Use of an electrical model of the left ventricle of the heart and the arterial system permits analysis of the changes which take place as the capacity of the myocardium for generation of force decreases. The model is simple in structure, and its construction and practical testing would not be difficult. It demonstrates that, as the heart muscle weakens, the peak of intracardiac force occurs later in systole, and the difference between the intracardiac pressure and the aortic pressure in the second half of systole is much greater than for the normal heart. The feedback mechanisms which are proposed to affect myocardial contractility would affect this compensation for cardiac weakening. Indices to categorize the behavior of the normal, compensated though weakened, and decompensated myocardium are proposed.

The pulmonary congestion, weight gain, ankle edema, and venous engorgement which result from a severely decompensated human heart have been the objectives of extensive studies (Friedberg, 1956). Yet the intermediary stages between a normal and failing heart have been poorly understood. Heart failure must occur as the end stage of a continuous process. Initially, compensatory adjustments prevent noticeable cardiac insufficiency. As the progression of time decreases the effectiveness of the compensatory mechanisms, overt heart failure becomes apparent. The present theoretical study utilizes an electrical model of the heart and systemic arteries to delineate the changes which result
from decreasing forces of cardiac contraction. An electrical circuit is advantageous as a model because its behavior is easily understood and analysed, both theoretically and practically. The model acquires value from the practical advances in understanding of the action of the heart which result from its use.

Theoretical results. The electrical model of the heart and arteries is represented in Figure 1. In this model, the battery $B$, resistance $R_1$ and capacitance $C_1$ represent the heart. At the end of diastole the voltage across $C_1$ is taken as $V_0$. A switching mechanism, not shown in the diagram, is considered to prevent current flow through $B$ during each diastole. In practice, a simple relay switch with a repetitive timing device can accomplish this task with ease.

![Figure 1. Diagram of the electrical model](image)

At the onset of isometric contraction, $i_5$ becomes positive, and the voltage $V_1$ across $C_1$ increases. When it reaches a value $V_{10}$ of adequate magnitude, the gas in the thyatron $T$ (or similar device) becomes conductive and $i_2$ becomes positive. In healthy hearts, the magnitude of $B$ is large enough so that the value of $V_{10}$ is only a small portion of the magnitude which $V_1$ would reach in the absence of the thyatron. As the heart decompensates, $V_{10}$ becomes an ever increasing fraction of the theoretically maximal value because of the decreasing value of $B$.

The presence of the inductance $L$, with its accompanying resistance, $R_3$, prevents any initial surge in the value of $i_2$ in agreement with the corresponding gradual increase in the blood flow from the heart when the aortic valve opens with its thyatron-like action. Because of the slow initial increase in the value of $i_2$, there is a continuing increase in $V_1$ after the thyatron begins to conduct. The nature of this increase depends upon the values of $L$ and $B$. A healthy heart, represented by a high value of $B$ and a low value of $L$, results in a peak value of $V_1$ early in systole and then a rapid decline in its value. On the other