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Nonlinear Heart Model Predicts the Range of Heart Rates for 2:1 Swinging in Pericardial Effusion

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Abstract

We analyze Rigney and Goldberger's [14] two mathematical models of the heart's swinging in large pericardial effusions. Both models represent the torques due to the outflow of blood from the heart. The first assumes that the duration of systole does not vary with heart rate, whereas the second assumes that it varies linearly with heart rate. We examine the motion of the heart for heart rates between 50 and 200 and for a range of initial positions and velocities. Both models predict that the heart swings once every other beat (2:1 swinging, giving rise to electrical alternans) in a discrete range of heart rates and swings once per beat otherwise, and explain the appearance and disappearance of 2:1 swinging mathematically.

The first model predicts a rate range from 105 to 116 for the occurrence of 2:1 swinging. The second model predicts the same qualitative behavior, but with 2:1 swinging occurring at heart rates between 88 and 119, which agrees well with published clinical data showing 2:1 swinging at heart rates between 90 and 144. We describe an analysis program for ordinary differential equations that analyzed the models quickly and automatically.

Index Terms: electrical alternans, nonlinear dynamics, cardiovascular models.

1 Introduction

Cardiac alternans is the phenomenon in which the observable manifestations of cardiac activity alternate between two modes in each successive cardiac cycle. It comprises electrical alternans at the cellular and body surface levels and mechanical alternans, which has been attributed to variations in hemodynamic variables, contractile state, and the interrelation between mechanical and action potential alternans [20]. Alternans in the body surface ECG can also occur in association with myocardial ischemia and with cardiac motion [20]. The latter phenomenon is the subject of this paper.

Electrical alternans can result from 2:1 swinging of the heart, or swinging once every other heart beat, in large pericardial effusions. This phenomenon has been reproduced in an isolated heart preparation, and has been shown to result from periodic changes in the location of the heart with respect to the recording electrodes rather than from changes in conduction within the heart [3]. The clinical association of electrical alternans with tachycardia in the range of 100 to 125 beats per minute was made in 1958 [10]. A mathematical model of the cardiac motion that predicted 2:1 swinging was proposed in 1989 by Rigney and Goldberger [14]. That paper focused on the physics of the model and the justification for their selection of parameter values and simplifying assumptions.

This paper extends Rigney and Goldberger's (R&G) [14] results to address several additional questions. What is the range of heart rates and initial conditions over which 2:1 swinging occurs? How does a change in heart rate induce 2:1 swinging? Does the heart exhibit other motions at other heart rates or initial conditions, such as swinging once every four beats or swinging erratically?

2 Methods

R&G hypothesize that the outflow of blood from the heart causes it to swing during pericardial effusion. The force of the blood flowing out of the left and right hearts produces torques around the pivot point where the great vessels are anatomically fixed (Figure 1). The pericardium and the weight of the heart normally counterbalance these torques, but neither factor applies when the pericardial space fills with fluid. The heart then floats freely, so the only resistance to the torques comes from the pericardial fluid and from the thoracic walls. The imbalance between the left and right torques causes the heart to swing. The forces vary nonlinearly as the heart swings.

R&G model this situation as a nonlinear oscillator. They model the myocardium and chamber cavities as two point masses, the centers of mass of the left and right hearts, attached to a pivot by rigid, weightless rods. They assume that the heart swings solely in the plane of the triangle. Figure 2 shows the model and Table 1 lists the parameter values chosen by R&G. They model the hemodynamic torques on the left and right hearts, $T_l'(t)$ and $T_r'(t)$, as periodic step functions

$$T_l'(t) = \begin{cases} 9 \times 10^6 \text{ dyn-cm} & \text{during systole} \\ 1.25 \times 10^6 \text{ dyn-cm} & \text{during diastole} \end{cases}$$

and

$$T_r'(t) = \begin{cases} 8 \times 10^5 \text{ dyn-cm} & \text{during systole} \\ 8 \times 10^4 \text{ dyn-cm} & \text{during diastole} \end{cases}$$

whose period, T, is one heart beat. They assume that systole lasts 0.3 seconds for all heart rates, hence that diastole lasts $T - 0.3$ seconds. (In their Appendix 2, they propose an

alternate model in which the duration of systole varies linearly with heart rate.) From the model, R&G infer a T-periodic second-order ordinary differential equation that governs the angle, θ_l, between the left heart and the direction of gravity. The equation is

$$\ddot{\theta}_l + a_1 \sin\theta_l + a_2 \sin(\theta_l + \Phi) + a_3(t)\sin(\theta_l - \phi_l) + a_4(t)\sin(\theta_l + \Phi - \phi_r) + \dot{\theta}_l/\tau = 0 \qquad (1)$$

with

$$\begin{aligned} a_1 &= (M_l g - b_l)/(M_l + M_r)L \\ a_2 &= (M_r g - b_r)/(M_l + M_r)L \\ a_3(t) &= T_l'(t)/(M_l + M_r)L^2 \\ a_4(t) &= T_r'(t)/(M_l + M_r)L^2 \\ 1/\tau &= (\eta_l + \eta_r)/(M_l + M_r)L. \end{aligned}$$

R&G modify the equation to model the fact that the torques cannot push the heart beyond the limiting wall of the pericardium. They set $\dot{\theta}_l = 0$ when the angle $\theta_l = 0.85$ and the torque is positive or when $\theta_l = -0.85$ and the torque is negative. The angles follow from their choice of values for α_l, α_r, and Φ.

The solutions to the differential equation depend on the heart rate, which determines T, and on the initial values of θ_l and $\dot{\theta}_l$. R&G integrate the equation numerically to determine the steady state solutions for heart rates of 75 and 105 ($T = 0.8$ s and $T = 0.57$ s). The integrations begin at the start of diastole and continue until the solution appears periodic. They observe a T-periodic solution for heart rate 75, meaning that the heart swings once every beat. They observe $2T$-periodic solutions for heart rate 105, meaning that the heart exhibits 2:1 swinging and that the steady state motion depends on the initial conditions.

They conclude that the simplified heart model explains the effect of heart rate on 2:1 swinging. Table 2 summarizes their results. They do not analyze the alternate model from their Appendix 2.

The first step in our analysis of the heart equation is to write it as a system of two T-periodic first-order equations

$$\dot{\theta}_l = \omega$$
$$\dot{\omega} = -a_1 \sin\theta_l - a_2 \sin(\theta_l + \Phi) - a_3(t)\sin(\theta_l - \phi_l) - a_4(t)\sin(\theta_l + \Phi - \phi_r) - \omega/\tau$$

on the (θ_l, ω) state space. The equations specify the time derivatives of the angular displacement θ_l and of the angular velocity ω as a function of their values and of the elapsed time. We can measure elapsed time either from the start of the first diastole in the simulation or from the start of the most recent diastole, since the functions are periodic in the period T of the heart. The solutions specify the evolution of θ_l and ω given their values at the start of the first diastole.

We need only sample the solutions at the start of each diastole to obtain the steady state motion. 1:1 Swinging (or other T-periodic motion) corresponds to states that recur after one heart beat, called *period-1 points*. 2:1 Swinging (or other $2T$-periodic motion) corresponds to states that recur after two heart beats, called *period-2 points*. Period-2 points come in pairs, since the intermediate point after one heart beat is also a period-2 point. The two points denote the same steady state behavior with a one heart beat phase difference. For example, R&G's solutions 1 and 4 appear as the period-1 point $(-0.70, -0.38)$ and as the period-2 pair $(-0.65, 0.50)$ and $(-0.75, -1.05)$. In general, kT-periodic solutions correspond to *period-k points* that recur after k heart beats and that come in k-tuples. We need not

discuss aperiodic motions, since the heart model exhibits periodic motions only. Figures 3 and 4 illustrate these concepts.

A periodic point is *stable* if all nearby states converge to it, and *unstable* otherwise. Unstable periodic points do not represent steady state behaviors of physical systems because they are unreachable from any (technically, almost any) initial state. No matter how close a system approaches an unstable periodic point, it eventually moves away and converges to another, stable behavior. Nevertheless, unstable points are important because they govern transient behavior and because they interact with stable points, as explained below.

We must calculate and classify the periodic points as a function of the heart rate, h. The periodic points are smooth functions of h. The graphs of these functions are called *branches*. A period-1 point generates a single branch. A pair of period-2 points generates a pair of branches, which we will refer to as a period-2 branch. A k-tuple of period-k points generates k branches. Each branch partitions into alternating stable and unstable segments.

Bifurcations occur at heart rates where branches appear, vanish, or change stability. In physical terms, the steady state solutions change qualitatively when the heart rate crosses a threshold. Two types of bifurcations occur in the heart model. A *saddle node* bifurcation occurs when a stable and an unstable point merge and vanish. A *period doubling* bifurcation occurs when a stable period-1 point becomes unstable and gives birth to a stable period-2 point, or when an unstable period-1 point becomes stable and gives birth to an unstable period-2 point.

Analyzing differential equations is challenging and time consuming. Sacks has developed a program, called POINCARE, that automates the analysis of one-parameter families of two

first-order equations, such as the heart equation, and assists in the analysis of families of three or more equations [16, 17]. The inputs are the family, bounding intervals for the state variables and parameter, and an error tolerance. POINCARE traces the branches of periodic points in the parameter interval and finds the bifurcations. It derived the branches of periodic points and the bifurcations for the heart equation, using error tolerances of 10^{-9} for individual solutions and of 10^{-5} for branches. The entire analysis took roughly one minute on a DEC 5000 workstation.

The POINCARE program runs under any system that supports Fortran-77 and Lucid or Allegro Lisp. The program is written in Lisp. It uses the ODESSA [9] integration package and the AUTO [4] continuation package, which are written in Fortran-77. It is available free of charge upon request.

3 Results

R&G predict the motion of the heart at the specific heart rates of 75 and 105 beats per minute. We extend their analysis by predicting the cardiac motion for heart rates between 50 and 200 in increments of 0.1 heart beat or less, and by explaining the behavior according to the mathematical theory of differential equations. The analysis is supported by extensive computation.

The results suggest that the heart swings once every other beat (giving rise to electrical alternans) in a discrete range of heart rates and swings once per beat otherwise. The choice of the constant-duration systole model rather than variable-duration systole model yields better correspondence with the clinical literature, which is presented in the Discussion.

Figure 5 shows the branches and the bifurcations of the heart equation as a function of heart rate. Branch 1 is period-1 and the other branches are period-2. Branches 2 and 4 are unstable, branch 3 is stable, and branch 1 consists of two stable segments and one unstable segment. The branches contain four bifurcations: a period doubling of branches 1 and 2 at $p = 104.4$, a period doubling of branches 1 and 4 at $p = 116.4$, a saddle node of branches 2 and 3 at $p = 104.0$, and a saddle node of branches 3 and 4 at $p = 116.6$. The model predicts a single stable motion at each heart rate: 1:1 swinging at heart rates of 104 and below, 2:1 swinging at heart rates between 105 and 116, and 1:1 swinging at heart rates of 117 and above. The only exceptions are the narrow ranges of 104.0 to 104.4 and 116.4 to 116.6 where 1:1 and 2:1 swinging coexist.

Figure 5 is consistent with bifurcation theory because the changes in the number and the stability of the periodic points all occur at bifurcations. Based on this consistency, POINCARE conjectured that these branches contain all the steady state solutions of the heart equation. It tested the results numerically by generating the branches two different ways, with the AUTO [4] continuation package and by Newton-Raphson iteration [12]. The outputs were equal to within five decimal places. It searched for missing branches by Newton-Raphson iteration and by integrating for 1000 heart beats, using 1000 initial points for each method. It found none. We conclude that Figure 5 accurately portrays all periodic solutions of the heart equation for heart rates between 50 and 200.

The discontinuities at $|\theta_l| = 0.85$, which reflect the mechanical constraint of the pericardium on the motion of the heart, complicate the analysis because most theoretical and numerical analysis tools assume continuity or even smoothness, which is continuity of all

orders of derivatives. The discontinuity does not affect the solutions on branches 1, 2, and 4 because they do not reach the limiting wall. The discontinuity dramatically affects the solutions on branch 3 because they bounce periodically off the limiting wall. (The bouncing is invisible in Figure 5 because it occurs part way through the heart beat, whereas the figure describes the start of diastole.) These solutions react discontinuously to changes in their initial conditions, rendering Newton-Raphson iteration and continuation inapplicable. We generated branch 3 by repeatedly increasing the heart rate, starting from the previous fixed point, and iterating the equations over one heart beat until the solution converged.

We analyzed several variants of the heart model.

One variant, described by R&G in Appendix 2, treats the duration of systole as a linear function of heart rate, $t_s = 7/15 - h/450$, rather than assigning it the constant value of 0.3 seconds. This model predicts the same qualitative results as does the original model, but with 2:1 swinging at heart rates between 88 and 119. The prediction is consistent with R&G's comparison of the two models in their Figure 8. R&G observe that the swinging amplitude of their solution 1 (our branch 1) differs little between the models, except at low heart rates. They do not examine the stability of solution 1 or measure the heart rate at which it bifurcates. We confirm that variable-duration systole does not affect the swinging amplitude of solution 1, while observing that variable-duration systole does affect its stability.

The other variants that we examined maintain the assumption of constant duration systole, but model the hemodynamic torques on the left and right hearts as continuous functions of time, rather than as step functions. We linked the step functions into continuous functions with lines and into smooth functions with cubic curves, and varied the width of

the connecting curves from 0.0001 seconds to 0.05 seconds. The constant systole, continuous torque models predict essentially the same range of 2:1 swinging as does the original model.

We assessed the sensitivity of the variable-duration systole model to the values of the underlying physiological parameters by varying the heart mass. We varied the left heart mass from 200 grams to 400 grams in increments of 50 grams and the right heart mass from 100 grams to 300 grams in increments of 50 grams. The qualitative behavior was unaffected by the heart mass. 2:1 Swinging appeared at heart rates between 76 and 122.

4 Discussion

The variable-duration systole model predicts 2:1 swinging at heart rates between 88 and 119, which agrees with the clinical data to within 21%. Of eight published cases of electrical alternans with echocardiographically proven 2:1 swinging and 13 additional cases of electrical alternans without echocardiography (all in sinus rhythm), 14 occurred at heart rates between 100 and 124, two occurred at heart rate 90 to 95, and five occurred at heart rate 130 to 167 (Table 3). The cases of electrical alternans without echocardiographically proven 2:1 swinging are not as useful for model validation because electrical alternans can occur in pericardial effusion without abnormal heart motion [7].

The existence of a critical range of heart rates within which 2:1 swinging can occur has been suggested for many years. The range of 100 to 125 beats per minute (bpm) was suggested in 1958 [10], and revised to the range 108 to 125 bpm by Usher et al. in 1972[21, p. 343]. The fixed-systole model predicts 2:1 swinging at heart rates between 105 and 116, which agrees less well with the clinical data.. The variable-duration systole model appears

superior, since it is more accurate and only slightly more complicated. The other variants appear inferior, since they are no more accurate and somewhat more complicated.

It is noteworthy that such simple models capture the motion of the heart so well. The remaining inaccuracy may reflect the many simplifying assumptions in the model. Sensitivity analysis suggests that it may simply reflect differences between the actual values of the physiological parameters in the clinical data and the estimates used in the model.

In particular, the sensitivity analysis by R&G in their Figure 6 shows that the frequency of 2:1 swinging is inversely related to the left and right ventricular masses. In the model, ventricular mass is inversely related to buoyancy, which is in turn directly related to pericardial fluid density. Density is measured as specific gravity. The relationship of specific gravity to protein concentration holds generally: for plasma, specific gravity is 1.025–1.029 and protein concentration 5.7–7.9 mg/dl; for transudates, 1.005–1.015 and 0.4–1.9; for pleural fluid, 1.013 and 0.3–4.1; and for peritoneal fluid, 1.012 and 0.02–4.50 [1]. The protein concentrations in pericardial fluid from autopsy cases of meningitis and hydrocephalus ranged from 2.94–4.13 mg/dl [11]. Normal pericardial fluid has a protein concentration of one-quarter to one-third that of plasma, and an osmolarity slight lower than that of plasma [18, p. 7–8]. In contrast, the fluid in chronic effusive pericarditis due to tuberculosis, neoplasm, and trauma is clear to straw-colored, and has protein concentration of 5–6 gm/dl, and specific gravity of about 1.020 [18, p. 146–150]. These data predict that the heart rate at which 2:1 swinging occurs would be higher in exudative effusions, which have protein concentrations similar to that of plasma, and lower in transudative effusions, which have low protein concentrations. Indeed, association of 2:1 swinging with viscous effusions was noted clinically in 1972 by Usher and

Popp [21]. Yet, our sensitivity analysis did not show a marked change in heart rate range when left ventricular myocardial mass was varied from 200 to 400 grams.

An instructive deviant case was presented, also by Usher and Popp, in which an exudative protein concentration (5.7 mg/dl) was associated with fluid of low viscosity and 2:1 swinging at a heart rate of 90 bpm. Because viscosity data have not been published in the clinical literature on 2:1 swinging, they are difficult to include in models of this phenomenon. This is particularly true in cases with metastatic tumor in the pericardial space. The fluid in these cases may contain substantial amounts of mucin, tumor-specific secretory proteins, and other substances that may affect buoyancy and viscosity independently of total protein concentration.

Another factor that is not included in the model is the amount of epicardial fat. Hearts with large amounts of epicardial fat, more common in obese people, would be more buoyant and thus would demonstrate 2:1 swinging at a higher heart rate. These variables, and perhaps others, may help account for the discrepancies between predicted and observed limits on the heart rate range at which 2:1 swinging occurs.

The sensitivity analysis examines the effect on the known periodic points of variations in individual parameters. It is possible that the variations produce new periodic points or aperiodic motions. Finding them requires either multiple parameter continuation, which poses hard mathematical problems, or exhaustive search of the (h, θ, ω) space, which is computationally infeasible. Similar considerations apply to multiple parameter sensitivity analysis in which several parameters vary simultaneously. We recommend deferring exhaustive analysis until clinical findings reveal behavior that our analysis misses.

The analysis of the original equation reproduces R&G's solutions 1 and 4 (on branches 1 and 3), but not solutions 2 and 3. (The four solutions appear in Table 2.) We tested these solutions by integrating the equations over many heart beats and measuring θ_l and ω at the start of each diastole. We performed the integrations with 1000 initial states near those reported by R&G to compensate for measurement error. We also searched for steady states with Newton-Raphson iteration, using an additional 1000 initial states. Neither solution appears to represent a periodic point, since all the tests converge to solution 4. Table 4 summarizes the results of repeated integration from an initial state near solution 2. The first 200 heart beats closely resemble R&G's solution, but 800 heart beats reveal the true steady state motion. The motion appears periodic for two minutes, evolves irregularly for six minutes, then stabilizes at solution 4. Solution 3 behaves similarly.

Bifurcation theory also suggests that solution 3 represents transient swinging rather than steady state motion. Branch 2 consists of solutions very similar to solution 3. Although the branch ends at $h = 104$, the continuity of solutions with respect to heart rate causes solutions at $h = 105$ to approach the vanished period-2 point before converging to solution 4.

The investigation of solutions 2 and 3 illustrates the physical significance of stability. We rarely observe unstable motions because the heart rarely enters these states and soon leaves them. We cannot force the heart into an unstable motion because we cannot control its state well enough. If we put the heart close to an unstable motion, it follows the unstable motion for a while then switches to a stable motion. Hence, physicians rarely observe the unstable solutions of the heart equation even though they persist for hundreds of heart beats given the right initial state.

POINCARE can also partition the state space of two first-order equations into regions of uniform asymptotic behavior. Each region consists of all the initial states that converge to a common steady state solution. We have already seen periodic steady states. The other possibilities are constant solutions, *quasi-periodic* solutions that contain two incommensurate periodic components, and *chaotic* solutions that wander erratically through state space. Partitioning is superfluous for the heart equation because only one stable solution exists at a given heart rate (except for 104.0 to 104.4 and 116.4 to 116.6), but it is crucial in models where several stable solutions coexist.

In summary, the model of R&G explains 2:1 swinging qualitatively. A variant of it predicts a range of heart rates over which 2:1 swinging occurs that corresponds well to the range observed clinically. Also, the POINCARE program can analyze the resulting differential equations. Using POINCARE, researchers can analyze differential equations that are otherwise intractable. They can determine the effects of varying the parameters or the structure of a model, as we did in changing the length of systole, the heart rate, and the heart masses. The program is freely available, easy to use, and runs on scientific workstations.

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parameter	value	meaning
L	18 cm	length of pendulum
d	5 cm	distance between centers of mass
Φ	0.28 rad	angle between centers of mass
M_l	300 g	mass of left heart
M_r	200 g	mass of right heart
g	980 cm/s^2	gravitational constant
b_l	2.76×10^5 dyn	buoyancy of left heart
b_r	1.84×10^5 dyn	buoyancy of right heart
ϕ_l	-0.75 rad	angle of the aorta
ϕ_r	-0.35 rad	angle of the pulmonary artery
η_l	NA	damping constant of the left heart
η_r	NA	damping constant of the right heart
$1/\tau$	2.4 s^{-1}	aggregate damping constant

Table 1: Parameter values chosen by R&G. They do not specify η_l and η_r.

			Cycle 1		*Cycle 2*	
#	h	n	θ_l	$\dot{\theta}_l$	θ_l	$\dot{\theta}_l$
1	75	T	-0.70	-0.38	—	—
2	105	$2T$	-0.69	-0.04	-0.70	-0.42
3	105	$2T$	-0.69	-0.17	-0.70	-0.23
4	105	$2T$	-0.65	0.50	-0.75	-1.05

Table 2: Solutions reported in Figure 5 of R&G: h the heart rate, n the period, and θ_l and $\dot{\theta}_l$ the angle and angular velocity of the left heart at the start of diastole. Solution 1 shows the heart swinging once per beat at heart rate 75 (1:1 swinging). Solutions 2, 3, and 4 show the heart swinging once every other beat at heart rate 105 (2:1 swinging). The three solutions correspond to steady state motions arising from different initial states.

case	heart rate	reference
Proven 2:1 swinging		
1	90	Usher et al. [21]
2	100	Usher et al. [21]
3	110	Ratib & Perrenoud [13]
4	111	Feigenbaum et al. [5]
5	119	Feigenbaum et al. [5]
6	120	Gabor et al. [6]
7	130	Rinkenberger et al. [15]
8	144	Kreuger et al. [8]
Electrical alternans without echocardiography		
1	95	Bashour & Cochran [2]
2	100	Bashour & Cochran [2]
3	102	Spodick [19]
4	108	Littmann et al. [10]
5	115	Bashour & Cochran [2]
6	115	Bashour & Cochran [2]
7	120	Bashour & Cochran [2]
8	120	Littmann et al. [10]
9	124	Littmann et al. [10]
10	124	Littmann et al. [10]
11	136	Bashour & Cochran [2]
12	138	Brody et al. [3]
13	167	Spodick [19]

Table 3: Clinical observations of 2:1 swinging or electrical alternans in sinus rhythm.

beats	$\theta_l(t)$	$\omega(t)$
0	-0.69	-0.04
200	-0.68	0.02
400	-0.68	0.10
600	-0.66	0.24
800	-0.64	0.46
1000	-0.64	0.46

Table 4: Simulation of R&G's solution 2 for 1000 heart beats shows that it appears periodic for 200 heart beats (two minutes), evolves irregularly for 600 heart beats (six minutes), then stabilizes at solution 4. Hence, solution 2 appears not to represent a periodic point.

FIGURE LEGENDS

Figure 1: Schematic diagram of the outflow of blood from the left and right hearts. The arrows show the direction of motion as blood moves from the chambers through the outflow tract. The resulting torques can rotate the heart around the pivot point where the vessels are anatomically fixed. Adapted from Figure 1 of R&G.

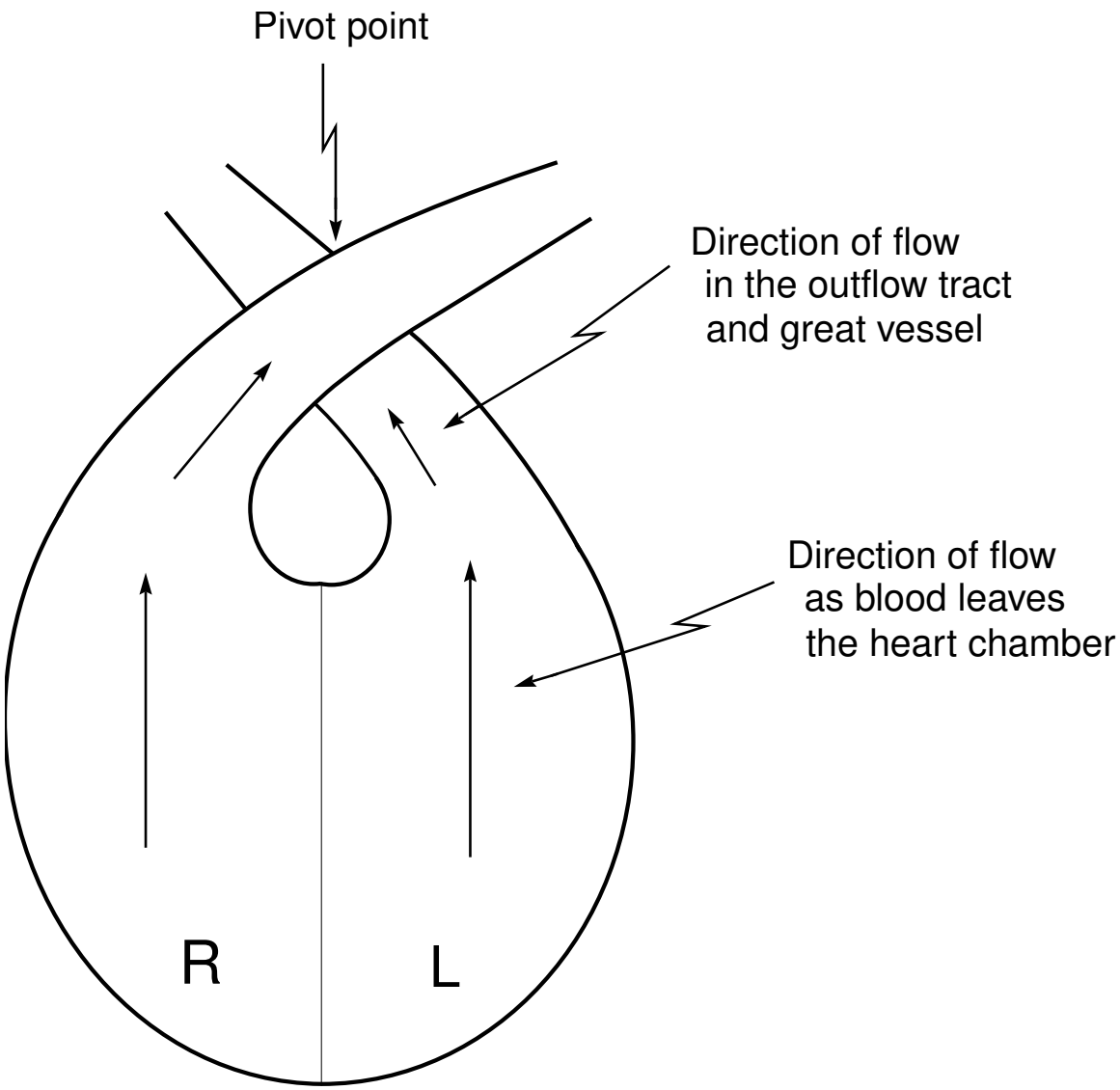
Figure 2: R&G's anatomical model of the heart as two point masses, the centers of mass of the left and right hearts, attached by rigid, weightless rods to the pivot point where the great vessels are fixed. L, distance from masses to pivot; d, distance between masses; Φ, angle between rods; M_l and M_r, masses of left and right hearts; θ_l and θ_r, angles between masses and direction of gravity; ϕ_l and ϕ_r, angles between aorta and pulmonary artery and the direction of gravity; α_l and α_r, angles at which left and right hearts hit the pericardial wall. All values are fixed, except for θ_l and θ_r. Adapted from Figure 2 of R&G.

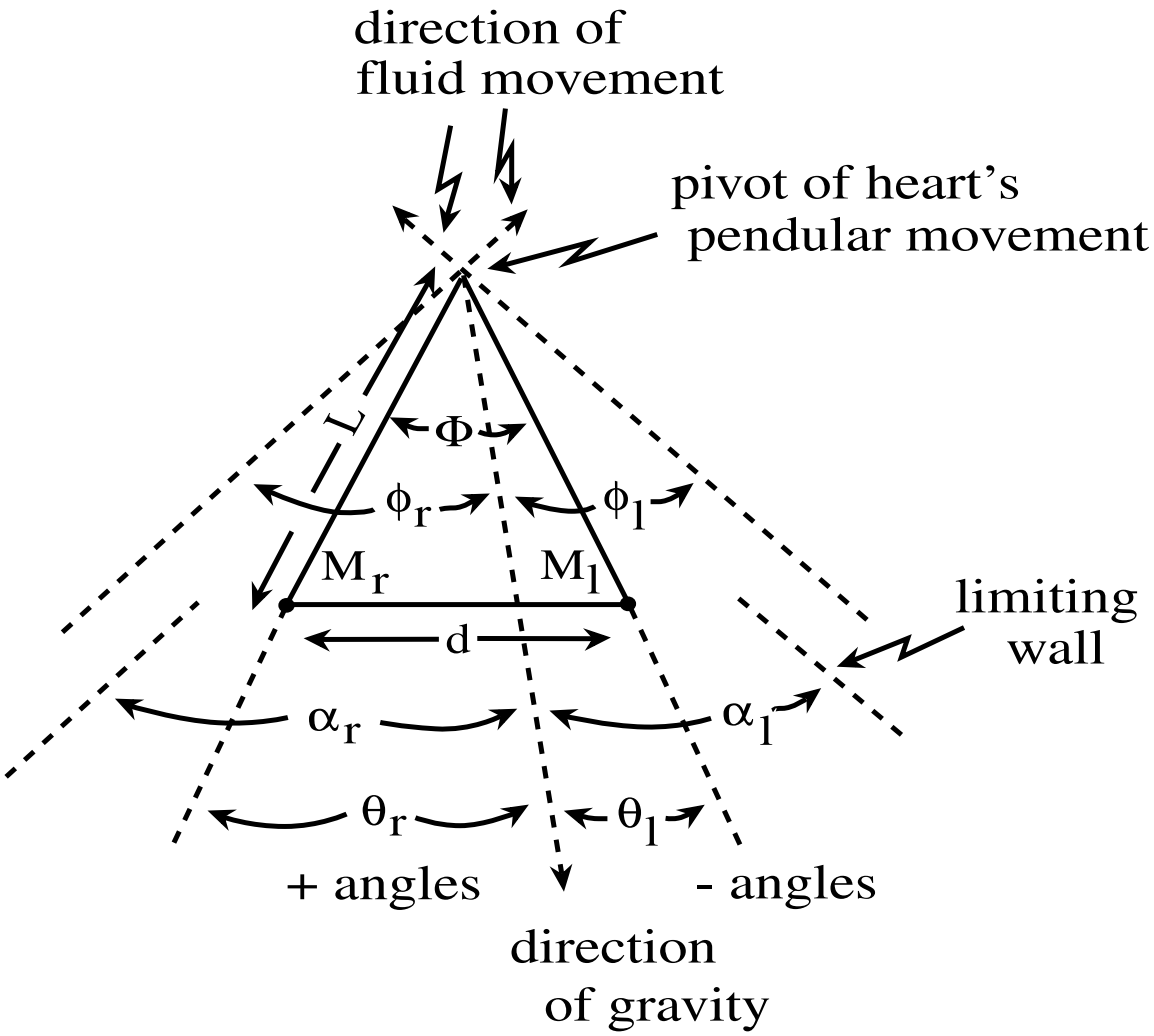
Figure 3: Solutions of the T-periodic system. The three planes represent the angular displacement θ_l and angular velocity ω of the heart at the start of three consecutive diastoles. The mapping F indicates the change in state after one heart beat. The state p is a period-1 point because $p = F(p)$, whereas q is a period-2 point because $q = F(F(q))$.

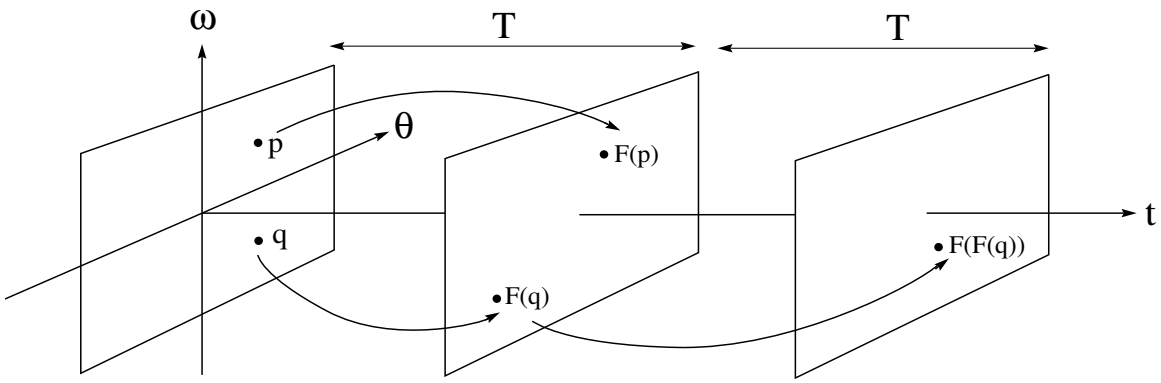
Figure 4: Sequence of initial states of the heart converging to the period-1 point $(-0.70, -0.38)$ on R&G's T-periodic solution for heart rate 75. The dots indicate the initial states, the first six of which are numbered. The closed curve indicates the T-periodic solution.

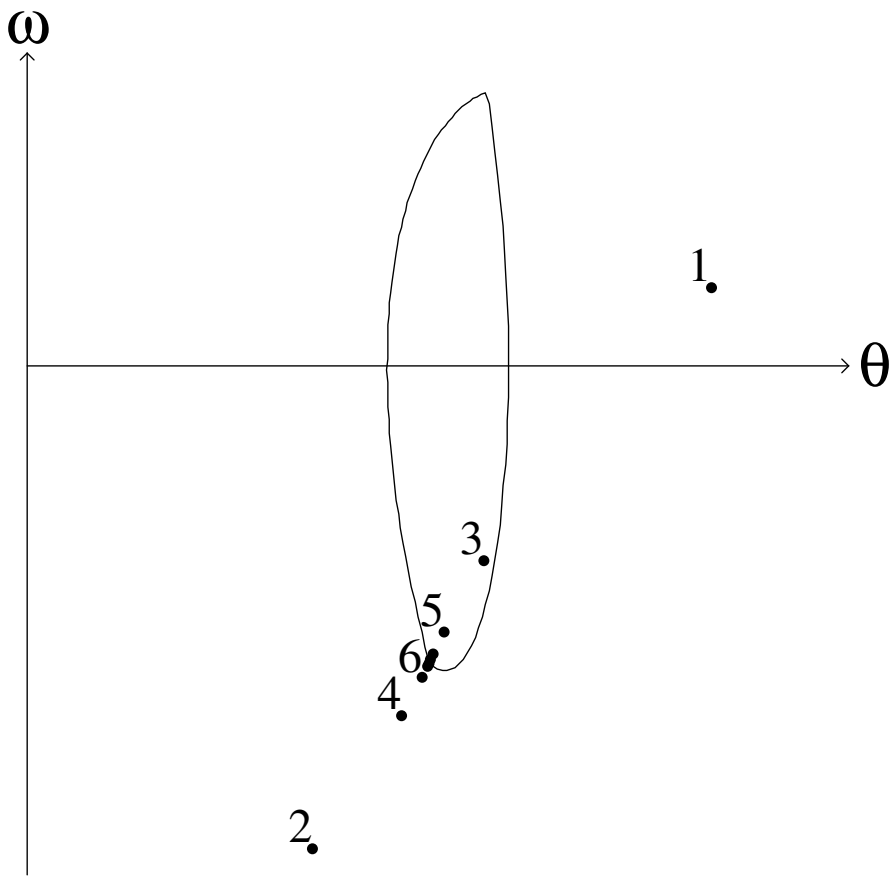
Figure 5: Branches of periodic points for the heart equation projected onto the (h, θ_l) and (h, ω) spaces with h the heart rate and θ_l and ω the angle and angular velocity at the start of diastole. Thick and thin lines indicate stable and unstable points. Branch 1 is period-1

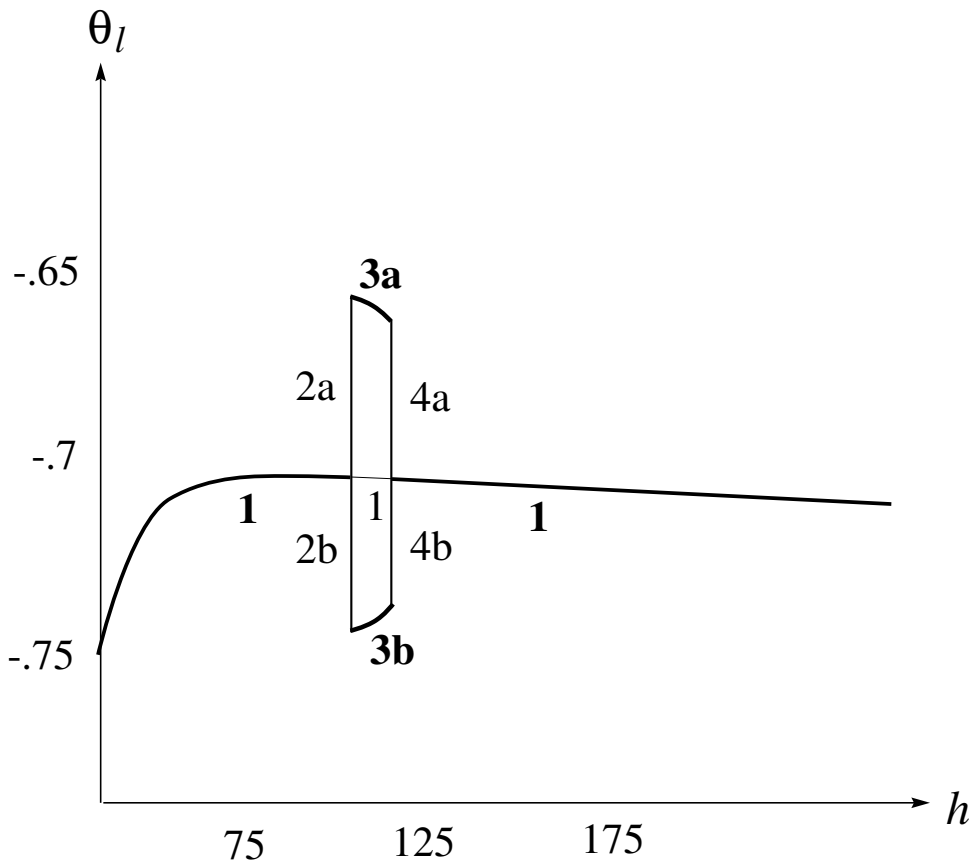
and consists of two stable segments separated by an unstable segment. Branches 2–4 are period-2. The results predict 1:1 swinging at heart rates of 104 and below, 2:1 swinging at heart rates between 105 and 116, and 1:1 swinging at heart rates of 117 and above.


Pivot point
Direction of flow
in the outflow tract
and great vessel
Direction of flow
as blood leaves
the heart chamber
R
L


direction of
fluid movement
pivot of heart's
pendular movement
L
Φ
ϕ_r
ϕ_l
M_r
M_l
limiting
wall
d
α_r
α_l
θ_r
θ_l
+ angles
- angles
direction
of gravity


ω
T
T
θ
p
F(p)
q
F(F(q))
F(q)
t


ω
θ
1
2
3
4
5
6


θ_l
-.65
-.7
-.75
3a
2a
4a
1
1
1
2b
4b
3b
h
75
125
175

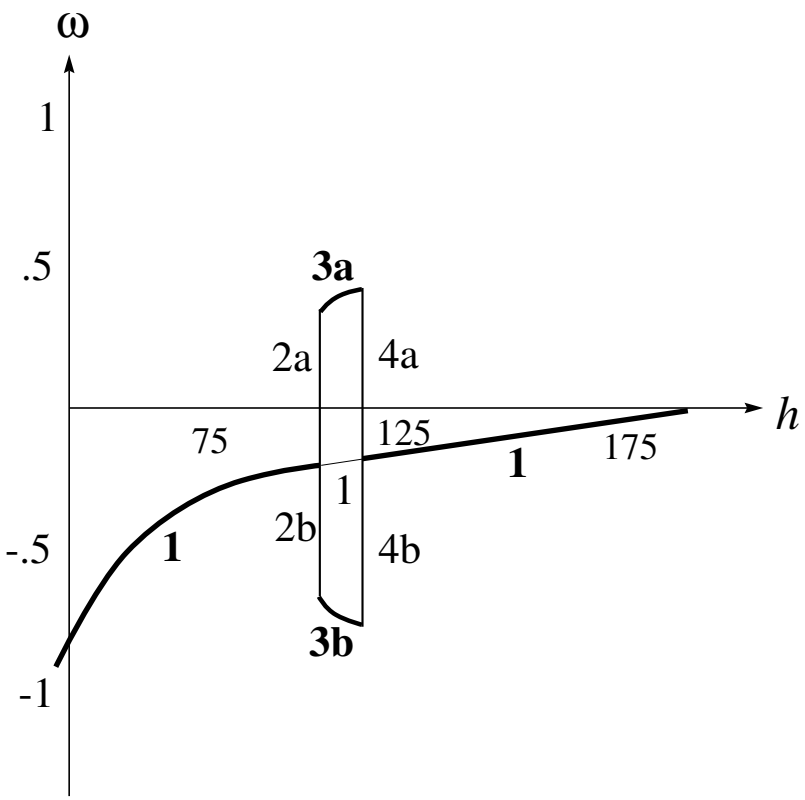

ω
1
.5
-.5
-1
3a
2a
4a
h
75
125
175
1
1
2b
4b
3b

Figure 1:

Figure 2:

Figure 3:

Figure 4:

Figure 5: