Slow Conduction and Reentry in the Ventricular Conducting System

II. SINGLE AND SUSTAINED CIRCUS MOVEMENT IN NETWORKS OF CANINE AND BOVINE PURKINJE FIBERS

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ABSTRACT

Closed loops of fibers of the ventricular conducting system of canine or bovine hearts were used to study circus movement of excitation. Action potentials were recorded at three sites with intracellular microelectrodes. Discrete segments were depressed by application of K⁺-rich agar or the entire loop was depressed by modified Tyrode's solution containing 15–17 mM K⁺ and 1 to 5 x 10⁻⁶ M epinephrine. The loops were 12–35 mm long and the effective conduction velocity was 0.02–0.08 m/sec. Impulses entering some loops traveled in one direction only, circling around the loop and returning to produce a second response at one or more sites (single circus movement). In other loops the impulse traveled around the circuit repeatedly (sustained circus movement). Circus movement around short loops requires a low conduction velocity and must be initiated by an impulse that travels around the loop in only one direction. Single circus movement can cause extrasystoles. Sustained circus movement can cause idioventricular rhythms and ventricular tachycardia.

KEY WORDS ventricular fibrillation slow conduction summation one-way block myocardial infarction depressed excitability mechanism of arrhythmias parasystole transmembrane potentials exit and entry block

That the cardiac impulse might become reentrant by means of a circus movement of excitation was implied by McWilliam in 1887 (1). Circus movement was demonstrated by Mayer (2, 3), Mines (4, 5) and Garrey (6). The hypothesis that circus movement of excitation might be the basis for atrial fibrillation was developed in detail by Lewis and his co-workers (7) but was also widely challenged (8). Ever since those early studies circus movement of excitation has been attractive as a possible explanation for various cardiac arrhythmias, while circus movement made possible by functional longitudinal dissociation offers much the most plausible explanation for many apparently reentrant nodal extrasystoles (9, 10). Nevertheless, circus movement has never actually been demonstrated in relatively short, grossly circuitous loops in the mammalian heart.

For a circus movement to occur in a closed ring of tissue, the time taken by the impulse to travel around the ring must at least exceed the longest refractory period in the ring. The path can be short if the transit time is lengthened either by uniform reduction of the conduction velocity or by the presence of delayed transmission in one or more regions of a ring the rest of which shows rapid conduction. Marked delays can be obtained by localized...
depression of the excitability of Purkinje fibers (11, 12) and depression of excitability induced by high K⁺ plus epinephrine (13, 14) can result in propagation with a very low conduction velocity. We now report circus movement in networks of Purkinje fibers in which delay is induced locally by depression of short segments or in which slow conduction is induced by high K⁺ and epinephrine.

Methods

The experiments were conducted on loops of cardiac tissue composed either entirely of fibers of the ventricular conducting system or partly of such fibers and partly of myocardium. In most instances, some part of the loop remained attached to one or more small pieces of myocardium to which the stimuli were applied. Each loop is shown in a diagram in the results section. The loops were obtained either from canine or bovine hearts; longer loops could be obtained from bovine hearts. Dogs or calves were anesthetized by the intravenous injection of 30 mg/kg sodium pentobarbital. The chest was opened and the heart was quickly removed and placed in a beaker of oxygenated Tyrode's solution. The loops were removed from the right or left ventricle within 5 minutes after the heart had been excised and were pinned to the wax bottom of a tissue bath in which they were perfused with Tyrode's solution of the following compositions in mM: NaCl, 137; KCl, 5.4; CaCl₂, 2.7; MgCl₂, 0.5; NaHCO₃, 12; NaH₂PO₄, 1.8; dextrose, 5.5. The solution was gassed with 95% O₂ and 5% CO₂ and maintained at 36° or 37°C.

Each preparation was driven at a rate of 60/min and explored with microelectrodes to verify that it showed normal action potentials before any depression was imposed. In some experiments depression of excitability was evoked in discrete segments of the loop by the application of two layers of K⁺-rich agar. The composition of the agar and the way in which it was applied have been described previously (11). In other experiments the entire loop was depressed by raising the concentration of K⁺ in the perfusing solution to a level that abolished excitability (usually 15–17 mM); after a few minutes, excitability was restored by the addition of L-epinephrine (1 to 5 × 10⁻⁶M) as described previously (13). After such restoration of excitability the action potential has a slow upstroke and a low conduction velocity (13, 14).

Stimulating and recording equipment and techniques were described previously (11). The preparations were often driven at a very low rate (from 30/min to as low as 6/min). This made it easy to turn off the drive in time to prevent the next stimulus from interrupting a circus movement when one appeared; it also served as a check on the absence of spontaneous activity; in addition, fibers exposed to high K⁺ plus epinephrine do not readily follow a high drive rate.

Results

In the preparation shown in Figure 1A, a network of Purkinje fibers from the canine heart was depressed by exposure to 15 mM K⁺ and 5 × 10⁻⁶M epinephrine. B shows that a single stimulus applied to the end of the main bundle at S₁ is followed at sites 1 and 2 by two action potentials. Initial activity at 1 was

![Figure 1](http://circres.ahajournals.org/content/bf1/1/164/1.f.jpg)
SLOW CONDUCTION AND CIRCUS MOVEMENT

followed by activity at 2 as would be expected if an impulse arose at S1 and was conducted from there to 1 and then to 2. The second impulse apparently returned toward the stimulus site, passing 2 first and then reaching 1. Only one action potential was, however, seen at 5 (bottom trace) and it appeared well after the initial responses at 1 and 2 but before the second responses at 1 and 2, suggesting that the second set of impulses at 1 and 2 arose because excitation was conducted around the loop to reenter the main branch. This interpretation requires that conduction around the loop occurs with delay, and in only one direction, from a to b to c, one-way block preventing direct excitation of branch c of the loop by activity in the main bundle.

To analyze this possibility, one electrode was moved from site 1 to site 3 in the main branch, one electrode was left in the lower part of the loop (branch c, site 5), and the third was placed in the upper part of the loop (branch a, site 4). Records from these sites are shown in C. An impulse evoked by stimulating at S1 always traveled along the main bundle and into a, but two types of response were seen at 4. One response, the first seen in C, consisted of a double deflection in which a rather slow depolarization was succeeded by a second upstroke. Such a second upstroke has been shown to be associated with certain types of slow conduction through a depressed segment (11). When the second upstroke was seen at 4, an action potential was seen at 5 and a second action potential appeared in the main bundle. The other response at 4 is the second seen in C in which the initial slow response is not succeeded by a second upstroke. In that situation no action potential was seen at 5 and no second action potential was seen in the main bundle. These records further suggest that the second response in the main bundle results from delayed and one-way transmission of the impulse around the loop; they also make more plausible the idea that excitation can reach 5 when it is conducted around the loop but not by direct conduction into branch c from the main bundle. The second upstroke at 4, the action potential at 5, and the second action potential at 3 were inseparable; either all three appeared or none appeared.

To test the validity of this analysis branch b was cut, as shown in the diagram at the top of Figure 2. In A one recording electrode was in the main bundle (top trace, 3) and the other was in branch a (bottom trace, 4). Two types of response were seen at 4. In one there were two upstrokes (the first set of action potentials in A), but in the other, there was only a single upstroke. These records confirm the fact that 4 was at or near a point of variable block. When the impulse succeeded in passing the block, the second upstroke appeared; when it did not, no second upstroke appeared.

FIGURE 2

The diagram shows the preparation shown in Figure 1 after branch b had been cut and a second set of stimulating electrodes had been placed at S2. The records show that (A) an impulse entering branch a may show a second upstroke or not at site 4 but that when a second upstroke is seen it does not lead to reentry at site 3. The records B and C show the presence of one-way block in branch c (see text). Vertical calibration 100 mv; horizontal, 500 msec.
However, conduction past 4 could not evoke a second upstroke in the main bundle because the connection between 4 and 5 had been cut.

To test the assumption that one-way block was present in branch c, a second set of stimulating electrodes, S2, was placed between the cut and branch c. When a stimulus was applied at S1, excitation traveled along the main bundle as seen in B (top trace) but did not enter branch c (bottom trace). On the other hand, when excitation was applied at S2 as seen in C, an action potential appeared at site 5 (bottom trace) and was conducted to 2 in the main bundle (top trace). Thus there was delay and variable block at or near 4 in branch a and one-way block in branch c, confirming the interpretation that the appearance of the second impulse in the main bundle resulted from delayed and one-way conduction around the loop. Only one such circuit was possible because conduction beyond 4 was rapid. Reentry of the main bundle was possible because the main bundle recovered its excitability during the delay at 4, but the impulse, once past 4, presumably returned to that site before it had recovered its excitability. A continuous circus movement thus could not occur in this preparation under these conditions.

Reentry into the main bundle can occur in this preparation only if the driven impulse conducts around the loop, which requires that it conduct past the site of variable block in branch a. Since conduction through depressed fibers may be subject to 2:1 or greater degrees of block according to the drive rate (11), reentry into the main bundle might be expected to occur with a frequency that varies with drive rate.

Records obtained before the loop was cut are shown in Figure 3 to illustrate this effect. At the rate of 50/min in A each driven impulse was followed by a second upstroke at site 4, an action potential at 5 and a second action potential in the main bundle at 3. In A there is a slight but progressive increase in delay at 4 with a corresponding progressive increase in delay of the appearance of activity at 5 and in the appearance of the second response in the main bundle. In B, where the drive rate for the first 4 responses was 80/min, the progressive increase in delay resembles

![Figure 3](http://circres.ahajournals.org/fig/3.png)

The records shown were obtained from the preparation shown in Figure 1 and are recorded from the same sites as those in Figure 1C. The recording sites are the same in A, B, and C and are labeled in A. The records show that as rate is increased delay and block near site 4 increase so that reentry into the main bundle becomes less frequent. See text for discussion. Calibrations: vertical, 100 mv; horizontal, 500 msec.
that seen in A; the rate then was increased to 88/min, leading to block of the second upstroke at 4 with dropping out of an impulse at 5 and a dropping out of the second response in the main bundle. At the drive rate shown in B, humps appear on the repolarization limb of the reentrant response recorded at 3; we are unable to explain this phenomenon. In C the rate varies between 109/min and 133/min and reentry to the main bundle is very infrequent. This dependence of the frequency of reentry on the drive rate resembles that seen in return extrasystole in unbranched bundles which we have described elsewhere (13).

The records shown in Figures 4 and 5 were obtained from the loop of canine Purkinje fibers depicted diagrammatically in A. The records in Figure 4 show the nearest approach to a full extra circuit of reentry that we were able to obtain by the depression of a discrete segment by high K+ agar. The first set of action potentials in Figure 4, B–E represents the response of the loop to the regular drive stimulus. The three recording sites show nearly simultaneous activity, and it is not possible to determine in what direction the impulse traveled around the loop; in all probability it traveled in both directions. The next set of action potentials in Figure 4, B–E is the response to a premature stimulus. In B the premature stimulus evokes a full response at site 2, a response of reduced amplitude at 3 and a very small deflection at 1. This sequence could result if an impulse originating at the stimulus site between 1 and 2 blocked before reaching 1 while spreading in the other direction past 2 toward 3, and blocking near 3. That path is shown in a diagram at the right of the tracings.

If the above assumption is correct, the tracings shown in C can be explained if the impulse that arose at the stimulating site and traveled toward 1 did not block but conducted through the agar with delay and returned...
around the loop to reexcite 3 and then 2, following the path shown in the diagram at the right of the tracings. The third impulse at 3 in C is reentrant because it appears at a site that has already produced an action potential in response to the premature stimulus. This reentrant impulse is preceded by a slow depolarization very like that seen at 4 in C, i.e., by a response suggestive of the impulse slowly traversing a site of depressed excitability.

The interpretation of the events seen in B and C is confirmed by D, in which the slow deflection at site 3 does not lead to an eventual fast upstroke. Under those circumstances, the response at 2 is much diminished in amplitude, as would be expected if the abortively reentrant impulse had all but blocked at or near 3. This pathway is shown in a diagram at the right of the tracings.

We were unable to record at enough sites to obtain definite proof of the existence and exact location of the sites of block or delay in the loop. The above interpretation requires the presence of two such sites, at one of which, within the agar, an impulse arising at the stimulus site and traveling clockwise may either die out near site 1 or be conducted past that site with delay. The assumption of such behavior is plausible in terms of our previous studies of conduction through depressed segments and in terms of the records shown in Figure 4, B and C. The other site of depressed conduction is assumed to be near 3. A premature impulse arising at the stimulating electrode and traveling counterclockwise is assumed to die out at or near 3 while an impulse traveling past site 1 clockwise is assumed to either die out at site 3 or pass that site with delay. The electrode was therefore moved from site 3 to site 4, slightly beyond site 3 in the counterclockwise direction.

E shows that the premature impulse at site 4 has a very low amplitude and short duration, confirming the fact that the premature impulse traveling counterclockwise is dying out between 3 and 4. The reentrant impulse at 4 is larger than that usually seen at 3, and it precedes the upstroke at 2, showing that the direction of spread of the reentrant impulse is indeed from 1 to 4 to 3 to 2, as shown in each of the diagrams. This confirms the interpretation that a premature impulse blocks in the counterclockwise direction while traveling with delay in the clockwise direction to return to 3 and then to 2. Refractoriness or some other sort of irresponsiveness evidently prevented the returning impulse from again reaching and conducting past 1, so no complete reentrant circuit was seen, nor was any continual circus movement seen. Even the partial reentrant passage of the impulse could, of course, lead to a premature impulse evoked at the stimulating site giving rise to reentry in the whole heart were the returning impulse to...
gain access to the heart via a branch arising at, e.g., 3.

After the studies using high K\(^+\) agar shown in Figure 4 were completed, the preparation was exposed to 15 mM K\(^+\) and epinephrine. The recording sites are shown diagrammatically in Figure 5A. B shows the most common response to the drive stimulus, namely a full response at site 1 and a smaller response at 2 with no response at 3. This suggests, as is shown in the diagram at the right of the tracings in B, that the impulse was blocked in both clockwise and counterclockwise directions. A premature stimulus evoked a different sequence of excitation, as shown in C. Conduction counterclockwise was much impaired as shown by the very small deflection seen at 2; clockwise conduction is demonstrated by the following of the response at 1 by a response at 3 (bottom trace) followed in turn by a response at 2 (middle trace). Occasionally a reentrant circuit was seen, as in D. The

**FIGURE 6**

*The preparation used (A) was taken from a calf heart. The stimulus site is S, the recording sites are a, b, and c. In this preparation the drive blocked in the clockwise direction near a while traveling in the counterclockwise direction. A partial circuit of reentry is shown in B, a full circuit in C. In B and C each action potential is numbered; the same numbers appear in the diagram to show in what order each response appeared at each site. In the diagram the base of the triangle corresponds to c, the left side to a and the right side to b. Calibration for B and C: vertical, 100 mv; time marks at 100-msec intervals. The records in D show regular repetition of the sequence of reentry shown in B; calibrations: vertical, 100 mv; horizontal, 3 sec.*

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sequence of events in D is exactly like that seen in C apart from the appearance of two additional responses (arrows). These responses are readily explained by the assumption that the impulse, having reached 2 by travel clockwise, continued on to 1 and then on to 3, completing an extra circuit around the loop. This pathway is shown in the diagram at the right of the tracings in D. The above interpretation requires that the premature impulse failed to conduct in the direction in which the driven impulse did conduct but was able to conduct in the direction in which the driven impulse could not conduct. The loop in this preparation was about 55 mm long and the transit time of the reentrant impulse in D was about 600 msec, so the average effective conduction velocity was approximately 0.08 m/sec.

In the records shown in Figure 6, the driven impulse initiated a circus movement in the network shown diagrammatically in A. The preparation, obtained from a calf heart, was depressed by exposure to high K⁺ and epinephrine. In B and C each impulse is numbered in the order of its appearance in time, and the site at which that impulse was recorded is similarly numbered in the diagrams at the right of the tracings. In B the initial driven impulse blocked in the clockwise direction, and failed to travel much beyond a, as suggested by the low amplitude of the response at a (impulse 2). That the driven impulse traveled counterclockwise is shown by activity at c being followed by activity at b and thereafter by activity at a, which in turn reexcited c (response 5). It thus appears that there is a site of one-way block that prevents excitation from reaching a when it approaches from c but can excite a when the approach is from b. The sequence shown in C includes a further complete circuit of the loop by the impulse. The conditions are the same as in B except that response 5 is followed by another circuit in the counterclockwise direction,
exciting $b$, $a$, and $c$ in that order to give rise to responses 6, 7, and 8.

At certain drive rates the degree of reentry seen in $B$ was evoked by every driven impulse, as is shown in $D$. The sequence seen in $B$ and $D$ represents a partial circuit of reentry with extra responses at $a$ and $c$. A branch arising at either of these sites could conduct the extra response into the heart to give rise to an extrasystole, so that the events shown in $D$ could produce a bigeminal rhythm. After the loop was cut between $b$ and $c$, reentry could not be obtained in this preparation.

The records shown in Figure 7 were obtained from the preparation shown diagrammatically at the top of the figure. The network of Purkinje fibers and myocardium was obtained from a calf heart; it was depressed by a 15-minute exposure to 15 mM K+ followed by the addition of $5 \times 10^{-4}$ M epinephrine. The position of the recording electrodes is shown in the diagram. Prior to exposure to high K+ and epinephrine, control records were obtained; they show (A) that the action potentials were normal at all sites and that conduction velocity was rapid. After exposure to high K+ and epinephrine, the preparation was stimulated at $S_1$ at a regular rate. Propagation appeared to occur through both main branches ($B$, first set of action potentials) since activity appeared more or less simultaneously at sites 1 and 2 and activity at 3 followed activity at 1 and 2. Activity could have reached 3 via either branch, as is shown diagrammatically in Figure 8A. The second set of action potentials in Figure 7B resulted when a premature stimulus was applied to the other end of the loop at $S_2$. The prematurity and the different site of stimulation resulted in propagation past 3 and 2 without activity reaching 1 either from the stimulating site or by conduction around the loop. At a slightly later interval (Fig. 7C) the response to a premature stimulus applied at $S_2$ traveled in one direction past all three sites, from $S_2$ to 3 to 2 to 1, as shown diagrammatically in Figure 8B. This is the necessary condition for establishing a reentrant circus movement. Figure 7D shows such a reentrant circus movement, the premature stimulus making a full circuit from 3 to 2 to 1 and then being followed by a partial circuit from 3 to 2. That sequence is shown diagrammatically in Figure 8C. A sustained circus movement is shown in Figure 7E. The first set of action potentials arose from a stimulus applied at $S_1$; the second set arose from a stimulus applied at $S_2$ and the rest resulted from reexcitation via a circus movement without further applied stimuli. The sequence of the circus movement is the same as that evoked by the premature stimulus: excitation travels from 3 to 2 to 1 and then from 1 to 3 and so on around the ring, for a total of seven circuits.

This sort of circus movement was evoked many times in the preparation; the impulse...
traveled around the loop four to seven times. The only peculiarity of the records worthy of special mention is the prolongation of the action potential at site 2; we have previously shown this sort of prolongation to be associated with marked slowing of forward conduction (11). The rate generated by this circus movement is not high, being about 50/min. The total path length was about 30 mm and the transit time was about 1.1 seconds, so the average conduction velocity around the loop was about 0.03 m/sec. In this preparation, stopping the drive for periods of 30 seconds–60 seconds revealed no spontaneous activity.

**Discussion**

*Conditions for Initiation of a Circus Movement.*—The relationship between path length, refractory period and conduction velocity that determines whether a circulating excitation can exist in a given loop is a simple one: the path length in meters equals the conduction velocity in meters/second times the duration of the full recovery time in seconds. In normal Purkinje fibers in which the refractory period is about 0.3 seconds and the conduction velocity is about 3 m/sec, the path length needed to permit a circus movement is thus 1 m. In Purkinje fibers depressed in the manner we have described, in which we have seen conduction velocities as low as 0.03 m/sec and refractory periods of about normal length, the path length needed to permit a circus movement is only 10 mm. We have not seen a circus movement in a path that short but we have found a circus movement in a path about 30 mm long (Fig. 7) and we have seen one-way conduction and delay sufficient to cause reentry occur around a loop about 12 mm long (Fig. 1).

Even if the necessary combination of path length, refractory period and conduction velocity is present, an impulse that arises at a point in a closed loop and travels in both directions from its point of origin will be extinguished when the impulses meet halfway round the loop from the starting point. A circus movement must thus be initiated by an impulse that travels around the loop in only one direction, as Mayer, Mines, and Garrey emphasized (1–6). In addition, unless the wave of excitation begins very near the site of one-way block, the impulse that travels around part of the loop to block at the depressed site may extinguish the impulse that traveled in the other direction. A circus movement can therefore begin in a loop only if a site of one-way block is at least transiently present and even then may fail unless the site of block is near the point at which excitation is initiated. The experimental initiation of a circus movement is thus often a matter of chance, just as it must be in the whole heart; and the above considerations may explain why it is difficult to obtain circus movement in a largely normal loop only one discrete segment of which has been depressed by high K+ agar.

*Criteria for Accepting Circus Movement as the Basis of Repetitive Activity.*—As far as we can determine it is impossible to prove that a sequence of activity that appears to depend on a circus movement of excitation really does depend on such a movement; as with any complex phenomenon it is always possible to invent alternative explanations. However, when the sequence of activation seen is that expected in a circus movement, when the sequence remains the same throughout the apparent circus movement, and when activity stops at each recording site in the expected sequence, the case for a circus movement is good. It is strengthened by showing the actual existence of slow conduction and by the demonstration of actual sites of one-way block and delay. If these latter phenomena persist and the circus movement cannot be elicited after the ring has been cut, the case becomes very strong.

The one finding that absolutely rules out circus movement is the persistence of excitation at one recording site in the ring when it has died out at other sites; such excitation clearly does not result from continuous conduction around the ring. In the present article we have presented only records in which the sequence of activation remains the same and in which activity dies in the correct
sequence. We have generally identified one or more of the sites of block and delay in the ring and in Figures 1–3 we have shown that such sites retain their properties after the possibility of circuitous reentry has been abolished by cutting the ring.

In the words of Mines (5): "The chief error to be guarded against is that of mistaking a series of automatic beats originating in one point in the ring and travelling around it in one direction only owing to a complete block close to the point of origin of the rhythm on one side of this point." To this caution we would add only that while the source of "spontaneous activity" might be pacemaker activity dependent on phase 4 depolarization, it might also be repetitive excitation being fed into the ring from any site, such as another circus in a complex network, or activity arising from oscillatory after-potentials. We have never seen pacemaker activity dependent on phase 4 depolarization in dog or calf Purkinje fibers subjected to high K+ agar or to 15 mM K+ plus $5 \times 10^{-6}$ epinephrine, nor have we ever seen oscillatory after-potentials in canine Purkinje fibers. Carmeliet and Vereecke have reported oscillatory after-potentials in calf Purkinje fibers subjected to high K+ and epinephrine (14); we have seen the same phenomenon under the same circumstances and it can lead to repetitive activity. In our experience, such activity is seen only in preparations that have previously shown subthreshold oscillatory after-potentials of the kind described by Carmeliet and Vereecke (14).

**SIGNIFICANCE FOR THE WHOLE HEART**

*Idioventricular Rhythms and Ventricular Tachycardia.*—Circus movement of the kind seen in Figure 6 could cause an idioventricular rhythm but only if it was evoked by an initiating impulse. Such initiation might result from the transient relief of entry block that had been shielding the ring from the activity of the rest of the heart. It might also result from a premature activation of the ventricle since some rings show circus movement in response to a premature excitation but not to the regular driven impulse, as shown in Figure 5.

None of the circus movements that we have obtained up to now show a very rapid rate and most of them are quite slow. We do not know whether rapid rates can be generated by slow conduction around a relatively short loop. On the other hand, the rate in many "tachycardias" is not really very rapid and the possibility that a circus movement could cause an idioventricular rhythm of 70 or 80 beats/min certainly seems reasonable as an extrapolation from the rates of 50/min that we have seen result from circus movement.

There are many possible mechanisms for ventricular tachycardia (16) including: (1) the onset of activity of a pacemaker dependent on phase 4 depolarization, (2) the unmasking of an already active pacemaker by relief of exit block, (3) activity resulting from oscillatory after-potentials, (4) excitation reciprocating between a pair of foci each capable of yielding return extrasystoles or single circus movement, (5) persistent circus movement. Our findings show that the two last mechanisms must be taken seriously as possible causes of idioventricular rhythms and ventricular tachycardias. They must also, of course, be considered to be possible mechanisms for the activity of parasystolic foci.

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