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## Expanding application of the Wiggers diagram to teach cardiovascular physiology

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**Mitchell JR, Wang J.** Expanding application of the Wiggers diagram to teach cardiovascular physiology. *Adv Physiol Educ* 38: 170–175, 2014; doi:10.1152/advan.00123.2013.—Dr. Carl Wiggers' careful observations have provided a meaningful resource for students to learn how the heart works. Throughout the many years from his initial reports, the Wiggers diagram has been used, in various degrees of complexity, as a fundamental tool for cardiovascular instruction. Often, the various electrical and mechanical plots are the novice learner's first exposure to simulated data. As the various temporal relationships throughout a heartbeat could simply be memorized, the challenge for the cardiovascular instructor is to engage the learner so the underlying mechanisms governing the changing electrical and mechanical events are truly understood. Based on experience, we suggest some additions to the Wiggers diagram that are not commonly used to enhance cardiovascular pedagogy. For example, these additions could be, but are not limited to, introducing the concept of energy waves and their role in influencing pressure and flow in health and disease. Also, integrating concepts of exercise physiology, and the differences in cardiac function and hemodynamics between an elite athlete and normal subject, can have a profound impact on student engagement. In describing the relationship between electrical and mechanical events, the instructor may find the introduction of premature ventricular contractions as a useful tool to further understanding of this important principle. It is our hope that these examples can aid cardiovascular instructors to engage their learners and promote fundamental understanding at the expense of simple memorization.

Wiggers diagram; energy wave; incisura; early diastolic filling

FOR >90 YEARS, the Wiggers diagram has been a fundamental tool for teaching cardiovascular (CV) physiology, with some of his earliest descriptions of the heart and circulation published in 1915 (18). The lack of significant additions or changes from Dr. Wiggers' original observations is a testament to his careful work. In describing the various auditory, electrical, pressure, volume, and blood flow changes, the novice learner is afforded an opportunity to view electrical and mechanical temporal relationships throughout a heartbeat. Depending on the academic level, the diagram can often be a student's first exposure to simulated recorded data. Importantly, it affords the opportunity for the CV instructor to introduce concepts through graphical interpretation versus strictly textual descriptors and flow charts.

A common challenge to the CV instructor is finding ways to engage the students and stress the importance of understanding the various traces as underlying determinants of significant cardiac events versus simply just memorizing the temporal deviations. In this regard, the CV instructor should not be hesitant to introduce some additions to the Wiggers diagram to aid in student engagement and understanding. For example, the

introduction of how the generation and reflection of an energy wave can influence blood pressure, and how its velocity changes in aged/diseased blood vessels, can be useful in CV pedagogy. Also, the use of a comparison of an athletic heart to a normal heart as it relates to ventricular filling, and therefore performance, could facilitate understanding and interpretation. Along these lines, the addition of an abnormal electrical event causing a premature ventricular contraction (PVC) can aid in understanding the important relationship between excitation-contraction coupling and ventricular filling time as it relates to cardiac performance as seen by electrical and blood pressure and flow changes. Thus, this report will highlight an opportunity to introduce energy waves in an attempt to better understand both normal and pathological pressure observances as well as incorporating how different perturbations from normal physiology can change the various recordings and encourage student engagement and, therefore, understanding.

*The Wiggers Diagram*

Depending on the source, Wiggers' diagrams can vary in detail and number of variables presented. Regardless, all provide essential information on how the normal heart functions with a minimum description of pressure changes during phases of diastole and systole. Figure 1A accomplishes this by showing the temporal relationships between left ventricular (LV) pressure ( $P_{LV}$ ), LV volume ( $V_{LV}$ ), left atrial (LA) pressure ( $P_{LA}$ ), and aortic pressure ( $P_{AO}$ ). Figure 1A also shows important events such as mitral valve opening and closing, aortic valve opening and closing,  $P_{AO}$  incisura, early diastolic filling (EDF), and the a, c, and v waves of  $P_{LA}$ . If diastole is considered to start from the time of aortic valve closure and ending upon the closure of the mitral valve, one can identify three distinct phases: 1) isovolumic relaxation, 2) EDF, and 3) atrial contraction. Figure 1A shows the period of isovolumic relaxation (denoted as the hashed area between aortic valve closure and mitral valve opening) describing a precipitous fall in  $P_{LV}$  with no change in  $V_{LV}$ , EDF occurring immediately after the mitral valve opens and accounting for ~80% of the end-diastolic volume (EDV), and LA contraction causing the a wave of  $P_{LA}$  accounting for the remaining 20% of the EDV. Calculating the stroke volume (SV) of the LV as EDV minus end-systolic volume (ESV), one could estimate it to be ~80 ml for this normal subject (125 ml – 45 ml = 80 ml). Finally, important auditory events are noted in Fig. 1A for *sounds 1* and *2* (mitral valve closure and aortic valve closure, respectively) and heart *sounds 3* and *4*. Although exaggerated heart *sounds 3* and *4* are most often associated to pathological conditions, one could comment on heart *sound 3* as being considered normal in young people (under 40 yr of age) with high early diastolic LV inflow velocity (11). This enhanced acceleration of flow into the ventricles during early diastole could provide

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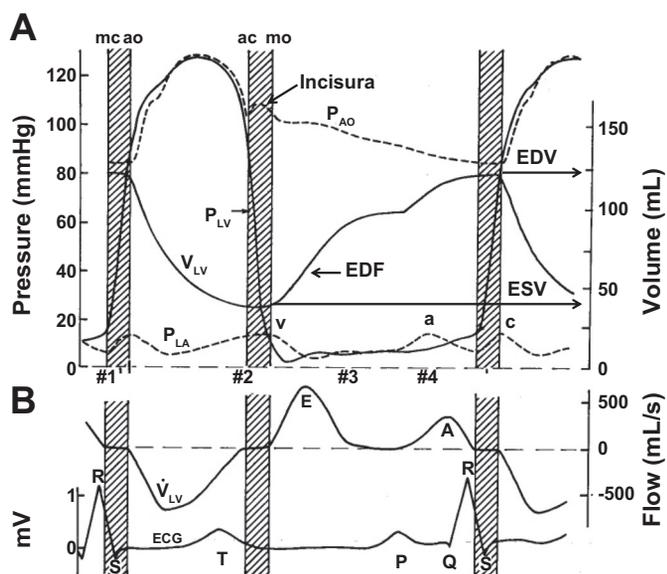


Fig. 1. A: temporal relationships between left ventricular (LV) pressure ( $P_{LV}$ ), LV volume ( $V_{LV}$ ), left atrial (LA) pressure ( $P_{LA}$ ), and aortic pressure ( $P_{AO}$ ). Shown in A are mitral valve opening and closing (mo and mc, respectively), aortic valve opening and closing (ao and ac, respectively),  $P_{AO}$  incisura, early diastolic filling (EDF), end-diastolic volume (EDV), end-systolic volume (ESV), and the a, c, and v waves of  $P_{LA}$ . Auditory events are shown in A for sounds 1 and 2 (mitral valve closure and aortic valve closure, respectively) and heart sounds 3 and 4. B: blood flow out of the LV during the ejection phase of systole ( $\dot{V}_{LV}$ ), early diastolic blood flow into the LV (E wave), blood flow into the LV during LA contraction (A wave), and a standard lead II ECG showing LA depolarization, LV depolarization, and LV repolarization (P wave, QRS complex, and T wave, respectively). (Unpublished figure provided courtesy of Dr. John V. Tyberg and Dr. Henk E. D. J. ter Keurs.)

the CV instructor an opportunity to introduce the concept of diastolic suction during ventricular relaxation. Briefly, relaxation of the ventricle, in a closed, fluid-filled system, causes a degree of suction. In young people with healthy myocardia, an enhanced LV relaxation, and therefore suction, is generally attributed to the enhanced acceleration of flow into the chamber and the creation of heart sound 3 (11). Figure 1B shows blood flow out of the LV during the ejection phase of systole, early diastolic blood flow into the LV (E wave), blood flow into the LV during LA contraction (A wave), and a standard lead II ECG showing LA depolarization, LV depolarization, and LV repolarization (P wave, QRS complex, and T wave, respectively).

As we can see, a student could learn much about the normal functioning of the heart as it relates to the periods of systole and diastole, influences and timings of different valves opening and closing, timing of pressure, and therefore volume and flow changes, as well as the important temporal relationship between the electrical and mechanical events of the heart. As stated earlier, the challenge for the CV instructor is to engage the students to a point in which they truly comprehend the important cardiac events versus simply memorizing the temporal deviations in the various traces.

### $P_{AO}$ and Energy Waves

All Wiggers' diagrams that portray  $P_{AO}$  throughout a cardiac cycle provide an opportunity to introduce the concept of energy waves and their possible implications in cardiac function and

hemodynamics. Often considered an esoteric, somewhat complex topic, energy waves can be simplified for the undergraduate, graduate, or medical student. It is important to, first, define what an energy wave is and how it is created and, second, indicate the potential implications for the study of cardiac function and hemodynamics.

### What Is an Energy Wave and How Is It Created?

There has been no single precise definition of a wave; however, an intuitive view is generally preferable: a wave is a disturbance transmitting energy but not necessarily matter as it propagates in time and distance. For example, a water wave can increase or decrease pressure, and accelerate or decelerate flow concurrently, with respect to the type (compression or decompression) and direction of a wave (17). One may also visualize such a phenomenon by initializing a disturbance transmitting across a "Slinky toy" without permanent translocation of any segment of the spring (Fig. 2). For example, a wave (disturbance), initiated by a push, generates a compression wave, which always increases pressure as it propagates (shown as the densely packed regions in Fig. 2). Note that when a compression wave transmits along the direction of flow, it simultaneously increases pressure and accelerates flow; when a compression wave transmits against the direction of flow, it increases pressure and decelerates flow. Conversely, a wave introduced by a pull generates a decompression wave, which always decreases pressure as it propagates (shown as the loosely packed spring regions in Fig. 2). Similarly, when a decompression wave transmits along the direction of flow, it concurrently decreases pressure and decelerates flow; when a decompression wave propagates against the direction of flow, it decreases pressure and accelerates flow simultaneously (17). The speed of a wave is associated with the restoring force yielded by the elastic property of the blood vessel and blood inertia, which makes wave velocity much faster than the velocity of blood itself. For example, the peak flow velocity in the central aorta is  $\sim 100$  cm/s, whereas the average aortic wave speed is  $\sim 5\text{--}8$  m/s at the same location (13).

There are two types of waves in blood vessels: compression and decompression waves. The former is created initially during LV contraction (15), which can be reflected positively as a backward compression wave (BCW) from a downstream "closed-end" reflection site (e.g., vessel bifurcation or a clotted vessel). A forward compression wave (FCW) may also be reflected as a backward decompression wave, reflected from an "open-end" reflection site. This often occurs at a higher daughter-to-parent cross-sectional area ratio such as at the junction of the abdominal aorta and renal arteries. Note that an arterial

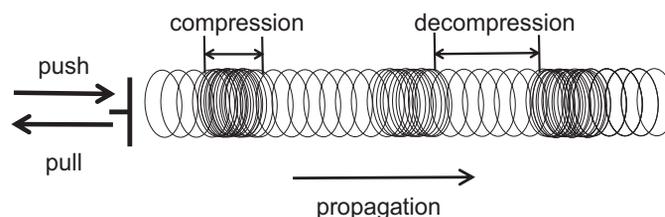


Fig. 2. The definition of a wave as a propagating disturbance is presented using a Slinky toy. A disturbance generated by a push is a compression wave (packed region), whereas a disturbance generated by a pull is a decompression wave (loose region).

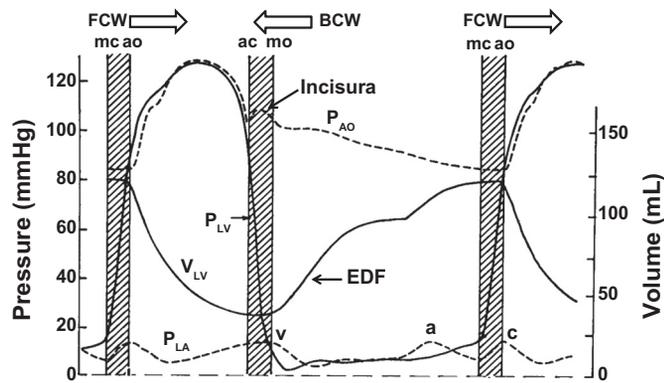


Fig. 3. The normal timing of the generation of a forward compression wave (FCW) and arrival of a backward compression wave (BCW). (Permission to alter the original unpublished figure provided courtesy of Dr. John V. Tyberg and Dr. Henk E. D. J. ter Keurs).

bifurcation can function as an open-end reflector (the type of the reflected wave is opposite to that of the incident wave), a closed-end reflector (the type of the reflected wave is the same as that of the incident wave), or a reflectionless junction [occurs when an area ratio of daughter-to-parent vessel equals 1.0 (the wall thickness of the daughter vessel is the same as that of the parent vessel) or equals 1.15 when wall thickness changes in proportion to the radius in the daughter vessel (14)]. To keep things simple for the novice learner, we will only focus on the possible hemodynamic impact of compression waves. In Fig. 3, we can see the timing of both the generation of a FCW and the arrival of a BCW in this hypothetical normal physiological example. As the BCW travels back toward the heart at the same speed as the FCW travels away, it consistently arrives at, or just after, the aortic valve closes (end systole) (16). The magnitude of a wave can be quantified by the amount of energy it carries passing through a unit area per unit time (in  $J \cdot m^{-2} \cdot s^{-1}$  or  $watts/m^2$ ). In practice, the energy of a wave in arteries can be calculated as the product of simultaneously measured pressure difference multiplied by the blood flow velocity difference, as detailed by Parker and Jones (15). In addition, the timings for the forward- and backward-traveling waves can be recognized using the same analysis, as identified by the onset of a surge in wave energy (16).

*Cardiac Function and Hemodynamic Implications*

The potential importance of energy waves, specifically, the arrival of the BCW and its influence on  $P_{AO}$ , is never more important than when we look at the possible implications in disease and aging. It has been well received that  $P_{AO}$  and flow waveforms, which can be assessed rather accurately using modern medical devices, can contain important information of the conditions of downstream organs. For example, an older subject, and/or one suffering from atherosclerosis, would have increased velocity of energy waves travelling in noncompliant, stiff and ridged vessels. In the case of a BCW, this could cause it to arrive sooner during the cardiac cycle, possibly during the ejection phase of systole. This can be demonstrated in an experimental animal model by artificially creating a closer reflection site versus increasing the wave speed per se. As shown in Fig. 4, a brief constriction of the upper abdominal aorta caused marked changes in  $P_{AO}$  (dashed trace in A) and

flow (dashed trace in B) compared with readings before the aortic constriction (solid traces). For convenience, Fig. 4C shows the cumulative changes in both  $P_{AO}$  ( $\Delta P_{AO}$ ) and flow ( $\Delta Q_{AO}$ ) caused by the earlier arrival of the BCW with aortic constriction [denoted in Fig. 4, A–C, by the vertical dashed line; the normal arrival of a BCW would be at, or just after, aortic valve closure (see Fig. 3)]. When analyzing the pressure effects of a BCW (Fig. 4A), we can see that peak systolic  $P_{AO}$  increased during constriction (dashed trace) compared with the normal beat (solid line), whereas LV output ( $Q_{AO}$ ) decreased (Fig. 4B, dashed trace). In our example, an earlier arriving BCW results in a cumulative  $P_{AO}$  increase of  $\sim 12$  mmHg and a flow decrease of  $\sim 1.5$  l/min (Fig. 4C, dashed trace and solid trace, respectively). Note that the timing of the aortic

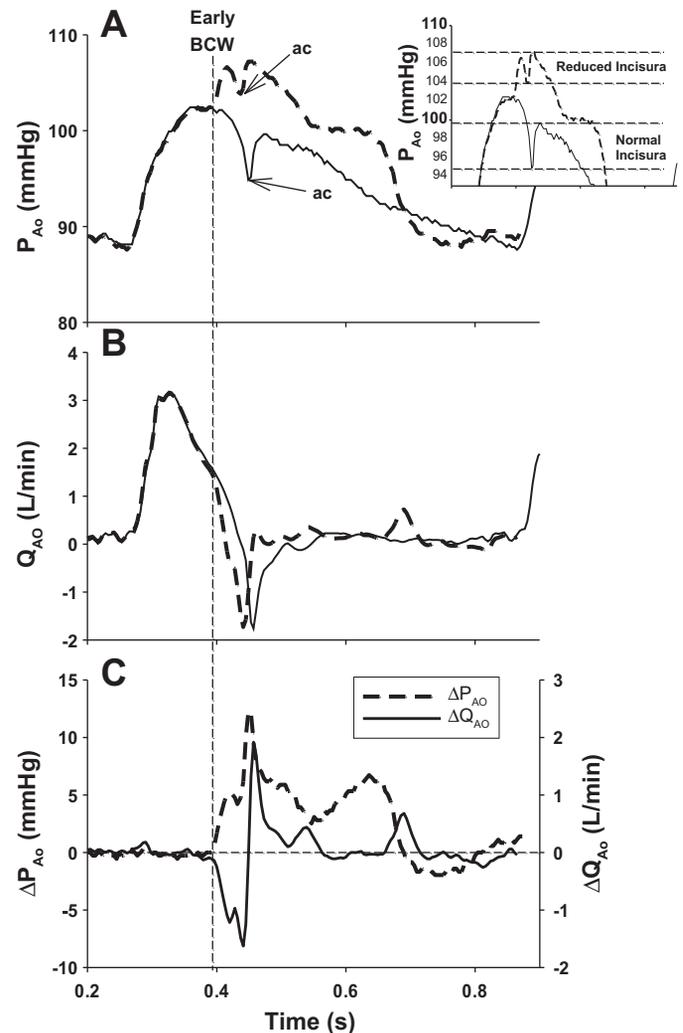


Fig. 4. Comparison of  $P_{AO}$  (A) and aortic flow changes ( $Q_{AO}$ ; B) between a normal beat (solid trace) and one taken from a simulated pathological example (dashed trace). Aortic constriction caused an earlier arrival of the BCW [normal arrival of BCW would be at, or just after, aortic valve closure (see Fig. 3)], increased peak systolic  $P_{AO}$  (A; dashed trace), and decreased  $Q_{AO}$  (B; dashed trace) compared with the normal beat (solid traces). C: cumulative increase in  $P_{AO}$  (dashed trace) and decrease in  $Q_{AO}$  (solid trace) caused by the earlier arrival of the BCW. Note that the magnitude of the incisura (A, inset) was smaller after the constriction of the aorta (dashed trace) compared with that of the control beat (solid trace) and that aortic valve closure occurred earlier in the cycle, suggesting that the BCW contributes to the magnitude and generation of the incisura itself and duration of the ejection phase of systole.

closure occurs sooner with the constriction compared with the normal beat. Also, the magnitude of the incisura, the pressure jump traditionally associated with the closure of the aortic valve, was smaller after the constriction of the aorta compared with that of the control beat (Fig. 4A, *inset*), suggesting that the BCW may contribute to the magnitude and generation of the incisura itself and, in the case of a pathological example, may shorten the ejection period. Highlighting these changes provides a good opportunity to aid in student engagement by introducing the pathophysiological implications of aging and/or atherosclerosis and the increased speed of the BCW (which would have the same effect by causing the BCW to arrive sooner during the cardiac cycle) in noncompliant vessels contributing to systolic hypertension (high blood pressure). Thus, the Wiggers diagram and a simple  $P_{AO}$  trace afford the opportunity to not only introduce a potentially complex concept in a very basic way but also to engage and challenge the student by understanding the implications in a pathological condition.

#### An Athlete's Heart and EDF

As many students partake in some form of recreational sporting activity or have previously/currently partake in elite athletics, providing just a small example of the possible CV differences between a hypothetical normal subject and endurance athlete exercising at the same submaximal intensity can prove immensely beneficial. Compared with the normal subject (Fig. 5A,a), the Wiggers diagram for the endurance-trained subject (Fig. 5B,a) can be manipulated to simply show an increased E wave (Fig. 5A,b and B,b) describing the increased EDF and, thus, EDV. Keeping the example simple by assuming the heart rate (HR; and therefore filling time) is matched for the exercise intensity and the atrial contribution to EDV and ESV do not change significantly between the subjects (8, 9), the student can now clearly recognize the greater SV (and therefore LV outflow) (5, 19) for the endurance-trained subject (160 ml – 25 ml = 135 ml; Fig. 5B) compared with the normal

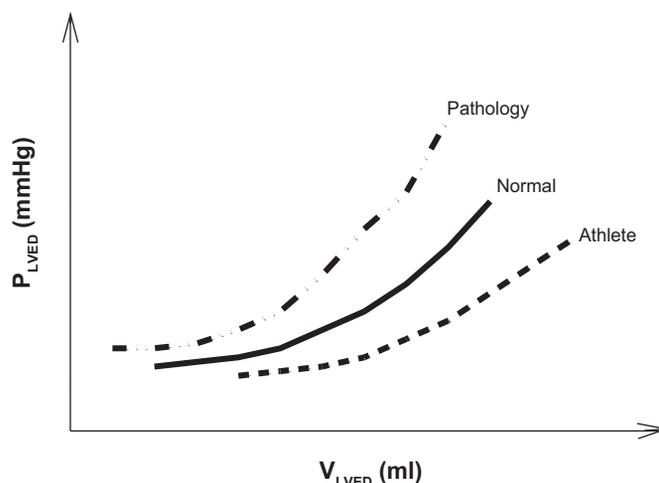


Fig. 6. Relationship between LV end-diastolic pressure ( $P_{LVED}$ ) and volume ( $V_{LVED}$ ) for normal (solid line), athlete (dashed line), and pathological (dashed-dotted line) subjects. The rightward-downward shift of the athlete demonstrates increased compliance, whereas the leftward-upward shift of the pathological subject demonstrates decreased compliance, compared with the normal subject.

subject (140 ml – 25 ml = 115 ml; Fig. 5A). Depending on the degree of depth and complexity that the instructor wishes to take, the pressure and volume differences between the examples could be highlighted to show the increased pressure gradient between the LA and LV during EDF resulting in the increased E wave/peak diastolic filling rate (4) and slope of the  $V_{LV}$  curve during EDF. A useful way to demonstrate the myocardial adaptation in the athlete compared with a normal subject and/or pathological subject would be to plot the pressure-volume curves. Upon viewing Fig. 6, the student can clearly see the increased compliance (rightward-downward shift) of the athlete (7) compared with a normal subject and that of a pathological subject demonstrating a noncompliant LV. One may choose to describe the relationship as a more compliant ven-

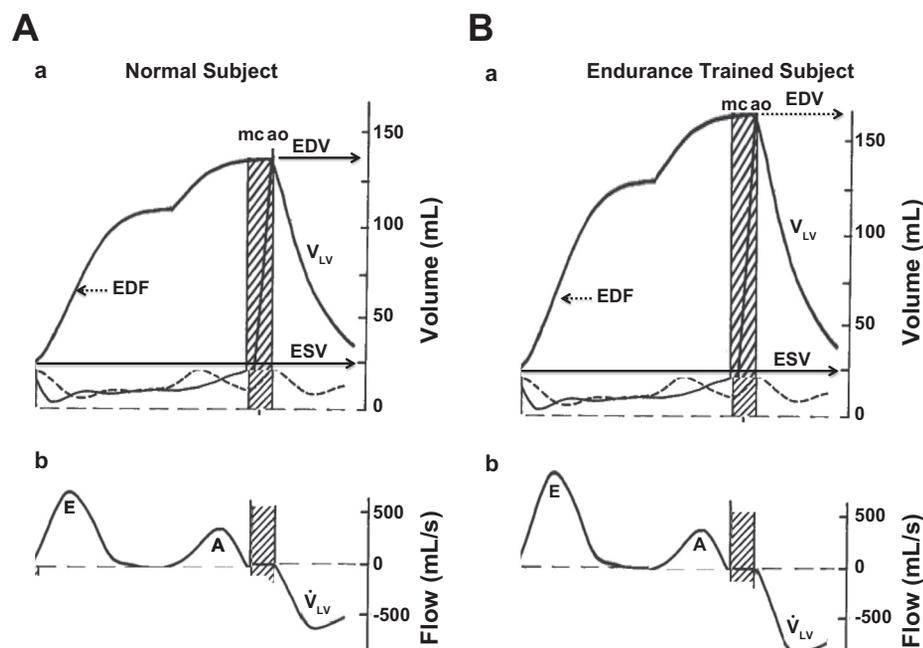


Fig. 5. A and B: hypothetical volume traces (a) and flows (b) for a normal subject (A) and endurance-trained subject (B) both exercising at the same submaximal intensity. The endurance-trained subject showed an increased E wave (b) compared with the normal subject describing the increased EDF (a) and, thus, EDV (a) (dotted line compared with solid line). Assuming heart rate (therefore filling time), atrial contribution (A wave; b) to EDV, and ESV (a) are the same for both subjects, the greater stroke volume (and therefore  $\dot{V}_{LV}$ ; b) for the endurance-trained subject (160 ml – 25 ml = 135 ml) compared with the normal subject (140 ml – 25 ml = 115 ml) can be deduced. (Permission to alter the original unpublished figure provided courtesy of Dr. John V. Tyberg and Dr. Henk E. D. J. ter Keurs.)

tricle will tend to fill greater at lower pressures compared with normal subjects or subjects with a diseased myocardium (pathology) characterized by stiff, ridged ventricles (6, 20). Naturally, the athletic example has many underlying causes as to “why” the changes occur [i.e., myocardial and pericardial adaptations, increased LV cavity dimension, increased rate of LV pressure decline (increased diastolic suction), increased rate of calcium uptake, etc.], which would serve as a natural segue to further exercise physiology instruction if appropriate. One could also include how the different parameters change from rest to maximum exercise for both subjects [i.e., influence of increasing HR on time spent in diastole (and therefore preload), systole, isovolumic relaxation and contraction [including indexes of ventricular relaxation (suction) and contractility, respectively], etc.], but that, again, would be up to the instructor to choose the degree of detail they wish to deliver to the specific class.

### Electrical and Mechanical Relationships

Upon reviewing Fig. 1, we can see an important tenet of CV physiology: electrical events always precede mechanical events. This is clearly shown when we compare the temporal relationship between the ECG (Fig. 1B) and various pressure changes (Fig. 1A). For example, ventricular depolarization (characterized by the QRS complex) occurs just before the mechanical event of isovolumic contraction (rise in  $P_{LV}$ ), whereas LV repolarization occurs just before the mechanical event of isovolumic relaxation (fall in  $P_{LV}$ ). Along these same lines, the electrical P wave (LA depolarization) occurs immediately before LA contraction (the a wave of  $P_{LA}$ ). Once again, a student who is a skilled memorizer could be successful in recreating these events without completely understanding the underlying importance governing the relationship. This only becomes apparent when examples of how changes in electrical activity can alter the mechanics, and therefore performance, of the ventricle.

### PVC

PVCs are extra heartbeats that begin in one of the ventricles thereby skipping the normal conduction pathway that starts from the sinoatrial node (1). These premature discharges are typically due to a general “irritability” of the ventricular myocardium brought on by a number of possible causes (examples including electrolyte imbalances, hypoxia, excessive caffeine intake, or certain medications). PVCs are often characterized by a subsequent resetting of the electrical system causing a brief prolongation before the resumption of the next heartbeat (2). Thus, this example of an electrical disturbance provides a unique opportunity to describe how changes in the time of ventricular depolarization and repolarization can impact ventricular filling and, therefore, performance (output). Of note, PVCs are common occurrences in both normal and athletic populations (3, 12) and are normally benign in nature with no implications to short- or long-term morbidity or mortality (10).

Figure 7 shows the temporal relationships between mechanical descriptors  $P_{LV}$  and  $P_{AO}$  (A),  $Q_{AO}$  (B), and an electrical marker lead II ECG (C) in an experimental animal model. The

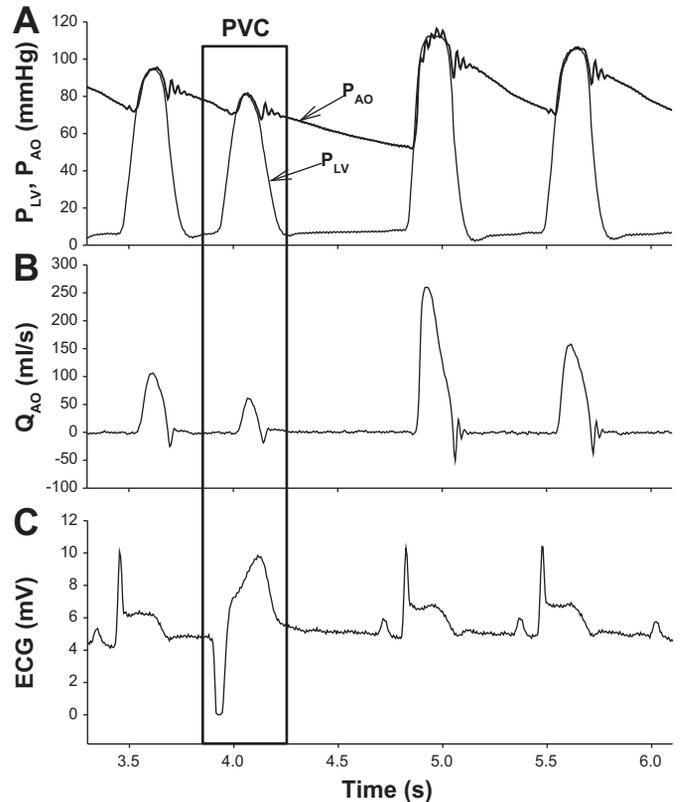


Fig. 7. An example of a premature ventricular contraction (PVC). Temporal relationships between  $P_{LV}$  and  $P_{AO}$  (A),  $Q_{AO}$  (B), and ECG (C) are shown for a single heartbeat immediately before a PVC, during a PVC (highlighted in box), and for two heartbeats immediately after a PVC. The ECG (C) distinguishes the abnormal electrical activity from the normal heartbeats. The shortened diastole due to the PVC results in decreased LV output (B; integrated area under  $Q_{AO}$ ) and force of contraction, as shown by reduced  $P_{LV}$  and  $P_{AO}$  (A). The prolonged interval during the electrical “resetting” of the ventricle affords an increased time spent in diastole and, therefore, increased  $Q_{AO}$  and both  $P_{LV}$  and  $P_{AO}$  compared with the normal beat preceding the identified PVC.

hemodynamic monitoring clearly demonstrates how an electrical abnormality can affect the mechanical performance of the LV *in vivo*. The ECG (Fig. 7C) clearly distinguishes the abnormal electrical activity from the normal heartbeats as highlighted by the large electrical deflection with no preceding P wave (highlighted in the box labeled PVC) and ubiquitously observed compensatory pause before the next beat. The shortened diastole due to the PVC results in decreased LV filling (preload not shown) and, therefore, decreased LV output (integrated area under  $Q_{AO}$  in Fig. 7B). The reduction in LV preload also manifests in a decreased force of contraction, as shown by reduced  $P_{LV}$  and  $P_{AO}$ . The utility of using a PVC is highlighted as the prolonged interval during the electrical “resetting” of the ventricle affords an increased time spent in diastole and, therefore, increased ventricular performance on the very next beat. As seen in Fig. 7B,  $Q_{AO}$  increased and both  $P_{LV}$  and  $P_{AO}$  increased compared with the normal beat preceding the identified PVC. Depending on the academic level, this would be an excellent opportunity to either introduce or reemphasize the Frank-Starling relationship and/or sarcomere length-force relationship as it relates to a changing ventricular preload.

### Conclusions

Teaching fundamental CV physiology to novice learners can be challenging. Many CV instructors use the ubiquitous Wiggers diagram as an essential tool to relate various electrical and mechanical events throughout a heartbeat. To the simple memorizer, the various events are nothing more than line deviations on paper that can be reproduced with ease. Based on experience, we have found that the introduction of different applications to the traditional Wiggers diagram can be an excellent instructional tool to recruit student engagement. We have provided just a sample of possibilities that could be added to push the novice learner into a willful effort to understand the important fundamental events governing the generation of a heartbeat and blood flow. It is the hope that the process further engages student interest in CV physiology and, more importantly, impresses the importance of conceptual realization and critical thinking versus simple rote learning.

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All animal experiments were approved by the institutional animal care committee whose criteria are consistent with those of the American Physiological Society.

### DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

### AUTHOR CONTRIBUTIONS

Author contributions: J.R.M. and J.-J.W. conception and design of research; J.R.M. and J.-J.W. analyzed data; J.R.M. and J.-J.W. interpreted results of experiments; J.R.M. and J.-J.W. prepared figures; J.R.M. and J.-J.W. drafted manuscript; J.R.M. and J.-J.W. edited and revised manuscript; J.R.M. and J.-J.W. approved final version of manuscript; J.-J.W. performed experiments.

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