

# Diagrammatic Reasoning with Interactive P-V Curves

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## Abstract

*In this paper, we describe a case study that supports pathophysiological reasoning using a combination of diagrammatic and qualitative reasoning. Pressure-Volume (P-V) representations of the cardiac cycle have been traditionally used to represent clinical situations, from textbooks to research articles. This has led us to develop an interactive version of the P-V curve which supports diagrammatic inference, based on user-driven modification of essential parameters of the P-V curve. Because diagrammatic inference cannot account for temporal adaptation of the cardio-vascular system, we have extended the diagrammatic reasoning system to incorporate qualitative simulation of adaptive mechanisms. We discuss detailed examples of the system in operation, which produces results fully consistent with the medical literature on some typical pathological situations, for a continuous range of user-driven modification of the P-V curve.*

## 1. Introduction

Generations of medical students will have encountered *Pressure-Volume diagrams* (or P-V diagrams) to explain the behaviour of the heart across the cardiac cycle [1]. These offer a snapshot of cardiac behaviour through the various phases of ventricular activity for a single beat, yet they encompass most determinants of ventricular ejection as well as giving an indication of ventricular properties (contractility, compliance) [2]. Statements such as “*end-diastolic stiffness of the ventricle is increased because the ventricle operates on the steep part of the curve*” [3], are used commonly in textbooks as a consequence of that form of diagrammatic reasoning.

In this paper, we describe a system based on an interactive version of the P-V cycle, where interactive modifications by the user support diagrammatic inference for pathophysiological situations in cardiology or critical care. In addition, we have incorporated a mechanism for simulating the short-term evolution of the situations created by users, using a technique known as qualitative simulation.

## 2. Previous and related work

Previous work in Intelligent Tutoring Systems has emphasized the use of P-V diagrams for visualization [4], entering physiological data in numerical form via a menu, without supporting direct interaction and manipulation that characterizes diagrammatic reasoning. Kelsey et al. [5] have described the use of a similar web-based simulator displaying its results in the form of a P-V curve, reporting excellent acceptance by first-year students. If compared to these previous simulation systems that produce a P-V curve in response to single data entry, our system allows continuous and interactive modification of the curve itself in a way that implements exactly the textbook examples.

## 3. The interactive P-V curve

Our system presents itself as an interactive P-V diagram, as depicted on Figure 1. Its interactive features were designed from the analysis of multiple examples from textbooks and tutorial articles in cardiac physiology, critical care and anesthesiology, from references such as [3] [6] [7] [8], encompassing both introductory and advanced presentations of the topic. To a large extent, it automates the manual reasoning described for P-V diagrams in textbooks, while also incorporating multiple-beats simulation and cardiovascular adaptive mechanisms. The interactive visualization tool was developed using C# (.Net Framework 4) and its graphical user interface was created using Windows Presentation Foundation (WPF).

Figure 1 shows an overview of the actual application window used to display the interactive P-V diagram, which is used both for user interaction and to display integrated results. To determine which portions of the curve should be made interactive, we observed how curves were modified in textbooks and in examples in articles together with the underlying explanations. We designed mechanisms for user interaction, which could reproduce the modifications reported in the traditional “manual” reasoning with the P-V diagram that are common to many textbooks. We then included mechanisms enforcing the basic properties of the P-V diagrams as constraints, so that user interaction would

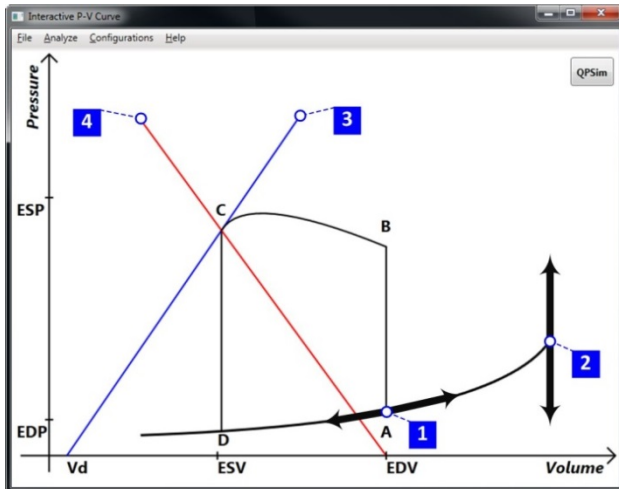


Figure 1. Overview of the Interactive P-V Curve. Control points make it possible to modify the most important physiological parameters in a way that reflects the use of the P-V curve in articles and textbooks. The “QPSim” button at the top right corner activates qualitative simulation for short-term adaptation to the changes created by altering the shape of the curve.

allow real-time redrawing with direct visual feedback, as well as automatic inference reporting the global variation of key parameters that followed P-V curve modification.

The user can modify key parameters of the P-V diagram by interacting with control points through mouse clicking and dragging. The interaction model offers control over the key variables that represent *preload*, *afterload*, *left ventricular compliance* (active and passive), and *contractility*. In Figure 1, the user interface displays highlighted dots which represent four main control points (numbered 1-4). As each of the above variables has a different geometrical interpretation on the P-V curve, appropriate interaction mechanisms have been devised. *Preload* is represented by End-Diastolic Volume (EDV) and is modified by interacting with point “A” (control point 1), whose movements are constrained alongside the End-Diastolic Pressure-Volume relation (EDPVR) curve, which represents left ventricular filling. This curve is usually described in the literature by the approximated formula  $EDP = a \cdot e^{kEDV} + b$  and corresponds to ventricular compliance. It can be modified through interaction with control point 2: the vertical component of any displacement is used to redraw the curve from the above equation, essentially through a modification of parameter  $k$ , resulting in exponentials of various steepness.

The blue line (Figure 1) represents the End-Systolic Pressure Volume Relation (ESPVR), which is taken as a measure of the left ventricle’s inotropic state, or cardiac contractility. The line is drawn from the invariant point on the volume axis that represents the volume of the

ventricle not subjected to any volume stress (labeled  $V_d$ ), and intersects the P-V curve at point C, which is therefore said to describe end-systolic elastance. Interacting with control point 3 modifies its slope by rotating around  $V_d$ .

Finally, the slope of the red line is considered as a measure of afterload, and can be modified by interacting with control point 4, through a rotation around EDV.

The system allows multiple simultaneous modifications to be combined into complex syndromes: for instance, it is possible to explore the effects of a decrease in afterload on a dilated, insufficient ventricle, etc. These complex modifications preserve the direct visual feedback to the user as well as the calculation of qualitative values. During several consecutive interactions, which can take place with the above-described control, the graphical system is constantly redrawing the overall diagram. For every interaction with the control points, the redrawing takes place whenever a control point is moved by a single pixel. This is an important feature to support diagrammatic reasoning as it offers potential for the exploration of configurations. Finally, a menu item on the interface offers the possibility to initialize the configuration of the P-V diagram to a typical physiological context, such as normal heart or an insufficient heart. This allows creating predefined clinical settings corresponding to typical syndromes.

#### 4. Extending diagrammatic reasoning with adaptive mechanisms

P-V diagrams give an integrated, synthetic view of cardiac performance. One core concept for understanding P-V curves is the notion of “single-beat” representation [9]. The P-V curve only represents one prototypical cycle; however, it does not by itself accommodate the propagation of any alterations over several cycles. This not only ignores active regulation mechanisms but also natural adaptation via Frank-Starling’s mechanisms, or adaptation to an afterload increase or a decrease in venous return.

This is why we have extended our interactive P-V curve by interfacing it to a qualitative simulation system that incorporates active and passive regulation mechanisms. This is adapted from our previous hemodynamics simulation system, which has been evaluated with medical students in Japan<sup>1</sup> [10]. The role of this qualitative simulator (QPSim) is to simulate the evolution of the same physiological parameters over time. It ensures that convergence of the qualitative simulation is based on the temporal simulation of several cardiac cycles, thus addressing the single-beat limitation of the initial P-V representation, which it complements ideally.

<sup>1</sup> We describe the underlying principles rather than technical details about its implementation, as they are described in previous publications [10].

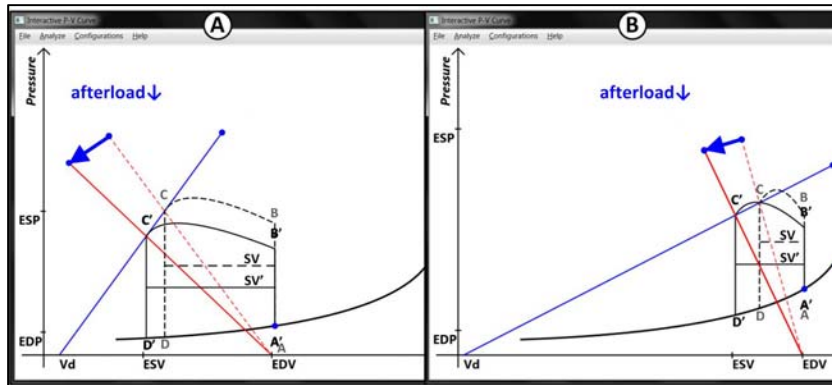


Figure 2. Results for a reduction of afterload on a normal heart (A) and an insufficient heart (B), which is a canonical use of the PV diagram. The comparative increase in Stroke Volume is significantly higher for the insufficient heart.

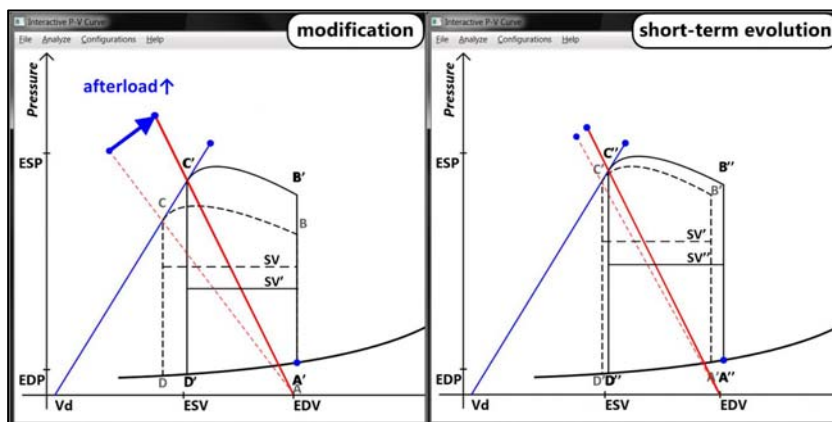


Figure 3. Results for the adaptation of a healthy heart to an increase in afterload through preload reserve.

## 5. Results and system validation

The basis for system validation is that no single pathophysiological syndrome has been directly encoded in the system, which is developed from first principles. For instance, whilst single-beat responses can be derived from the inherent properties of the P-V curve, adaptive responses can only be obtained through integration with qualitative simulation.

Single-beat representations do not give access to all variables when reasoning only on the P-V diagram, nor do they give the most accurate picture, even if the trend they signal is correct, somehow reflecting comments from Shishido et al. [9] on an apparent dissociation between the P-V curve and some key physiological measurements. Our first (classical) example is the improvement in SV obtained by reducing afterload in case of chronic heart failure. Figure 2 shows how beneficial a reduction of afterload can be on an insufficient ventricle. Because the ventricle operates on the steep part of the EDPVR curve, a small reduction in afterload will result in a significant increase in SV. The user reduces afterload by interacting with the red line (dashed line): this generates an increase

of SV, however the situation further improves after a few beats (A'-B'-C'-D') as shown by activating QPSim.

To illustrate the integrated behaviour of the system, let us consider an isolated increase in afterload on a healthy (normal) heart (Figure 3). It gives rise to a progressive adaptation through which SV is partially restored by an increase in preload. This can be explained as follows: the increase in Afterload causes an increase in ESV, which in turn increases EDV (or preload) during the subsequent beats. This increase in preload activates the Frank-Starling mechanism to partially compensate for the reduction in stroke volume caused by the increase in afterload [8]. This compensates the fall in SV, although in general not entirely<sup>2</sup>. This mechanism is based on preload reserve [11]. System output on Figure 3 illustrates this behaviour: the dashed P-V cycle represents the initial condition. The first A-B-C-D cycle, the decrease in SV following increase in afterload (slope of the red line, modified interactively by the user), which is the single-

<sup>2</sup> Afterload is unchanged (as it is defined by the slope of the red line, not by its position).

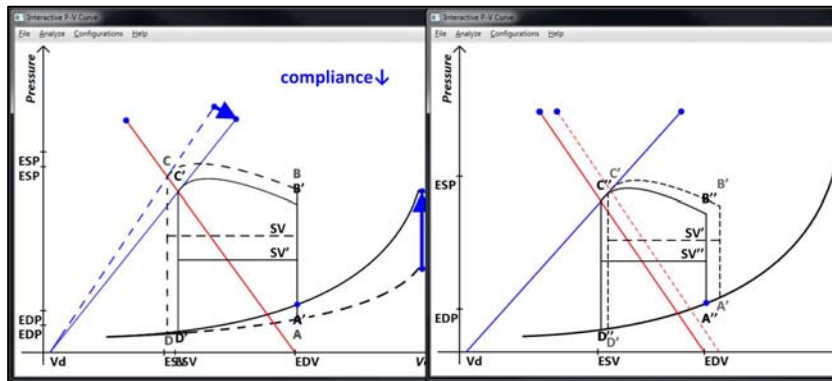


Figure 4. Modelling diastolic cardiac failure: a mild decrease in contractility is associated to a significant decrease in ventricular compliance, which can be entered by shifting upwards the EDPVR curve. The right-hand side of the figure shows the situation evolving through time, with the decrease in compliance affecting the stroke volume.

beat response obtained through the P-V diagram alone. After activation of QPSim to calculate the response of the cardiovascular system over several beats, the final A''-B''-C''-D'' cycle shows the improvement in SV, short of a total restoration. This behaviour we have successfully reproduced was not directly encoded in the system (as an adaptive mechanism), but was an outcome of the qualitative simulation from first principles through several cardiac cycles, with the final output displayed on the P-V diagram. Figure 4 shows a different example of evolution, in the case of diastolic cardiac failure [3]. In this case, the real impact on SV cannot be immediately derived from the curve properties and is only visible after simulating a few cycles.

## 6. Conclusion

We have successfully automated the reasoning principles described in the medical literature for “single-beat” alterations of the P-V cycle, and our system is able to operate successfully on all typical pathophysiological situations that are normally illustrated through P-V diagrams. In addition, we have introduced a principled mechanism to incorporate short-term adaptive mechanisms into P-V diagrams via qualitative simulation using an extended set of physiological parameters. The behaviour of the integrated system preserves the most important properties of the heart, while also being consistent over a range of typical clinical situations. One limitation of the system consists in the lack of long-term adaptation mechanisms (e.g. cardiac hypertrophy following prolonged increase in afterload, cardiac dilatation in some cases of congestive heart failure) in the simulation part of the system, which was designed to simulate acute situations, hence short-term adaptation over several beats.

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