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The effect of interrupting normal nasal breathing on the brain temperature and cerebrospinal fluid pressure in the sheep

Verlin Arnold Krabill
Iowa State University

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by

Verlin Arnold Krabill

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DEDICATION

This thesis is dedicated to my
father, Verlin Christian Krabill,
who endeavored to bring up his
children in the way of the Lord
and to be victorious in His service.

INTRODUCTION

The nasal mucosa has been the object of study by many investigators, especially with reference to its role in the regulation of brain temperature. Some of the first reports on the countercurrent heat exchange between cerebral arterial blood and cranial venous blood were by Taylor (1966) in the goat, Magilton and Swift (1967) in the dog, and Baker and Hayward (1967) in the cat. This functional significance was later demonstrated in the sheep by Baker and Hayward (1968b) and in the antelope by Taylor (1969). Magilton and Swift (1968, 1970b) described two physiologic heat exchange systems in the dog: 1) an "external heat exchange system" between the venous system and the ambient air passing over the nasal mucosa (either by conduction or by evaporation, or both), and 2) an "internal heat exchange system" between the warm blood in the internal carotid artery (destined for the brain) and cool venous blood from the nasal area, which flows around the artery in the cavernous sinus (i.e., countercurrent heat exchange).

Further, Baker and Hayward (1968a, b) noted a decrease in brain temperature in the region of the hypothalamus when air was blown over the nasobuccal surfaces of the sheep. On the other hand, Young et al. (1976) prevented breathing in the panting sheep by mechanically occluding the nostrils, as well as by chemical means, which caused an immediate increase in the hypothalamic temperature. From their work they concluded that evaporation from the surfaces of the upper respiratory tract has an immediate and local effect on the brain temperature. In the cavernous sinus, cool venous blood, draining the nasal mucosa of the nose of the

sheep, dissipates the heat of the warmer arterial blood in the carotid rete; so, there is direct cooling of the brain when the sheep is panting. Also, the results of Cabanac and Caputa (1979) suggested that there is a selective cerebral cooling in humans due to venous blood returning from facial skin via the ophthalmic vein to the cavernous sinus, where a cooling of arterial blood ascending to the brain can take place.

In addition, Baker et al. (1974), while studying the effects of a tracheostomy on brain temperature, reported that when the dog breathed directly through the tracheal opening, there was an immediate rise in the temperature of the cerebral arterial blood and the hypothalamus. Kluger and D'Alecy (1975) had rabbits with tracheal bypass canulas, which enabled them either to breathe normally with the bypass "closed," or to breathe through it with the bypass "open." They found that hypothalamic temperature was influenced by the upper respiratory cooling of venous blood and that the subsequent transfer of heat from the warmer internal carotid artery to the cooler venous blood in the cavernous sinus could effectively cool the brain. Carithers and Seagrave (1976) irrigated the nasal alar fold of dogs having body core temperature elevated to 42° C. At this elevation, a difference of 0.5-1.0° C between brain temperature and body core temperature was maintained for up to 1.5 hours. Caputa et al. (1976b) found that vasodilatation of the nasal mucosa of rabbits paralleled a drop in brain temperature and, conversely, its vasoconstriction paralleled an increase in the brain temperature. The nasal mucosa, therefore, plays an important role in brain temperature regulation.

Heat dissipation of the brain is also dependent upon an increase in the rate of blood flow; however, toleration limits on brain temperature

are important. Carithers and Seagrave (1976) stated that the cells of the central nervous system appear to be among those which are most prone to thermal damage, and the thermal damage is permanent because the cells cannot regenerate. Hayward and Baker (1969) administered 10% CO_2 to monkeys through breathing and the gas produced cerebral vasodilatation, accompanied by an increase in the arterial flow through the brain. By so doing, they were successful in lowering brain temperature toward the arterial blood temperature, which was due to the increase in arterial flow accelerating the removal of metabolic heat. From this experiment, they verified that the arterial blood serves an important role in the removal of heat from the brain and that fluctuations in brain temperature are greatly determined by the temperature of the blood. Abrams et al. (1965) reported that the rate of heat transfer from a heat-producing mass (hypothalamus) to a coolant fluid (arterial blood) must depend, in part, on the rate of flow of that coolant through the heated mass. Therefore, a relatively high temperature difference will exist between the heat-producing mass and the coolant fluid when either the rate of fluid flow is relatively low or the rate of production of heat by the mass is relatively high, or both. So, when the brain becomes warm, under normal physiologic conditions, the arterial blood acts in such a way that the heat is removed from the brain parenchyma, thus preventing an excessive, or perhaps fatal, rise in the brain temperature.

Several investigators demonstrated a linear correlation between cerebral vasodilatation, increased blood volume, and an increase in cerebrospinal fluid pressure (Langfitt and Kassell, 1968), and that the cerebrospinal fluid pressure parallels roughly the changes in the diameter

of the pial artery (Forbes and Wolff, 1928). Further, Magilton and Swift (1970b) and Sawada and Tazaki (1977) attributed both decreases and increases in the cerebrospinal fluid pressure to indicate cerebral vasoconstriction and cerebral vasodilatation, respectively. Even the amount of heat reaching the skin from the deep tissues can be varied by changing the rate of blood flow to the skin (Ganong, 1977). When the cutaneous vessels are dilated, warm blood raises the skin temperature, whereas, in the maximally vasoconstricted state, heat is held centrally in the body.

The purpose, then, of this experiment was to demonstrate that the sheep, when placed on upper respiratory bypass breathing: 1) would exhibit an increase in brain temperature due to a decrease in heat loss in the nasal mucosa; and 2) cerebral vasodilatation would occur as evidenced by an increase in cerebrospinal fluid pressure.

REVIEW OF LITERATURE

Over the years, many investigators have presented many theories concerning the functional significance of the carotid rete of some domestic animals. In the sheep, the carotid rete or rete mirabile epidurale rostrale (International Committee on Veterinary Anatomical Nomenclature, 1973) consists of a compact network of intertwined, freely anastomosing arteries at the base of the brain. It is triangular in outline and lies intracranially between the foramen orbitorotundum, rostrally, to just beyond the foramen ovale, caudally. The hypophysis cerebri (pituitary gland) is situated between, but not surrounded by, the two bilaterally symmetrical halves of the rete, which communicate across the midline. The rete is bathed in venous blood which drains the nasal and facial area and flows through the cavernous sinus. Blood destined for the more dorsally located cerebral arterial circle must first pass through this venous sinus via the well-developed carotid rete (Daniel et al., 1953; Baldwin, 1964).

The main blood supply to the carotid rete in the sheep is from the external carotid artery, via the caudal and rostral rete branches of the maxillary artery. The rete develops as a result of the dividing of these caudal and rostral rete branches into small vessels in the cavernous sinus. The large vessel which emerges from the dorsomedial aspect of the rete is called the internal carotid artery. It pierces the internal layer of the dura mater and gives off a branch, the caudal communicating artery, which joins the basilar artery. The internal carotid artery turns rostrally and, after coursing along the ventral surface

of the optic tract, gives off the middle cerebral and continues further as the rostral cerebral artery. There is no rostral communicating artery, connecting the right and left rostral cerebral arteries (just rostral to the optic chiasma), and no caudal epidural rete mirabile as found in other domestic mammals, such as the ox, cat, dog, and pig (Baldwin, 1964; Getty, 1975).

The arterial blood that flows through the carotid rete, does not mix with the venous blood pool of the cavernous sinus (Baker, 1979). However, the rete arteries are very thin as compared to the thick wall of the internal carotid artery and the cavernous sinus acts as a counter-current heat exchanger. The warmer arterial blood thus loses heat to the cooler venous blood in which the rete is bathed (Baker, 1979).

Venous blood is drained into the cavernous sinus from several sources. Some of it comes from the base of the brain; much of it comes from outside the cranial cavity (Baker, 1979). To outline some of these routes, Baker and Hayward (1968b) injected colored latex rostrally into the angular vein (of the eye), and it entered the nasal cavity through the dorsal and lateral nasal veins, and thus filled the superficial venous plexuses of the nasal mucosa (Dawes and Prichard, 1953) of the same side over the dorsal and ventral maxillary turbinates (conchae), the lateral wall and median septum, and portions of the ethmoturbinates. Latex injected caudally into the angular vein entered the supraorbital vein, which traverses the supraorbital canal and anastomoses with the ophthalmic veins caudal to the orbit, and filled the cavernous sinuses bilaterally. While these injection studies by Baker and Hayward (1968b) demonstrated one pathway for nasal venous drainage to the cavernous sinus, they

stated that "it is likely the ethmoidal and sphenopalatine veins, which also drain the nasal mucosa in sheep (Dawes and Prichard, 1953), are also connected to the cavernous sinus." Taylor (1966) demonstrated a venous pathway draining from the horns of the goat to the cavernous sinus. Magilton and Swift (1969) suggested that the dorsal nasal, angularis oculi, and ophthalmic veins, which form a venous pathway from the nasal area to the cavernous sinus, play a part in the brain temperature regulation in the dog. Robertshaw (1976) and Baker (1979) reported that venous blood from the nose and parts of the mouth of ruminants drain into several intracranial dural sinuses, including the cavernous sinus.

Many investigators have demonstrated the possible role of the nasal passages in brain temperature regulation in various mammals. For instance, Hemingway et al. (1966) reported that the hypothalamic temperature in the sheep, which is supplied by the blood in the internal carotid artery after it emerges from the cavernous sinus (and has, therefore, been cooled by countercurrent heat exchange), is cooler than deep body temperature under normal physiologic conditions. Also, Hellstrom and Hammel (1967) stated that the control of the rate of respiration in the dog and in other panting animals, which has been shown to lower brain temperature, is strongly dependent upon both hypothalamic temperature and ambient temperature. Additionally, Taylor (1969) reported that in the gazelle the hypothalamus was as much as 2.9° C cooler than the carotid arterial blood and he attributed this to the cool venous blood from the nasal passages draining into the cavernous sinus. Baker and Hayward (1968a) found that, in the resting sheep, shifts in hypothalamic and other brain temperatures paralleled temperature shifts in the cerebral arterial

blood, which was cooler than central arterial blood. Further, Baker and Hayward (1968d) demonstrated that, in the recumbent sheep at 20° C, cerebral arterial blood was 0.5° C cooler than carotid blood. During periods of arousal and paradoxical sleep, vasoconstriction of the nasal mucosa and the ear skin occurred and temperatures of the cerebral arterial blood and brain rose without a comparable rise in central arterial blood temperature (Baker and Hayward, 1968c). Baker and Hayward (1968b) stated that the venous blood returning from the nasal mucosa and skin of the head to the cavernous sinus cools the central arterial blood in the carotid rete. Just as the cerebral arterial blood cooled by countercurrent heat exchange with venous blood bathing the rete in the cavernous sinus is considered the "internal heat exchange system," the venous blood cooled by the ambient air passing over the surface of the nasal mucosa is considered the "external heat exchange system" (Magilton and Swift, 1968, 1970a). This is an important factor in the maintenance of hypothalamic temperature in the wool-covered, long-nosed, panting sheep and affects hypothalamic thermoreceptors and temperature regulation in species with a variably developed carotid rete, especially the cat, dog, and sheep (Baker and Hayward, 1968a, b). Fluctuations in cerebral arterial blood as well as the brain temperature occur quite independent of the steady central arterial temperature. Baker and Hayward (1968b) observed that, when air was blown over the nasal passages and buccal surfaces of the sheep, there was a local fall in brain temperature in the region of the hypothalamus. This was attributed to a transfer of heat from the relatively warm arterial blood passing through the carotid rete to the relatively cool venous blood draining from the nasobuccal

surfaces into the cavernous sinuses surrounding the rete. They concluded that the heat exchange between the central arterial blood in the carotid rete and the cranial venous blood in the cavernous sinus is the major factor regulating cerebral arterial blood and brain temperatures in the sheep.

In the sheep, the mucosa, lining the turbinates (conchae) and the rest of the nasal cavity, is highly vascular and contains a large number of arteriovenous anastomoses (Dawes and Prichard, 1953). There are also species differences in the methods of breathing that will have an influence on the level of heat transfer from these mucosal surfaces to the ambient air. For example, in dogs, which pant due to a heat load, most of the respired air enters through the nose and leaves via the mouth (Schmidt-Nielsen et al., 1970). Different patterns of air flow are possible, however, in this species. Also, Blatt et al. (1972) found that two lateral nasal (Steno's) glands, opening in the nasal vestibule, appear to provide a large part of the water for evaporative cooling in the panting dog. The lateral nasal gland is found in a variety of animals (dog, cat, pig, sheep, goat, and small antelopes), which utilize thermal panting for evaporative cooling. Scott (1954) stated that the nasal mucous membrane of man contains many mucus secreting glands, which, by their activity, keep its surface moist. Thus, the modes of breathing and the presence of the nasal and mucous glands greatly influence the effectiveness of the nasal mucosa in brain temperature regulation. But, Robertshaw (1976) observed that ruminants pant with the mouth closed and heat exchange must, therefore, take place at the nasal mucosa, especially since Bligh (1957) demonstrated that there is no

change in the temperature of the blood as it passes through the lungs of calves during panting. Baker and Hayward (1968d) also reported that the closed mouth panting, which occurs in the heat-stressed sheep, accelerates countercurrent heat exchange between arterial blood in the internal carotid and the venous blood in the cavernous sinus. This allows the sheep to maintain a relatively cool brain in the face of a rising body temperature. The panting mechanism, thus, allows for localized cooling of the most sensitive brain tissue.

The heat loss, which has been demonstrated to take place at the nasal mucosa, depends not only upon the rate and pattern of air flow over the nasal passages, but also upon the rate of blood flow through its mucosal surfaces. In a cool environment, when the respiratory rate is relatively constant, vasoconstriction of the mucosal vessels decreases the nasal heat loss and vasodilatation increases it. In a warm environment, panting increases the evaporation in the nasal cavity, but vasomotor activity can still influence the heat loss there. When the nasal mucosa is constricted, the amount of cool venous blood bathing the rete decreases and cerebral arterial blood temperature rises toward central arterial temperature; when the nasal mucosa is dilated, the amount of cool venous blood bathing the rete increases and blood in the rete is cooled below central arterial temperature (Baker, 1972).

Intracranial pressure and its relationship to the diameter of cerebral vessels have been the interest and concern of many investigators. Magilton and Swift (1970b) concluded that changing the temperature of the vascular plexus at the tip of the nose of dogs produced repeatable variations in the cerebrospinal fluid pressure. The response of the

cerebral vasculature to hot water irrigation was an increase in cerebrospinal fluid pressure. Conversely, the response of the cerebral vasculature to cold water irrigation was a decrease in cerebrospinal fluid pressure. They demonstrated that there was no correlation between blood gas levels and the changes in cerebrospinal fluid pressure (Magilton and Swift, 1970b), and they considered changes in cerebrospinal fluid pressure as an indication of constriction and dilatation of cerebral vessels similar to other workers in the field. Forbes and Wolff (1928) demonstrated that an increase in pial vessel diameter was accompanied by an increase in the cerebrospinal fluid pressure. Langfitt and Kassell (1968) stated that a dilatation of cerebral vessels causes an increase in cerebral blood volume and a rise in intracranial pressure. Risberg et al. (1969) noted that acute transient rises of intracranial pressure in the lateral ventricle of the human brain were accompanied by an increase in cerebral blood volume. While studying the effects of CO_2 on collateral circulation in the human brain, Sawada and Tazaki (1977) measured the cerebrospinal fluid pressure to determine the extent of cerebral vasodilatation.

The demonstration by Baker and Hayward (1968b), that brain temperature decreased with an increase in the rate of air flow through the nasal passages, as previously mentioned, indicates that an increase in the rate of air flow through the passages results in accelerated heat loss from the blood circulating in the nasal mucosa, which, in turn, causes a decrease in the temperature of the venous blood flowing toward the cavernous sinus. Consequently, more heat is transferred from the arterial to venous blood in the cavernous sinus, resulting in a lowering of brain

temperature.

An increase in intracranial pressure is reflected in an increase in the cerebrospinal fluid pressure. Cobb and Fremont-Smith (1931) demonstrated that the cerebrospinal fluid pressure in man could be reduced to zero in some cases with 20 to 30 deep breaths. They attributed the fall in pressure partly to the withdrawing of blood from the cerebral veins as a result of the increased negative intrathoracic pressure and, perhaps, partly to a cerebral vasoconstriction that was observed in similar experiments in cats (Wolff and Lennox, 1930). The possible causes for the reduction of cerebrospinal fluid pressure as shown by Cobb and Fremont-Smith have never been fully resolved. Most workers, however, attribute this change to blood gas levels.

MATERIALS AND METHODS

This experiment has been designed to compare brain temperature and cerebrospinal fluid pressure changes as they relate to zero air flow and the air flow during normal nasal breathing as they relate to changes in the rate of air flow through the nasal passages.

Six sexually matured Rambouille ewes (30 to 60 kg body weight), aged 14 months to three years, were acquired from the National Veterinary Services Laboratory in Ames. The animals were housed in the LAR facilities and, prior to investigation, they were moved to the veterinary anatomy holding room for acclimatization. During this period of acclimatization, rectal temperatures, pulse rates, respiration rates, and heart rates were taken on each animal three times a day.

An upper respiratory bypass canula, which was designed by Kluger and D'Alecy (1975) for rabbits, was modified and adapted to the sheep (Figure 4). Several days were allowed to elapse after implantation of the modified canula in order for the animals to recover and become accustomed to the implant. Then, an indwelling catheter was placed in the cerebellomedullary cistern according to the method of Buck (1964) and a thermistor was placed in the brain employing a technique which was designed by the author and coworkers.

In all cases, each sheep was held off feed 24 hours prior to surgery. Thirty minutes before the induction of halothane anesthesia, atropine was injected intramuscularly at a rate of approximately 1/4 mg/kg body weight to reduce salivation during the operation. The surgical area was first clipped and scrubbed with several applications of surgical soap

until cleaned and then swabbed with a 1:750 aqueous solution of benzalkonium chloride ("Zephiran" chloride; Winthrop Laboratories, Division of Sterling Drug, Inc., New York).

The techniques, employed for implantation, are described in greater detail as follows.

1) For the implantation of the upper respiratory bypass canula, a ventral midline incision of the caudal one-third of the neck was made and the trachea exposed. The trachea was transected and the cranial and caudal tubal extensions of the canula (Figure 4) were inserted into the cut ends of the cranial and caudal segments, respectively, of the trachea. A double loop of No. 2 surgical silk was fastened about each cut end of the trachea, securing an airtight seal between the cut ends of the trachea and the tubal extensions of the canula. Supporting sutures were placed through the cut ends of the trachea along the lateral sides of the canula in order to draw the ends of the trachea toward the body of the canula. All sutures were placed to prevent the trachea from sliding off the tubal extensions of the canula when the animal forcefully extended its neck. The wound was then closed, leaving the body of the canula extending beyond the surface of the skin. Each day, the cap of the body of the canula was unscrewed and the flow-through insert (Figure 4) was removed for cleaning.

2) The technique for placing the indwelling catheter required a six-inch vinyl catheter, having an outside diameter (O.D.) of 0.088 inch and an inside diameter (I.D.) of 0.054 inch. One end

was bent at a right angle and this short end (1.5 cm) would subsequently be placed through the spinal dura mater so that it would be just dorsal to the spinal cord with the free end pointed cranially into the cerebellomedullary cistern. Just above the angled tip of the catheter, two collars were made by linearly compressing the tubing; both the angled tip and the collars were formed by heating the tubing after placing a stiff wire stylet in the lumen. After halothane anesthesia was attained by using a mask over the muzzle, the animal was placed on the surgery table and a plastic tube to the tracheal canula (with the bypass insert in place) was substituted for the mask. The sheep was placed on its sternum and its muzzle (planum nasale) was placed in a moderately tapered funnel. This funnel was firmly fixed in the table by placing the tapered end into a hole bored in the table. A strap, fastened to the edge of the large opening of the funnel on one side, was pulled tightly over the dorsum of the head and buckled on the other side to hold the head firmly in the funnel. With the head securely held in the funnel, the animal was pushed forward to flex the head and widen the space at the atlantooccipital junction as desired for optimal surgical accessibility. Wooden blocks, mounted on screw-adjustable metal rods, were used to maintain the animals in ventral recumbency by exerting external pressure on the thorax. After the animal was surgically prepared, a three-inch skin incision was made along the dorsal midline of the neck beginning at the external occipital protuberance and continued

caudally to expose the nuchal ligament. The right and left halves of the nuchal ligament were separated and retracted, exposing the extensor muscles of the head (rectus capitis dorsalis major and minor). These muscles were bluntly dissected from their insertions with a periosteal elevator, exposing a tough, dorsal atlantooccipital membrane over the caudal portion of the cerebellomedullary cistern. Two lengths of suture material (000 cardiovascular, black, braided silk with a size 20, Ferguson, 1/2 circle, taper-point needle) were placed sagittally (one on each side of the dorsal midline) through the exposed dorsal atlantooccipital membrane. A sagittal midline incision was subsequently made between the two sutures. It was important that this incision be made exactly on the midline, which is at the center of the triangle bounded by the "V" shaped notch separating the articular surfaces of the atlas and the caudal edge of the external occipital crest, to avoid severing blood vessels. The incision was extended cranially and caudally with a hemostat. The free ends of the suture material which had been passed through the atlantooccipital membrane, were pulled laterally and served to enlarge the operative area. The epidural fat was removed and the dura mater was exposed over the cerebellomedullary cistern. A sterile 25 gauge needle on a 12 cc syringe was used to withdraw 5 to 8 cc of cerebrospinal fluid from the cistern. The puncture hole in the dura mater, made by the needle, was enlarged to allow the passage of the catheter. The catheter tip was then passed through the opening in the

atlantooccipital membrane, inserted through the dura mater, and directed rostrally into the cerebellomedullary cistern until the first collar rested on the dura mater. The lengths of suture material, which had been passed through the atlantooccipital membrane, were then drawn around the catheter in such a way as to anchor the second collar to the dorsal atlantooccipital membrane. The retractors were released, allowing both parts of the nuchal ligament to return to the midline, and the skin was closed with a continuous mattress suture using No. 3 silk. The cerebrospinal fluid, previously withdrawn from the cerebellomedullary cistern, was replaced through the catheter.

A Luer stub adapter was placed on the external end of the catheter to facilitate its attachment to a pressure transducer or Luer-lock syringe. When it was not in use, the adapter was capped.

3) While the sheep was still under anesthesia, the thermistor was placed in the brain. A stereotaxic instrument designed for the beagle was used (with modification of technique) for placement of the thermistor at the site where the internal carotid artery emerges from the cavernous sinus. The sheep was placed on the table in ventral recumbency and the mouthbar was placed caudal to the dental pad. The orbital fixation bars were placed on the nose to hold the nose firmly against the mouthbar. The orbits were aligned equidistant from the stereotaxic tracks. The dorsum of the skull was leveled from side to side by visual inspection.

A midline incision was made from a transverse line between the most dorsal parts of the orbits and was extended caudally to a transverse line between the rostral margin of the base of the ears. The skin and fascia were reflected laterally with a self-retaining wound retractor and the periosteum was then reflected laterally with a periosteal elevator. The area of convergence of the parietofrontal sutures as well as the interfrontal suture was identified.

A vertically oriented stereotaxic drill bit (0.1360 inch, No. 29) was placed on the midline 10 mm rostral to the point of convergence of the parietofrontal suture. The drill was then moved laterally 10 mm from the midline and a drill bit marker was used to mark the spot (and to make a depression), so that the drill bit would not slide on the convex external surface of the skull. The skull was then trephined to the level of the dura mater. Three additional trephinations, in the form of a triangle around the first, were made to assist in anchoring cranioplastic to the surface of the skull. All four trephinations were tapped (size 8-32 x 3/8") for subsequent threadings of flat headed nylon screws.

The nylon screw that was placed in the trephination for the thermistor was center drilled. The size of the hole was determined by the size of the thermistor to insure a snug fit as the thermistor was passed through the screw for placement in the brain.

A two and one-half inch thermistor, previously plumbed, was passed through the hole drilled in the screw and continued until the base of the skull was encountered. The thermistor was then withdrawn a distance of two mm. The placement of the thermistor at the exact (desired) site was not possible because of the difference in size and shape of the heads of the sheep.

For protection, brass sleeves were fitted around the extracranial portions of the thermistor. Cranioplastic was then applied to the surface of the skull, around the heads of the four nylon screws, and built up around the brass tubing.

After the cranioplastic had hardened, the top of the brass tubing was crimped around the hub of the thermistor with needle-nose pliers to keep it from, (1) moving up and down, and, (2) turning around on its long axis. The thermistor (and its encasement) was then padded with cotton and the latter was held in place by adhesive tape. A piece of stockinette was then slipped over the head and neck of the sheep and held in place by plastic tape.

When the animal was able to stand erect, during recovery from the anesthesia, the thermistor was attached directly to the Grass polygraph machine (Model 5c; Grass Instrument Company, Quincy, Massachusetts) and the catheter was attached to this recorder by way of a pressure transducer. These connections monitored the brain temperature and the cerebrospinal fluid pressure, respectively.

The brain temperature and the cerebrospinal fluid pressure were first recorded with the flow-through insert (on normal breathing) in the

canula and then with the bypass insert. A bead thermistor, placed inside the nasal vestibule, was used to determine the efficacy of the bypass (no oscillation demonstrable on the polygraph). While on a bypass period, cool air (at approximately 45° C) was introduced cranially over the nasal mucosa through a needle embedded into the wall of the upper respiratory bypass canula to obtain results on subsequent changes in the temperature of the brain and the cerebrospinal fluid pressure.

The sheep were usually eating within a few hours after the initial readings which were taken during recovery from anesthesia. Antibiotics were given I/M immediately after the operation and were continued throughout the postsurgical recovery and the experimental periods. Small amounts of fluid surfaced externally around the catheter, in some cases. To prevent local infection from occurring, the area was cleansed and local antibiotics were applied daily.

After the experimental trials were completed on each sheep, it was sacrificed and the head was embalmed and injected to outline the cerebral vascular network (Figure 6). Then, after decapitation, the head was sagittally sectioned with a band saw, approximately two cm on either side of the midline (Figure 7). This facilitated dissection of the cerebral structures in order to determine the location of the tips of the thermistors and their relative proximity to the cerebral arterial circle, and, in particular, to the internal carotid artery (Figure 1).

RESULTS

A total of 20 trials were made on six sheep: three trials on each of four sheep and four trials on each of the remaining two sheep. A trial consists of four phases comprising: (1) a period of normal breathing (via the nasal cavity), (2) followed by a period of tracheal breathing (via the upper respiratory bypass canula), (3) then a second period of normal breathing, and (4) a second period of tracheal breathing. Brain temperature and cerebrospinal fluid pressure were not recorded simultaneously in all animals because of the inability of some to endure the stress.

The trials were conducted on the resting and unanesthetized sheep in a standing position. The room, where polygraph recordings were made, was kept as quiet as possible in order to minimize false recordings. It was found that even the least movement within the field of vision of the experimental animal or slightest audible sound would cause a change in the cerebrospinal fluid pressure and brain temperature (the latency of the latter depended upon the distance of the thermistor from the desired site). The desired site was the point of emergence of the internal carotid artery from the cavernous sinus (Figure 1.A).

The location of the tips of the thermistors (represented in Figure 1 by black dots) as determined on postmortem, was as follows:

1) Sheep No. 53--the thermistor tip was one mm medial to the internal carotid artery where the caudal communicating artery branches off toward the basilar artery (B).

2) Sheep No. 37--the thermistor tip was located one mm caudal to the caudal cerebral artery and two mm lateral to the caudal communicating artery (C).

3) Sheep No. 150--the thermistor tip was located caudal, lateral, and adjacent to the junction of the arterial branch to the rostral mesencephalic tectum and the caudal communicating arteries (D).

4) Sheep No. 76--the thermistor tip was located caudally, adjacent to the arterial branch to the rostral mesencephalic tectum, and one mm lateral to the caudal communicating artery (E).

5) Sheep No. 174--the thermistor tip was rostral and lateral to the junction of the rostral cerebellar and caudal communicating arteries (F).

6) Sheep No. 26--this animal died (after recordings were completed) and, therefore, was not embalmed and injected in order to locate the tip of the thermistor.

In the sheep (No. 150), where the thermistor was adjacent to the cerebral arterial circle (Figure 1.D), 30 seconds elapsed before the first detectable change in brain temperature was recorded, with the first change from nasal to bypass breathing. In another sheep (No. 174), where the thermistor was 1 mm lateral to the cerebral arterial circle (Figure 1.F), the first detectable change took place after two minutes and 40 seconds following a similar change in the breathing route.

Only four brain-temperature recordings were considered in the analyses of the results. These temperatures correspond to:

1) normal breathing (N1);

2) bypass breathing, temperature at maximum change (BP1);

3) normal breathing, temperature at maximum change (N2); and

4) bypass breathing, temperature at maximum change (BP2).

On all sheep and trials (Table 1), the average temperatures for these four phases of each trial (based on 12 observations) were:

$N1 = 38.56^{\circ}$ C

$BP1 = 38.89^{\circ}$ C

$N2 = 38.51^{\circ}$ C

$BP2 = 38.90^{\circ}$ C

Four questions were constructed concerning the differences among the four phases mentioned above and, for each question, a test statistic (a t-value) was calculated and a probability attached. The probability is considered as the strength of the evidence for the supposition that the calculated difference examined represents a true underlying difference of zero. The questions were as follows.

1) Does N1 differ from N2? $(N1 \neq N2)$

2) Does BP1 differ from BP2? $(BP1 \neq BP2)$

3) Does the change from N1 to BP1 differ from the change from N2 to BP2? $(N1 - BP1 \neq N2 - BP2)$

4) Does the average of the two bypass measures differ from the average of the two normal measures?

$$\frac{N1 + N2}{2} \neq \frac{BP1 + BP2}{2}$$

Table 1. Brain temperature (^{o} C)

Sheep No.	Phase[a]	Trial			
		No. 1	No. 2	No. 3	No. 4
37	1	38.833	38.667	38.000	
	2	38.833	38.667	38.000	
	3	38.666	38.334	37.666	
	4	38.750	38.501	38.000	
26	1	38.333	38.833	Trial not	
	2	38.167	38.917	used for	
	3	38.833	39.000	analysis	
	4	39.334	38.916		
174	1	39.501	Trial not	38.667	
	2	39.501	used for	38.833	
	3	39.501	analysis	38.585	
	4	39.752		38.833	
150	1	Trial not	38.400	38.501	38.835
	2	used for	39.000	39.167	39.500
	3	analysis	38.500	38.500	38.668
	4		39.000	39.167	39.668
76	1	Trial not	Trial not	37.000	
	2	performed	performed	37.800	
	3			37.200	
	4			37.600	
53	1	Trial not	Trial not	Trial not	38.670
	2	performed	performed	performed	39.330
	3				38.670
	4				39.330

[a]Phase 1 = normal breathing (N1); Phase 2 = bypass breathing (BP1); Phase 3 = normal breathing (N2); Phase 4 = bypass breathing (BP2).

Question	t-value	Probability	Remarks
$N1 \neq N2$	0.625	$0.5 < P < 0.6$	Very probably no difference
$BP1 \neq BP2$	0.125	$0.8 < P < 0.9$	Almost assuredly not a difference
$N1 - BP1 \neq N2 - BP2$	0.54	$0.5 < P < 0.6$	No evidence for a difference
$\frac{N1 + N2}{2} \neq \frac{BP1 + BP2}{2}$	7.2	$P < 0.01$	Almost unquestionably there is a difference

Therefore, the brain temperatures, measured at the sites near the cerebral arterial circle in these sheep (Figure 1), were significantly higher when the animals were breathing through the upper respiratory bypass canula and, when the sheep were placed on normal nasal breathing, they were able to maintain a relatively lower brain temperature. This occurred in every trial of all sheep even though the degree of change varied with the location of the thermistor and the behavior of the individual animal. In the sheep, where the thermistor was nearest the desired site in the brain, the brain temperature difference between tracheal breathing and normal nasal breathing registered a high of 1^{o} C in these resting, standing, unanesthetized sheep. The average difference in brain temperature in all trials, between normal breathing and tracheal bypass breathing, however, was 0.37^{o} C.

The same statistical method was used for evaluating the results regarding the changes in the cerebrospinal fluid pressure. On all sheep and trials (Table 2), the average pressures (in mm Hg) for the four phases of each trial (based on 17 observations) were:

Table 2. Cerebrospinal fluid pressure (mm Hg)

Sheep No.	Phase[a]	Trial			
		No. 1	No. 2	No. 3	No. 4
37	1	-42.306	-38.460	23.076	
	2	-38.460	-30.768	34.614	
	3	-38.460	-34.614	38.460	
	4	-34.614	-26.922	42.306	
26	1	69.228	30.768	Trial not	
	2	73.074	24.999	used for	
	3	69.228	28.845	analysis	
	4	73.074	28.845		
174	1	73.074	Trial not	80.766	
	2	65.382	used for	80.766	
	3	76.920	analysis	80.766	
	4	80.766		76.920	
150	1	57.690	65.382	46.152	57.560
	2	49.998	79.966	60.736	61.406
	3	42.306	65.382	49.198	61.406
	4	56.890	50.798	49.198	75.990
76	1	-07.692	38.460	-19.230	
	2	-15.384	53.044	-07.692	
	3	-15.384	76.120	-15.384	
	4	-15.384	64.582	-03.846	
53	1	-23.076	Trial not	11.538	-03.846
	2	-11.538	used for	-19.230	-18.430
	3	23.076	analysis	-49.998	-22.276
	4	03.846		-53.844	-26.122

[a]Phase 1 = normal breathing (N1); Phase 2 = bypass breathing (BP1); Phase 3 = normal breathing (N2); Phase 4 = bypass breathing (BP2).

N1 = 24.652

BP1 = 26.028

N2 = 25.623

BP2 = 26.028

The same four questions were answered similar to those concerning the brain temperature.

Question	t-value	Probability	Remarks
$N1 \neq N2$	0.24	$P > 0.5$	Most likely no difference
$BP1 \neq BP2$	0.0	$P > 0.5$	No difference
$N1 - BP1 \neq N2 - BP2$	0.17	$P > 0.5$	Practically no difference
$\frac{N1 + N2}{2} \neq \frac{BP1 + BP2}{2}$	0.31	$P > 0.5$	No evidence for difference

The average pressures of the four phases registered a higher cerebrospinal fluid pressure with the animal on bypass breathing ($\frac{26.028 + 26.028}{2}$) than when the sheep was breathing normally through the nasal cavity ($\frac{24.652 + 25.623}{2}$) even though the difference (0.8905) is not statistically significant.

Although some differences exist between the average pressures of the four phases of the trials (N1, BP1, N2, and BP2), there are no significant differences in the correlation between certain combinations of the four phases as exemplified in the answers to the four questions listed previously. The probability is such that very likely no difference occurs in the answers to all these questions. The cerebrospinal fluid pressure

varied considerably and in some cases it fluctuated as much as 40 to 75 mm Hg between the high and low pressures within a trial. In most cases, each rise and fall in the pressure was seemingly accompanied by its subsequent fall and rise, respectively.

A correlation was then made between brain temperature and the cerebrospinal fluid pressure values (a = correlation coefficient; b = probability):

Comparison	Correlation of CSF pressure and brain temperature
Of total values	a = 0.38393 b = 0.0071
Of normal nasal breathing values	a = 0.36987 b = 0.0752
Of tracheal bypass breathing values	a = 0.41543 b = 0.0435

When total values for brain temperature and cerebrospinal fluid pressure were considered, there was a significant, positive, linear correlation between the two (significant at 99%), i.e., when the brain temperature increased, the cerebrospinal fluid pressure also increased, and, correspondingly, when the brain temperature decreased, the cerebrospinal fluid pressure also decreased. This same positive, linear correlation was observed when the normal nasal breathing values of both brain temperature and cerebrospinal fluid pressure were considered (significant at 90%), and, also, when the tracheal bypass breathing values of both brain temperature and cerebrospinal fluid pressure were considered (significant at 95%).

A bead thermistor was placed (hand-held) in the nasal vestibule for the expressed purpose of verifying the efficacy of the upper respiratory bypass canula; for example, when the sheep was placed on tracheal breathing, there would be no oscillation registered on the polygraph paper that would be indicative of breathing through the nasal cavity and, conversely, oscillation would, of course, appear on the paper with nasal breathing. On several trials, the temperature at the nasal vestibule was 6^{o} C higher on tracheal breathing compared to normal nasal breathing. It is recognized for this observation to be meaningful, the rate of air flow and blood flow in the nasal mucosa would also have to be considered.

It is worthwhile mentioning here that, in three trials on two sheep (Nos. 26 and 37), the air flowed more freely through the right nostril than through the left. The use of the bead thermistor, however, was restricted, in all other cases, to the right nasal vestibule of the sheep in order to obtain the results from uniformity.

DISCUSSION

The use of the upper respiratory bypass canula (Figure 4) has proven to be an effective way to interrupt normal nasal breathing in the sheep. When the "flow-through insert" was placed in the canula, the sheep breathed normally through its nasal passages, and when the "bypass insert" was put in place, the sheep breathed through the tracheal opening. To check for the complete cessation of air flow through the nasal cavity during bypass breathing, a bead thermistor was placed in the nasal vestibule of the sheep. The sensitivity of the bead thermistor was such that air movements resulting from inspiration and expiration could be detected. This served to check the efficacy of the tracheal bypass canula.

In the course of this investigation, when the sheep breathed directly through the tracheal bypass canula, there was an increase in the brain temperature (av. 38.90^{o} C), whereas, when normal breathing was restored, the brain temperature decreased (av. 38.53^{o} C)(Figure 2). This is in agreement with the findings of Kluger and D'Alecy (1975) on the rabbit. Further, the temperature at the nostril was higher when the brain temperature increased (bypass 36^{o} C) and was lower when the brain temperature decreased (normal breathing 30^{o} C). This demonstrates the conspicuous difference in the temperature of the nasal cavity with air flowing over the nasal mucosa during normal breathing and the lack of air flow through the nasal cavity during tracheal breathing. The higher temperature of the air at the nostril during bypass breathing would, at first impression, indicate that relatively more heat is being lost from the circulating

blood in the nasal mucosa during this phase of breathing. One must bear in mind, however, that the only air that is being heated during this phase is that which is lying static within the nasal passages whereas, in normal breathing, air is being heated as it is circulating through the entire nasal passage. From the above, it can be inferred that, when the animal was placed on tracheal bypass breathing, there would be no significant loss of heat from the circulating venous blood in the nasal mucosa to the ambient air. Consequently, the warmer venous blood passes from the nasal area to the cavernous sinus, which could not lower the core temperature blood of the carotid rete. Therefore, the arterial blood reaching the cerebral circle after passing through the carotid rete (bathed in the venous blood of the cavernous sinus) would register a higher cerebral arterial blood temperature as compared to the lower temperature registered on normal breathing with air flowing over the moist, nasal passages, where loss of heat from the nasal mucosa takes place.

The observations from this investigation, using more precise methods for measurement, substantiate work that has previously been done. For example, Baker and Hayward (1968b) found that, when air was blown rapidly over the nasal mucosa of unanesthetized sheep, temperatures dropped in the cavernous sinus, and the cerebral arteries and in the brain. They demonstrated by injecting colored latex into the nasal veins of embalmed heads that the venous blood, affecting the temperature of the arterial rete in the cavernous sinus, came from the nasal passages of the sheep. Inasmuch as the venous blood in the cavernous sinus is cooler than the arterial blood traversing the rete, countercurrent heat exchange takes

place between them. Baker et al. (1974) noted an immediate rise in cerebral arterial blood temperature while dogs were breathing through a tracheostomy. The cerebral arterial blood was, however, cooler when they breathed normally through the nasal passages. Young et al. (1976) mechanically occluded the nostrils of the sheep, resulting in an immediate rise in hypothalamic temperature. On the other hand, this temperature decreased to control values when the nostril occlusion ceased.

These results are further substantiated by several investigators (Baker and Hayward, 1968a, in the sheep; Magilton and Swift, 1968, in the dog; Robertshaw, 1976, in ruminants). These workers stated that the cool venous blood from the nasal passages enters the cavernous sinus at the base of the brain. The carotid arterial supply to the head also passes through the cavernous sinus where it breaks up into the carotid rete. This arrangement allows the exchange of heat between the two blood streams which is enhanced by the fact that they are flowing in opposite directions and are, in effect, a countercurrent heat exchanger. Magilton and Swift (1968) described two physiologic heat exchange systems in the dog for the control of brain temperature. One is a heat loss from the venous blood in the nasal mucosa to the ambient air or "external heat exchanger," and the other is a heat loss from the arterial blood in the carotid rete to the venous blood in the cavernous sinus or "internal heat exchanger." Since the sheep pants with its mouth closed, all heat exchange from the venous blood in the nasal mucosa to the ambient air would take place in the external heat exchange system. Bligh (1957) demonstrated that there is no blood temperature change in the lungs during panting in calves; Ingram and Whittow (1962), however, stated

that there is considerable cooling of blood draining from the head in the ox. The findings of these workers demonstrated the fact that the heat exchange is a local condition and not a systemic one and that fluctuations in brain temperature under normal conditions are independent of the deep body or the core temperature.

In the present study, during two trials, rectal temperature was taken on one sheep during normal as well as bypass breathing. On one trial, even though the brain temperature increased (0.58° C) during tracheal breathing, there was no difference in the rectal temperature (103.2° F = 39.6° C)(Figure 3). On the other trial, using the same method, the rectal temperature increased 0.2° C in comparison to the increase in brain temperature of 0.83° C, which could not be explained within the framework of this experiment. The work of Baker and Hayward (1968b) showed that central arterial blood temperature tended to remain steady even at times when cerebral arterial temperature showed pronounced thermal shifts associated with cranial peripheral vasomotor activity consequent to behavioral activity. Again, these results showed that the rise and fall in brain temperature is a local change and that it occurs independently of the body (core) arterial blood temperature.

In the course of this investigation, a marked variation in the temperature of the cerebral arterial blood was observed, which was attributed to the location of the thermistor in the brain. For instance, in the change from normal nasal breathing to bypass breathing, the time for the recorded increase in brain temperature varied from several seconds to a few minutes (10 seconds to 12 minutes and 30 seconds). This delayed response to brain temperature increase was greatly influenced

by the site of the implanted thermistor in relation to the vessels of the cerebral arterial circle (Figure 1). The increase in the temperature of the blood in the cerebral arterial circle was greater at the desired site (described previously), but it was progressively less as the site of the implanted thermistor was placed caudally toward the basilar artery. This observation is concordant with the findings of Andersson and Jewell (1956) in the goat. The location of the tip of the thermistor was, however, dependent on the size and shape of the sheep heads and on visual inspection employed in the threading of the skull for the screw supporting the thermistor.

This variation in brain temperature, with reference to the site of the implanted thermistor, can be explained in light of the following. The brain of the sheep receives its major blood supply from the carotid arteries, contributing vessels to the cerebral arterial circle. In small ruminants, blood flows caudally in the basilar artery away from the cerebral arterial circle (Andersson and Jewell, 1956; Baldwin and Bell, 1963). The blood in the carotid system comes into intimate contact with cool venous blood in the cavernous sinus and, therefore, a major portion of the brain of the sheep is under the influence of the internal heat exchange system as previously described.

In the brain stem of the sheep, according to Baker and Hayward (1968b), a gradient of increasing temperature exists from the cerebral arteries in the basal subarachnoid space toward the center of the brain. They concluded that, in general, the warmest brain sites are those which are farthest from the source of cool blood in the subarachnoid space surrounding the brain and the resistance to a change in temperature was

most marked in the warm, deep brain sites, and least marked in the cooler hypothalamic area which is near the cerebral arterial circle. Also, the degree and rapidity of the temperature drop in any brain site was related to the thermal inertia of the site and the degree of the temperature drop in the cavernous sinus. Besides, they (1968a) stated that the level of temperature in any brain site above the cerebral arterial blood temperature appears to be dependent not only, in part, on the distance of the site from the source of cool blood, but also, in part, on the rate of local heat production and blood flow. Pasztor et al. (1965) reported that, when the surface of the brain is chilled, the temperature of the deeper structures is not reduced. They further asserted that evidence had been published to indicate that, if the temperature is not reduced to below 0^{o} C at the surface and so without destruction of cortical cells, the cooling does not extend deeper than 3 to 5 mm. This would account for many of the observed latent changes in the brain temperature with reference to the location of the thermistor.

Another objective of this experiment was to demonstrate that cerebral vasodilatation as evidenced by an increase in cerebrospinal fluid pressure would occur during bypass breathing. In this study, the results, although not as dramatically conclusive as anticipated, revealed a linear accompaniment between brain temperature and cerebrospinal fluid pressure. However, within the framework of this experiment, it would be difficult to show conclusively that the increased intracranial pressure was the evidence of cerebral vasodilatation. There was a trend, though, in that direction: when brain temperature elevated, the cerebrospinal fluid pressure also increased and vice-versa.

The results of some investigators, who worked previously in this related field, showed a linear correlation between vessel diameter and cerebrospinal fluid pressure (Forbes and Wolff, 1928) as well as increased intracranial pressure and cerebral vasodilatation (Risberg et al., 1969; Sawada and Tazaki, 1977). The above changes can be explained by the displacement of the space in an immovable, nonexpansive cranial vault by the increase in the size of the vessels which, in turn, exert pressure on the cerebrospinal fluid; conversely, when cerebral vasoconstriction takes place, a decrease in cerebrospinal fluid pressure results. Conceivably, then, with an increased arterial blood flow through the cerebral structures in an effort to lower the brain temperature, an increase in intracranial pressure might occur. The increase in blood volume which might accompany an increased arterial flow in an already compact and peripherally-limited cranial cavity would cause compression of surrounding tissues, especially the ventricles and the brain parenchyma.

The polygraph recordings of this aspect of the study, though somewhat significant, were not as clear-cut and defined in their trajectory as compared to those that were made on brain temperature. Investigations concerning other parameters, such as pressure-dependent outflow resistance (via arachnoid granulations) controlling the rate of cerebrospinal fluid absorption, intracranial compliance through distention of meningeal membranes and the compression of cerebral veins (Johnson et al., 1978), and arterial blood pressure and cardiac output (Chao and Hwang, 1972), would, perhaps, yield a meaningful correlation to complement future studies in this field. Further, Chao and Hwang (1972) listed a variety of other factors that would regulate the intracranial cerebral blood

flow. Michenfelder et al. (1969) indicated certain techniques associated with neuroanesthesia and their relevance to intracranial pressure, cerebral blood flow, and cerebral metabolism.

In support of the hypothesis already stated, cool air was mechanically introduced through the nasal passages, via the special device on the bypass canula (Figure 4), to decrease the temperature of the arterial blood destined to supply the brain and to reduce the brain temperature, and, thus, to reduce intracranial pressure. Some attempts had been made, in three sheep, to reverse the trend by lowering the brain temperature, even when the animals were on tracheal breathing. To accomplish this objective, cool compressed air from a refrigerated pressure bottle at approximately 7.0° C was introduced initially at the rate of 10 liters per minute, which was later increased to 50 liters per minute. In this procedure, the air was passing in a direction contrary to the animals' natural way of breathing to which the sheep apparently could not adapt. The excitation that accompanied this procedure was thought to increase brain temperature which masked decreases in brain temperature that might have occurred. Consequently, the use of this device was discontinued.

Lastly, it would be worth noting that, in three trials on two sheep, while using the bead thermistor there was less resistance observed to the air flow through the right nostril than through the left. Besides individual variation between experimental animals, this difference could, perhaps, be attributed to several factors, such as the anatomic structure and pathologic conditions of the nasal cavity, including the presence of some foreign body obstructing the free flow of air, and the like.

Therefore, to use bead thermistors in the nostrils of sheep, abnormal resistance to air flow could be an important factor to avoid misleading results.

CONCLUSION

The objectives of this study, previously stated, were to demonstrate that the sheep, when placed on upper respiratory bypass breathing: 1) would exhibit an increase in brain temperature due to a decrease in heat loss in the nasal mucosa; and 2) cerebral vasodilatation would occur as evidenced by an increase in cerebrospinal fluid pressure. Within the framework of this investigation, the following conclusions have been formulated:

1) The cessation of air flow over the nasal passages caused a decrease in heat loss from the venous blood of the nasal mucosa, when the sheep breathed through the tracheal bypass canula (not via the nasal cavity). The brain temperature rose concurrently.
2) Conversely, the air flow over the nasal passages caused an increase in heat loss from the venous blood of the nasal mucosa, when the sheep breathed normally through the nasal cavity. The temperature of the brain decreased concurrently.
3) These results, then, are evidence of the important role of normal nasal breathing in the control of cerebral arterial blood temperature.
4) Although several investigators have associated cerebral vasodilatation with an increase in cerebrospinal fluid pressure, this experiment was unable to conclusively demonstrate this correlation due to insufficient data for analysis, in particular, the parameter of systemic arterial blood pressure of the experimental sheep.

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APPENDIX: ILLUSTRATIONS

Figure 1. The location of implanted intracranial thermistors at the cerebral arteries in the sheep (Getty, 1975. Courtesy of W. B. Saunders Co.)

A	Desired site of thermistor
B	Sheep No. 53
C	Sheep No. 37
D	Sheep No. 150
E	Sheep No. 76
F	Sheep No. 174

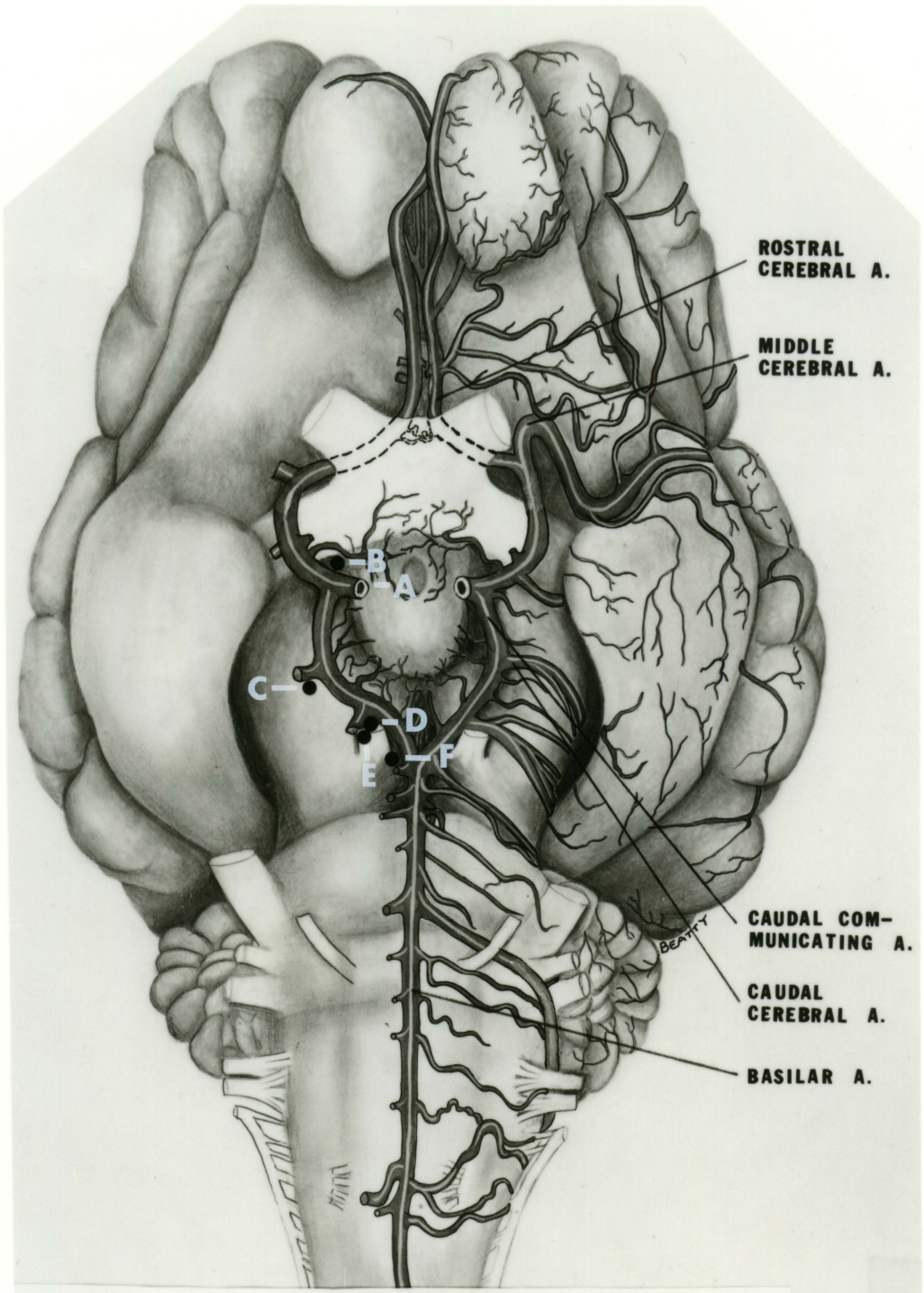

FIG. 1 The location of implanted intracranial thermistors at the cerebral arteries in the sheep.

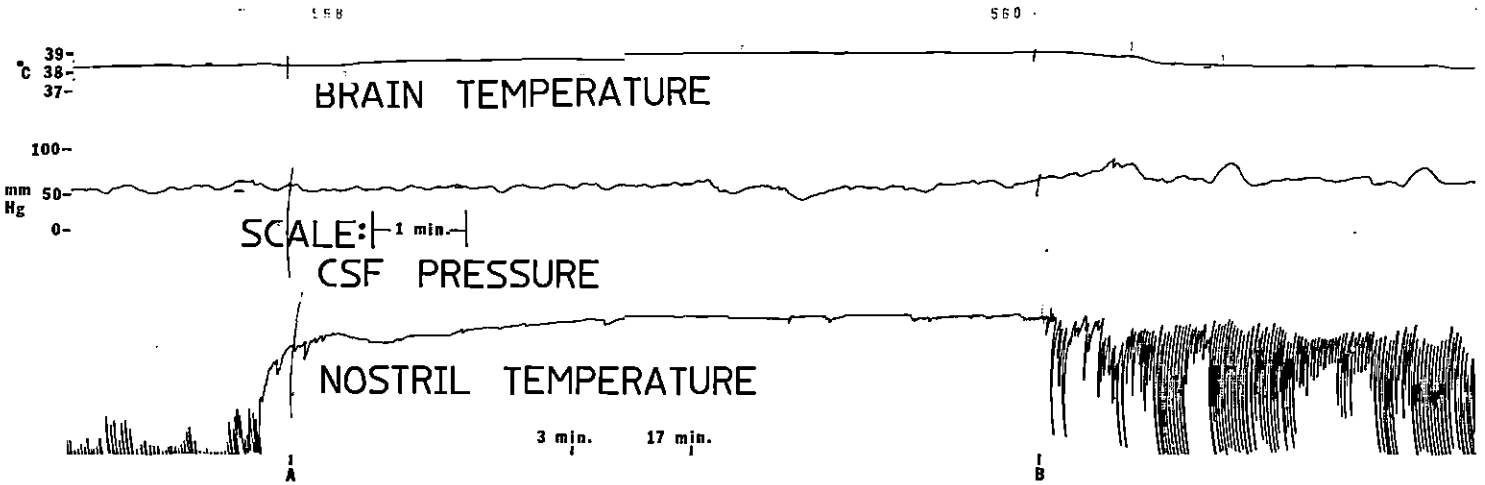

FIG. 2 *A REPRESENTATIVE PART OF A TRIAL MADE ON THE SHEEP.*

A = on bypass breathing
B = on normal breathing

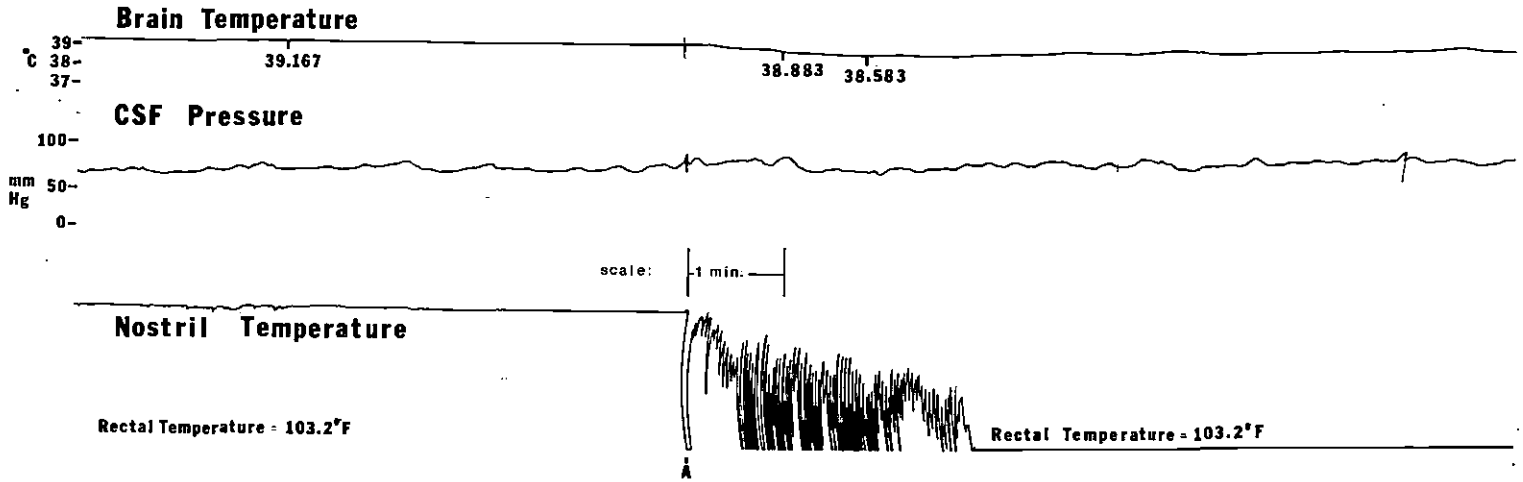

FIG. 3 *A REPRESENTATIVE PART OF A TRIAL MADE ON ONE SHEEP.*

A = on nasal breathing

Figure 4. Upper respiratory (tracheal) bypass canula (modified from Kluger and D'Alecy, 1975). Solid arrows denote bypass breathing with insert (A); broken arrows denote normal breathing with insert (B) (manufactured by the work shop of the Engineering Research Institute, Iowa State University, Ames, Iowa)

A Bypass insert

B Flow-through insert

C Cranial tubal extension

D Caudal tubal extension

E Body

F Cap

G Leur lock needle with cap

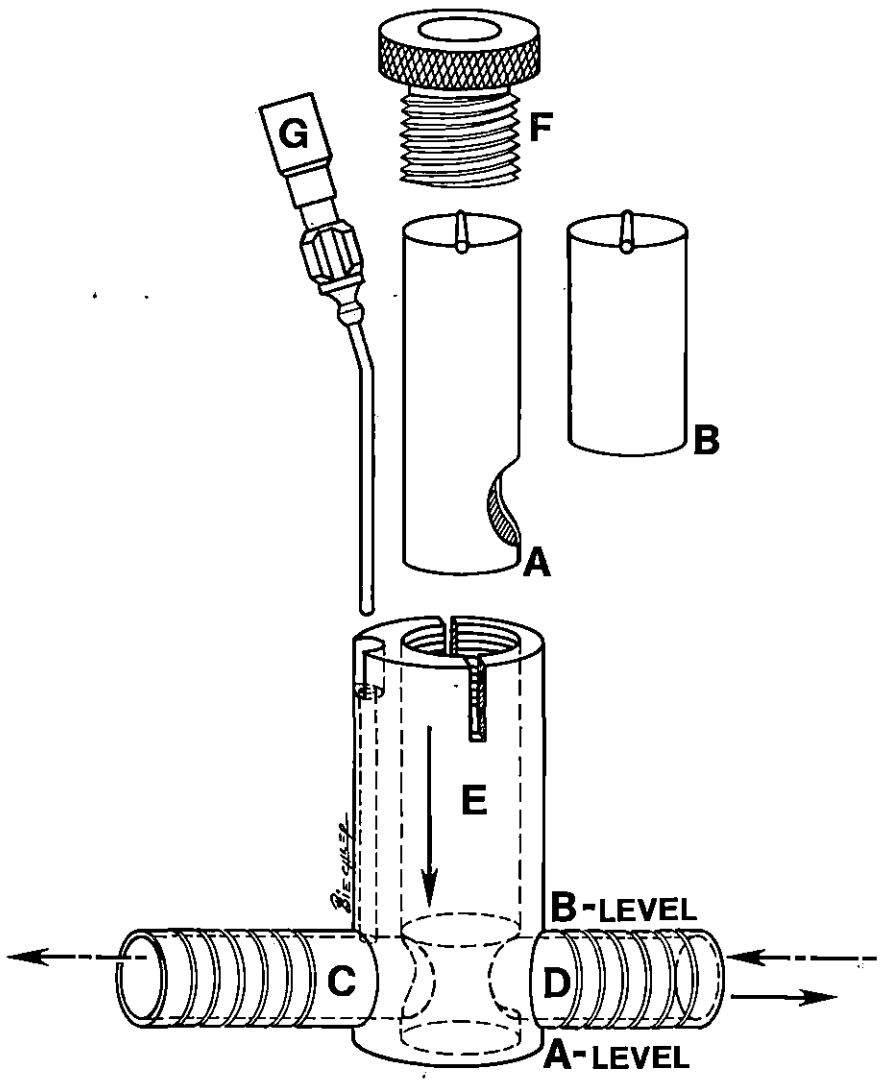

FIG. 4 **UPPER RESPIRATORY (TRACHEAL) BYPASS CANULA (MODIFIED FROM KLUGER AND D'ALECY, 1975). SOLID ARROWS DENOTE BYPASS BREATHING WITH INSERT (A); BROKEN ARROWS DENOTE NORMAL BREATHING WITH INSERT (B).**

A - BYPASS INSERT
B - FLOW-THROUGH INSERT
C - CRANIAL TUBAL EXTENSION
D - CAUDAL TUBAL EXTENSION
E - BODY
F - CAP
G - LEUR LOCK NEEDLE WITH CAP

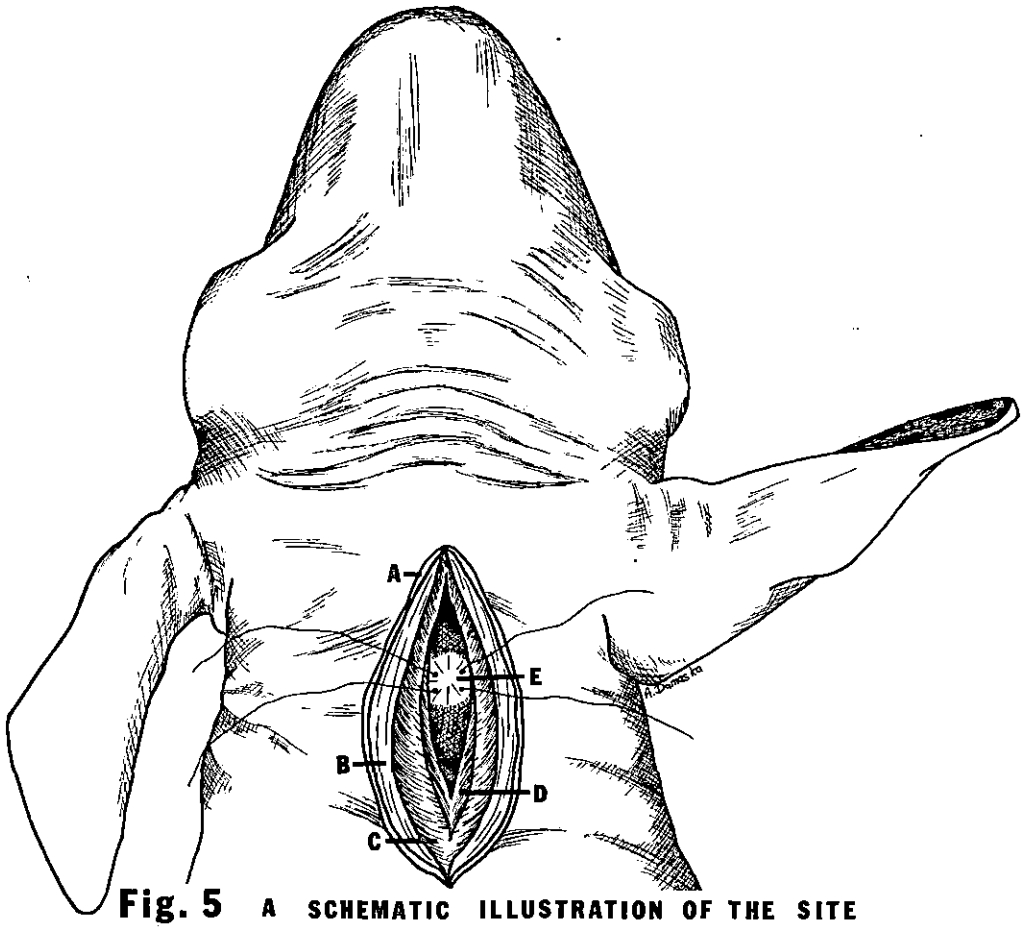

Fig. 5 **A SCHEMATIC ILLUSTRATION OF THE SITE FOR THE INSERTION OF THE CATHETER INTO THE CEREBELLOMEDULLARY CISTERN.**

A - Skin B - Nuchal Ligament

C - Rectus Capitis Dorsalis Major M.

D - Rectus Capitis Dorsalis Minor M.

E - Dorsal Atlantooccipital Membrane

Figure 6. A dissected sagittal section of the cranial cavity showing the location of a thermistor in relation to the cerebral arterial circle (right side = rostral; left side = caudal)

A	Thermistor
B	Caudal cerebral artery
C	Arterial branch to the rostral mesencephalic tectum
D	Rostral cerebellar artery
E	Caudal communicating artery

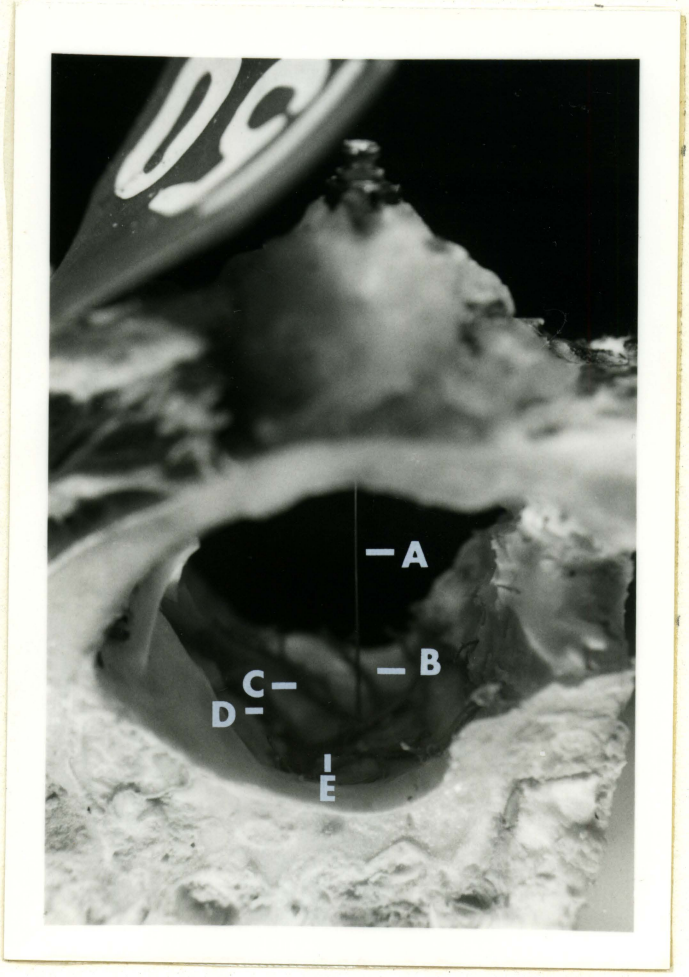
A
B
C
D
E

Figure 7. A sutured site around a cerebellomedullary catheter on the dorsal surface of the neck of a sheep