Correspondence

Letters to the Editor must not exceed 400 words in length and may be subject to editing or abridgment. Letters must be limited to three authors and five references. They should not have tables or figures and should relate solely to an article published in Circulation within the preceding 12 weeks. Only some letters will be published. Authors of those selected for publication will receive prepublication proofs, and authors of the article cited in the letter will be invited to reply. Replies must be signed by all authors listed in the original publication.

Atrial Flutter Cycle Length Oscillations and Role of the Autonomic Nervous System

To the Editor:

In a recent issue of Circulation, Stambler et al1 provide an intriguing but possibly misleading set of results regarding the variability of atrial flutter cycle length (AFCL) and the role of the autonomic nervous system in AFCL modulation. Previous studies2-5 identified in the AFCL variability the existence of 2 rhythmic oscillations, 1 controlled by the ventricular rate and 1 by respiratory activity, suggesting a modulatory effect of atrial stretch. Using spectral analysis, Stambler et al claimed the existence of an additional low-frequency oscillation (band 1) mediated by sympathetic tone. Their conclusions are not supported by their study design and results.

In fact, the newly proposed low-frequency oscillation band was concentrated in the extremely low-frequency region of the spectrum and on average was centered around 0.006±0.002 Hz (Table 2 of the article by Stambler et al). This should correspond to an AFCL oscillation with a period of 166 seconds, clearly longer than the 2 minutes of analyzed recording time. This means that we are dealing with a baseline trend rather than a rhythmic oscillation. In other words, band 1 is simply a DC component of the spectrum reflecting the nonstationary nature of the analyzed time series and thus should be avoided in the interpretation of the power spectra.6

In their study, Stambler et al also examined the role of the autonomic nervous system in the modulation of AFCL by studying the effect of pharmacological interventions on the power-band distribution of AFCL spectra. Stimulation and inhibition of sympathetic tone obtained by intravenous infusion of isoproterenol and long-term therapy with β-adrenergic blockers affected only the newly proposed low-frequency band, leaving unchanged the respiratory and ventricular rate–related oscillations. These spectral changes were accompanied in patients receiving isoproterenol by a decrease of the mean AFCL. From these results, the authors conclude that band 1 is mediated by sympathetic tone.

By the above methodological considerations, isoproterenol and β-blockers, by changing only the DC component of AFCL spectra, seem to be effective only on the stationarity of the time series by slowly changing the level of AFCL. Thus, it seems probable that the increase of power in band 1 caused by isoproterenol may simply reflect the trend of shortening of the mean AFCL rather than indicate a modulatory role of sympathetic tone on short-term AFCL variability. This is clearly evident in Figure 2 of the article by Stambler et al, which shows the effects of isoproterenol on band 1, in which the spectral estimator is not able to attribute a frequency different from zero to this spectral component (see the numerical data inserted in the figure). On the other hand, modulatory sympathetic activity on the cardiovascular variables is reported to be active at frequencies around 0.1 Hz,7 at least 1 order of magnitude greater than the average frequency of the band 1 given by Stambler et al. In addition, the authors point out that vagal stimulation by intravenously infused adrenaline does not modify either overall AFCL variability or power-band distribution.

Thus, from these results it seems hard to conclude a possible role of the autonomic nervous system in beat-to-beat modulation of AFCL. On the other hand, the additional data given by the authors (that in transplant patients, well-defined AFCL oscillations exist, although the global variability was less than in control patients) do not seem to offer any additional element to support the hypothesis of an autonomic modulation of AFCL. In fact, the existence in transplant recipients of AFCL fluctuations may strengthen the hypothesis of a mechanical modulation of AFCL. The authors suggest that the diminished variability may reflect the lack of innervation in transplant recipients compared with preserved autonomic function in control patients, indicating a role of autonomic tone in AFCL variability. However, it is known that orthotopic heart transplant results in an altered anatomy and function of the atria. In the hypothesis that AFCL variability is caused by atrial stretch,7-4 the reduced AFCL variability in transplant recipients may also be explained by impaired mechanical atrial function.

In conclusion, we believe that spectral analysis can be a powerful tool for studying cardiovascular variability series.6-8 However, it should be used with full knowledge of the methodology, especially if commercially available systems for heart rate variability analysis are used. In the application of this technique to a novel variability series such as AFCL, generated by the modulation of a reentrant mechanism clearly different from the physiological mechanism of sinus rhythm, attention should be paid to avoid tout court transposition of the heart rate variability analysis as the strained search of a sympathetically mediated oscillation as well as the arbitrary introduction of a sympathovagal balance index (page 2517: Methods; page 2519: Results1). However, we believe that if the identification of rhythms and their physiological interpretation is correctly performed, spectral analysis may provide some insight into the intriguing mechanisms of AFCL oscillations.

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Response

In response to the comments offered by Ravelli, our study applied power spectral analysis techniques to characterize the oscillations in atrial flutter cycle length variability in both the time and frequency domains. Previous attempts to investigate the mechanisms governing atrial flutter cycle length variability have only used time-domain methods and importantly have concluded that the beat-to-beat variability in atrial flutter cycle length could be explained entirely by an effect of the ventricular rate mediated via atrial stretch. Thus, previous studies had failed to consider the role of other potential control mechanisms, such as respiration and the autonomic nervous system.

In our study, DC filtering was used to remove the DC component of the data before power spectral analysis was performed. A detailed analysis of 3 frequency bands in the spectra was performed (band 1, <0.18 Hz; band 2, 0.18 to 0.60 Hz; and band 3, 0.6 to 2.2 Hz) at baseline and in response to a variety of interventions (isoproterenol, long-term β-adrenergic blockade, edrophonium, heart transplantation, and changes in respiratory rate).

The findings of our study provided additional confirmatory evidence of the importance of the ventricular contraction in mediating atrial flutter cycle length variability but also suggested that other mechanisms may be involved. We showed that band 3, which accounted for >60% of total spectral power, was significantly correlated with the ventricular rate. However, the lower-frequency bands (ie, <0.6 Hz) were not related to or altered by changes in the ventricular rate. Furthermore, we also demonstrated that band 2, which accounted for on average ~25% of total spectral power, appeared to be related to respiration because it could be modified in a similar direction as changes in respiratory rate (ie, increases in respiratory rate shifted the band 2 frequency peak to higher frequencies). Finally, we also found that on average ~10% of total spectral power was in frequencies <0.18 Hz. In about half the patients studied, this was associated with a distinct spectral peak in these very low frequencies, although in other patients no distinct peak was observed at these frequencies. On the basis of several observations, we concluded that although activity at frequencies <0.18 Hz was a relatively minor component of atrial flutter cycle length variability, it appeared to be related to changes in autonomic or sympathetic tone. These observations included the following findings: (1) isoproterenol altered the spectral characteristics of the band 1 component (ie, increased the power in band 1); (2) patients receiving long-term therapy with β-adrenergic blockers had a similar atrial flutter cycle length as patients not receiving β-blockers but virtually absent band 1 spectral activity; and (3) heart transplant recipients had markedly diminished atrial flutter cycle length variability.

Thus, in conclusion, our data are in agreement with Ravelli’s comments that atrial flutter cycle length variability is primarily modulated by both the ventricular rate and respiration. However, we do not believe that a potential role for autonomic nervous system effects has been fully excluded. We hope and expect that future studies will continue to examine this issue using the methodologies that we have newly applied to analysis of atrial flutter.

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Reappraisal of Mouth-to-Mouth Ventilation During Bystander-Initiated CPR

To the Editor:

The “reappraisal” of the literature on mouth-to-mouth ventilation during bystander-initiated CPR, by a working group of the Basic Life Support and Pediatric Life Support subcommittees of the American Heart Association (AHA), is misleading and incomplete. There is no convincing evidence that the low incidence of initiation of CPR out of hospital by lay bystanders is the result of fear of becoming infected by mouth-to-mouth ventilation. Such fear should not be promoted. If such fear exists, however, it should be mitigated by explaining that initiating CPR is safe and by carrying a pocket-size barrier for ventilation of strangers. The errors in this article concerning behavioral, educational, epidemiological, and logistics issues will be summarized in a separate letter by Braslows and Brennan.

Although the article says “. . . it is not intended to change any current AHA recommendations,” its publication has created confusion and the erroneous impression for laypersons and the media that in sudden coma, bystanders will save lives by merely pushing on the sternum (step C, circulation support). In cardiac arrest, oxygenated blood must be circulated to restore heartbeat and to keep the brain viable, requiring “head tilt plus blowing plus pumping.” The article suggests that mouth-to-mouth ventilation can be omitted in various forms of sudden loss of consciousness. Laypersons cannot differentiate between various forms of sudden coma and between the absence versus presence of a weak pulse. Coma always results in upper airway obstruction, which in turn can lead to apnea (particularly head trauma with impact apnea), intoxication, asphyxiation (common in children), and other causes of sudden coma with or without pulselessness. Indeed, step A was not even suggested by the authors during sternal compressions alone; at least the shoulders should be elevated so the head assumes a backward-tilt position that might support a backward tilt of the head; reference 26 in the article by Becker et al) will harm not only victims of sudden cardiac death but also those of trauma (particularly head trauma with impact apnea), intoxication, asphyxiation (common in children), and other causes of sudden coma with or without pulselessness. Indeed, step A was not even suggested by the authors during sternal compressions alone; at least the shoulders should be elevated so the head assumes a backward-tilt position that might support a backward-tilted position that might support a patent hypopharyngeal air passage (references 26 to 31 in the article by Becker et al). If there is still a faint pulse, steps A and B alone can often reverse the dying process. Even in cases of out-of-hospital sudden ventricular fibrillation, reoxygenation...
should precede countershocks, which will not result in heartbeat after >2 to 3 minutes of untreated ventricular fibrillation (reference 33 in the article by Becker et al).

This article’s “historical rationale” includes errors and omissions. For comatose humans with pulse and a natural air passage, the failure of chest-pressure methods to ventilate was documented in 1957. For humans both with or without pulse, the need to combine steps A, B, and C was documented in 1960: in 30 anesthetized and curarized adult patients and in 12 patients with cardiac arrest, ventilation produced by sternal compressions (step C) alone was measured, with or without backward tilt of the head, and with or without tracheal tube. In all 30 supine, horizontal, curarized patients, the unsupported head assumed a semiflexed position that resulted in zero tidal volumes by sternal compressions. With backward tilt of the head by elevation of the shoulders (a measure ignored in the article by Becker et al), tidal volumes were zero in 16 and only 25 to 200 mL in 14 patients. In all 12 patients with cardiac arrest, in spite of tracheal tube in place, forceful sternal compressions alone moved essentially no ventilation. The authors listed this article (reference 31) but might not have studied these, the only data published thus far on ventilation produced by sternal compressions in humans, because that article predates the earlier citations in MEDLINE. If chest pressure alone ventilates via an open airway, it would be with unpredictably inadequate tidal volumes below resting lung volume, which causes lung collapse, particularly in terminal patients with congested lungs. Hence, not elastic recoil of the chest, but positive-pressure inflations are needed to ventilate and oxygenate.

In discussing ventilation requirements, we agree with the article by Becker et al that low minute ventilation is sufficient to normalize arterial PO\textsubscript{2} and PCO\textsubscript{2}, during the low blood flow produced by external CPR in patients with sudden cardiac arrest. However, reoxygenation in asphyxiation (reference 29 of the article by Becker et al) and ventilation after restoration of spontaneous circulation, both with high blood flow, require hyperventilation, which is possible even with exhaled air (references 27, 29, and 30 of the article by Becker et al). Also, the authors’ concern about gastric insufflation by mouth-to-mouth ventilation is self-limiting (reference 29 of the article by Becker et al). Furthermore, the presently recommended ventilation:compression ratios of 2:15 or 1:5, which is a compromise, were based on a study in dogs, which was not quoted.

Animal data of ventilation by chest compressions alone, the main argument presented for the “reappraisal,” have no clinical relevance. Rats, pigs, and dogs have straight airways that do not obstruct, even in the absence of a tracheal tube. Humans have kinked airways that are obstructed even in the absence of a tracheal tube in human patients. Hence, forceful sternal compressions alone moved essentially no ventilation. Once the head is elevated such that the head cannot rest on the chest, the unsupported head assumes a semiflexed position that resulted in zero tidal volumes by sternal compressions. In 16 to 200 mL in 14 patients. The unsupported head assumes a semiflexed position that resulted in zero tidal volumes by sternal compressions. With backward tilt of the head by elevation of the shoulders (a measure ignored in the article by Becker et al), tidal volumes were zero in 16 and only 25 to 200 mL in 14 patients. The unsupported head assumes a semiflexed position that resulted in zero tidal volumes by sternal compressions. With backward tilt of the head by elevation of the shoulders (a measure ignored in the article by Becker et al), tidal volumes were zero in 16 and only 25 to 200 mL in 14 patients.

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In sudden ventricular fibrillation cardiac arrest with healthy lungs, arterial oxygen levels indeed remain near normal during a long period of no blood flow without CPR. This occurrence does not obviate the need for mouth-to-mouth ventilation, because sternal compressions alone in the presence of complete airway obstruction recirculate venous blood and cause arterial deoxygenation within <60 seconds.

Clinical studies by the excellent CPCR registry of Belgium (references 38 and 39 in the article by Becker et al) have been overinterpreted. The fact that 10% of prehospital cardiac arrest patients survived among those who were found on arrival of the ambulance with bystanders doing only chest compressions does not prove that mouth-to-mouth ventilation is not needed; it may have been used earlier or was not necessary because of short

arrest times with continued breathing (gasing). Moreover, the efficacy of individual resuscitation measures must be determined by physiological measurements in humans, rather than clinical, epidemiological, statistically significant outcome correlations, which do not prove cause-effect relationships on mechanisms.

What and how to teach the public must be extremely simple, with only 1 sequence of steps, ie, A-B-C. We agree with the authors’ first recommendation that CPR guidelines should not be changed at this time. Their second recommendation, to restudy details of mouth-to-mouth ventilation, is laudable but has low priority. Higher priority should be given to promoting the motivation and skill acquisition of the public with use of media and self-training systems; to early automatic external defibrillation by first responders; and to clinical documentation of cerebral resuscitation with mild hypothermia and blood pressure support.

We strongly disagree with their third recommendation that “clinical trials of chest compressions without mouth-to-mouth ventilation are ethical.” For the obvious reason that randomly withholding ventilation and thereby condoning the moving of deoxygenated blood would be irresponsible.

Response
We appreciate the thoughtful comments from Dr Safar and his colleagues regarding the role of mouth-to-mouth ventilation in adult CPR. The world will remain forever in the debt of James Elam, MD, who rediscovered the value of mouth-to-mouth ventilation in the 1950s, and Dr Safar, who meticulously documented the superiority of expired air ventilation to maintain blood oxygen levels. The combination of the Safar-Elam techniques with the chest compression rediscovered by Kouwenhoven, Jude, and Knickerbocker ushered in the modern era of CPR in the 1960s and has saved literally thousands of lives.

By the 1990s, however, a number of questions had arisen about CPR: Why is CPR so difficult for laypeople to learn and
remember? Why is CPR started on such a small percentage of witnessed cardiac arrests? Is poorly performed CPR perhaps capable of doing harm? Are there different causes of cardiac arrest that merit modifications of the traditional “pump and blow” of basic CPR?

Our Ventilation Working Group started its work with open minds. For this report, we decided to address the particular CPR feature of mouth-to-mouth ventilation. This focus was because of indirect evidence suggesting that the disagreeable features of mouth-to-mouth ventilation, combined with a growing fear of disease transmission, were making people reluctant to start CPR. Adding mouth-to-mouth ventilation to the task of chest compression may create a complex psychomotor skill that is simply too difficult for lay rescuers to remember at the time of an event as dramatic as sudden cardiac arrest.

We decided to review the entire body of research regarding chest compression and ventilation in CPR. We noted a scarcity of human research on this topic and think there are areas that merit further investigation. The purpose of the article was to stimulate interest and promote CPR research. We reached the following conclusions:

1. Two-person CPR with chest compression and mouth-to-mouth ventilation, performed by experienced professionals, is indisputably the most effective method to maintain brain and heart viability and to prolong ventricular fibrillation during cardiac arrest.

2. Single-person CPR with chest compression and mouth-to-mouth ventilation, performed by inexperienced laypeople, is probably less effective but should be taught without reservation in all AHA CPR courses.

3. We completely reject the hypothesis that mouth-to-mouth ventilation should be eliminated from CPR training. This conclusion is a distortion of our work and is without valid scientific support.

4. Single-person CPR with chest compression but without mouth-to-mouth ventilation is suboptimal and is not recommended by the AHA. However, the optimal single rescuer technique for lay rescuers is not known because it must both “optimize” the physiology of promoting oxygenated blood flow as well as promote maximal community-wide implementation (which may be affected by ease of training, skill retention, willingness to act, and ability). Animal studies suggest that mouth-to-mouth plus chest compression improves oxygen saturation. But as the single rescuer switches from compression to ventilation, fewer compressions are performed, which may adversely impact circulation of blood and myocardial perfusion. The attainment of additional knowledge on these competing processes seems important, and these processes have not yet been well studied.

5. Human data on lay single-rescuer CPR without mouth-to-mouth ventilation are scant. The data from the Belgian CPR registry and reports from the Netherlands fail to find any disadvantage for patients who received compression but not mouth-to-mouth ventilation. Moreover, our BLS instructors need practical guidance when confronted with a CPR learner they are certain will not perform mouth-to-mouth ventilation in an emergency. In such situations, the CPR learner must be encouraged to “at least do something.” There is good evidence that chest compression alone is far better than no rescue attempts at all.

6. We found a theme in the research regarding adults who collapse with sudden fibrillation and well-oxygenated blood. These individuals compose a significant proportion of adult out-of-hospital arrests and may maintain acceptable blood oxygen levels for a few minutes after collapse with chest compression alone. We could hypothesize that mouth-to-mouth ventilation could be delayed for these patients for a few minutes. This window of time, however, is limited, and ventilation will eventually be required. These data suggest an avenue for future research. We encourage creative research on this hypothesis; it is ethical if properly designed.

7. Further research is needed to improve community CPR rates. We cannot expect lay rescuers to make judgments about the cause of collapse nor to switch from 1 rescue sequence to another based on the passage of time. For successful community CPR, the techniques must be easy to learn, remember, and perform during a crisis. We have called for more research because we agree that our current data do not provide complete answers to some important questions. We believe that the public is best served by conducting research to answer unresolved issues.

8. Finally, readers should not mistake “confusion” with legitimate controversy. That letters with divergent viewpoints are printed reflects the fact that scientific controversy still exists. In our deliberations, we adhere to the principles of evidence-based decision making. CPR has been advocated for nearly 40 years, yet most victims of cardiac arrest never receive CPR efforts, and survival rates are poor. These controversies have existed for too long without resolution. Let us resolve to perform the research to turn scientific controversy into consensus among scientists. This duty falls to all of us as guardians of public health and safety.

The Safar Resuscitation Research Center has been a beacon of quality research on the topic of CPR. We appreciate their comments and their interest in this topic. We are sure they will join us in encouraging more research evaluation in this rich and dynamic area of resuscitation.

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Layperson CPR

To the Editor:

“A Reappraisal of Mouth-to-Mouth Ventilation During Bystander-Initiated Cardiopulmonary Resuscitation”1 argues that laypersons do not perform CPR because of concerns about mouth-to-mouth resuscitation. On the basis of this conclusion, the authors explore physiological issues related to exclusion of mouth-to-mouth as a component skill. With media coverage focusing on the notions that mouth-to-mouth resuscitation poses significant risk of infection and CPR without mouth-to-mouth breathing can be effective, the Special Report’s publication has done more harm than good to efforts to train laypersons and encourage them to initiate CPR. We believe the following facts about out-of-hospital cardiac arrest and bystander action are more explanatory of the failure of bystander CPR than the hypothesis offered in the Special Report:

1. CPR is not performed by bystanders because most laypersons are not trained, and training is not targeted to those likely to be present at the scene of a cardiac arrest.—At least 74% of arrests occur in the home2 where the typical family bystander is 55 years old.3 CPR trainees average 31, with a small minority 55 or older. Fewer than 8% of participants take CPR training because they live with someone at elevated risk of heart attack.4,5
2. A layperson’s decision to respond to an emergency depends on a set of factors unlike those affecting medical professionals.—According to the psychological research on “helping behavior,”6,7 the decision to act depends on acknowledging that the situation exists and having confidence in one’s abilities. An unaccustomed concern of laypersons, such as disease transmission, will be but one, most likely trivial, factor. One article8 cited in the Special Report confirms that reluctance to perform mouth-to-mouth on family members and friends is uncommon among laypersons. The only study cited in which lay bystanders were interviewed9 reveals that they do not hesitate in helping the victim even in the presence of disagreeable stimuli, nor do they advance concerns about HIV.

3. Trained laypersons cannot competently perform CPR.—Immediately after training, CPR trainees are not competent in performing ventilations of sufficient volume and compressions of sufficient depth.10 Because feelings of competence are critical to the decision to take action,6,7 lack of competence may be responsible in part for low rates of initiation.

We urge that the American Heart Association (AHA) promulgate the authors’ recommendation that “current CPR guidelines for performing mouth-to-mouth ventilation during CPR should not be changed” and maintain that the risk of infection presented by mouth-to-mouth ventilations is minimal. We further urge that (1) the argument that laypersons do not initiate CPR because of fear of performing mouth-to-mouth breaths be disavowed unless new studies reveal this to be a significant contributory cause; (2) CPR training be targeted at laypersons with high exposure to individuals with heart disease; (3) CPR training programs produce, at the very least, competent CPR performance immediately after training; (4) the Emergency Cardiovascular Care Committee and training organizations address laypersons and medical professionals as separate populations requiring different curricula, teaching methods, and expert committees; and (5) the AHA initiate and support research related to lay bystander response.

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7. Mogilner and Brennan. The importance of educational research on the teaching of bystander CPR. Although the issue of “why people don’t perform CPR” was not the focus of our recent article, we agree that these are important issues. Braslows and Brennan have made many critical contributions to our understanding of how people learn CPR. They have been pioneers in developing techniques for objective evaluation of CPR training outcomes, peer-training methods, and video self-teaching methods. As experts in the education of layperson CPR, their insights and perspective are valuable.

We appreciate the opportunity to expand several points of the article:

1. “Fear of AIDS” is not the primary reason people fail to perform CPR, it is only one of the barriers to CPR performance. We contend that the primary reason CPR is not performed is related to training obstacles, not fear of infection. Failure to provide effective CPR by the lay rescuer represents a real failure to save lives. Additional resources must be targeted to improve lay bystander response.

2. In our article, we discuss fear of disease transmission along with several other issues (including pulmonary aspiration, carbon dioxide, and time taken away from compression). These are possible adverse side effects specific to mouth-to-mouth ventilation. Few medical therapies are without side effects; CPR and mouth-to-mouth ventilation are no exception. Our responsible reappraisal of CPR had to include a consideration of these side effects. After careful review, the Task Force concluded that “Current guidelines for performing mouth-to-mouth ventilation during CPR should not be changed.” Suggestions that the article states otherwise misrepresent the article.

3. We think bystanders considering mouth-to-mouth contact do voice concerns about HIV. The available data support this statement. Locke et al reported on 975 survey respondents, of whom 80% were laypersons (see article referenced above). They concluded that laypersons are reluctant to provide mouth-to-mouth ventilation and that this layperson reluctance is a barrier to CPR provision even in relatives and close friends. Moreover, we know from our training network that concerns over disease transmission (and most specifically HIV) continue to be an issue raised by participants taking CPR courses. To answer these concerns, we first respond that most CPR will be performed on family members and that the actual risk of disease transmission is extremely low. However, for those who may delay or not begin CPR because of persistent fears, we have a duty to at least provide clear instructions to begin immediate chest compression. We know that chest compression alone has been lifesaving for many. We know that the lowest survival rate occurs when no efforts at resuscitation are attempted.

4. Are research funds better spent on determining improved methods to teach CPR to the public, or should we be concentrating on further research into the physiology and improved mechanics of the “pump and blow” techniques? There has been a scarcity of research on the methodology of teaching CPR to the public. By contrast, relatively more research has been published on the physiology of blood flow.
and survival with CPR techniques. However, it seems to us that both are still required. Although educational issues are important, no one would advocate the teaching of an ineffective technique simply because it was easy to teach, well retained, and easy to perform. Likewise, no technique would be useful, even if it resulted in near-perfect blood flow during CPR, if bystanders could never acquire the skills to perform the task. Some balance is necessary. Our position on funding is that the entire field of resuscitation and CPR research has been underfunded relative to its importance for public safety. It doesn’t make sense to argue “over mere nickels” when we need serious dollars to make headway and save more lives.

We look forward to continued contributions and perspectives on CPR training and techniques, for this intervention is a major public health issue that can directly impact the lives of many citizens.

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Circulation. 1998;98:608-610
doi: 10.1161/01.CIR.98.6.608

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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