Active and passive control of spiral turbulence in excitable media

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(Received 29 April 2007; revised manuscript received 8 October 2007; published 23 April 2008)

The influence of a spatially localized heterogeneity defect, defined by failure of the diffusion effect, on spiral turbulence suppression in two-dimensional excitable media is studied numerically, based on the Bär model. It is shown that in certain parameter regions spiral turbulence without the defect can be suppressed by a boundary periodic forcing (called active control) if the forcing frequency is properly chosen. However, with a sufficiently large defect this active control method no longer works due to the wake turbulence following the defect. We suggest an auxiliary method of enclosing the defect with a thin layer of material of high excitability (called passive control) to screen the interaction between the defect and the turbulence and to restore the global control effect of the periodic forcing. The possible application of the method in cardiac defibrillation is discussed.

DOI: 10.1103/PhysRevE.77.046217 PACS number(s): 05.45.−a, 82.40.Ck, 82.20.−w

I. INTRODUCTION

Excitable media are extended spatial systems that support propagation of waves including pulses and rotating spirals. Thus various models of excitable media have been used to account for nonlinear wave phenomena in many fields of biology, chemistry, and physics [1]. Wave propagation in inhomogeneous media has attracted much attention in the past decades because some natural reaction-diffusion media such as biological excitable tissues are strongly heterogeneous [2–7]. Experimental observations of spiral waves (SWs) in excitable media have demonstrated significant effects of heterogeneities on the motion of SWs. These effects include drift of SWs due to parameter gradients [8] or external fields [9], and anchoring of SWs on localized heterogeneities [8]. Numerical results show that in certain conditions heterogeneities of the media can either cause the breaking up of SWs into spatiotemporal chaos [i.e., spiral turbulence (ST)] [10] or prevent this breakup in some other conditions [6].

The above wave phenomena are particularly interesting in the dynamics of cardiac tissue [11–15]. It is believed that SWs and ST are associated with the most dangerous cardiac arrhythmias, including ventricular tachycardia and fibrillation. For example, in many cases spirals can drift and eventually dissipate at tissue borders, whereupon the heart returns to normal [16]. However, if there exist defective regions in a heart (such as some diseased cells due to myocardial infarction), spirals may be trapped by the defects, and thereby cause permanent tachycardia, and even induce transitions from scroll waves to turbulence, leading to cardiac fibrillation [17,18]. Many control methods have been developed for ST suppression in homogeneous media in the past decades [12,19–26]. However, the control of turbulence caused by heterogeneities of the media has been studied much less.

It has been shown that ST can be effectively suppressed by the active control method of local periodic forcing [26–28]. However, we find that, if there exists a sufficiently large heterogeneity defect (e.g., some dead cells without the function of diffusion) in the excitable medium, the above active control method cannot succeed in ST suppression. In this paper, we focus on how to eliminate the negative influence of the heterogeneity defect on ST suppression. We propose an auxiliary method (a passive control method) to screen the interaction between the defect and the turbulent waves by enclosing the defect within a patch of medium having high excitability. It is shown that with the active and passive (AP) control methods together we can completely suppress ST. The method may have potential applications in cardiac defibrillation.

In the next section, the dynamical model, the active control method, and the numerical scheme are introduced. The influences of defects on ST suppression are also studied. In Sec. III we describe the scheme for the combined application of the active and passive control methods and present the results of these methods in ST suppression. The physical mechanism underlying the efficiency of AP control is analyzed in Sec. IV. Section V gives some discussion of the implications of its application in cardiac defibrillation.

II. MODEL AND NUMERICAL METHOD

The excitable medium to be studied is the Bär model [29], i.e., the following two-dimensional (2D) reaction-diffusion equation:

$$\frac{\partial u}{\partial t} = -\frac{1}{\varepsilon}u(u-1)\left(u-\frac{v+b}{a}\right) + D\bar{\delta}(\Omega_1)\nabla^2 u + \delta(\Omega_2)F(t), \tag{1a}$$

$$\frac{\partial v}{\partial t} = f(u) - v, \tag{1b}$$

$$\bar{\delta}(\Omega_1) = \begin{cases} 0 & \text{if } (x,y) \in \Omega_1, \\ 1 & \text{if } (x,y) \notin \Omega_1, \end{cases},$$

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$$\delta(\Omega_2) = \begin{cases} 1 & \text{if } (x,y) \in \Omega_2, \\ 0 & \text{if } (x,y) \notin \Omega_2, \end{cases} \tag{1c}$$

$$F(t) = F\cos(\omega t). \tag{1d}$$

When $F=0$ Eq. (1a)–(1d) describes the interaction between an activator $u(x,y,t)$ and an inhibitor $v(x,y,t)$. In Eq. (1a)–(1d) the function $f(u)$ takes the following form:

$$f(u) = \begin{cases} 0 & \text{if } u < 1/3, \\ 1 - 6.75u(u-1)^2 & \text{if } 1/3 \le u \le 1, \\ 1 & \text{if } u > 1. \end{cases}$$

In Eq. (1a)–(1d) a, b, and ε are system parameters, and D is the diffusion coefficient. Throughout the paper the system size is taken as $L_x \times L_y = 240 \times 120$. The last term of Eq. (1a) is a periodic forcing applied to a local region Ω_2. In our study, the positive parameters D, a, and b are fixed at $D=1$, $a=0.84$, and $b=0.07$ to take advantage of the many known results that have been obtained in substantial earlier work using this parameter set [3,29–31]. It is verified that a change of (a,b,D) does not qualitatively change the features shown in the following work as far as ST suppression is involved. The forcing area Ω_2 is located at the left boundary stripe of $x=0$. We vary ε in [0.03,0.10] and ω in [1.0,2.6]. In this paper we introduce to the system (a conductive medium with $D=1$) a square heterogeneity defect, a part of the medium, Ω_1, with $D=0$. The size of the defect is $l_x = l_y = l = n\Delta$ with n being the number of space steps in the numerical discretization. The center of the defect is located at $(x^*, y^*) = (L_y/2, L_y/2)$. Equation (1a)–(1d) is integrated by the second-order Runge-Kutta scheme together with a second-order accurate finite-difference method, with fixed time step $\Delta t = 0.02$ and spatial step $\Delta x = \Delta y = \Delta = 0.4$, i.e., we divide the $L_x \times L_y$ rectangular physical domain into $N_x \times N_y = 600 \times 300$ grid points. No-flux boundary conditions are used in all simulations. All the numerical results in this paper are checked with finer space grids and smaller time step Δt.

The parameter ε in Eq. (1a) determines the ratio of time scales of the fast field u and the slow field v. Therefore, it can determine the excitability of an excitable medium. The excitability of an excitable medium may be defined by the inverse of ε [29]. Upon increase of ε the ability of an excitable medium to propagate waves is usually reduced, and this may lead to the onset of ST [32]. Precisely, in model (1a)–(1d) SWs are generated for $\varepsilon < \varepsilon_c \approx 0.07$ with suitable initial conditions and ST is observed for $\varepsilon \ge \varepsilon_c$ [30,31]. The typical states of this classification without (with) the heterogeneity defect are shown in Figs. 1(a) and 1(b) [Figs. 1(c) and 1(d)].

Now we show how the presence of the defect can affect the control performance. With local periodic forcing, we excite some local space area periodically to generate regular propagating planar waves which have the frequency of the periodic forcing (called controlling waves), and we use these waves to suppress ST. The control signal is turned on after the system has evolved to a developed ST state. In Fig. 2, we present the results of the periodic forcing control (active control) without a defect. The control signal is applied to the

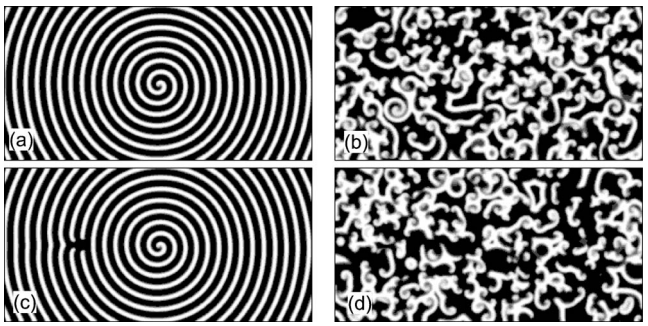

FIG. 1. Numerical results of Eq. (1a)–(1d) with $F(t)=0$. Asymptotic spatial contour patterns of variable u for different ε. (a),(b) Without defect. ε=(a) 0.06; (b) 0.08. (c),(d) The same as (a),(b), respectively, with the presence of a square heterogeneity defect centered at $x=60$ and $y=60$. The size of the defect is $n\Delta$, $n=21$, with Δ being the space step for simulation.

turbulent state of Fig. 1(b) at $t=0$. It is obvious that the active control alone can successfully suppress ST in the homogeneous medium. In Figs. 2(a) and 2(b) we show how periodic forcing with $\omega=1.3$ generates controlling waves which wipe away ST waves. In Fig. 2(c) we lift the periodic signal (i.e., set $F=0$) at $t=2000$, and the system approaches the desired rest state as the remaining planar waves leave the conducting medium through the right boundary. In Fig. 2(d) we plot the controllable region in the ω-ε parameter plane where in the shaded region successful ST and SW suppression is achieved, while in the blank region the boundary active control fails in ST and SW suppression. These results are well known from our previous investigations [27,28].

In Fig. 3, we do the same as Fig. 2 except for the presence of a heterogeneity defect. If the defect is small [Figs. 3(a) and 3(b), $n=5$], it does not change the control effect, and ST suppression can be achieved, similarly to the case of Fig. 2. However, when the defect is sufficiently large ($n \ge n_c = 8$ in our case) we can find some serious influence of the large heterogeneity defect on ST suppression [Figs. 3(c) and 3(d), $n=21$] which makes the active control of Fig. 2 fail. First, in Fig. 3(c) the presence of the defect prevents the complete ST suppression of Figs. 2(a) and 2(b). A certain wake turbulence remains behind the large defect in the asymptotic state under the active control. Second, this wake turbulence returns to the full ST (rather than evolving to the rest state) after the periodic boundary forcing $F(t)$ is lifted, as shown in Fig. 3(d).

In order to investigate the influence of various control parameters on the control effects, we plot Figs. 4 and 5 where controllable regions in different parameter plans are demonstrated. From these figures the effects of pacing amplitude and defect size are clearly shown. For small amplitude, an increase in F can effectively enlarge the controllable regions [Figs. 4(c), 5(a), and 5(b)). This improvement saturates for sufficiently large F [Figs. 4(a)–4(c), 5(b), and 5(c), $F \ge 1.4$]. When saturation is reached, an increase in F can no longer compensate further for the defect. In all the following investigations we fix $F=2$, which already represents saturation of F. With respect to defect size, we observe that increasing n can obviously reduce the efficiency of the pacing

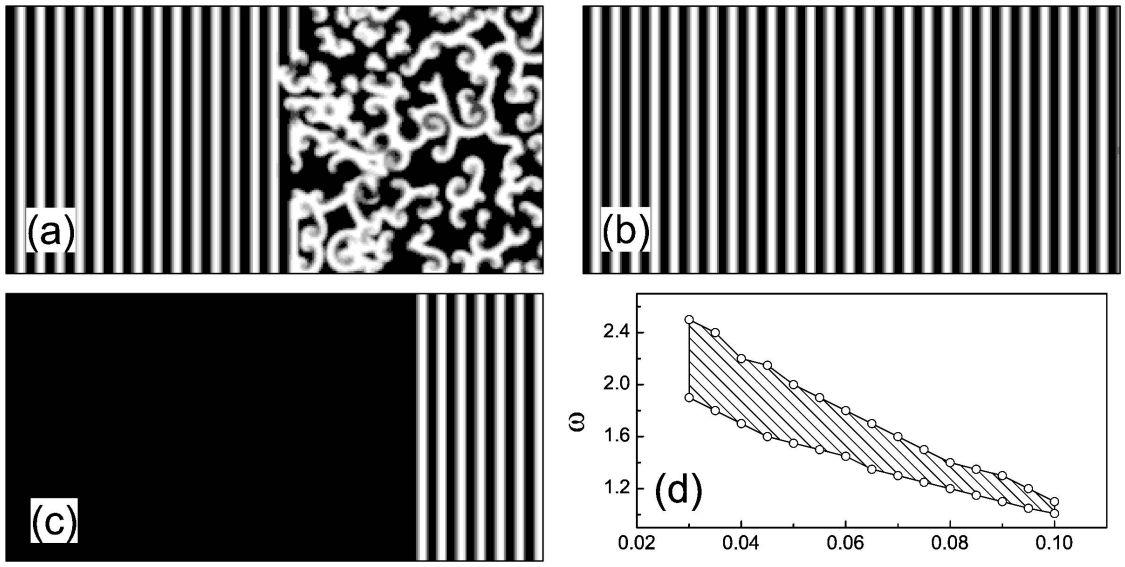

FIG. 2. Dynamic behavior of system (1a)–(1d) without defect. $F=2$, $\omega=1.3$. The periodic control signal is applied to the left boundary $x=0$ of the system after $t=0$. The initial state for the control is Fig. 1(b). (a)–(c) Spatial contour patterns of u at different time moments. (a) $t=1000$. Controlling waves generated by the boundary pacing partially suppress ST. (b) $t=2000$. The whole system is dominated by the controlling waves and ST is completely suppressed. (c) The control signal $F(t)$ is lifted at $t=2000$. At $t=2100$, the system decays to the rest state in a large space area (black area), and the rest state is completely realized after $t>2200$. (d) The controllable region in the ε-ω parameter plane. The shaded region represents successful ST and SW suppression while in the blank region the boundary control fails in ST and SW suppression.

control. The shrinkage of the controllable regions saturates also for sufficiently large n [Figs. 5(b), 5(d), and 5(e), $n \geq 20$]. When the control parameters ε and ω are chosen in the controllable region of Fig. 5(e), successful ST and SW suppression is observed even if we take $n=141$. In the next section we focus on the suppression of wake turbulence in the presence of a sufficiently large heterogeneity defect (e.g., $n=21$).

III. ACTIVE AND PASSIVE CONTROL OF SPIRAL TURBULENCE

The central task of the present paper is to eliminate the undesired influence of the heterogeneity defect on ST suppression. That is, we are interested in finding an effective method to recover the global control effects of the active control in the presence of the heterogeneity defect. Here we propose an auxiliary passive control method of ST suppression by changing the excitability parameter ε of a part of the conductive medium around the defect from ε to ε'. The medium with ε' (called the screening medium) is used to screen the interaction between the defect and the ST waves. A schematic of the passive control method is shown in Fig. 6. The thickness of the screening medium, δ, is equal to $m\Delta$ (m is an integer), which is considerably smaller than the size of the defect ($m \ll n \ll N_x, N_y$).

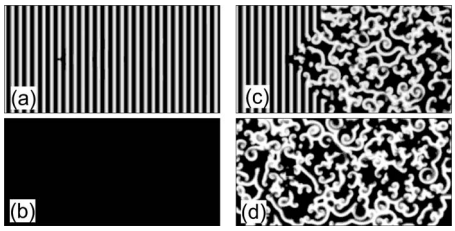

FIG. 3. The same as Figs. 2(b) and 2(c) except for the presence of the heterogeneity defect. The periodic pacing $F(t)$ is applied to the state of Fig. 1(d) at $t=0$. (a),(b) $n=5$. Wake turbulence does not exist and ST is successfully suppressed. (a) $t=2200$. ST suppression. (b) The rest state (black area) is realized at $t=2340$. The pacing $F(t)$ is lifted from the state of (a) at $t=2200$. (c),(d) $n=21$. Wake turbulence exists and ST suppression fails. (c) $t=2200$. ST is suppressed before the defect while wake turbulence remains after the defect. (d) ST contour pattern at $t=2340$. $F(t)$ is turned off at $t=2200$. After lifting the pacing [$F(t)=0$] the system returns back to the fully developed ST state in the whole space.

Now let us study systematically the effectiveness of the joint application of the active control method of Eq. (1a) together with the passive control method of Fig. 6. The turbulent state of Fig. 1(d) is chosen as the initial state ($\varepsilon=0.08$ and $n=21$) to which the control is applied. In Figs. 7(a) and 7(b) we show the results of the joint AP control method with $\omega=1.3$, $m=6$, and $\varepsilon'=0.06$. It is observed that the AP control can successfully suppress ST. When we lift the periodic signal at the moment $t=2200$ while the passive control is kept working until the controlling waves pass the defect completely, and then delete the passive control (i.e., change ε' back to ε), the system returns to the desired rest state after some transient process [Figs. 7(c) and 7(d)]. However, if the active and passive controls are turned off simultaneously [i.e., by setting both $F(t)=0$ and $m=0$ at $t=2200$], the system will return to the ST state after some transient process, due to the breakup of the controlling planar waves by the defect. It should be emphasized that the results of Fig. 7 are rather general and independent of the size of the defect. In Fig. 8 we do exactly the same as in Fig. 7 but changing the size of the defect from 21Δ to 61Δ, and the results obtained are similar to those of Fig. 7.

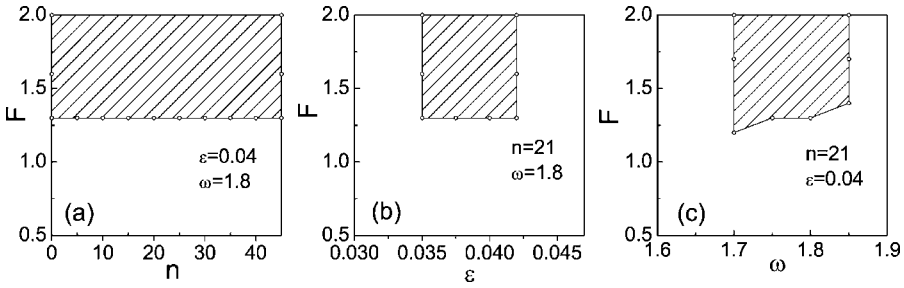

FIG. 4. Controllable regions of SW suppression in the presence of a heterogeneity defect: (a) in the F-n plane, $\varepsilon=0.04$, $\omega=1.8$; (b) in the F-ε plane, $n=21$, $\omega=1.8$; (c) in the F-ω plane, $n=21$, $\varepsilon=0.04$. In the shaded region SWs can be successfully suppressed. In (c) for small amplitude, increasing F can effectively improve SW suppression, and this improvement saturates for sufficiently large F (the saturated F value is about 1.4 in our system).

For the passive control there are some relevant control parameters, such as the excitability ε' and the thickness of the screening medium, m. In order to find how these two quantities influence the effect of ST suppression near the heterogeneity defect, we show in Fig. 9 the controllable domains in the ω-ε plane for different (ε',m)'s, and in Fig. 10 the controllable domains in the m-ε' plane for different (ω,ε,n)'s. It is found that in the shaded domains of Figs. 9 and 10 ST can always be successfully suppressed by the joint application of both the active and passive methods. Some characteristic features in Figs. 9 and 10 are worthy of remark. Comparing Fig. 9 with Fig. 5, we find that, although the active control alone completely fails for ST suppression in the presence of the heterogeneity defect [Fig. 5(b)], a small patch of screening medium can greatly enlarge the domain of successful ST suppression with the same defect, namely, the large ε of the control domain is considerably increased. In Fig. 10 we find three different characteristic boundaries of the controllable regions: right (large ε'), left (small ε'), and bottom (small m) boundaries, among which the first two are vertical to the ε' axis for sufficiently large m [i.e., the left and right boundaries are independent of the width of the screening medium for $m\geq 8$ in Figs. 10(d)–10(f)]. These features will be explained in the next section in exploring the mechanism of AP control.

IV. MECHANISM UNDERLYING THE EFFICIENCY OF AP CONTROL

It is interesting to understand the mechanism underlying wake turbulence control by the AP method. In order to do so, first we should understand why wake turbulence appears in ST suppression when the controlling planar waves pass a sufficiently large heterogeneity defect. For system (1a)–(1d) SWs can develop to ST waves for weak excitability $\varepsilon>\varepsilon_c \approx 0.07$, as shown in Fig. 1(b) ($\varepsilon=0.08$, $F=n=0$). Without the heterogeneity defect, we can use periodic boundary pacing to generate planar controlling waves to successfully suppress

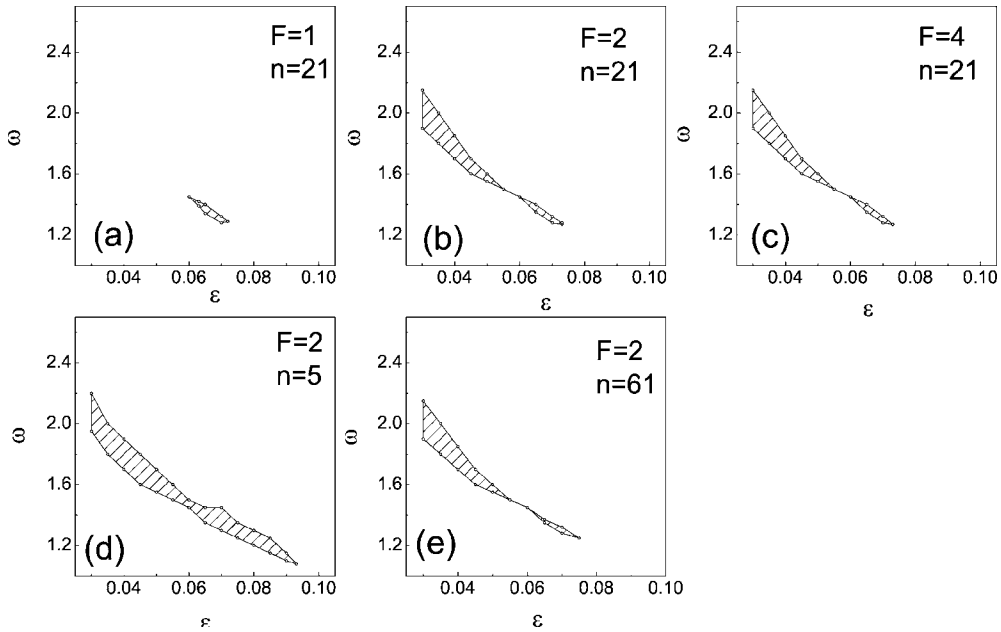

FIG. 5. Controllable regions in the ω-ε plane for various F and n in the presence of a medium defect. (a)–(c) $n=21$ and $F=1$ (a), $F=2$ (b), and $F=4$ (c). (d),(e) $F=2$ and $n=5$ (d), and $n=61$ (e). The controllable regions in (b), (c), and (e) are practically the same, and saturation is observed for $F>1.4$ and $n\geq 20$.

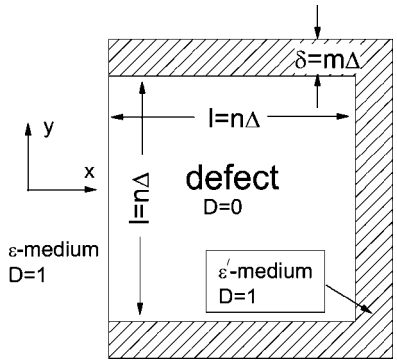

FIG. 6. Schematic of the passive control.

ST [Figs. 2(a)–2(c), $F=2$, $\omega=1.3$, $n=0$]. In the presence of a heterogeneity defect, each planar wave is broken by the defect into two segments which create two tips moving down along the left and right defect boundaries, respectively [see Figs. 3(a) and 3(c)]. If the defect is small ($n<n_c=8$ in our case), the two tips can merge with each other after the defect, recovering a smooth planar wave, and no wake turbulence is formed after the defect [Fig. 3(a), $n=5<n_c=8$]. However, for sufficiently large n ($n>n_c$), the tips develop into ST before they merge with each other as demonstrated in Fig. 3(c).

From the above understanding of wake turbulence we can explain why a combined AP control with a suitable screening medium can avoid wake turbulence. When the screen parameters ε' and m are chosen in the controllable region of Fig. 10, the tips of the wave segments broken by the heterogeneity defect move within the ε' medium after the defect, where these tips do not develop to ST because ε' is smaller than ε. These tips merge in pairs to recover smooth planar waves before they move into the ε medium [see Figs. 7(b) and 8(b)], and thus no wake turbulence is formed after the defect.

From the above qualitative analysis we can understand the three characteristically different boundaries of Fig. 10. If the thickness of the screen medium is too small ($m<2$ in our case), the tips cannot be confined within the ε' medium, and

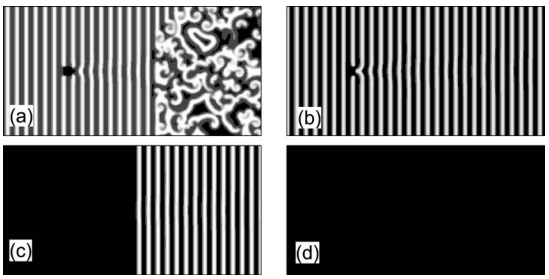

FIG. 7. Spatial contour patterns of u at different time moments under AP control. $\omega=1.3$, $\varepsilon'=0.06$, $n=21$, and $m=6$. The active and passive control methods are applied jointly to the state of Fig. 1(d) at $t=0$. (a) $t=1100$. The controlling waves partially suppress ST. Unlike in Fig. 3(c), here the heterogeneity defect does not cause wake turbulence. (b) $t=2200$. ST is completely suppressed. (c) The periodic pacing is lifted at $t=2200$ while passive control is kept working. At $t=2270$, half of the space area of system decays to the rest state (black area). (d) The passive control is lifted at $t=2300$. At $t=2340$ the whole system returns to the rest state.

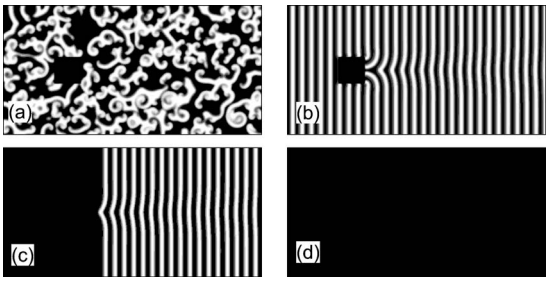

FIG. 8. The same as Fig. 7 except $n=61$. (a) $t=0$. Initial ST state of the system. The active and passive control are applied jointly to this state at $t=0$. (b) $t=3000$. ST is completely suppressed by AP control. (c) The periodic pacing is lifted at $t=3000$ while passive control is kept working. At $t=3050$, some space area of the system decays to the rest state (black area). (d) The passive control is lifted at $t=3100$. At $t=3200$ the whole system returns to the rest state.

they have a large probability and sufficiently long time to move outside the screening region. In the ε region these tips can develop to ST before they merge to smooth waves in the ε' screening region. An interesting observation in Fig. 10 is that for both too large and too small ε' AP control fails in ST suppression. The failure of the large-ε' screen can be easily understood. For too large ε' ($\varepsilon_{\max}<\varepsilon'<\varepsilon$ where $\varepsilon_{\max}$ is the largest ε' in Fig. 10), the screening medium itself has too low excitability, and tips can develop into ST even in the screening ε' domain. In this case the features of wake turbulence shown in Fig. 3(c) can also be observed in a similar manner, as shown in Fig. 11. Therefore, the screening effect of the ε' medium cannot play a role beyond this large-ε' boundary. For too small ε', tips can no longer develop ST in the screening ε' medium. However, waves propagate in the ε' medium with much greater velocity than in the conductive ε medium. Therefore, a too large difference between the velocities of the two media breaks the waves around the boundaries between the ε' and ε media and forms new tips [see the arrow in Fig. 12(c)]. These new tips develop into ST in the ε medium as shown in Fig. 12. It is clear that the mechanism of the wake turbulence of Fig. 3(c) with no screening effect (or similarly, for too small m and too large ε' of the screening medium) is essentially different from that of Fig. 12 with a screen of too small ε'. The former fails due to too weak and the latter due to too strong screening. The above descriptions of the left and right boundaries are not related to the width of the screening ε' medium. This is why both boundaries are parallel to the m axis for sufficiently large m ($m\geq 11$).

V. CONCLUSION

In conclusion, we have investigated the influence of a heterogeneity defect on the efficiency of ST suppression. We find that the presence of the heterogeneity defect can considerably decrease the control effects of the periodic pacing method (so-called active control) due to the wake turbulence downstream of the defect, and combined applications of both active and passive control methods can efficiently suppress such wake turbulence. The passive control method increases

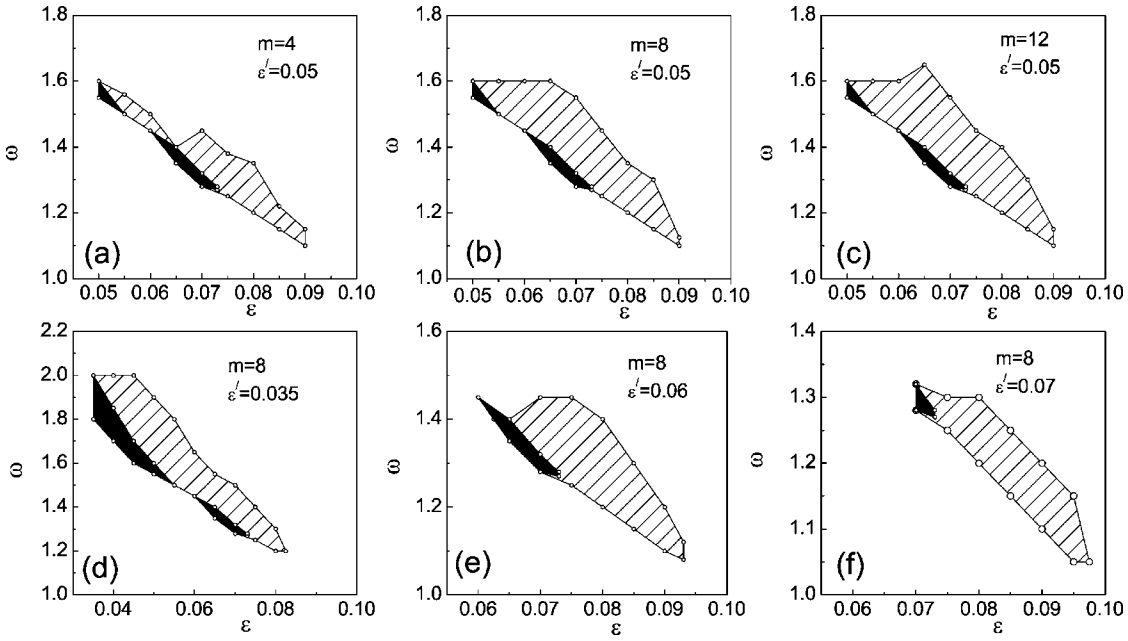

FIG. 9. Controllable regions (shaded areas) in the ω-ε plane for various (ε', m)'s $(\varepsilon \geq \varepsilon')$ under AP control. $n=21$, $F=2$. (a)–(c) $\varepsilon'=0.05$ and $m=4$ (a), $m=8$ (b), and $m=12$ (c). (d)–(f) $m=8$ and $\varepsilon'=0.035$ (d), $\varepsilon'=0.06$ (e), and $\varepsilon'=0.07$ (f). The controllable regions in (b) and (c) are considerably enlarged in comparison with that of Fig. 5(b) for $\varepsilon \geq \varepsilon'$ (dark areas which are overlapped by shaded areas).

the excitability of a small part of the medium enclosing the defect to screen its effects. Low-amplitude defibrillation of cardiac tissue is an important task, and it has attracted much attention of scientists in the past decade. We expect that in the presence of heterogeneity defects of dead or ill tissue cells some medical drugs may be injected to increase the excitability of a small part of the cardiac tissue enclosing the dead tissue defects and to essentially improve the efficiency

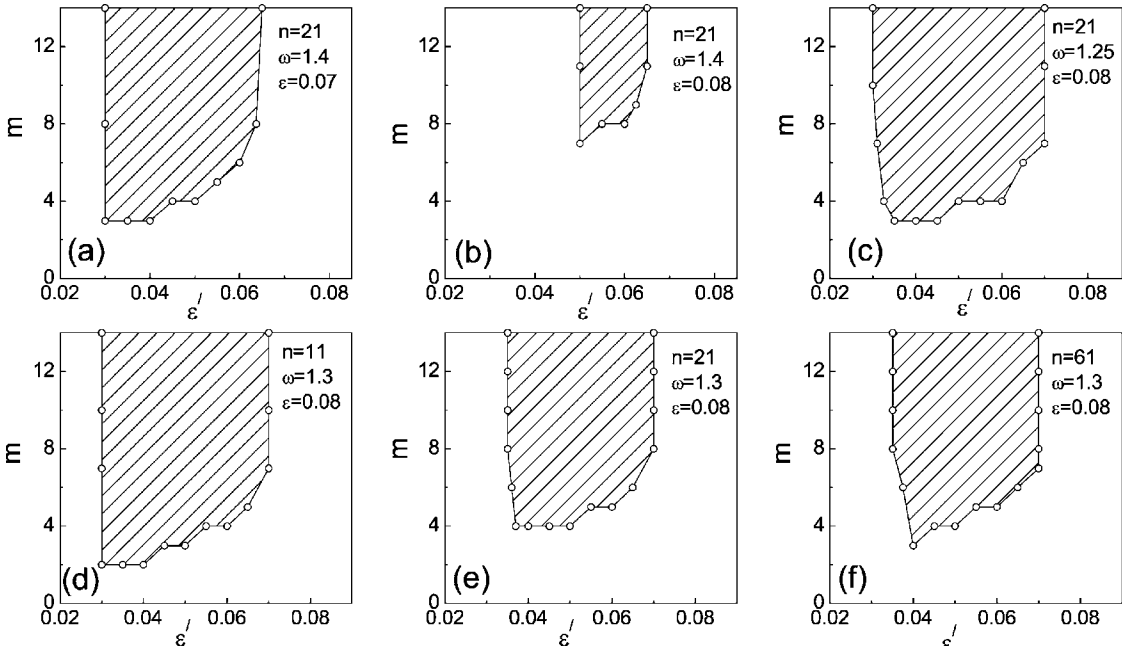

FIG. 10. Controllable region in the m-ε' parameter plane for various n, ε, and ω. (a) $n=21$, $\omega=1.4$, and $\varepsilon=0.07$; (b) $n=21$, $\omega=1.4$, and $\varepsilon=0.08$; (c) $n=21$, $\omega=1.25$, and $\varepsilon=0.08$; (d) $n=11$, $\omega=1.3$, and $\varepsilon=0.08$; (e) $n=21$, $\omega=1.3$, and $\varepsilon=0.08$; (f) $n=61$, $\omega=1.3$, and $\varepsilon=0.08$. Note that both the left and right boundaries of the controllable regions are vertical to the horizontal axis. This indicates that with given system parameters the controllability is independent of the screen width m for sufficiently large m. The area of the AP controllable region in the m-ε' plane is reduced as the distance between the given (ε, ω) set and the controllable domain of Fig. 5 increases [compare (a), (b), (c), and (e) with Fig. 5(b)]. Moreover, increasing the defect size can considerably reduce the controllable area for small n [see (d) and (e)], while the controllable area becomes independent of n for sufficiently large n [compare (e) and (f)].

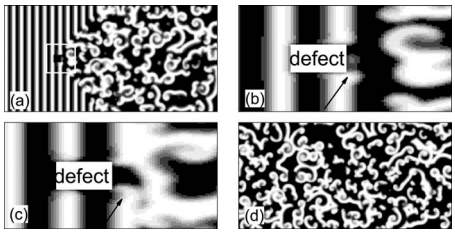

FIG. 11. Failure of AP control in ST suppression with too large $\varepsilon'=0.073$. (a) Asymptotic contour pattern of variable u. Wake turbulence remains under the given AP control. (b) Blowup of the framed region of (a). The two tips caused by the defect cannot merge. (c) The successive wave pattern after (b). The original tips develop to wavelet turbulence downstream. (d) The complete ST pattern is observed after lifting the pacing from state (a).

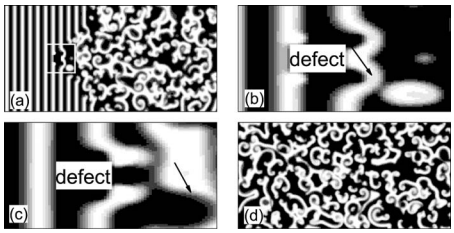

FIG. 12. The same as Fig. 11 with too small $\varepsilon'=0.033$. Note that in (b) the two tips caused by the heterogeneity defect have already merged to form a smooth wave, while in (c) new breakup of waves indicated by arrows creates new tips which develop to ST on further propagation.

of active low-amplitude pacing control by wiping away the wake turbulence. We should emphasize that it is still not clear how one can perform the passive control shown in Fig. 6 for cardiac defibrillation with medical drugs in practice, the idea of joint application of active and passive methods is only suggestive for the task of cardiac defibrillation in the presence of strong heterogeneity. Moreover, apart from cardiac systems, the present method is expected to be useful for the general task of ST suppression in other heterogeneous excitable systems, such as chemical reaction-diffusion systems.

ACKNOWLEDGMENTS

This work was supported by the Natural Science Foundation of China (Grant Nos. 10765002, 10335010, and 10347001) and the Nonlinear Science Project.

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