Rapid brain cooling in diving ducks

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Caputa, Michał, Lars Folkow, and Arnoldus Schytte Blix. Rapid brain cooling in diving ducks. Am. J. Physiol. 275 (Regulatory Integrative Comp. Physiol. 44): R363–R371, 1998.—Hypothermia may limit asphyxic damages to the brain, and many small homeotherms have been shown to use anapyrexic strategies when exposed to asphyxic conditions. Larger homeotherms do not seem to use the same strategy, but could save oxygen and prevent hypoxic brain damage by employing selective brain cooling (SBC) in connection with asphyxia. To test the hypothesis that selective brain cooling may take place in connection with asphyxia, we have recorded brain [hypothalamic (THyp)] and body [colonic (Tc)] temperatures and heart rates in four Pekin ducks during 5-min simulated (head submersion) diving in cold water (10°C). Diving resulted in a drop in THyp (3.1 ± 1.4°C) that continued into the recovery period (P < 0.001). Restricting heat loss from the buccal cavity and eyes during diving compromised brain cooling in an additive manner. Tc was not influenced by diving. Control cooling of the head with crushed ice during a 5-min period of undisturbed breathing had no effect on THyp. Warm water (35°C) markedly reduced brain cooling, and dive capacity was reduced by ~14% (P < 0.05) compared with diving in water at 10°C. The data suggest that SBC is used in ducks during diving, and we propose that this mechanism may enable the bird to save oxygen for prolonged aerobic submersion and to protect the brain from asphyxic damages.

selective brain cooling; dive capacity; hypoxia; neuroprotection; hypothermia

THE MAIN MECHANISMS OF defense against asphyxia are similar among reptiles, birds, and mammals. They include peripheral vasoconstriction, which favors the brain and the heart at the expense of skeletal muscles and abdominal organs. Simultaneously, cardiac output is dramatically reduced through a profound bradycardia. This is true not only for diving mammals and birds (5) but for hypoxemic fetal sheep (34), turtles under anoxic conditions (12), and armadillos covered by soil (9) as well.

A decrease in body temperature represents another adaptive response to oxygen shortage in some species (40). Recent neurological studies show that moderate hypothermia has very important neuroprotective effects (10, 13, 32, 41). Both accidental (36) and experimentally induced (10, 13, 25, 29, 32, 41) hypothermia have been shown to prevent hypoxic-ischemic damages to the brain. The mechanism of neuroprotection by moderate hypothermia is complex: it reduces the cerebral demand for oxygen through the Q10 effect (6, 40); it suppresses massive cerebral release of excitatory amino acids (14); it prevents an excessive rise in Ca2+ within neurons (25), thereby delaying hypoxic depolarization in the brain (19); it reduces the disruption of the blood-brain barrier (21); and it suppresses free radical production in the brain (14, 28, 42). Thus small rodents, when exposed to hypoxia and to a variety of other traumatic agents, display a drop in body temperature that is accomplished through both autonomic and behavioral heat-dissipating responses (15). Relatively large homeotherms, including humans (23), on the other hand, do not show any substantial change in core temperature under such circumstances, but hypoxia lowers body temperature thresholds for vasoconstriction and shivering in nondiving homeotherms (23). Obviously, a huge body cannot be cooled as quickly as a small one because of its much higher thermal inertia, but large mammals (4, 7) and birds (2, 3, 26, 31, 35) are able to cool their brain quickly and markedly. They use efficacious mechanisms of selective brain cooling, which manifests itself, usually under hyperthermic conditions, in that cerebral temperature is maintained at a lower level than that of the rest of the body, and Caputa (8) suggested that this might be used also to protect the brain against hypoxic damage during prolonged diving.

To test the hypothesis that selective brain cooling may take place in connection with diving, we have studied changes in brain and body temperatures in Pekin ducks that were subjected to simulated (head submersion) diving. In ducks, as in other birds, selective brain cooling is achieved by countercurrent heat exchange in the ophthalmic rete, where the warm arterial blood is cooled by cold blood from the eyes and the nasal and palatine mucosa (2, 3, 31, 35). Therefore, we also investigated the effects of reducing heat loss from the eyes and buccal cavity on brain temperature changes during diving. One more aim of the present study was to determine the effects of changing water temperatures on brain temperature and on the diving capacity of the ducks.

MATERIALS AND METHODS

Animals. The experiments involved a total of seven adult white Pekin ducks (Anas platyrhynchos) of either sex, weighing 2.3–3.4 kg. Four of the birds were implanted with hypothalamic guide tubes and used in temperature-recording experiments. Two of these and another three nonimplanted ducks were subjected to dive-capacity experiments. All birds were kept in single pens and fed commercial feed and provided with water ad libitum between experiments.

Guide tube implantation. Hypothalamic glass guide tubes were implanted into the rostral brain stem of four of the ducks under general ketamine anesthesia [15 mg/kg, Ketalar (50 mg/ml); Parke-Davis, Barcelona, Spain] combined with diazepam [2.5 mg/kg, Vival (10 mg/ml); Apothekernes Laboratorium, Oslo, Norway], according to the following procedure. First, a sagittal incision was made in the skin of the occipital part of the skull. To avoid conductive cooling of the hypothalamic thermocouple during diving, a hole was made in the
posterior part of the occipital bone and the tube (27 mm long) was pushed 25 mm rostroventrally to reach the hypothalamic area. The external end of the tube, which was connected to a piece of polyethylene cannula (8–10 cm long), was cemented to the skull using dental cement (Meliodent; Bayer Dental, Newbury, UK). Finally, the skin wound was sutured, so that the cannula protruded from the neck. The placement of the guide tubes was verified by postmortem examinations, which confirmed that their tips were positioned just above the optic chiasma.

Temperature measurements. Brain, colonic (Tbc), and water bath (Twb) temperatures were measured to an accuracy of ±0.1°C by means of copper-constantan thermocouples. Hypothalamic temperature (THyp) was measured at the tip of the implanted hypothalamic reentrant tube, and basal cerebellar temperature (TBC; see below) was measured at intervals by withdrawing the thermocouple 12.5 mm from the tip of the guide tube. During diving, care was taken to avoid submergence of the entrance of the guide tube. TC was measured with a guide tube. During diving, care was taken to avoid submergence of withdrawing the thermocouple 12.5 mm from the tip of the guide tube. TC was measured with a thermocouple that was introduced 10–11 cm into the colon via the cloacal opening. Twb was measured at the level of, but 10 cm away from, the submersed head of the duck. All thermocouples were connected to a data acquisition system (Dianachart model PCA-48; Dianachart, Rockway, NJ), which in turn was connected to a desktop computer containing software for linearization according to Toien (39). Temperature data were collected at 10-s intervals.

All thermocouples were regularly calibrated using a thermostatically controlled calibration water bath (model 6025; Hart Scientific, Pleasant Grove, UT) and a 0°C ice point dry-well reference chamber (model 5115, Hart Scientific).

Heart rate recordings. Heart rate (HR) was monitored using bipolar leads with stainless steel needle electrodes that were inserted subcutaneously, one between the wings and the other on one of the thighs. Signals were fed into a Gould Universal Amplifier (Gould Electronics, Cleveland, OH) and displayed on a chart recorder (model TA 4000, Gould Electronics).

Experimental procedure. Experiments started 5–7 days after the surgery. The ducks were fastened to a specially designed aluminum board in a ventral recumbent posture as described by Andersen (1). The board was placed over a water tank (5 cm above water surface). After the thermocouples were introduced into the hypothalamic guide tube and into the colon and after attachment of HR leads, the birds were left undisturbed at room temperature (18–20°C) until body and brain temperatures had been stable for 30 min. Then the duck’s head was submerged in the water tank by tilting the board, as is the standard procedure for experimental diving of ducks. To prevent conductive cooling of the extracranial part of the brain thermocouple by cold water, the temporal and occipital parts of the head were never submerged.

Experiments were performed in two parts. In the first part, three experimental series were conducted to study the effect of restricting heat loss from the buccal cavity and the eyes on THyp during diving. They consisted of 5-min submergences in water at 10°C 1) with bill unrestrained and eyes under water surface, 2) with bill restrained (tightly closed) and eyes under water, and 3) with bill restrained and eyes above water. Thermal effects of these dives were compared with the effects of 5-min external cooling of the head (including the bill and the eyeballs) with crushed melting ice in nondiving, freely breathing ducks. This was achieved by putting crushed ice underneath, on top of, and along both sides of the head, so that also the eye area was in close contact with ice. Moreover, a small polyethylene bag, filled with crushed ice, was wrapped around the bill, care being taken not to cover the nares. The area of the control cooling was exactly the same as in submergences with eyes under water surface. In the second part, three series of dives (with bill unrestrained and eyes under water surface) were performed at water temperatures of 10, 25 (2 ducks only), and 35°C to study the effect of water temperature on brain cooling and on diving capacity, which was estimated from the HR changes, based on the following criterion: Hudson and Jones (20) showed that a sudden onset of tachycardia during endurance diving in Pekin ducks marks the limit of their diving capacity. In the present study, submergence was maintained, under electrocardiograph control, until the diving bradycardia was replaced by a sudden sustained tachycardia. The time interval between the start of the dive and the onset of the tachycardia was defined as the diving capacity of the duck. These experiments were performed without any signs of ill effects in any of the five participating ducks.

All temperatures were recorded throughout the entire experiment, whereas HR recordings started 5 min before diving and continued for at least 5 min into the recovery period.

Because an accidental pulling of the hypothalamic thermocouple away from the tip of the guide tube during one of the experiments resulted in a considerable change in temperature, we also decided to examine regional differences in brain temperature after withdrawal of the thermocouple by 12.5 mm (putative placement of the thermocouple was then basal part of the cerebellum) in three ducks. The thermocouple withdrawal for 1–2 min was repeated five times during experiments in which ducks were subjected to diving with bill unrestrained and eyes under water 1) during initial steady state, 2) 1 min before the end of the dive, 3) 5 min after surfacing, 4) 10 min after surfacing, and 5) 20 min after surfacing.

All experiments were performed under permit from the Norwegian Committee on Ethics in Animal Experimentation.

Statistics. Values reported in the present paper are means ± SE unless otherwise specified. Data were analyzed for statistical significance using the Student’s t-test or the paired t-test. Differences between groups were considered significant when P < 0.05.

RESULTS

The brain-to-body (colon) temperature difference was moderate in resting nondiving ducks (Tbc 0.6–1.0°C below Tc) and quite stable in each experimental series (Figs. 1–7).

Diving in cold water resulted in a rapid brain cooling which started 1–3 min after the beginning of a dive, depending on its type (Figs. 1–3 and 5).

Effect of restricting buccal and ocular heat loss on brain cooling during diving. When ducks were submerged with their bill unrestrained, they immediately opened it, exposing the buccal cavity to cold water. In this situation, THyp rapidly declined (Fig. 1), starting in the second minute of the dive and continuing until the third minute after the end of the dive. Brain temperature remained significantly decreased throughout the 15-min recovery period. There was no change in Tc during the dive, whereas a slight tendency for a decrease (nonsignificant) in Tc was apparent during the recovery period. As a result, the brain-to-body (colon) temperature difference increased substantially during the dive and at the start of the recovery period. It
reached its maximum average of 3.1 ± 1.4°C in the third minute of the recovery period.

The simultaneously recorded HR changes (Fig. 1, bottom) demonstrate that diving bradycardia was fully expressed during the first 2 min after submersion and maintained until the end of the dive. There was a transient postdive tachycardia that culminated 2 min after the dive and then slowly faded.

Diving with the bill restrained and the eyes under water (Fig. 2) also produced a substantial drop in $T_{\text{hyp}}$, although it started 1 min later (in the third minute of the dive) and at the end of the 5-min submergence period was significantly ($P < 0.05$) reduced compared with open-bill diving. Brain cooling in this type of experiment continued also during the first 3 min of the recovery period. Because there were no significant changes in $T_C$ throughout diving and recovery, the brain-to-body (colon) temperature difference increased progressively from the start of the dive, reaching its maximal average value of 2.4 ± 1.0°C in the third minute of the recovery period. A significant decrease in cerebral temperature was maintained throughout the 15-min recovery period.

The cardiac responses in this experimental series were the same as those recorded in the previous series (cf. Fig. 1).

The drop in brain temperature during the third series of experiments in which the ducks were diving with the bill restrained and the eyes above water (Fig. 3) was the least pronounced. It started 3–4 min after submersion and continued during the initial 4 min of the recovery period. Brain temperature remained significantly below the predive value from the 2nd until the 10th minute of the recovery period. There was no change, however, in $T_C$ either during the diving or the recovery period. The brain-to-body (colon) temperature difference reached its maximal average value of 1.5 ± 0.8°C in the third minute of the recovery period. The decrease in brain temperature at the end of the 5-min submergence period in this series of experiments was

![Fig. 1. Mean values (and SE) of hypothalamic (brain) and colonic (cloacal) temperatures and heart rate in ducks subjected to 5-min diving (water temperature 10°C) with the bill unrestrained and the eyes under water. bpm, Beats/min. Significance of decreases in brain temperature: *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$ compared with time 0 value ($n = 4$).](image1)

![Fig. 2. Mean values (and SE) of hypothalamic (brain) and colonic (cloacal) temperatures and heart rate in ducks subjected to 5-min diving (water temperature 10°C) with the bill restrained (tightly closed) and the eyes under water. Significance of decreases in brain temperature: *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$ compared with time 0 value ($n = 4$).](image2)
significantly less than during both open-bill (P < 0.01) and closed-bill (P < 0.05) diving with the eyes under water. Comparing this with the first two series of experiments showed that there was also a slight difference in cardiac responses to diving. Thus, in the third series of experiments, diving bradycardia was again fully developed within the initial 2 min of submergence, but surfacing in each of the four trials was preceded by signs of an acceleration of HR (Fig. 3, bottom).

Control cooling of the head with ice in nondiving, freely breathing ducks (Fig. 4) produced no significant changes in \( T_{\text{Hyp}} \) and \( T_C \), either during or after the 5-min period of head cooling. The brain-to-body (colon) temperature difference was almost constant throughout the experiment, with an average maximum of 1.1 ± 0.3°C. No bradycardia was observed, but HR showed some oscillatory changes during the head cooling.

Effect of water temperature on brain cooling during diving and on diving capacity. The effect of water temperature on brain and body temperature changes during endurance diving was studied in two ducks only. These experiments were aimed to elucidate the effects of increasing water temperatures on \( T_{\text{Hyp}} \) during diving, which then would allow us to evaluate potential changes in diving capacity, in relation to the thermal state of the brain also in nonimplanted ducks.

Endurance diving in cold water (Fig. 5) was accompanied by a progressive decrease in brain temperature. The rate of brain cooling decreased slightly toward the end of the submersion period. On average, diving was terminated after 9 min 51 s (mean value) at the onset of tachycardia (Fig. 5, bottom). Brain cooling continued for another 4 min after surfacing, whereafter \( T_{\text{Hyp}} \) started to rise. \( T_C \), on the other hand, was unchanged throughout the dive period, but decreased slowly during 7–8 min of the recovery period and then remained stable and low for the duration of the experiment. The brain-to-body (colon) temperature difference reached its average maximal value of 4.3°C in the fourth minute of the recovery period.

The lukewarm water (25°C), used in the second series of endurance dives (Fig. 6), did not influence the time courses for hypothalamic and colonic temperature changes much from those recorded at 10°C water temperature.
temperature (Fig. 5). On average, diving was inter-
rupted because of the onset of tachycardia (Fig. 6, 
bottom) after 9 min 5 s, and the average maximal 
brain-to-body (colon) temperature difference was 4.0°C 
after the first minute of recovery.

Increasing water temperature to 35°C (Fig. 7) in the 
third series of endurance dives, however, resulted in 
the maintenance of brain temperature close its predive 
level during diving. However, THyp dropped somewhat 
at the end of the dive and during the initial 6 min of the 
recovery period. On average, diving in such warm 
water was terminated due to the onset of tachycardia 
(Fig. 7, bottom) after 7 min 59 s, and the maximal 
brain-to-body (colon) temperature difference averaged 
1.7°C after 6 min of recovery.

Endurance dives were also performed in nonim-
planted ducks. The average dive capacity of five ducks 
in 10°C water was 7.9 ± 0.9 min (Fig. 8). In 35°C water, 
however, it was reduced in each animal, the mean value 
being 6.8 ± 0.6 min. This difference is significant (P < 
0.05) and implies that the diving capacity in 35°C 
water was shortened by 14% compared with in 10°C 
water.

Intracerebral thermal gradients: the effect of diving. 
Table 1 presents changes in the temperature difference 
between THyp and TBC during the course of experiments. 
During the predive period, THyp was always lower than 
TBC. However, this THyp-TBC gradient became reversed 
during diving and the cooling of the central parts of the 
brain progressed in the early postdive period, when TBC 
in some cases was 1°C lower than THyp. This gradient 
was subsequently reduced, but it was still present 20 
min after the dive.

DISCUSSION

Brain cooling in diving ducks as an active phenom-
enon. Brain cooling during diving apparently depends 
to a great extent on heat loss from the buccal cavity and 
eyes, because reduced cooling of these areas (Figs. 2 
and 3) compromised brain cooling in an additive man-
ner. These areas, together with the nasal cavity, are 
known as heat-dissipating organs participating in selec-

during diving with the bill unrestrained and the eyes under water 
(10°C) lasting 9 min 51 s on average (n = 2).

Fig. 5. Mean values and ranges of hypothalamic (brain) and colonic 
(cloacal) temperatures and heart rate in ducks subjected to endur-
ance diving with the bill unrestrained and the eyes under water 
(10°C) lasting 9 min 51 s on average (n = 2).

Fig. 6. Mean values and ranges of hypothalamic (brain) and colonic 
(cloacal) temperatures and heart rate in ducks subjected to endur-
ance diving with the bill unrestrained and the eyes under water 
(25°C) lasting 9 min 5 s on average (n = 2).

Fig. 7. Mean values and ranges of hypothalamic (brain) and colonic 
(cloacal) temperatures and heart rate in ducks subjected to endur-
ance diving with the bill unrestrained and the eyes under water 
(35°C) lasting 9 min 59 s on average (n = 2).
tive brain cooling in birds, including ducks (2, 31, 35). Such studies have shown that the venous blood returning from these heat-dissipating surfaces cools, by countercurrent heat exchange, the arterial blood supplying the brain. The vascular heat exchanger is the rete mirabile ophthalmicum. There is a double shunt mechanism that allows both the venous (via the v. ophthalmica and v. maxillaris) and the arterial blood (via the cerebral carotid artery) to bypass the rete (31). Altogether, there are three ways to control the degree of selective brain cooling in birds:

1) by changing the temperature of the venous blood returning from the eye, bill, and nasal as well as buccal cavities through adjustments of heat loss rates from these areas;

2) by graded bypassing or perfusion of the ophthalmic rete with cool venous blood; and

3) by changing the proportion of arterial blood that supplies the brain from the ophthalmic rete. Yet, compared with mammals (4, 7, 8), the brain-to-body temperature difference in birds exposed to various thermal conditions (26) is much less variable. This was also the case in the present study during control periods preceding diving, during which brain-to-body temperature differences of 0.6–1.0°C were generally maintained. In contrast, an abrupt change in the brain-to-body temperature difference was recorded in our experiments a few minutes after diving had started.

The fact that cooling of the duck head with ice did not cause THyp to drop, whereas head submersion in cold water (at 10°C; Figs. 1–3) and in lukewarm water (at 25°C; Fig. 6) and even in warm water (at 35°C; Fig. 7) did, suggests that brain cooling during diving is not a result of passive heat loss from the surface of the head. During both head cooling with ice and head submersion, the head feathers were wet, and it is therefore highly unlikely that the observed differences in responses could be due to a lower rate of heat transfer from the ice-covered head in air than from the head during submersion in 10°C, not to mention 25 and 35°C, water. From this, we conclude that brain cooling during head submersion cannot be explained in terms of passive cooling, but rather seems to be an active cooling mechanism associated with the long-duration diving. It is even possible that the selective brain cooling is activated by local vascular control, as part of the diving response, and in a manner reminiscent of the control of selective brain cooling in reindeer (22). Accordingly, there is no substantial brain cooling in ducks repeatedly dabbling for food in ice-cold water (37). It should be kept in mind, however, that diving Pekin ducks maintain a dramatically reduced cardiac

Table 1. Changes in $T_{Hyp} - T_{BC}$ in various periods of experiments in which ducks were diving in cold water

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<th>Predive</th>
<th>Dive 5 min</th>
<th>Dive 10 min</th>
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<tr>
<td>$T_{Hyp} - T_{BC}$, °C</td>
<td>−0.28 ± 0.16</td>
<td>+0.11 ± 0.02</td>
<td>+0.99 ± 0.52</td>
<td>+0.64 ± 0.44</td>
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Values are means ± SE for 3 ducks. $T_{Hyp} - T_{BC}$ is temperature difference between the hypothalamus and the basal cerebellum. *P < 0.05 vs. predive.
output (24), and it may be argued that in the event of a substantial reduction in the supply of warm arterial blood to the head, passive heat loss over the surface of the head could alone explain the fall in brain temperature. However, despite the dramatic reduction in cardiac output in diving ducks, brain blood flow is reportedly increased during diving (24), which again makes passive cooling during diving rather unlikely. The delayed and then quite abrupt onset of brain cooling observed in the present study provides further indirect evidence for an active cooling mechanism (Figs. 1–3). Moreover, in our endurance diving experiments with bill unrestrained and eyes under water, the brain cooled equally well in cold (Fig. 5) and lukewarm (Fig. 6) water, which suggests the use of efficient regulatory mechanisms to compensate for the substantial difference in thermal conditions in the two situations. Finally, brain cooling always continued for several minutes after the end of diving, despite the fact that head cooling was terminated. Interestingly, Jones and colleagues (24) also reported that a high blood flow is maintained to the eye, even during prolonged diving in ducks. The high flow of blood will contribute to the heat-dissipating function of the eye, which has been demonstrated in diving ducks in the present study. On the basis of all these observations, we will suggest that brain cooling in diving ducks is an active and controlled mechanism.

If this is so, the reduced brain temperature of diving ducks should not be referred to as hypothermia. To describe regulated decreases in core temperature, the term anapnoxia has been recommended (27).

The observed rapid changes in $T_{Hyp}$ during diving could suggest that the hypothalamic area shows higher thermal lability than the rest of the brain, but measurements of brain temperature halfway from the surface of the occipital bone to the tip of the hypothalamic cannula (i.e., in the basal part of the cerebellum) showed that this is not the case (Table 1). Under predive resting conditions, the basal cerebellum was warmer than the hypothalamic area, which confirms previous observations that the warmest brain regions are those near the center of the brain (4, 7). However, the intracerebral temperature gradient reversed during diving and even progressed during the first 5 min of the postdive recovery period. We are, at present, unable to propose any explanation for this observation.

Selective brain cooling and diving capacity. There are at least two positive advantages of selective brain cooling in diving animals: 1) saving oxygen for prolonged aerobic submergence and 2) protecting the vulnerable cerebral tissue against asphyxic damage.

In birds and mammals, diving capacity is mainly determined by oxygen availability to the brain and heart (5, 17). Accordingly, in diving ducks the brain and heart are virtually the only organs that are supplied with the oxygen stored in the blood (17). From this point of view, reduction of the cerebral metabolic rate should result in a substantial saving of the blood oxygen stores. In normoxic piglets, a forced 5°C decrease of core temperature has been shown to reduce cerebral oxygen consumption by as much as 53% (6). In this context, selective brain cooling in diving ducks may prove to be an important component of their oxygen-saving strategy. In fact, we were able to show that restriction of brain cooling during submergence by use of warm water (Fig. 7) led to a 14% reduction of diving capacity (Fig. 8). A slight cardiac acceleration was also seen at the end of the 5-min dives with the bill restrained and the eyes above water (Fig. 3). This may be related to the markedly reduced drop in brain temperature that was observed during this type of diving.

Expert divers like seals and penguins appear to rely on similar mechanisms as demonstrated for ducks in the present study. Scholander and co-workers (38) reported temperature drops of >2°C in the liver, brain, and abdomen of harbor seals (Phoca vitulina) subjected to 13–15 min of simulated diving in 20°C water. Their measurements were made using glass thermometers and are open to criticism, but more recent studies have shown that aortic temperature dropped by ~2°C in a freely diving adult Weddell seal (Leptonychotes weddelii) in connection with dives of >30 min duration (18), and the brain temperature of a young harp seal (Phoca groenlandica) that was subjected to simulated dives of up to 9-min duration in 5°C water dropped by as much as 2.4°C (33). It is also worth mentioning that recent observations of abdominal temperature changes in freely diving penguins (Aptenodytes patagonicus) indicate a substantial cooling of body core, independent of food ingestion, during diving (16).

Neurobiological and clinical implications. Mild-to-moderate degrees of brain cooling (2–4°C) during hypoxia (19, 41), cerebral ischemia (10, 13, 25, 28, 41), or trauma (14) have been shown to reduce the extent of histopathological damage to the brain, and, conversely, mild degrees of temperature increase may markedly aggravate the outcome (13, 36). Neuroprotective effects of the decreased temperature include attenuation of excitotoxic glutamate release (14), reduction of free radical formation (14, 42), prolonged delay of a spread depolarization of cerebral neurons (19), and prevention of massive calcium entry into neurons (25). The high protective value of brain cooling is a consequence of the interruption of vicious circles in which those neurotoxic disturbances interact with each other in the propagation of hypoxic-ischemic neuronal damage (14). This is why artificially induced brain cooling is known to be the single most potent neuroprotective factor (14, 32). Even neuroprotective effects of other measures, such as glutamate antagonists (NBQX or MK-801), are correlated with the level of long-lasting brain cooling they induce (32).

The brain temperature drops recorded in the present study during and a couple of minutes after endurance dives (Fig. 5) reached values comparable to those reported above to provide effective neuroprotection in other species. In fact, the brain-to-body (colon) temperature difference of 4.3 ± 0.4°C recorded in the present paper 198 ± 34 s after endurance dives in water of 10°C...
is, to the best of our knowledge, the highest degree of selective brain cooling ever reported.

Artificially reduced cerebral temperature has a neuroprotective value not only when applied during asphyxic events (in experiments performed on various mammalian species) but also during the recovery period (11, 14, 28, 30), during which brain cooling may provide protection against damaging effects of free radicals (14, 28). Such a protection might be advantageous during the hyperventilation following emergence from the dive because it has been shown that production of reactive oxygen species rapidly increases in the gerbil brain up to 20 min after a 5-min ischemic insult (28). In this context one of the most interesting (and also one of the most reproducible) phenomena observed in the present study was a continuation of selective brain cooling into the postdive recovery period.

Comparative implications. The ability to initiate and rapidly achieve a substantial degree of selective brain cooling in large homeotherms may be compared with the susceptibility for whole body cooling in small mammals and birds. The former seems to be related both to the efficacious thermolytic effectors of the head (8) and the relatively small mass of the brain, compared with body mass. In fact, the brain of the duck is smaller than the whole body of a small passerine bird, and the brains of some seal species are comparable to the body size of common laboratory rodents. From this point of view one might expect that selective brain cooling responses to neurotraumatic agents play the same role in large homeotherms as the whole body cooling reported under such circumstances in small mammals (15, 27).

Conclusion. The results of the present study suggest that active, controlled brain cooling is employed during long-duration diving in ducks. We propose that this mechanism is important in reducing the rate of oxygen consumption and, hence, increasing diving capacity, and that it most likely provides protection against asphyxic damage to the brain during and after prolonged dives.

Perspectives

Our paper is the first to announce selective brain cooling during diving. Further studies are necessary to verify the role of this physiological mechanism in defense against asphyxic brain damage. This may have an impact on future comparative studies because many homeotherms and poikilotherms have developed a variety of selective brain cooling mechanisms and in their habitats they are subjected to various neurotraumatic influences. On the other hand, our paper should encourage thermal physiologists to search for efficacious mechanisms of selective brain cooling in mammalian species that repeatedly have to cope with acute hypoxic stress in their natural environment. Further studies are also necessary to specify the role of selective brain cooling in diving strategy of elite divers such as seals (selective brain cooling has not been studied in aquatic mammals so far) and penguins. In common avian species that can fly at extreme altitudes, effect of a simulated altitude hypoxia on the brain cooling could easily be investigated. An involvement of selective brain cooling should be taken into account also in animal models of focal cerebral ischemia, hypoglycemia, and other neurotraumatic disturbances. Effects of blocking selective brain cooling, if it is used under any of the above-mentioned conditions, should clarify the question of its biological significance. Altogether, we hope our study will contribute to strengthening integrative trends in animal physiology.

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