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WITH THE CARDIOVASCULAR SYSTEM

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INTERACTION OF STATIONARY MAGNETIC FIELDS WITH THE CARDIOVASCULAR SYSTEM

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Introduction

The electrodynamic interaction of a stationary magnetic field with blood flow leads to induced electrical potentials within the circulatory system, and to a resultant magnetohydrodynamic effect on the velocity of blood flow. The magnetically-induced flow potentials and the alteration in hemodynamic parameters that occurs in an applied magnetic field are well understood theoretically, and the characteristics of induced flow potentials have been systematically investigated in experimental animal studies. Both the theoretical background and experimental observations of magnetic field interactions with the circulatory system are presented in this chapter. The influence of these interactions on cardiac performance is also considered from the perspective of evaluating potential cardiovascular stress effects.

Magnetically-induced flow potentials

A. Theoretical aspects. Because the blood circulation involves the flow of an electrically conductive fluid, the application of a homogeneous, stationary magnetic field will induce an electrical potential, ψ, along an axis that is orthogonal to both the flow velocity, $\vec{v}$, and the magnetic induction, $\vec{B}$. The magnitude of ψ can be predicted from the Lorentz force law governing the electrodynamic interaction between the magnetic field and the flowing electrolytes in the blood. Under steady state conditions the net macroscopic force on any volume element of the flowing conductive fluid must be equal to zero, and the Lorentz force law predicts that the magnitude of the induced electric field, $\vec{E}$, is

$$|\vec{E}| = |\vec{v} \times \vec{B}| = |\vec{v}|\,|\vec{B}| \sin\theta \qquad (1)$$

where θ is the angle between $\vec{v}$ and $\vec{B}$. If the fluid flow occurs between two

parallel walls separated by a distance, d, that is small compared to the dimensions of the walls, then $|\vec{E}| = \psi/d$ and the magnetically-induced flow potential predicted from eqn. (1) is

$$\psi = |\vec{v}||\vec{B}|d \sin\theta \tag{2}$$

For the specific case of blood flow within a cylindrical vessel of diameter d, it has been shown by Kolin (1945) that eqn. (2) is rigorously valid.

From eqn. (2) it can be predicted that the magnitude of the induced potential is a function of the relative directions of the blood flow and the applied field, and that $\psi = 0$ when $\vec{v}$ and $\vec{B}$ are parallel. It can be further predicted that the sign of ψ will reverse when the orientation of $\vec{v}$ and $\vec{B}$ is reversed, i.e., when one vector is rotated by π radians relative to the other. Both of these predictions are amenable to experimental tests, as described below.

It is also of interest to calculate the magnitude of the blood flow potentials that would be predicted from eqn. (2) to occur in the ascending aortic vessels of various species of mammals with widely differing sizes. The results of this calculation are shown in Table 1 for a rat, monkey and man exposed to a 1.0 Tesla field with an orientation that is perpendicular to the direction of blood flow in the ascending aortic vessel. It is evident that the predicted magnitude of the induced flow potential is significantly greater in the primate species than in the rodent, and experimental data presented in a later section of this chapter are consistent with this theoretical prediction.

B. Experimental observations. The occurrence of magnetically-induced potentials associated with pulsatile blood flow into the aortic vessel have been demonstrated from electrocardiogram (ECG) measurements on rats (Gaffey and Tenforde, 1981), rabbits (Togawa et al., 1967), dogs (Gaffey and Tenforde,

1979), baboons (Gaffey et al., 1980), and monkeys (Beischer and Knepton, 1964; Beischer, 1969; Tenforde et al., 1983) exposed to stationary magnetic fields. The primary change in the ECG recorded in the field is an alteration of the signal amplitude at the locus of the T-wave. It was first recognized by Togawa et al. (1967) that this apparent change in the T-wave reflects a superposition of the magnetically-induced aortic blood flow potential onto the normal T-wave signal. Because the repolarization of ventricular heart muscle, which gives rise to the T-wave signal in the normal ECG, occurs at approximately the same time in the cardiac cycle as the pulsatile ejection of blood into the aortic vessel, it is reasonable to expect that the magnetically-induced flow potential and the T-wave should be superimposed. This superposition of potentials at the T-wave locus in the ECG is illustrated in Fig. 1.

Four additional sets of experimental observations lend support to the concept that the T-wave alteration is the result of an electrical potential induced by the electrodynamic interaction between an applied magnetic field and aortic blood flow:

(1) From the theoretical considerations discussed above, it is predicted that the magnitude and the sign of the induced flow potential should be a function of the angle between the aortic blood flow and the direction of the applied field. Consistent with this prediction, it was shown by Togawa et al. (1967) for rabbits and by Gaffey and Tenforde (1981) for rats that the amplitude of the T-wave signal can be increased, decreased, or unchanged by the superimposed flow potential depending upon the orientation of the animal within the applied magnetic field. It was also demonstrated by Gaffey and Tenforde (1981) that the maximum change in the T-wave amplitude occurs when the long axis of a rat, and hence its ascending aortic vessel, is oriented perpendicular to the field. The observation is completely consistent with the theoretical

prediction from eqn. (2) that the magnitude of the magnetically-induced aortic blood flow potential should achieve its maximum value when the flow velocity vector and the magnetic field vector are orthogonal.

(2) Because an induced flow potential is the manifestation of a physical interaction between an applied magnetic field and the charged electrolyte species in a flowing fluid, it would be expected that the induced aortic blood flow potential, and hence the alteration in the T-wave signal strength, should be immediately and completely reversible upon termination of the field exposure. Consistent with this expectation, ECG measurements on rats (Gaffey and Tenforde, 1981), dogs (Gaffey and Tenforde, 1979), baboons (Gaffey et al., 1980), and monkeys (Beischer, 1969; Tenforde et al., 1983) have demonstrated the complete reversibility of the magnetically-induced change in the T-wave amplitude.

(3) From eqn. (2) it is predicted that an induced flow potential should have a linear dependence on magnetic field strength. Experimental tests of this prediction have been carried out by recording the ECG of rats (Gaffey and Tenforde, 1981), dogs (Gaffey and Tenforde, 1979), baboons (Gaffey et al., 1980), and monkeys (Tenforde et al., 1983) during exposure to graded field intensities. In these studies, the orientation of the experimental subject was chosen to produce an augmentation of the T-wave amplitude in an applied magnetic field. From the ECG records of rats exposed to stationary fields ranging from 0.1 to 2.0 Tesla (Gaffey and Tenforde, 1981), a field-strength-dependent increase in T-wave amplitude was observed at field levels greater than 0.3 Tesla (see Fig. 2). The T-wave signal increase was a linear function of the applied field strength up to 1.4 Tesla. For dogs (Gaffey and Tenforde, 1979), baboons (Gaffey et al., 1980), and monkeys (Tenforde et al., 1983), the threshold for detection of the T-wave amplitude change was 0.1 Tesla, and the

increase in signal strength was a linear function of the magnetic field strength up to 1.0 Tesla. These data support the concept that the T-wave alteration is a consequence of the superposition of an induced aortic blood flow potential, which from eqn. (2) should have a strictly linear dependence on the magnetic field strength.

As shown in Fig. 2, the increase in T-wave amplitude observed in the rat ECG exhibits a steeper slope at field levels above 1.4 Tesla. A similar change in slope has been observed with dogs (Gaffey and Tenforde, 1979) and monkeys (Tenforde et al., 1983) at field levels exceeding 1.0 Tesla. Gaffey and Tenforde (1981) have proposed that this effect may result from the superposition of one or more additional blood flow potentials that have thresholds for detection at high field levels. They have suggested that magnetically-induced potentials associated with pulsatile blood flows into the pulmonary, carotid and subclavian arteries could appear at the T-wave locus in the ECG record. Because of the smaller diameters of these vessels, the associated blood flow potentials would be expected to be significantly smaller than the aortic flow potential. These magnetically-induced flow potentials may therefore be detectable in the external ECG only at field strengths exceeding 1.0 Tesla in the rodents and small primates that have been studied to date.

(4) The theoretical calculations presented in Table 1 suggest that the magnitude of induced aortic blood flow potentials should be significantly greater for large animal species in comparison with the rodent. From ECG measurements on animals exposed to a 1.0 Tesla field with an orientation perpendicular to the body axis, the maximum aortic flow potentials recorded at the body surface were 75 μV for rats (Gaffey and Tenforde, 1981) and 200 μV for monkeys (Tenforde et al., 1981). The greater magnetically-induced blood flow potential observed with the larger species of animal thus conforms to

theoretical expectations. It should be noted that the aortic blood flow potentials measured in the external ECG records of rats and monkeys were, respectively, 5 and 20 times less than the values predicted to occur within the ascending aortic vessels of these animals (see Table 1). However, a significant reduction in the magnitude of the induced blood flow potential between its locus in the ascending aorta and the body surface would be expected to occur because of the high electrical resistance of the conductive pathway joining these locations.

Magnetohydrodynamic interactions with blood flow

A. Theoretical aspects. The electrodynamic interaction of stationary magnetic fields with flowing, electrically conductive fluids has a direct effect on the fluid velocity and pressure. For the case of laminar blood flow in a stationary field, the magnetohydrodynamic interaction leads to a "flattening" of the parabolic velocity profile within the blood vessel, and a resultant reduction in the mean axial flow velocity. The physical mechanism underlying this effect can be explained in the following qualitative terms. As discussed above, the interaction of a stationary magnetic field and a moving, electrically-conductive fluid produces induced electrical potentials and currents within the solution. By Lenz's law, an electrical volume force is established within the fluid that opposes the induced currents. The hydrodynamic consequences of this electrical volume force is a reduction in the rate of movement of the fluid.

The mathematical foundation for quantitatively describing this magnetohydrodynamic phenomenon was originally developed by Hartmann (1937). These theoretical concepts were first applied by Belousova (1965) and by Korchevskii and Marochnik (1965) to predict the extent to which blood flow

would be retarded by stationary magnetic fields with intensities in the range to which humans are exposed. Belousova (1965) calculated that a 2 Tesla field would reduce the flow velocity of blood in a major vessel of 1 cm radius by 9%, and a qualitatively similar conclusion was drawn by Korchevskii and Marochnik (1965). It should be noted, however, that both sets of authors used values of electrical conductivity and kinematic viscosity for serum rather than for whole blood. As shown by Tenforde et al. (1983), the use of appropriate physical parameters for whole blood leads to the prediction of a 5% decrease in blood flow velocity within a 2 Tesla field, as contrasted to the 9% decrease calculated by Belousova (1965).

The decrease in flow velocity resulting from a magnetohydrodynamic interaction must be compensated by an elevation in pressure if the volume of circulating blood per unit time is to remain constant, i.e. if a homeostatic condition is to be maintained. A calculation of the predicted increase in blood pressure resulting from exposure to a homogeneous, stationary magnetic field was first made by Vardanyan (1973), and subsequently modified by Abashin and Yuvtushenko (1974). Vardanyan's theory considers the magnetohydrodynamic perturbation of laminar blood flow within a rigid cylindrical vessel. The assumption of laminar flow is reasonable on the basis of calculations by Tenforde et al. (1983). However, the pulsatile nature of blood flow is neglected in Vardanyan's theory, and the assumption that the walls of blood vessels are rigid is also incorrect. The effects of the unsteady nature of blood flow and the elasticity of blood vessel walls have been taken into account in the theoretical treatments of magnetohydrodynamic interactions given by Sud et al. (1974, 1978) and Kumar (1978). Although these theoretical calculations are more refined than the earlier theory of Vardanyan (1973), they lead to qualitatively similar conclusions when the magnetohydrodynamic

interaction with blood flow is weak. As will be shown below, this situation pertains at the magnetic field levels to which humans are generally subjected.

For the case of blood flow in a cylindrical vessel oriented with its longitudinal axis perpendicular to a homogeneous, stationary magnetic field, Vardanyan's theory predicts that the instantaneous blood pressure, P, at any point in the vessel must be modified in accord with the following equation if the volume flow rate of blood is to remain constant:

$$P(B) = P(B=0) \left[\frac{H_a^{\,2}\, I_0(H_a)}{8 I_2(H_a)} \right] \tag{3}$$

where I_0 and I_2 are modified Bessel functions of the zero and second order, respectively, and H_a is the Hartmann number [9]:

$$H_a = (Bd/2)(\sigma/\eta)^{\frac{1}{2}} \tag{4}$$

In eqn. (4), d = vessel diameter, σ = electrical conductivity and η = kinematic viscosity. For values of H_a significantly less than unity, eqn. (3) can be expanded as a power series. Including terms up to second order in H_a, eqn. (3) becomes:

$$P(B) = P(B=0)[1+(H_a^{\,2}/4)] \tag{5}$$

Two aspects of the blood pressure modification predicted by eqn. (5) are particularly noteworthy. First, the magnetohydrodynamic effect varies approximately as $H_a^{\,2}$, and hence as d^2. Accordingly, the greatest effect will occur in the large vessels of the central circulatory system. Secondly, the pressure change predicted from eqn. (5) varies approximately as B^2, and hence will rapidly increase as the field intensity is elevated.

It is of interest to calculate from eqn. (5) the pressure change occurring in a vessel of 1 cm radius, similar to the human aorta, within a 2 Tesla

stationary field. Using values for the electrical conductivity and the kinematic viscosity of human whole blood (Tenforde et al., 1983) of 0.52 S/m and 4.6×10^{-3} kg/m•s, respectively, then $H_a = 0.212$ and P(B = 2 Tesla) = 1.011 P(B = 0). The pressure alteration is therefore about 1%. It can also be shown from eqn. (5) that a magnetic field intensity of 6 Tesla would be required in order for a 10% change in pressure to occur.

B. Experimental observations. The only direct experimental test of potential alterations in blood pressure resulting from magnetohydrodynamic interactions was made by Tenforde et al. (1983), who exposed three Macaca monkeys to homogeneous, stationary fields ranging in strength from 0.1 to 1.5 Tesla. Direct intraarterial measurements of blood pressure were made during the field exposure using a cannula that was inserted 15 cm into the femoral artery and interfaced with a linear pressure-voltage transducer exterior to the body. Within the ± 2 mm Hg accuracy with which the systolic and diastolic blood pressures could be determined by this technique, no measurable alteration in blood pressure was recorded in fields up to 1.5 Tesla. This observation was fully consistent with a theoretical calculation that the maximum pressure elevation within the monkey's ascending aortic vessel should be 0.6 mm Hg in a 1.5 Tesla stationary field.

Cardiac performance in stationary magnetic fields

Available data on the response of the circulatory system to stationary magnetic fields are sufficient to permit an assessment of potential stress effects resulting from electrodynamic and magnetohydrodynamic interactions with blood flow. The indices of cardiac performance that have been studied include blood pressure, heart rate and the bioelectric activity of heart muscle. As described above, there is no measurable alteration in the blood pressure of

monkeys exposed to a 1.5 Tesla stationary field. The heart rate and electrical properties of heart muscle have been determined from ECG measurements on rats exposed to stationary fields up to 2.0 Tesla (Gaffey and Tenforde, 1981), rabbits in a 1.0 Tesla field (Togawa et al., 1967), dogs (Gaffey and Tenforde, 1979) and baboons (Gaffey et al., 1980) in fields up to 1.5 Tesla, and monkeys exposed to fields up to 1.5 Tesla by Tenforde et al. (1983) and to a 10.0 Tesla field by Beischer (1969). In none of these studies were significant changes in heart rate observed during acute magnetic field exposures. Similarly, the amplitudes of the P, Q, R and S waves of the ECG were not altered, indicating that the applied magnetic field had no effect on the depolarization characteristics of auricular and ventricular heart muscle. The data from these studies with various species of animals also indicate that no cardiac arrhythmias occur during acute exposures to the field levels indicated above.

The set of experimental observations summarized here therefore provide evidence that little or no cardiovascular stress should result from exposure to the highest stationary magnetic field levels ($\lesssim$ 2 Tesla) routinely encountered by man. This conclusion must be tempered, however, by the recognition that there are no data available in the literature relating to cardiovascular performance during protracted exposure to large stationary magnetic fields. Also, from the theoretical considerations discussed in this chapter, it would be anticipated that significant hemodynamic perturbations could occur during exposure to stationary fields exceeding approximately 5 Tesla. This factor should be considered in any application of superconducting magnets that might involve exposing human subjects to exceedingly high fields.

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TABLE 1. Calculated aortic blood flow potentials in a 1.0 Tesla stationary magnetic field

Species[a]	Cardiac Output[b] (cm^3/min)	Aortic Diameter[c] (cm)	Blood Flow Velocity[d] (cm/s)	Induced Flow Potential[e] (mV)
Rat	47	0.26	15	0.4
Monkey	1060	0.57	69	3.9
Man	5100	1.6	42	6.7

(a) Parameters used in the flow potential calculations are appropriate for adult mammals of the three species.

(b) Taken from Spector (1956).

(c) Values for the rat, monkey and man were taken from Gaffey and Tenforde (1981), Tenforde et al. (1983) and Togawa et al. (1967), respectively.

(d) The aortic blood flow velocity was calculated from the ratio of the cardiac output to the cross-sectional area of the ascending aorta.

(e) Magnetically-induced flow potentials were calculated from eqn. (2) of the text for a 1.0 Tesla stationary magnetic field.

Figure Legends

Fig. 1. Schematic diagram of a magnetically-induced aortic blood flow potential superimposed on the normal ECG record. A_o and A_c denote the approximate times at which the aortic valve opens and closes.

Fig. 2. The percent increase in T-wave amplitude observed in the ECG of an adult rat is plotted as a function of magnetic field strength. The percentage increase is defined as $100(T_m - T_c)/T_c$, where T_c and T_m are the T-wave amplitudes in the control state and during magnetic field exposure, respectively. [From Gaffey and Tenforde (1981)]

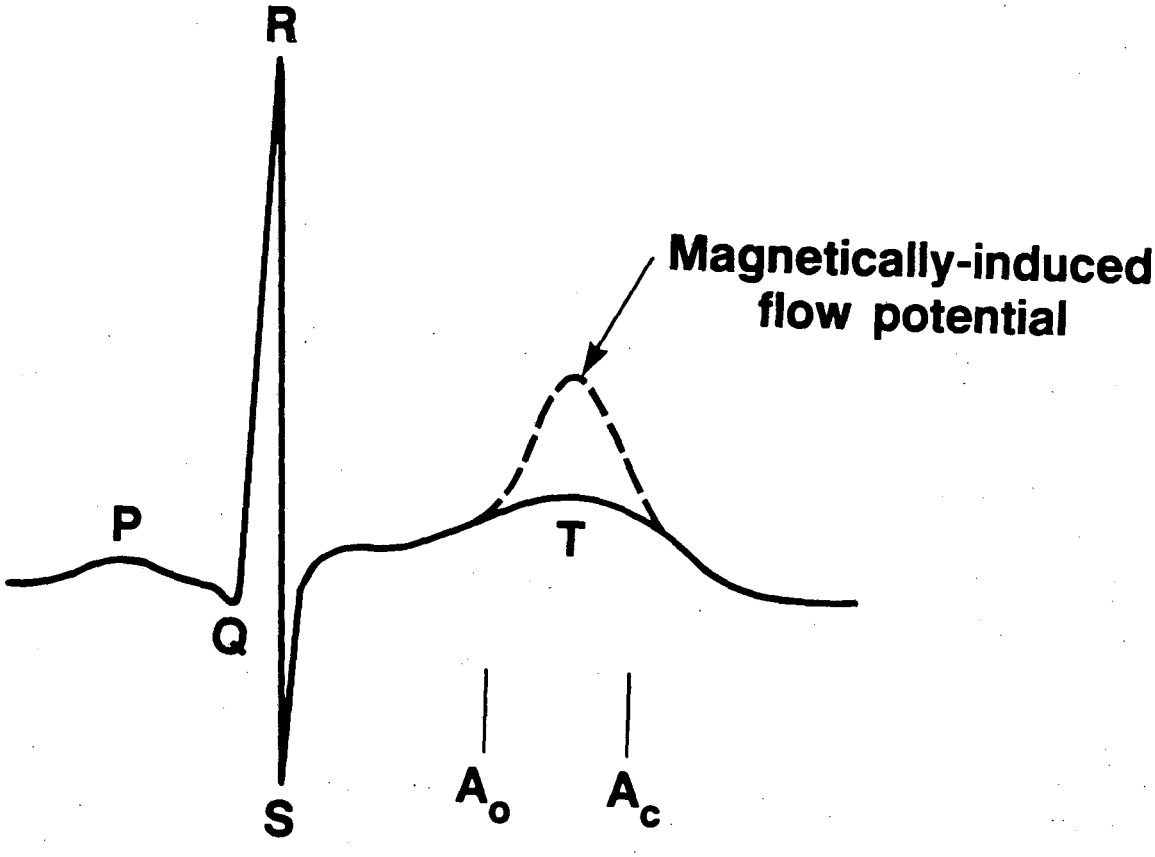

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FIGURE 1

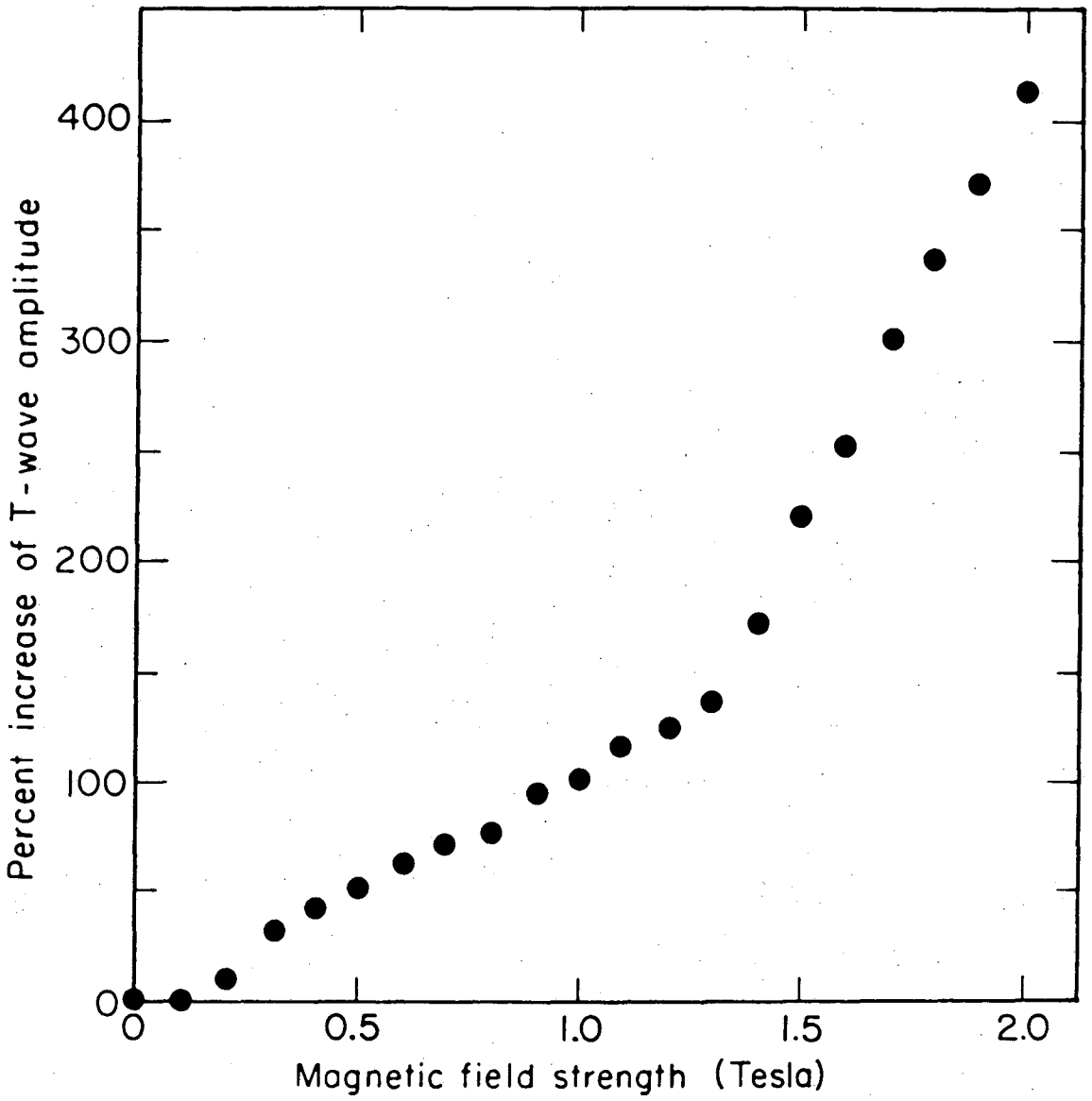

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FIGURE 2

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