Examination of the Windkessel Effect Using a Two-element System

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Windkessel; one-element; two-element; capacitance; compliance; heart; cardiac circuit

ABSTRACT: The 2-element Windkessel model describes load on the heart during the cardiac cycle with respect to peripheral resistance and arterial compliance. Compliance allows for the Windkessel effect, which helps the body to combat the pulsating nature of blood flow and maintain a relatively constant arterial pressure. This study attempts to measure the effect of compliance on fluid flow by comparing 1-element and 2-element Windkessel models. Experiment results were compared to simulations of the cardiac circuit, it was revealed that the experimental Windkessel models explored bear close resemblance to their simulated counterparts. The addition of capacitance to the Windkessel model resulted in a reduction in pressure-time signal energy for both simulated and practical models as well as an increase in the time required to return to resting pressure.

BACKGROUND

The Windkessel model describes load on the heart during the cardiac cycle with respect to input impedance. The two-element Windkessel model describes impedance with two parameters: peripheral resistance and arterial compliance. In the commonly applied circuit analogy, peripheral resistance is represented by a resistor and arterial compliance by a capacitor and these two elements are linked in parallel [2]. For this study, “compliance” is interchangeable with “capacitance”.

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*Figure 1. Circuit analogy of two-element Windkessel model. Circuit element values are arbitrary.*

Peripheral resistance is the resistance of the arteries to blood flow. Arterial resistance is determined by blood viscosity, blood vessel length, and blood vessel width. While viscosity and length are generally constant, blood vessel width is subject to change through vasodilation (expansion) and vasoconstriction (contraction) of the blood vessel. The second Windkessel element, arterial compliance, is defined as the ratio of change in volume to change in pressure. Compliance allows for the Windkessel effect, which helps the body to combat the pulsating nature of blood flow and maintain a relatively constant arterial pressure [3].

Arterial compliance is an important cardiovascular risk factor that can cause a cycle of elevating blood pressure and atherosclerosis (arterial hardening). Increased blood pressure can cause blood vessels to weaken and rupture (aneurysm) due to excess strain and may also increase susceptibility to several other organ diseases [1].

The intent of this study was to measure the effect of compliance (or capacitance) on fluid flow. Results were compared to Simulink simulations of the heart. It was hypothesized that the simulations would successfully approximate the experimental models. It was expected that the 2-element Windkessel pressure wave would have a lower amplitude and a slower return to rest than the 1-element Windkessel. The 1 and 2 element pressure-time signals were predicted to exhibit similar energies, or area under the curve following the assumption that energy is not lost through the addition of capacitance.

METHODS

Simulated Windkessel models were made for each of the following systems using Simulink: 1-element, 2-element, 3-element, 4-element. Plots were generated in MATLAB using these models. Heart rate was held at a constant 70BPM across simulations and initial pressure was set to 0 mmHg. These controls were set to enhance comparability across simulations.

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*Figure 4. 2-element Simulink circuit used to conduct simulations.*

A 1-element Windkessel model was built without the presence of a capacitive circuit element. Two tubes were connected in parallel to two exits. One circuit exit led to a pressure transducer which read to an ADInstruments PowerLab. At the same exit, a beaker was set up to passively catch the water as it passed the transducer. The PowerLab was configured to measure and record voltage (mV) of the pressure transducer with respect to time (s). A hand-operated syringe was filled with 25 mL of water and connected to the free circuit inlet. Air bubbles were removed, and water was injected by the syringe into the system in bursts. Approximately 5 mL of water was syringed with each burst and the intervals between bursts were intended to resemble a consistent heartbeat.

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*Figure 2. 1-element Windkessel experimental setup.*

The 2-element Windkessel model was adapted from the 1-element model. One of the two parallel tubes from the 1-element model was replaced by a rubber balloon. The rest of the circuit was left unaltered as a control. Once again, air bubbles were removed, and water was injected by hand into the system in 5 mL bursts at relatively consistent intervals.

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*Figure 3. 2-element Windkessel experimental setup.*

Data was exported from the PowerLab and cleaned using Python. For each circuit, the data was cropped by varying lengths at each end to remove random artifacts recorded before and after each trial. For both the 1-element and 2-element models, a moving average was used to smooth the data and average out any outliers. Moving average window sizes were selected to improve visibility while maintaining underlying trends. A window of 50 datapoints was used to average the 1-element data and a window of 100 datapoints was used for the 2-element data. The 2-element data looked considerably noisier which provoked the window size discrepancy between each model. Discrete integrals were calculated using Python and Simpson’s rules. The difference between initial and max y-axis values was calculated for each simulation and experiment. The smoothed datasets were plotted, alongside their integrals, using the Python Matplotlib package.

RESULTS

|  |  |
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| Chart, line chart  Description automatically generated  *Figure 5. Results from Simulink simulations of 2-element, 3-element, and 4-element Windkessel models. Plotted in MATLAB.* | Chart  Description automatically generated  *Figure 6. Results from Simulink simulations of Windkessel models. The blue plot depicts the results of a 1-element model using only total peripheral resistance (TPR). The orange plot depicts the results of a 2-element model using TPR as well as a capacitive element. Plotted in MATLAB.* |
| Chart  Description automatically generated  *Figure 7. Pressure simulation of 1-element Windkessel model at a heart rate of 70BPM. The model was simulated in MATLAB and Simulink. The data, along with its integral, was plotted using the Matplotlib Python package.* | Chart, line chart, histogram  Description automatically generated  *Figure 8. Pressure simulation of 2-element Windkessel model at a heart rate of 70BPM. The model was simulated in MATLAB and Simulink. The data, along with its integral, was plotted using the Matplotlib Python package.* |
| Chart, histogram  Description automatically generated  *Figure 9. Recorded pressure transducer voltage with respect to time in the Windkessel circuit without a capacitive element (1-element Windkessel). Data was recorded with the PowerLab and plotted using the Matplotlib Python package alongside its integral.* | *Chart, line chart, histogram  Description automatically generated*  *Figure 10. Recorded pressure transducer voltage with respect to time in the Windkessel circuit with a capacitive element (1-element Windkessel). Data was recorded with the PowerLab and plotted using the Matplotlib Python package alongside its integral.* |

*Table 1. Energy/integral calculations for each simulation and experiment.*

|  |  |  |
| --- | --- | --- |
|  | Simulation | Experiment |
| 1-Element | 445.09 mmHg | 4.09 mV |
| 2-Element | 334.87 mmHg | 1.73 mV |
| Ratio | 0.75 | 0.42 |

*Table 2. Difference between initial and max pressure/voltage for each simulation and experiment.*

|  |  |  |
| --- | --- | --- |
|  | Simulation | Experiment |
| 1-Element | 432.72 mmHg | 1.98 mV |
| 2-Element | 101.74 mmHg | 0.16 mV |
| Fold change | 4.2 | 12.5 |

DISCUSSION

The simulation results from *Figure 5* demonstrate only subtle differences between simulated 2-element, 3-element, and 4-element models. In contract, the results from *Figure 6* show drastic differences between simulated 1-element and 2-element models. Differences are consistent with the hypothesized capacitive effect. These observations, along with the simplicity of the 2-element model, motivated the decision to only observe the behavior of the 1 and 2 element models experimentally.

A few observations become immediately apparent upon comparison of the 1-element and 2-element simulations in *Figure 6*. Firstly, the waveform frequency of each signal was identical, this was expected since the heart rate was controlled across simulations. Additionally, the 1-element signal pressure returns to 0 mmHg every cycle while the 2-element signal never returns to 0 mmHg. In fact, upon further examination of the 2-element signal in *Figure 8*, it is observed that the signal continues to increase, seemingly logarithmically, over the length of the simulation.

It was hypothesized that the simulations would successfully approximate the experimental models. Similarities are observed between the 1-element experiment (*Figure 9*) and the 1-element simulation (*Figure 7*). Both models exhibited similar waveforms and periodicity suggesting that the 1-element simulation is a good predictor of the experimental model. The only apparent discrepancy is that the simulation showed identical amplitudes each cycle while the amplitudes varied during the experiment. This result is unsurprising given that water was pumped through the system by hand. Similar waveforms are also observed between the two-element simulated (*Figure 8*) and experimental (*Figure 10*) models. However, the signal frequency was lower in the experimental model than the simulated model owing to discrepancies in the “heart rate”, an uncontrolled experimental variable. As such, the overall slope (end pressure/voltage – start pressure/voltage) of the experimental plot was less than the slope of the simulated plot.

It was expected that the 2-element Windkessel pressure wave would have a lower amplitude than the 1-element Windkessel. This prediction generally held true for both the simulated and experimental Windkessel models, and, in fact, the difference was significant. The peak experimental amplitudes found in *Table 2* yielded a 12.5-fold decrease in amplitude from 1-element to 2-element models with respect to the resting voltage. For the peak simulated amplitudes only a 4.2-fold decrease is observed. A reduction in sensitivity due to the transistor intermediary is a possible culprit of this observation. As is the assumption of laminar flow in the simulation as excess turbulence in the 2-element experimental Windkessel may have impeded flow through the circuit. Additional investigation is needed to determine the cause of this phenomenon.

It was expected that the 2-element Windkessel pressure wave would exhibit a slower return to rest than the 1-element Windkessel. This prediction was supported by the 2-element models (*Figures: 8, 10*). Additionally, the return to rest of the experimental signal behaves like a negative exponential while it descends linearly in the simulation. This phenomenon may be contributed to changing “arterial” circumferential stress in response to pressure in the experimental model which was not accounted for in simulation.

The 1 and 2 element pressure-time signals were predicted to exhibit similar energies. The data disproves this hypothesis. In *Table 1*, signal energies are shown to vary significantly between the 1-element and 2-element circuits for both the simulated and experimental models. For the simulations, the 2-element model had a 25% reduction in energy from the 1-element model. For the experiments, the 2-element model had a 58% reduction in energy from the 1-element model. A possible explanation for this phenomenon could be that some energy is lost to the capacitor. Experimentally, the wider diameter of the balloon could account greatly for this due to total volume “loss” (stored in balloon).

Several limitations of this study are of note. Firstly, arterial behavior is not precisely mimicked by a single capacitive element since arterial elasticity is a global property of arteries. This could contribute to differences between the model pressure wave and the actual cardiac circuit pressure wave. Additionally, although its impact can likely be inferred, “heart rate” was not controlled for across the experiments as it was across the simulations. Ultimately, this contributed to some inconsistencies mentioned between the 2-element simulated and 2-element experimental models. Furthermore, the syringe was operated by hand. If a syringe pump were used this could resolve inconsistencies in heart rate as well as volume per cycle. Again, the effect of these factors is inferable however they do likely contribute to discrepancies in the data. Finally, this study inferred cardiac behavior solely on systemic peripheral resistance and systemic arterial compliance. Other factors are involved in the cardiac circuit including aortic/pulmonary resistance and total arterial inertance. This study assumed negligible differences between 2, 3, and 4-element Windkessel models based on the results of *Figure 5*.

CONCLUSION

Arterial compliance is an important cardiovascular risk factor that can cause a cycle of elevating blood pressure and atherosclerosis (arterial hardening). The results of this study suggest that compliance/capacitance plays a large role in decreasing maximum pressure reached within arteries during each heart pump cycle. Additionally, both the simulations and experiments suggest that compliance helps the body to combat the pulsating nature of blood flow and maintain a more constant arterial pressure. Increased blood pressure can cause blood vessels to weaken and rupture (aneurysm) due to excess strain and may also increase susceptibility to several other organ diseases [1]. The results of this study show that pressure can greatly be reduced through the addition of compliance to both the simulations and experiments. Additional investigation into differences in relative peak amplitudes between (see *Table 2*) should be explored. Additionally, this study inferred cardiac behavior solely on systemic peripheral resistance and systemic arterial compliance. Other factors aortic/pulmonary resistance and total arterial inertance should be examined before making anatomical inferences.

AUTHOR INFORMATION

Author Contributions

The manuscript and all graphics were written by J.G. solely. J.G, S.D, M.L, and D.R. contributed equally to the lab experiments.

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ABBREVIATIONS

TPR, total peripheral resistance; BPM, beats-per-minute; mV, milli-volts; s, seconds.

REFERENCES

[1] Glasser SP, Arnett DK, McVeigh GE, Finkelstein SM, Bank AK, Morgan DJ, Cohn JN. The importance of arterial compliance in cardiovascular drug therapy. J Clin Pharmacol. 1998 Mar;38(3):202-12. doi: 10.1002/j.1552-4604.1998.tb04417.x. PMID: 9549658.

[2] Her, K., Kim, J.Y., Lim, K.M. et al. Windkessel model of hemodynamic state supported by a pulsatile ventricular assist device in premature ventricle contraction. BioMed Eng OnLine 17, 18 (2018). <https://doi.org/10.1186/s12938-018-0440-5>

[3] Tucker WD, Arora Y, Mahajan K. Anatomy, Blood Vessels. [Updated 2021 Aug 11]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470401/