uncorrelated data. This miniscule portion of the brain has been deluged with responsibility for a large number of apparently unrelated functions (*1*); it has been considered the head-ganglion of the autonomic nervous system, responsible for the regulation of body temperature, blood pressure, respiration, appetite, the diurnal rhythm of sleep and wakefulness, the sexual cycle, and the control of the metabolism of sugar, fat, and water. On the basis of the approach presented above, these functions may be considered as parts of an integrative mechanism. Thus, it is apparent that as the neo-amphibian leaves the water and is exposed to large changes in body temperature, immense variations may occur in the metabolic rates of the numerous tissue and organ systems which might lead to maladjustments of the internal economy. An internal environment optimal for 5° C might be wholly unsuitable for survival at 30° C. Since temperature variations of such magnitude may occur rapidly in terrestrial poikilotherms under natural conditions, the development of a coordination center for metabolic adjustment would provide survival value for its possessor. In such poikilotherms this center would be charged, not with the maintenance of the constancy of the internal environment (homeostasis) but with the coordination of changes in the internal environment (homeodynamics) to meet the demands placed by other changes such as temperature. Such a rapidly reacting center for homeodynamic control appears to have been developed in the hypothalamus. The rich vascularity of this organ makes it eminently suitable for such a role.

Our studies suggest that some homeodynamic regulations depend upon the independent variable, the temperature of the temperature-sensitive center in the brain. In the course of time, heat conservation, thermogenic and thermolytic mechanisms apparently were laid down proximal to the homeodynamic center, introducing a new factor: the relative constancy of body temperature. Under these conditions, the homeodynamic adjustments became minimal during the diurnal cycle, although the adjusting mechanisms persisted. Patterns of behavior of warm-blooded animals suggest retentions of some elements of the diurnal cycle of the poikilotherms, such as the diurnal variations in body temperatures and the period of torpor (sleep). Phenological functions, such as the reproductive cycle, which previously depended in part upon the diurnal variations and their annual precession, may escape and establish a reproductive calendar independent of the solar year.

Although begun as an analysis of a relationship between body temperatures and blood pressure, our studies have not only indicated that the relationship depends on the central nervous system, but have suggested that it may be but a portion of a more general integration. Some of these integrating mechanisms may be approached experimentally in the poikilotherm by producing variations in body temperature. In homeotherms, the superimposition of a relatively constant body temperature serves to mask the basic integrative mechanisms, but induced hypothermia causes the animal to revert to a more primitive condition and thus exposes the homeodynamic apparatus to experimental analysis.

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Indole-3-Acetic Acid and Flowering

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The experiments described briefly in this paper were conducted to test the assumption by Thurlow and Bonner (*2*) that, since a marked decrease in the auxin content of plants at the time of their flowering has been demonstrated (*1*), it might be possible to delay flowering by externally supplying plants with auxin. Thurlow and Bonner sprayed soybean and *Xanthium* plants, grown under flower-inducing short photoperiods, with indole acetic and naphthalene acetic acid solutions (500 ppm) and observed inhibition of production of flower primordia, a result which lends support to Bonner's assumption. Since the leaves of the treated plants showed pronounced epinasty and other growth deformities and since the inhibitory effect of the spray might thus be an indirect leaf-injury effect, it was thought desirable to treat plants with indole acetic acid by immersing their roots or the cut ends of their stems in water solutions of this auxin in order to avoid direct auxin contact with leaves.

In the first set of experiments the roots of petunia plants (var. Topaz Queen) which were 7 weeks old and which were approaching flowering time, though still without flower primordia, were immersed for 24 hrs in water solutions of indole-3-acetic acid (200 ppm). The plants were then potted in rich loam soil. The first visible flower buds appeared in these treated plants 23 days after the first flower buds were visible on the control plants, roots of which were treated for 24 hrs with water. Thus, the indole acetic acid treatment distinctly delayed flowering, a result which agrees with that of Thurlow and Bonner. No signs of leaf epinasty or other types of deformity appeared in the leaves of the treated plants, nor was there any apparent difference in root development between treated plants and controls.

This experiment was repeated on Lincoln soybeans,

with a similar result. The treated plants produced their first flower buds 12 days after the controls.

A second set of experiments was devised to determine whether or not indole acetic acid solutions might inhibit the development of already-formed flower buds into fully opened flowers. Young inflorescences bearing flower buds in various stages of development but no open flowers were cut from plants of stocks, snapdragons (Rose Queen), annual larkspur (Blue Bell), blue salvia, and iris (Sierra Blue), and were immediately placed with their cut ends in water solutions of indole acetic acid (25, 50, 100, 150 ppm) for 24 hrs; following this treatment, the inflorescences were placed in containers with the cut ends in tap water. On successive days, counts were made of the numbers of buds which developed into fully opened flowers. The results indicate that, in all treated plants, the indole acetic treatment retarded the development of flower buds into open flowers, as compared with that of untreated controls. In iris, the growth of flower buds was completely inhibited; all buds of the controls opened. The retarding effect of the auxin solutions upon bud development was more pronounced at the higher concentrations used; also, at the higher concentrations, some signs of leaf epinasty and of abnormal stem twisting were observed. These growth abnormalties were less pronounced than those of leaves directly treated with auxin sprays.

Although these experiments differed somewhat from those of Thurlow and Bonner, the results are similar to theirs. It cannot be assumed that the results of such experiments demonstrate a causal relationship between auxin content of a plant and its flowering, but these preliminary tests may open a way to more precise approaches to the suggested relationship.

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Observation on the Mechanism of Action of Dicoumarol

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The mechanism of the action of dicoumarol is obscure, and many clinicians hesitate to use it for this reason. Quick (*5*) presents evidence that the prothrombin time, as measured by the one-stage method, may be prolonged by diminution of prothrombin component A, component B, or a labile factor. According to Quick, component B is reduced in dicoumarol administration.

Loomis and Seegers (*2*) consider prothrombin to be a unitary principle and receive support from Munro and Munro (*3*), who have demonstrated that A and B cannot be regarded as separate components.

Accepting the hypothesis of a unitary principle, the clotting time has been studied when dicoumarol plasma is mixed with whole normal plasma, Seitz-filtered plasma, defibrinated plasma, and serum.

Experiment 1—Mixture of Dicoumarol Plasma and Normal Plasma

Dicoumarol plasma (14% prothrombin)		Dilution of normal plasma				
Mixture		80%	50%	25%	12.5%	6.25%
Prothrombin percentage	Observed %	50	42	38	34	22
	Arithmetic avg. %	47	32	19	13	10

Experiment 2—Dicoumarol Plasma and Seitz-filtered Normal Plasma

Dicoumarol plasma (5% prothrombin)		Dilution of Seitz-filtered plasma (free from prothrombin)				
Mixture		80%	50%	25%	12.5%	6.25%
Prothrombin percentage	Observed %	3	3	3	2	3
	Arithmetic avg. %	3	3	3	3	3

Experiment 3—Dicoumarol Plasma and Defibrinated Normal Plasma

Dicoumarol plasma (25% prothrombin)		Dilution of defibrinated plasma* (100% prothrombin)			
Mixture		50%	25%	12.5%	6.25%
Prothrombin percentage	Observed %	30	24	20	17
	Arithmetic avg. %	37	25	18	15

* Plasma was defibrinated by the addition of 1/10 volume of Parke Davis & Co. Thrombin Topical: One ampoule was made up to 200 ml with 0.15 N sodium chloride. The excess thrombin was inactivated by keeping the plasma at 37° C for 1 hr.

Experiment 4—Dicoumarol Plasma and Normal Serum

Dicoumarol plasma (24% prothrombin)		Dilution of 24-hr serum* (trace of prothrombin)			
Mixture		50%	25%	12.5%	6.25%
Prothrombin percentage	Observed %	44	38	32	24
	Arithmetic avg. %	12	12	12	12

* The serum was allowed to stand for 24 hrs. It contained a trace of prothrombin by the one-stage method, but no thrombin.

Dilutions of normal were made with 0.9% sodium chloride; 0.1 ml of dicoumarol plasma and 0.1 ml of diluted normal plasma were added to 0.2 ml of thromboplastin (Difco) and the mixture activated by 0.2 ml of 0.025 M calcium chloride solution. The clotting time was measured at 37° C and the corresponding prothrombin percentage read from a dilution curve. This value was compared with the arithmetic average of the prothrombin concentrations of the dicoumarol plasma and the normal plasma, measured separately.