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Inferior olive excitability after high frequency climbing fibre activation in the cat

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Summary. 1. Climbing fibre responses (CFRs) were evoked by limb nerve stimulation and recorded from the cerebellar surface in barbiturate anaesthetized cats. Climbing fibres were activated at frequencies of usually 2.5–7.5 Hz for periods of 15–30 s, after which the stimulation frequency was reduced to below 1 Hz. 2. The high-frequency stimulation induced a strong depression of CFR-amplitude, lasting up to 60 s. The magnitude of this depression was dependent on both the frequency and the duration of the high-frequency stimulation. 3. The depression occurred in the c1, c2 and c3 zones of the pars intermedia and in the x zone in the vermis but not in the b zone in the vermis. 4. Recordings of olivary reflex responses demonstrated that the depression occurred in the inferior olive. 5. It is suggested that the inhibition of the inferior olive occurs because the high-frequency stimulation leads to a disinhibition of neurones in the interpositus nucleus which inhibit the olivary neurones.

Key words: Cerebellum – Inferior olive – Interpositoolivary pathway

Introduction

It is well known that peripherally evoked climbing fibre responses (CFRs) recorded as field potentials from the cerebellar surface are characterized by variable amplitudes, which presumably reflect variations in the excitability of the inferior olive (Ito et al. 1966; Miller and Oscarsson 1970; Armstrong et al. 1973). When climbing fibres are activated at a low frequency of approximately 1 Hz, periods of high responsiveness alternate in a slow and irregular manner with periods during which CFRs are depressed. Such periods may last from a few seconds to half a minute or more. Oscarsson and Sjölund (1977) showed that the fluctuations in different cerebellar zones were independent and also that the effects of anaesthetics on these fluctuations differed between zones.

It has been shown (Carrea et al. 1954; Miller and Oscarsson 1970) that, when climbing fibres are continuously activated at high frequencies (> 3 Hz), amplitude variations occur in a more regular "on-off" manner, i.e. alternations between periods of maximal CFR amplitude and periods during which CFRs were almost completely suppressed. The durations of the "on" periods were shortened when stimulation frequency was increased. It is not known if these cyclical fluctuations are related to the variations observed at lower stimulation frequencies.

With the exception of these studies, very little research has been directed at systematic investigations of the modulation of inferior olive excitability. The findings of Miller and Oscarsson (1970) suggest that high frequency activation of the climbing fibre system may interfere with some mechanism which regulates the excitability of the inferior olive. The present investigation was undertaken to study systematically the effects of various stimulation frequencies on olivary transmission.

Methods

The experiments were performed on 22 cats under deep pentobarbitone anaesthesia (initial dose 40 mg/kg i.p.; supplementary doses of 5 mg i.v. as required). The level of anaesthesia was adjusted such that the pupils were maximally constricted. The animals were paralyzed with gallamine triethiodide and artificially ventilated. The end-expiratory CO_2 concentration, arterial blood pressure and rectal temperature were continuously monitored and kept within physiological limits.

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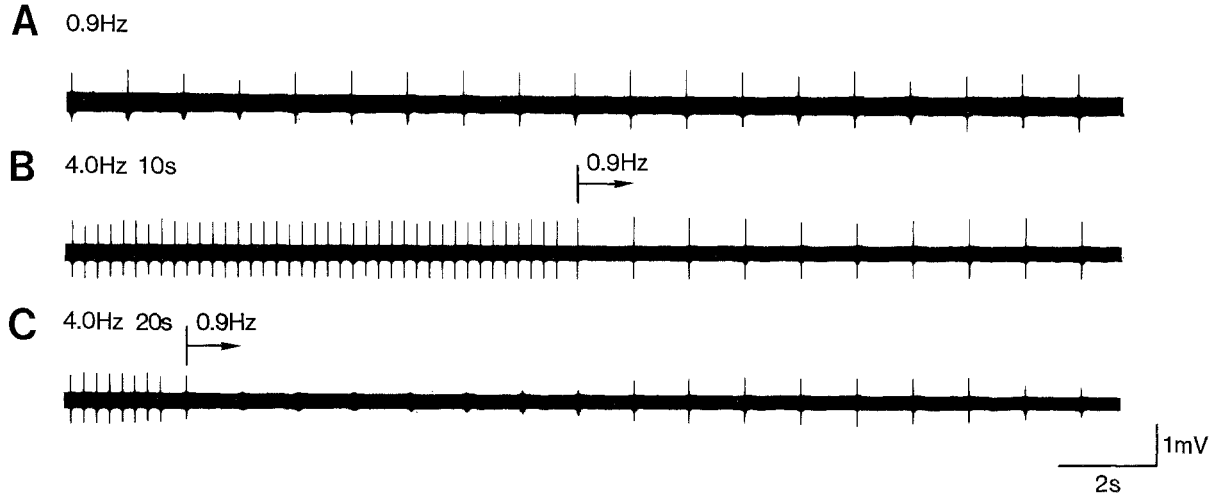

Fig. 1A–C. Records of climbing fibre responses (CFRs) in the c3 zone of the cerebellar cortex on stimulation of ipsilateral superficial radial nerve. **A** Stimulation at 0.9 Hz. **B** Conditioning stimulation at 4.0 Hz for 10 s followed by 0.9 Hz test stimulation. Arrow indicates time at which stimulation frequency was changed. Record shows most of the conditioning period and part of the test period. **C** Conditioning stimulation at 4.0 Hz for 20 s. Record only shows the last seconds of the conditioning period

The left anterior lobe of the cerebellum was exposed and the cortex was covered with warm mineral oil. The left common or superficial radial nerve and the left sciatic nerve were dissected and mounted for stimulation.

Climbing fibre responses (CFRs) were evoked by bipolar stimulation of limb nerves, with square pulses of 100 μs duration. Stimulation strength was slightly above that which evoked a maximal climbing fibre response. Climbing fibres were also activated by monopolar stimulation of the cerebellar surface in order to evoke an olivary reflex response (Armstrong et al. 1973). Recordings from the cerebellar surface were made with silver ball electrodes. An indifferent electrode was placed in the neck musculature. All recordings were made in the anterior lobe, in the x, b, c1, c2 and c3 zones (for references see Ito 1984).

Results

Effect of stimulation frequency on CFR amplitude

In all experiments, a peripheral nerve was continuously stimulated at low frequencies of approximately 1 Hz for 1–2 min and field responses were recorded from the cerebellar surface. This stimulation evoked CFRs with stable amplitudes. A typical example is illustrated in Fig. 1A, which shows CFRs in the c3 zone during continuous stimulation of the ipsilateral superficial radial nerve (iSR) at 0.9 Hz. The standard deviation of the amplitude was less than 5% of the mean amplitude for a period of 2 min. There were thus no indications that this stimulation frequency influenced the CFR amplitude. When the stimulation frequency was increased above 2 Hz, cyclical variations occurred and became progressively more pronounced (cf. Miller and Oscarsson 1970).

All the tests described below started with a period of 5–30 s 'high-frequency' conditioning stimulation at frequencies between 2.5 and 7.5 Hz. The duration of the conditioning period was always kept short enough that the above-mentioned cyclical variations did not occur. The effect of this stimulation was then studied by examining CFR amplitude during a period of 'low-frequency' or test stimulation (0.5–0.9 Hz), which usually lasted for approximately 1 min. These test frequencies were chosen because they did not seem to influence the amplitude of CFRs (cf. Fig. 1A).

Figure 1B shows part of a recording from the same experiment as Fig. 1A. Conditioning stimulation at 4.0 Hz was applied for 10 s and was followed by test stimulation at 0.9 Hz. The record only shows the last part of the conditioning period and the beginning of the test period (the complete time course is shown as a curve in Fig. 2A). In this case, the conditioning stimulation resulted in a small increase in the CFR amplitude. When the duration of the conditioning stimulation was increased to 20 s, the amplitudes of the test CFRs were decreased (Fig. 1C and 2C).

Figure 2A–C illustrates results from the same experiment as in Fig. 1. CFR amplitude is plotted against time. The diagrams show the CFR amplitudes during 4.0 Hz conditioning stimulation of 10, 15 and 20 s, respectively, and during the following 0.9 Hz test stimulation. The interrupted line indicates the average CFR amplitude of 0.9 Hz control stimulation shown in Fig. 1A. The curve in Fig. 2A corresponds to the record in Fig. 1B (the line indicates the

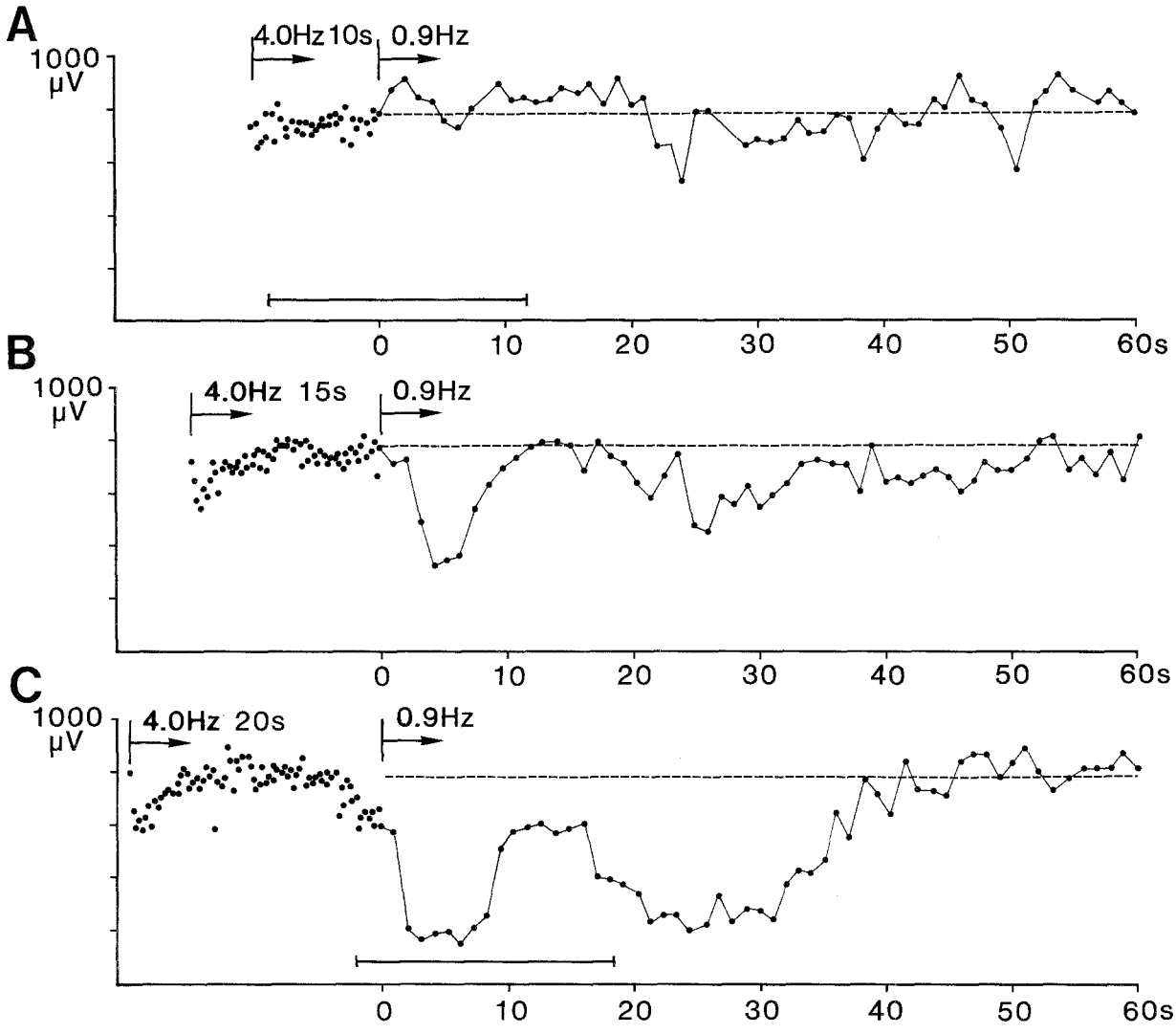

Fig. 2A–C. Time course of depression after high-frequency conditioning stimulation with conditioning period of different durations. Same experiment as Fig. 1. **A** Stimulation of superficial radial nerve at 4.0 Hz for 10 s followed by 0.9 Hz test stimulation for 60 s. Each dot represents a CFR. Interrupted line indicates average CFR amplitude during control stimulation at 0.9 Hz. Arrows show start of stimulation at indicated frequency. Record in Fig. 1B corresponds to the period indicated by line above the time scale. **B** Duration of conditioning period increased to 15 s. **C** Duration of conditioning period increased to 20 s. Record corresponding to the period indicated by the line above the time scale is shown in Fig. 1C

segment shown in 1B). With a 10 s conditioning stimulation, there was a small increase in the CFR amplitude, which lasted for about 20 s. In this experiment, conditioning stimulation for 7–10 s regularly resulted in a small but consistent increase in CFR amplitude.

When the duration of the conditioning stimulation was increased to 15 s or 20 s, as shown in Figs. 1C and 2B–C, the CFR amplitudes were markedly depressed during the test stimulation. Two interesting features of this depression are illustrated in Fig. 2. Firstly, the CFR amplitude remained large throughout the conditioning stimulation and was not depressed until the stimulation frequency was reduced.

Secondly, the time course of the CFR depression often had two components. In Fig. 2B, it can be seen that, during test stimulation, the CFR amplitude was initially depressed for nearly 10 s and that the response amplitude returned to pre-conditioning levels before a later, small depression lasting for an additional 30 s occurred. In Fig. 2C (corresponding to Fig. 1C), where conditioning stimulation was applied for 20 s, the depression was more pronounced and the recovery at approximately 10 s was not complete.

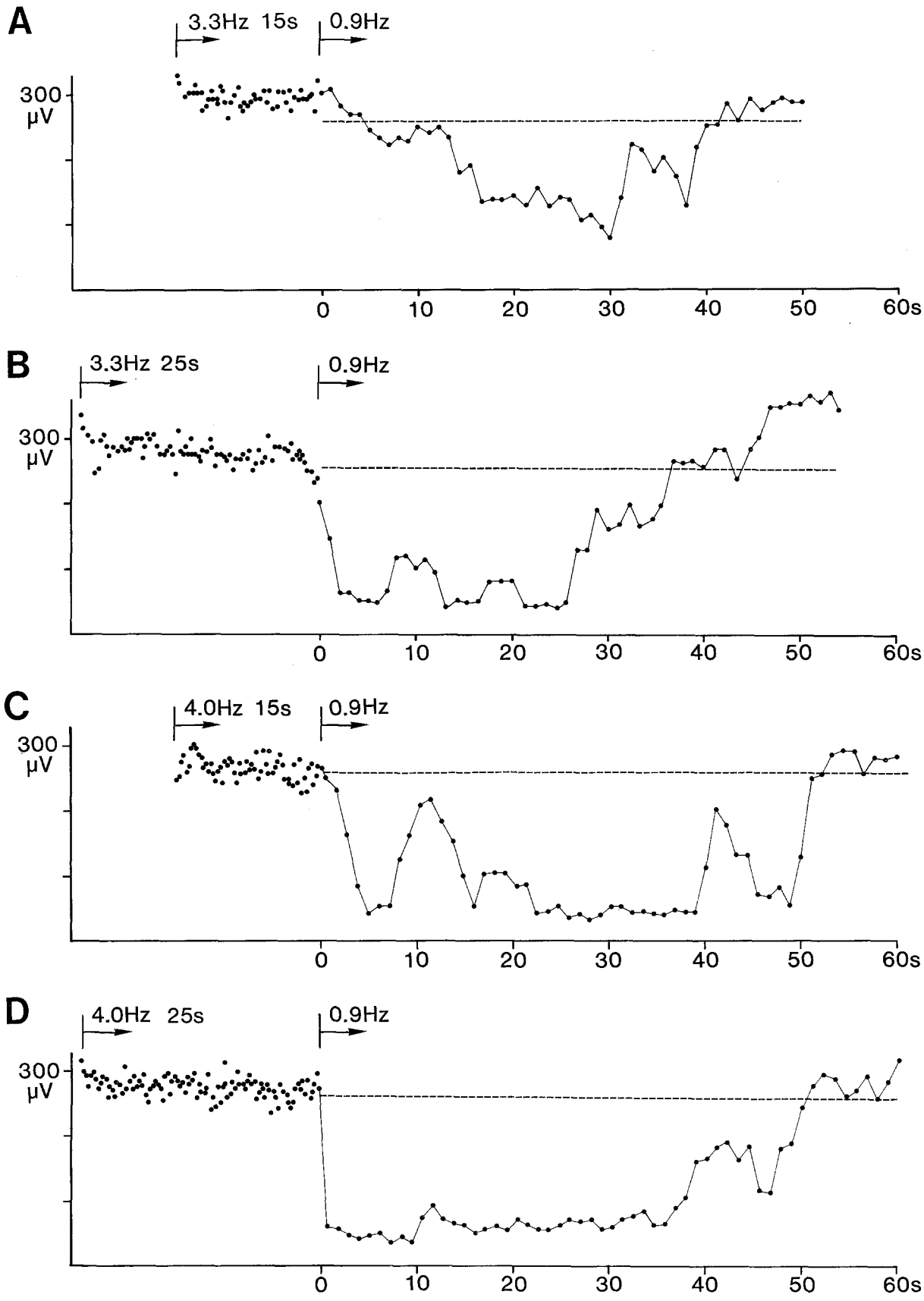

Fig. 3A–D. Effect of varying frequency and duration of the conditioning stimulation. All curves are from the same experiment. CFRs evoked by stimulation of the ipsilateral sciatic nerve and recorded in the c1 zone. Conventions as in Fig. 2. **A, B** Conditioning stimulation at 3.3 Hz, 15 and 25 s, respectively. **C, D** Conditioning stimulation at 4.0 Hz, 15 and 25 s

The curves in Fig. 2 suggest that the effectiveness of high frequency conditioning stimulation is dependent on the duration of the conditioning stimulation. The effects of both the duration and the frequency of the conditioning stimulation were studied systematically in a number of experiments, one of which is illustrated in Fig. 3. CFRs were evoked by stimulation of the ipsilateral sciatic nerve and recorded in the c1 zone. Figure 3A shows the CFR amplitude during conditioning stimulation at 3.3 Hz for 15 s and test stimulation at 0.9 Hz. The depression consisted mainly of the late component. In Fig. 3B, 3.3 Hz stimulation was applied for 25 s and the CFR amplitude was more strongly depressed. The early component was well developed and the recovery at approximately 10 s was small. In the same experiment, 4.0 Hz stimulation was applied for 15 and 25 s (Fig. 3C, D). Once again, the longer conditioning stimulation produced a more pronounced depression. A comparison of Fig. 3A with 3C, B with D reveals that increasing the frequency of the conditioning stimulation also increases the degree of depression.

As shown in Figs. 2 and 3, when the frequency and duration of the conditioning stimulation were just above threshold for producing a depression, one of the components was dominant. With an increase in frequency and/or duration, the other component of the depression often developed. With a further increase of either parameter, the magnitude of the depression was usually increased while any effect on the duration was generally small. The depression in Fig. 3D, for instance, lasted only about 10 s longer than in 3A.

An interesting feature of the time course of the depression was the appearance of brief recoveries in the CFR amplitude. A recovery around 10 s after the termination of the conditioning period was nearly always present, as seen in Figs. 2B, C and 3A–D, although it was sometimes quite small, as in Fig. 3D. Sometimes, additional recoveries followed by renewed depressions occurred later. Examples are seen in Fig. 3A, C, D. In a few cases, up to four or five oscillations could occur during the depression period.

Conditioning stimulation applied for periods shorter than 10 s either had no effect on CFRs or was, in some experiments, followed by small increases in the CFR amplitude (cf. Figs. 1B and 2A).

Generally, the frequency of the conditioning stimulation had to be at least 2 Hz in order to depress the CFR amplitude. With such low frequencies, the duration of the stimulation had to be at least 45 s. When the stimulation frequency was increased to 4 Hz, a conditioning period of 10–15 s was usually sufficient. These parameters, however, varied between cats and were also influenced by the level of anaesthesia.

It was a general finding that the CFR amplitude remained high throughout the conditioning period and decreased when the conditioning stimulation was terminated. This can be seen, for instance, in Fig. 3C, D. After 15 s of 4.0 Hz stimulation, CFR depression started immediately when the stimulation frequency was reduced to 0.9 Hz (3C), but when stimulation was continued for 25 s (3D), the CFR amplitude remained stable throughout the conditioning period. There were two exceptions to this rule. Firstly, as already mentioned, the later component of the depression sometimes appeared without a preceding early component (Fig. 3A), but this was not very frequent. Secondly, the ability of the CFRs to follow high-frequency stimulation varied considerably. The CFRs would never follow stimulation at frequencies above 2–3 Hz indefinitely, but when the depression occurred varied between cats and with the level of anaesthesia. In a few cases, CFRs followed frequencies of 4–5 Hz for up to 1 min with only small amplitude variations, whereas in others the amplitude would decrease after 15–30 s. An example is shown in Fig. 2C, where the CFRs began to decrease in amplitude just before the stimulation frequency was reduced.

Localization of inhibitory effect

In order to determine where the inhibition leading to the amplitude depression occurs, climbing fibres were antidromically activated by cathodal stimulation of the cerebellar surface. Some of the olivary axons innervating the c3 zone have collaterals in the c1 zone (Armstrong et al. 1973; Ekerot and Larson 1982). In the experiment illustrated in Fig. 4, monopolar cathodal stimulation, which is known to activate climbing fibres with a lower threshold than anodal stimulation (Armstrong et al. 1973), was applied to the c1 zone while evoked responses were recorded in the c3 zone. As seen in the control records in Fig. 4, the stimulation resulted in two responses. The first, with a latency of 4 ms, was due to an axon reflex in the climbing fibres. The second response, with a latency of 8–9 ms, was an olivary reflex due to antidromic invasion of cells in the inferior olive and spread of depolarization through electrical synapses (Llinás et al. 1974). The figure shows 3–4 superimposed records taken before a conditioning stimulation (Control), at around 20 and 30 s during conditioning and at 10 s intervals during

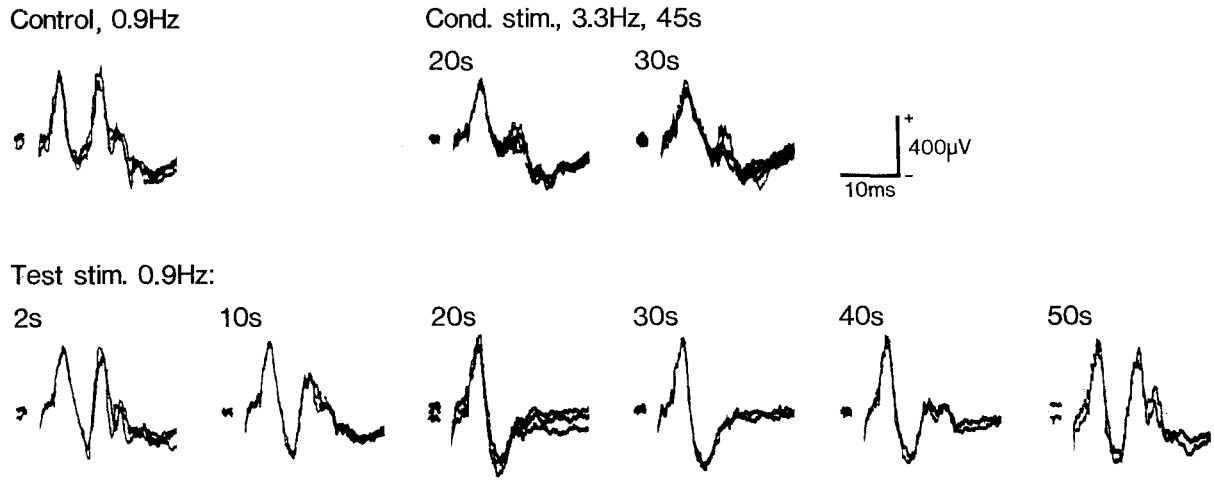

Fig. 4. Effect of high-frequency conditioning stimulation of climbing fibre collaterals. CFRs evoked in c3 zone by stimulation of c1 zone. Records are shown of control responses, responses from 20 s and 30 s after beginning of conditioning stimulation and at 10 s intervals during test stimulation

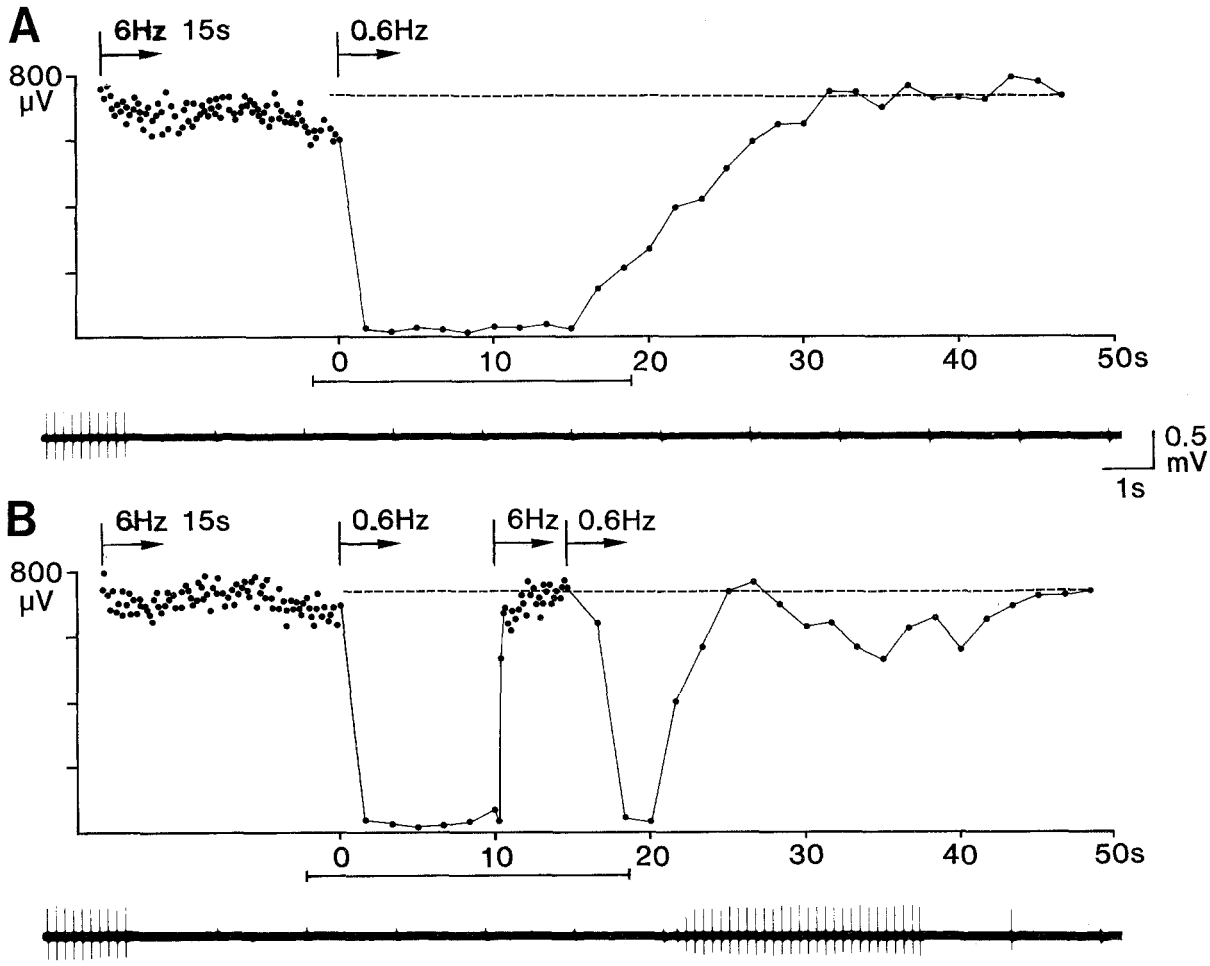

Fig. 5A, B. Disinhibition of CFR amplitudes with high-frequency stimulation during CFR depression. Stimulation of the ipsilateral superficial radial nerve. Recording from c3 zone. **A** Conditioning stimulation at 6.0 Hz for 15 s followed by test stimulation at 0.6 Hz. Record corresponds to period indicated by the line under the time scale. **B** Conditioning stimulation identical to that shown in **A**, but 6.0 Hz stimulation resumed for 5 s after 10 s of test stimulation

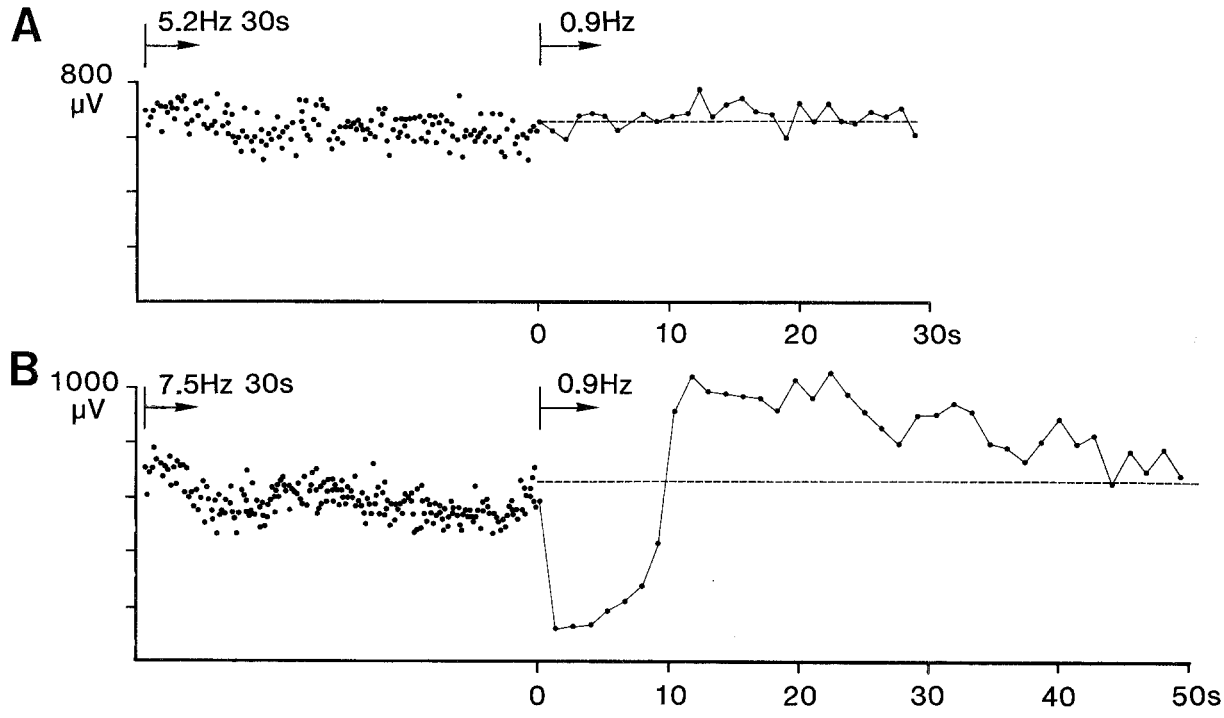

Fig. 6A, B. Effect of high-frequency conditioning stimulation in the b zone. Stimulation of the ipsilateral sciatic nerve. Recordings are from the same experiment as Fig. 3. **A** Conditioning stimulation at 5.2 Hz for 30 s. **B** Conditioning stimulation at 7.5 Hz for 30 s

test stimulation. Conditioning stimulation at 3.3 Hz was applied for 45 s. During this stimulation, the amplitude of the olivary reflex response was reduced. This was presumably due to the fact that the antidromic impulses in the olivo-cerebellar fibres provide a relatively weak activation of the olivary cells. Hence, fewer cells were able to follow the higher frequency. There was also a change in the axon reflex during conditioning stimulation. The responses were slightly depressed in amplitude and more dispersed temporally. Since the area under the response remained about the same, this presumably reflects an increased variation in latency and not in response probability.

During test stimulation at 0.9 Hz, the olivary reflex response amplitude first returned to control level. After approximately 10 s, it started to decline and was completely depressed between 20 and 30 s. Recovery began at about 40 s and was complete at 50 s. The time course of the depression was similar to that shown in Fig. 3A, i.e. with a delay in the onset of the depression of about 10 s. In contrast, the axon reflex responses were unaffected. The latter observation excludes the possibility that the depression of CFRs after conditioning stimulation is mediated by synaptic events in the cerebellar cortex. Together with the fact that the olivary reflex responses were inhibited in the same way as CFRs evoked by nerve stimulation, this shows that the depression must be due to inhibition of the olivary neurones.

Disinhibition during high frequency CF stimulation

The observation that the above-mentioned depression did not start until the stimulation frequency was reduced (cf.Fig. 3), suggests that high frequency stimulation may counteract the inhibitory mechanism. To test whether high frequency stimulation could enhance the amplitude of depressed CFRs, high frequency stimulation was resumed during the period of depression. In the experiment illustrated in Fig. 5, 6.0 Hz stimulation was applied for 15 s and then followed by 0.6 Hz test stimulation. In A, it can be seen that the CFR amplitude was strongly depressed for about 15 s and then gradually recovered, reaching control level after about 30 s. In B, the frequency was increased to 6.0 Hz after 10 s of 0.6 Hz stimulation. This resulted in a rapid and complete recovery of the CFR amplitude. 0.6 Hz stimulation was then resumed and the CFR amplitude decreased again. Such tests were performed in 3 cats and similar results were obtained in all of them.

Comparisons between sagittal zones

High frequency conditioning was tested in the c1, c2 and c3 zones with similar results in all zones. The c2

zone was difficult to study systematically, however, since CFRs did not follow high frequencies in all experiments. Tests were also made in different parts of these zones with both forelimb and hindlimb nerve stimulation, again with the same results. Tests in the x zone revealed a similar pattern, although the depression of the CFR amplitude was weaker here than in the other zones.

In the b zone, a different pattern appeared. When stimulation parameters were employed which produced strong depression in the c1 or c3 zones, no depression occurred in the b zone. Figure 6 shows results from the b zone from the same experiment as that illustrated in Fig. 3. Even when conditioning stimulation of 5.2 Hz was applied for 30 s, no depression of the CFR amplitude was observed (Fig. 6A). This should be compared to the very strong depression observed in the c1 zone after conditioning stimulation at a lower frequency (3.3–4.0 Hz) and of shorter duration (15–25 s) (see Fig. 3). However, in 3 cats in which the stimulation frequency was increased to 7.5 Hz, a short-lasting (5–10 s) and relatively weak depression was observed. Figure 6B illustrates the strongest depression observed in the b zone. It should be pointed out that similar short-lasting periods of depression also occurred during prolonged continuous high frequency stimulation. Furthermore, high frequency conditioning stimulation at about 7 Hz was usually followed by a period of strongly enhanced CFR amplitude which lasted up to 1 min. This is also evident in Fig. 6B.

Discussion

The major result of this investigation of surface recorded CFRs is that activation of climbing fibres at frequencies of at least 2–3 Hz for a period of at least 15 s leads to a depression of the CFR amplitude lasting up to about 60 s.

When climbing fibres were activated antidromically (cf. Fig. 4), the olivary reflex responses was inhibited, while the axon reflex response remained unaffected. Thus, the depression of the CFR amplitude cannot be caused by a change in the responsiveness of Purkinje cells. Since the olivary reflex response was inhibited, it can be concluded that at least part of the inhibition occurred in the olivary cells.

It is difficult to explain these findings in terms of any known mechanisms of the inferior olive. Previously demonstrated inhibitory influences on the inferior olive, such as recurrent inhibition (Armstrong and Harvey 1966; Armstrong et al. 1968) and mutual inhibition between olivary cells (Andersson 1984) are effective for only about 100 ms and are clearly insufficient to explain the effects described here.

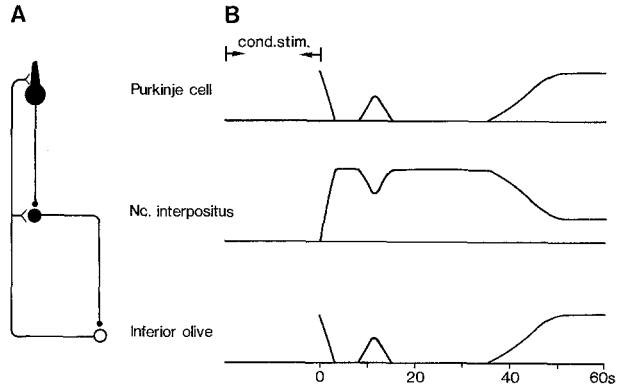

Fig. 7A, B. Hypothetical explanation of the effects of high-frequency stimulation. **A** Postulated circuitry with inhibitory neurones in the interpositus nucleus projecting to the inferior olive. **B** Postulated changes after high-frequency conditioning stimulation in the activity of Purkinje cells, interpositus neurones and inferior olivary neurones

A tentative hypothesis to explain these findings is illustrated in Fig. 7, where it is suggested that high frequency conditioning stimulation depresses simple spike firing of Purkinje cells, which leads to a disinhibition of interpositus neurones and, via an inhibitory nucleo-olivary pathway, to inhibition of the inferior olive. This hypothesis is based on two findings reported in the recent literature.

Firstly, Rawson and Tilokskulchai (1981) have shown that high frequency climbing fibre stimulation (4–10 Hz) leads to a strong suppression of simple spike firing in the Purkinje cells. The duration of this suppression could, to some extent, be graded with stimulation frequency and duration, but usually it lasted up to 20–40 s. Although the anaesthesia and the stimulation parameters were somewhat different in the present experiments, the conditioning stimulation presumably led to a suppression of SS firing for a similar period of time.

Secondly, recent evidence suggests that the interpositus nucleus may inhibit the inferior olive. It is well established that the interpositus nucleus, which is the target of the Purkinje cells of the pars intermedia, projects to the inferior olive (Graybiel et al.1973; Tolbert et al. 1974). Nelson et al. (1984) have reported that cells in the interpositus nucleus, retrogradely labelled after HRP injections in the inferior olive, are GAD-positive and thus, presumably, GABA-ergic and inhibitory.

Figure 7A shows the neuronal circuitry required by the hypothesis (inhibitory neurones shown in black). Climbing fibres project to the Purkinje cells

and send collaterals to the interpositus nucleus. Purkinje cells project to the cells of the interpositus nucleus which in turn project to the inferior olive. The internal circuitry of the interpositus nucleus is not well known, but some evidence suggests that both climbing fibre collaterals and Purkinje cells terminate on the olive-projecting neurones (McCrea et al. 1978).

The schematic diagram in Fig. 7B shows the hypothesized changes in the activity of Purkinje cells, interpositus neurones and inferior olivary neurones after high frequency conditioning stimulation. According to the hypothesis, the conditioning stimulation will suppress the Purkinje cell SS discharge for about 50 s. Correspondingly, nuclear cells will increase their firing for a similar period, while the excitability of the inferior olive will be depressed.

This hypothesis explains the difference observed between the b zone and the pars intermedia (Fig. 6). The Purkinje cells of the b zone project to the lateral vestibular nucleus (Groenewegen and Voogd 1977; Andersson and Oscarsson 1978), but there does not seem to be any projection from this nucleus to the inferior olive corresponding to the interposito-olivary projection. Thus, one would not expect high frequency conditioning stimulation to have the same effects in the b zone. This leaves unexplained, however, the very brief depression of CFRs sometimes observed in the b zone after more extreme high frequency conditioning stimulation.

In order to explain other features of the post-conditioning effects, some assumptions must be made concerning the effects of high-frequency stimulation on Purkinje cells and interpositus neurones. For instance, it was a regular finding that the CFR amplitude after conditioning stimulation recovered briefly after about 10 s. If this recovery was due to decreased activity in the nucleo-olivary pathway, the hypothesis predicts that a similar recovery occurs in Purkinje cell SS discharge.

In order to explain the graded effects of varying duration and frequency of the conditioning stimulation, it must be assumed that the Purkinje cells behave in a corresponding manner. Thus, it is predicted that conditioning stimulation with lower frequencies or shorter conditioning periods will lead to a more short-lasting and smaller suppression of Purkinje cell SS firing and increase in nuclear cell firing, corresponding to the different effects of conditioning illustrated in Figs. 2 and 3. The hypothesis also predicts that very short-lasting conditioning stimulation (< 10 s) can, at least in some preparations, lead to an increased SS firing and decreased nuclear cell firing in accordance with the effects illustrated in Figs. 1A and 2A.

A more difficult task is to explain the fact that the depression of the CFR amplitude usually occurred only when the high frequency conditioning stimulation was terminated. In the experiment illustrated in Fig. 3, for instance, the depression started when the conditioning stimulation was terminated after 15 s, but when this stimulation was continued for 25 s, the CFRs were large throughout the conditioning period. A possible explanation is that the output generated in the Purkinje cells in response to climbing fibre activation, the complex spikes, substituted for the simple spikes during high-frequency stimulation. Thus, although simple spikes may have disappeared before the end of the conditioning stimulation, the complex spike discharge in the Purkinje cells would keep the nuclear cells depressed. When the stimulation frequency was lowered to below 1 Hz, the CFRs would no longer be a sufficient inhibitory input to the nuclear cells, which would then increase their activity and inhibit the inferior olive.

The suggestion that high-frequency complex spike discharge in the Purkine cells could depress the nuclear cells may explain the observation that high frequency climbing fibre stimulation during a period of depression caused a recovery of CFR amplitude (Fig. 5). One should not expect this effect to be immediate, because, when CFRs are strongly depressed, very little transmission through the inferior olive can occur and thus stimulation generates very little output from the Purkinje cells. However, once some activity is generated, the postulated circuit will function as a positive feedback system and CFRs will recover quickly.

Preliminary experiments have shown that lesions of the brachium conjunctivum eliminate the depression of CFRs after high frequency stimulation (Andersson and Hesslow 1986 and in preparation). With the exception of the observations by Rawson and Tilokskulchai (1981), there is no evidence for any of the assumptions made above about the behaviour of Purkinje cells and interpositus neurones. However, the hypothesis presented here makes specific predictions which are open to straightforward experimental testing. Results from such tests are presented in an accompanying paper (Andersson and Hesslow 1987).

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