feature at 47 km/sec from the position W3 OH (Fig. 1) by assuming the intensities and line spacings in Table 4 and taking various (equal) line widths and Gaussian profiles for the transitions. We were unable to obtain a satisfactory fit in this way. Hence, we may conclude, independent of considerations of source-brightness and polarization, that we are not observing a source in thermal equilibrium. A single Gaussian profile is an excellent fit to the feature profile above the half-maximum level, but below this level the observed profile is broader than the Gaussian profile, particularly on the low-velocity (high-frequency) side. However, we estimate that these features could be fitted very accurately if we relaxed the constraint that the three most intense transitions in Table 4 have the equilibrium intensity ratios. The high observed antenna temperatures and the small angular size of the regions of water emission imply high brightness temperatures and suggest a maser-type emission. The relative intensities could, in fact, be altered by the ratios of the maser gains for various hyperfine transitions, but we cannot distinguish between saturated and exponential gains on the basis of these data. Nevertheless, the presence of hyperfine splitting must be taken into account if we are to interpret the apparent feature widths.

M. L. MEEKS
J. C. CARTER
Lincoln Laboratory, Massachusetts Institute of Technology, Lexington
A. H. BARRETT
P. R. SCHWARTZ, J. W. WATERS
Department of Physics and Electrical Engineering and *Research Laboratory of Electronics, Massachusetts Institute of Technology, Cambridge*
W. E. BROWN III
Optics and Microwave Laboratory, Electronics Research Center, National Aeronautics and Space Administration, Cambridge, Massachusetts 02139

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10. The effective area of the Haystack antenna at a wavelength of 1.35 cm appears to be approximately equal to that of the 85-foot (26-m) antenna of the Naval Research Laboratory (*2*). We believe the surface tolerance of these two antennas is nearly equal because the predicted loss from the Haystack radome at this wavelength (2.4 db) approximately offsets the difference in diameter. The half-power beam widths, of course, scale as 85:120.
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13. Weather conditions during the observations on 10 February 1969 were unusually bad. The W49 spectrum shown for that date in Fig. 2 was taken during the worst Boston-area blizzard in 20 years.
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Primary Afferent Depolarization Evoked by a Painful Stimulus

Abstract. *Pulses of intense radiant heat applied to the plantar pad of unanesthetized spinal cats produced negative dorsal root potentials, increased excitability of cutaneous A fibers, and marked activation of ipsilateral flexor motoneurons. The same effects were obtained during cold block of A fiber conduction in the appropriate peripheral nerve. We conclude that adequate noxious activation of cutaneous C fibers depolarizes cutaneous A fibers.*

A basic tenet in the theory of pain mechanisms proposed by Melzack and Wall (*1*) is that cutaneous afferent fibers of large and small diameter have opposing effects on the spinal mechanisms which presynaptically modulate synaptic transmission in other cutaneous afferents. This assumption was derived from the observation (*2*) that selective electrical stimulation of small-diameter unmyelinated cutaneous fibers (C fibers) produced positive dorsal root potentials (DRP's) and hypoexcitability of cutaneous afferent terminals. However, this is in conflict with studies (*3*, *4*) which report that selective electrical C fiber stimulation resulted in negative DRP's similar to those produced by the large diameter myelinated A fibers from the skin.

Since electrical stimulation of skin nerves may not initiate patterns of afferent activity similar to those produced by natural painful stimuli, we have investigated the effects of intense radiant heat pulses applied to the plantar pad of spinal cats on the polarization of afferent fibers. This stimulus is clearly noxious and produces intense pain when applied to human skin, and the stimulus appears to activate primarily, if not exclusively, C fibers (*5*).

Under halothane anesthesia, the lumbosacral spinal cord of adult cats was exposed and sectioned at the first lumbar segment (L1). The ventral roots (VR) of L6 through S1 were cut and a small filament of the L6 dorsal root (DR) was cut and prepared for recording. All sciatic nerve branches were cut in the left leg except for the posterior tibial nerve, and some were placed on platinum wire electrodes. After completion of the dissection the animals were decerebrated and, with anesthesia discontinued, paralyzed with gallamine triethiodide and artificially respired. The cord and nerves were covered with warm mineral oil. Blood pressure and body temperature were monitored continuously and remained within normal limits. The posterior tibial nerve containing the afferents from the central plantar pad was preserved intact and in some experiments was mounted on a cooling device to block nerve conduction (*4*). Hair around the left central plantar pad was removed and a small thermocouple was placed on the pad surface for temperature recording. Pulses of radiant heat were directed exclusively to the pad by a focusing projection lamp covered with a movable shutter.

Intense heat pulses delivered to the plantar pad consistently evoked nega-

tive DRP's in ipsilateral DR filaments (Fig. 1A, upper trace). It may be inferred from this that some of the afferent terminals were depolarized (had undergone primary afferent depolarization) at some point along their intraspinal course (*6*). Onset of the negative DRP waves was coincident with the beginning of markedly increased motoneuron discharge in VR filaments (Fig. 1, lower traces). Both effects began when the surface temperature of the pad reached 45° to 55°C (Fig. 1, middle traces). The VR activity was due to repetitive firing in flexor motoneurons (*7*).

Negative DRP's produced by A fiber volleys in a cutaneous nerve (sural) were decreased in amplitude when superimposed on a heat-evoked negative DRP (Fig. 1B). The reduction in A fiber DRP's during heat-evoked DRP's varied in magnitude in different experiments, but the two responses were always of the same polarity, that is, negative.

To specify which types of afferent fibers were depolarized by the heat stimulus, we studied the excitability changes of afferent terminals within the spinal cord with the technique developed by Wall (*8*). This technique is based on the notion that when afferent terminals undergo primary afferent depolarization (PAD), they become more easily excited to discharge when stimulated directly within the cord, leading to an increase in the antidromically conducted response which can be recorded from peripheral nerves. The records in Fig. 2A show antidromic A fiber responses, and their corresponding areas, recorded from one branch of the sural nerve during application of a heat pulse to the ipsilateral plantar pad. Both the peak and the area of the antidromic responses increased during the rising phase of the heat pulse. The time course of the changes in the antidromic response and the pad surface temperature are shown in Fig. 2, B and C, respectively. The duration of the increase in cutaneous afferent terminal excitability was approximately the same as that of the heat-evoked DRP (*9*).

Impulses in cutaneous A fibers cause PAD in other cutaneous afferents, resulting in negative DRP's (Fig. 1B) and increased excitability (*8, 10*). When the terminals of fibers in one branch of the sural nerve were maximally depolarized by conditioning volleys in another sural branch at about three times threshold intensity, there was neither addition nor subtraction of excitability (Fig. 2E, dots and solid line) during a superimposed heat pulse (Fig. 2F). Decreasing the strength of sural conditioning to about 1.3 times threshold produced an intermediate level of hyperexcitability. A superimposed heat pulse then resulted in an additional excitability increase (Fig. 2E, open squares). These results indicate that the same cutaneous afferent fibers are depolarized by both types of stimulation.

To ascertain the relative contribution of large and small diameter afferent fibers to the effects produced by plantar pad heat pulses, we attempted to elim-

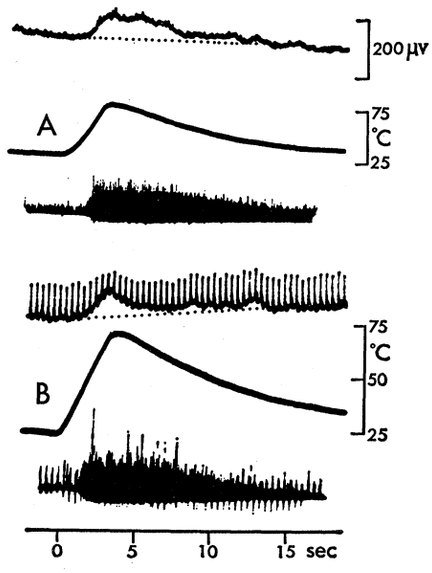

Fig. 1. Negative dorsal root potentials (DRP's) produced by radiant heat pulses to central plantar pad in an unanesthetized spinal cat. (A) Top trace; potential recorded from L6 dorsal root filament with d-c coupled amplifier, negativity at proximal electrode indicated by upward deflection. Middle trace; skin surface temperature of plantar pad. Lower trace; activity in cut L7 ventral root filament. (B) as in (A), but with negative DRP's evoked by single sural nerve volleys superimposed every 400 msec. Note skin temperature scale change. The dotted lines in DRP records indicate presumed baselines. Spinal cord temperature, 36.4°C; body temperature, 37.2°C.

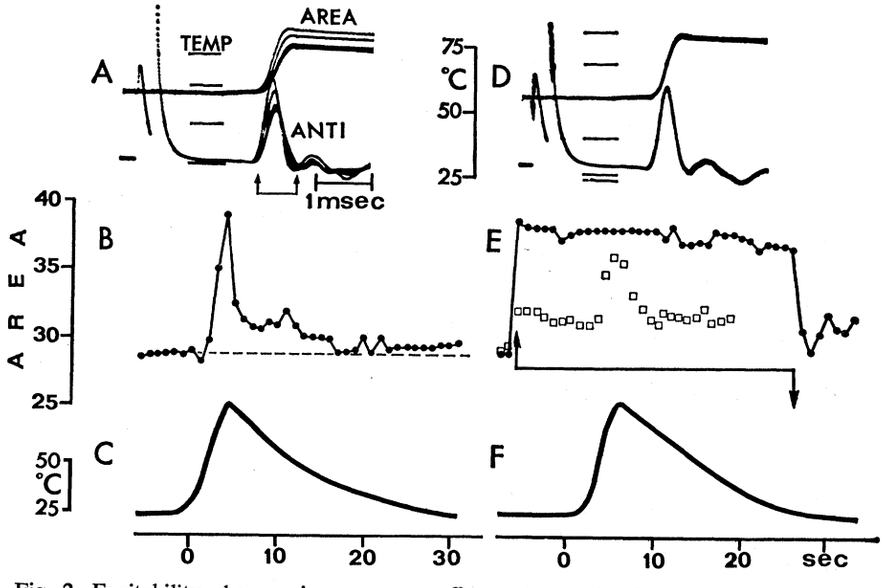

Fig. 2. Excitability changes in cutaneous afferent terminals. (A) Four superimposed records taken at intervals of 1/sec of the antidromic responses (*ANTI*, lower traces) recorded from one branch of the sural nerve, produced by constant current local stimulation in the L7 dorsal horn by a micropipette filled with 3*M* NaCl. The antidromic spikes were integrated over the interval indicated by arrows with an analog network, and resulting areas are shown in the upper traces (*AREA*). The records were obtained during the rising phase of a plantar pad heat pulse, and skin temperature during each fast sweep is indicated by the short lines labeled *TEMP*. (B) and (C) The complete time course of changes in area of the antidromic responses and pad surface temperature during heat pulse, respectively. (D) Format as in (A), except that each antidromic response was preceded by 23 msec with a single conditioning shock to another branch of the sural nerve, giving maximum excitability increase. Note that heat pulse caused no additional change in excitability. The time course of antidromic response area changes is shown in (E) by filled circles and solid line; sural conditioning applied between arrows. Open squares, another trial showing area changes when conditioning sural volley was reduced to give submaximal excitability increase. (F) Pad surface temperature. Temperature records for two trials in (E) were essentially the same. Cord temperature, 37.5°C; body temperature, 37.0°C.

inate any possible A fiber contamination in the input from the pad by cooling the posterior tibial nerve above the ankle. Franz and Iggo (*4*) have shown that controlled cold block can abolish conduction in A fibers, leaving only C fibers conducting. Figure 3A shows the negative DRP and VR discharge produced by a heat pulse before cold block. When the temperature of the posterior tibial nerve was reduced to 5.3°C, the heat pulse produced similar although somewhat delayed effects (Fig. 3C). At this temperature, A fiber conduction was blocked, as shown by the absence of both the early action potential and the negative DRP (Fig. 3D) when the posterior tibial nerve was stimulated distal to the block at six times threshold. Rewarming the nerve established conduction in the myelinated fibers again (Fig. 3F), but there was little change in the heat-evoked DRP (Fig. 3E) other than some decrease in latency. In similar experiments we have shown that heat-evoked increases in excitability of cutaneous afferent terminals (Fig. 2) remain essentially the same during cold block of A fiber conduction.

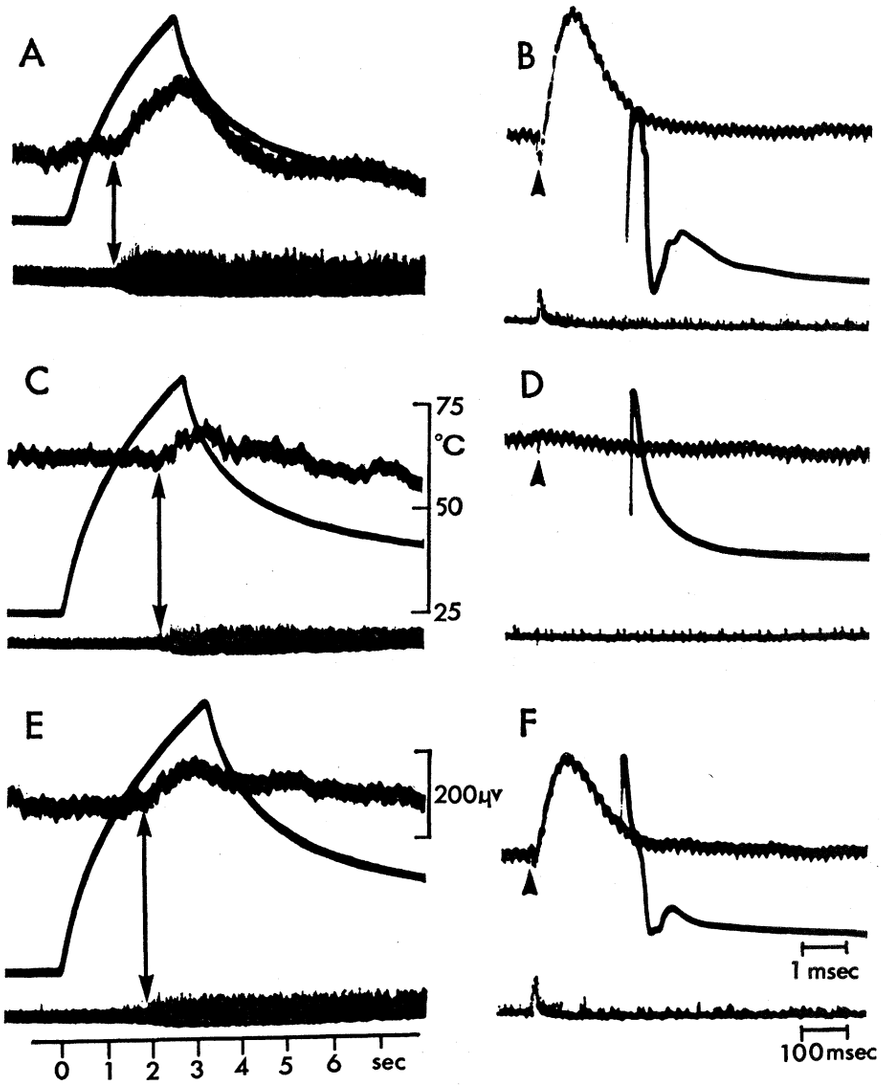

Fig. 3. Effect of A fiber conduction block on DRP's evoked by plantar heat pulses and electrical stimulation. (A) Negative DRP's in L6 DR filament (upper trace) and flexor VR discharge (lower trace) during plantar pad heat pulse (middle trace) before cold block of posterior tibial nerve. (B) Negative DRP (upper trace) and ventral root discharge (lower trace) evoked by electrical stimulation of posterior tibial nerve at six times threshold before blocking. Middle trace shows, on fast sweep [1 msec calibration in (F)], orthodromic volley in the nerve recorded at a point 5 cm proximal to stimulating electrode. (C) and (D) as in (A) and (B), but during cold block of tibial nerve (nerve temperature 5.3°C). (D) Negative DRP and A fiber volley evoked by stimulation distal to the site of block [as in (B)] were eliminated with nerve cooled to 5.3°C (only stimulus artifact remained in fast trace). (E) and (F) as in (A) and (B), after rewarming tibial nerve to 24°C. Other evidence indicated that conduction in A and C fibers was incompletely reestablished on rewarming. Note that DRP and VR discharge evoked by heat pulse during A fiber blockade (C) were somewhat delayed in onset but were qualitatively similar to the effects obtained before conduction block (A). Cord temperature, 37.2°C; body temperature, 35.6°C.

These results show that pulses of intense radiant heat applied to the plantar pad of spinal cats produce PAD in cutaneous afferent terminals as well as intense discharge of flexor α motoneurons. The heat-evoked PAD occurs in the same fibers which are depolarized by A fiber volleys and the effect is essentially unmodified after blockade of A fiber conduction from the skin. These findings are in agreement with those of Zimmermann (*3*) and of Franz and Iggo (*4*). With a natural painful stimulus, we have found no evidence for the existence of a presynaptic gating mechanism activated by painful stimuli such as that proposed by Melzack and Wall (*1*).

L. Vyklický,* P. Rudomin†
F. E. Zajac, III, R. E. Burke
Laboratories of Neurophysiology and *Neural Control, National Institute of Neurological Diseases and Stroke, Bethesda, Maryland 20014*

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* On leave from the laboratory of cellular and comparative neurophysiology, Academy of Sciences, Institute of Physiology, 4 KRC Budjovícka 1083, Praha, Czechoslovakia.
† On leave from the Department of Physiology, Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, México 14, D. F.

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