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Influence of atrio-ventricular conduction on the statistical properties of ventricular beat intervals during atrial fibrillation

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Abstract: Atrial fibrillation is commonly described by irregular electrical excitations circulating in the atria. Developing methods to characterise and classify various kinds of atrial fibrillation is an important task, since it allows one to improve the diagnosis and to support the choice of suitable therapies. While most methods use invasive intraatrial measurements by catheter electrodes, one can obtain valuable information also from the noninvasive surface ECG by studying the statistics of ventricular impulses. Such approach, however, requires a good understanding of the conduction of impulses through the atrio-ventricular node. Based on a simple conduction model we study how the distribution of ventricular interbeat intervals is affected by the atrial fibrillation. Due to the nonlinear properties of the AV node, a surprisingly rich pattern of behaviours is found, even if the fibrillation is characterised in terms of a Poisson process with only one characteristic fibrillation rate.

Keywords – atrial fibrillation, fibrillation rate, AV node, AV nodal recovery curve, refractory period, conduction model, distribution of ventricular interbeat intervals

Conduction model

We calculate the sequence of ventricular responses to atrial excitations by employing the conduction model proposed by Zeng and Glass [1], which relies on the conduction properties of the atrio-ventricular (AV) node. The time it takes for an atrial excitation to be transferred to the ventricle is the conduction time $t_{\rm con}$. During this time and the refractory period θ following it, atrial impulses are blocked. Another atrial impulse is conducted if it arrives at the AV node at a time larger than $t_{\rm con} + \theta$. The time interval between $t_{\rm con} + \theta$ and the next atrial impulse conducted to the ventricle is the recovery time $t_{\rm rec}$.

Measurements revealed [2] that the conduction process speeds up with the recovery time $t_{\rm rec}$. This effect is described by the AV nodal recovery curve $t_{\rm con}(t_{\rm rec})$ [3], which in the simplest case can be expressed by a single exponential relaxation with relaxation time $\tau_{\rm rec}$:

$$t_{\rm con} = \alpha + \beta e^{-t_{\rm rec}/\tau_{\rm rec}}.$$
 (1)

Here α and $\alpha + \beta$ are the minimal and maximal conduc-

tion time, respectively. In the following we define $\tau_{\rm rec}$ as our time unit (typically $\tau_{\rm rec} = 0.3$ sec). The intervals between two atrial impulses (AA) are denoted by a and the intervals between ventricular excitations (VV) by v.

Unfortunately, the statistical properties of the atrial intervals have so far not been investigated in much detail. Atrial signals, usually obtained by a bipolar catheter electrode brought into the atria, are almost uncorrelated and exhibit a unimodal distribution with a peak at the inverse fibrillation rate of about 0.2 sec. However, measurements at different places in the atria have shown that the atrial fibrillation rate is not unique. This is important since the AV node is an extended region, whose exact specification is still under discussion. Thus it is not clear presently how the atrial intervals arriving at the AV node are distributed in general.

In view of this limited knowledge we here start with the simplest model, where atrial impulses are characterised by a Poisson process with an effective fibrillation rate f. The distribution of a then is

$$p_a(a) = fe^{-fa}, \quad 0 \le a < \infty. \tag{2}$$

Due to the absence of memory in the Poisson process, the transition probability for the first conducted atrial impulse to occur at a certain time after a ventricular excitation is independent of the previous atrial excitation. Hence the distribution of the ventricular intervals is given by

$$p_{\nu}(\nu) = \int_{0}^{\infty} dt_{\rm rec} f e^{-ft_{\rm rec}} \delta\left(\nu - \theta - t_{\rm rec} - t_{\rm con}(t_{\rm rec})\right). \quad (3)$$

By performing the integral in eq. (3) we obtain the following distributions dependent on the parameter β ,

$$\beta \le 1: p_{\nu}(\nu) = p_2(\nu), \quad \nu_2 < \nu$$
 (4a)

$$\beta > 1: \ p_{\nu}(\nu) = \begin{cases} p_1(\nu) + p_2(\nu), & \nu_1 < \nu < \nu_2, \\ p_2(\nu), & \nu_2 \le \nu, \end{cases}$$
(4b)

where $v_1 = \theta + \alpha + 1 + \ln \beta$, $v_2 = \theta + \alpha + \beta$,

$$p_1(v) = \frac{fe^{-fu_1}}{\beta e^{-u_1} - 1}, \qquad p_2(v) = \frac{fe^{-fu_2}}{1 - \beta e^{-u_2}},$$
 (4c)

and $u_1 < u_2$ are the positive roots of

$$g(u) = u + \beta e^{-u} = v - \alpha - \theta. \tag{5}$$

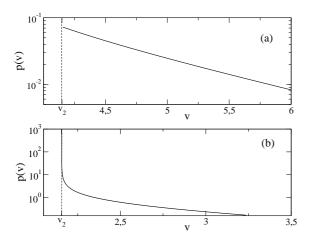


Figure 1: Semi-logarithmic plot of the probability density $p_{\nu}(\nu)$ for (a) $\beta=0.9$ and (b) $\beta=1$ (according to eq. (4a)). The parameters are f=3.3, $\alpha=0.147$ $\theta=0.2$ (note that all times are given in units of $\tau_{\rm rec}$).

Note that the times v_1 and v_2 are the smallest possible interbeat intervals in the cases $\beta > 1$ and $\beta < 1$, respectively.

For large intervals v, p(v) decays exponentially,

$$p_{\nu}(\nu) \sim f e^{f(\theta + \alpha)} e^{-f\nu}, \qquad \nu \to \infty.$$
 (6)

Accordingly, the decay rate equals the fibrillation rate f, while the amplitude $fe^{f(\theta+\alpha)}$ contains information about the conduction parameters α and θ . The asymptotics can be understood from the fact that large intervals a are not affected by the detailed form of the AV nodal recovery curve.

For small ν , in contrast, the shape of $p_{\nu}(\nu)$ is more sensitive to the conduction parameters. Three different cases dependent on the values of β are obtained,

$$\beta < 1: \ p_{\nu}(\nu) = \frac{f}{1-\beta} - \frac{f[f(1-\beta)+\beta]}{(1-\beta)^{3}} (\nu - \nu_{2}) \\ + \mathcal{O}((\nu - \nu_{2})^{2}), \qquad (7a)$$

$$\beta = 1: \ p_{\nu}(\nu) = \frac{f\beta^{-f}}{\sqrt{2(\nu - \nu_{2})}} - f^{2}\beta^{-f} \\ + \mathcal{O}((\nu - \nu_{2})^{1/2}), \qquad (7b)$$

$$\beta > 1: \ p_{\nu}(\nu) = \frac{2f\beta^{-f}}{\sqrt{2(\nu - \nu_{1})}} + \mathcal{O}((\nu - \nu_{1})^{1/2}). \quad (7c)$$

Considering the overall shape of $p_v(v)$ even four different patterns of behaviour can be distinguished: (a) For $\beta < 1$, $p_v(v)$ monotonously decreases from a finite value (cf. Fig. 1a). (b) For $\beta = 1$, $p_v(v)$ also decays monotonously but has a square root singularity for $v \rightarrow v_1$ (cf. Fig. 1b). (c) For $\beta > 1$ and f smaller than a critical frequency $f_c > \beta/(\beta-1) > 1$ has a behaviour as in case (b) but in addition there is a discontinuous jump at $v = v_2$ (cf. Fig. 2c). (d) For $\beta > 1$ and $f > f_c$ the behaviour is the same as in case (c) but $p_v(v)$ exhibits a minimum in the intervall $v_1 < v < v_2$ (cf. Fig. 2d).

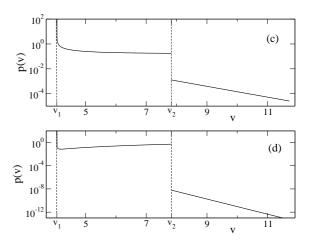


Figure 2: Semi-logarithmic plot of the probability density $p_{\nu}(\nu)$ for $\beta = 2$, and for (c) $f = 3.3 < f_c$ and (d) $f = 8.3 > f_c$ (according to eq. (4b)). The other parameters are the same as in Fig. 1.

In view of the simplicity of the model the shape sensitivity of the VV interval distribution is remarkable. More realistic models may lead to an even richer behaviour. Hence there is hope to extract important physiological parameters by a detailed shape analysis of the distribution of ventricular beat intervals.

It is clear that the distributions shown in figures 1,2 are not realistic. Measured distributions $p_{\nu}(\nu)$ do not show a monotonous decrease but exhibit a maximum close to the mean VV interval. However, such distributions can be obtained already by slight modifications of the simple model studied above. For example, the refractory period θ does not have a unique value in reality but will fluctuate in time. Taking a uniform distribution in some interval $[\theta_1, \theta_2]$ to account for these fluctuations, gives the more realistic distribution shown in Fig. 3. These data were obtained by a simulation of the model (an analytical calculation is also possible based on performing the average over the distribution of θ in eq. 3).

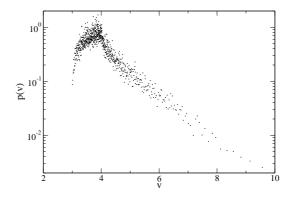


Figure 3: Semi-logarithmic plot of the probability density $p_{\nu}(\nu)$ for the conduction model discussed in the text, with a variable refractory period drawn from a uniform distribution in the interval [1,2]. The parameters with unique values are $f=\alpha=\beta=1$. $\tau_{\rm rec}$ is used as time unit.

Summary

We modelled distributions of ventricular interbeat intervals based on a simple model for the conduction of electrical impulses through the AV node. Irregular atrial excitations arriving at the AV node were described in terms of a Poisson process with a rate f specifying the effective atrial fibrillation rate. We found that the distribution of VV intervals decays exponentially with the rate f and is largely unaffected by the details of the conduction process for large VV intervals. By contrast, for small VV intervals, the distribution is more sensitive to the AV conduction parameters. We conclude that a detailed shape analysis of the VV distribution should allow one to extract important physiological parameters, both for the fibrillatory dynamics in the atria and for the AV conduction processes. Further work in this direction offers a route for a better classification of atrial fibrillation.

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