than 1 dB. When the eye was in a fixational pause with close temporal proximity to a saccade, the pattern motions were plainly seen but evoked different (smaller) sway responses. These findings are consistent with a special role of the motor commands per se.

We infer that the central nervous system responds differently to visual image motion accompanying a saccade than to that which is externally produced. Neither visual masking effects nor the effects of retinal shear can explain our results.

Currently accepted views of the changes in visual sensitivity during saccades attribute saccadic-suppression effects predominantly to sensory factors. These include visual masking, optical blur, and perhaps proprioceptive feedback from the extraocular muscles. The role of efferent commands in reducing visual sensitivity is believed to be minor. For the visual control of sway, however, the relative contributions of afferent and efferent factors appear to be reversed. The need to omit inappropriate responses to self-produced visual motion during eye movements appears to be satisfied in distinctive ways by the mechanisms underlying visual sensitivity and those underlying postural stability.

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5. The results were qualitatively similar but less reliable when subjects stood on both feet. One subject could support himself on his hands and also showed the basic effects reported.
6. Supplementary experiments alternated the pattern between stationary and moving at rates from 20 to 0.05 per second. Sway frequency components from 2.5 to 3.5 Hz always showed high gain; thus, frequency dependence in the response is not controlled by stimulus timing per se. Timing did affect the amplitude of induced sway, however, which tended to increase with stimulation frequency. The larger sways evoked by aperiodic motions may reflect less habituation, since these occurrences were less predictable than periodic occurrences of the pattern motion.
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Opioid and Nonopioid Mechanisms of Stress Analgesia

Abstract. *Inescapable foot shock in rats caused profound analgesia that was antagonized by naloxone or dexamethasone when shock was delivered intermittently for 30 minutes, but not when it was delivered continuously for 3 minutes. Thus, depending only on its temporal characteristics, foot-shock stress appears to activate opioid or nonopioid analgesia mechanisms. Certain forms of stress may act as natural inputs to an endogenous opiate analgesia system.*

Early work on the analgesic effect of electrical brain stimulation led to the suggestion that portions of the central nervous system normally function to inhibit pain (*1*). This hypothesis gained considerable support from the discovery of opioid peptides with analgesic properties (*2*) and a distribution encompassing regions of the nervous system implicated in stimulation-produced and opiate analgesia (*3*). Suggesting that opioid peptides act as chemical mediators in this endogenous system of pain inhibition are studies showing that an opiate antagonist can block stimulation-produced analgesia (*4*), that cross-tolerance develops between stimulation-produced and opiate analgesia (*5*), and that opioid peptide concentration in human cerebrospinal fluid is altered by chronic pain and by analgesic central or peripheral stimulation (*6*).

To establish that opioids serve a biologically significant role in pain inhibition, the natural factors activating their release need to be identified. So far, only fragmentary or contradictory evidence related to this point is available (*7*). Perhaps the most promising lead comes from the recent demonstration that various stressors can cause analgesia (*8, 9*). Whether opioids mediate stress analgesia, however, has remained in doubt. Some reports indicate that stress analgesia is attenuated by the opiate antagonist naloxone (*8, 10, 11*) and that cross-tolerance develops between opiate and stress analgesia (*10, 12*). Moreover, stressors that induce analgesia alter brain and plasma indices of opioid activity (*13*). Other studies, however, indicate that stress analgesia neither manifests cross-tolerance with morphine nor is antagonized by naloxone (*14, 15*).

Consideration of the stress paradigms used in these studies reveals both qualitative and quantitative differences that may account for the disparity in the findings. The principal aim of the present experiment was to investigate the possibility that both opioid and nonopioid systems influence stress analgesia and that quantitative characteristics of a given stressor can determine which system is predominantly engaged. We find that, depending on its temporal parameters, inescapable foot shock can cause either an opioid or a nonopioid type of analgesia, as defined by susceptibility to naloxone blockade (*16*).

Because the analgesic effects of stress (*17*) and acupuncture (*18*) are reduced by hypophysectomy, it has been suggested that pituitary hormones mediate these forms of analgesia. Some pituitary cells contain both adrenocorticotropic hormone (ACTH) and β-endorphin (*19*) and release them concomitantly in response to stress (*20*). The synthetic glucocorticoid dexamethasone blocks the stress-induced rise in plasma ACTH (*21*) and β-endorphin (*22*) and decreases acupuncture analgesia in mice (*23*). Therefore, a second aim of our experiment was to in-

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vestigate the effects of dexamethasone on stress analgesia of both the naloxone-sensitive and naloxone-insensitive type.

Subjects were 60 male Sprague-Dawley albino rats (350 to 400 g) maintained on a 12-hour light-dark cycle (lights on at 11:00 p.m.) and tested during the dark phase of the cycle. Inescapable foot shock (60-Hz sine waves, 3-mA constant current) was delivered through a scrambler to the grid floor of a Plexiglas chamber (23 by 23 by 20 cm). Animals were exposed to one of three procedures: (i) brief, continuous foot shock for 3 minutes; (ii) prolonged, intermittent foot shock for 30 minutes (1-second pulses delivered every 5 seconds), as previously described by Azil *et al.* (*8*); and (iii) a no-stress condition in which animals were handled similarly but received no shock. Baseline pain responsiveness was measured with the tail-flick test (*24*) immediately before shock administration. Five tail-flick trials were conducted at 1-minute intervals, and the baseline was defined as the mean tail-flick latency for the last three trials. An analysis of variance of the baseline data revealed no significant difference between groups. Tail-flick testing resumed 1 minute after the stress or control procedure, continuing at 1-minute intervals for 9 minutes and subsequently at 2-minute intervals until 15 minutes had elapsed since the procedure. A 7-second limit of exposure to the radiant heat stimulus was imposed to minimize tissue damage to the tail.

To assess the effects of naloxone and dexamethasone, 12 groups of five animals each were given one of the two drugs or their appropriate vehicle controls and exposed to one of the three procedures. Naloxone HCl (10 mg/kg) or isotonic saline was injected intraperitoneally immediately after the baseline testing and again 30 minutes later, just before analgesia assessment. The prolonged, intermittent foot shock was applied during the entire 30-minute interval between injections; the brief, continuous foot shock, during the final 3 minutes before the second naloxone injection. Alternatively, dexamethasone (0.4 mg/kg) was injected intraperitoneally 24 hours before stress was applied; and 0.2 mg/kg was injected 1 hour before the procedure. [This dexamethasone administration regimen was found effective by French *et al.* (*22*) in suppressing a foot shock–induced rise in plasma β-endorphin levels.] Equivalent volumes of isotonic saline were injected simultaneously into the control animals.

Figure 1 shows the major results of these experiments. A 4 × 3 × 12 analysis of variance with two between-group independent variables (drug regimen and stress procedure) and one repeated factor (time) revealed significant main effects for drug, stress procedure, and time ($P < .001$ in each case). The only significant interactions found were stress procedure × drug ($P < .001$) and stress procedure × time ($P < .001$). Specific comparisons reported below are based on F-tests for simple effects.

Both stress conditions caused strong and long-lasting analgesia. Thus, among saline-treated animals, tail-flick latencies were significantly longer for those subjected to either prolonged, intermittent foot shock ($P < .01$) or brief, continuous foot shock ($P < .05$) than for nonstressed controls. Animals subjected to the prolonged, intermittent foot shock manifested analgesia of significantly longer duration ($P < .05$) and appeared behaviorally depressed (*25*).

Naloxone and dexamethasone both blocked analgesia for rats receiving prolonged, intermittent foot shock (Fig. 1A). Compared to saline-treated controls, the naloxone- and dexamethasone-treated groups had significantly shorter tail-flick latencies ($P < .01$). In fact, neither drug-treated group subjected to this stress procedure showed tail-flick latencies that were different from those of drug-treated but nonstressed controls (*26*).

By contrast, neither naloxone nor dexamethasone significantly altered the analgesic effect of brief, continuous foot shock (Fig. 1B). Significant elevations in tail-flick latencies were obtained under this stress condition in groups receiving naloxone ($P < .01$), dexamethasone ($P < .01$), and saline ($P < .05$ for each group) compared with latencies in similarly treated but nonstressed controls.

Finally, no significant between-group differences in tail-flick latency were found among nonstressed animals subjected to the different drug regimens (Fig. 1C). It may be concluded that neither naloxone nor dexamethasone affected baseline pain responsiveness under the test conditions.

These results provide clear evidence for the involvement of two possibly independent substrates in stress analgesia, only one of which appears to be acted on by opioids. Which substrate is predominantly acted on during foot shock depends on the temporal properties of the shock: its duration (3 versus 30 minutes), its pattern (continuous versus intermittent), or both. By defining and bringing under control a set of parameters that determine whether naloxone-sensitive or naloxone-insensitive mechanisms of stress analgesia are engaged, these findings not only help explain discrepancies among the results of previous studies of naloxone's effect on stress analgesia (*8, 10, 11, 14, 15*), they also reinforce the view that there exists an opioid-mediated system of pain inhibition with physiological inputs that are activated by certain stress conditions.

It has been shown that hypophysectomy can block stress analgesia (*17*) and that stress causes a rise in plasma ACTH and β-endorphin levels (*27*) that is blocked by dexamethasone (*22*). We observed that dexamethasone blocks only the type of stress analgesia that is also blocked by naloxone. These findings are consistent with the hypothesis that pituitary hormones, presumably β-endorphin, mediate at least certain forms of stress analgesia. It has been pointed out, however, that plasma concentrations of β-endorphin after stress are well below those needed to produce analgesia with systemic β-endorphin administration (*27*). A possible resolution of this paradox may lie in studies that suggest there is a blood flow toward the brain through

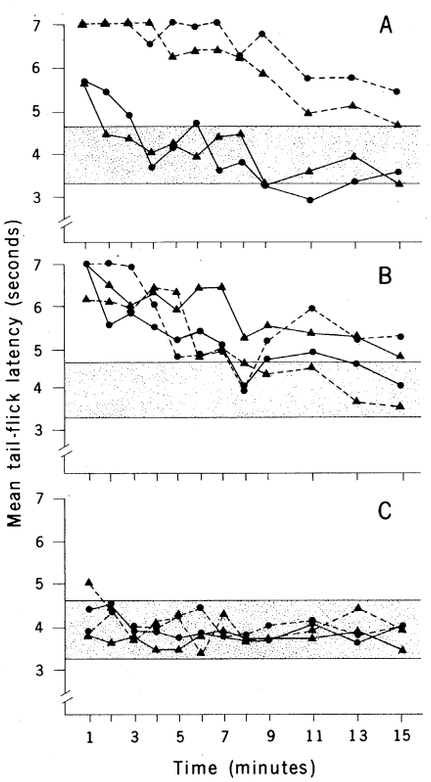

Fig. 1. Mean tail-flick latencies of groups treated with naloxone (●——●), naloxone vehicle (●----●), dexamethasone (▲——▲), and dexamethasone vehicle (▲----▲) at 12 time periods beginning 1 minute after exposure to (A) prolonged, intermittent foot shock, (B) brief, continuous foot shock, and (C) no foot shock. The shaded areas represent ± 2 standard errors of the grand mean calculated for the four nonstressed groups shown in (C).

the hypophyseal portal system (*28*). Thus it may be that brain areas mediating pain inhibition are reached via this route by opioids of pituitary origin in concentrations sufficient to cause analgesia. The intriguing possibility also exists that the ventricular system normally serves as a conduit for transporting pituitary opioids from ventrobasal regions of the hypothalamus to the more distant periventricular and periaqueductal structures thought to be involved in endogenous mechanisms of analgesia.

It is also possible that naloxone-sensitive stress analgesia is mediated in part by opioid release directly within the brain. In fact, it has recently been reported that lesions of the arcuate nucleus that deplete brain levels of β-endorphin can disrupt some forms of stress analgesia (*29*). The demonstration of specific dexamethasone binding sites in the brain (*30*) suggests a means by which this drug could inhibit centrally released opioids and hence block the analgesic effect of prolonged, intermittent foot shock.

Our findings suggest that both opioid and nonopioid mechanisms underlie stress analgesia. To evaluate this hypothesis, studies are needed in which other criteria for opioid involvement (*16*) and other stressors are used. It will also be important in future work to assess the specificity of the analgesic effect of stress. Severe stress causes a constellation of physiological changes (thermoregulatory, motoric, hormonal, respiratory, and cardiovascular), some of which, like analgesia, have been found to be anatagonized by naloxone (*22, 25, 31*). The possibility that naloxone-sensitive stress analgesia is secondary to one or more of these other physiological effects cannot yet be dismissed.

Note added in proof: In work completed since this report was submitted, we found that rats receiving five daily injections of morphine (5 mg/kg) show less analgesia to prolonged, intermittent foot shock than saline-injected controls (*32*). The same morphine regimen does not affect analgesia to brief, continuous foot shock. This demonstration of cross-tolerance between the analgesic effects of morphine and the naloxone-sensitive (but not naloxone-insensitive) form of stress analgesia provides further evidence for the existence of opioid and nonopioid mechanisms of stress analgesia.

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Rings of Uranus: Proposed Model Is Unworkable

In a recent report (*1*), Van Flandern restates some of the difficulties with a conventional picture of Uranus's rings and proposes that they are the gaseous tails of unobserved small satellites. No quantitative support for this hypothesis is given; the only substantiation is a spectacular cover sketch. Van Flandern's hypothesis raises far more objections than it answers. A gaseous torus must have a minor diameter of thousands, not tens, of kilometers; very large densities are required to produce the postulated angles of refraction; and making up the mass losses, even with a generous lifetime, would use up quite a large body in a few million years.

In McDonough and Brice's study (*2*) of a possible torus associated with Titan, the minor diameter was found to be large, of the order of a Saturn radius or more. The ionized Io torus (*3*) is of a similar dimension. The difficulty with a very narrow torus can be readily illustrated, if we use the η ring of Uranus as an example; it has major and minor radii of

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