Concluding Remarks

Lightning studies are difficult to carry out because of the unpredictable nature of thunderstorms and the variety and complexity of measurements necessary to derive significant information. It is perhaps remarkable that the various independent measurements obtained in this study led to results that are entirely consistent with one another. For example, the location of the ground strike point was accurately determined by both thunder and magnetic measurements. The locations of the charged regions determined from independent sets of data were in good agreement with each other and with the channel geometry determined from thunder measurements. Cooperative work such as that reported here can provide much more insight about lightning discharges and their relationships to the thunderstorm environment than could have been obtained with only independent observations. The results described here represent the most comprehensive study ever made of a single lightning flash and its relation to the thunderstorm which produced it.

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Neural Organization and Evolution of Thermal Regulation in Mammals

Several hierarchically arranged integrating systems may have evolved to achieve precise thermoregulation.

E. Satinoff

Much of the work in the field of temperature regulation has been devoted to elucidating the nature of the central thermostat. According to the generally accepted model of thermoregulation, the temperature of the body is detected by thermosensitive neurons which bring autonomic and behavioral mechanisms into play to counteract any deviations from the optimal state (see Fig. 1A). Inputs from the skin, brain, and body are summed in a comparator which also receives input from an intrinsic reference signal, the set point. The output of this comparator, or integrator (the two terms will be used interchangeably), is a single error signal which serves as an input to the controllers of the appropriate thermoregulatory responses, both autonomic and behavioral (*1*). The alteration in body temperature resulting from the performance of these responses is fed back to the comparator which, in turn, adjusts the error signal. Because the same signal activates all thermoregulatory responses the fact that some responses in an animal's thermal repertoire appear earlier

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than others would depend on the threshold of the particular response.

The thesis of the present article is that this model, essentially a single integrator with multiple inputs and outputs, is inadequate to account for several new findings. In its place, an essentially Jacksonian multiple integrator model will be proposed, in which sensing and integrative functions occur at many levels of the nervous system (Fig. 1C), with higher levels facilitating, inhibiting, and coordinating those below. Finally, the evolution of endothermy in mammals will be discussed with a view toward understanding why so apparently simple a function as temperature regulation should be represented at so many different levels of the nervous system.

Summary. This article proposes a modification of the currently accepted view of the central neural integration of body temperature. In place of a single integrator with multiple inputs and outputs, the new model includes as many integrators as there are thermoregulatory responses. Furthermore, these integrators are postulated to be represented at many levels of the nervous system, with each level facilitated or inhibited by levels above and below. The purpose of such a complicated arrangement is to achieve finer and finer control over body temperature. A consideration of how endothermy might have evolved, with originally nonthermally related responses gradually coming under thermal control, makes such a brain organization highly reasonable.

One Thermostat

A commonly held view of the central nervous control of temperature regulation holds that the integrator controlling thermoregulatory responses—the thermostat—is in the preoptic area of the hypothalamus (*2*). There is much evidence that supports this. The preoptic area contains a very high percentage of thermosensitive cells relative to other parts of the brain, and many of these cells change their firing rates in response to changes in the temperatures of the skin, body core, and other parts of the brain (*3*). Localized heating and cooling lead to appropriate autonomic and behavioral responses (*4*), and if lesions are made in the preoptic area, animals can no longer maintain normal body temperatures in warm or cool environments (*5*). This last effect is not caused by motor deficits. Hemingway (*6*) traced a major pathway for shivering that originated in the posterior hypothalamus, and there was some evidence from early work of Keller (*7*) and Bard (*8*), among others, that decerebrate preparations can shiver and pant and change vasomotor state appropriately in thermally extreme environments. However, all these responses were fragmentary and ineffective and the animals would die from hypo- or hyperthermia unless they were housed in incubators.

Given this model, the problem of understanding the central nervous control of temperature regulation becomes very straightforward. On one side we must find out where extrahypothalamic thermal inputs exist, and how much they contribute to informing the preoptic area of the state of the animal's body temperature. On the other side we must work out the controller equations for the individual effectors to see how they vary with the direction and magnitude of the error signal.

There is no question that this model successfully describes many facts about thermoregulation. Fever, in this framework, is a rise in the intrinsic reference input, or set point. This leads to a change in the error signal which then activates all effector responses, both autonomic and behavioral, in the direction of increasing heat production and decreasing heat loss. Local heating or cooling of the preoptic area artificially displaces hypothalamic temperature and again, activates all effector responses. Most important of all, the model adequately describes the normal sequence and intensity of thermoregulatory responses that appear in a thermally stressed animal by taking into account both differences in thresholds of activation and slopes of the various responses. However, in the past few years, there have been a number of experiments that require a reevaluation of the central nervous integration of temperature regulation (*9*). Here I suggest that the hypothalamus is not the sole integrator of body temperature. Rather, it is the most important among many in that it coordinates the activity of other integrating mechanisms at lower levels of the neuraxis. In its absence the other systems can still function, although not as efficiently.

Two Thermostats

At first it appeared that it would be sufficient to postulate two independent integrators, one for behavioral responses and one for autonomic responses (see Fig. 1B). The reason for this change came from experiments on behavioral thermoregulation in rats with preoptic lesions. Because thermal stimulation of the preoptic area elicits both autonomic and behavioral thermoregulatory re-

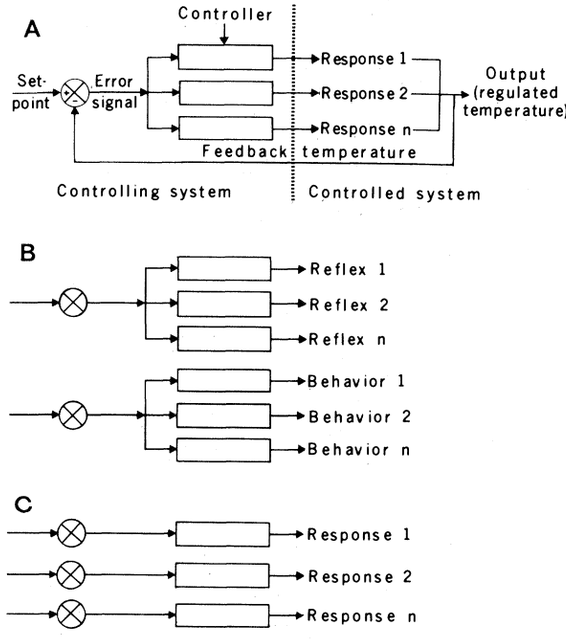

Fig. 1. Schematization of three possible control systems for thermoregulation. The comparators (circles) are mixing points. Whenever the combination of pluses and minuses do not cancel one another, an error signal is generated. When this occurs a response is activated which alters the regulated body temperature (whatever temperature or combination of temperatures that may be). The output of that response is fed back to the comparator and the error signal is adjusted. Feedback temperatures and the separation between controlling and controlled systems have been drawn only in the top model for clarity. (A) One central thermostat whose output activates all relevant behavioral and autonomic responses. (B) Two central thermostats, one activating all behavioral, the other all autonomic responses. (C) Each thermoregulatory response can be elicited independently of any other. This system may be multiply represented at several levels of the nervous system, and the individual integrators at lower levels would then receive input from higher levels (see text).

sponses (*4*), it was reasonable to assume that lesions of that area, which greatly damage autonomic responses, would also impair behavioral responses. Surprisingly, this was not the case. Rats with preoptic lesions placed in a hot environment used an operant response to turn the heat lamp off and a cooling fan on, and thereby avoided certain death from hyperthermia (*10*). In the cold, rats whose body temperatures otherwise dropped as much as 6.5°C in an hour were able to maintain their body temperatures for 2 hours within 0.75°C of normal by pressing a bar to turn a heat lamp on (*11*). These experiments indicate that there are sufficient thermosensitive cells and integrative neurons outside the preoptic area to enable rats to react to the discomfort of a cold environment by maintaining nearly normal body temperatures behaviorally even though their autonomic responses are either nonexistent or highly inadequate.

In a later study (*12*) it was demonstrated that lateral hypothalamic lesions in rats can leave thermoregulatory reflexive responses intact while specifically abolishing operant thermoregulatory responses. Some of the rats had very small lesions; they were able to maintain normal body temperatures in the cold, they ate and drank normally, and pressed a bar to avoid shock. Thus, whatever inputs are involved in maintaining motivated behavior in general were still operating in these animals. Yet none of them pressed the bar to get heat until many weeks had passed after the lesions had been made. Van Zoeren and Stricker (*13*) have recently replicated these findings in rats with preoptic or lateral hypothalamic lesions.

Adair (*14*) demonstrated a separation between behavioral and physiological thermoregulation in squirrel monkeys. Posterior hypothalamic thermal stimulation was as effective as preoptic stimulation in controlling operant thermoregulatory behavior, yet it had no effect on any autonomic responses.

Cabanac and his colleagues (*15*) reported a phenomenon in humans that is analogous to that seen in rats with lateral hypothalamic damage. They studied two subjects who were congenitally indifferent to pain. When the subjects were immersed in a hot bath they sweated, and when immersed in a cool bath they exhibited piloerection and shivering, and their core temperatures did not change any more than normal subjects did. This demonstrated that their autonomic responses to a changing core temperature were normal. However, these people reported no, or only very slight, feelings of discomfort toward any peripheral thermal stimulus, regardless of what their internal temperatures were. All normal subjects report that warm peripheral stimuli are very unpleasant when their core temperatures are high and cold stimuli are very unpleasant when their body temperatures are low. If the subjects who were indifferent to pain did not feel uncomfortable, we can assume that they would not have been motivated to thermoregulate behaviorally. These people, probably with deficits in peripheral pathways, are functionally very much like rats with lateral hypothalamic lesions that maintain their temperatures autonomically in the cold but do not use an operant to get heat.

In summary, it would appear that the nervous networks for behavioral and autonomic responses are functionally and neuroanatomically separate. The work both with laboratory animals and with humans leads to the conclusion that we must distinguish at least two independent systems (*16*).

Multiple Thermostats

However, two integrators, one controlling all autonomic responses, the other controlling all behavioral responses (Fig. 1B), are not adequate to describe the central nervous integration of body temperature. When several behavioral or several autonomic responses are examined simultaneously it becomes apparent that a further reevaluation is necessary.

For example, Roberts and Mooney (*17*) diathermically warmed various areas of the diencephalon and mesencephalon of rats and measured three responses in the rats' heat-loss repertoire—prone body extension, grooming, and locomotion. If there were a single integrator for all thermoregulatory behaviors, then if the rat's brain were heated locally, there should be a fixed order of activation of each response, depending on their individual thresholds. A normal rat in the heat first grooms, then becomes active, and finally lies quietly in a sprawled position with locomotor activity appearing intermittently as the animal remains in the heat (*18*). If the brain were heated locally, one would expect to see the same sequence of events: at low intensities of heating the rat should groom, as the intensity increased it should become active, and at higher intensities activity should alternate with prone body extension. However, this is not what Roberts and Mooney (*17*) found. They reported that 72 of the 74 electrode placements in various areas of the brain produced only one or two of the three heat-loss behaviors. Sprawled extension was elicited only from electrodes in the preoptic area, grooming only from more posterior placements in the posterior hypothalamus and ventral medulla, and they were never seen together. Locomotion was elicited in zones extending from the septal area through the midbrain to the medulla and was seen in combination with sprawling or grooming. Similar experiments in opossums demonstrated that warming of the medial preoptic area and anterior hypothalamus by means of radio frequencies elicited either grooming or sleeplike relaxation, depending upon electrode placement (*19*).

It must be emphasized that these results of diathermic warming cannot be attributed to stimulation of effector cells rather than thermoreceptive elements. When the rats were electrically stimulated through the same electrodes, they showed a variety of nonthermoregulatory responses, such as rolling, turning, and eye closure, that were never seen during warming. Furthermore, electrical stimulation in areas that were sensitive to diathermy never produced prone body extension or grooming. The same was true for the opossums; grooming was evoked only rarely, and a variety of other responses, such as sniffing, mouth opening, and various motor movements that were never seen during warming were regularly induced by electrical stimulation.

This work is puzzling if one assumes that thermodetectors all feed into one comparator for behavior, whose error signal then activates separate response components, as illustrated in Fig. 1B. According to this model, wherever the electrode placement, a range of stimulus intensities should yield the fixed normal sequence—groom, locomote, sprawl—because all inputs sum in the one integrator whose single output should then activate the response components according to their individual thresholds. Roberts and Mooney interpret their data as indicating multiple controllers with separate and independent channels from thermal detector to motor effector for each of these behaviors, and I agree with their interpretation. This model is represented by Fig. 1C. As will be discussed later, these parallel systems probably exist at every level of the neuraxis.

Lesion experiments also support a multiple integrator model. Of all the thermoregulatory responses, both autonomic and behavioral, available to rats (the most studied species), some are unaffected by hypothalamic lesions, whereas others are grossly disturbed. For ex-

ample, operant responding for warmth or coolness is left almost intact after preoptic lesions (*10, 11*), as are locomotion and grooming (*20*), two of the major behavioral responses to heat stress in rats (*21*). However, other behaviors are clearly impaired. In the cold, rats with preoptic lesions build very poor nests compared with controls (*13*), eat less food, and are less active than normal (*22, 23*). In the heat, prone body extension, or sprawling, is greatly reduced (*20*) and animals do not reduce their food intake nearly as much as do controls (*22*).

In studies of the autonomic cold-defense deficits in rats with preoptic lesions, it was found (*24*) that over the course of many months the animals gradually recovered the ability to maintain their body temperatures in the cold, but the three major responses measured—shivering, nonshivering thermogenesis, and vasoconstriction—appeared to recover independently. If these autonomic responses were organized in separate pathways, it could be argued that the differential recovery times were caused by more or less damage to those pathways under the electrode tip. In any case, it is not obvious how damage to a single integrative center could produce such results.

Again, it must be emphasized that the various thermoregulatory deficits in animals with preoptic hypothalamic lesions are not caused by damage to effector pathways rather than, or in addition to, integrative circuits. There is ample evidence that there is a disruption in activation, not execution, of motor behaviors in animals whose hypothalamus is completely disconnected from the rest of the brain (*25, 26*), and the same is true for most thermoregulatory effectors (*27*). Although the body temperatures of rats with such hypothalamic islands dropped more than 4°C upon a first exposure to an ambient temperature of 0°C, by the third exposure they were able to maintain their body temperatures in the cold nearly as well as controls (*28*). Cats decerebrated at the level of the lower pons or medulla could still exhibit shivering, piloerection and vasoconstriction in response to spinal cord cooling (*29*), and even spinal preparations can shiver below the level of the transection (*30*). These responses may be only fragmentary in response to peripheral cooling, and they do not occur as promptly as in normal animals even during spinal cooling. Nevertheless, these experiments, as well as others [for reviews, see (*9*)], conclusively demonstrate that animals without a hypothalamus can respond appropriately to thermal stresses.

Sensory Input to the Multiple Integrators

Signals from thermoreceptors do not influence the parallel integrating systems equally. From experiments on peripheral and localized central cooling in newborn guinea pigs, it appears that nonshivering thermogenesis is largely controlled by the temperatures of the skin and hypothalamus, whereas shivering is mainly influenced by the temperatures of the skin and cervical spinal cord (*31*). This may also be the case for adult rats (*32*). In other experiments on rats, grooming, tail vasodilation, and locomotion could be regularly elicited by differential heating of several superficial and deep peripheral sites, but prone body extension was never seen (*33*). These results, together with the fact that locomotor and grooming responses are not impaired after preoptic lesions, although pronation is (*20*), suggest that grooming, locomotion, and tail vasodilation can be elicited by either peripheral or central thermoreceptors, whereas prone body extension seems to depend mainly on input from central thermosensitive units.

Given the assumption of separate integrators, a reasonable question to ask is why all thermoregulatory responses are seen in sequence when a normal animal is in a thermally extreme environment. Part of the answer has just been discussed—the individual integrators are not affected equally by all thermosensitive inputs. A particular behavior like grooming, which depends to some extent on peripheral input, will appear before a behavior like sprawling which may require a high internal temperature before it is activated. Another part of the answer is that all the separate controllers for effector responses, even if they have identical inputs, may have different thresholds of activation. Heating, cooling, and ablating particular parts of the brain are very good methods of examining the central nervous system, but they are extreme and special situations. Usually when thermoregulatory responses are initiated the animal is in a hot or cold environment. When that is not the case, as with fever, the leukocytic pyrogen travels through the bloodstream and affects sensitive elements in the whole brain. In a normal animal in the cold, or with an infection, the progression from, say, vasoconstriction to huddling to piloerection to shivering, and the simultaneous occurrence of all these responses as the animal remains in the cold or remains febrile, can be explained by a single integrator and different thresholds of activation for each individual response. But the effects of lesions or localized heating or cooling do not fit such a model. This brings me to the major part of the answer to the question of how orderly thermoregulatory responses are obtained: the separate thermoregulatory systems are not independent of each other but are hierarchically controlled.

Hierarchical Control of the Parallel Systems

Thus far, the model of the thermoregulatory system has appeared to consist of independent parallel feedback circuits for each available thermoregulatory response. However, the parallel miniature thermoregulatory systems, each able to transduce thermal information into appropriate effector output, are not independent of each other. Rather, the activity of lower structures appears to be facilitated and inhibited by those above. Chambers *et al.* (*29*) demonstrated that in cats whose spinal cords were transected at the level of T-6, spinal cooling below the level of transection induced shivering and vasoconstriction of the hind limbs. (Whole-body cooling elicited only slight shivering in the hind limbs, possibly because the stimulus was not as effective, but, as would be expected, it elicited strong shivering in the forelimbs.) Decerebration at the level of the superior colliculus and rostral pons in these animals abolished thermoregulatory responses to whole-body cooling in the forelimbs, while still permitting shivering below the level of transection in response to spinal cord cooling. Most important, lowering the level of transection to the lower pons or medulla, again in the same animals, reinstated shivering, vasoconstriction, and piloerection in the forelimbs. These data show that there is a region in the midbrain and upper pons which tonically inhibits lower regions in the lower pons, medulla, and spinal cord. When the influence of the upper areas is abolished, the lower areas are themselves capable of organizing and facilitating thermoregulatory responses.

A similar organization can also account for the results of a striking experiment on thermoregulatory behaviors. In 1973, Carlisle and Ingram (*34*) examined the effects of thermally stimulating the spinal cord and hypothalamus on two thermoregulatory behaviors in pigs—operant responding for heat and postural changes. Cooling the spinal cord or preoptic area led to an increased rate of barpressing for heat, whereas heating the cord or preoptic area decreased responding. When the two temperatures were opposed, the operant response was de-

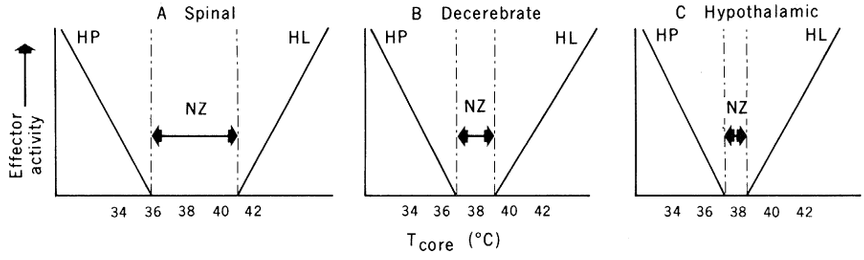

Fig. 2. The width of the thermoneutral zones (*NZ*) in spinal, decerebrate, and hypothalamic preparations. In intact animals there may be no neutral zone because either heat loss (*HL*) or heat production (*HP*) mechanisms may always be operating. The slopes of heat production and heat loss are drawn as equal for simplicity, but they probably are not; this is a matter for experimental determination. T_{core}, core temperature.

termined by the temperature of the hypothalamus, but the postural response was controlled more by spinal cord temperature. In a cool environment with a locally cooled spine the pigs spent 95 percent of the time in a cold-defense position with their legs retracted. With a cooled hypothalamus, they spent only about 50 percent of the time in the cold-defense position and, moreover, their legs were frequently extended. When the temperatures of the spinal cord and hypothalamus were made to go in opposite directions, the results were apparently paradoxical. A cooled hypothalamus increased the rate of working for heat, which implies that the pigs felt cold, but at the same time, if the spinal cord was warmed, the pigs lay down, not in a cold-defense position, which implies that the pigs did not feel cold. What kind of integration is going on where the operant response is determined by the temperature of the preoptic area, the postural response relates to the temperature of the spinal cord, and both responses can be in opposite directions at the same time? These results also imply a hierarchical control of the parallel individual circuits because in a normal animal in a hot or cold environment one would never see such opposing behaviors.

In these experiments, contrary to the normal situation, the spinal cord is acting independently of and, indeed, antagonistically to the preoptic area. In effect, local thermal stimulation in each area has overriden the integration that normally exists between them. Thus, intense local thermal stimuli appear to act not only as inputs into a given system, but also as transections, in the sense that they disrupt hierarchical coordinating pathways that would ordinarily integrate both spinal and forebrain mechanisms. The effect of a transection was beautifully stated by John Hughlings Jackson (*35*) about 80 years ago. "Roughly we say that there is a gradual 'adding on' of the more and more special, a continual adding on of new organizations. But this adding on is at the same time a 'keeping down.' The higher nervous arrangements evolved out of the lower keep down those lower, just as a government evolved out of a nation controls as well as directs the nation. If this be the process of evolution, then the reverse process of dissolution is not only a 'taking off' of the higher, but is at the very same time a 'letting go' of the lower. If the governing body of this country were destroyed suddenly, we should have two causes for lamentation: (1) The loss of the services of eminent men; and (2) the anarchy of the now uncontrolled people."

Thermoneutral Zones and the Concept of Set Point

In Jacksonian terms of levels of function, the hypothalamus is simply at the top of a thermoregulatory hierarchy. So why has it been thought of for so long as the only thermostat in the brain? To answer this question let us reconsider the concept of set point. As mentioned earlier, decerebrate and spinal preparations have no adequate temperature regulation—the animals die in the cold or heat, presumably for lack of a set point, a function governed by the hypothalamus. However, as many investigators have demonstrated (*7, 8, 29*), if such animals are cooled they shiver and show vasoconstriction. If they are heated they pant and sweat and exhibit vasodilation. In adults these responses are fragmentary and not very effective. But what does that mean? Considering Bard's work (*8*) on pontile and low mesencephalic cats, it means that they begin to exhibit shivering and piloerection at temperatures colder than those that activate shivering in normal animals, and that they begin to pant and exhibit vasodilation at hotter temperatures than normals do. This is especially clear in Keller's dogs in whom the entire hypothalamus was removed (*7*). Such animals exhibited a permanently raised heat-dissipation threshold. That is, they did not begin panting in the heat until core temperatures were raised 2.5° to 3.0°C above the normal panting threshold. In other words, such animals have a much wider neutral zone, or indifferent range, than normals, but they do defend their body temperature, however ineffectively, at both ends of their neutral zone. That defense of a neutral zone is the set point, only it is not as narrow as we usually think of it when we use the term.

In recent work Bignall and Schramm (*36*) have described temperature regulation in decerebrate kittens. These kittens maintain their body temperatures, both autonomically and behaviorally, much better than animals decerebrated when adult. This work implies that we will have to reconsider all our concepts of what the lower levels of the nervous system can do when these concepts are drawn from experiments on adults. Our present estimates of what subhypothalamic tissue is capable of may be drastically inadequate.

Even the simplest levels of the nervous system can be considered to have set points. The body temperature limits within which no thermoregulatory responses are activated—the neutral zone—are wider, and we are accustomed to thinking of a set point as a very narrowly regulated range as it is in the intact adult. But there is nothing inherent in the definition of set point that dictates the width of the neutral zone—the term is just as valid to use from the points of the upper and lower limits of the defended range. Figure 2 summarizes this idea. The leftmost graph is Bligh's (*37*) representation of the neutral zone in a spinal preparation—about 4° to 5°C. The next two graphs are hypothetical pictures of the neutral zones in animals in which all neural tissue above the midbrain or hypothalamus has been removed. (Presumably the neutral zone would be narrower or even nonexistent in an intact adult.) The difference between the preparations is in the width of the neutral zones. The hierarchical organization of the parallel integrative systems, which yields a progressive narrowing of the neutral zone, can thus give the illusion of a single integrator for a single narrow set point.

What, then, is the function of the hypothalamus in temperature regulation? Within the framework outlined above, it serves to coordinate and adjust the activity of thermoregulatory systems located at several lower levels of the neuraxis. Selective facilitation and inhibition from the hypothalamus to lower levels would ensure that appropriate thermoregulatory reflexes are activated and in-

appropriate ones suppressed when thermoregulation is the paramount consideration. Facilitatory influences from the hypothalamus would also ensure that thermoregulatory responses are initiated promptly in the presence of an appropriate stimulus. In this sense the hypothalamus would play a similar role in thermoregulation to the one it plays in aggression. Ellison and Flynn (*26*) reported that cats in whom the hypothalamus had been disconnected from the rest of the brain were still capable of showing well-organized aggressive responses. However, these responses were elicitable only in the presence of very strong stimuli; the rest of the time the animals sat immobile and showed no spontaneous behavior. Translating these results into thermoregulatory terms, it means that, in the absence of the hypothalamus, thermoregulatory responses would not be initiated except in the presence of very strong (that is, very hot or cold) stimuli [see (*7*)].

The Evolution of Endothermy in Mammals

A major problem remaining is to consider why the brain should be organized in such a fashion; why should there be many independent integrators arranged in parallel at every level of the nervous system, each level facilitated or inhibited by the levels above and below, controlling every conceivable thermoregulatory response? It seems like an unnecessarily complicated way to design a brain. However, if we consider how endothermy probably evolved, it becomes very reasonable.

There are three relevant points. First of all, there are many changes needed to evolve from an ectotherm to a well-regulating endotherm—from chemical thermogenesis and shivering, to panting and sweating, to thermal control of peripheral circulation, to the development of fur, feathers, or fat (*38*). Second, evolution takes a great deal of time. Each one of these mechanisms took millions of years to become efficient for thermoregulatory purposes. Third, by no stretch of the imagination could all of them have developed concurrently. No animal has all of them and some animals have developed one mode of regulation to a much greater degree than other modes. Heath (*39*) gives three examples of endothermic extremes: the arctic fox, an insulation specialist; the shrew, a metabolic specialist; and the elephant, a surface-to-volume specialist; all other mammals fall somewhere within these extremes. Now most, if not all, thermoregulatory reflexes evolved out of systems that were originally used for other purposes. To give just two examples of this, Cowles (*40*) has argued that the peripheral vasomotor system, the basic mechanism for changing blood flow at the surface, first served as a supplemental respiratory organ in amphibia. It then became a heat collector and disperser in reptiles (regulating the flow of heat from outside the body to inside) and finally an essential temperature regulatory mechanism for endotherms (regulating heat flow from inside the body to outside). Heath (*39*) has argued that the change in posture from the sprawling stance of a reptile to the limb-supported posture of the therapsids, the mammal-like reptiles, and subsequently the mammals, and the consequent changes in muscular organization and muscle tension provided the basis for a high internal heat production. This illustrates the principle of evolutionary coadaptation: a mechanism evolved for one purpose has as a side benefit an adaptive value in an entirely different system.

So we start with an organism that senses temperature and has some form of thermoregulatory behavior. That is one integrating system. If later on it develops another form of thermoregulatory behavior, there is another integrating system in parallel with the first. In changing its posture it accidentally develops a system for producing heat. Eventually the temperature sensors gain control over this new form of heat production and that is yet another integrating system. At some point it becomes advantageous to lose some of that heat more quickly. The animal already breathes and for that purpose has a good vasomotor system, so changes in peripheral blood flow and respiratory rate simply need to come under the influence of thermal detectors. When they do, however long it takes, we have two more integrating systems. And so on. The same principle of new controls over an already existing mechanism for a new function can be used to understand the nervous organization of many forms of motivated behavior (*41*).

It would be unnecessarily burdensome to require the evolutionary process to create new systems to solve a problem already solved by an existing system. If a function is already perfectly well handled by the midbrain, and another by the spinal cord, there would be no point in transferring all of these separate integrations to the preoptic area of the hypothalamus. But if these functions are not perfectly well handled at lower levels—if, for instance, a narrower set point conferred a selective advantage on the organism that had it—then we would be on the way toward a hierarchical organization of thermoregulatory responses for the purpose of achieving finer and finer tuning of the thermoneutral zone.

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Nuclear Waste: Increasing Scale and Sociopolitical Impacts

The shorter-term impacts of large-scale nuclear waste operations should be examined.

Todd R. La Porte

Summary. The article argues that insufficient attention has been paid to the operational aspects of the U.S. radioactive waste management system when it grows to the scale necessary to handle wastes produced by a fully deployed plutonium economy. Without such information, many of the unsettling speculations which have become part of the public debate and are summarized herein cannot be clearly addressed. The article then outlines the types of information necessary to begin estimating the costs and consequences of radioactive waste management. Finally, an index of social exposure to radioactive hazard is proposed to improve the basis for policy decisions in this area.

Planning for radioactive waste management has been criticized primarily because estimates of potential environmental damage have been inadequate (*1*) and, secondarily, because the impact of radioactive waste management systems on social and political development has been ignored (*2*). Here I address the latter consideration and begin by noting several speculations that have colored the public debate about the social and political consequences of radioactive waste management. Implicit in each speculation is the expectation of a large-scale waste management system. I then propose some first steps toward providing a firmer basis for estimating social, economic, and political, as well as environmental, impacts of managing increasingly large volumes of radioactive wastes. The argument calls, first, for developing reasonably detailed descriptions of each of the waste management systems that might be used and, second, for estimating the organizational requirements for each of these systems when they are faced with increasingly large volumes of waste. In addition I suggest that for each potential waste management and organizational system, estimates be made of the chances that human beings would be exposed to radioactive hazards.

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Speculations on Sociopolitical Consequences

Most frequently, questions about the management of radioactive wastes are associated with what might be called the "1000-year problem." That is, how can we develop highly reliable, socially acceptable technical systems that are so effective that they will nearly eliminate for at least 1000 years the long-term risks to those generations who will not benefit from the processes that produced the risk (*3*). These are, of course, very important problems, but there are a number of equally important, more immediate matters. These are, in a sense, the "10-year problems"—those associated with the handling of spent fuel, especially if reprocessing is involved, during the 10 years or so before wastes can be safely stowed away in permanent repositories—and they involve the shorter-term social, economic, and political consequences likely to result from actually developing the organizational systems required as the scale of radioactive waste production and management greatly increases.

Discussions about the shorter-term consequences of radioactive waste management have stimulated some unsettling speculations about the various effects on social, economic, and political aspects of life were a "plutonium economy" or any large nuclear economy actually to be developed (*4*, *5*). A notable characteristic

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