

Quantitative evaluation of the systemic arterial bed by parameter estimation of a simple model

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Abstract—The parameters of a simple model (r - L - C - R) of the systemic circulation are estimated from aortic root pressure and flow, which are either simulated by a complex model of the systemic circulation or measured in dogs. This model contains one additional parameter (inductance L) as compared with the r - C - R model proposed by Westerhof; it allows for a better representation of the input impedance of the complex model and of the systemic circulation in dog, resulting in meaningful values for the parameters r , C , R . Because there is a good relation between C and the sum of the compliances of the complex model, and because C varies in the direction of the expected changes in compliance following angiotensin and sodium nitroprusside administration in dogs, C appears to be a valid estimate of the total systemic arterial compliance. The good relation between r and the characteristic impedance in the complex model or in the upper thoracic aorta of the dog indicates that r is a good measure of the characteristic impedance. The r - L - C - R model therefore appears to provide a better characterisation of the left ventricular afterload than the r - C - R model. The identification of this r - L - C - R model also permits a more convenient quantification of the afterload than the classical computation of input impedance.

Keywords—Simple model-parameter estimation, Systemic arterial bed

1 Introduction

IN a recent review, MILNOR (1975) wrote that 'afterload must be measured and taken into account in any evaluation of ventricular performance'. Assuming that the systemic arterial bed can be considered as a linear system, the input impedance of the arterial bed which is certainly the more complete description of the left ventricular afterload should therefore be computed. However the input impedance which by definition can represent a system of infinite order is difficult to interpret. Moreover the scatter on the input impedance curves obtained from aortic pressure and flow harmonics is generally large (NICHOLS *et al.*, 1977; WESTERHOF *et al.*, 1973) at least above the third harmonic. For these reasons, the physical interpretation of the input impedance is difficult and generally restricted to two quantities (NICHOLS *et al.*, 1977): the total peripheral resistance (modulus of the input impedance at zero frequency) and the characteristic impedance (average of impedance moduli between about 3 to 10 Hz).

The systemic arterial bed has also been described by simple models amongst which the 'windkessel' model (FRANK, 1899) and the extended 'windkessel'

model or 'Westkessel' model (WESTERHOF, 1968) are certainly the more commonly used. *A priori*, the advantage of such kind of simple models in comparison with the input impedance, consists in the finite number of parameters. Indeed, if from pressure and flow measured at the root of the aorta, the parameters of such simple models are estimated and if these estimated values admit some physical meanings, these parameter values could be used as quantitative indices measuring the afterload (BENEKEN, 1972).

This paper deals with the study of the possibilities to 'measure' left-ventricular afterload and its modifications by the estimation of the parameters of a simple model of the systemic arterial bed from measurements of instantaneous pressure and flow at the root of the aorta. To study the validity of a simple model and the physical meanings of its parameters, we proceed with two complementary approaches

(a) 'Complex model to simple model' approach (or simulation approach)

Different circulatory states are simulated by a complex model (CHANG, 1973; CHANG *et al.*, 1974; SIMS, 1972) which represents the main parts of the systemic circulation. The solutions of the equations of this complex model are computed to obtain simulated aortic pressure and flow signals. The

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parameters of the simple model are then estimated from these simulated signals and the meaning of the different parameters is studied.

(b) *Experimental approach*

The parameters of the simple model are estimated from experimental measurements of aortic root pressure and flow under different circulatory conditions in dogs. We then examine whether the parameter values allow us to quantify the different circulatory states and in particular whether their physical interpretations are still valid.

This paper is organised in the following manner. The first Section briefly presents the choice of the simple model and the estimation procedure. The second and third Sections deal with the simulation and experimental approaches, respectively. A discussion is finally given.

2 Simple model of the systemic arterial bed and estimation procedure

The systemic arterial bed has been represented by the 'windkessel' model (Fig. 1a) which contains one capacitance C and one resistance R . LANDES (1943) has proposed modifications to this model by adding one resistance r (Fig. 1b) or one resistance r and one inductance L (Fig. 1c). The model r - C - R (Fig. 1b) has been extensively studied by WESTERHOF (1968) who showed that this model gives a better representation of the pressure-flow relation at the aortic root than the 'windkessel' and concluded that the adjunction of an inductance L (Fig. 1c) does not improve the model. Other simple models containing more parameters have been proposed by SPENCER and DENISON (1963) (Fig. 1d) and by GOLDWYN and

WATT (1967) (Fig. 1e). For all these models, the input voltage U_{in} is the analogue of the aortic root pressure p_{ao} and the input current i_{in} is the analogue of the aortic root flow q_{ao} .

Our preliminary studies (DESWYSEN, 1977a) have indicated that the parameters of Goldwyn and Watt's model are difficult to determine univoquely and, for this reason, cannot admit any physical meaning. A good fit of the input impedance of the systemic arterial bed indeed does not require the use of this more complete model nor, *a fortiori*, the use of Spencer and Denison's model.

The following physical interpretation has been given to the parameters of the r - C - R model (WESTERHOF, 1968):

- (a) the sum $r + R$ is equal to the total peripheral resistance, i.e. the ratio between mean aortic pressure and mean flow (for periodical pressure and flow signals)
- (b) r , called the characteristic resistance, is the model representation of the characteristic impedance of the aorta where pressure and flow are measured; its value would be related to the mechanical and geometrical properties of the aorta at this level.
- (c) C is the total compliance of the arterial bed, i.e. the ratio of the change in volume to the change in pressure of all arteries lumped together.

For the reasons explained above, we have chosen to study the r - C - R model. However, the main contribution of this paper will be to show that the addition of an inductance L (Fig. 1c) gives a significantly better representation of the aortic root pressure-flow relation, resulting in more meaningful values for the parameters r , C and R when estimated from p_{ao} and q_{ao} .

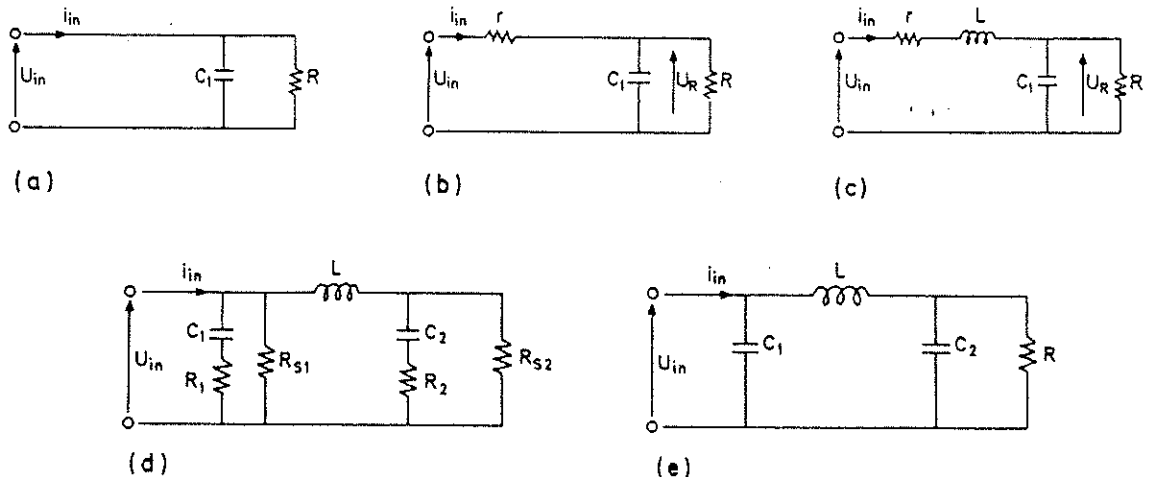


Fig. 1 Simple models of the systemic arterial bed
 U_{in} = analogue of the aortic root pressure p_{ao}
 i_{in} = analogue of the aortic root flow q_{ao}
 (a) 'Windkessel' model (Frank, 1899)
 (b) Model proposed by Landes (1943) and

extensively studied by Westerhof (1968)
 (c) Model proposed by Landes (1943)
 (d) Spencer and Denison model (1963)
 (e) Goldwyn and Watt model (1967)

The estimation procedure is now briefly described. From measurements of the instantaneous pressure p_{ao} and flow q_{ao} at the root of the aorta during one beat, we want to estimate the parameters r , C , R and r , L , C , R of the models illustrated in Figs. 1b and c. The two models can be described by a state equation that represents the system dynamics, and by a measurement equation relating the measurement q_{ao} with the state variables of the model and with a noise term W_m . These equations are

(i) *r-C-R model*

$$\text{state equation: } dU_R/dt = -U_R(1/r + 1/R)/C + p_{ao}/r.C \quad (1)$$

$$\text{measurement equations: } q_{ao} = i_{in} + W_m \quad (2)$$

where

$$i_{in} = (p_{ao} - U_R)/r$$

where

U_R is the single state variable, this model being of order one

(ii) *r-L-C-R model*

state equations:

$$\left. \begin{aligned} di_{in}/dt &= -U_R/L - r \cdot i_{in}/L + p_{ao}/L \\ dU_R/dt &= (i_{in} - U_R/R)/C \end{aligned} \right\} \quad (3)$$

$$\text{measurement equation: } q_{ao} = i_{in} + W_m \quad (4)$$

where i_{in} , U_R is the state vector, this model being of order two.

The estimation problem consists in determining the values of the parameter vectors, respectively

$$\theta = \begin{bmatrix} r \\ R \\ C \end{bmatrix} \quad \text{and} \quad \theta = \begin{bmatrix} r \\ R \\ C \\ L \end{bmatrix}$$

that minimise the output error J during the beat under study:

$$J = \sum_{k=0}^N W_m^2(kT) = \sum_{k=0}^N (q_{ao}(kT) - i_{in}(kT))^2$$

where T is the sampling period and NT is the duration of the beat, with time origin ($t = 0$) at the beginning of the beat, that we arbitrarily fixed at the beginning of the ejection. In the exp. 5, $q_{ao}(kT)$ is the measured or simulated aortic rootflow, while $i_{in}(kT)$ is the solution of the model equations.

A recursive procedure is used to compute θ . The chosen minimisation procedure is a direct search method that only requires the evaluation of the error cost J without computing the gradient of J .

The parameters are adjusted one by one: each component of θ is taken successively and its value is brought to a point for which J is minimum, the other components of θ being kept constant. This procedure is repeated a number of times until a joint minimum is reached. The minimum in one direction is reached by using the algorithm of DAVIES, SWANN and CAMPAY (1964). The choice of this procedure and the way it operates for the estimation of θ are discussed in detail elsewhere (DESWYSEN, 1977b).

The parameter values estimated on the basis of the *r-C-R* model shall be indicated by the index W (r_W , C_W , R_W) and the values estimated on the basis of the *r-L-C-R* model by the index D (r_D , C_D , R_D , L_D). The same notation is used for the values of J (J_W or J_D).

The parameters R and C are also computed in the 'usual manner':

(a) R is computed as the ratio between mean aortic pressure and mean flow during the beat; this value is indicated by R_p

(b) C is computed from R_p and the diastolic p_{ao} decay, which is assumed to be a negative exponential function with time constant $\tau = R_p \times C_e$. This time constant is evaluated and an estimate C_e of C is obtained by dividing τ by R_p .

The units used in this study and their S.I. equivalents (given in brackets) are as follows: pressure in mmHg (133.32 Pa) and flow in mls⁻¹ (10⁻⁶ m³ s⁻¹). This gives for the different parameters the following units:

resistance in mmHg s ml⁻¹,
capacitance in ml mmHg⁻¹, and
inductance in mmHg s² ml⁻¹.

3 'Complex model to simple model' approach

We want to use a complex model that simulates the behaviour of the systemic circulation from the point of view of the pressure and flow dynamics, particularly at the root of the aorta. The chosen model is presented in Fig. 2. This model was developed by Professor Rideout's group (CHANG, 1973; CHANG *et al.*, 1974; SIMS, 1972). In this model, the voltage P_1 and the current F_1 are the analogue of p_{ao} and q_{ao} , respectively. The left ventricle is represented by a periodically time varying capacitance $C(t)$. The shape of $e(t)$, the inverse of $C(t)$, has been taken from the work of SUGA *et al.* (1973).

Normal values (DESWYSEN, 1977b) of the parameters of this complex model, corresponding with the systemic circulation of a healthy dog, have been taken from the literature, mainly from Chang's study (1973). These values are listed in Table 1.

The estimation procedure described above has been applied to the determination of the parameter values r_W , C_W , R_W , r_D , C_D , R_D , L_D , R_p and C_e from the pressure P_1 and the flow F_1 simulated by the

complex model. From P_1 and F_1 simulated with the normal values of the parameters (Table 1), the estimated values and the error function are:

r - C - R model: $r_W = 0.158$, $C_W = 1.029$, $R_W = 3.01$, $J_W = 85\ 700$

r - L - C - R model: $r_D = 0.136$, $C_D = 0.726$, $R_D = 2.85$, $L_D = 0.00201$, $J_D = 7\ 880$.

J_W is much greater than J_D . *A priori*, it was expected that the adjunction of one supplementary parameter to the r - C - R model would reduce the error function J . However, it is interesting to note that this reduction amounts to 91%: $(J_W - J_D)/J_W = 0.91$.

Fig. 3 shows the input impedance of the optimum simple models in comparison with the input impedance

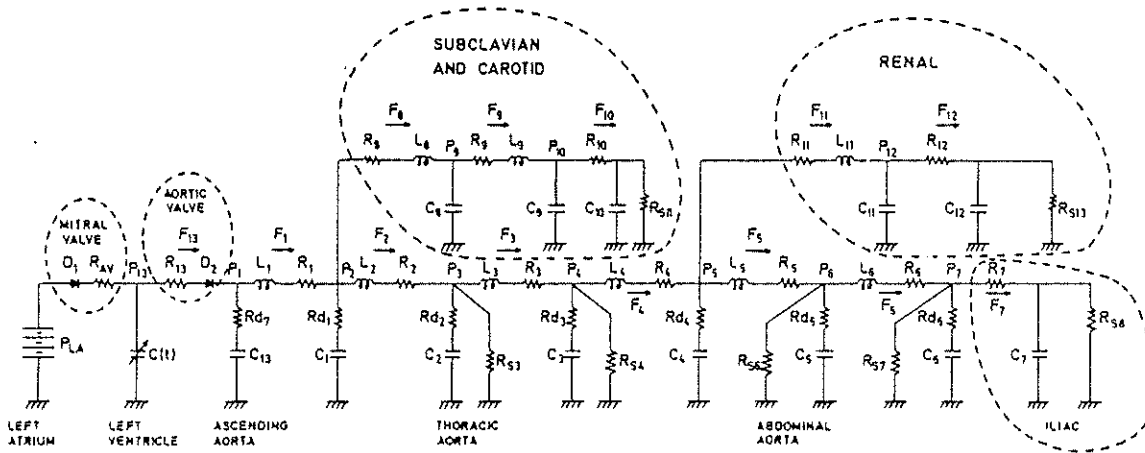


Fig. 2 Complex model of the systemic circulation (Chang, 1973; Sims, 1972)

Table 1. Normal values of the parameters of the complex model

$C_{13} = 0.023$	$R_{d7} = 0.0882$		
$C_1 = 0.087$	$L_1 = 0.0027$	$R_1 = 0.0012$	$R_{d1} = 0.023$
$C_2 = 0.059$	$L_2 = 0.0033$	$R_2 = 0.0019$	$R_{d2} = 0.034$
$C_3 = 0.041$	$L_3 = 0.0041$	$R_3 = 0.0030$	$R_{d3} = 0.049$
$C_4 = 0.028$	$L_4 = 0.0053$	$R_4 = 0.0050$	$R_{d4} = 0.072$
$C_5 = 0.018$	$L_5 = 0.0071$	$R_5 = 0.0090$	$R_{d5} = 0.109$
$C_6 = 0.012$	$L_6 = 0.0101$	$R_6 = 0.0180$	$R_{d6} = 0.171$
$C_8 = 0.004$	$L_8 = 0.0046$	$R_8 = 0.0328$	
$C_9 = 0.002$	$L_9 = 0.0089$	$R_9 = 0.1265$	
$C_{11} = 0.003$	$L_{11} = 0.0160$	$R_{11} = 0.4058$	
$C_7 = 0.133$	$R_7 = 1.351$	$R_{s8} = 10.5$	
$C_{10} = 0.133$	$R_{10} = 0.481$	$R_{s11} = 10.5$	
$C_{12} = 0.133$	$R_{12} = 0.398$	$R_{s13} = 6.6$	
$R_{s3} = R_{s4} = R_{s6} = R_{s7} = 105$			
$R_{s3} = 0.0075$ $R_{AV} = 0.0038$			
$f = 120$ beats/min			
$P_{LA} = 6$ mmHg			
$E_{max} = 6$ mmHg/ml			

Resistances in mmHg s ml^{-1} , capacitances in ml mmHg^{-1} , inductances in $\text{mmHg s}^2 \text{ml}^{-1}$

E_{max} is the maximum of the ratio between ventricular pressure and ventricular volume during the beat (Suga *et al.*, 1973)

dance of the complex model obtained by Fourier analysis of P_1 and F_1 . The moduli of the impedances of the simple models are quite similar to the one of the complex model but the phase of the r - C - R impedance, due to the lack of inductance, is always negative and cannot follow the phase of the complex model.

To derive a physical interpretation for the parameters of the simple models and to check whether these parameters are insensitive to modifications of the state of the heart, a number of different circulatory states are simulated by the complex model. This is achieved by varying the values of several parameters of the complex model, while keeping the other parameters at their normal values.

The different circulatory states simulated are summarised in Table 2. The parameters of the simple models are estimated from P_1 and F_1 in these different circulatory states.

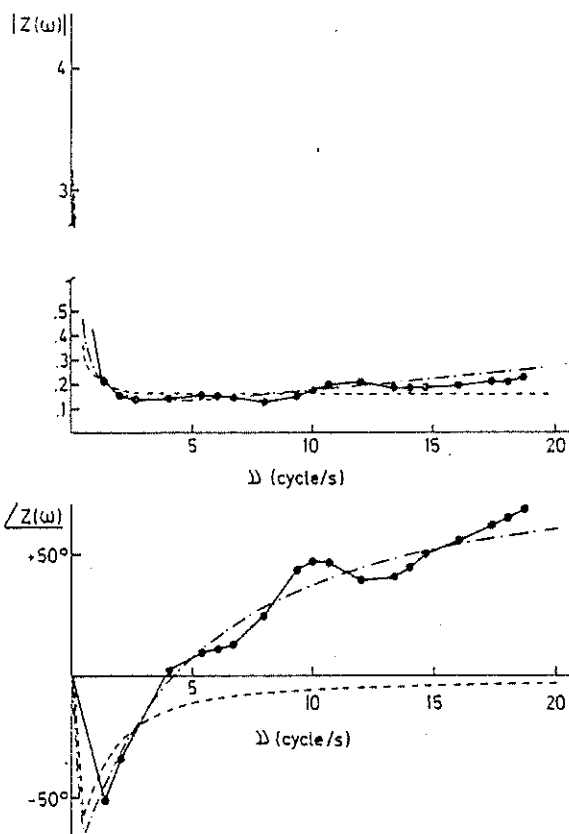


Fig. 3 Input impedance of the complex model (—●—●—), computed by Fourier analysis of P_1 and F_1 (normal values of the parameters at two cardiac frequencies: 120 and 80 beats/min.), in comparison with the input impedances of the optimised r - C - R (---) and r - L - C - R (- · - · -) models

3.1 Validity of the parameter values of the simple models

If the parameter values $r_w, C_w, R_w, r_D, C_D, R_D, L_D, R_p, C_e$ have to be used as indices measuring the afterload, these values must be insensitive to cardiac changes in the complex model. For 11 different simulations for which the contractility, the preload or the cardiac frequency are changed (cases 1 to 11, Table 2), the values of the vascular parameters in the complex model being kept at their normal values, the mean and maximum deviation from the mean of the estimated parameters are

r - C - R model

$$\begin{aligned} \bar{r}_w &= 0.158 - 0.011 (-7\%) \\ \bar{R}_w &= 3.067 + 0.983 (+32\%) \\ \bar{C}_w &= 1.06 + 0.72 (+68\%) \end{aligned}$$

r - L - C - R model

$$\begin{aligned} \bar{r}_D &= 0.135 - 0.005 (-3.7\%) \\ \bar{R}_D &= 2.849 + 0.061 (+2.14\%) \\ \bar{C}_D &= 0.724 + 0.109 (+15\%) \\ \bar{L}_D &= 0.00205 + 0.00025 (+12\%) \end{aligned}$$

Let us note that with the 'usual computation', we obtain the following values:

$$\begin{aligned} \bar{R}_p &= 2.879 - 0.11 (-4\%) \\ \bar{C}_e &= 0.707 + 0.182 (+26\%) \end{aligned}$$

These results show that, to measure the afterload, the parameter values obtained on the basis of the r - C - R model are much less adequate than those obtained with the r - L - C - R model because the former appears to be much more sensitive to cardiac changes.

When pressure and flow are measured *in vivo* at the root of the aorta, it is usually not possible to take measurements at the same level. For this reason, there is often a phase displacement between pressure and flow that is difficult to correct. Therefore, the influence of a time shift between P_1 and F_1 has been tested. We have observed (DESWYSEN, 1977b) that the values r_D, C_D and R_D are less sensitive to time shifts than the values of r_w, C_w and R_w . However, as was expected, L_D is very sensitive: as an example, a time shift of 2 ms between P_1 and F_1 produces a change of 15% in L_D .

The estimation results presented above are obtained from periodical P_1 and F_1 signals. Measured p_{ao} and q_{ao} are not always periodical, for example in cardiac arrhythmias and during transient states caused by changes in contractility, preload or afterload. For this reason, the influence of a non-periodicity of P_1 and F_1 on the parameter values has been tested (DESWYSEN, 1977b). The following

observations have been made:

- (a) As could be theoretically expected, the value of R_p is strongly affected by the nonperiodicity of the signals.
- (b) Nonperiodicity also strongly affects R_W while R_D is relatively insensitive.
- (c) C_D and C_W are only slightly affected, C_D being less influenced than C_W while C_e is highly affected.

3.2 Physical interpretation of the parameters

3.2.1 *Physical meaning of the resistances R and r :*
 For periodical pressure and flow signals, the sum $r + R$ is theoretically equal to the total peripheral resistance R_p , i.e. the ratio between mean pressure and mean flow. Neglecting the value of r (small in comparison with R), R is very close to R_p . The physical meaning of the total peripheral resistance has been sufficiently discussed in the cardiovascular literature and is of current use in clinical practice.

Table 2. Different circulatory states simulated by the complex model

Case number	Circulatory state Modifications of the left ventricle
1	Normal values of the parameters (from Table 1)
2	$P_{LA} = 4$
3	$P_{LA} = 12$ changes in preload
4	$E_{max} = 4$
5	$E_{max} = 12$ changes in contractility (Suga <i>et al.</i> , 1973)
6	$E_{max} = 12$ $P_{LA} = 12$ change in preload and contractility
7	$f = 180$
8	$f = 150$
9	$f = 90$
10	$f = 80$ changes in cardiac frequency
11	$f = 60$
Modifications of the systemic vascular bed	
12	all the peripheral resistances $R_{st, i}$ multiplied by 2
13	1.5
14	0.75
15	0.5
16	all capacitances C_i divided by 1.25
17	1.15
18	0.75
19	0.70
20	0.65
21	0.60
22	0.55
23	0.50
24	C_i with $i = 1$ to 6 and 8, 9, 11 divided by 1.25
25	0.75
26	0.50
27	C_7, C_{10}, C_{12} (terminal compliances) divided by 1.25
28	0.75
29	0.50

For these reasons, we shall not discuss specifically the meaning of R .

The resistance r is generally considered as the model representation of the characteristic impedance of the aorta where pressure and flow are measured (WESTERHOF, 1978). This means that, in this approximation of the complex model by a simple r - C - R or r - L - C - R model, r must be correlated with $Z_0 = \sqrt{L_1/C_1}$ (WESTERHOF, 1968). Fig. 4 shows the relations between the estimated values r_D , r_W and the complex model value Z_0 for eight simulations corresponding to various values of Z_0 . As it can be seen, there is a good relation between r_D or r_W and Z_0 .

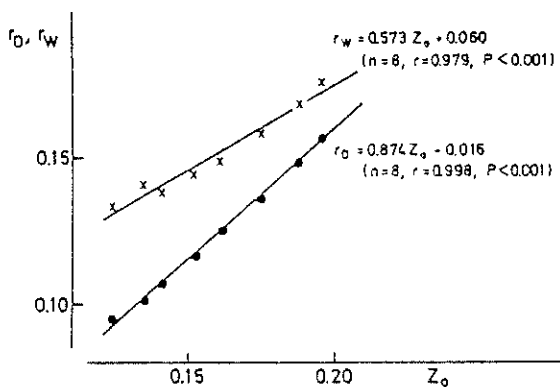


Fig. 4 Relation between r_W (\times), r_D (\bullet) and Z_0 when Z_0 is changed in the complex model

r_D = value of the characteristic impedance estimated on the basis of the r - L - C - R model

r_W = estimated value on the basis of the r - C - R model

Z_0 = value of the characteristic impedance of the aortic root in the complex model at the level of P_1 and F_1 ($Z_0 = \sqrt{L_1/C_1}$)

3.2.2 Physical meaning of C : C is usually considered as the total arterial compliance. This means that in the case of the approximation of the complex model by the simple model, C should be related to the sum ΣC of the capacitance of the complex model downstream of the measure of P_1 and F_1 .

Fig. 5 shows the estimated values C_D , C_W and C_e as a function of the values ΣC ($\Sigma C = \Sigma C_i$ with $i = 1$ to 12) for the 15 simulations for which the capacitances C_1 are fixed at various values. The correlation coefficient between C_e and ΣC is much smaller than the one between C_D and ΣC . There exists a good relation between C_D and ΣC . However, the values C_D are different from the 'actual' values ΣC , but the C_W values differ much more from the actual ΣC values.

3.3 Conclusions of the model to model approach

The results discussed above have shown that the r - L - C - R model is much more adequate than the r - C - R model to represent the relation between the fictitious aortic pressure and flow simulated by the complex model. The parameter values R_D , r_D , C_D estimated from P_1 and F_1 during one beat, on the basis of the r - L - C - R model, provide a valid measure for some characteristics of the state of the afterload in the complex model. The usual physical meanings of r_D , R_D and C_D have therefore been confirmed. On the other hand, these conclusions obviously do not apply to the R_W and C_W values estimated on the basis of the r - C - R model, these values being highly affected by heart function changes in the complex model.

It was indicated above that L_D is strongly affected by a time shift in the pressure and flow signals. For this reason, we do not want to give any physical

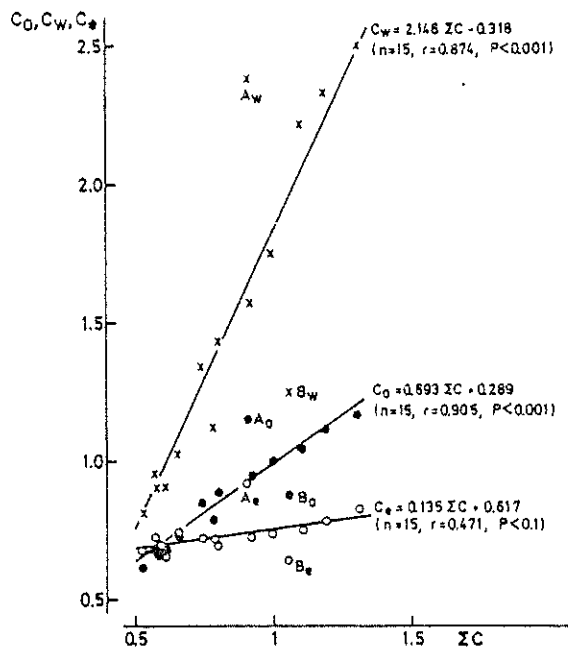


Fig. 5 Relation between C_D (\bullet), C_W (\times), C_e (\circ) and ΣC when capacitances C_i are changed in the complex model.

C_D = value estimated on the basis of the r - L - C - R model

C_W = value estimated on the basis of the r - C - R model

C_e = value computed from R_p (total peripheral resistance = \dot{P}_1/\dot{F}_1) and the diastolic decay of P_1

ΣC = sum of the capacitances of the complex

$$\text{model} \left(\Sigma C = \sum_{i=1}^{12} C_i \right)$$

interpretation to the values of L . We consider L as an additional parameter which only allows us to obtain a better representation of the input impedance of the complex model as well as more correct values for the parameters r , R , C .

4 Experimental approach

4.1 Method

Experiments have been performed on three dogs (G, I, J). After anaesthesia by intravenous injection of sodium pentobarbital (25 mg/kg), the dog is placed under artificial ventilation. One catheter tip pressure transducer (PC 470, Millar Instr., Inc., Houston) introduced through the left femoral artery is located at the root of the aorta to measure p_{ao} , the instantaneous pressure. Another identical catheter introduced through the right femoral artery is placed in the aorta, a few centimetres downstream from the tip of the first catheter to measure $p_{ao, th}$, the instantaneous pressure in the thoracic aorta. After thoracotomy, an electromagnetic cuff-flow

probe (SEM 275, Feltham, England) is surgically implanted around the root of the aorta for measuring the instantaneous blood flow q_{ao} at this level.

Two small ultrasonic transducers (o.d. 5 mm; thickness 2 mm) are fixed at two diametrically opposite sites on the external wall of the thoracic aorta where $p_{ao, th}$ is monitored. Measurement of the transit time of the ultrasonic wave gives a measure of $\phi_{ao, th}$, the instantaneous external diameter of the thoracic aorta.

These measurements, illustrated in Fig. 6, are first recorded on analogue tape (Precision Instr., mod. 6000) and then sampled every 2 ms and stored on digital tape. These signals are transferred in a file on the disk of the computer (Hewlett-Packard, 2100A) to be used by the estimation program.

The circulatory variables are recorded during control steady states, during the administration of drugs (angiotensin and sodium nitroprusside) producing vascular effects and during infusion or withdrawal of blood (and/or physiological solution). By increasing the smooth muscle tone of the vessels, angiotensin (GILLESPIE *et al.*, 1972; O'ROURKE *et al.*,

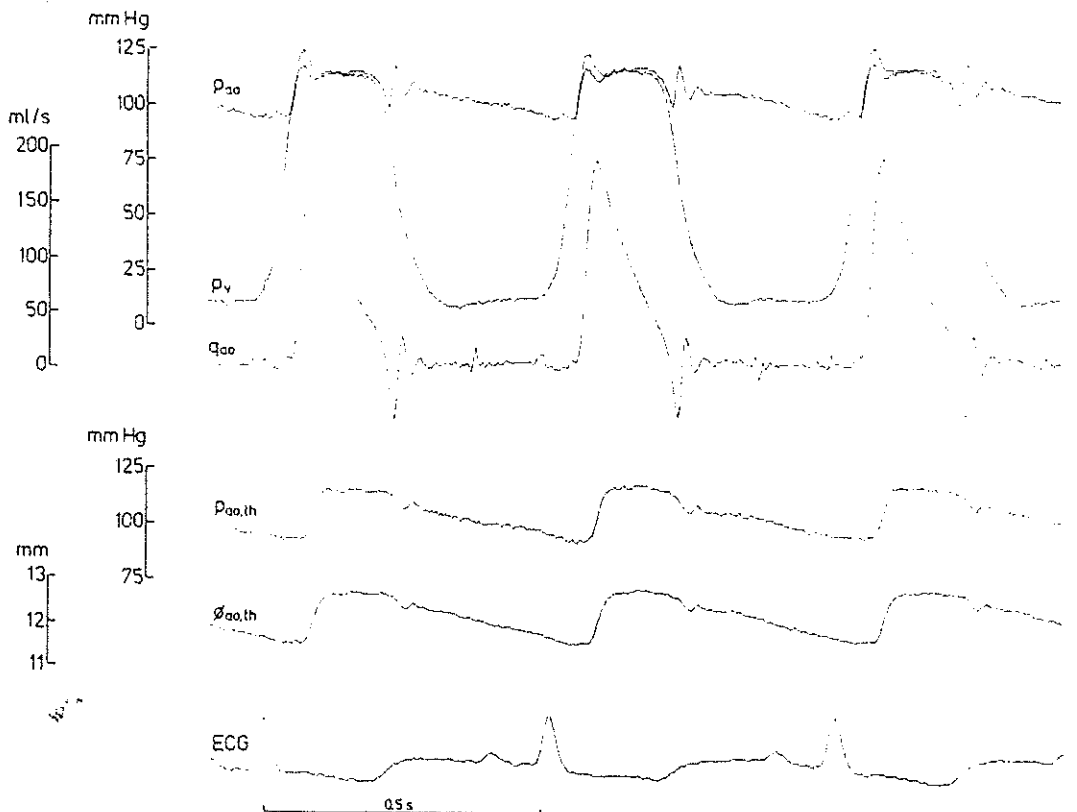


Fig. 6 Examples of measurements recorded on dog G in control state at the beginning of the experiment:

p_{ao} = root aorta pressure

p_v = left ventricular pressure

q_{ao} = root aorta flow

$p_{ao, th}$ = thoracic aortic pressure

$\phi_{ao, th}$ = thoracic aortic diameter

1972) produces an increase in vascular resistance and a decrease in vascular compliance. This decrease in vascular compliance results also from the non-linearity of the mechanical properties of the arterial wall (BERGEL); indeed, an increase in pressure produces an increase in the stiffness of the vascular wall. On the other hand, sodium nitroprusside (PALMER and LASSETER, 1975) is known to produce opposite effects on vascular properties. Angiotensin and sodium nitroprusside are used specifically to produce their expected effects on the resistance and compliance characteristics of the systemic arterial

bed. Blood infusion or withdrawal were performed to change the mean pressure level (mainly by changing the venous return) without directly acting on the vascular properties.

Groups of ten successive beats (steady states) are selected at different levels of pressure and flow induced by the circulatory interventions. For each beat, the parameters of the simple models ($r-L-C-R$ and $r-C-R$) are estimated by the procedure described above. Means and standard deviations of the estimated parameter values are calculated for each group of ten beats.

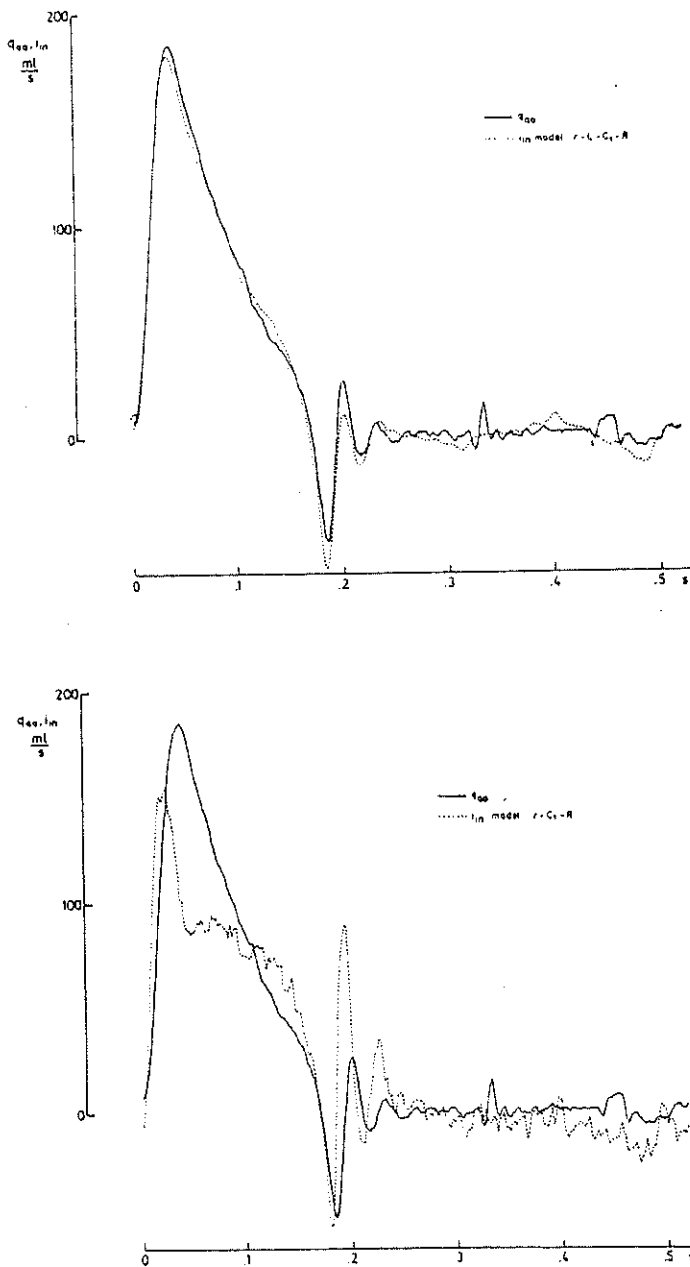


Fig. 7 Comparison between q_{a0} , the measured aortic flow, and i_{in} , the flows reproduced by the simple optimised models $r-L-C-R$ and $r-C-R$, respectively (Dog G: one beat in control state at the beginning of the experiment)

5 Results

5.1 Validity of the $r-L-C-R$ model

Just as for the model to model approach, we study whether the $r-L-C-R$ model compared with the $r-C-R$ model, gives a better representation of the relation between p_{ao} and q_{ao} measured at the root of the aorta. For one beat recorded on dog G at the beginning of the experiment, the error cost J is

$$r-L-C-R \text{ model: } J_D = 7\,960$$

$$r-C-R \text{ model: } J_W = 209\,000$$

This difference between the performance of the two models is illustrated by Fig. 7 that gives, for the same beat, the true q_{ao} in comparison with the flows i_w simulated by the two optimum simple models. For this beat also, Fig. 8 shows the input impedance of the systemic arterial bed (obtained by Fourier analysis) in comparison with the input impedances of the optimised simple models. These two figures, as well as the values of J_W and J_D , clearly show that

the $r-L-C-R$ model gives a more adequate representation of the relations between p_{ao} and q_{ao} than the $r-C-R$ model. This finding together with the conclusions of the simulation approach justifies the fact that we shall from now on consider only the values r_D , R_D and C_D obtained with the $r-L-C-R$ model.

It is also important to note that the standard deviations of the values r_D , R_D and C_D obtained for ten successive beats (in steady state) are generally small; for example, the values obtained for the first ten beats analysed in control condition at the beginning of the experiment on dog G are

$$\bar{r}_D = 0.087 \pm 0.006$$

$$\bar{R}_D = 3.31 \pm 0.09$$

$$\bar{C}_D = 0.582 \pm 0.029$$

$$\bar{L} = 0.00147 \pm 0.00006$$

5.2 Physical interpretation of the parameters

5.2.1 a/C_D . The model-to-model approach has shown that the correlation between C_D and ΣC is good; C_D can therefore be considered as a measure of the total systemic arterial compliance. Because the total compliance of the systemic arterial bed cannot be measured, it is not possible to correlate C_D with an objective measurement and to verify quantitatively whether it has been given a correct interpretation. Nevertheless, a qualitative verification of this meaning can be made by observing whether C_D varies in the direction of the expected changes in compliance following the administration of angiotensin and sodium nitroprusside.

Table 3 gives the mean values of C_D on ten beats recorded during the administration of angiotensin in comparison with the mean values on ten beats before the administration of the drug for each of the three dogs. Similarly, Table 4 gives the mean values of C_D for the same number of experiments with sodium nitroprusside. Table 3 shows a clear decrease in C_D during each angiotensin administration, while Table 4 shows a clear increase in C_D caused by sodium nitroprusside. These changes in C_D are in concordance with the expected changes in total systemic arterial compliance following the administration of the two drugs.

5.2.2 b/r_D . From the work of RIDEOUT and DICK (1967), we can write that the characteristic impedance of a vessel is proportional to:

$$Z_0 \div \sqrt{(h/r_i)} \sqrt{(E_t/r_i)} \dots \dots \dots (6)$$

where h_i , r_i , E_t are, respectively, the thickness of the wall, the radius and the Young's modulus of the wall of the vessel. Moreover, if, around a pressure p which corresponds to a radius r_i , a segment of vessel is submitted to a change in pressure Δp , the radius is changed by Δr . Then an incremental

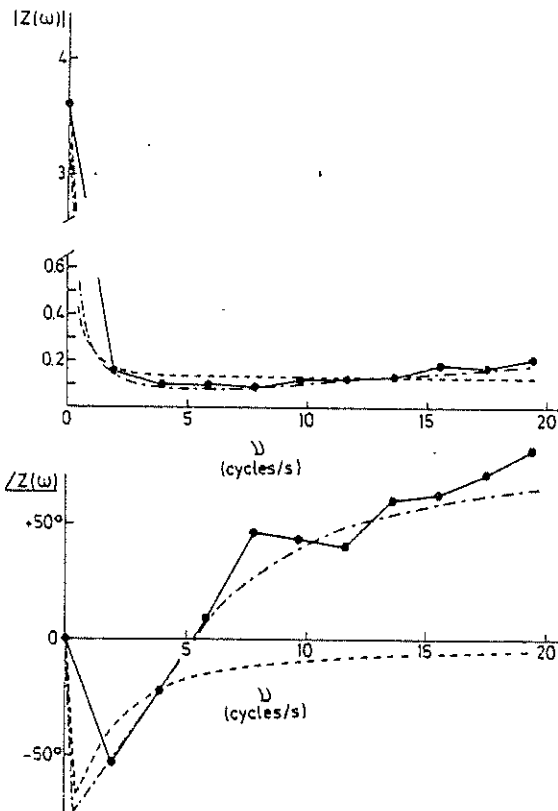


Fig. 8 Comparison between the input impedance of the systemic arterial bed (●—●—●—), computed by Fourier analysis of p_{ao} and q_{ao} with the input impedance of the optimised $r-L-C-R$ (— · — · —) and $r-C-R$ (— · —) models (Dog G: the same beat as for Fig. 7)

Young's modulus can be approximated (BERGEL, 1961) by

$$E_t = (\Delta p / \Delta r) (r_t^2 (1 - \sigma^2) / h_t) \quad (7)$$

where σ is the Poisson's ratio.

From eqns. 6 and 7, the following relation can be written:

$$Z_0 \div \sqrt{(\Delta p_{ao, th} / \Delta \phi_{ao, th})} / \bar{\phi}_{ao, th}^{1.5} \text{ def } r_m \quad (8)$$

where $\Delta p_{ao, th} / \Delta \phi_{ao, th}$ is the ratio between the maximum changes in $p_{ao, th}$ and in $\phi_{ao, th}$ during one beat, $\bar{\phi}_{ao, th}$ is the mean $\phi_{ao, th}$ during the beat. The mean values of $\bar{\phi}_{ao, th}$ and $\Delta p_{ao, th} / \Delta \phi_{ao, th}$ on ten beats are computed for 42 groups of ten beats on dog C at different levels of pressure and flow resulting from the administration of angiotensin and sodium nitroprusside and from the infusion or withdrawal of blood (and/or physiological solution).

For each group of beats, a value of r_m can therefore be computed. Fig. 9 shows the correlation between r_m and the mean values of the estimated parameter r_D on the corresponding ten beats. As it can be seen, there is a good correlation between r_D and r_m .

It was not possible to compute correlations between r_D and r_m for dogs I and J because r_m was little affected by the circulatory interventions on these dogs. This results from the higher mean aortic pressure levels during most of the circulatory interventions on dogs I and J than on dog G. As was indicated by Cox (1975), the characteristic impedance is approximately constant between 80 and 150 mmHg. For our dogs, we have observed that r_m was little affected above about 100 mmHg.

6 Discussion and conclusions

Measuring the afterload has become an important

Table 3. Changes in C_D , R_D and p_{ao} following the administration of angiotensin

Dog	Doses (μ g/min)	$C_D (\pm SD)$		$R_D (\pm SD)$		p_{ao}	
		Control	Angiotensin	Control	Angiotensin	Control	Angiotensin
G	5.7	1.05 \pm 0.07	0.31 \pm 0.05	2.9 \pm 0.5	7.7 \pm 0.7	99	192
	5.7	1.03 \pm 0.05	0.38 \pm 0.01	1.8 \pm 0.01	4.6 \pm 0.1	99	170
I	11.4	0.52 \pm 0.01	0.25 \pm 0.01	3.7 \pm 0.1	7.9 \pm 0.3	134	180
	11.4	0.78 \pm 0.04	0.28 \pm 0.01	3.9 \pm 0.2	10.9 \pm 0.3	111	179
J	5.7	0.72 \pm 0.03	0.22 \pm 0.01	5.3 \pm 0.4	11.9 \pm 0.3	112	187
	23.0	0.78 \pm 0.03	0.32 \pm 0.01	3.7 \pm 0.2	5.1 \pm 0.2	102	157

Mean values of C_D , R_D (\pm standard deviation) and p_{ao} on ten beats before and during intravenous perfusion of angiotensin for the three dogs (G, I, J). The drug has been administered two times for each dog

Table 4. Changes in C_D , R_D and p_{ao} following the administration of sodium nitroprusside

Dog	Doses (μ g/min)	$C_D (\pm SD)$		$R_D (\pm SD)$		p_{ao}	
		Control	Nitroprusside	Control	Nitroprusside	Control	Nitroprusside
G	570	0.65 \pm 0.01	0.79 \pm 0.03	2.8 \pm 0.1	1.3 \pm 0.1	114	75
	570	0.85 \pm 0.01	1.09 \pm 0.05	2.1 \pm 0.1	1.2 \pm 0.1	83	60
I	570	0.36 \pm 0.02	0.88 \pm 0.09	3.9 \pm 0.1	1.1 \pm 0.1	163	77
	570	0.46 \pm 0.01	1.19 \pm 0.10	4.4 \pm 0.3	2.1 \pm 0.1	137	73
J	570	0.45 \pm 0.01	1.41 \pm 0.11	3.3 \pm 0.1	2.1 \pm 0.2	130	84
	570	0.53 \pm 0.03	0.90 \pm 0.08	4.1 \pm 0.1	2.4 \pm 0.2	101	74

Mean values of C_D , R_D (\pm standard deviation) and p_{ao} on ten beats before and during intravenous perfusion of sodium nitroprusside for the three dogs (G, I, J). The drug has been administered two times for each dog

objective for many people as well in research as in medical practice, e.g. in a coronary care unit (CHATTERJEE and SWANN, 1973). We have studied the feasibility of measuring the afterload by the estimation of the parameters of a simple model.

The r - L - C - R model is shown to perform significantly better than the r - C - R model for the representation of the relation between instantaneous pressure and flow at the root of the aorta and therefore of the afterload. This is also shown by the complex model to simple model approach and by the experimental approach. This model has been rarely used in the cardiovascular literature. WESTERHOF (1968), referring to LANDES (1943), has computed the input impedance of the r - L - C - R and has concluded that the addition of an inductance L gives a completely inadequate model; the reason for this is that he used a too large value for L . Recently, WESTERHOF *et al.* (1977) have compared in one figure the input impedance of the r - L - C - R

with the mean input impedance of the systemic arterial bed for 7 dogs but they make no comment; their figure shows good agreement between the actual impedance and the one of the r - L - C - R model; in fact in this work they have computed the impedance of the r - L - C - R model with a value for L of the same order of magnitude as the values we have estimated. PIENE (1976) has used the r - L - C - R model to approach the input impedance of the pulmonary circulation and showed that this model performs well; in his work, he did not try to give any physical meaning for L . In most of the published literature R is computed as $R_p = \bar{p}_{ao} / \bar{q}_{ao}$. C is obtained from R_p and from the exponential decay of p_{ao} during diastole, and r is computed as the mean of the input impedance modulus between about 3 and 10 Hz (NICHOLS *et al.*, 1977; WESTERHOF *et al.*, 1973). In our study, the characteristic resistance r , the total systemic arterial compliance C and the total peripheral resistance R are computed using a different method. We compute r , C and R by the direct adaptation of the r - L - C - R model to the instantaneous p_{ao} and q_{ao} . Our results show that this procedure gives parameter values with physical meanings. The simulation approach has given a quantitative validation of the meanings of r_D and C_D while the classical interpretation of R_D as an approximation of the total peripheral resistance has been confirmed. In addition, the experimental approach has given a quantitative validation for r_D and a qualitative validation for C_D . The number of experimental results we have presented is relatively limited, but we think that the concordance between the results of the two approaches confirms the meanings previously proposed (WESTERHOF, 1968) for r and C : r_D can be considered as a measure of the characteristic impedance of the aorta near the measurement site of p_{ao} and q_{ao} , while C_D is a measure of the total compliance of the systemic arterial bed.

Our results allow us to introduce here a complementary remark concerning the determination of cardiac output by pulse-pressure methods. In these methods, the systemic arterial bed is implicitly (BOURGEOIS *et al.*, 1976)* or explicitly represented by a simple model of the windkessel type. In a calibration step, the instantaneous pressure p_{ao} and the mean flow are measured and used to compute the compliance C of the model; after that, assuming that this compliance is constant, its value is used to estimate the stroke volume from the instantaneous pressure only. Because the total compliance can change strongly (Tables 2 and 3), it is difficult for these methods to give accurate results. Note that our experimental results are in opposition to those from BOURGEOIS *et al.* (1974) who affirm that C is constant even after angiotensin administration.

* Note that the 'new' formula presented by Bourgeois *et al.* (1976) can be directly obtained by integration of the equation $i_{in} = C dU_{in}/dt + U_{in}/R$ of the windkessel model during the ejection.

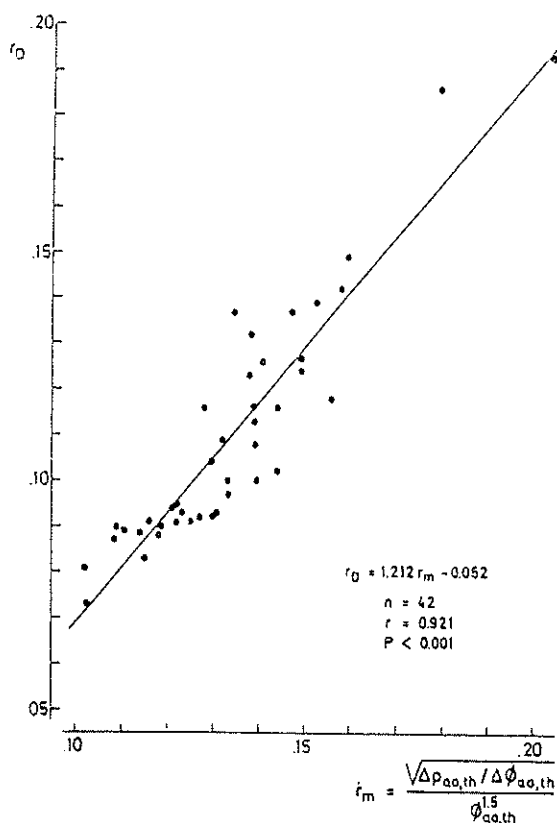


Fig. 9 Relation between the estimated characteristic impedance r_D and the measured characteristic impedance r_m for 42 groups of ten beats recorded on dog G at different levels of pressure and flow following the circulatory interventions. Each value of r_D and r_m is a mean value computed on ten beats. The regression line is also illustrated

The input impedance of the $r-L-C-R$ model gives a good approximation of the input impedance of the systemic arterial tree; this has been shown by the two approaches presented above. It must be noticed also that the input impedance of the $r-L-C-R$ model is qualitatively similar to the recently published impedance curves of the systemic circulation in man (NICHOLS *et al.*, 1977). The scatter on the input impedance curves (computed by Fourier analysis of p_{ao} and q_{ao}) being generally large, and the input impedance representing by definition a system of infinite order, its physical interpretation is usually restricted to two quantities (at least for the systemic circulation): the total peripheral resistance and the characteristic impedance. Because the total compliance C mainly determines the input impedance at very low frequencies (0–2 Hz), this compliance cannot be directly deduced from the input impedance which does not give information between 0 Hz and the first harmonic. On the other hand, the identification of the $r-L-C-R$ model gives directly and simultaneously valid estimation of the total peripheral resistance, of the characteristic impedance and of the total compliance. For these reasons, to characterise the systemic arterial bed as ventricular afterload, the identification of the $r-L-C-R$ model seems to be more advisable than the computation of the input impedance from pressure and flow harmonics. Moreover, as it has been shown above, the estimation of r , R , C on the basis of the $r-L-C-R$ model gives better estimates of these parameters than if estimated on the basis of the $r-C-R$ model or by the usual computation (R_p , C_c). In conclusion, we suggest that the estimated parameters of the $r-L-C-R$ model should be used for future studies on the left ventricular afterload.

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