Physiological measurement

Spectral analysis and acoustic transmission of mitral and aortic valve closure sounds in dogs

Part 2 Effects of neuromuscular blockade, sternotomy and pacemaker control, and a two-week recovery period

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Abstract—*The paper describes the effects of neuromuscular blockade, sternotomy and atrio-ventricular pacing, and a two-week recovery period on the spectra and acoustic transmission of mitral M1 and aortic A2 sound components in dogs. Results indicate that neuromuscular blockade does not affect the attenuation properties of the heart/thorax acoustic system even if it modifies the intensity of M1 and the phase of the transfer function. The immediate effect of sternotomy and cardiac pacing is an important increase in the attenuation of the heart/thorax acoustic system. This increased attenuation is different for both sounds (20 dB for M1 and 11 dB for A2) and disappears after a two-week recovery period. However, the resulting controlled dog model shows slightly different acoustic characteristics than those of the normal animal model.*

Keywords—*Aortic valve, Heart sounds, Heart/thorax acoustic system, Mitral valve, Neuromuscular blockade, Pacemaker control, Spectral analysis, Sternotomy*

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1 Introduction

THE EFFECTS of open heart surgery on the transmission of heart sounds and murmurs through the heart/thorax acoustic system have not been systematically documented in the past (RANGAYYAN and LEHNER, 1988). In the course of a series of dog experiments designed to measure the influence of cardiac chronotropic and inotropic variations on the spectral properties of the mitral and aortic valve closure sounds, we had to produce a chronic controlled animal model in which the heart rate and the atrio-ventricular sequential pacing were controlled by a telemetric pacemaker programmer. We have taken the opportunity to study the effect of neuromuscular blockade induced after anaesthesia, that of immediate sternotomy and cardiac pacing, and the effect of a two-week recovery period on the spectral properties of the mitral and aortic valve closure sound components as well as on their acoustic transmission through the heart/thorax system. This paper documents our findings, which may have implications in the recording and analysis of the human valvular sound components after open-heart surgery.

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2 Materials and methods

Two experimental investigations were carried out in seven mongrel dogs. The purpose of the first experiment was to produce a chronic animal model for controlling P-R interval and heart rate by using a programmable sequential atrioventricular (AV) pacemaker. The objective of the second experiment was to study the influence of changes in P-R interval, heart rate and left ventricular inotropy on the spectra of the mitral and aortic valve sound components as well as on their transmission through the heart/thorax acoustic system.

2.1 *First experiment*

Anaesthesia was induced with sodium thiamylal (7–14 mg kg^{-1}) given intravenously. The dog was then intubated and anaesthesia maintained with isofluorane

(0·73–1·37 MAC) in oxygen using an anaesthetic breathing circle system. Each anaesthetised dog (18–35 kg) was put in a supine position, immobilised, and the neck and thorax shaved. The animals were then instrumented according to the protocol described in Part 1 of this study (DURAND *et al.*, 1990). After a period of 5 min allowed to each dog for stabilisation, two samples of eight cardiac signals were recorded on an eight-channel FM tape recorder to represent the basal state of the normal dog. The first sample, taken when the pressure tracings of the Millar catheter showed that the distal micromanometer was in the left ventricle and the proximal one into the aorta, was composed of the following signals: the ECG, the two thoracic PCGs, the left ventricular and aortic PCGs, the left ventricular and aortic pressures, and the left ventricular dP/dt. The catheter was then pulled back by 3 cm, thus providing a recording of the intra-aortic signals without interference of the catheter with the aortic valve. The second sample was thus composed of the following signals: the ECG, two thoracic PCGs, two aortic PCGs, two aortic pressures, and the aortic dP/dt. Each recording had a duration of 2 min and was started only when all physiological signals had reached steady-state values.

After the initial signal recordings, neuromuscular blockade was induced with atracurium (200 $\mu g\,kg^{-1}$, IV) while ventilation was controlled by using a mechanical volume-cycled ventilator. A peripheral nerve stimulator (Life-Tech, Inc., MiniStim Model MS-1) was used to assess the need for any additional doses of the neuromuscular blocking agent. A period of 8 min was allowed to ensure complete effect of the drug. Then, a second set of two recordings was acquired for studying the effects of neuromuscular blockade on the cardiac signals recorded.

A sternotomy was then performed and a telemetric AV sequential rate-programmable pacemaker (Medtronic Model 7005C) implanted after the production of a chronic complete heart block as described below. First, an epicardial unipolar lead (Medtronic Model 4951) was fixed on the left atrium and another on the epicardium near the apex of the left ventricle. The two leads were routed from the thorax to a lateral abdominal wall opening and connected to the AV sequential pacemaker. The normal function of the bundle of His was then destroyed with one to four formaldehyde injections of 0·1 ml each, according to the method of STEINER and KOVALIK (1968). Ventricular and atrial pacing were then resumed by inserting the pacemaker into a pocket located within the muscles of the abdominal wall. The P-R interval was adjusted between 75 and 125 ms and the heart rate at 100 or 120 beats min^{-1}. Finally, the abdominal wall opening was sutured and a trocar catheter was inserted within the thorax through a small opening between the ninth and tenth rib. The sternum was closed and a vacuum pump connected to the trocar catheter, which was then clamped when a sufficient negative intrathoracic pressure was restored. The contact microphones were repositioned and a third set of two recordings was acquired to evaluate the effects of immediate sternotomy and cardiac pacing on the cardiac signals. Neuromuscular blockade was then reversed and anaesthesia discontinued. Two weeks of recovery were then allowed to the dog before the second experiment was carried out.

2.2 *Second experiment*

After anaesthetising and instrumenting the animals with the same procedure used during the first experiment, a telemetric pacemaker programmer (Medtronic model 9710) was used to program the P-R interval and heart rate independently. Cardiac inotropy was varied by using injections or infusions of cardiotonic drugs. One or two signal recordings were made after each chronotropic or inotropic alteration of the heart function. The protocols used during these two substudies will be described in Parts 3 and 4 of this series of papers.

2.3 *Signal processing and spectral analysis*

Signal processing was done on an IBM-PC/AT. The objective was to characterise the spectral density functions of the mitral component M1 of the first heart sound and of the aortic component A2 of the second heart sound. Estimates of the transfer function (gain and phase) and of the coherence function of the heart/thorax acoustic system during the production of M1 and A2 were also computed. The reader is referred to Part 1 of this series for details related to signal processing and spectral analysis (DURAND *et al.*, 1990).

2.4 *Quantification of the spectral changes*

The influence of neuromuscular blockade and that of sternotomy and cardiac pacing before and after the recovery period of two weeks were evaluated by using each dog as its own reference. For each intervention and for each dog investigated, difference spectra were computed by subtracting the spectral functions describing the status of the heart/thorax system after a given intervention from those recorded before by using:

$$D_{qi}(Sx, f) = X_{qib}(Sx, f) - X_{qia}(Sx, f) \tag{1}$$

where q = the type of spectral function on which the measurement is performed (the intracardiac sound spectrum, the thoracic sound spectrum, the gain and phase of the transfer function and the coherence function)
i = dog number
b = relative temporal index for recordings made before the intervention
a = relative temporal index for recordings made after the intervention
Sx = the sound component analysed (M1 or A2)
f = frequency.

Bias and standard error of the mean spectra were also computed from these difference spectra. The bias spectra were defined by

$$B_q(Sx, f) = \frac{1}{M} \sum_{i=1}^{M} D_{qi}(Sx, f) \tag{2}$$

and the standard error of the mean spectra were defined by

$$SEM_q(Sx, f) = \frac{1}{\sqrt{M}} \left\{ \frac{1}{M-1} \sum_{i=1}^{M} [D_{qi}(Sx, f) - B_q(Sx, f)]^2 \right\}^{0.5} \tag{3}$$

where M is the total number of dogs investigated. To obtain a quantitative evaluation of the effect of each intervention, the mean value of each bias spectrum ($B_q(Sx, f)$) were computed between 20 and 100 Hz and between 100 and 500 Hz.

2.5 *Validation of the spectral changes*

The spectral changes observed after a given intervention were validated by comparison with boundaries defining

the normal baseline variation of the heart/thorax acoustic system as a function of time. For this purpose, two temporal references were selected from three recordings of the second experiment. It is important to emphasise that these three recordings were made under identical electrophysiological conditions (heart rate of 100 beats min^{-1} and P-R interval of 75 ms).

The first reference interval had a duration of 12 min and was used to validate the effect of neuromuscular blockade. During this reference interval, the heart rate was varied between 100 and 130 beats min^{-1} while the P-R interval was varied between 25 and 75 ms, before returning to their initial values (HR = 100 beats min^{-1} and PRI = 75 ms). The second reference interval had a duration of 50 min and was used to validate the effect of immediate sternotomy and AV sequential pacing, and that of the two-week recovery period. During this second interval, the heart rate was varied between 70 and 130 beats min^{-1} while the PR interval was varied between 25 and 175 ms.

Because there was no other variable than heart rate and P-R interval modified during these intervals, and also because all recordings were made with identical heart rate and P-R interval, we believed that the difference spectra of these intervals could be used as references to determine the normal variability of the heart/thorax acoustic system. This hypothesis is supported by the work of McDonald (1974) who has shown that, following a sudden change in cardiac rhythm or loading impedance, the steady state of the cardiac system is recovered within only one cardiac cycle.

The variability of the difference spectra of the reference intervals was determined by computing a minimum ($B-$) and a maximum ($B+$) boundary function by using $B \pm = B_q \pm 2SEM_q$ (see eqns. 2 and 3). The spectral boundary functions were then averaged between 20 and 100 Hz and 100 and 500 Hz to generate two reference boundary parameters ($RB-$ and $RB+$).

3 Results

The frequency bandwidth parameters obtained from the bias spectra of the three interventions (neuromuscular blockade, immediate sternotomy and recovery period) and the boundary parameters of the two reference intervals (12 and 50 min) are presented in Tables 1 and 2. The content of these Tables will be used in the following sections to assess the significance of the effect of each intervention studied.

Table 1 Comparisons of bias parameters (BP) computed from the difference spectra of M1 obtained after three interventions with the reference boundary parameters (RB±) of two reference intervals (R12 and R50). R12 and R50 are reference intervals having a duration of 12 and 50 min, respectively. The three interventions studied are: neuromuscular blockade (NB), sternotomy and atrio-ventricular pacing (SAVP), and a two-week recovery period (2WRP). Two frequency bands are analysed: 20–100 Hz and 100–500 Hz

Spectral function	Intervention/ reference	20–100 Hz			100–500 Hz		
		BP	*RB−*	*RB+*	*BP*	*RB−*	*RB+*
$M1_{lv}$	NB/R12	−2·83	−3·20	1·20	−3·19	−1·89	0·71
dB	SAVP/R50	2·29	−10·42	1·02	4·07	−10·11	−0·87
	2WRP/R50	−3·87	−10·42	1·02	−5·82	−10·11	−0·87
$M1_{ap}$	NB/R12	−1·67	−2·30	2·04	−1·57	−3·59	1·35
dB	SAVP/R50	−16·59	−9·68	7·80	−9·14	−8·56	8·06
	2WRP/R50	−1·34	−9·68	7·80	1·31	−8·56	8·06
$TF_{(M1)}$	NB/R12	0·49	−3·60	4·82	−0·35	−6·34	6·54
gain	SAVP/R50	−24·18	−7·48	12·60	−19·43	−7·69	17·89
dB	2WRP/R50	−0·39	−7·48	12·60	0·75	−7.69	17·89
$TF_{(M1)}$	NB/R12	−103·7	−44·9	278·7	−186·7	−307·1	862·6
phase,°	SAVP/R50	−82·7	−165·5	267·6	108·1	−1559·4	1813·6
	2WRP/R50	−95·8	−165·5	267·6	70·9	−1559·4	1813·6
$CF_{(M1)}$	NB/R12	−0·07	−0·22	0·12	−0·07	−0·04	0·06
	SAVP/R50	−0·43	−0·44	0·28	−0·19	−0·06	0·06
	2WRP/R50	−0·27	−0·44	0·28	−0·19	−0·06	0·06

Table 2 Comparisons of bias parameters (BP) computed from the difference spectra of A2 obtained after three interventions (NB, SAVP, 2WRP) with the boundary reference parameters (RB±) of two reference intervals (R12 and R50). See Table 1 for the meaning of the abbreviations

Special function	Intervention/ reference	20–100 Hz			8–500 Hz		
		BP	*RB−*	*RB+*	*BP*	*RB−*	*RB+*
$A2_{ao}$	NB/R12	0·44	−2·61	0·75	0·95	−3·53	1·71
dB	SAVP/R50	−0·19	−5·97	3·35	−1·15	−6·31	7·37
	2WRP/R50	0·04	−5·97	3·35	4·69	−6·31	7·37
$A2_{aa}$	NB/R12	−1·93	−4·81	0·59	0·18	−5·06	0·90
dB	SAVP/R50	−14·89	−8·08	0·78	−4·45	−7·47	1·43
	2WRP/R50	−5·52	−8·08	0·78	1·42	−7·47	1·43
$TF_{(A2)}$	NB/R12	−2·89	−5·61	2·01	−2·99	−7·43	5·37
gain	SAVP/R50	−16·60	−9·36	4·80	−10·13	−16·69	7·13
dB	2WRP/R50	−5·84	−9·36	4·80	1·41	−16·69	7·13
$TF_{(A2)}$	NB/R12	−22·8	−204·2	71·5	−6·6	−493·3	712·7
phase,°	SAVP/R50	−9·1	−42·4	261·5	−190·0	−760·0	196·1
	2WRP/R50	9·0	−42·4	261·5	253·3	−760·0	196·1
$CF_{(A2)}$	NB/R12	−0·07	−0·24	0·16	−0·06	−0·07	0·11
	SAVP/R50	−0·19	−0·22	0·26	−0·13	−0·12	0·10
	2WRP/R50	−0·03	−0·22	0·26	−0·10	−0·12	0·10

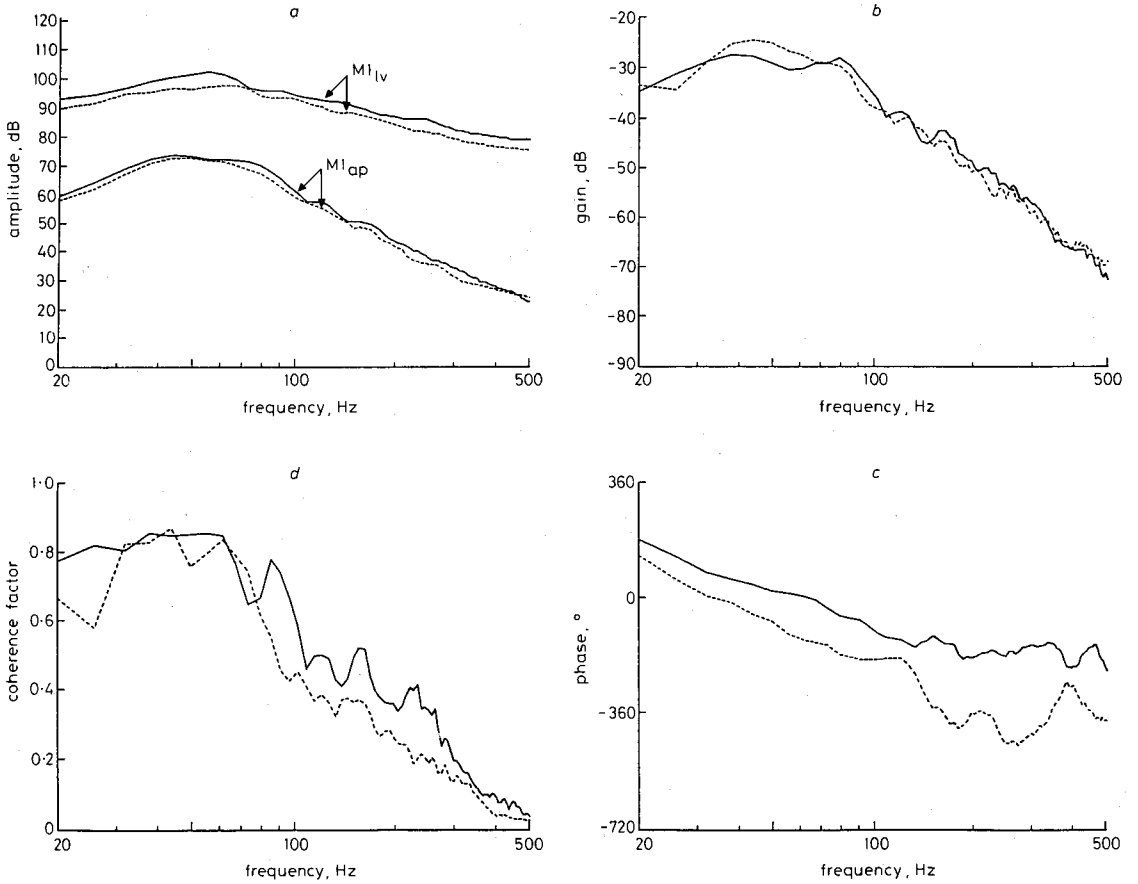

Fig. 1 *Influence of neuromuscular blockade on the status of the heart/thorax acoustic system during the production of M1. Results are presented for seven dogs. (a) Mean spectra of the left ventricular and apical M1 components ($M1_{lv}$ and $M1_{ap}$); (b) gain and (c) phase of the transfer function; (d) mean coherence function. Full lines represent data obtained before the neuromuscular blockade, and broken lines represent data obtained after the neuromuscular blockade*

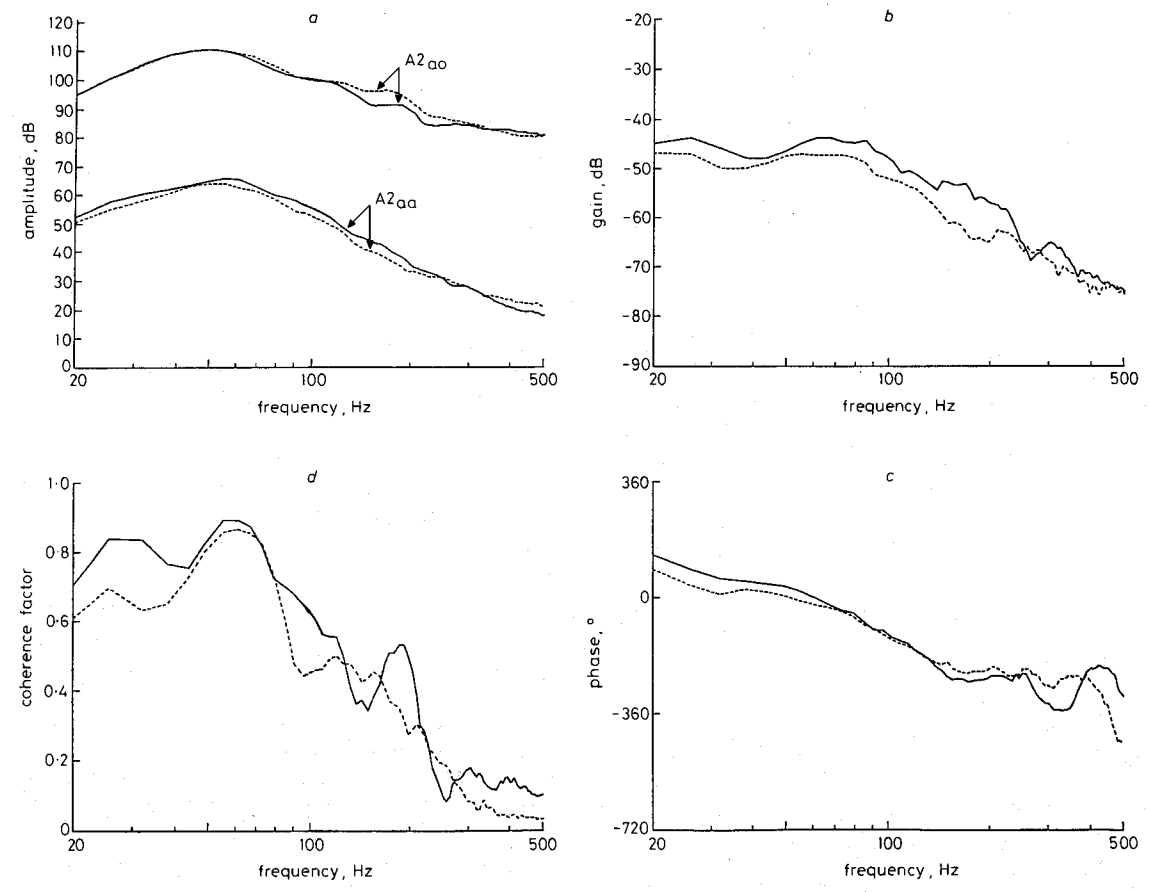

Fig. 2 *Influence of neuromuscular blockade on the status of the heart/thorax acoustic system during the production of A2. Results are presented for seven dogs. (a) Mean spectra of the A2 sounds recorded within the aorta ($A2_{ao}$) and at the aortic area ($A2_{aa}$) on the body surface; (b) gain and (c) phase of the transfer function; (d) mean coherence function. Full lines represent data obtained before the neuromuscular blockade, and broken lines represent data obtained after the neuromuscular blockade*

3.1 *Effect of neuromuscular blockade*

The spectra describing the status of the heart/thorax acoustic system before and after neuromuscular blockade are shown in Fig. 1 during the transmission of M1 and in Fig. 2 during the transmission of A2. As this intervention had a duration varying between 10 and 15 min, the frequency bandwidth parameters of the difference spectra were compared with the boundary parameters of the 12 min reference interval (see NB/R12 in Tables 1 and 2).

Fig. 1*a* shows a 3 dB reduction in the intensity of the spectra of the left ventricular M1 $M1_{lv}$ and a 1·6 dB reduction of the apical M1 $M1_{ap}$ in the 20–500 Hz band. According to Table 1, these changes are not higher than those observed after the 12 min interval, except for the left ventricular M1 in the 100–500 Hz band. Fig. 1*b* shows that the gain of the transfer function is not modified by the neuromuscular blockade. However, the phase of the transfer function (Fig. 1*c*) is changed in the 20–100 Hz band. Finally, the coherence function shown in Fig. 1*d* appears to be slightly reduced above 100 Hz. According to Table 1, this reduction is not negligible and is also in agreement with the reduced intensity of left ventricular M1 above 100 Hz.

Similarly to Fig. 1, Fig. 2 suggests that neuromuscular blockade does not affect the spectra describing the status of the heart/thorax acoustic system during the production of A2. This visual interpretation is confirmed by the parameters of Table 2. Even the 3 dB reduction of the gain of the transfer function is negligible compared with the reference boundary parameters of the 12 min reference interval.

3.2 *Effect of immediate sternotomy and AV sequential pacing*

The influence of sternotomy and AV pacing on the spectra of the sound components and on the status of the heart/thorax acoustic system is shown in Fig. 3 for M1 and in Fig. 4 for A2. As the sternotomy had a duration around one hour, the comparison of its influence on the frequency bandwidth parameters describing the status of the heart/thorax acoustic system was made with the boundary parameters of the 50 min reference interval (see SAVP/R50 in Tables 1 and 2).

Both figures clearly demonstrate that the attenuation of the heart/thorax acoustic system is highly increased immediately after the sternotomy. This increase in sound attenuation results in a decrease of the amplitude of the coherence function and of the intensity of the thoracic M1 and A2 ($M1_{ap}$ and $A2_{aa}$), even if the intensity of the left ventricular M1 $M1_{lv}$ is increased in both the lower (2·3 dB) and the higher (4·1 dB) frequency ranges while that of the intra-aortic A2 $A2_{ao}$ is not modified.

The increased sound attenuation due to the sternotomy seems to vary as a function of the frequency. According to Table 1, the intensity of M1 recorded on the body surface $M1_{ap}$ is reduced by a factor of 17 dB below 100 Hz and 9 dB above that frequency. Similarly, Table 2 shows that the intensity of A2 recorded on the body surface $A2_{aa}$ is reduced by a factor of 15 dB below 100 Hz and 4 dB above that frequency. Comparison of panels (*a*) and (*b*) of both figures shows that the reduction of the intensity of the external sound components is lower than the reduction of the gain of the corresponding transfer functions. During the transmission of M1, the mean sound intensity between 20 and 500 Hz is reduced by 11 dB while that of the transfer function is reduced by 20 dB. During the transmission of A2, the mean sound intensity (20–500 Hz) is reduced by 6 dB while that of the transfer function is reduced by 11 dB. These results show that there is a difference between the increase in sound attenuation and the reduction of the gain of the transfer functions.

According to Tables 1 and 2, the changes observed in the phase of the transfer function during transmission of M1 and A2 are negligible (see also Figs. 3 and 4). As shown by the coherence functions of panels (*d*) of both figures, the relative contribution of the intracardiac sound components to the thoracic PCGs is highly decreased in both cases. According to Tables 1 and 2, this decrease in coherence amplitude is more important for M1. It is just above the negative reference boundary ($RB-$) for frequencies below 100 Hz and below it and thus not negligible for the higher frequencies.

3.3 *Effect of a recovery period of two weeks after sternotomy*

The spectra describing the status of the heart/thorax acoustic system before and after the recovery period are shown in Fig. 5 for the transmission of M1 and in Fig. 6 for the transmission of A2. The frequency bandwidth parameters extracted from the difference functions of the recovery period were compared with the boundary parameters of the 50 min reference interval (see 2WRP/R50 in Tables 1 and 2).

As shown by Fig. 5 and Table 2, the spectral intensity of M1 recorded on the body surface $M1_{ap}$ and the gain and phase of the transfer function (Figs. 5*b* and 5*c*) after the recovery period are not different from those recorded before the sternotomy. According to Table 1, the 5·8 dB intensity reduction of the left ventricular M1 spectrum $M1_{lv}$ above 100 Hz is also not important. However, Fig. 5*d* and the quantitative parameters of Table 1 show that the coherence function remains rather low in the higher frequency range.

According to the intensity of A2 $A2_{aa}$ recorded on the body surface and to the gain and phase of the transfer function Figs. 6*b* and 6*c*, the acoustic transmission path of A2 does not appear to have returned to its normal attenuation level after the recovery period. Table 2, however, shows that these residual changes are small compared with the boundary parameters of the 50 min reference, except for the phase above 100 Hz. Finally, the amplitude of the coherence function is not modified except, perhaps, for the decrease in the resonant contribution of the intra-aortic A2 to the thoracic phonocardiogram around 187 Hz.

4 Discussion and conclusion

Even if the drug used to induce neuromuscular blockade has a strong effect on the tonus of the skeletal muscles, its effect on the acoustic transmission of the sound components through the heart/thorax system is negligible. The influence of the drug is limited to a change of phase in the 20–100 Hz band and a small reduction of cardiac inotropy (the maximum value of the dP/dt was reduced by 12 per cent) during the production of M1. This conclusion is supported by the fact that the spectral intensity of M1 recorded within the left ventricle and its coherence contribution to the external PCG were slightly reduced after neuromuscular blockade. This observation is in accordance with the studies of SAKAMOTO *et al.* (1965), STEPT *et al.* (1969) and GENEST and DURAND (1985) which have demonstrated that a reduction in cardiac inotropy results in a decrease of the intensity of the first heart sound. No similar effect is observed for the A2 component.

Our study has demonstrated that the immediate sternotomy drastically increases the attenuation of the heart/thorax system and reduces the intensity of the sound

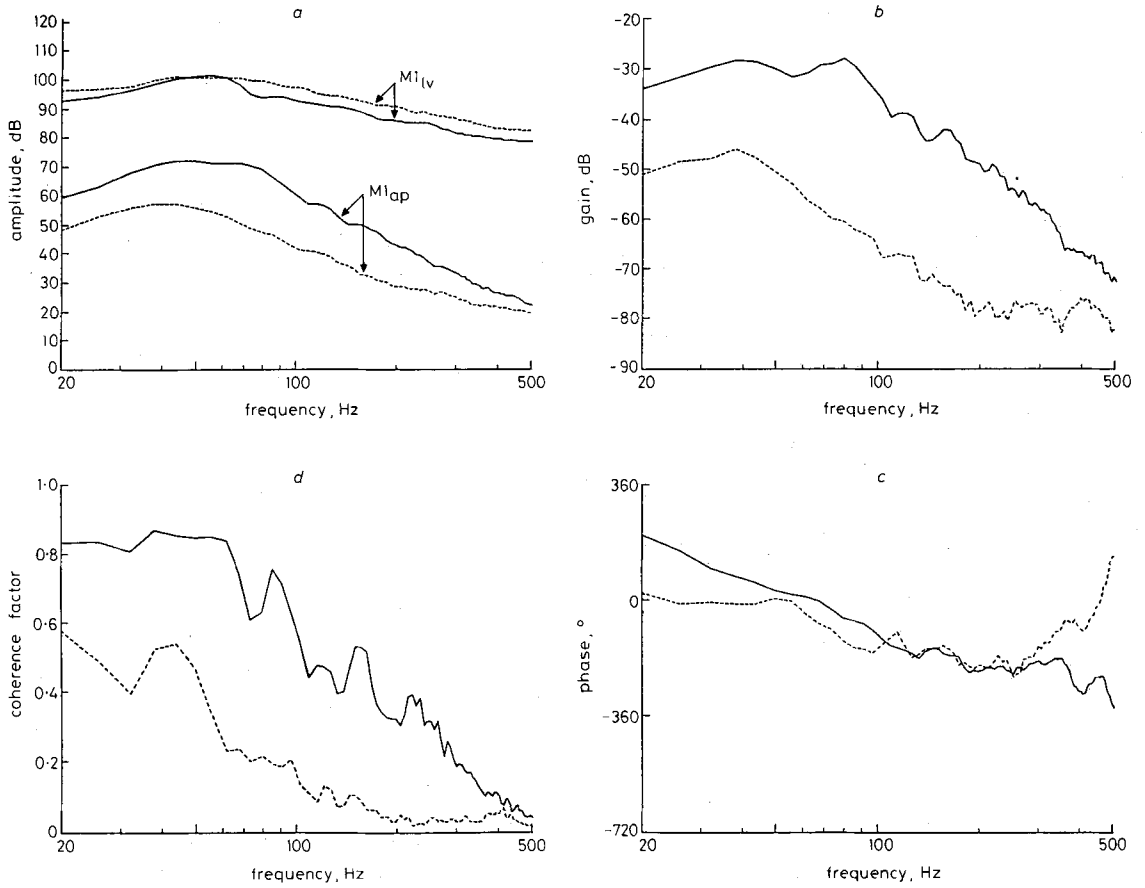

Fig. 3 *Influence of immediate sternotomy and cardiac pacing on the spectra describing the status of the heart/thorax acoustic system during the production of M1. Results are presented for seven dogs. Full lines represent data obtained before the sternotomy, and broken lines represent data obtained after the sternotomy. A format similar to that of Fig. 1 is used*

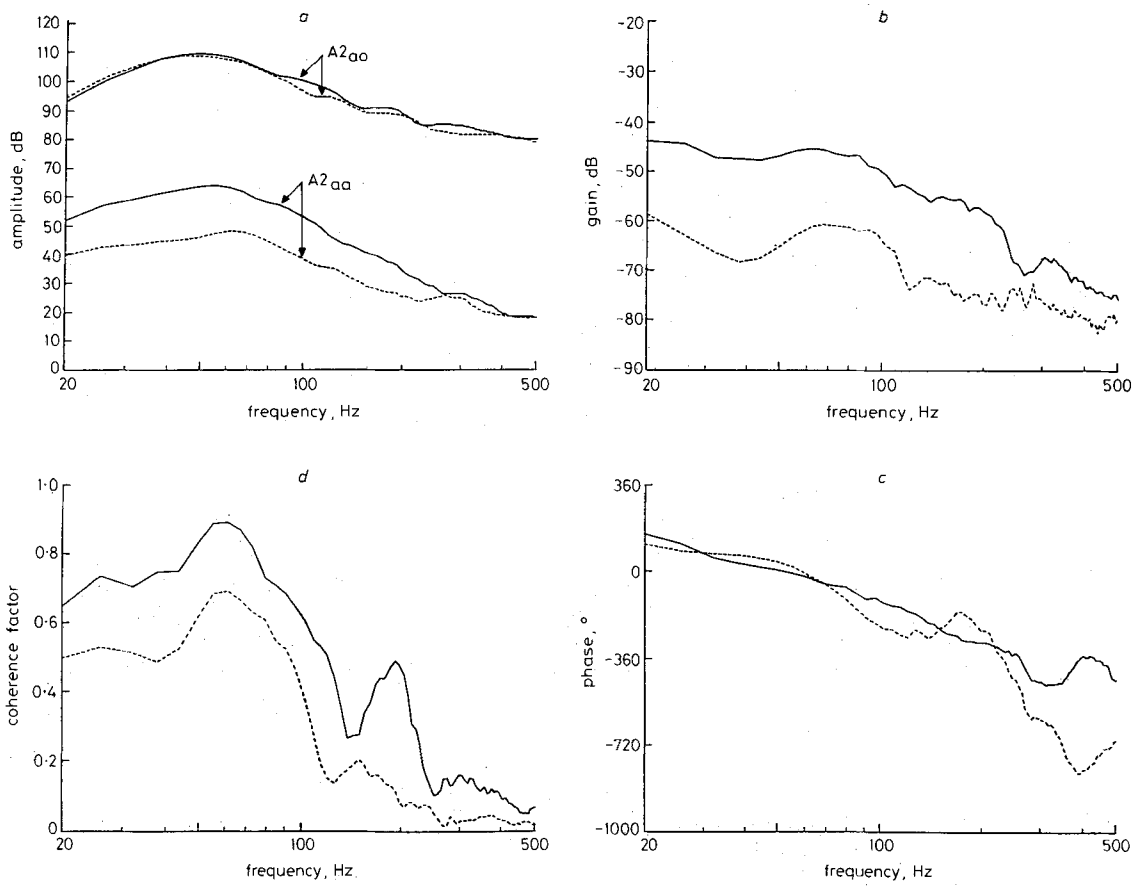

Fig. 4 *Influence of immediate sternotomy and cardiac pacing on the spectra describing the status of the heart/thorax acoustic system during the production of A2. Results are presented for seven dogs. Full lines represent data obtained before the sternotomy, and broken lines represent data obtained after the sternotomy. A format similar to that of Fig. 2 is used*

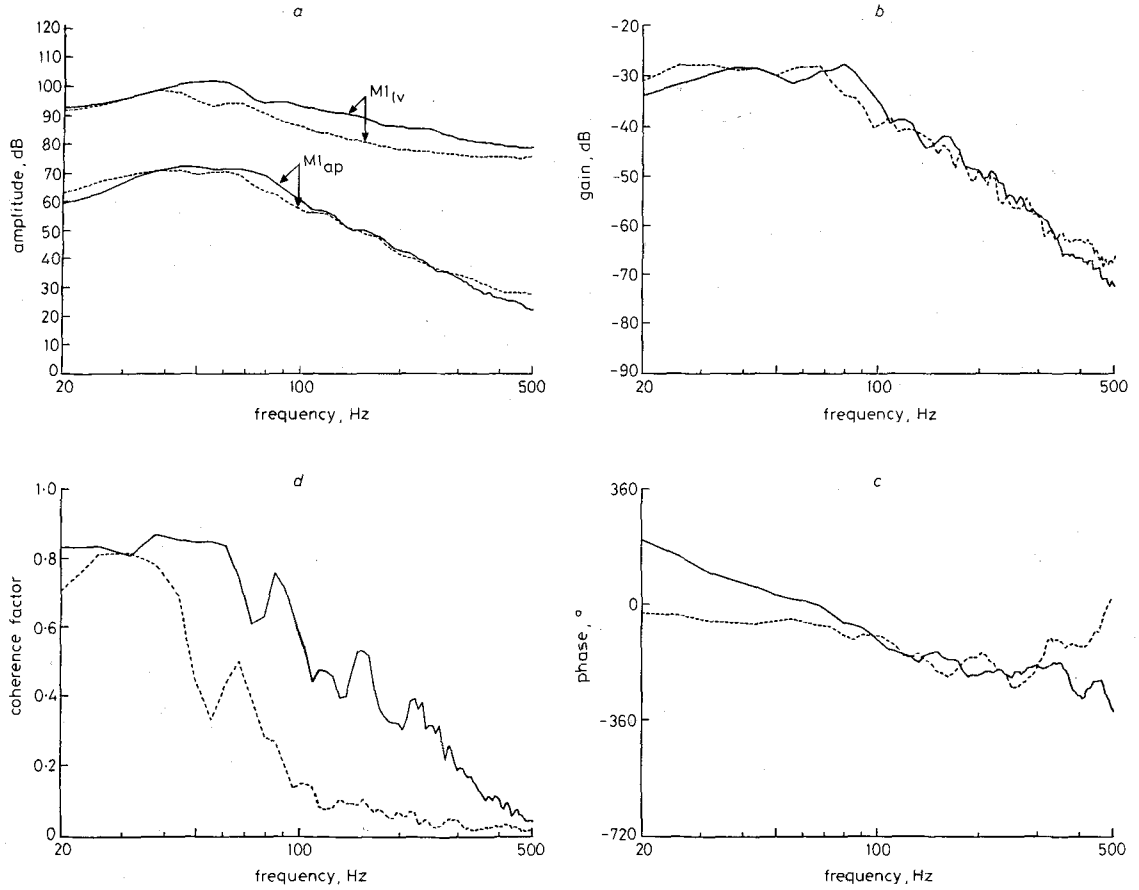

Fig. 5 *Effect of a recovery period of two weeks after the sternotomy on the spectra describing the status of the heart/thorax acoustic system during the production of M1. Results are presented for six dogs. Full lines represent data obtained before the sternotomy, and broken lines represent data obtained after the recovery period. A format similar to that of Fig. 1 is used*

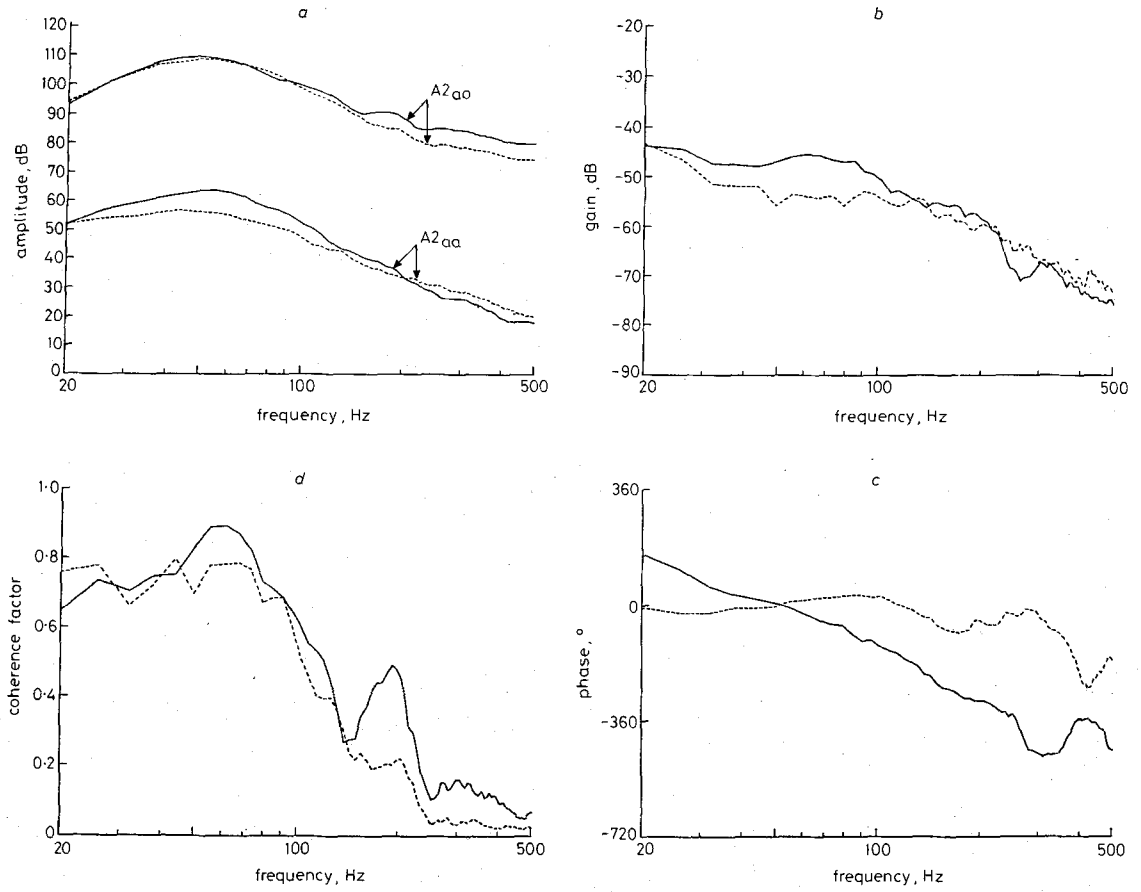

Fig. 6 *Effect of a recovery period of two weeks after the sternotomy on the spectra describing the status of the heart/thorax acoustic system during the production of A2. Results are presented for six dogs. Full lines represent data obtained before the sternotomy, and broken lines represent data obtained after the recovery period. A format similar to that of Fig. 2 is used*

components recorded on the body surface. This reduction in the gain of the transfer function probably results from a bad acoustic coupling between the heart and thoracic structures following dehydration of the cardiac and thoracic tissues. Bad acoustic coupling may be due to presence of residual air between cardiac, pericardial or pulmonary tissues and the chest wall even after regeneration of the negative intrapleural pressure at the end of the sternotomy. We believe that a two-week recovery period allows the tissues sufficient time to resorb the residual air and retrieve their natural perfusion and hydration which provide normal acoustic coupling between the various structures composing the heart/thorax system.

The reduced coherence contribution of the left ventricular M1 to the thoracic PCG shown in Fig. 5 may be due, in part, to the reduced intensity of the left ventricular M1, and also to the complete modification of the AV depolarisation sequence of the heart. The new AV excitation scheme resulting from the destruction of the bundle of His and the initiation of artificial pacing may have strongly altered the cardiac mechanics and markedly affected the linear properties of the cardiac acoustic system during ventricular isovolumetric contraction.

Our results clearly show that the increased attenuation of the transfer function after immediate sternotomy seems to be higher than, and thus in contradiction with, the decreased intensity of the external sound components. However, this is not the case. For instance, the noise $n(t)$ recorded on the thorax may contain, in addition to the contribution of intracardiac, respiratory and intestine peristaltic noises, other significant sources of noise such as muscular thoracic noise and ambient vibrations mostly transmitted by the surgery table (floor noise). Thus, a reduction in the gain of the transfer function will not automatically result in a proportional reduction of the thoracic noise of the system. Consequently, the thoracic noise is a limiting factor in the reduction of the intensity of the external sound components. However, the cross-correlation technique used to estimate the gain of the transfer function is independent of any thoracic noise not correlated to the intracardiac signal and can thus provide a very good estimate of the real gain of the transfer function, even in the presence of a thoracic PCG having a low signal-to-noise ratio. This observation is in perfect agreement with the work of BENDAT and PIERSOL (1980), who have shown that, in the presence of uncorrelated noise, the cross-correlation method is highly superior to the division of the output spectrum by the input spectrum to estimate the gain of the transfer function of the system.

On the other hand, the spectral changes observed after the sternotomy and cardiac pacing are not due to a change in P-R interval and heart rate. For instance, the mean P-R interval and heart rate of the seven dogs were 96 ms and 117 beats min^{-1} before the sternotomy and 92 ms and 107 beats min^{-1} after the sternotomy. The spectral changes of Figs. 5 and 6 are probably due to the important changes in the atrio-ventricular depolarisation sequence following the destruction of the bundle of His. The reduced coherence function observed during the production of M1 can also be attributed to the dramatic changes in the ventricular excitation path. The reverse excitation pattern of the ventricles may have increased the nonlinear characteristics of the heart/thorax acoustic system during ventricular contraction. This effect is not apparent for A2 because the transmission path studied is completely different.

The various peaks observed on the waveform of the coherence functions of Figs. 1–6 reflect the resonant properties of the valvular sounds filtered by the frequency response of the heart/thorax acoustic system. One of the most striking observations of this study is the permanent change in the phase of the transfer function of A2 obtained after the recovery period. Individual inspections of the results of each dog showed that, in some animals, the phase slope was reversed after the recovery period. This can be due to various factors (BENDAT and PIERSOL, 1980). For instance, dispersive propagation (i.e. the propagation speed proportional to frequency), which is a phenomenon resulting from an increase of the surface wave components of the PCGs, could reverse the phase relationship with the frequency. Reverberation and obstruction or lack of homogeneity in the transmission path may cause scattering of sound waves, a phenomenon which is also very detrimental to the accuracy of the phase estimate. Finally, the phase estimate of the transfer function is also very sensitive to the presence of noise in the input and output signals as well as to nonlinearities of the system.

The present study also shows that the surgical technique and instrumentation used to produce a controlled chronic animal model have resulted in a model slightly different from that of the intact normal animals. The gain of the transfer function is not changed significantly, but the coherence function of the left ventricular M1 and intra-aortic A2 remains lower. This could be due to change in the linearity of the system. In addition, the resulting changes in the spectral properties of the mitral and aortic valve closure sound components and their acoustic transmission were always more important for M1 than for A2. While being still valid for understanding the transmission of sounds in normal patients under various electrophysiological conditions, this chronic dog model should be of great interest for transposing the results of heart sound analyses in patients having had a sternotomy and/or a cardiac pacemaker implantation.

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References

BENDAT, J. S. and PIERSOL, A. G. (1980) *Engineering applications of correlation and spectral analysis.* John Wiley & Sons, New York.

DURAND, L.-G., LANGLOIS, Y.-E., LANTHIER, T., CHIARELLA, R., COPPENS, P., CARIOTO, S. and BERTRAND-BRADLEY, S. (1990) Spectral analysis and acoustic transmission of mitral and aortic valve closure sounds in dogs. Part 1 Modelling the heart/thorax acoustic system. *Med. & Biol. Eng. & Comput.*, **28**, 269–277.

GENEST, J. Jr and DURAND, L. G. (1985) Relationship of the left ventricular and apical first sounds to the left ventricular pressure derivative. *Ibid.*, **23**, 95–98.

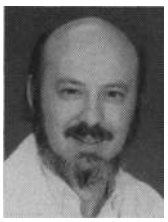
MCDONALD, D. A. (1974) *Blood flow in arteries.* Edward Arnold, London, 164–165.

RANGAYYAN, R. M. and LEHNER, R. J. (1988) Phonocardiogram signal analysis: a review. *CRC Crit. Rev. Biomed. Eng.*, **15**, 211–236.

SAKAMOTO, T., KUSUKAWA, R., MACCANON, D. M. and LUISADA, A. A. (1965) Hemodynamic determinants of the amplitude of the first heart sound. *Circ. Res.*, **16**, 45–57.

STEINER, C. and KOVALIK, T. W. (1968) A simple technique for production of chronic complete heart block in dogs. *J. Appl. Physiol.*, **25**, 631–632.

Authors' biographies



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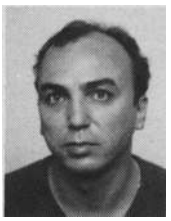

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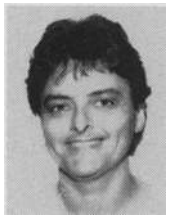

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STEPT, M. E., HEID, C. E., SHAVER, J. A., LEON, D. F. and LEONARD, J. J. (1969) Effect of altering P-R interval on the amplitude of the first heart sound in the anesthetized dog. *Circ. Res.*, **25**, 255–263.

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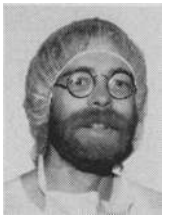

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