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Characteristics of Interhemispheric Impulse Conduction Between Prelunate Gyri of the Rhesus Monkey

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Summary. Cells of origin of the corpus callosum (callosal efferent neurons) in prelunate gyrus (area OA) of the rhesus monkey were studied using electrophysiological techniques. Monkeys were chronically prepared and callosal efferent neurons were identified by their antidromic activation following electrical stimulation of the contralateral prelunate gyrus and/or the splenium of the corpus callosum. Interhemispheric antidromic latencies ranged from 2.6–18.0 ms (median = 7.0 ms) while the conduction velocity along the length of the axon ranged from 2.8 to 22.5 M/s (median = 7.4 M/s). Following the relative refractory period of a single prior impulse, all but one of 61 callosal efferent neurons studied showed a supernormal period of increased axonal conduction velocity and excitability. Following several prior impulses, the supernormal period was followed by a subnormal period of decreased axonal conduction velocity and excitability, which, depending on the number of prior impulses, lasted from several hundred ms to nearly 2 min.

Key words: Corpus callosum – Callosal efferent neurons – Visual association cortex – Conduction velocity – Supernormal impulse conduction.

Both the conduction velocity and excitability of callosal axons in the rabbit vary with the history of impulse conduction along the particular axon under study (Swadlow, 1974a, 1974b, 1977; Swadlow and Waxman, 1976; Waxman and Swadlow, 1976). Systematic increases and decreases in conduction velocity and excitability occur, and in some cases, last for more than 1 min following prior impulse activity. In central nervous system, a similar history dependence in conduction velocity had been previously descrived only in parallel fibers of cat cerebellum (Gardner-Medwin, 1972), which have a distinct morphology (Palay

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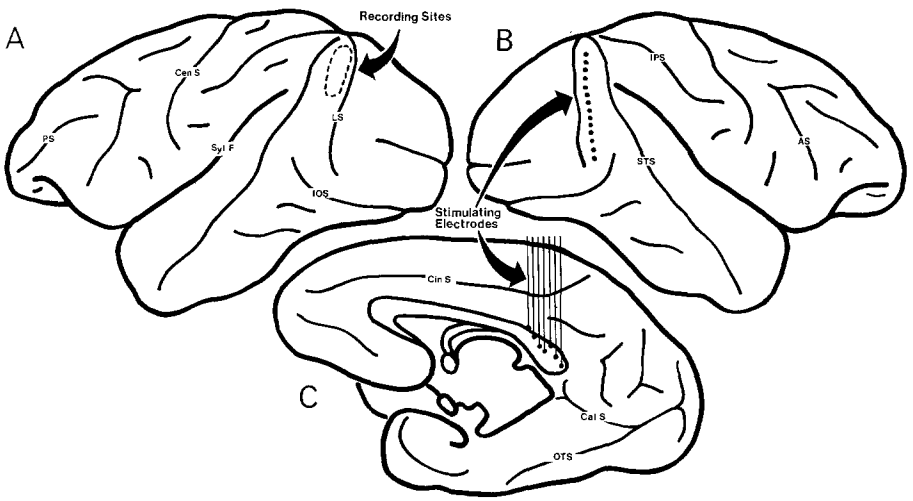

Fig. 1. Schematic illustration made from photographs of the brain of one of the rhesus monkeys used in these experiments. **A** The area in the central portion of the prelunate gyrus from which all microelectrode recordings were taken. **B** The location of cortical stimulating electrodes in the contralateral prelunate gyrus. **C** The location of the midline stimulating electrodes in the splenium of the corpus callosum. Abbreviations: AS – arcuate sulcus; Cal S – calcarine sulcus; Cen S – central sulcus; Cin S – cingulate sulcus; IPS – intraparietal sulcus; IOS – inferior occipital sulcus; LS – lunate sulcus; OTS – occipital temporal sulcus; PS – principal sulcus; SylF – Sylvian Fissure; STS – superior temporal sulcus

and Chan-Palay, 1974). Such history dependence of conduction properties may have significance with respect to temporal summation and coding of neural information (Chung et al., 1970; Swadlow and Waxman, 1975). In the present study we examine characteristics of impulse conduction along callosal axons originating in prelunate gyrus of rhesus monkey (area OA of Bonin and Bailey, 1947). This cortical region projects directly to contralateral prelunate gyrus (see below) and to ipsilateral inferotemporal cortex (Kuypers et al., 1965) and may play a role in the elaboration of visual discriminations (see Review by M. Wilson, 1978).

Methods

Extracellular single-unit recordings were obtained from callosal efferent neurons in prelunate gyrus of the rhesus monkey (M. mulatta). Several days prior to microelectrode recording, under sodium pentobarbital anesthesia, a steel bar (6 mm diameter, 20 mm in length) was attached to the skull with stainless steel screws and acrylic cement. During subsequent recording sessions the head was immobilized by clamping this bar to a rigid frame while the body rested on a foam rubber pad. During initial surgery, the skin and fascia were permanently removed from the region of skull that lay over the area of cortex to be recorded from. This region of skull was protected by a very thin layer of acrylic cement. In each monkey, a row of 12–17 stimulating electrodes was implanted along the prelunate gyrus of the contralateral hemisphere, at a depth of approximately 1.5 mm and within 2.0 mm of the lunate sulcus (interelectrode distance was approximately 1.5 mm). In one monkey a

row of 7 stimulating electrodes was also implanted, under visual control, into the splenium of the corpus callosum (1.0 mm between electrodes). Figure 1A presents a schematic illustration of recording sites in the prelunate gyrus of one hemisphere. In Fig. 1B and 1C, stimulation sites in the contralateral hemisphere and splenium of the corpus callosum are shown. Stimulating electrodes were, in most cases, constructed of sharpened platinum-iridium wire, insulated to within 0.75 mm of the tip. Stimuli consisting of 0.1 ms rectangular pulses of constant current were delivered to the stimulating electrodes through a stimulus isolation unit. Stimuli were either presented singly or a conditioning stimulus (CS) was followed by a test stimulus (TS). In some cases the CS consisted of a train of pulses. Stimuli were also triggered at various intervals following a spontaneous spike.

Beginning several days after the initial surgery, each monkey participated in 4–8 recording sessions, with 1–4 days usually allowed between sessions. During recording sessions, monkeys were either anesthetized with sodium pentobarbital or tranquilized with phenycyclidine HCL. Body temperature was maintained at 36–38°C. Recordings were obtained via tungsten-in-glass microelectrodes, through a small entrance in the skull and through an intact dura.

The principal criterion for the identivication of neurons, which were antidromically activated, was the test for collision of impulses. This test has been thoroughly described previously (see, e.g., Bishop et al., 1962; Fuller and Schlag, 1976; Swadlow, 1974a). The test for post-collision recovery (Swadlow, 1974a), a corollary of the test for collision of impulses, was also used. Additional confirmation of the antidromic activation of most neurons was provided by examination of refractory periods, and, in many cases, by examination of spike waveform changes following the second of two closely spaced volleys (Bishop et al., 1962; Phillips, 1969; Phillips et al., 1963). Some units could not be tested by collision techniques due to very low rates of spontaneous impulse activity. These units were considered to be antidromically activated if they satisfied two ancillary criteria: (a) a refractory period of 1.2 ms or less and (b) latency variability of 0.1 ms or less to a TS that followed a CS by 8.0 ms (Swadlow et al., 1978).

Antidromic latency was determined by examination of spike waveform and was measured at the first detectable deviation from baseline. The modal value of 10–15 antidromic latency measurements was assigned as the latency for a given neuron so as to eliminate disparate latency measurements which occur during periods of spontaneous impulse activity (see below).

In estimating axon conduction velocity, conduction time was estimated by subtracting 0.2 ms from the antidromic latency in order to compensate for utilization time at the site of stimulation and delay of invasion near the cell body (Takahashi, 1965). The conduction distance for stimulating electrodes located in the midline of the corpus callosum was estimated by measuring the distance from the stimulating electrode to the tip of the microelectrode and subtracting 0.5 mm (to compensate for spread of stimulus current). This value was then multiplied by 1.2 to compensate for the curved path of the callosal axon. The factor of 1.2 was derived from measurements of dissected brains. For stimulating electrodes located in the contralateral hemisphere, 0.5 mm was subtracted from the distance from the recording microelectrode to the stimulating electrode and this value was multiplied by 1.5. The factor of 1.5 was derived from measurements of electrode location in a case in which stimulating electrodes were placed in both the midline of the corpus callosum and the contralateral hemisphere, as well as from gross dissection of unoperated brains. We feel that our estimates of axonal conduction velocity are accurate ± 20%.

Results

Sixty one callosal efferent neurons were antidromically activated via contralateral cortical and/or midline callosal stimulating electrodes. Antidromic latency following stimulation of the contralateral hemisphere (N = 51) ranged from 2.6–18.0 ms (median = 7.0 ms) while antidromic latency following stimulation of the midline corpus callosum (N = 19) ranged from 1.2–5.9 ms (median = 3.2 ms). Figure 2A presents a histogram of estimated conduction velocity based on contralateral cortical stimulation while Fig. 2B shows estimated velocity based on stimulation of the corpus callosum near the midline.

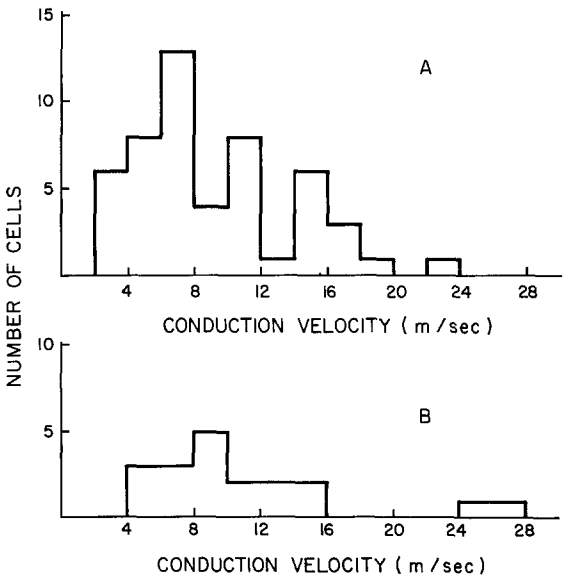

Fig. 2. Histogram of estimated conduction velocity following stimulation of the contralateral hemisphere (**A**) and midline corpus callosum (**B**)

Since changes in axonal geometry (and therefore conduction velocity) probably occur near the callosal terminals, estimates of conduction velocity based on contralateral cortical stimulation might best be considered as the mean conduction velocity along the length of the callosal axon.

The Refractory Period

The refractory period of each neuron was obtained by presenting two pulses (each at either 2X threshold or, in a few cases, 1.5 threshold) at decreasing intervals. The refractory period was defined as the shortest interval which yielded a response to the second stimulus for three consecutive stimulus presentations. Refractory periods to contralateral cortical stimulation ranged from 0.55–1.3 ms and were generally shorter for faster conducting axons. This relationship was also evident following midline callosal stimulation.

The Supernormal Period

All but one of the callosal efferent neurons studied showed a supernormal period, manifested by a decrease in antidromic latency and threshold to a TS which followed an antidromic CS at appropriate intervals. Figure 3A–B each present superimpositions of three oscilloscope tracings from a callosal efferent neuron which demonstrated collision of impulses, postcollision recovery and a

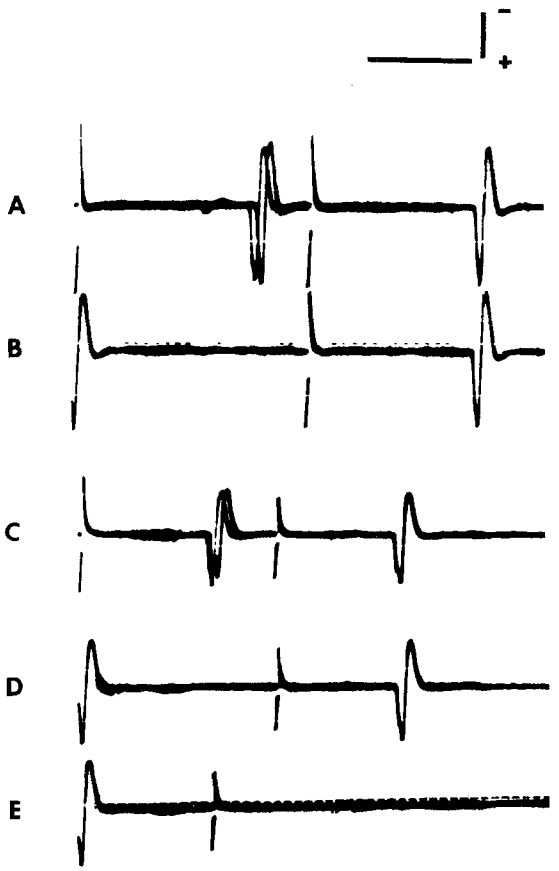

Fig. 3. A antidromic TS is preceded by an antidromic CS at an interval of 11.1 ms. The CS results in a response at a latency of 8.3 to 8.65 ms while the TS results in a spike at a latency of 8.0 ms. Under these conditions the threshold to the TS is less than when no CS is presented. The intensity of the TS was therefore similarly reduced. **B** decrease in latency to a TS is shown to follow a spontaneous spike. **C–D** For another neuron, the same phenomena are shown as were shown in **A** and **B**, respectively. **E** Collision of impulses. No spike results when a TS follows a spontaneous spike at an interval of 6.5 ms. Calibration Bar = 5 msec

refractory period of < 1.0 ms. In Figure 3A, an antidromic TS was preceded by an antidromic CS at an interval of 11.1 ms. Pairs of stimuli (CS and TS) were presented randomly with respect to the spontaneous activity of the cell, at a rate of 1/3 s. While the CS resulted in an antidromic latency of 8.3–8.65 ms, the TS resulted in a response of reduced (8.0 ms) and relatively invariant latency. Threshold to the TS was reduced under these conditions. For this neuron, and for all neurons studied, threshold to the TS was measured at each CS-TS interval and the intensity of the TS was always presented at 1.2 x this threshold intensity. This procedure minimized the possibility of activating the axon at varying distances from the stimulating electrode. Figure 3B shows that the decrease in latency to the TS following a *spontaneous impulse* is similar to that found following an electrically elicited impulse (Fig. 3A). Figure 3C–D shows, for another neuron, the same phenomenon shown in Fig. 3A–B, respectively. Figure 3E shows for the neuron shown in Fig. 3C–D that no spike results when a TS follows a spontaneous spike at an interval of 6.5 ms (due to collision of impulses).

The maximal decrease in antidromic latency to the TS ranged from 1.9–10.3% of the control latency, with one neuron showing no decrease in latency. Figure 4 relates the magnitude of the decrease in latency to the conduction velocity of the callosal axon. Points represent the decrease in latency (%) to a TS presented near the peak of the supernormal period which follows the impulse initiated by the CS. Figure 4A shows neurons activated by stimulation of contralateral prelunate gyrus while Fig. 4B shows neurons activated by stimulation of the corpus callosum near the midline. The magnitude of the decrease in latency was generally greater in the slower conducting axons.

In Fig. 5A–B, the closed circles represent, for two units, the time course of the decrease in latency to a TS presented at various intervals following a single CS. The solid horizontal line represents the control antidromic latency (modal value of 10–15 latency measurements). At CS-TS intervals of 1–2 ms, latency was greater than control values. This is probably due to slowed conduction during the relative refractory period of the axons. At intervals of 6–10 ms antidromic latency reached a low value and slowly increased to reattain control levels at CS-TS intervals of approximately 160 ms and 50 ms in A and B, respectively. Closed circles in C and D represent threshold of the TS for the same units represented in A and B, respectively. Note that variations in threshold closely mirror the variations in antidromic latency.

For several units the effects of increasing the number of conditioning pulses was examined. This resulted in either a decrease, or no change in the magnitude and duration of the supernormal period.

The Subnormal Period

When a CS consisting of several pulses was presented, a subnormal period of increased antidromic latency and threshold followed the initial supernormal period and lasted as long as several seconds. The closed triangles and squares of Fig. 5A–B represent the antidromic latency to a TS presented at various intervals following a CS consisting of 10 or 20 pulses (330/s), respectively. Closed triangles and squares in C and D represent concomitant variations in threshold. Note that the magnitude of the increase in both latency and threshold (in B and D, respectively) is augmented by an increase in the number of conditioning stimuli.

Increases in antidromic latency may last for more than one minute following tetanic stimulation. Figure 6A–D shows four units with control antidromic latencies of 4.4, 5.7, 2.95, and 17.3 ms, respectively. Closed circles to the left of the solid bar represent antidromic latency to a TS presented at 1/3 s. During the period represented by the width of the solid bar (45, 45, 60, and 20 s in A, B, C, and D, respectively) tetanic stimuli were presented at 33 pulses/s (antidromic latency to the tetanic stimuli was not measured). Closed circles to the right of the bar represent antidromic latency to test stimuli presented again at 1/3 s. For the four units, latency increased fom 17 to 27% of control values and returned to control levels in 1–2 min. In Fig. 6A–D, every third TS (triangles) was preceded by a suprathreshold CS (CS-TS = 8 ms). As would be expected, antidromic

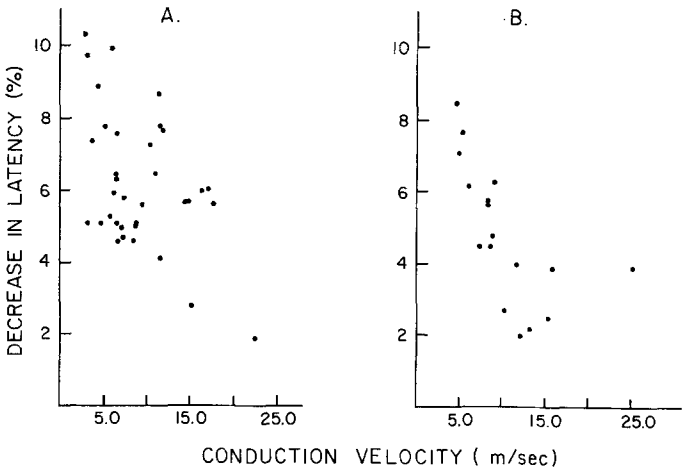

Fig. 4. Relationship of the magnitude (% of control value) of the decrease in latency (near the peak of the supernormal period) to the conduction velocity of the axon following contralateral cortical stimulation (A) and midline callosal stimulation (**B**). Each point represents a single cell

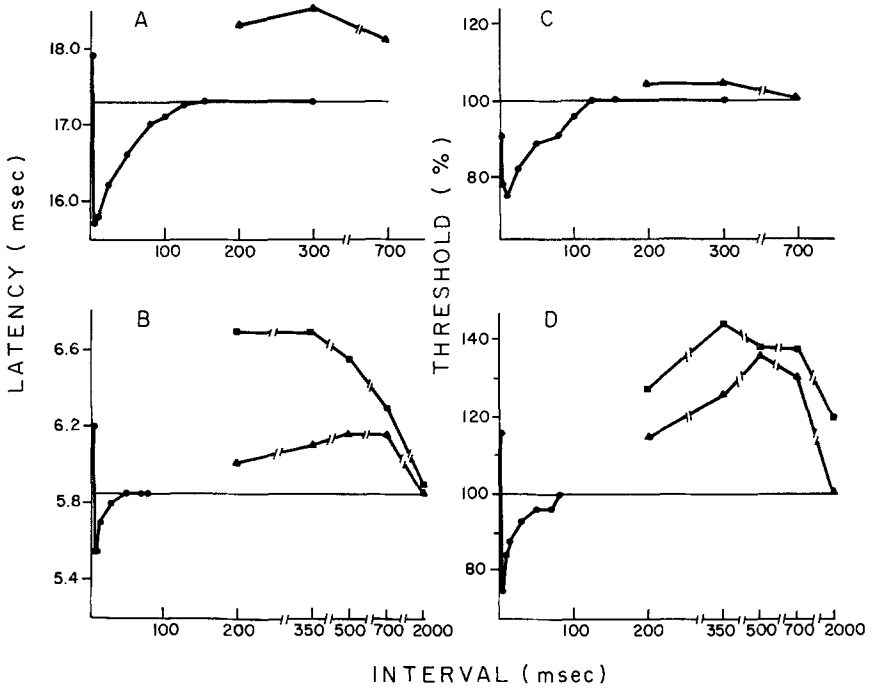

Fig. 5. A–B For 2 units, antidromic latency to a TS which is presented at various intervals following either a single CS (●–●), a train of 10 pulses (▲–▲) or a train of 20 pulses (■–■). **C–D** For the same 2 units presented in A and B respectively variations in threshold of the TS are shown. The horizontal lines represent control latency in **A** and **B** and control threshold in **C** and **D**

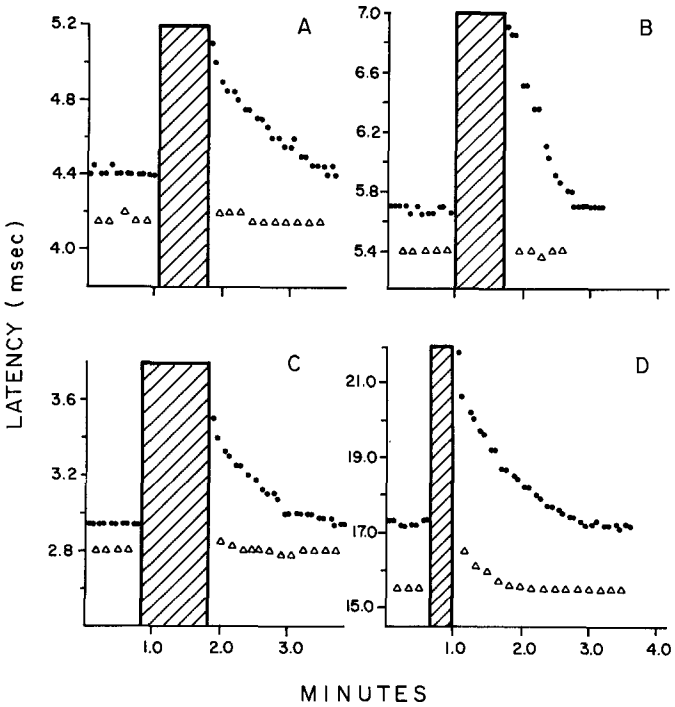

Fig. 6. For 4 units, long-term variations in antidromic latency following tetanic stimulation are shown. For each unit, closed circles to the left of the solid bar represent antidromic latency to a TS presented at $^1/_3$ s. Test stimuli were presented at 33/s during the period represented by the width of the solid vertical bar. To the right of the solid bar, test stimuli were again presented at $^1/_3$ s. For the above units, approximately every third stimulus (represented by triangles) is preceded by a supra-threshold CS (CS-TS interval = 8 ms). Note the minimal variations in antidromic latency under these conditions

latency was reduced under these conditions. Note, however, that for the units shown in Fig. 6A–C following tetanic stimulation, the antidromic latency of the TS following a CS does not appreciably increase (less than 0.1 ms variability). In Fig. 6D, latency to the TS increased somewhat under these conditions, but far less than observed when no CS was presented (closed circles).

As would be predicted from the above considerations, the "control" antidromic latency of a neuron may vary significantly with the rate of stimulus presentation. Figure 7A–B presents, for two units, the antidromic latency to a TS presented at rates of 1/3 s–10/s. Closed circles represent the latency to the TS when presented in the absence of a CS. Antidromic latency to the TS was allowed to stabilize for 2 min at each stimulation rate before obtaining these measurements. In B, open circles represent a repetition of this measure obtained 20–30 min later. Triangles in A and B represent the latency to the TS when preceded by a CS (CS-TS interval = 8 ms). Note that, as in Fig. 6, the variation in antidromic latency to a TS was far less when preceded by a CS than when presented alone.

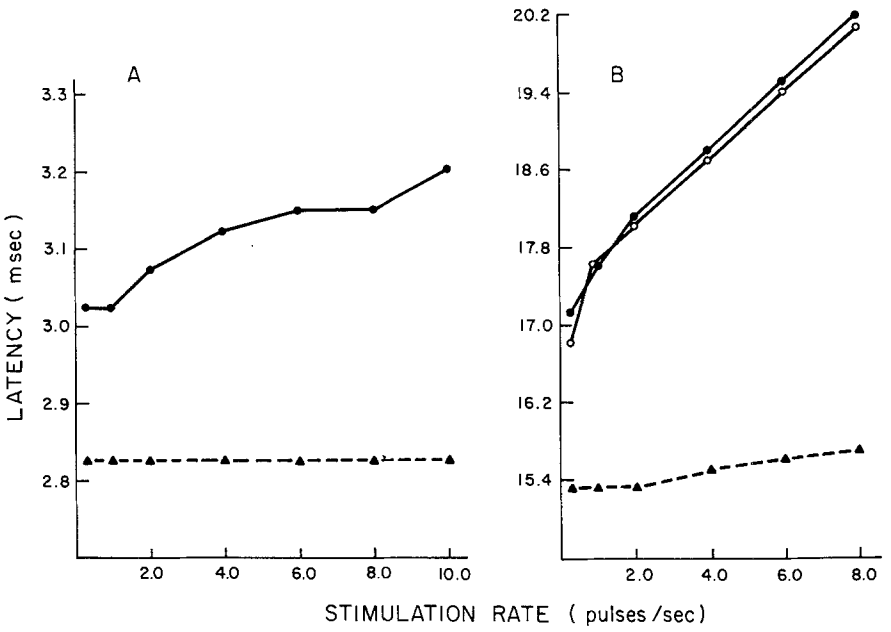

Fig. 7. For 2 units (**A** and **B**), antidromic latency is shown to vary with the rate of stimulus presentations. For each unit, closed circles represent latency to the TS when presented at various frequencies in the absence of a CS. In **B**, the open circles represent a repeat of this measure obtained 20–30 min later. In **A** and **B**, triangles represent latency to the TS when preceded by a CS (CS-TS interval = 8 ms). Note again, as in Fig. 6, the lesser magnitude of the variations in antidromic latency under these conditions

Control Experiments

The finding that antidromic latency is reduced following a spontaneous impulse (see Fig. 3B) as well as following an electrically elicited impulse strongly suggests that the variability in antidromic latency is due to the prior impulse conducted along the axon under study, and not to either ephaptic interactions between axons or some other artifact of the prior electrical stimulation. Further experiments showed that variations in the intensity of a supra-threshold CS have no bearing on the magnitude or time course of the reduction in latency to the TS or to the threshold of the TS, and that when the CS was presented at threshold intensity, a reduction in latency to the TS occurred only when the CS resulted in an antidromic spike.

If activity-dependent variations in antidromic latency are due to variations in conduction velocity distributed along the axon, then the magnitude of the variations observed in a given callosal efferent neuron ought to be dependent on the length of the conduction path. This was found to be the case for each of several neurons which were antidromically activated via both contralateral cortical and midline callosal stimulating electrodes. For each neuron, the decreases in latency to stimulation at each site were approximately proportional to the control latency at that stimulation site. Furthermore, a CS applied to one

stimulation site along the axon always resulted in a decrease in latency to a TS applied at a second point on the axon. The finding that variations in antidromic latency are greater following contralateral cortical stimulation than following midline callosal stimulation, and in fact are approximately proportional to conduction path length indicates that *activity-dependent variations in antidromic latency are due to variations in conduction velocity distributed along the axon.* The finding that antidromic latency is reduced following a spontaneous impulse (see Fig. 3B) and the other control experiments reported above show that these *variations in conduction velocity are due to the prior impulse conduction along the callosal axon.*

Discussion

A series of altered conduction properties (refractory period → supernormal period → subnormal period) follows impulse activity along callosal axons in the monkey. A supernormal period of increased axonal conduction velocity and excitability follows the refractory period and may last for more than 150 ms. The maximal magnitude of the supernormal period usually occurs at 6–12 ms following a prior impulse and is negatively correlated with axonal conduction velocity.

Following several prior impulses, the supernormal period is followed by a subnormal period of decreased conduction velocity and excitability. While the supernormal period is not augmented by an increase in the number of prior impulses, both the magnitude and duration of the subnormal period are augmented by an increase in the number of prior impulses. The subnormal period may last for more than 1 min.

In addition to visual callosal axons of monkey and rabbit, the supernormal period has been described in somatosensory callosal axons of the rabbit (Swadlow and Waxman, 1976), parallel fibers of the cat cerebellum (Gardner-Medwin, 1972) and in caudato-fugal axons of the cat (Kocsis et al., 1977). Furthermore, we have recently observed such variations in conduction velocity and excitability in axons projecting from visual area I to ipsilateral visual area II of the rabbit. In the peripheral nervous system, activity-dependent variations in conduction properties have been documented in sciatic nerve of frog (Bullock, 1951; Lass and Abeles, 1975; Newman and Raymond, 1971; Raymond and Lettvin, 1978), olfactory nerve of tortoise (Bliss and Rosenberg, 1974) and radial and ulnar nerve of humans (Bergmans, 1973; Gilliatt and Willison, 1963). Activity-dependent variations in conduction properties may therefore represent a rather general feature of axonal physiology. This view is supported by ultrastructural studies which show that the morphology of both myelinated and non-myelinated callosal axons of rabbit (Waxman and Swadlow, 1976) and monkey (in prep.) are similar to those of axons found in other regions of the central nervous system (see, e.g., Hirano and Dembitzer, 1967).

It is interesting that while interhemispheric conduction distances are 2–3 times greater in the macaque than in the rabbit, the antidromic interhemispheric latencies of callosal efferent neurons are considerably shorter in the monkey

(range = 2.6–18.0 ms, median = 7.0 ms) than those found in a comparable study in the rabbit (range = 2.4–39.8 ms, median = 17.0 ms, Swadlow, 1974a). The conduction velocity of callosal axons in rabbit range from 0.3–12.9 ms (Swadlow, 1974a, Swadlow and Waxman, 1976) and electron microscopy reveals that the splenium of the corpus callosum of the rabbit contains both myelinated (55%) and non-myelinated (45%) axons (Waxman and Swadlow, 1976). Thus, in the rabbit, both physiological and anatomical results are consistent with the view that the visual callosal efferent system consists of both myelinated and non-myelinated axons. In contrast, in the monkey, though electron microscopy (in prep.) reveals both myelinated and non-myelinated axons in the splenium, the axonal conduction velocities (see Fig. 2) suggest that all axons studied in the present experiment were myelinated. Although the results obtained from visual areas I and II of the rabbit are not strictly comparable with those obtained from prelunate gyrus of monkey, the above considerations suggest the possibility of a fundamental species difference in the composition of the visual component of the corpus callosum.

A crucial question concerns the functional significance, with respect to interhemispheric communication, of the observed history-dependent variations in conduction properties of callosal axons. One distinct possibility is that they simply introduce noise into the system and thereby result in limitations in the information carrying capacity of the smaller callosal axons, due e.g., to the ambiguity in the arrival time of the presynaptic impulse at the axon terminal. Alternatively, the historical information inherent in the instantaneous conduction velocity and excitability of callosal axons might somehow be utilized by the callosal system. In some callosal axons of macaque, history-dependent variations in interhemispheric conduction time may exceed 6 ms (see Fig. 6). Such variations might be detected by a post-synaptic element sensitive to small fluctuations in the arrival time of the presynaptic impulse, perhaps by comparing it to the relatively invariant input from a coupled, faster conducting axon. Another possibility has been put forth by Chung et al. (1970), who suggested that small history-dependent variations in axonal excitability might be highly significant at axonal branch points, which may be regions of low safety factor where failure of conduction is likely to occur. Variations in axonal excitability could serve to block an impulse, or to route an impulse down one branch or another, depending on the history of impulse activity along the axon. This would result in a transformation of the temporal distribution of impulses to a spatial distribution of impulses along the axonal tree. Such history dependent routing of impulses has, in fact, been observed in spiny lobster (Grossman et al., 1973), and activity-dependent conduction block has been shown to account for some adaptation phenomena in sensory fibers of the leech (Van Essen, 1973; Yau, 1976). Finally, variations in excitability, if present at axon terminals, may modulate the dynamics of transmitter release (Hubbard and Willis, 1962, 1968; Takeuchi and Takeuchi, 1962). Whether the historical information inherent in axonal conduction velocity and excitability of callosal axons is utilized by the callosal system is a question whose answer awaits a more complete understanding of the detailed nature of neural codes and of the mechanisms by which post-synaptic elements receive and interpret afferent trains of impulses.

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