sequent leukocytes, or (ii) damage in the G_1 period to long-lived lymphocytes, the damage not being observed as chromosomal abnormalities until mitosis. The latter may be the more likely hypothesis.

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Swimming Sea Anemones of Puget Sound: Swimming of Actinostola New Species in Response to Stomphia coccinea

Abstract. *Swimming as a response of the sea anemone* Actinostola *new species can be elicited as a result of contact with the submarginal surface of another swimming sea anemone* Stomphia coccinea. *However,* Stomphia *does not swim as a result of contact with* Actinostola. *In all other known respects, swimming is caused in both species by the same stimuli, including certain starfishes, a nudibranch, and electrical stimuli. No agent that causes* Actinostola *to swim has been detected in extracts, rubbings, or dried matter from* Stomphia.

Since the observation that the sea anemone *Stomphia coccinea* swims in response to certain starfishes (*1*), a number of other circumstances that cause swimming have been discovered and investigated. These are (i) electrical stimuli of the appropriate strength, number, and frequency (*1, 2*), (ii) extracts from the aboral surface of one of the active starfish, *Dermasterias imbricata* (*3*), and (iii) contact with a nudibranch *Aeolidia papillosa* (*4*).

The swimming sea anemones of Puget Sound were first regarded as members of a single species, *Stomphia coccinea.* However, it was noted that two groups exist, differing from each other in size and color, and investigators and collectors referred to these groups as "large" and "small" *Stomphia.* The latter corresponded with published descriptions of *S. coccinea* (*4, 5*). The former has now been identified as a new species of *Actinostola* (*6*). Besides differences in appearance and morphology, there are certain differences between the swimming movements of the two forms (*6, 7*). Both anemones belong to the large family Actinostolidae.

We have now discovered that the bigger swimming sea anemone, *Actinostola,* swims in response to contact with the smaller *Stomphia. Actinostola* has no such effect on *Stomphia,* nor does one *Stomphia* cause another *Stomphia* to swim. This response was obtained in more than 50 percent of matched trials (Table 1), though it seemed to be a more frequent occurrence in some animals than in others. In some respects, swimming of *Actinostola* is more easily evoked by *Stomphia* than by *Dermasterias* or *Aeolidia.* Thus *Stomphia* brought into contact with a single tentacle of *Actinostola* will frequently cause the latter to swim, but this rarely happens when one of the active starfishes is brought into contact with a single tentacle of *Actinostola.*

The surface just below the margin of the oral disc of *Stomphia* is most effective in causing *Actinostola* to swim. When ten animals were tested against the tentacles, the lower column, and the submarginal region of *Stomphia,* none swam in response to the tentacles, one swam on contact with the lower column, but nine of the ten *Actinostola* swam in response to contact with the submarginal region. Figure 1 shows a typical response under the most effective circumstances, namely when tentacles of *Actinostola* are in contact with the epidermis immediately above the marginal sphincter of a partly closed *Stomphia.*

Some features of the response have emerged from attempts to detect a chemical substance in *Stomphia* that causes swimming in *Actinostola.* Rubbing the active area of *Stomphia* with absorbent objects (such as pipe cleaners or swabs) does not pick up active materials as in the case of *Dermasterias* or *Aeolidia.* Sea-water and alcohol extracts and freeze-dried powders of *Stomphia* do not elicit swimming of *Actinostola* as corresponding preparations of *Dermasterias* and *Aeolidia* (*3, 4*) do. Nevertheless, since the two animals must be brought into contact if swimming is to occur, it is reasonable to suppose that *Actinostola* swims in response to some substance in *Stomphia* to which it is highly sensitive. The failure to detect activity in extracts and rubbings would suggest that this substance is present only in small quantities in the animal as a whole, and that perhaps it can be delivered locally at high concentrations when the two animals are brought into contact in the appropriate way.

The most obvious functional explanations for the swimming responses to certain starfishes, for example, as escape reactions, are not consistent with certain facts (*2, 8*). It is even more

Table 1. Matched trials of ten single *Stomphia* tested against ten *Actinostola* (*Actinostola* closed on eight occasions and not available for testing).

Swim to *Stomphia*	Responses of individual *Actinostola*									
	1	2	3	4	5	6	7	8	9	10
Positive	10	8	0	8	8	7	8	1	3	0
Negative	0	1	8	2	2	3	2	7	6	8

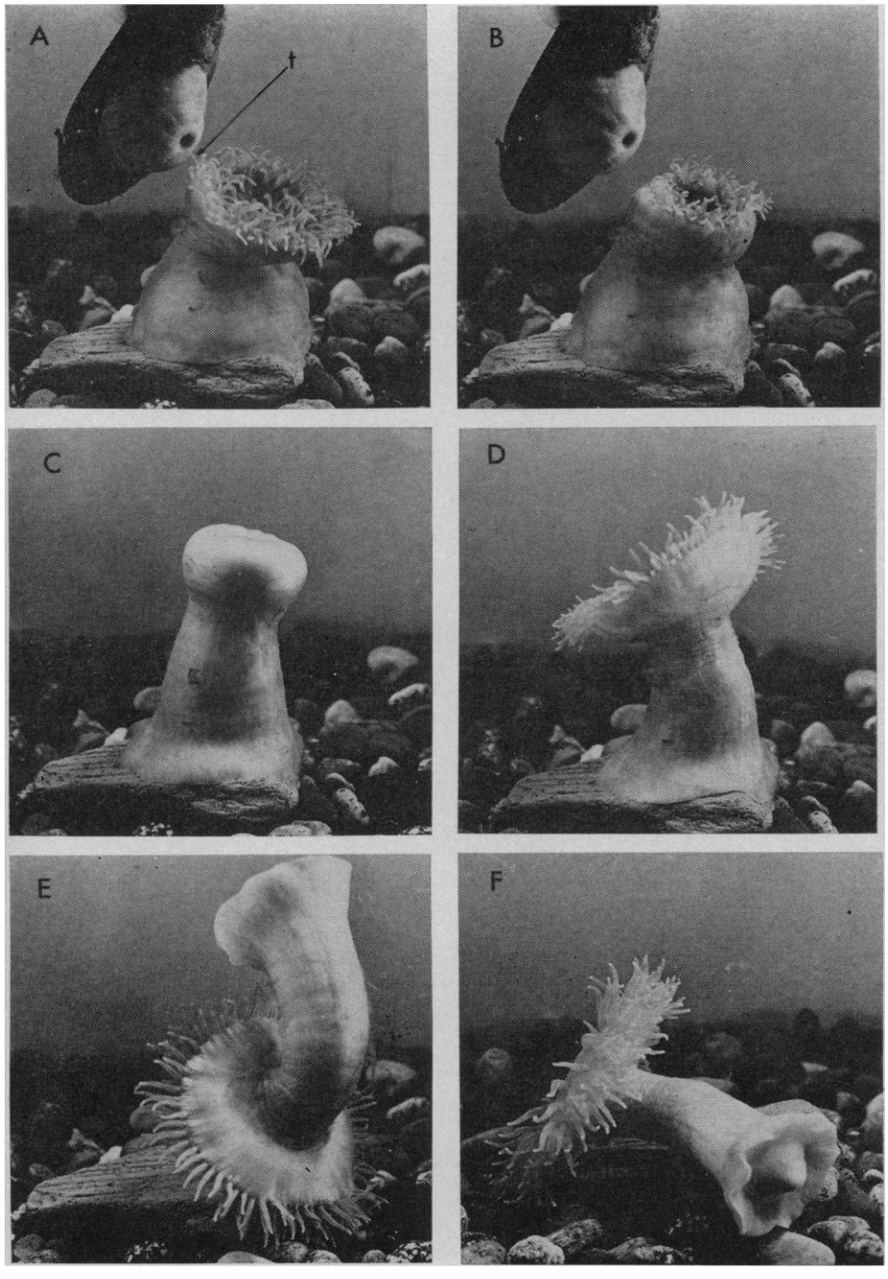

Fig. 1. Swimming response of *Actinostola* new species on contact with *Stomphia coccinea*. (A) Single tentacle (*t*) of *Actinostola* in contact with surface above the sphincter in semiclosed *Stomphia* (on *Modiolus* shell). (B) *Actinostola* closing [1 second after (A)]. (C) *Actinostola* extending after closure [3 seconds after (B)]. (D) *Actinostola* reopened, bending and whirling prior to detachment [lifting of base beginning to be visible; 3 seconds after (C)]. (E) Detached *Actinostola* showing typical swimming flexion; anemone lifted above substratum by these movements [5 seconds after (D)]. (F) *Actinostola* at end of swimming sequence, showing typically extended column and conical protuberance from base [22 seconds after (E)].

difficult to see a functional role for the swimming of *Actinostola* in response to *Stomphia*. The two anemones are rarely collected in the same hauls and individuals of both species are widely scattered in their natural habitats in the deeper waters where they occur (usually below 100 meters). The most acceptable explanation at the moment is that this behavior pattern is due to the fortuitous existence in *S. coccinea* of some substance closely resembling a substance in some other organism to which *Actinostola* has developed an escape reaction.

Before this observation was made, the swimming responses of the two anemones were thought to occur under identical circumstances, namely upon contact with the starfishes *Dermasterias imbricata*, *Hippasteria spinosa*, and *H. phrygiana* (*4*); upon contact with the nudibranch *Aeolidia papillosa*; and in response to electrical stimuli of high intensity (*2*). Evidently the swimming response of *Actinostola* differs in at least one respect, since it can be evoked by some agent occurring in *Stomphia*. Moreover, the more varied the circumstances that cause the swimming response, the more reasonable it is to suppose that this behavior pattern serves a variety of important functions in these anemones in their natural habitats.

The neuromuscular mechanisms that release the base and that perform the swimming flexions are very complex; these are perhaps the most highly coordinated fast activities so far described in actinians. It would be surprising if this behavior pattern did not have very important functions in the lives of these animals. We may have to wait for satisfactory explanations of these behavior patterns until more is known about the behavioral and ecological relationships of some common animals in deeper waters.

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Circadian Pattern of Plasma 17-Hydroxycorticosteroid: Alteration by Anticholinergic Agents

Abstract. *Atropine, administered to cats just prior to the time of the expected circadian rise in levels of 17-hydroxycorticosteroid in plasma, blocks this rise. Atropine does not alter this circadian pattern when administered at other times in the circadian cycle. Results similar to those obtained with atropine have been observed with short-acting barbiturates. Dibenzyline administered just prior to the time of the expected circadian rise is ineffective in blocking this rise. These findings support the hypothesis that the circadian pattern of plasma 17-hydroxycorticosteroid levels reflects activation, by the central nervous system, of the hypothalamic-pituitary-adrenal axis during a "critical time period" in the circadian cycle.*

The occurrence of a circadian pattern in levels of plasma 17-hydroxycorticosteroids (17-OHCS) and in other parameters of adrenal cortical function has been well documented in man (*1*), monkeys (*2*), rats (*3, 4*), and mice (*5*). The constancy of this pattern in man during illness (*6, 7*), night work (*1*), and total bed rest (*8*), as well as the difficulty of altering this pattern with change in activity-sleep routine (*9*), have been commented upon. Alterations in this pattern have been reported in patients with chronic, diffuse, central nervous system disease accompanied by impairment of consciousness and by delirium or restlessness (*2*). Alterations in this pattern have also been reported in our studies (*10*) which described such abnormalities in patients with central nervous system disease localized in the temporal lobe or in pretectal or hypothalamic areas. These observations suggest that the central nervous system plays a dominant role in determining this daily variation in plasma 17-OHCS levels.

The phenomenon of sleep, and its accompanying rapid-eye-movement periods (*11*), suggest circadian function of the nervous system (*12*), as does the circadian variation of the human electroencephalogram (*13*). Central nervous system regulation of pituitary adrenocorticotropin (ACTH) secretion is well established (*14*). Integrity of some portion of the hypothalamus seems to be a necessary factor in this regard (*10, 15*). There is preliminary evidence from the mouse (*5*) and from the rat (*3*) of circadian variation in the hypothalamic content of pituitary corticotrophic stimulatory factor.

The nature of the mechanisms initiating the release or elaboration, or both, of hypothalamic releasing factors has still to be elucidated. Some workers (*16*) have introduced the concept of a "variable set point" component in the central nervous system. It is possible that this set point, in addition to being "reset" by steroid levels, may also be reset by a variety of changes in the internal or external milieu acting on the central nervous system, as well as by the state of synaptic activity at the hypothalamic secretory cell involved. We have demonstrated (*17*) that implantation of minute amounts of cholinergic and adrenergic agents in certain areas of the hypothalamus is capable of eliciting an abrupt, prompt rise in plasma 17-OHCS levels in the cat. Similar results have also been reported by Endroczi (*18*).

Our hypothesis in the present investigation is that the observed circadian pattern in levels of plasma 17-OHCS reflects the release of corticotrophic-releasing factor, and consequently ACTH, at one *critical period* in the 24-hour cycle, immediately preceding the period when levels of plasma 17-OHCS begin their circadian rise. If we accept the findings that activity of the nervous system is circadian, and that pituitary adrenal activation can be effected by synaptically active agents, then it should be possible to abolish the circadian rise in plasma 17-OHCS levels by administering, just prior to the postulated "critical period," drugs which can block synaptic transmission and thereby block release of corticotrophic-releasing factor.

This concept is analogous to the "critical period" described for release of luteinizing hormone in the rat (*19*). Such release can be blocked by the administration of anticholinergic and anti-

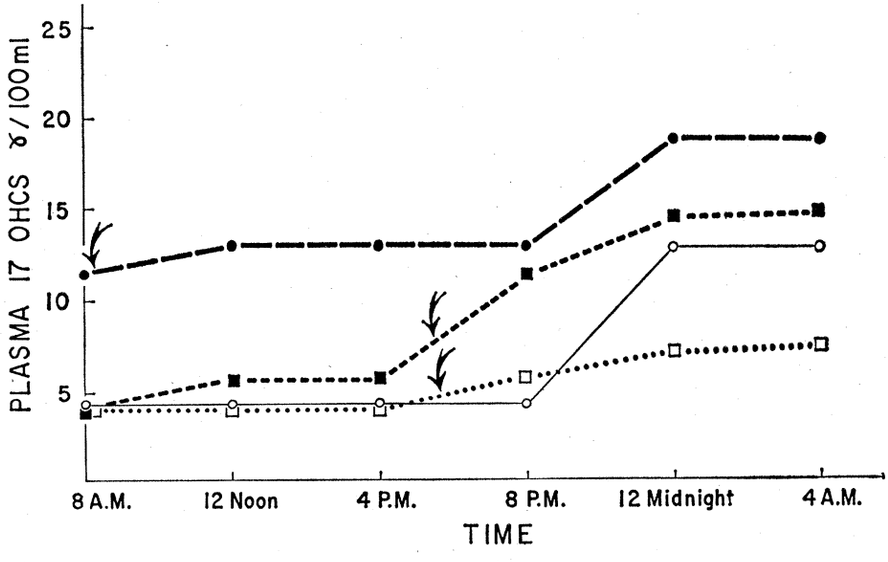

Fig. 1. Effect of atropine administered in different dosages and at different times of day on circadian pattern of plasma 17-OHCS in a cat (No. 224). Arrows indicate time of administration of drug. Key: ○——○, control day; □ · · · · □, atropine, 1.2 mg subcutaneously (0.4 mg/kg) at 6 p.m.; ■- - - - -■, atropine, 0.6 mg subcutaneously (0.2 mg/kg) at 6 p.m.; ●— — —●, atropine, 1.2 mg subcutaneously at 8 a.m.