Heat conservation during cold exposure in birds (vasomotor and respiratory implications)

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The mechanisms by which peripheral circulation and respiration serve in maintaining thermal homeostasis in birds living in cold climates are reviewed. Three types of arteriovenous heat exchanger (an *elaborate rete*, a *simple rete*, and a *venae comitantes* system) are found in the legs of birds. The anatomical differences between the different types of A-V associations are described, and the regulation of peripheral blood flow, in respect to maximal heat conservation and prevention of tissue damage, is discussed. A nasal temporal counter current heat exchanger, lowering the temperature of the expired air to values considerably below the body temperature, is the most important mechanism for minimizing the respiratory heat and water loss. In addition, a decreased ventilatory requirement, caused by a changed respiratory pattern and an increased parabronchial oxygen extraction, lowers the amount of air ventilated relative to the amount of oxygen uptake. Thus, the relative loss of heat and water is reduced.

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Introduction

Homeotherms from polar and higher temperate latitudes are typically very well insulated and have low critical temperatures serving to maintain deep body temperature without increased heat production during cold exposure. During activity, however, when flying or running, the high insulative value of the fur or plumage requires efficient heat loss mechanisms. Such heat loss is mainly localized to portions of extremities like the legs or the nose, tail, beak, or other protruding parts which are generally much less insulated than the rest of the body or have no insulation in the form of fur or feathers at all. These extremities essential for heat dissipating purposes become prone to excessive heat loss and tissue injury from freezing during severe cold exposure. The many birds with unfeathered feet are cases in point, particularly species in polar regions. Regulation of blood flow to birds' feet must hence encompass consideration of competing homeostatic drives such as heat dissipation and conservation as well as protection against freezing and maintenance of adequate nutritional blood flow.

The thermal balance in homeotherms also depends on the heat and water exchange associated with ventilation of the airways and lungs. Depending on ambient conditions, more than 25% of the total heat production of a bird may be involved in the heat and water vapor exchanges with the ventilated air.

This paper will attempt to evaluate how the circulation of blood through the extremities of birds is regulated to serve in thermal homeostasis during cold exposure. Similarly, it will be discussed how the heat exchange between the ventilated air and the respiratory passages is regulated to conserve body heat during cold exposures.

Blood circulation and thermal homeostasis

Anatomical considerations

The vascular system of the leg and foot in birds and the anatomical basis for its heat transport and exchange efficiency have been excellently described by Midtgård (1980 a & b, 1981). Based on investigations of 66 species of birds representing 21 orders, Midtgård (op. cit.) categorized birds according to the degree and type of arteriovenous contact in the leg and foot vasculature. In species having an *elaborate* rete tibiotarsale, more than seven collateral arteries are in close opposition to a larger number of veins. Both vessel types usually anastomose extensively in the rete region. A rete system classified as *simple* has

3–5 arteries in close association with a larger number of anastomosing veins, but possesses no apparent arterial anastomosis. The *non-rete* type vascular arrangement has only one or two collateral arteries running counter current to an anastomosing venous network surrounding the artery and forming a *venae comitantes system*. In addition to the deeply running veins closely associated with the tibial arterial system, all birds have a large system of superficial medial and lateral metatarsal veins without immediate contact with the leg and foot arterial system. In the unfeathered section of the feet, birds show a profuse arrangement of arteriovenous anastomoses (Schumacher 1916; Midtgård 1980b). When dilated, these are much larger (25–120 μm) than the capillaries supplying the nutritional circulation. The larger superficial veins as well as the deeper veins associated with the tibial arterial system are perfused from the same arterial system in such a way that the two types of venous drainage are interconnected. Figure 1 from Midtgård (1981) shows the vascular arrangement in A: a

bird lacking a rete tibiotarsale, and B: a bird having an elaborate rete (e.g. a mallard duck). In the latter type the main tibial artery from which the rete arterial inflow is derived is very muscular and densely innervated, presumably to partition the arterial inflow in a regulated way between the rete and the more distal vascular beds. As pointed out by Midtgård (1981), the rete system offers an anatomical advantage if the species is in need of maximal heat dissipation, since the counter current heat exchange, i.e. heat conserving function of the rete, can be bypassed. The partitioning of the venous return between the arteriovenous contact surfaces of the rete or venae comitantes system and the superficial vessels having no thermal contact surface with the arterial system, provides an anatomical basis for heat conservation which would be favoured by venous return through the deeper veins associated with the arterial outflow forming a counter current heat exchange system. Conversely, heat dissipation would be maximized if venous drainage predominantly occurred through the superficial veins. If arterial blood could be shunted through A-V anastomosis proximal to the rete system or in deeper portions of the legs, there would be a third option for heat conservation.

Polar species of birds do not typically have an elaborate rete system which might anatomically be deduced to provide an optimal basis for both heat conservation and heat dissipation. However, as pointed out by Midtgård (1981), no physiological study has addressed the efficiency of heat exchange in an elaborate rete system compared to a venae comitantes system. Thus, the elaborate rete system in arctic eider ducks is comparable to that in other ducks; a similar situation obtains when comparing the montane and arctic ptarmigan with relatives from warmer climates. There appears, however, to be a larger contact surface between the tibial artery and associated veins (vena comitantes) in charadriiform species from arctic regions (arctic gulls and guillemots) compared to non-arctic species (Midtgård 1981).

Fig. 1. Diagram showing the relation of the heat exchanger to the large arteries and veins (black) in birds lacking the *rete tibiotarsale* (A) and in birds with elaborate retia (B). H—heat exchanger, cc—counter-current veins, s—superficial vein, m—main tibial artery, p—peroneal artery and vein (collateral vessela). From Midtgård (1981).

Physiological aspects, regulation of peripheral blood flow, heat dissipation, conservation, tissue damage, nutrition

The importance of blood circulation in thermal homeostasis is likely to be amplified in birds from polar regions due to the extreme exposure to both wet and dry cold (Irving 1972). Thus, maximum
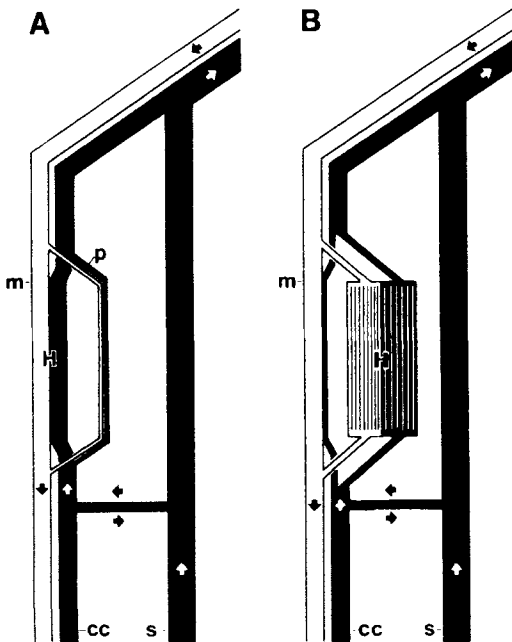
blood flow to the webbed feet of the antarctic giant fulmar *Macronectes giganteus* could exceed 10 litres/m²/min (Johansen & Millard 1973), which is about twice the value reported as maximum to human skin during vasodilatation elicited by heat exposure (Folkow & Neil 1971).

Conversely, thermal homeostasis during a steady state cold exposure may involve regulation of foot blood flow to very low levels expressed by skin and subcutaneous temperatures only slightly higher than tissue freezing temperature. A direct effect of low temperature on foot blood may also be important and could depend on the large increase in blood viscosity at reduced blood temperature. Guard & Murrish (1975) have compared the viscous properties of blood in antarctic birds and mammals with those in animals in temperate regions. The antarctic species showed a higher apparent viscosity but a lower yield shear stress at low temperature than the non-antarctic species. The authors argue that the higher apparent viscosity will reduce flow and thus diminish heat loss from extremities in the antarctic species, but the low yield shear stress will prevent stasis at low flows and thus reduce the likelihood of tissue freezing.

The differences between maximum and minimum blood flow to birds' feet are likely to exceed those of any tissues in other vertebrates. Such large and fluctuating blood flow is accommodated through the agency of the conspicuous presence of A-V anastomosis in bird legs, particularly superficially on the plantar surface, but also elsewhere in the leg vasculature distal to the feathered portion of the leg. Since blood flow is proportional to the fourth power of the radius, an A-V anastomosis of diameter 100 μm can accommodate a blood flow 10,000 times greater than a capillary of diameter 10 mm, provided the pressure drop along the two types of vascular channel and their lengths were similar. That the extravagantly high blood flow which can occur in birds' feet is predominantly channelled through A-V anastomosis is also clear from the very small arteriovenous differences in blood P_{O_2}. At the lowest level of leg blood flow recorded, Johansen & Millard (1973) reported an A-V P_{O_2} difference between 4 and 10 mmHg. At peak blood flow, however, there was no detectable difference in blood A-V P_{O_2} values. The large complement of vascular smooth muscle in A-V anastomosis is richly innervated (Grant 1930; Grant & Bland 1931) and reported to form sphincter-like structures in

mammals (Plenck & Püschman 1971). The anastomosis contains large amounts of cholinesterase suggestive of cholinergic innervation (Hurley & Mescon 1956), but also has a dense adrenergic innervation (Molyneux 1977). Based on pharmacological evidence, Guard & Murrish (1974) claimed that dilatation of A-V anastomosis in the legs of the giant fulmar *Macronectes* could be prevented by propranolol, a beta-adrenergic blocking agent. High flow could be restored by injection of isoproterenol, a beta-adrenergic mimetic agent. They claimed that normal dilatation depends on a stimulus originating in sympathetic nerves rather than from circulating catecholamines, since vasodilatation could be prevented by procaine blockage of the tibial nerves. Regulation of blood flow through the A-V anastomosis in birds is likely to be influenced by a dual type innervation, since AVA flow, selectively determined by a radioactive microsphere technique, revealed a decrease in response to α-adrenergic agonists (norepineperine), and an increase in flow could be elicited either by nerve blockage or by the α-adrenergic blocking agent, phenoxybenzamine (Hillman *et al.* 1982).

By contrast it has been demonstrated that a vasomotor constrictor tonus to the nutritional vascular beds (true capillary circulation) is not present at thermoneutrality in either birds (Hillman *et al.* 1982) or mammals (Hales *et al.* 1978).

McGregor (1979), working on ducks and chickens, also reported A-V anastomosis dilatation in response to β-adrenergic agonists but he was unable to block a vasodilatation produced by faradic nerve stimulation by a β-adrenergic antagonist. Johansen & Millard (1974), working on the antarctic giant fulmar *Macronectes*, also demonstrated active leg vasodilatation from faradic nerve stimulation and claimed this response to be blocked after atropinization. The latter effect, however, has been refuted by McGregor (1979), who offered evidence that the active vasodilatation in ducks and chickens is due to neither cholinergic nor adrenergic nerves. His studies leave open the possibility of a purinergic type innervation. Hillman *et al.* (1982) also excluded that the active vasodilatation in the chicken is due to dopaminergic neurons such as suggested for AVA dilatation in the dog hindlimb (Bell & Stubbs 1978).

In homeotherms from low temperate and southern latitudes, sudden exposure to cold, particularly wet cold, causes an immediate vasocon-

strictor response (Lewis 1930). This cold vaso-constriction is described as stimulation of α-adrenergic receptors. In man and most other mammals this cold vasoconstriction is followed after variable time, usually 4–5 minutes, by a vasodilatation referred to as cold vasodilatation (Krog et al. 1960). Following its onset, periodic changes in cutaneous blood flow produce fluc-tuations in skin temperature referred to as the 'hunting response' (Lewis 1930; Greenfield & Sheperd 1950). In birds from polar regions a similar if not homologue vascular response has been described. The most notable difference is the sudden onset of vasodilatation at the time of immersing the feet in ice. Figure 2 shows an ice immersion experiment of the foot of a giant ful-mar *Macronectes*. Johansen & Millard (1973) termed this response a 'cold flush'. It has also been demonstrated in penguins and confirmed for *Macronectes giganteus* by Guard & Murrish (1974). Peak blood flow to one leg during a cold flush could reach 100 ml/min. Typically, the change in foot blood flow was parallelled by a rise

in pressure in the matatarsal superficial veins. Venous pressure and flow also became distinctly pulsatile, synchronous with the arterial pulse, sug-gesting that the increased foot blood flow associ-ated with a cold flush is channelled through 'open' A-V anastomosis. Guard & Murrish (1974) reported that the marked vasodilatation of the large bored A-V anastomosis during a 'cold flush' was expressed by a marked rise in web venous pressure bringing it to within 5 mm Hg of the web arterial pressure. These authors claim that the cold flush response is elicited by stimulation of β-adrenergic receptors.

It appears a reasonable assumption that the suddenly onset cold induced vasodilatation is adaptive to the prevention of cold injury to the peripheral tissues. Heat loss from the unfeathered extremities having such a high thermal conduc-tivity, however, would be detrimental to central thermal homeostasis if it continued long. Thus at peak blood flow during a cold flush a temperature gradient of about 40°C may exist between web arterial blood and the ambient ice water separated by no more than 1–2 mm of tissue (Fig. 2). Con-sequently, the high flow during a cold flush sub-sides after 30–60 seconds and transcends into lower flow values. If ice immersion is maintained, foot blood flow typically starts to fluctuate as demonstrated in Fig. 3. This response type is similar to the hunting phenomenon described by Lewis (1930) for the human hand.

Reite et al. (1977) have offered an alternative explanation for the mechanisms underlying the blood flow exchanges to birds' feet during cold exposure. Earlier studies on mammals (arctic seals) (Johansen 1969) and birds (ducks) (Millard & Reite 1975) have demonstrated that vascular smooth muscle from peripheral segments of the vasculature in the leg responds to catecholamines at lower temperatures than smooth muscle from more thermally protected vessels. Yet at very low tissue temperatures vascular smooth muscle from peripheral tissues also becomes irresponsive to vasoconstrictor stimuli and stays dilated. Unlike conditions in mammals, in birds a relatively large fraction of the vascular resistance to blood flow is caused by smooth muscle constriction in arteries much larger than arterioles. When a bird foot is cooled, nerve stimulation as well as the smooth muscle response to circulating catecholamines are gradually lost in the most peripheral portions of the foot like the web of ducks. Reite et al. (1977) reported that at a tissue temperature below 8°C,
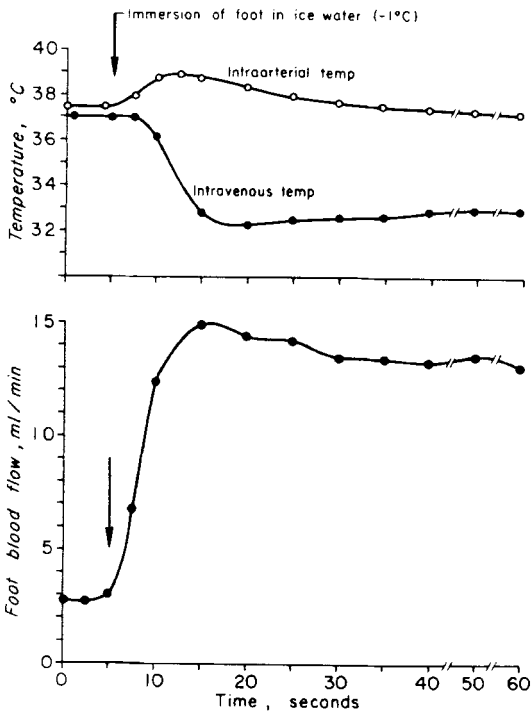

Fig. 2. A comparison of foot blood flow and intravascular blood temperatures in the foot of the giant fulmar *Macronectes gigan-teus* during sudden immersion of the foot in ice water. From Johansen & Millard (1973).
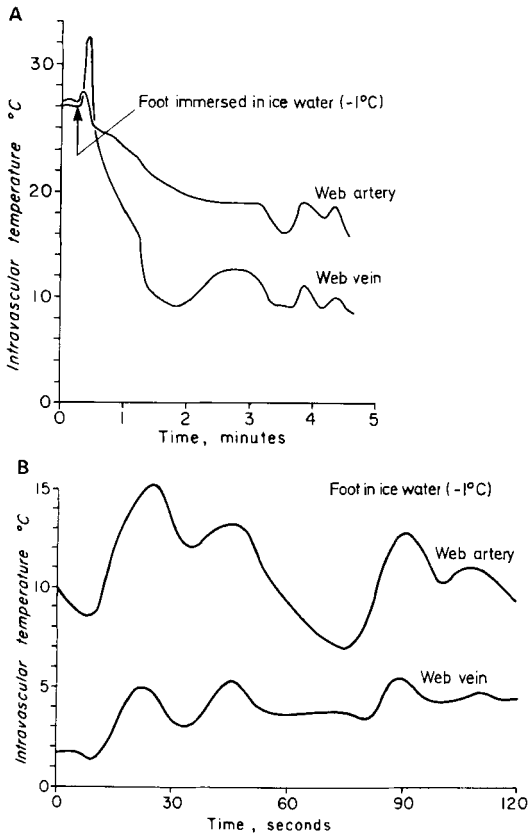
Fig. 3. Foot blood temperatures in the giant fulmar *Macronectes giganteus* during ice-immersion. A. Intravascular blood temperatures in the foot during five minutes of immersion in ice water. Note the sudden transient increase in blood temperature at ice immersion and the simultaneous fluctuations in blood temperatures (hunting response) after three minutes ice immersion. B. Typical 'hunting response' in foot blood temperatures after prolonged ice immersion. From Johansen & Millard (1973).

the vascular smooth muscle response of ducks was near negligible. This implies that when a neurogenic or humoral vasoconstrictor stimulus is applied during severe cold exposure, the ensuing response will cause vasoconstriction mainly in the warmer segments of the leg. This in turn will raise the central systemic blood pressure and increase blood flow to the distal portions of the cold exposed legs where the vasoconstrictor stimulus is without a response. In this manner the maintenance of an adequate blood supply protecting the cold exposed feet against freezing is not regulated by humoral or neurohumoral mechanisms operating in the coldest tissues. The authors point out the usefulness of controlling blood flow through cold exposed tissues without

depending on local mechanisms per se since such would be so long lasting and slowly onset that tissue protection against freezing would be impaired.

Once the delicate naked tissues of bird feet have been protected against injury from sudden ice immersion, the primary homeostatic drive influencing foot blood flow will be the maintenance of central core temperature when the cold exposure continues. This call for heat conservation depends crucially on counter current exchange of heat. This process will precool arterial blood flowing to the foot and rewarm venous blood before it reenters the central veins. Its anatomical basis must in *Macronectes* be the venae comitantes system surrounding the main tibial artery. In other birds from polar regions it could alternatively be a simple or elaborate rete system such as described by Midtgård (1981). Figure 4 illustrates the efficiency of heat conservation in *Macronectes* expressed by the temperature of venous blood when measured along veins from the feathered portion of the leg stepwise onto a distal vein in the ice-immersed web. A conspicuous increase in intravenous temperature is seen to occur just distal to and along the metatarsal section. This must reflect the heat exchanging function of the venae comitantes system. Rewarming of the cold blood draining the web could additionally occur through A-V shunt flow through anastomosis in deeper and thermally more protected portions of the leg.

Information is scarce about the central nervous integration and control of heat loss and conservation via the extremities of birds. Hammel *et al.* (1977) have reported a thermoregulatory change in wing and foot circulation of adelie penguins in response to heating and cooling the preoptic region of the anterior hypothalamus. Rautenberg (1969) has demonstrated that selective heating of the spinal cord in pigeons causes feet vasodilatation and thermal panting.

Respiratory heat exchange

The dissipation of heat and water vapour from the respiratory tract during breathing depends on the temperature and relative humidity of the inhaled and exhaled air as well as on the volume of air ventilated. During exposure to low ambient temperature, typically associated with a low relative humidity, the potential heat loss via the
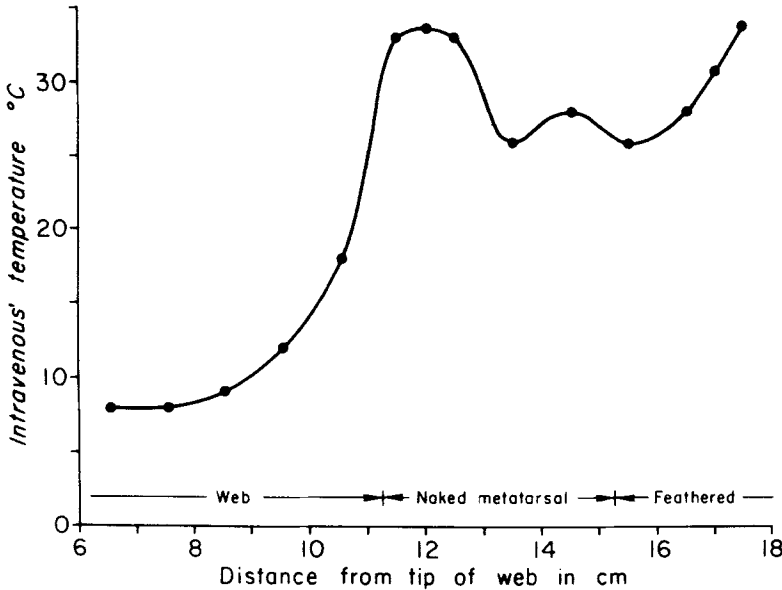
Fig. 4. Foot intravenous blood temperature in the giant fulmar *Macronectes giganteus* during gradual stepwise withdrawal of thermistor catheter. The lower foot (web) was immersed in ice during the experiment. From Johansen & Millard (1973).

expired air can be considerable and can theoretically exceed 25–30% of the total metabolic heat production in birds.

Conservation of this heat during cold exposure is physiologically regulated, however, and this regulation invloves both the temperature of the exhaled air and the total volume of air ventilated relative to the amount of O_2 taken up from this ventilated volume.

Cooling of expired air

Cold ambient air is warmed and saturated with water vapour during inhalation. The heat and water vapour required are transferred from the mucosal surfaces of the nasopharyngeal passages which are thus concomitantly cooled. When reaching the lung the inhaled air has been prewarmed to about deep body temperature. However, in all species of birds studied, a large fraction of the heat added to the inhaled air is recovered in the upper respiratory tract during expiration. The mechanism behind this heat retention depends on a form of temporal counter current heat exchange (Schmidt-Nielsen *et al.* 1970b). When expired across the cool nasopharyngeal surfaces the warm air will give up heat by convection and conduction and also release latent heat when water vapour is condensed at the same cool surfaces. As a consequence, exhaled air temperature is typically considerably below the deep

body temperature (Schmidt-Nielsen *et al.* 1970b; Murrish 1973; Brent *et al.* 1984). Figure 5 shows the relationship between exhaled air temperature and ambient temperature for the European coot *Fulica atra*. Between ambient temperatures of 35°C and 10°C the exhaled air temperature is seen to nearly parallel that of ambient temperature, with a gradient between the two of less than 5°C. At lower ambient air temperature, the gradient expanded but the efficacy of this nasal heat exchange is so high that when the inhaled air was at −10°C the exhaled air temperature was within 2°C of zero. Figure 6 shows in column A the maximum theoretical heat loss if the inspired air temperature was −25°C and all heat added would be lost with expired air.

The black segment of column B shows the heat recovered due to the cooling of expired air. This represents about 80% of the heat added to the air during inhalation. Similarly, in penguins 81.9% and 83.4% of the water and heat respectively added to inhaled air were recovered during exhalation. In penguins this heat retention corresponded to 17% of the metabolic heat production (Murrish 1973).

If a need for heat dissipation during cold exposure arises, e.g. following exercise, the heat recovery by nasal counter current exchange can be bypassed by an alteration of the respiratory flow directions. Working with dogs, Schmidt-Nielsen *et al.* (1970a) showed that when in a
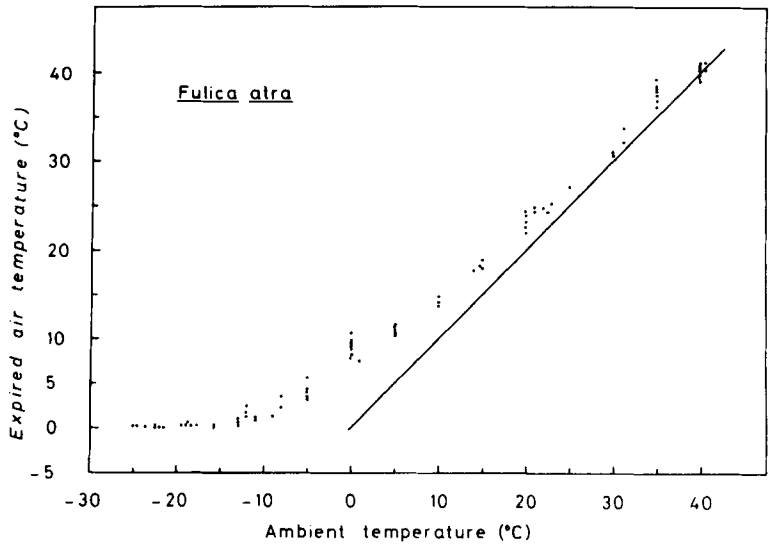
Fig. 5. The relationship between expired air temperature and ambient (inspired) air temperature in the European coot *Fulica atra*. The diagonal line shows $T_E = T_A$. From Brent *et al.* (1984).

Fig. 6. Column A shows the maximum possible theoretical heat loss from ventilated air in the European coot *Fulica atra* at ambient temperature of −25°C. Column B shows the heat actually retained because of precooling of expired air (black segment) and the fraction retained due to the lower air convection requirement (cross hatched segment). The top segment shows the actual respiratory heat loss to be about 10% of the possible maximum. From Brent *et al.* (1983a).

positive heat load, inspiration through the nostrils was followed by expiration through the mouth, a situation that would cancel the nasal heat conservation. A more recent study (Goldberg *et al.* 1981) has demonstrated more complex patterns of respiratory flow directions involving combinations of nasal and mouth in- and expirations. The breathing patterns were in all cases organized to maximize heat conservation or heat dissipation in accordance with thermoregulatory requirements. There are no data available on birds showing changes in respiratory flow directions. Murrish (1973), however, working on penguins, showed that the nasal heat retaining mechanism can be short circuited. The penguins were able to increase the expired air temperature to near body temperature during periods of external body heating while still inhaling cold ambient air. Such a regulation is probably mediated through blood-flow changes since injections of phenoxybenzamine, an α-adrenergic blocker, also caused a rise in the temperature of the exhaled air, indicating that vasodilatation had occurred in the nasal region (Murrish 1973).
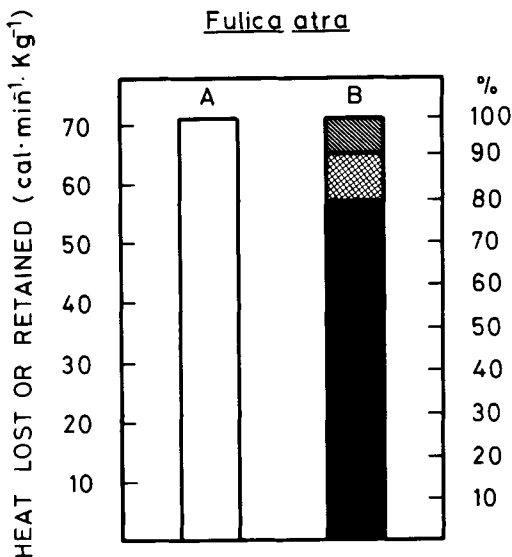
Heat conservation by a reduced ventilatory requirement

Heat and water loss through the respiratory tract will also depend on the total volume of air ventilated relative to the rate of O_2 uptake. The ratio between these parameters, referred to as the ventilatory requirement, has actually been demonstrated to decline when the ambient air temperature falls below the thermoneutral zone (Bucher 1981; Brent *et al.* 1983, 1984; Bech *et al.* in press). A reduced ventilatory requirement implies that the O_2 extracted from the air reaching the gas exchange surfaces of the lung must increase. This

in turn could depend on either an increased tidal volume and a reduced breathing rate or an actual increased efficiency in the gas exchange between blood and the parabronchial ventilation at the low ambient temperature. Thus, in the domestic duck *Anas platyrhynchos* in which the overall O_2-extraction increases from 28.5% at 20°C to 41.4% at −20°C, a changed breathing pattern accounts for 21% of this increase, while 79% of this increase in oxygen extraction is due to a more efficient oxygen exchange in the lung (Bech *et al.* in press). The breathing frequency was relatively stable and changed from 13.6 min^{-1} to 14.3 min^{-1}, whereas the tidal volume showed a considerable increase from 19.0 ml kg^{-1} to 30.2 ml kg^{-1} during exposure to an ambient temperature of −20°C. This much deeper breathing pattern obviously caused a relatively smaller fraction of the total ventilation to be dead space ventilation with a resultant increase in the overall oxygen extraction.

Similarly, in the kittiwake *Rissa tridactyla*, where there was an increased overall oxygen extraction from 19.7% to 25.5% when moved from an ambient temperature of 22°C to 6°C, the change was in part due to an altered breathing pattern consisting of slower and deeper ventilation (f fell from 23.2 min^{-1} to 16.4 min^{-1} and V_T increased from 9.0 ml to 12.1 ml) and partly due to an increased parabronchial oxygen extraction (Brent *et al.* 1983).

A more efficient parabronchial gas exchange operating during cold exposure has recently also been demonstrated in the domestic duck. Catheters were implanted in the interclavicular air sac. Samples drawn from this air sac showed an oxygen tension of 101.6 mmHg (S.D. = 5.5, n = 4) at 20°C and a tension of only 86.6 mmHg (S.D. = 4.8, n = 4) at −20°C (Bech *et al.* in press). This corresponds to parabronchial oxygen extractions (E_{O_2}) of 30.9% and 41.1% respectively. Thus, the O_2-extraction operating over the gas exchange surfaces (parabronchii) is obviously higher than the overall oxygen extraction. Parabronchial E_{O_2} increased in the kittiwake from 26.9% at 22°C to 30.6% at 6°C (Brent *et al.* 1983b) and in the European coot *Fulica atra* from 27.5% at thermoneutrality to the conspicuously high value of 62.0% at −25°C (Brent *et al.* 1983).

The significance of the decreased ventilatory requirement during cold exposure has been evaluated in a study on the European coot (Brent *et al.* 1984). At an ambient temperature of −25°C,

the coots decreased the exhaled air temperature to near 0°C concomitant to the increased oxygen extraction (Fig. 5). It was calculated that the coots regained about 90% of the heat added to the inhaled air. Most of this (79%) was due to the reduced exhaled air temperature, while a smaller fraction (11%) was a result of the lower ventilatory requirement (Fig. 6). The heat retained by the lowering of the ventilatory requirement thus corresponded to 2.6% of the total heat production at −25°C (Brent *et al.* 1984). Although this might seem of limited importance, the recovered heat actually represents more than 50% of the heat that otherwise would have been lost if the lung oxygen extraction had been held constant at low ambient temperatures.

Bucher (1981) reported that the oxygen extraction in the linneated parakeet *Bolborhynchus lineola* increased from 29% at thermoneutrality (28°C) to 37% at 5°C. The changed oxygen extraction accounted for a saving of 21% of the heat that otherwise would have been lost with the expired air.

So far, a reduced ventilatory requirement during cold exposure has been described in four species of birds: the linneated parakeet, the European coot, the kittiwake, and the domestic duck (Bucher 1981; Brent *et al.* 1983, 1984; Bech *et al.* in press). Two earlier studies involving measurements of ventilation in birds at low ambient temperatures, i.e. on the fish crow *Corvus ossifragus* by Bernstein and Schmidt-Nielsen (1974) and on the pigeon *Columba livia* by Bouverot *et al.* (1976), did not report changes in the oxygen extraction, although there was a small tendency for an increased E_{O_2} at the lowest ambient temperatures in the fish crow. Figure 7 illustrates lung oxygen extraction as a function of ambient temperature in birds, for which ventilatory data from below thermoneutrality are available.

No data to explain the mechanisms underlying the changes in the parabronchial oxygen extraction at cold exposure are available. An increased effectiveness of the parabronchial gas exchange could depend on a changed pattern of perfusion and ventilation, i.e. a reduction of the physiological perfusion and ventilation shunts (a reduced ventilation-perfusion inequality). Whereas the parabronchial perfusion shunt is reported to represent only a small part of the lung perfusion in birds (2.7% in ducks (Burger *et al.* 1979) and 0.4% in geese (Powell & Wagner 1982)), the ventilation shunt is of a much higher

Fig. 7. Relationship between ambient temperature and oxygen extraction in birds. The oxygen extraction is calculated as $(\dot{V}_{O_2} \times 0.095)/(\dot{V}_I \times 100)$, where $\dot{V}_{O_2}$ is the oxygen uptake and $\dot{V}_I$ is the inspired ventilation. Sources of data: ○ linneated parakeet *Bolborhynchus lineola* (Bucher 1981), ● European coot *Fulica atra* (Brent *et al.* 1984), □ domestic duck *Anas platyrhynchos* (Bech *et al.* in press), △ kittiwake *Rissa tridactyla* (Brent *et al.* 1983), ☆ fish crow *Corvus ossifragus* (Bernstein & Schmidt-Nielsen 1974), ⊗ pigeon *Columba livia* (Bouverot *et al.* 1976).
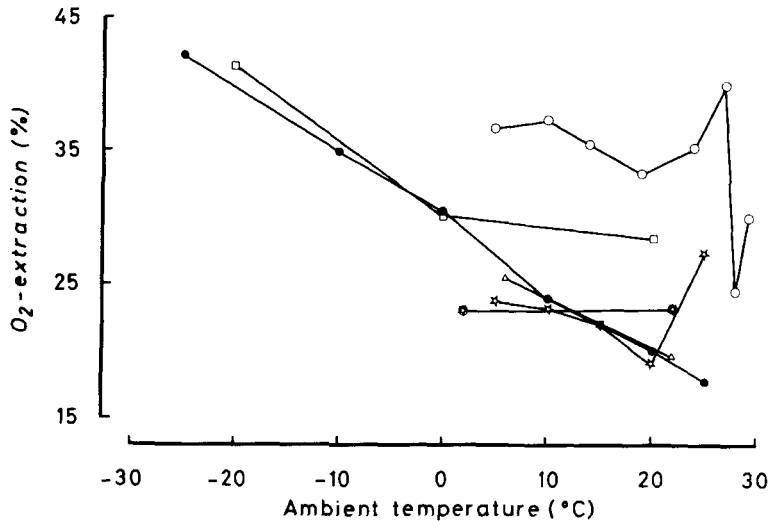
magnitude. Thus, Burger *et al.* (1979) calculated a ventilation shunt of 9.7% in ducks and Powell & Wagner (1982) found a high proportion of the 'lung units' in geese to have a high V/Q ratio (totally receiving 10.6% of the ventilation but only 0.3% of the cardiac output). If this large effective ventilation shunt is under physiological control and could be reduced under cold stress, it could well explain the observed increase in parabronchial oxygen extraction.

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