hours, redistribution of blood flow away from the kidney and viscera might be inappropriate or even disastrous, since damage to these organs would inevitably result from the prolonged periods of anoxia.

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