different depths are compared to find the depth with the largest response. Llinás *et al*, however, ask *when* does the largest response occur at each depth and then compare these peak latencies between depths. To calculate this temporal maximum, we set $\partial V/\partial T = 0$.

7. For longer conductance changes, the rate is not significantly altered. In figure 1, for example, we use a square conductance change of 0.2 τ duration.
8. The apparent propagation rate for a passively spreading spike will be faster than for a PSP. For example, a biphasic current stimulus which produces a spike of 0.2 τ duration at the origin will spread with an apparent propagation rate about twice as fast as a PSP caused by a monophasic pulse.
9. Supported by NIH grants NB 04053 and GM 33139.

* Present address: Department of Physiology and Biophysics, University of Washington, Seattle.

30 September 1968

Calvin and Hellerstein claim that the negativities observed at the molecular layer of the alligator cerebellum after parallel fiber activation are generated by the synaptic current as the synaptic potentials are decrementally conducted toward the soma of the Purkinje cells. They state a well-known set of equations for the electrotonic decrement of amplitude and change of time course for the intracellularly recorded synaptic potentials, but these are not directly applicable to the case in question.

The spatial orientation of the Purkinje cells is of the open-field type (*1*) so that it permits a simplification of the dendritic trees to a series of vertical core conductors (*2*). This particular organization resembles closely a muscle-nerve preparation, where the only region in which an excitatory synaptic current generates a negative extracellular field is in the vicinity of the synaptic region (*3*). The inward current across the postsynaptic-subsynaptic membrane is simultaneously accompanied by an outwardly directed current which generates the postsynaptic potentials and which produces in the extracellular media a positive field as the external longitudinal current moves toward the synaptic sink. This type of field is, in fact, the most commonly observed in other vertebrate cerebella (*4, 5*). Our findings, however, relate to a negative field recorded extracellularly, that is, a sink which moves in time from its origin at the surface of the molecular layer to the layer of Purkinje cell somas. Furthermore, their argument is faulty, even if it were to be applied to an extracellular positivity. The extracellular field potentials are generated by transmembrane currents and not by transmembrane potentials. To calculate the time course of the extracellular field one should take the second derivative of the intracellular potential with respect to space in order to estimate the transversal current density along the cable (*6*). The results would have reminded them that there is a marked phase shift between the transmembrane potential and the transmembrane current and thus that the time course of an electrotonically conducted EPSP does not reflect directly the time course of the extracellular field potential.

The aforementioned negativity is blocked by a preceding stimulus to the surface of the cerebellar cortex. This stimulus generates large inhibitory synaptic potentials in alligator Purkinje cells (*5*) similar to those observed in other species (*4*). Excitatory synaptic currents are increased, not blocked, by membrane hyperpolarization since the electromotive force is larger under such conditions than at the normal resting potential level. Since our negative fields are blocked by a preceding parallel fiber stimulation which does not have any presynaptic action on the test EPSP (*7*), the large dendritic negativity cannot be ascribed to synaptic currents.

As shown in figures 1, B and C, and 2A of our paper (*8*), the amplitude of the negativity does not reach its maximum value at the region of synaptic impingement but at an appreciable distance from this site; thus it cannot be generated by decrementally conducted potentials, given that, in such a situation, the maximum amplitude of the potential should correspond to the locus of synaptic input.

For the reasons cited in the first paragraph, if the negativity recorded at the molecular layer were generated by synaptic currents, a reversal to a positive field should be evident as the microelectrode moves away from the site of synaptic impingement along the length of the cell. Such reversal was never observed. A reversal from a negative field at the surface of the molecular layer to a positive field at 100 μ and all levels below this depth was shown, however, for the synaptic currents generating the dendritic spikes after these spikes were blocked by a preceding local stimulation (Fig. 2B).

Finally, the presence of large all-or-none negativities at the level of the Purkinje cell dendrites (*8*, figure 2, E to I) and their inhibition by a preceding local stimulation is, in fact, a direct demonstration of dendritic spikes in Purkinje cells. The other neuronal elements of the molecular layer, the stellate cells, display a very different behavior following paired Loc stimulation (*9*).

We do not consider the criticisms of Calvin and Hellerstein as truly pertinent to the question of dendritic spikes, and we feel that our data can best be explained by the presence of propagating dendritic spikes in Purkinje cells.

RODOLFO LLINAS
CHARLES NICHOLSON
JOHN A. FREEMAN
DEAN E. HILLMAN
Institute for Biomedical Research,
535 North Dearborn Street,
Chicago, Illinois 60610

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23 October 1968

Educational Status and Risk of Coronary Heart Disease

Hinkle *et al.* (*1*) have investigated the occurrence of death and disability from coronary heart disease (CHD) in relation to occupational and educational status. Their sample includes few men with angina pectoris as the only manifestation of CHD because angina often does not result in prolonged absence from work. Thus, their observations have direct relevance primarily for the epidemiology of myocardial infarction and sudden death, the other two major clinical forms of CHD. However, Hinkle *et al.* generalize their results to risk of CHD without qualification. They seem to overlook the possibility that angina pectoris may be related to the antecedent conditions in a different manner than is myocardial infarction and sudden death.

Previous investigations (*2*) have shown that a factor can be related to one clinical form of CHD but not another. Excess body weight in the Framingham study bears little relation to risk of myocardial infarction, although it is associated with occurrence of angina pectoris and sudden death. Cigarette smoking is related to increased risk of myocardial infarction but is unrelated to risk of angina pectoris.

Data from the Western Electric study (*3*) suggest that educational status is related to incidence of CHD but that the form of the relationship varies according to the clinical form by which the disease is first manifested. In this cohort the incidence of myocardial infarction and death from CHD is less in the upper than in the lower educational strata. This finding agrees with the data presented by Hinkle *et al.* (*1*, Fig. 2). However, the incidence of angina pectoris in the Western Electric cohort follows the opposite pattern; angina pectoris as the first manifestation of CHD occurs more frequently in the upper than in the lower educational strata.

For these reasons the conclusions of Hinkle *et al.* should not be generalized at this time to risk of CHD, but should be limited to those clinical forms actually studied by them, myocardial infarction and sudden death.

RICHARD B. SHEKELLE
Department of Preventive Medicine and Community Health, University of Illinois, Chicago 60612

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11 October 1968

Angina pectoris is not a form of coronary heart disease; it is a pain in the chest which occurs in association with a number of cardiovascular diseases. Its association with coronary heart disease in American men is based on evidence that men who have no other obvious cause for angina pectoris have a greater likelihood of dying of coronary heart disease (*1*), and of having significant areas of atherosclerosis found in their coronary arteries at autopsy (*2*).

We have no data on the prevalence of the complaint of angina pectoris in the whole population of 270,000 Bell System men. However, we do have data on 407 events of angina pectoris which led to periods of sickness disability in this population during 5 years. At every age from 30 to 65 the rates are higher among no-college men than among college men (see Table 1).

Table 1. Rate of disability attributed to angina pectoris among Bell System men, 1963–1966 (annual number of events per 1000 men). Comparing number of events observed with number expected in each age band, on the assumption of a single population with equal risk at each age, yields $\chi^2 = 13.190$ (d.f. $= 7$), $P = .05$.

Age	"No-College" men		"College" men	
	Men at risk	Rate	Men at risk	Rate
30–34	41,423	0.02	3453	
35–39	45,126	.09	3501	
40–44	42,234	.22	2865	0.09
45–49	25,076	.36	1847	.27
50–54	12,944	.73	1195	.42
55–59	15,599	1.23	2292	.65
60–64	11,375	2.44	2153	.93

We also have essentially complete data on the 30-year mortality from age 30 to age 60 among an age cohort of 1160 men in New Jersey; and we have data on the prevalence of angina pectoris, of definite and probable myocardial infarction, and of electrocardiographic changes indicative or suggestive of coronary heart disease, among the survivors (*3*). Mortality data from this cohort are in accord with the findings of the national survey that coronary deaths are more frequent in no-college men than in college men. The data from the survivors suggests that the other indicators of coronary heart disease are also more common among no-college men than among college men. Preliminary results from other studies of younger men, from several states throughout the System, are in accord with this finding.

Based on our experience, we believe that all forms of coronary heart disease are more prevalent among "no-college" than among "college" men in the Bell System. However, we also think it quite possible that a complaint of chest pain, which could be interpreted as angina pectoris, might be more readily elicited from "college" men than from "no-college" men in the course of a survey conducted in an industry. This opinion is based upon our experience with our intensive surveys of small samples of Bell System men, in the course of which medical questionnaires, asking for specific items of information relating to 219 syndromes and to many symptoms, were administered in a standard manner to all subjects; and all subjects were interviewed at length by physicians. Despite the fact that the prevalence of coronary heart disease, as detected on examination, was lower in the college men, these men gave a greater number of positive answers relating to past illnesses and to present symptoms that were of concern to them, and during the interviews they responded more freely and completely to the questions of the physicians than did the men at the lowest educational levels.

Shekelle bases his conclusion on a sample of 1472 men from the Western Electric Company (*4*). The men in this sample are roughly similar in many respects to the men of the Bell System Operating Companies whom we have examined. There are 154 men in his sample who have approximately the same educational characteristics as our "college" men. The 13 complaints of angina pectoris recorded for this group during the survey were 6 more than would have been expected on the assumption of equal risk, but only a single subsequent event of myocardial infarction was found, where three and a half would have been expected. It seems to us quite possible that these findings may be related to the greater willingness and ability of the college men to communicate with the examining physicians in an industrial setting.

LAWRENCE E. HINKLE, JR.
Department of Medicine, New York Hospital–Cornell Medical Center, New York 10021

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12 November 1968