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Comparison of three artificial models of the MHD effect on the electrocardiogram

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Abstract

The Electrocardiogram (ECG) is often acquired during Magnetic Resonance Imaging (MRI) for both image acquisition synchronisation with heart activity and patient monitoring to alert for life-threatening events. Accurate ECG analysis is mandatory for cutting-edge applications, such as MRI guided interventions. Nevertheless, the majority of the clinical analysis of ECG acquired inside MRI is made difficult by the superposition of a voltage called the MagnetoHydroDynamic (MHD) effect. MHD is induced by the flow of electrically charged particles in the blood perpendicular to the static magnetic field, which creates a potential of the order of magnitude of the ECG and temporally coincident with the repolarisation period.

In this study, a new MHD model is proposed which is an extension of several existing models and incorporates MRI-based blood flow measurements made across the aortic arch. The model is extended to several cardiac cycles to allow the simulation of a realistic ECG acquisition during MRI examination and the quality assessment of MHD suppression techniques. A comparison of two existing models is made with our new model and with an estimate of the MHD voltage observed during a real MRI scan.

Results indicate a good agreement between our proposed model and the estimated MHD for most leads, although there are clearly some discrepancies with the observed signal which are likely to be due to remaining deficiencies in the model. However, the results demonstrate that our new model provides a closer approximation to observed MHD effects and a better depiction of the complexity of the MHD effect compared to the previously published models. The source code will be made freely available under and open source license to facilitate collaboration and allow more rapid development of more accurate models of the MHD effect.

Keywords

Electrocardiogram; Modelling; Magnetic Resonance Imaging; Magnetohydrodynamic Effect

1. Introduction

The 12-lead electrocardiogram (ECG) is an important tool for measuring cardiovascular activity during medical procedures. However, some procedures require an environment which is hostile to ECG acquisition. Magnetic Resonance Imaging (MRI), a noninvasive radiological technique, is a particular case in point. ECG acquisition is required during MRI examinations for two major reasons. Firstly during cardiac MRI applications, acquisitions have to be synchronised with heart activity to reduce motion artefacts in the images (Scott et al. 2009). The ECG is thus used as information on cardiac activity and the QRS complexes are used to initiate MRI acquisitions. This task is known as triggering or gating, and can be performed prospectively or retrospectively. Secondly for safety reasons, one wants to ensure that the patient condition does not deteriorate during the MRI examination, so that physiological parameters need to be acquired throughout the process (Shellock 2001). Among these physiological parameters, the ECG is one of the most important, since it provides a rapid and standard method to detect conduction and perfusion issues.

Electrocardiography and MRI make use of electromagnetic fields whether by measuring the fields induced by the heart directly or by exciting the spins of the water particles of the body with the MRI scanner. The ECG is therefore susceptible to interference from the MRI system since the presence of the high static magnetic field distorts the ECG signal, the Magnetohydrodynamic (MHD) effect, which results from the flow of ionically charged blood in the magnetic field as a direct consequence of the Lorentz force $\mathbf{F} = q\mathbf{u} \wedge \mathbf{B}$. The MHD effect thus increases in regions with increased blood velocity and volume and if the flow is primarily perpendicular to the magnetic field. Therefore it has been assumed that the effect is mainly induced during blood ejection through the aortic arch (Gupta 2007; Gupta et al. 2008).

Analysis of the MHD effect has been conducted in part due to fears of the hazardous consequences on human physiology, namely the possibility of increased blood pressure, decreased blood flow and interactions between the MHD effect and cardiac pacing. Different studies, ranging from experiments on animals to realistic models with rigid tubes or theoretical analysis with rigid vessels, have concluded that the immersion of human bodies in magnetic field up to 15T is safe. The signal distortion of the ECG has been demonstrated with *in vivo* experiments in various animals (Beischer and Knepton 1964; Togawa et al. 1967; Gaffey et al. 1980; Gaffey and Tenforde 1981; Tenforde et al. 1983; Jehenson et al. 1988; Tenforde 1992, 2005). Noise on the ECG signal has been shown to be correlated with blood flow between heart chambers and also with rapid ejection of blood into pulmonary and aortic vessels. To better understand the phenomenon and predict the physiological effect, theoretical analysis have been conducted, first by approximating the aorta with a rigid vessel with a non conducting wall (Keltner et al. 1990), then by taking into account the conductivity of the vessel wall (Kinouchi et al. 1996; Tenforde 2005) and solving the fluid dynamics with finite element methods. The results of these studies have shown no significant effects on cardiac hemodynamics or function, with a slowing of the blood flow of less than 5% at 10T, an increase in blood pressure of 3% at 8T and the coupling of the magnetic field and blood flow creating current densities at the sinoatrial node region unlikely to produce effects on cardiac pacing under clinical conditions. The

major contribution to the MHD effect has been identified as being due to the blood flow in the aortic arch, resulting from geometrical and physiological considerations.

As a consequence, the MHD effect corrupts the ECG immediately after ventricular ejection, which is simultaneous with the ST segment on the ECG. Detection of acute ischemia is thus practically impossible, since the MHD effect can have a larger amplitude than the T-waves. False triggering can also occur if the R wave amplitude is exceeded leading to motion artefacts on images, especially with the growing use of higher static magnetic field in clinical applications (3 to 7T).

Some specific QRS detectors have been designed to discard MHD effect from QRS complexes (Fischer et al. 1999; Oster et al. 2009). The replacement of the ECG by other cardiac signals, such as heart sounds (Frauenrath et al. 2008) or photoplethysmography has been proposed in order to avoid the distortions due to the MHD effect (Scott et al. 2009). Nevertheless no current methods lead to a signal which provides an accurate diagnosis of ischemic events. Effective methods to produce a diagnostic quality ECG would be of great clinical benefit. There are three main applications during which this knowledge is useful: 1) MRI guided surgery (McVeigh et al. 2006; Saikus and Lederman 2009; Lederman 2005; George et al. 2011), 2) intracardiac electrophysiology (Susil et al. 2002) and 3) stress testing by MRI (Cheng et al. 2005; Jekic et al. 2008, 2010). However, in order to develop a tool which can provide a useful ECG during MRI, realistic models of the MHD effect are required. Gupta et al. (2008) suggested the use of the solution for steady flow in rigid vessels and realistic aortic and torso models. Abi-Abdallah et al. (2009) proposed to solve the MHD blood flow problem in the presence of a physiological pressure gradient. Nijm et al. (2008) suggested the use of realistic inhomogeneous torso model with a simulated blood flow. More recently, some groups have proposed the use of finite element techniques in order to simulate the blood flow in arteries and the corresponding potentials on the patient's torso. In particular, Kyriakou et al. (2012) proposed to solve the MHD blood flow in a decoupled way, first simulating the blood flow in the primary arteries (aorta, pulmonary arteries) and secondly solving the electromagnetic problem to simulate the MHD effect measured by the ECG electrodes. In a recent work, Oster *et al.* (Oster et al. 2012) proposed a model of the MHD effect based upon a sum of Gaussians, motivated by the work of Clifford and McSharry (2004). since any morphological blood pressure or ECG characteristic can be modelled by an arbitrary number of Gaussians (Clifford (2006)), blood flow and the the MHD effect could be well modelled with arbitrary accuracy, if the underlying ECG is known. However, this work aims to identify a suitable model from which an estimate of the MHD effect can be estimated, in order to then identify the myocardial activity. Therefore we will not consider this model further.

Lastly, Martin et al. (2012) developed a simulation based on finite element techniques, addressing the blood flow, the heart activity and the diffusion through the torso in a coupled fashion. In this last simulation, the authors concluded that the ECG acquired inside the MR bore can be approximated by the superposition of the ECG without magnetic field and of the MHD signal, which is proportional to the magnetic field $\mathbf{B}$ and to the flux $\mathbf{q}$ in cm^3s^{-1} as follows;

$$V_i^{ECG}(B_0) \approx V_i^{ECG}(0) + \alpha_i B_0 q, \quad (1)$$

where V_i^{ECG} represents the signal measured on lead i of the 12-lead set and α_i are scaling constants and are given in table 1.

In this paper we explore and extend a recently described model, indicating deficiencies, and suggesting updates to improve correspondence with real data. ECGs recorded during MRI were used to qualitatively validate our model, and the remaining deficiencies are described.

2. Methods

Three approaches for generating a realistic model of the MHD effect were implemented. First, realistic geometric models of the torso, heart and aorta are required for accurate simulation of both the ECG and the MHD. These models are presented in subsection 2.1 and will be used for simulating the modelings. The three models compared are:

1. The theoretical solution of the Navier-Stokes equation is presented with some assumptions, which allows for the simulation of the first MHD modelling in subsection 2.2
2. A new modelling based on MRI 4D phase-contrast blood flow measurements across the aortic arch is proposed in subsection 2.3
3. A previously presented model from the literature (Martin et al. 2012, 2011) is utilised.

These three models are compared with an estimation of the MHD effect extracted from real ECG acquisitions during MRI, the method of estimating the MHD effect is presented in subsection 2.4. This section ends with a description of the methodology for the comparison of the models in the subsection 2.5.

2.1 Realistic geometric models

In order to simulate surface ECG measurements, the open source ECGSim package was used to provide a forward model (van Oosterom and Oostendorp 2004). ECGSim contains a realistic 3D model of the heart and the torso, and the electrical activity of the heart's ventricles and provides a solution to the forward problem, resulting in realistic QRST waves on the surface of the body. P waves can be modelled in the recent version of the ECGSim software but were not included in this study, where the focus is on the distortion of ST segment by the MHD effect.

A model of the aortic arch, developed by Gupta *et al.* (Gupta 2007; Gupta et al. 2008) can describe the three dimensional topology of the vessel as:

$$\begin{aligned} x_A &= R_a(\Omega) \cos(\Psi_a(\Omega)) \cos(\Omega) \cos(\pi/4), \\ y_A &= -R_a(\Omega) \sin(\Psi_a(\Omega)) \sin(\pi/4), \\ z_A &= R_a(\Omega) \cos(\Psi_a(\Omega)) \sin(\Omega), \end{aligned} \quad (2)$$

where Ω describes a 3D grid such that $\Omega \in [-\frac{3\pi}{24}, -\frac{\pi}{24}, \frac{\pi}{24}, \dots, \frac{27\pi}{24}]$, R_a is the radial function given by

$$R_a = 6.513 \times 10^{-2} \Omega^4 - 4.215 \times 10^{-1} \Omega^3 + 7.732 \times 10^{-1} \Omega^2 - 3.255 \times 10^{-1} \Omega + 2.759 \quad (3)$$

and Ψ_a is the azimuthal angle which is described by

$$\Psi_a = -(\Psi_1 + (\Psi_2 - \Psi_1) \chi(\frac{\Omega}{\Theta})) \cos(2\Omega), \quad (4)$$

with $\Psi_1 = 9.559 \times 10^{-2}$, $\Psi_2 = 1.598 \times 10^{-1}$, $\Theta = 3\pi/4$ and

$$\begin{cases} \chi(\frac{\Omega}{\Theta}) = 0 & (\frac{\Omega}{\Theta} < 0) \\ \chi(\frac{\Omega}{\Theta}) = 3(\frac{\Omega}{\Theta})^2 - 2(\frac{\Omega}{\Theta})^3 & (0 \leq \frac{\Omega}{\Theta} < 1) \\ \chi(\frac{\Omega}{\Theta}) = 1 & (\frac{\Omega}{\Theta} \geq 1) \end{cases} \quad (5)$$

By translating the model so that the aortic arch starts from the approximate center of the heart, one can make the different models (heart, torso and aorta) match together (see figure 1).

The aortic model was translated such that it originates from the 51st node of the heart model. The model of the aorta with its 16 points in the arch therefore provides 15 sections of one centimeter length. The direction of each section is then computed. The models are oriented such that the backbone is along the z axis and the magnetic field is also oriented along the z axis.

The simulation of the MHD effect can be computed by adding the contributions of each of these sections. These contributions can be simulated by computing the pulsed blood flow in this rigid vessel (the aortic arch) under a physiologically realistic pressure gradient (Abi-Abdallah et al. 2009). This pressure profile is the same for each section of the arch except for a delay due to the propagation of the pulse through the aortic arch (Gupta et al. 2008). This delay between two consecutive sections is computed by:

$$\delta t = \frac{L}{V_{PW}}, \quad (6)$$

where V_{PW} is the pulse wave velocity, set to 6.5 ms^{-1} and L is the length of the section (1cm).

Once the body potentials are computed, the resulting ECG signals can be found by using the formulae provided in the ECGSim package. These formulae are determined by the nodes corresponding to the standard electrode placement, which is given in table 2.

2.2 Theoretical solution using Navier-Stokes

In this section, we present a theoretical MHD model Abi-Abdallah et al. (2009) to solve the MHD problem along the aortic arch under simplified assumptions.

2.2.1 General Equations—Consider a rigid vessel oriented along an arbitrary direction $\mathbf{k}$, such that $\mathbf{k} = \sin \varphi_V \cos \theta_V \mathbf{x} + \sin \varphi_V \sin \theta_V \mathbf{y} + \cos \varphi_V \mathbf{z}$ and immersed in a static magnetic field $\mathbf{B} = B_0 \mathbf{z}$, as shown in figure 2. The Navier-Stokes equation is given by:

$$\rho \left(\frac{\partial \mathbf{u}}{\partial t} + (\mathbf{u} \cdot \nabla) \mathbf{u} \right) = -\nabla p + \eta \nabla^2 \mathbf{u} + \sigma (\mathbf{u} \wedge \mathbf{B}) \wedge \mathbf{B}, \quad (7)$$

where $\mathbf{u}$ is the velocity profile, p is the pressure, η the blood viscosity, σ its conductivity and ρ the blood density. Induced fields are neglected (Abi-Abdallah et al. 2009).

An analytical solution of equation (7) can be calculated (see appendix A) and the flow rate at a cross-section of the arteries can be deduced by integrating over the area of the cross sectional area of the aortic arch as follows

$$q(t) = \int_A \mathbf{u} \cdot \mathbf{k} dA, \quad (8)$$

where A is the cross section of the segment of the aortic arch.

Abi Abdallah *et al.* (Abi Abdallah 2007; Abi-Abdallah et al. 2009) have shown that the resulting dipolar electrical moment is given by

$$\mathbf{p} = \varepsilon B_0 \sin \phi L q(t) \mathbf{i}, \quad (9)$$

where ε is the dielectric constant of the blood.

The surface body potential, at a point $M = (x_M, y_M, z_M)$ can then be computed as

$$\Phi(M, t) = \frac{B_0 \sin \phi L}{4\pi} \frac{\sin(\theta_V) x_M - \cos(\theta_V) y_M}{d(M, A)^3} q(t), \quad (10)$$

where $d(M, A) = ((x_M - x_A)^2 + (y_M - y_A)^2 + (z_M - z_A)^2)^{1/2}$ and $A = (x_A, y_A, z_A)$ are the coordinates of the center of the section of the aortic arch.

2.2.2 Pressure profile model—The last component to be computed in order to assess the potential induced by the blood flow in a section of the aortic arch is the pressure gradient, $g(t)$. A widely used model for the aortic pressure is a three-element Windkessel lumped model (Westerhof et al. 1971), which draws an analogy between the pressure profile inside the arteries and an electrical circuit. The equivalent diagram is depicted in figure 3.

We then define the ventricular pressure to be

$$P_V(t) = \begin{cases} \frac{P_{max}}{2}(1 - \cos(2\gamma t)) & 0 \leq t \leq t_p \\ 0 & t_p \leq t \leq T \end{cases} \quad (11)$$

with T being the period and t_p being $1/2T$, $\gamma = \pi/t_p$ and P_{max} being the systolic pressure.

Given the Windkessel model, the aortic pressure P_A follows the relationship

$$\frac{dP_A(t)}{dt} + \frac{P_A(t)}{R_p C} = \frac{Q(t)}{C}, \quad (12)$$

where the output flow $Q(t)$ is defined by:

$$Q(t) = \begin{cases} \frac{(P_V(t) - P_A(t))}{R_a} & \text{while } P_V(t) > P_A(t) \\ 0 & \text{else} \end{cases} \quad (13)$$

An analytic solution of equation (12) has been given by Abi-Abdallah et al. (2009). The flow rate in the aorta can thus be calculated and is shown in figure 4.

By assuming that the flow is laminar in the aortic section (with a radius a) the pressure drop can be approximated by solving Poiseuille's equation, which is given by:

$$\frac{\Delta P_A(t)}{L} = -\frac{8\eta}{\pi a^4} Q(t) = -\frac{8\eta}{\pi a^4} \frac{\max(0, (P_V(t) - P_A(t)))}{R_a} \quad (14)$$

The pressure gradient profile is defined as $g(t) = -\frac{a^2}{\eta} \frac{\partial p}{\partial t}$ (Abi-Abdallah et al. 2009), and therefore

$$g(t) = \frac{8}{\pi a^2} \frac{\max(0, (P_V(t) - P_A(t)))}{R_a}. \quad (15)$$

2.3 MHD modelling based on 4D blood flow measurements from MRI

The theoretical solution of the MHD effect is based on the assumptions that the blood flow is laminar and unidirectional. These assumptions have been shown to be inaccurate since the blood flow generates vortices and twists, especially in the ascending aorta (Markl et al. 2005; Hope et al. 2007). In order to show these perturbations in the aortic blood flow, Markl et al. (2005) have used 4D blood flow measured with phase-contrast MRI.

In order to improve the simulation of the MHD effect given in the last section, we made the same type of measurements across the aortic arch. These measurements were then used to determine the dipolar moment the blood flow creates by adapting equation (9). The use of a 4D blood flow measurement made it possible to observe the nonlinearities in the blood flow. The flow rate thus had components not only along the $\mathbf{k}$ direction, but also in the $\mathbf{i}$ and $\mathbf{j}$

directions. These two new components will contribute to the MHD potential on the body torso such that

$$\Phi(M, t) = \Phi_k(M, t) + \Phi_j(M, t) + \Phi_i(M, t) \\ = \frac{B_0 \sin \phi L}{4\pi} \frac{\sin(\theta_V) x_M - \cos(\theta_V) y_M}{d(M, A)^3} q_k(t) + \frac{B_0 \cos \phi L}{4\pi} \frac{\sin(\theta_V) x_M - \cos(\theta_V) y_M}{d(M, A)^3} q_j(t) + \frac{B_0 L}{4\pi} \frac{-\cos(\theta_V) x_M - \sin(\theta_V) y_M}{d(M, A)^3} q_i(t), \quad (16)$$

where $q_i(t)$, $q_j(t)$ and $q_k(t)$ are the blood flow rate in the three different directions measured with MRI.

Measurements were carried on a single subject on a 1.5T General Healthcare (Wawkesha, WI) MRI scanner, after receiving institutional review board approval. Blood directional velocity over the cardiac cycle measurements were performed at four cross sections of the aortic arch with a Phase Contrast Steady State Free Precession (SSFP) Cine sequences (Repetition Time, TR = 10.48ms, Echo time, TE = 5.04ms, flip angle = 20, Field Of View, FOV = 350 × 350mm, 30 cardiac phases, 8 views per segment and Velocity Encoding = 150cm/s).

The imaging slices were placed such that two measurements were realised in the ascending and descending aorta, and two were located in the aortic arch. Their placements are depicted in figure 5. For each plane, the corresponding cross section of the aorta were delineated by hand and the flow rate in the three different directions were computed, by integrating the phase values in the delineated region of interest.

The aortic model described in section 2.1 was used to compute the contribution of each aortic section to the MHD effect, which contained 15 consecutive segments. Four measurements were taken and so each measurement was used for several sections of the model. The first five aortic contributions were computed by using the flow rate measured in the ascending aorta, the sixth to the eighth sections' contributions by using the cross section in the ascending aortic arch, the ninth to eleventh sections by the measurements in the descending aortic arch and the last sections (i.e. twelfth to fifteenth) were computed with the measurements in the descending aorta.

2.3.1 MHD amplitude correction factor—In our modelling we simplified the forward propagation of the potential on the torso induced by the MHD effect, by computing the torso potential resulting from the radiation of the dipolar electrical moment. Abi Abdallah (2007) has shown that such simplification yields a good approximation of the MHD contribution, but with a slight underestimation of its true value. Recently, modelling of the MHD effect has been recomputed by using realistic geometrical models and numerical methods the MHD model can be assumed (to a first approximation) to be linearly mixed with the myocardial electropotential sources (Martin et al. 2012, 2011).

However, the computed magnitude of the MHD effect has been found to be different from observations and therefore an amplitude correction factor is needed. Equation (1) can be used in order to find a proportionality factor needed to adjust the amplitude of the simulated MHD effect, $\mathbf{V}^{\text{MHD}}$. This amplitude correction factor, denoted a_{norm} , can be computed by solving first the following linear equation:

$$\mathbf{V}^{\text{MHD}} = \alpha_{\mathbf{q}} \times B_0 \times \mathbf{q}^T, \quad (17)$$

where $\mathbf{q}$ is the norm of the measured flow rate in the second slice. The $\alpha_{\mathbf{q}}$ components represent the equivalent proportionality coefficients as in equation (1), by assuming the principal contribution of the MHD comes from the flow in the aortic arch (rather than any other arterial system). This value can be confirmed by computing the Singular Value Decomposition (SVD) of the MHD effect, $\mathbf{V}^{\text{MHD}}$, and the eigenvector associated with the largest eigenvalue turns out to be approximately colinear with the vector $\alpha_{\mathbf{q}}$.

Once the $\alpha_{\mathbf{q}}$ components have been found, the MHD amplitude correction factor, α_{norm} , can be easily computed with the following relation;

$$\alpha_{\text{norm}} = \frac{\|\alpha\|}{\|\alpha_{\mathbf{q}}\|}. \quad (18)$$

2.3.2 RR interval time series generation—The main purpose of this research was to build a realistic simulation of ECG acquired during MRI. Applying the above described methodology, we were able to build an artificial signal representing one cardiac cycle of such signal. Nevertheless, we wish to build a continuous signal in order to assess the quality of any denoising method. It is therefore important to build a signal containing several successive cardiac cycles, with their characteristic beat-to-beat (RR) time interval variations. A realistic RR interval time series was simulated with the method proposed by McSharry et al. (2003). This time series was simulated such that its power spectrum consists of a bimodal power spectrum with a sum of two Gaussians distribution. The parameters of these Gaussians determine the frequencies for simulation of Respiratory Sinus Arrhythmia (RSA) and the Mayer waves, but also the relative balance of the contributions of the sympathetic and parasympathetic branches of the autonomic nervous system.

2.3.3 QT interval correction factor—The ECG modelling used in this study only takes into account the ventricular depolarisation, that is only the QRS and the T waves are modelled. It has been shown that the diastole and the systole durations do not evolve linearly with the RR interval duration. Several models of QT interval evolution have been presented, among them one of the most famous linking the RR duration with the QT interval through the Bazett formula. However, because we want to be able to study extreme heart rate cases, we have chosen to implement the QT interval relationship with the Fridericia formula (Fridericia 2003), given by

$$QT_c = QT \times \beta \sqrt[3]{RR}, \quad (19)$$

where β is a constant, which was determined through simulation using ECGsim performed with $RR = 100\text{ms}$, such that $\beta = 44.5\text{ms}^{2/3}$. The modelling of the ECG signal has thus been linearly stretched between the onset of the Q wave and the offset of the T wave. As the atrial depolarisation period has not been modelled in this study, this period was set to 0V.

2.3.4 MHD amplitude fluctuations with the Heart Rate—In this study the modelling of the MHD has been assumed to be induced only by the flow through the aortic arch, which is clearly dependant on the heart rate. The heart can be compared to a pump and the cardiovascular mechanics can easily be modelled. The cardiac output (CO) can be linked to the heart rate (HR) (Mark 2004):

$$CO = \frac{6.9}{0.08 + \frac{3}{HR}} \quad L/min. \quad (20)$$

Since the stroke volume (SV) is the cardiac output divided by the heart rate, one can write

$$SV = \frac{6.9}{0.08HR + 3} \quad L/beat \quad (21)$$

and the stroke volume can be computed from the flow rate by

$$SV = \int q(t) dt \quad (22)$$

By assuming that the MHD effect is only induced by the flow rate, then its integral over the cardiac cycle is proportional to the SV and is thus subject to the same dependency on HR.

The easiest way of taking this dependency into account is to multiply the MHD effect by a SV correction factor and stretch the MHD effect so that it fits the duration of the RR interval (as the stretching procedure does not have an effect on the integral value). The flow measurements have been performed while the subject had a mean HR of 60bpm, thus the SV correction factor, α_{SV} , can be computed from (21) as follows:

$$\alpha_{SV} = \frac{SV(HR)}{SV(60)} = \frac{0.08 \times 60 + 3}{6.9} \frac{6.9}{0.08HR + 3} = \frac{780}{8HR + 300}. \quad (23)$$

2.4 Estimation of MHD using subtraction

The estimation of the MHD model was computed by a simple linear combination of two recordings. The first recording was performed while the subject held his breath inside the MR bore in a supine position feet-first (ECG_{ff}). The second recording was performed under the same conditions in a head-first position (ECG_{hf}). Both recordings were 20s long.

The ECG template for each recording was estimated by detecting the QRS complexes and averaging over all cardiac cycles. The two templates were then subtracted and this new template was divided by two. This new template corresponds to a combination of the MHD effect since each recording can be assumed to correspond to ECG plus or minus given the orientation of the patient (Tse et al. 2012).

$$\begin{aligned}
 ECG_{hf} &= ECG + MHD \\
 ECG_{ff} &= ECG - MHD \\
 MHD &= \frac{1}{2}(ECG_{hf} - ECG_{ff})
 \end{aligned} \quad (24)$$

The MHD template is depicted in figure 6 with the 95% confidence interval.

2.5 ECG recordings and MHD estimation for comparison of models

As was described in subsection 2.3, a set of blood flow measurements were recorded from a healthy subject. For this subject, ECG recordings have also been realised by using a newly designed and MRI compatible 12-lead ECG monitoring device (Tse et al. 2012). Anatomical imaging of the aortic arch was performed so that one of the parameters of the geometrical model, the angle of the aortic arch, could be set.

The comparison of the MHD model to real recordings was performed first on a single cardiac phase. Three different models, the one described in subsection 2.2 (*theoretical solution*), the one created with the technique described in subsection 2.3 (which is the *proposed model*) and the model computed by equation (1) (*INRIA's model*), were compared with the MHD estimated from the recording (*extracted MHD*).

Quantitative comparisons were also conducted by measuring successively the Pearson cross-correlation factor and the coefficient of determination between the *extracted MHD* and the different modellings on each lead and finally the complexity of the different models. This can be measured by the number of eigenvectors which are required to account for a certain percentage of the energy. The more eigenvectors are required, the more complex is the model.

3. Results

Figure 7 illustrates the derived templates of the 12 standard leads from each of the three implemented MHD models. The primary and obvious comparisons of the different models to be made are the qualitative similarities and differences. First, all models have the same approximate amplitudes, showing the amplitude correction factor for the *proposed model* provides a good approximation and is consistent with the literature. This correction factor, α_{norm} , is approximatively equal to 5, which is within the range described by Abi Abdallah (2007). Moreover, the approximate structure of the resultant effect for the *proposed model* appears to be consistent with the observed or *extracted MHD* effect. In contrast to this, the *theoretical solution* seems to be far too smooth, failing to generate high frequencies present in the *extracted MHD*.

The *proposed model* and *INRIA's model* both appear to be in good agreement with the *extracted MHD*. This is especially true for leads AVL, AVR, AVF, I, II and III. However, these models seem less in agreement with the *extracted MHD* in the precordial leads.

The polarity of the *proposed model* is also in good agreement with the *extracted MHD* effect. One can notice that for most leads, the first wave of the *extracted MHD*, with its peak around 0.15s is well simulated with the *proposed model*. However, we note that the

simulated peaks of both the *proposed model* and *INRIA's model* are slightly smoother than the extracted ones.

Notably, the *extracted MHD* also shows a peak in most leads at around 0.6s. These peaks are not present in the three models under consideration, since those are only based on the blood flow in the aortic arch and this peak is not present in the flow measurements across the aortic arch, indicating that this later peak may be due to flow in other vessels.

Cross-correlation factors between the *extracted MHD* and the different modellings are depicted in table 3. It can be shown that the polarity error on the precordial leads between the *extracted MHD* and both the *INRIA's model* and *proposed model* is characterised by negative cross-correlation factors.

It is possible to compare the *INRIA's model* and *proposed model* on the limb and augmented limb leads by computing the mean of the cross-correlation factors, whose value is 0.51 and 0.43 respectively. This shows that the linear relation is slightly better between the *extracted MHD* and the *INRIA's model* than with the *proposed model*.

The mean over all the leads of the coefficients of determination, R^2 , between the *extracted MHD* and the *theoretical solution*, the *INRIA's model* and the *proposed model* is 0.16, 0.36 and 0.31. This result highlights that the *INRIA's model* and the *proposed model* have approximatively the same level of agreement with the *extracted MHD* and both outperforms the *theoretical solution*.

By performing an SVD of the resultant signals for each model, we can tabulate the cumulative energy distributions as a function of number of eigenvectors used (see table 5). The results show that five components are needed to account for 99% of the energy of the *extracted model*, whereas only one component is needed for both *INRIA's model* and the *theoretical solution*, and two components for the *proposed model*. To represent 100% of the energy, nine eigenvectors are needed for the *extracted model*, only one eigenvector for *INRIA's model*, two for the *theoretical solution* and four for the *proposed model*.

4. Discussion

The smoothness of the *theoretical solution* can be understood by the fact that the rate of flow is a filtered form of the pressure gradient. The filter has a low pass effect, characterised by an impulse response $f(t)$ (See Appendix A). The fact that $f(t)$ corresponds to such a low pass filter comes from the numerous assumptions made when solving the Navier-Stokes equation, especially the laminarity and unidirectionality of the flow. As shown by Markl et al. (2005), the blood flow in the aortic arch presents vortices and is twisted, especially in the ascending aorta, which is a result of the manner in which the heart twists as it contracts. These conclusions have been made possible by 4D blood flow measurements in MRI, and therefore the use of these measurements should allow for a better simulation of the MHD effect.

Although the *proposed model* is in good agreement for most leads, morphology was not properly simulated in the precordial leads. This could be due to the way of simulating the

propagation through the human torso. Using the ECGSim package, we assumed a linear propagation of the potentials through an homogenous torso. However, if we consider a more complicated propagation of this potentials, such as in Kyriakou et al. (2012), the potentials in the electrodes close the heart are very sensitive to slight positioning errors. Therefore, by using a standard average torso model which does not correspond to the patient's anatomy, dramatic changes in the MHD effect morphology will result.

The first peak of the *extracted MHD* is well simulated in most leads with the *proposed model*, nevertheless the *extracted MHD* contains higher frequencies and the *proposed model* seems to be a smoothed version of the *extracted MHD*. One explanation of such a phenomenon could come from the MRI based method for measuring the blood flow in the aortic arch. One limitation of this method is the relatively low temporal resolution achievable for the measurement due to the heart motion and the intrinsic properties of MRI acquisition. Moreover the blood flow is measured over several cardiac cycles (approximately twenty) and the blood flow pattern obtained is thus averaged over these cycles, which could explain the smoothing. The same conclusion can be made with the *INRIA's model* since it is also based on the blood flow measurement in the ascending aorta.

A second important peak is also present in the *extracted MHD* around 0.6s, which peak is not simulated with the proposed method. This can be explained by the fact that the proposed method only considers the contribution from the blood flow in the aortic arch. Kyriakou et al. (2012) have analysed the contributions of other arteries, such as the pulmonary arteries. These contributions have been shown not to contribute greatly, and they cannot explain the presence of this second peak, since the blood flow in these arteries occurs simultaneously with the flow in the aortic arch. Nevertheless, the MHD is the sum of the contributions from all the arteries in the human body and the 0.6s peak could represent a cumulative contribution from major arteries such as the carotid, femoral, subclavian and perhaps even brachial arteries. Adding all these contributions could allow for a better simulation of the high frequencies present in the extracted MHD effect. One contribution that has not been considered in known simulations is the blood filling of the ventricles since this flow is important and located near the electrodes and can thus have an important influence over the MHD effect. It would be interesting to simulate this effect by measuring the blood flow inside the ventricles, by locating the measurement plane just after the valves. Unfortunately these measurements were not available from our recordings.

The introduction of the *proposed model* has been interesting for several reasons. First it increases our understanding of the phenomenon, and in particular shows that the main contribution of this MHD effect comes effectively from the aortic arch. However other contributions should be considered for complete simulation of the MHD phenomenon. Even if the linear relation between the *extracted MHD* and the *INRIA's model* is slightly better than between the *extracted MHD* and the *proposed model*, it has been shown that the *proposed method* reflects more adequately the reality that the simplified equation proposed by Martin et al. (2012). Applying SVD to the *extracted MHD* clearly shows that this effect is more complex than a simple eigenvector as suggested by Martin et al. (2012). This property is important when assessing the quality of a denoising technique for ECG acquired during MRI, since the reduced complexity of the model on INRIA's model can bias the

denoising results when applying dimensionality reduction techniques such as SVD or Independent Component Analysis.

5. Conclusion

In conclusion, this study allows for the development of a realistic MHD model. The *proposed model* focuses on the aortic arch contribution, which is the most important in the situation where the magnetic field orientation is along the body axis, as in most clinical MRI. The MHD was modelled with blood flow measurements performed along the aortic arch, and the *proposed model* was shown to be in good agreement with the extracted MHD. Such agreement shows that if we are able to simulate the MHD effect from blood flow measurements in the aortic arch, then conversely we should be able to extract important clinical information like the Cardiac Output from the extracted MHD, as suggested by Tse et al. (2012).

Improvements could be added to this modelling by considering blood flow circulation in more arteries as was suggested by Kyriakou et al. (2012). It seems however that given the localisation in time of an important peak in the MHD, the blood circulation in the heart ventricles should be playing an important role in the creation of the MHD effect.

Lastly, although we have identified several deficiencies in current MHD modelling techniques, the new model presented here captures much of the MHD effect superimposing the ECG during MRI and should provide a useful tool for preliminary assessment of ECG denoising and MHD suppression techniques (Oster et al. 2012).

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Appendix A. Analytical solution of the Navier Stokes equation

Let (r, θ, l) be the cylindrical coordinates in the $(O, \mathbf{i}, \mathbf{j}, \mathbf{k})$ reference frame. Let us also assume that the flow is laminar, axisymmetric and unidirectional. The fluid velocity can then be written as $\mathbf{u} = u_l(r, l, t)\mathbf{k}$. As the blood can be considered as an incompressible fluid, $\nabla \cdot \mathbf{u} = 0$ and as the flow is considered unidirectional $u_\theta = u_r = 0$. One can then conclude that since $\nabla \cdot \mathbf{u} = \frac{1}{r} \frac{\partial}{\partial r}(ru_r) + \frac{1}{r} \frac{\partial u_\theta}{\partial \theta} + \frac{\partial u_l}{\partial l} = 0$ then $\frac{\partial u_l}{\partial l} = 0$, and thus $\mathbf{u} = u_l(r, t)\mathbf{k}$. The vector

Laplacian of the fluid velocity is also given by $\nabla^2 \mathbf{u} = \left(\frac{\partial^2 u_l}{\partial r^2} + \frac{1}{r} \frac{\partial u_l}{\partial r} \right) \mathbf{k}$.

Taking into account that:

$$\begin{aligned}
\mathbf{u} \wedge \mathbf{B} &= u_l(r, t) \mathbf{k} \wedge B_0 \mathbf{z} \\
&= u_l(r, t) B_0 (\sin \phi_V \cos \theta_V \mathbf{x} + \sin \phi_V \sin \theta_V \mathbf{y} + \cos \phi_V \mathbf{z}) \wedge \mathbf{z} \quad (\text{A1}) \\
&= u_l(r, t) B_0 (\sin \phi \sin \theta_V \mathbf{x} - \sin \phi \cos \theta_V \mathbf{y}),
\end{aligned}$$

one have

$$\begin{aligned}
(\mathbf{u} \wedge \mathbf{B}) \wedge \mathbf{B} &= -u_l(r, t) B_0^2 (\sin \phi \cos \theta_V \mathbf{x} + \sin \phi \sin \theta_V \mathbf{y}) \\
&= -u_l(r, t) B_0^2 \sin \phi (\cos \theta_V \mathbf{x} + \sin \theta_V \mathbf{y}) \quad (\text{A2}) \\
&= -u_l(r, t) B_0^2 \sin \phi (\sin \phi \mathbf{k} + \cos \phi \mathbf{j}).
\end{aligned}$$

By projection onto the vector $\mathbf{k}$, equation (7) can then be rewritten as:

$$\rho \frac{\partial u_l}{\partial t} = -\frac{\partial p}{\partial l} + \eta \nabla^2 \mathbf{u} - \sigma B_0^2 \sin^2 \phi u_l. \quad (\text{A3})$$

Multiplying equation (A3) by $\frac{a^2}{\eta}$ and defining $y = \frac{r}{a}$, where a is the radius of the vessel, we have

$$\frac{a^2}{\nu} \frac{\partial u_l(y, t)}{\partial t} = -\frac{a^2}{\eta} \frac{\partial p}{\partial l} + \nabla^2 \mathbf{u} - \sigma \frac{a^2}{\eta} B_0^2 \sin^2 \phi u_l(y, t). \quad (\text{A4})$$

Defining $g(t) = -\frac{a^2}{\eta} \frac{\partial p}{\partial l}$ and $\sigma \frac{a^2}{\eta} B_0^2 \sin^2 \phi = H_a^2$ (the squared Hartmann number), equation (??) becomes

$$\frac{a^2}{\nu} \frac{\partial u_l(y, t)}{\partial t} = g(t) + \nabla^2 \mathbf{u} - H_a^2 u_l(y, t). \quad (\text{A5})$$

To solve this equation, we can employ a method based on Fourier and Hankel transforms. Applying the following Fourier transforms:

$$u_l(y, t) = \frac{1}{2\pi} \int_{-\infty}^{\infty} U_l^y(\omega) e^{j\omega t} d\omega, \quad (\text{A6})$$

$$g(t) = \frac{1}{2\pi} \int_{-\infty}^{\infty} G(\omega) e^{j\omega t} d\omega, \quad (\text{A7})$$

equation (??) becomes:

$$\frac{a^2}{\nu} j\omega U_l^y(\omega) = G(\omega) + \nabla^2 U_l^y(\omega) - H_a^2 U_l^y(\omega), \quad (\text{A8})$$

where,

$$\nabla^2 U_l^y(\omega) = \frac{\partial^2 U_l^y(\omega)}{\partial y^2} + \frac{1}{y} \frac{\partial U_l^y(\omega)}{\partial y}. \quad (\text{A9})$$

The Hankel transform is defined as:

$$H\{f(r)\} = F^*(n) = \int_0^1 r f(r) J_0(r\lambda_n) dr, \quad (\text{A10})$$

and

$$f(r) = 2 \sum_{n=1}^{\infty} \frac{J_0(\lambda_n r)}{J_1(\lambda_n)^2} F^*(n), \quad (\text{A11})$$

and where J_n is the n^{th} order Bessel functions, λ_n are the roots of the J_0 . The Hankel transform has the following properties:

$$H\{\nabla^2 f(r)\} = -\lambda_n^2 F^*(n) \text{ (if } f(1)=0), \quad (\text{A12})$$

$$H\{c\} = \frac{c}{\lambda_n} J_1(\lambda_n), \quad (\text{A13})$$

with c being a constant.

Applying Hankel transform to equation (A8) yields

$$\frac{a^2}{\nu} j\omega U_{l\omega}^*(n) = \frac{G(\omega)}{\lambda_n} J_1(\lambda_n) - \lambda_n^2 U_{l\omega}^*(n) - H_a^2 U_{l\omega}^*(n). \quad (\text{A14})$$

The value of $U_{l\omega}^*(n)$ can thus be computed:

$$U_{l\omega}^*(n) = \frac{J_1(\lambda_n)}{\lambda_n \left(\frac{a^2}{\nu} j\omega + \lambda_n^2 + H_a^2 \right)} G(\omega). \quad (\text{A15})$$

Applying the inverse Hankel transform then gives

$$U_l^y(\omega) = 2 \sum_{n=1}^{\infty} \frac{J_0(\lambda_n r)}{J_1(\lambda_n)^2} \frac{J_1(\lambda_n)}{\lambda_n \left(\frac{a^2}{\nu} j\omega + \lambda_n^2 + H_a^2 \right)} G(\omega). \quad (\text{A16})$$

Finally, we obtain the velocity profile by applying the inverse Fourier transform to $U_l^y(\omega)$ such that

$$u_l(y, t) = \frac{1}{2\pi} \int_{-\infty}^{\infty} 2 \sum_{n=1}^{\infty} \frac{J_0(\lambda_n y)}{J_1(\lambda_n)} \frac{G(\omega)}{\lambda_n \left(\frac{a^2}{\nu} j\omega + \lambda_n^2 + H_a^2 \right)} e^{j\omega t} d\omega, \quad (A17)$$

and

$$u_l(y, t) = \frac{1}{\pi} \sum_{n=1}^{\infty} \frac{J_0(\lambda_n y)}{J_1(\lambda_n) \lambda_n} \int_{-\infty}^{\infty} \frac{G(\omega)}{\left(\frac{a^2}{\nu} j\omega + \lambda_n^2 + H_a^2 \right)} e^{j\omega t} d\omega. \quad (A18)$$

The integral

$$\int_{-\infty}^{\infty} \frac{G(\omega)}{\left(\frac{a^2}{\nu} j\omega + \lambda_n^2 + H_a^2 \right)} e^{j\omega t} d\omega \quad (A19)$$

is equivalent to the inverse Fourier transform of

$$FT^{-1} \left\{ G(\omega) \frac{1}{\left(\frac{a^2}{\nu} j\omega + \lambda_n^2 + H_a^2 \right)} \right\}, \quad (A20)$$

which in the time domain can be written

$$g(t) * FT^{-1} \left\{ \frac{1}{\left(\frac{a^2}{\nu} j\omega + \lambda_n^2 + H_a^2 \right)} \right\}, \quad (A21)$$

where $*$ denotes the convolution operator.

To solve the inverse transform in equation (A21), we apply the following Fourier transform pair:

$$h(t) e^{-\alpha t} \rightarrow \frac{1}{\alpha + j\omega}, \quad (A22)$$

where h is the Heaviside step function, the integral of the Dirac delta function,

$$h(t) = \int_{-\infty}^t \delta(x) dx.$$

We can therefore express the inverse Fourier Transform of equation (A21) as:

$$FT^{-1} \left\{ \frac{1}{\frac{a^2}{\nu} j\omega + \lambda_n^2 + H_a^2} \right\} = FT^{-1} \left\{ \frac{\nu}{a^2} \cdot \frac{1}{j\omega + \frac{\nu}{a^2} (\lambda_n^2 + H_a^2)} \right\} \quad (A23)$$

$$= \frac{\nu}{a^2} e^{-\frac{\nu}{a^2} (\lambda_n^2 + H_a^2) t} h(t).$$

Finally, we obtain the velocity profile as

$$u_l(y, t) = g(t) * \frac{1}{\pi} \sum_{n=1}^{\infty} \frac{J_0(\lambda_n y)}{J_1(\lambda_n) \lambda_n} \frac{\nu}{a^2} e^{-\frac{\nu}{a^2} (\lambda_n^2 + H_a^2) t} h(t). \quad (\text{A24})$$

Given this velocity profile, the rate of flow, q , corresponds to:

$$q(t) = 2\pi a^2 \int_0^1 u_l(y, t) y dy. \quad (\text{A25})$$

which is

$$q(t) = g * 2 \sum_{n=1}^{\infty} \left(\frac{1}{J_1(\lambda_n) \lambda_n} \nu e^{-\frac{\nu}{a^2} (\lambda_n^2 + H_a^2) t} h(t) \times \int_0^1 J_0(\lambda_n y) y dy \right). \quad (\text{A26})$$

Taking into account that $\int x^n J_{n-1}(x) dx = x^n J_n(x)$, the flow rate becomes

$$q(t) = (g * f)(t), \quad (\text{A27})$$

where $g(t) = -\frac{a^2}{\eta} \frac{\partial p}{\partial t} \text{ m/s}$, and $f(t) = 2 \sum_{n=1}^{\infty} \frac{1}{\lambda_n^2} \nu e^{-\frac{\nu}{a^2} (\lambda_n^2 + H_a^2) t} h(t)$.

Finally, the electrical potential induced on the vessel walls is given by

$$V(t) = \frac{2B_0 \sin \phi}{\pi a} q(t). \quad (\text{A28})$$

Figure 1.

Multiple View of the Geometrical Models. a) Front view, b) lateral view (right side), c) top view, d) lateral view (left side)

Figure 2.

Illustration of the MagnetoHydrodynamic model parametrisation in the aortic arch with a blood flow at velocity $\mathbf{u}$ in direction $\mathbf{k}$. The magnetic field is along the $\mathbf{z}$ direction, and the vessel is along the $\mathbf{k}$ direction.

Figure 3.

Electrical circuit analogy of the windkessel lumped parametric model, where $P_v(t)$ is the left ventricular pressure, $P_a(t)$ the aortic pressure, $Q(t)$ the output flow rate of the left ventricle, $Q_1(t)$ the input flow rate of the peripheral vessel, R_a the resistance of the aorta, C compliance of the aorta and R_p the resistance of the peripheral arteries.

Figure 4. Ventricular and aortic pressures computed by using the three-element Windkessel model (left). Flow rate in the aorta (right).

Figure 5.

Left: MRI image of region around heart with four hand-selected planes for extracting flow measurements. Colours indicate planes corresponding to ascending aorta (solid cyan), ascending aortic arch (dotted blue), descending aortic arch (dashed green) and descending aortic arch (dot dashed red). Right: Corresponding flow rate measurements for each of the four chosen planes with same colour coding recorded over one cardiac cycle (from QRS upslope trigger point).

Figure 6.

Template of the extracted MHD effect (solid black line) and the 95% confidence interval (dotted blue line).

Figure 7.

Template of the simulations and estimations of the MHD effects on the 12 standard leads. (solid line blue: *extracted MHD*, dashed line green: *theoretical solution*, dot dashed line red: *proposed model* and dotted line black: *INRIA's model*).

Table 1

Proportionality coefficients in equation (1) (in m^{-1}).

$I(V_1^{ECG})$	$aVR(V_4^{ECG})$	$V_1(V_7^{ECG})$	$V_4(V_{10}^{ECG})$
1.0	-0.67	-0.43	-0.67
$II(V_2^{ECG})$	$aVL(V_5^{ECG})$	$V_2(V_8^{ECG})$	$V_5(V_{11}^{ECG})$
0.35	0.82	-0.14	0.05
$III(V_3^{ECG})$	$aVF(V_6^{ECG})$	$V_3(V_9^{ECG})$	$V_6(V_{12}^{ECG})$
-0.65	-0.15	-0.075	0.12

Table 2
Correspondance between node number and standard ECG electrode potential locations.

Electrode Label	RA	LA	LL	V1	V2	V3	V4	V5	V6
Torso node number	1	2	56	19	26	isocenter(33,34)	41	48	54

Table 3

Pearson cross-correlation factor between the extracted MHD and the different modellings.

Lead	V1	V2	V3	V4	V5	V6	AvL	AvR	AvF	I	II	III
<i>proposed model</i>	0.085	0.515*	-0.095	-0.755*	-0.818*	-0.765*	0.636*	0.538*	-0.099	0.590*	0.215*	0.675*
<i>theoretical solution</i>	0.541*	0.228*	0.239*	-0.216*	-0.313*	-0.309*	0.098	-0.145*	-0.507*	0.230*	0.440*	0.206*
<i>INRIA's model</i>	-0.773*	-0.666*	-0.047	0.654*	-0.753*	-0.728*	0.623*	0.479*	0.606*	0.552*	0.108	0.661*

An asterix indicates statistical significant values (p values < 0.005).

Table 4

Coefficient of determination (R^2) between the *extracted MHD* and the different modellings.

Lead	V1	V2	V3	V4	V5	V6	AvL	AvR	AvF	I	II	III
<i>proposed model</i>	0.0072	0.265	0.010	0.570	0.670	0.586	0.404	0.290	0.010	0.348	0.046	0.456
<i>theoretical solution</i>	0.293	0.052	0.057	0.047	0.098	0.152	0.010	0.021	0.257	0.053	0.193	0.042
<i>INRIA's model</i>	0.597	0.444	0.002	0.428	0.566	0.530	0.388	0.230	0.367	0.305	0.012	0.437

Table 5

Fraction of energy in the signal as a function of number of eigenvectors used.

number of eigenvectors	Cumulative normalised energy <i>theoretical solution</i>	<i>INRIA's model</i>	<i>proposed model</i>	<i>extracted MHD</i>
1	0.9997	1	0.8915	0.7066
2	1	1	0.9992	0.8888
3	1	1	0.9998	0.9438
4	1	1	1	0.9860
5	1	1	1	0.9968
6	1	1	1	0.9990
7	1	1	1	0.9996
8	1	1	1	0.9998
9	1	1	1	1
10	1	1	1	1
11	1	1	1	1
12	1	1	1	1