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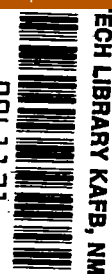
THE STUDY OF HUMAN VENOUS SYSTEM DYNAMICS USING HYBRID COMPUTER MODELING

by

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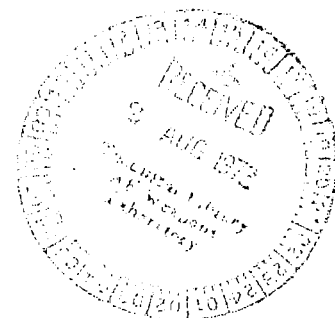
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15. Supplementary Notes

16. Abstract

A computer-based model of the cardiovascular system was created emphasizing effects on the systemic venous system. Certain physiological aspects were emphasized: effects of heart rate, tilting, changes in respiration, and leg muscular contractions. The results from the model showed close correlation with findings previously reported in the literature.

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THE STUDY OF HUMAN VENOUS SYSTEM DYNAMICS USING HYBRID COMPUTER MODELING.

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Introduction:

Simulation studies of the venous system have lagged behind similar studies of the arterial system, largely because of difficulties due to the presence of nonlinearities in the veins, resulting from the phenomenon of collapse and the effects of venous valves. Time-varying changes in parameters representing effects of venous tone and peripheral resistance add to the difficulties of simulation. However, it is precisely these complexities which make simulation necessary if the interacting effects in response to various inputs are to be understood. Early attempts at system simulation, including the veins,were made in which either linearity was assumed[1], or in which some nonlinearities were included, but in which the modeling was not very detailed[2]. A simulation study was begun at Wisconsin[3,4], therefore, with the initial objective of developing a model which would include the following features:

1) Simulation of the fluid mechanics of the complete cardio-vascular loop, with greatest detail in the systemic venous system.

2) Inclusion of the effects of venous collapse in the larger vessels[5].

3) Inclusion of venous valves in the leg veins.

4) Inclusion of a control loop for heart rate and strength of contraction control[6,7] as determined by carotid pressure. Also, atrial as well as ventricular pumping was used in the modeling of the heart.

5) Inclusion of effects on the venous system of changes in pressure due to breathing (in the thorax), abdominal contractions[8], and leg muscle contractions.

6) Inclusion of venous tone control effects, based largely on the work of Alexander[9,10], as well as peripheral resistance control based on work by Sagawa[11] and Dick[7].

7) Inclusion of the effects of gravitational forces in such a way that the effects of tilt-table tests as well as acceleration changes could be studied.

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After the first reports[3,4] on the studies made with the model, further detailed studies were made in which an improved model was used which had eight parallel pathways (see Figs. 1 and 2) in the systemic circuit, and in which certain overload problems were overcome to permit better simulation of the Valsalva maneuver. Some of the response curves were re-run, and a number of new studies of the effects of valve defects etc. were made. Descriptions of only a few of these new results have been published[12,13], and this report will serve to give a complete picture of studies made with the model at various times during the last 12 months throughout which it was kept in operating condition.

RESULTS:

New results obtained with the model of Fig. 2 for a Valsalva maneuver in horizontal position are shown in Fig. 3 (and repeated for a greater paper speed in Fig. 4). They compare closely with reported dynamics. The cardiac output curves for the left and right ventricles are particularly interesting. Figure 5 shows a number of venous variables during the same maneuver, not previously reported. Note the comparison between the velocity W_{AVC}[*] in the abdominal vena cava from the model, and the IVC velocity reported by Wexler (shown in Fig. 3).

Fig. 6(a) shows a Valsalva transient in the head-up position, showing interesting differences (as compared with the horizontal position) in heart rate, and in P_{LOLV}, the lower leg vein pressure. Figure 6(b) shows still greater heart rate change for a Valsalva in the horizontal position after a hemorrhage of about 10 % of total blood volume, while P_{LOLV} is sharply reduced.

Figures 7 and 8 show Müller maneuver results (for horizontal position). This maneuver was achieved by making intrathoracic pressures $P_{ITH}=-10$ mmHg and intra-abdominal pressure $P_{IAB}= +10$ mmHg for about 12 sec. Note the increase in cardiac output and decrease in heart rate in this maneuver, which is essentially the opposite of the Valsalva. The great increase in W_{AVC} (abdominal vena cava velocity) in Fig. 7 shows that this vein is close to collapse during this maneuver.

Figure 9 shows the Müller maneuver in the head up position, with resultant increase in P_{LOLV} and reduced change in right atrial pressure P_{RA}. Figure 10 is for the same condition, but with changes in P_{ITH} and P_{IAB} doubled.

[*]See glossary of symbols

Figure 11 shows the result on venous variables of a normal tilt (from horizontal to vertical position and return) followed by a second similar maneuver with 500 cc of blood removed. It can be seen by examining F_{AVC} that a much longer time is needed to achieve steady state in the head-up position. Also note that pulsations more completely disappear from the jugular vein as shown by its pressure trace, P_{JV}.

Figure 12 taken at faster paper speed, shows various venous variables under normal respiration in the horizontal position.

In Figure 13 a slightly different intrathoracic pressure wave due to breathing was used following a suggestion by Dr. W.B. Youmans. This more correct pressure pattern for P_{ITH} (in place of a rectified half-sine wave) has not greatly affected the venous waveforms.

The success of this model in reproducing the responses corresponding to several standard maneuvers in considerable detail encouraged us to try further experiments with the model as suggested by various physiologists. The remaining figures show the effects of various heart defects on response to tilt-table and Valsalva maneuvers.

Figure 14 again shows normal system responses for a Valsalva transient(V) followed by a tilt (T) to the head-up position and back. Figure 15 shows the effect of the introduction of an aortic valve insufficiency with a back resistance[*] of R_{ai} = 1000 at a point in time marked by an arrow at the bottom of the figure. Note that this defect, which gives the usual increase in arterial pulse pressure, results in very little change in the response to Valsalva and tilt. Even with a more severe aortic valve insufficiency of back resistance R_{ai} ▫ 100 in Fig. 16, with considerably increased pulse pressure variations these major transients are not much affected.

Figure 17 shows the effects of a mitral valve insufficiency (R_{mi} = 500). Here the effects on pulsatile waveforms are small, but some slight effect on the heart rate transient as compared with the normal response in Fig. 14 can be seen. Much the same result is obtained (Valsalva only) for a mitral valve back resistance of 200 (Fig. 18).

In Figure 19 the effects of the introduction of an aortic valve stenosis of resistance R_{as} ▫ 133 are shown. This defect causes increased left ventricular pressure, but otherwise little change in pulsatile response. In Valsalva and tilt the heart rate transient shows most variation from normal, with little downswing but a considerable increase.

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[*]Resistance values in this paper are c.g.s. units of $\dfrac{dynes/cm^2}{ml./sec.}$

Two defects were introduced simultaneously in Fig. 20, aortic stenosis (R_{as} = 133) and mitral insufficiency (R_{mi} = 100). Here a number of differences in response to a Valsalva maneuver may be seen if this result is compound with the normal results of Fig. 14. Heart rate shows no negative swing, and has an even larger increase than in Fig. 19. However all transient changes appear to be slowed, as can be seen most clearly by examining the left ventricular pressure.

In Figure 21 two defects are again introduced simultaneously, this time aortic stenosis (R_{as} = 133) and aortic value insufficiency (R_{ai} = 1000). Although these defects result in a considerable change in ventricular and aortic pressure waves, the Valsalva and tilt transients do not differ greatly from normal system responses.

In Figure 22 the effects of Valsalva and tilt in a system with an atrial septal defect, first of resistance R_{asd} ▫ 100 then of resistance R_{asd} ▫ 10, are shown. Here some changes in transient response may be noted — pulsatility tends to disappear from the R_{RV} trace during Valsalva, and the transient changes adjust more rapidly than in the normal case. Right ventricular output drops by more than a normal amount and left ventricular output by less, showing that the shunt is effectively from right to left.

If a ventricular septal defect is introduced (first for R_{vsd} = 10000, then for R_{vsd} = 1000), then as shown in Fig. 23 the main effect is an increase in right ventricular pulse pressure; transient waveforms are not much affected. Right ventricular output is now larger than left ventricular output because of left-to-right shunt through the ventricular septal defect. If R_{vsd} = 500, as in Fig. 24, it can be seen that various effects are greater. Here it is of interest to examine the values of right ventricular output before the defect is introduced, after it is introduced, and at the end of the Valsalva maneuver; the values are 80, 123 and 80 ml/sec. Corresponding values for the left ventricle are 11, 68 and 35. Thus with a large ventricular septal defect the right ventricular output is greatly reduced during Valsalva, but the proportional change is less for the right ventricle (80/123) than for the left (35/68). (The slight difference in normal left and right ventricular outputs results from dome difficulty with gating of the output flow pulses, but does not seriously affect results).

Figure 25 shows the same transients for a still larger ventricular septal defect of R_{vsd} ▫ 250.

Discussion:

Mathematical models of the cardiovascular system of considerable
complexity may be set up to run in real time on a modern hybrid
computer. Such simulations are particularly interesting if the
nonlinear and time-varying components of the heart and venous
system are included in the model. A first attempt has been made
to do this kind of modeling, and verification of the model has
been based on waveform comparison and comparison of response
to Valsalva, Müller and tilt-table disturbances of the model with
those in a normal human, as reported in the literature.
Parameter estimation schemes[14],[15] may be used in the future
to more closely match a model to an individual from certain data
is available.

This model has been used to study the effects of certain heart
defects on the circulation during transients (Valsalva and tilt)
which have a major effect upon the systemic venous blood pressure
and flow.

It should be emphasized that results obtained with the model
are only as good as the structure and parameter values of the
model permit. However, it is quite possible to improve the model
in form by adding components to it as needed, even to the extent
of adding mass transport circuits[16], which can be adapted to sys-
tems with collapsing vessels[17]. The possibility of improvements
in parameter estimation has been referred to above. The model
reported here should be regarded as an elementary framework which
may be adapted or augmented for future modeling studies: directions
which these changes take should be guided much more by physiolo-
gists than engineer-modelers.

Acknowledgements:

GLOSSARY OF SYMBOLS

HSV	- Head small veins
JV	- Jugular vein
SVC	- Superior Vena Cava
UPC, LOC	- Upper and lower carotid artery
RA, LA	- Right and left atrium
LA, LV	- Right and left ventricle
PA, PC, PV	- Pulmonary artery, capillaries and veins
AsA	- Ascending aorta
TVC, ThA	- Thoracic vena cava and aorta
CSMV, CSMA	- Celiac, superior mesenteric vein and artery
PORV	- Portal vein
RENV, RENA	- Renal veins and artery
AVC, AbA	- Abdominal vena cava and aorta
IMV, IMA	- Inferior mesenteric vein and artery
AbSKV	- Abdominal skin venules
FEV, FA	- Femoral vein and artery
IILVE	- Internal Iliac venules
UPLV, UPLA	- Upper leg veins and arteries
UPLVE	- Upper leg venules
LOLV, LOLA	- Lower leg veins and arteries
LOLVE	- Lower leg venules
ITh	- Intrathoracic
IAb	- Intra-abdominal

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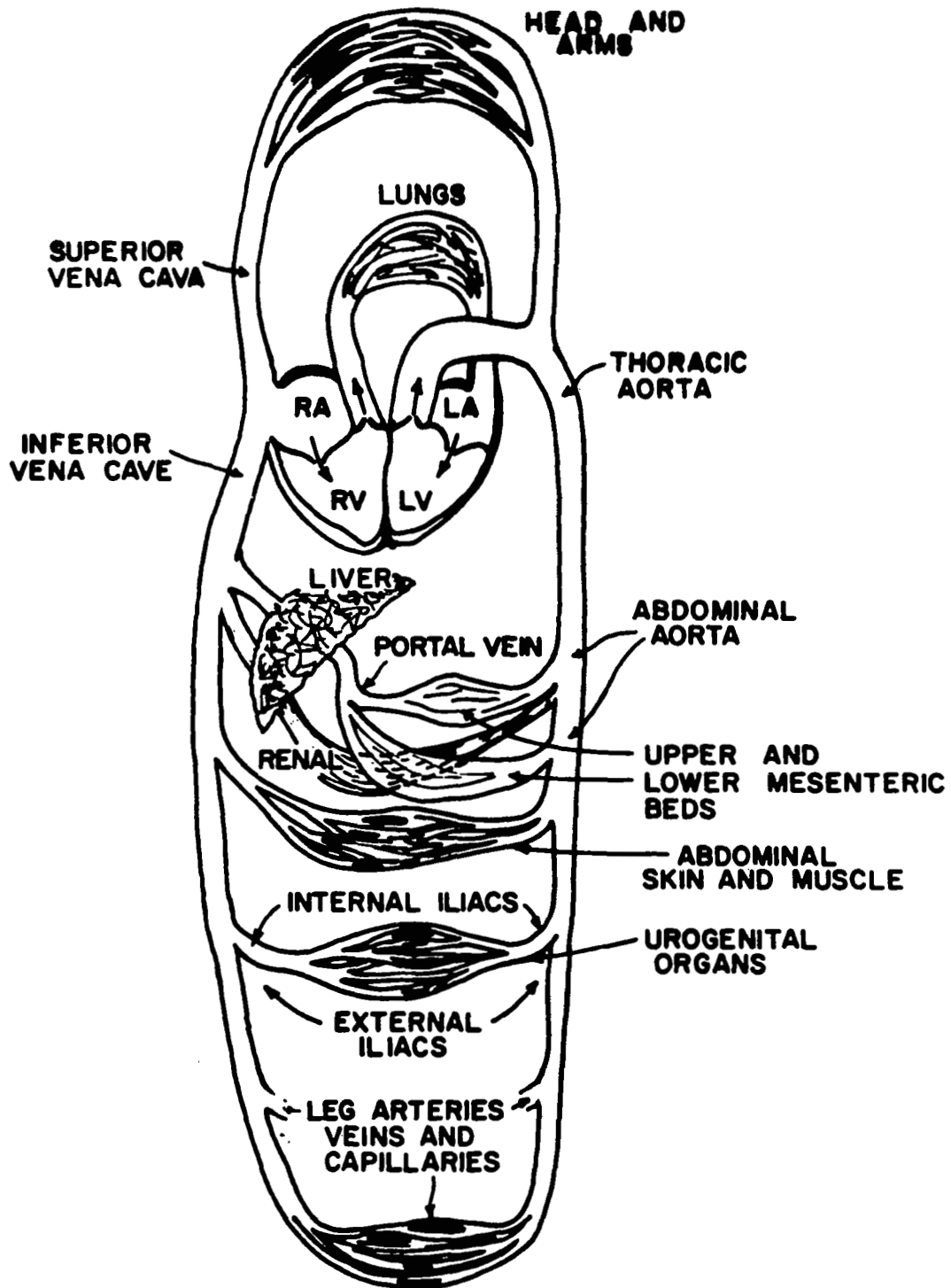
Fig.1. Simplified diagram showing the main pathways in the human cir-
culatory system incorporated into the model.

Fig.2. Model of the human cardiovascular system used in these studies
(circuits for control of heart, venous tone and peripheral resis‧
tance are not shown.
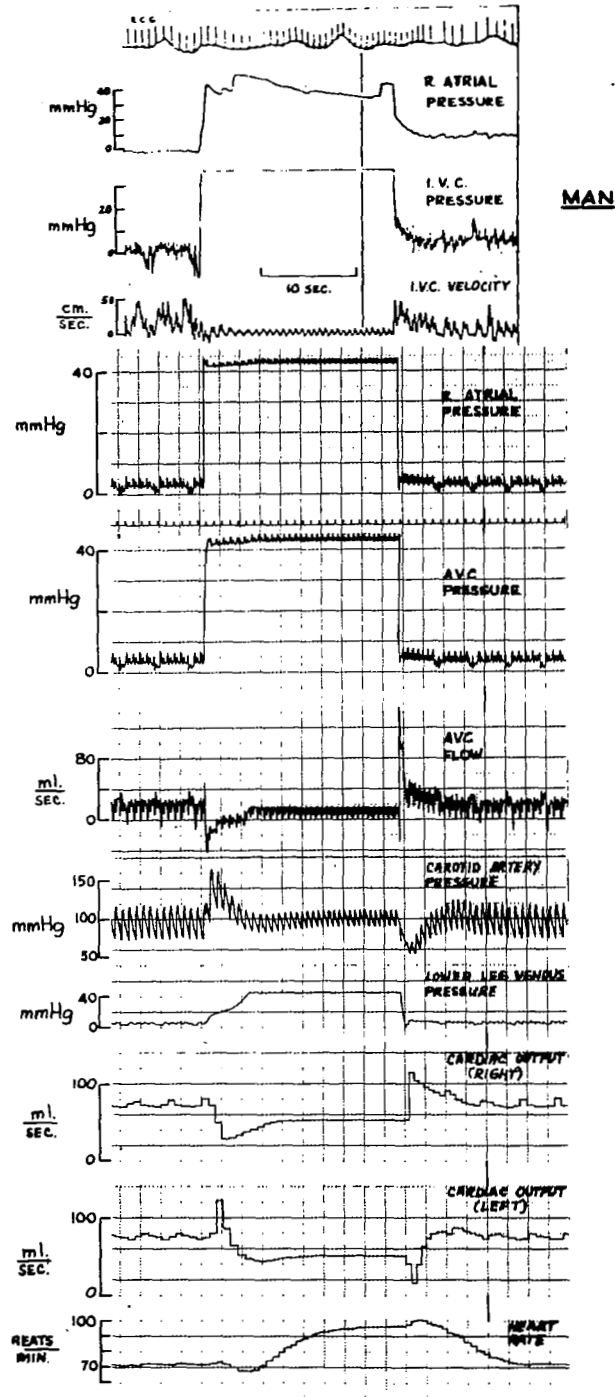
Fig.3. Valsalva maneuver in the horizontal position(in man, from Wexler et al,[18] upper four waves) and in the model (lower traces)
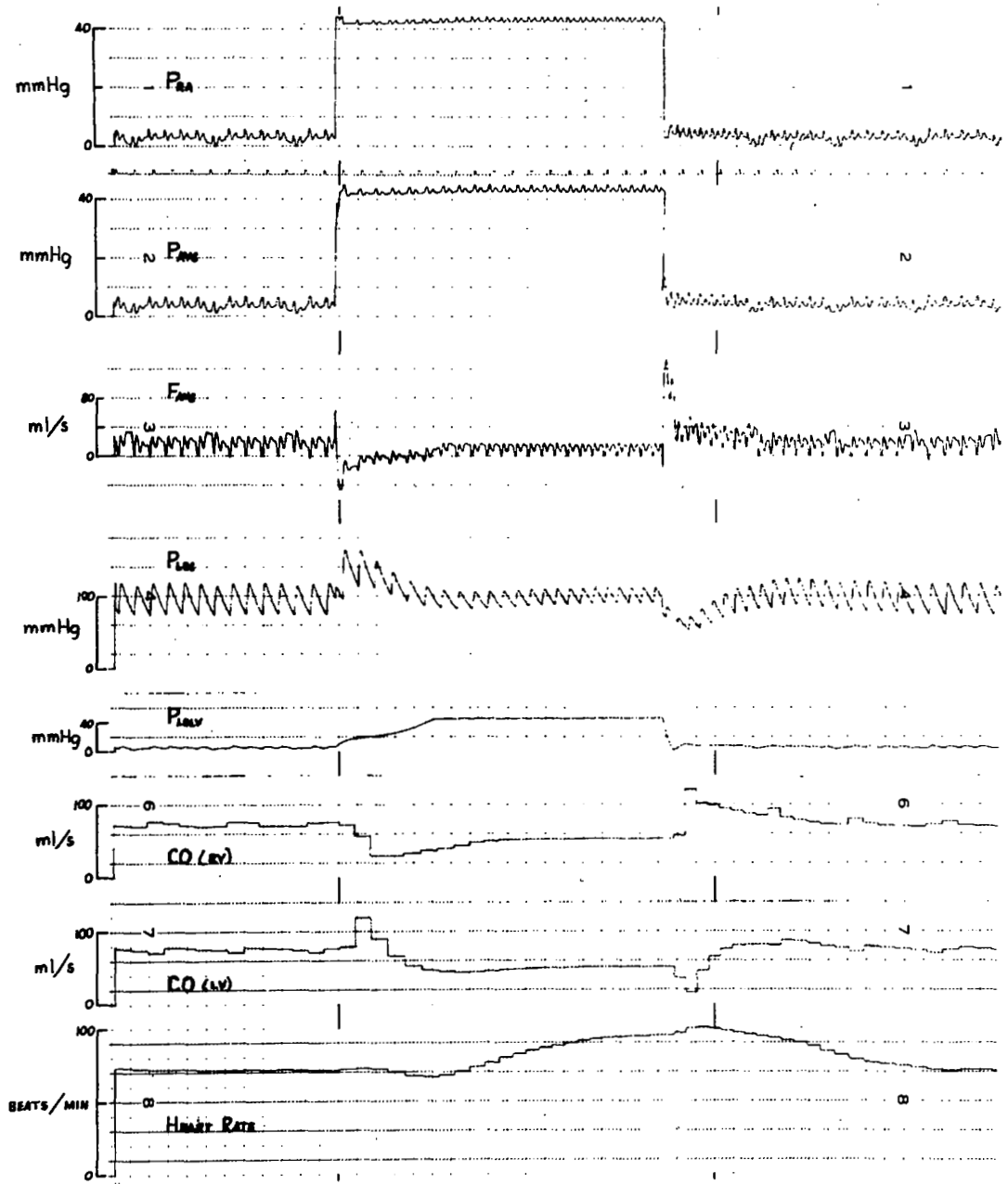
Fig.4. Same as model results of Fig. 3, but at faster recorder speed.

Fig.5. Valsalva meneuver in the horizontal position - venous variables
(this and all remaining figures measured from the model of Fig.2)
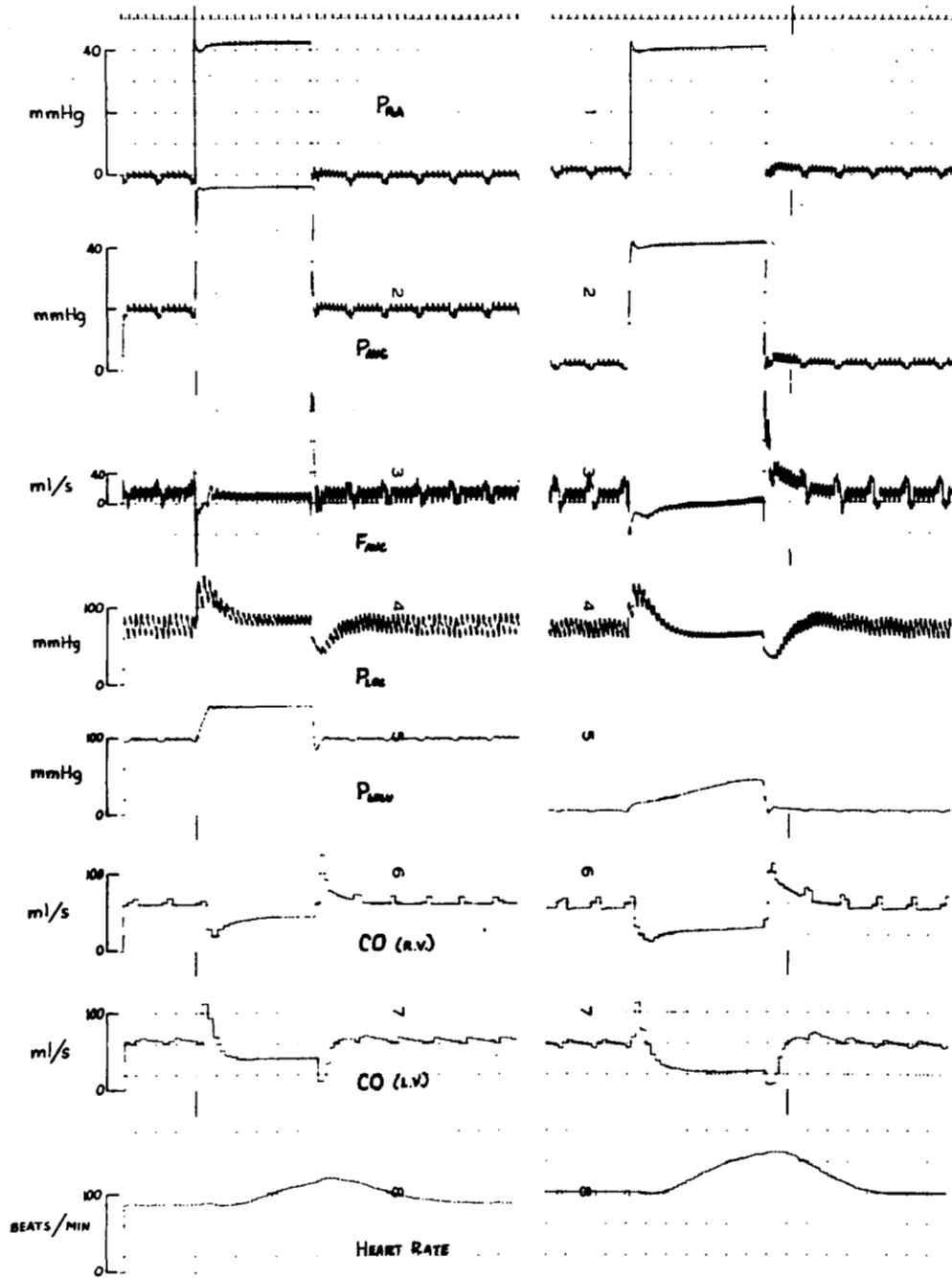
Fig.6. Valsalva in head-up tilt position(left) and same after 500 ml
hemorrhage (right)
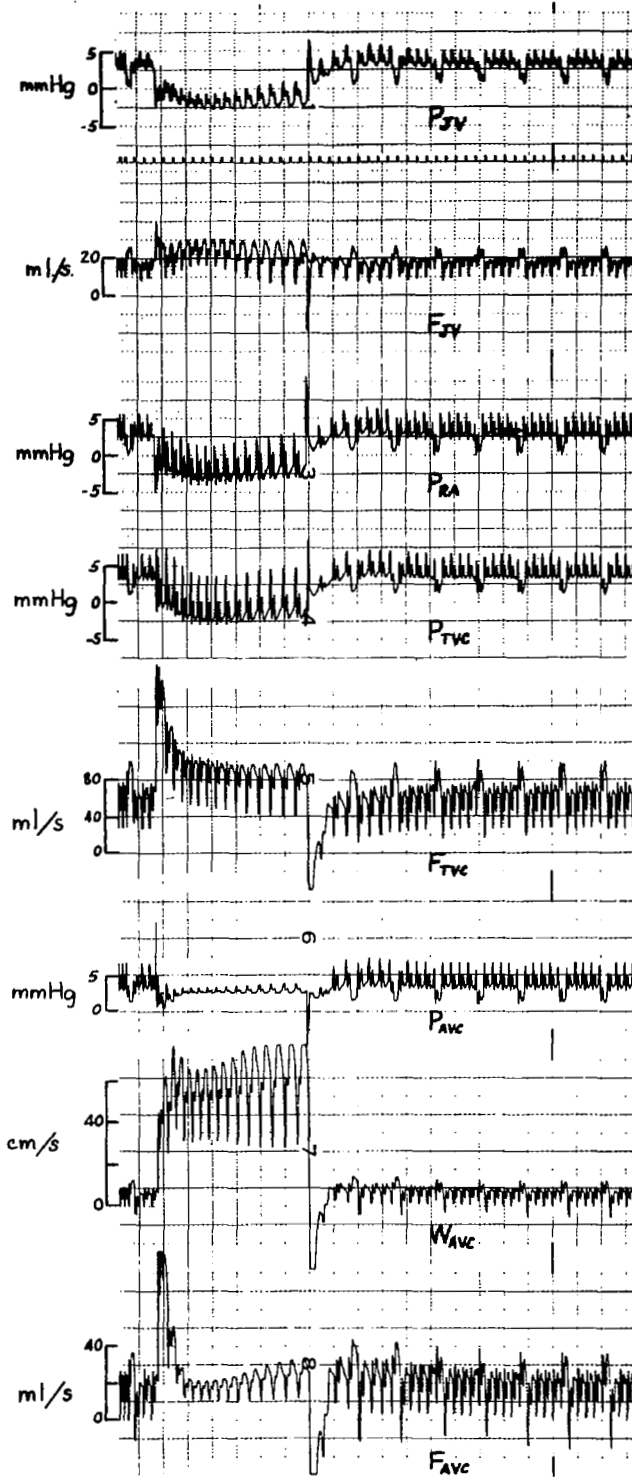
Fig.7. Müller maneuver in horizontal position(P_{ITh}=-10mmHg,P_{IAb}=+10mmHg)
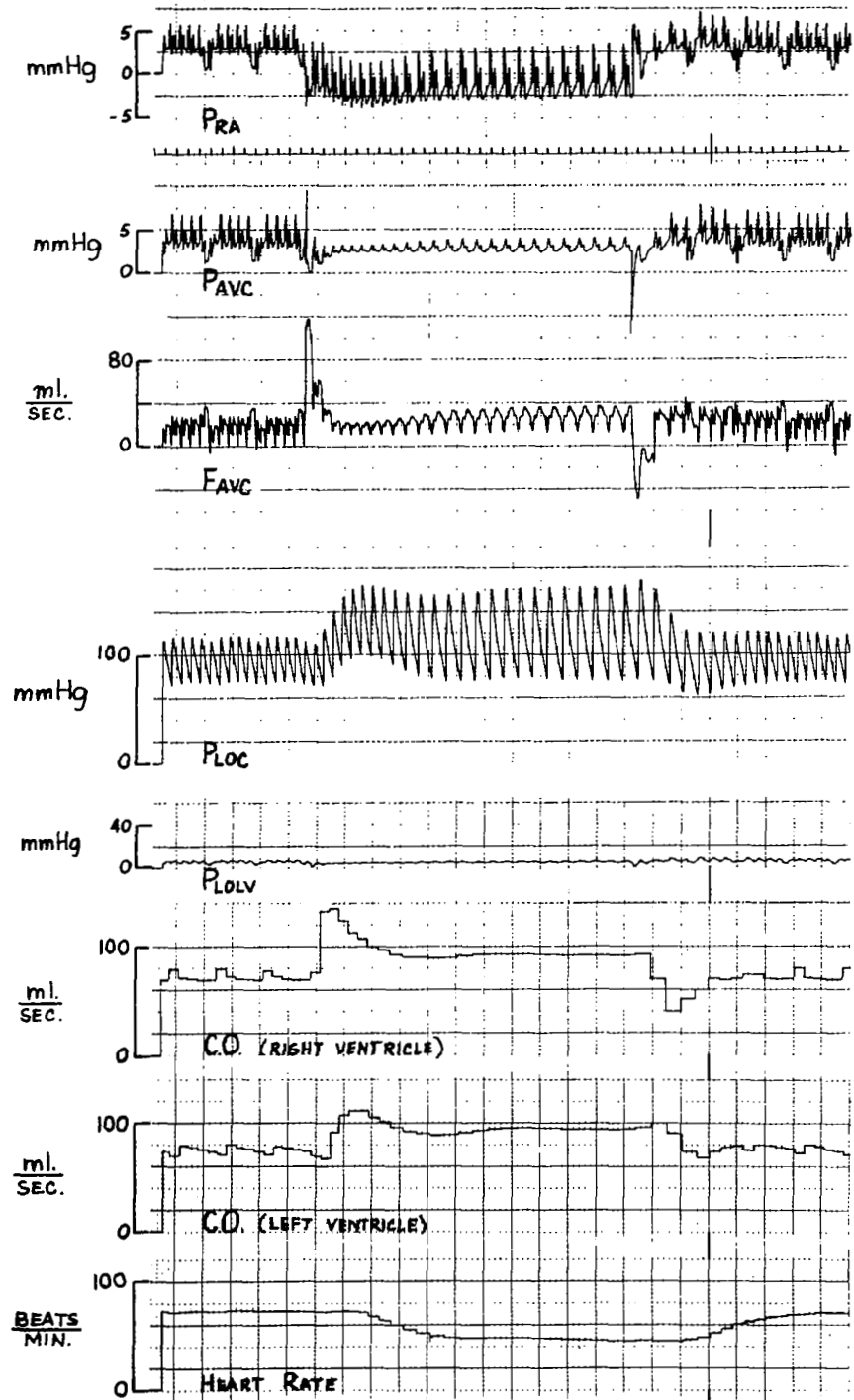
Fig.8. Müller maneuver as for Fig.7, but showing different variables.
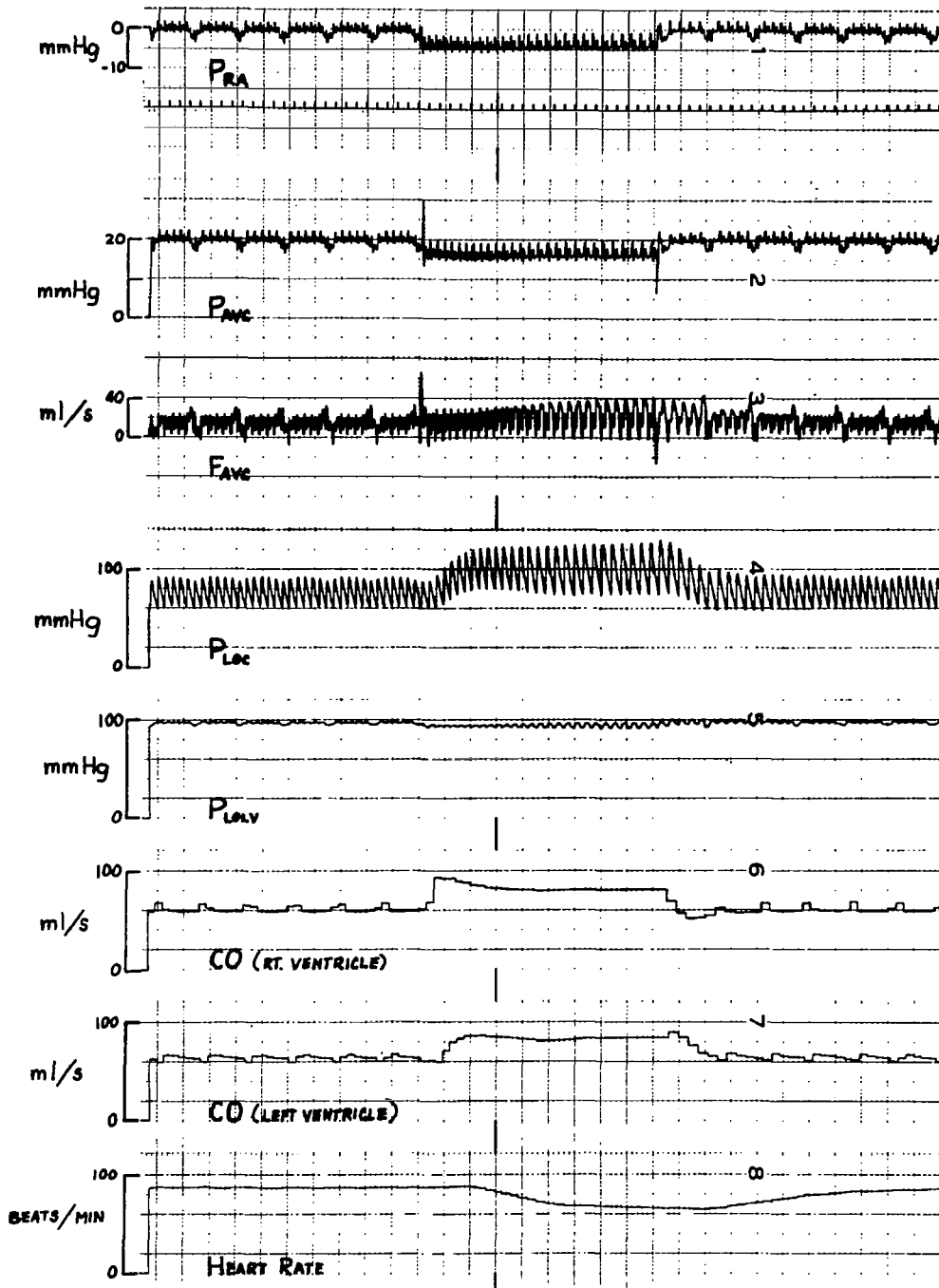
Fig.9. Müller maneuver in head-up tilt position(P_{ITh}=-10mmHg,P_{IAb}=+10mmHg)
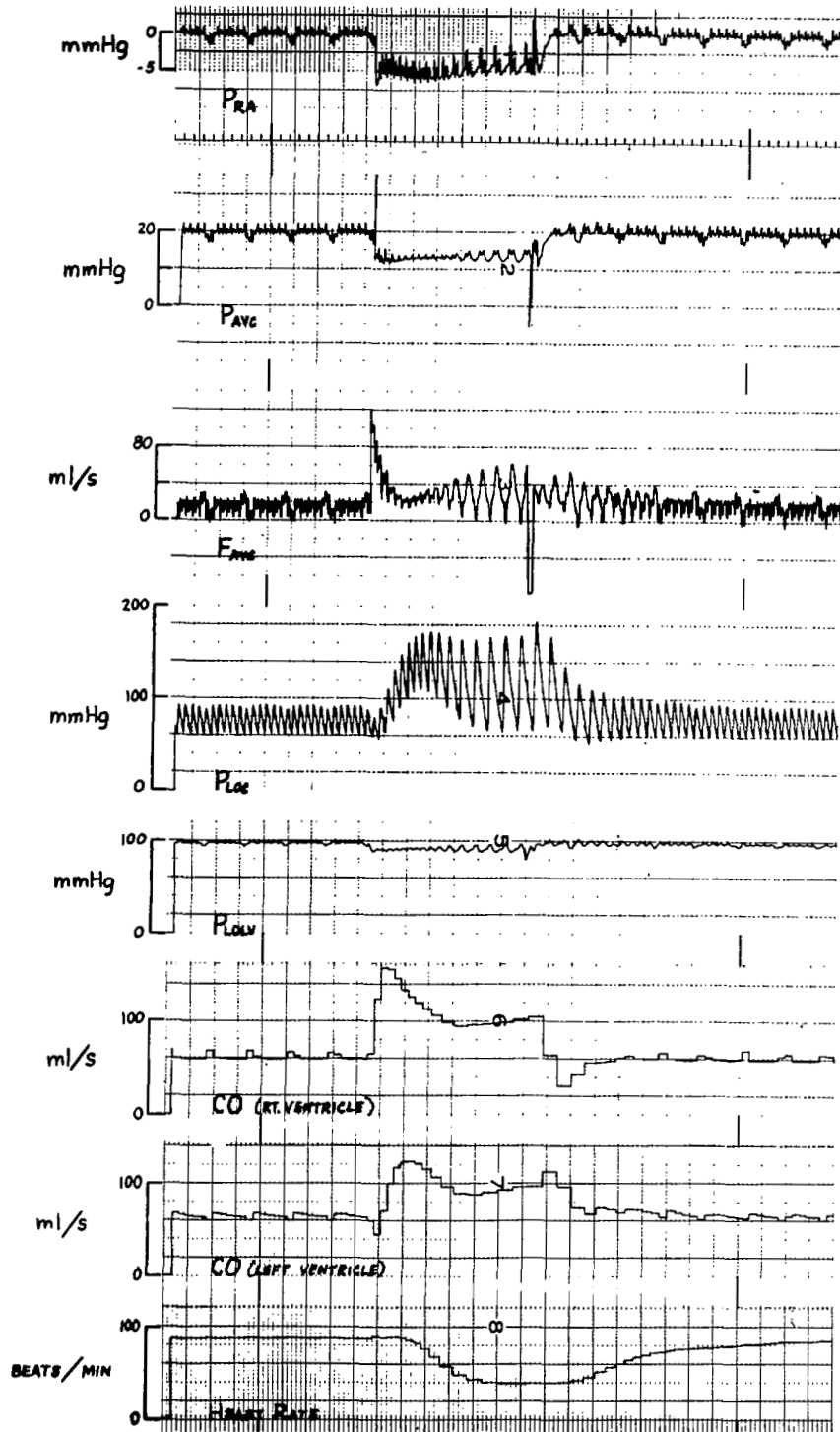
Fig.10. Same as Fig.8 but with P_{ITh}=-20 mmHg, P_{IAb}=+20 mmHg.
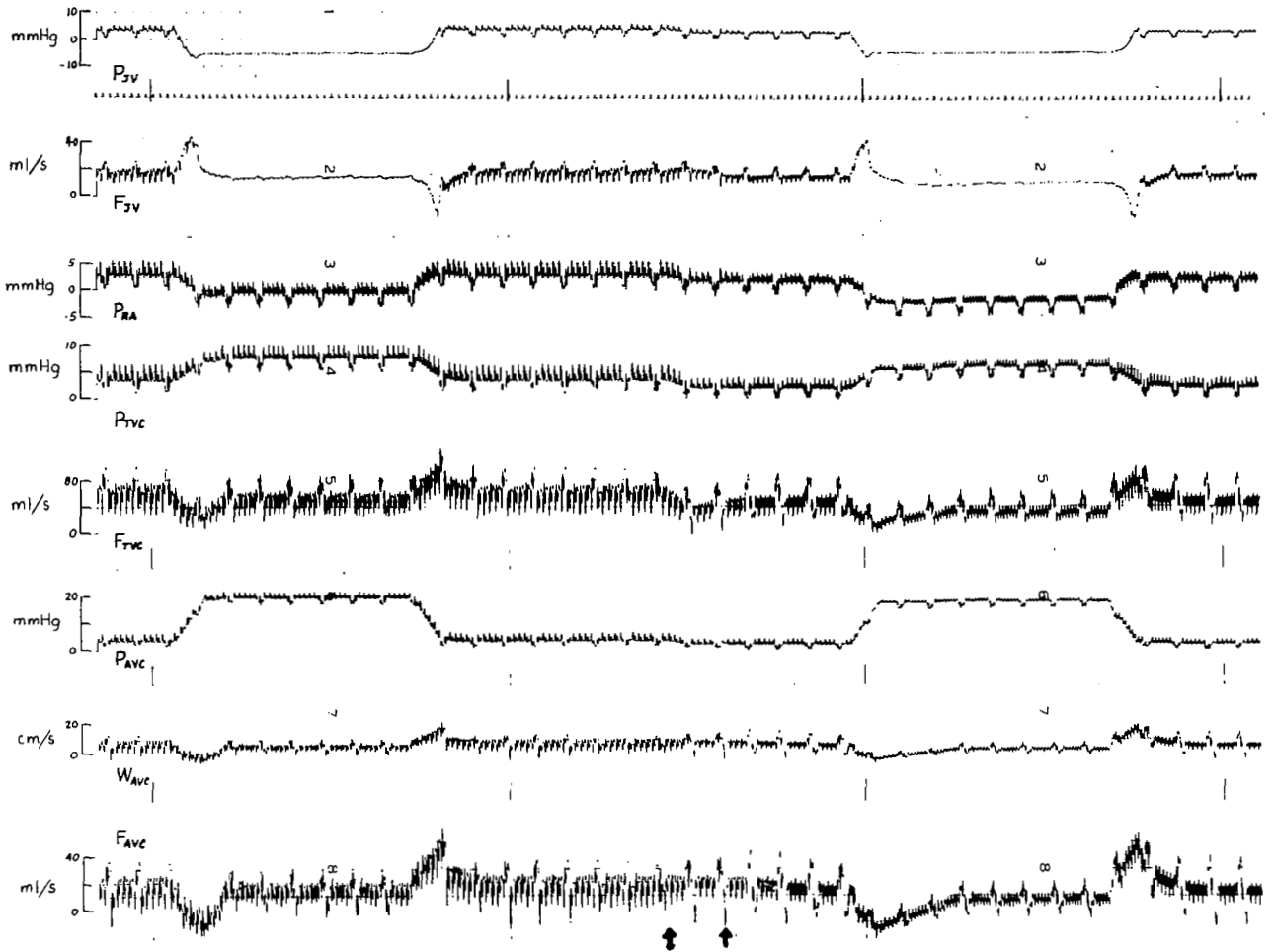
Fig.11. Tilt to head-up tilt position before and after 500 ml hemorrhage
(time of hemorrhage between arrows at bottom of figure)
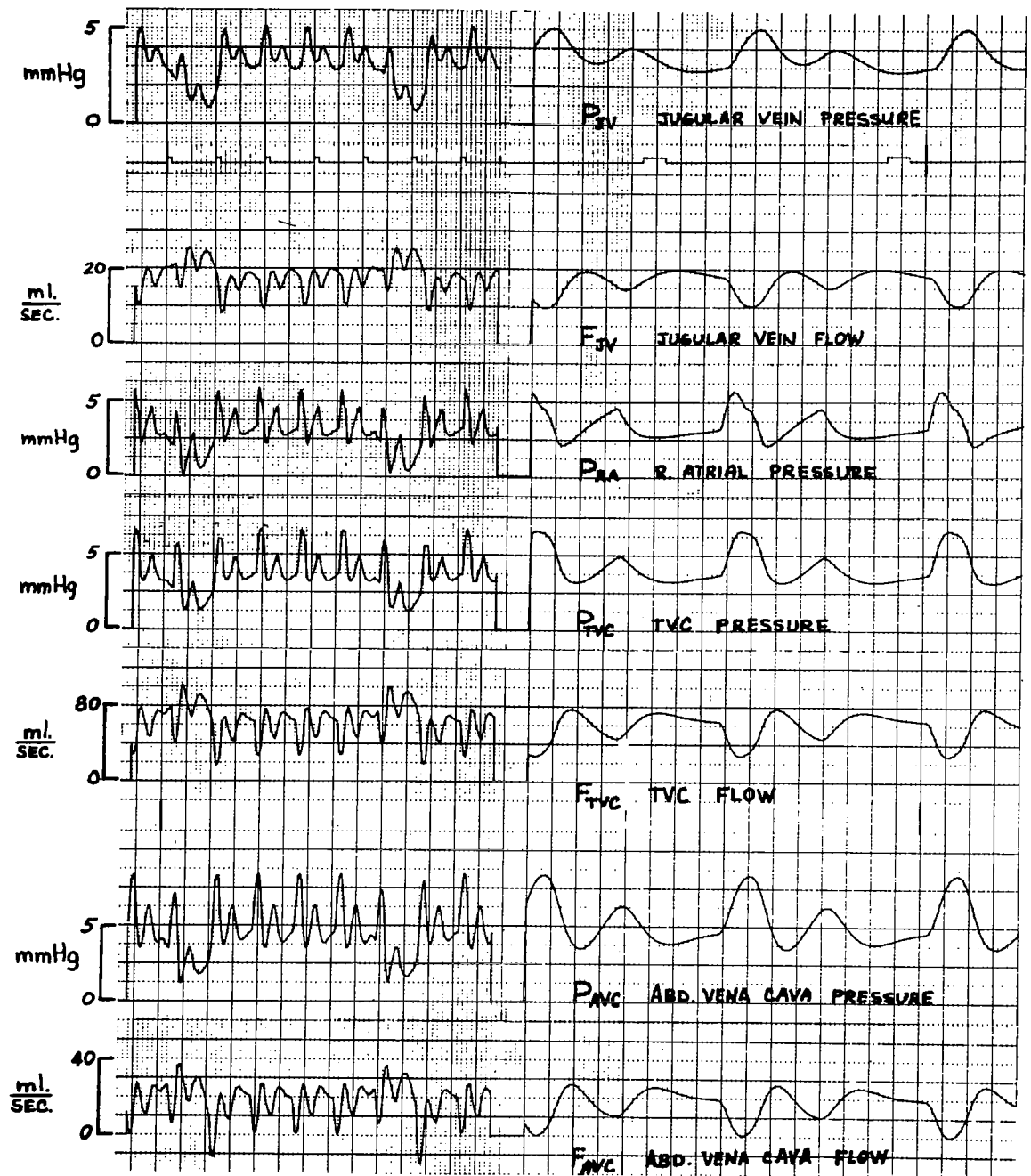
Fig.12. Various variables measured in the venous system of the model under normal respiration in the horizontal position.
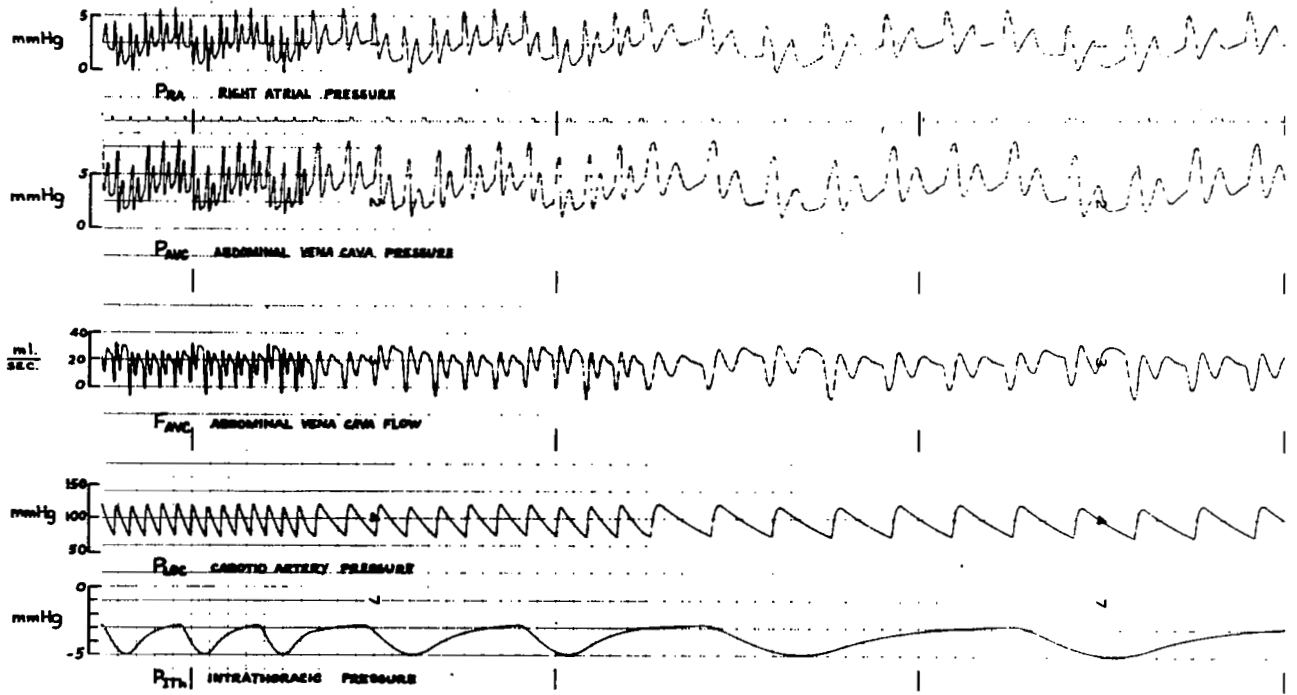
Fig.13. Several venous variables using a slightly modified P_{ITh} waveform.
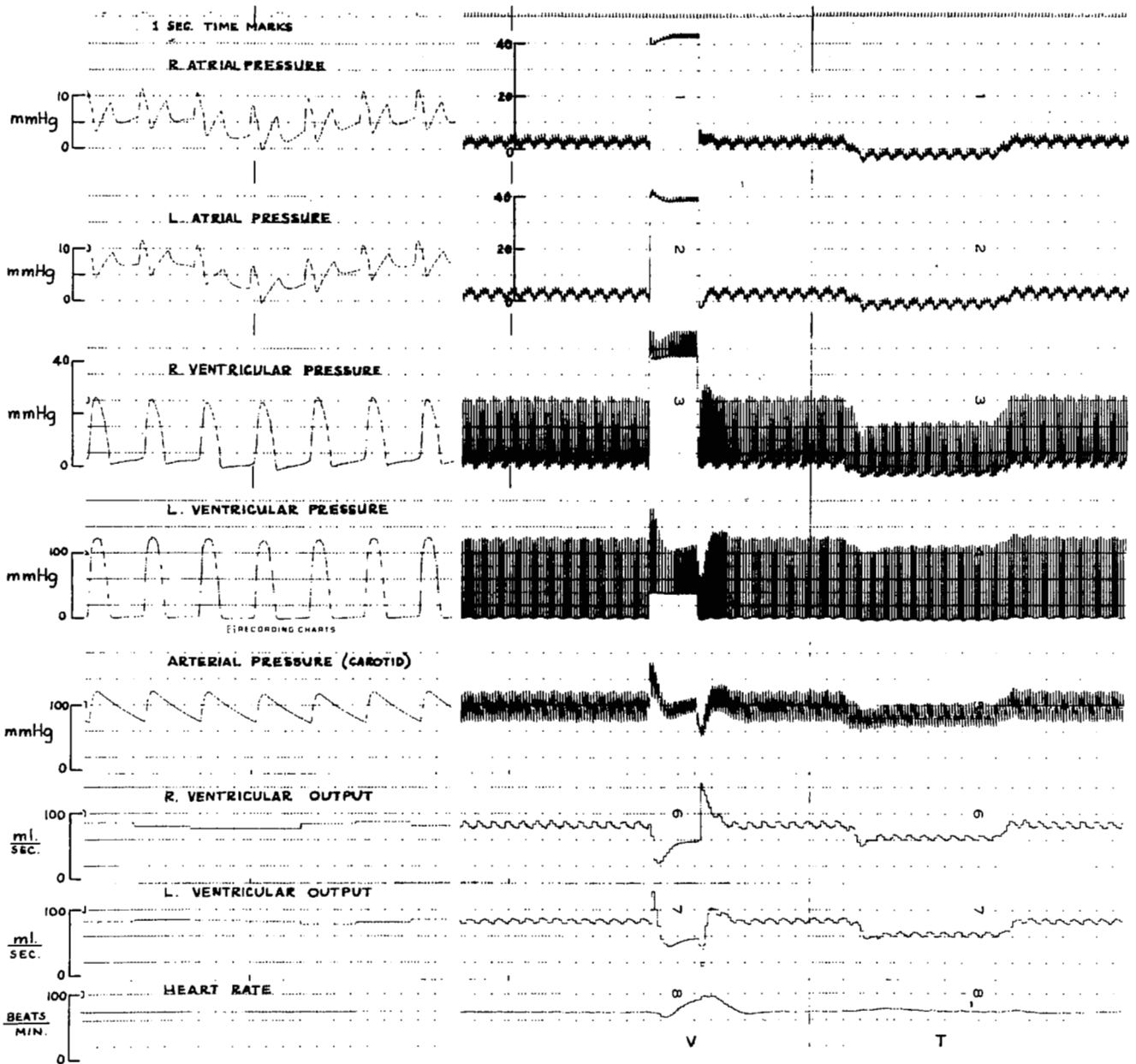
Fig.14. Various normal pressures from the model and a normal response to a Valsalva(V) and a tilt(T) maneuver.
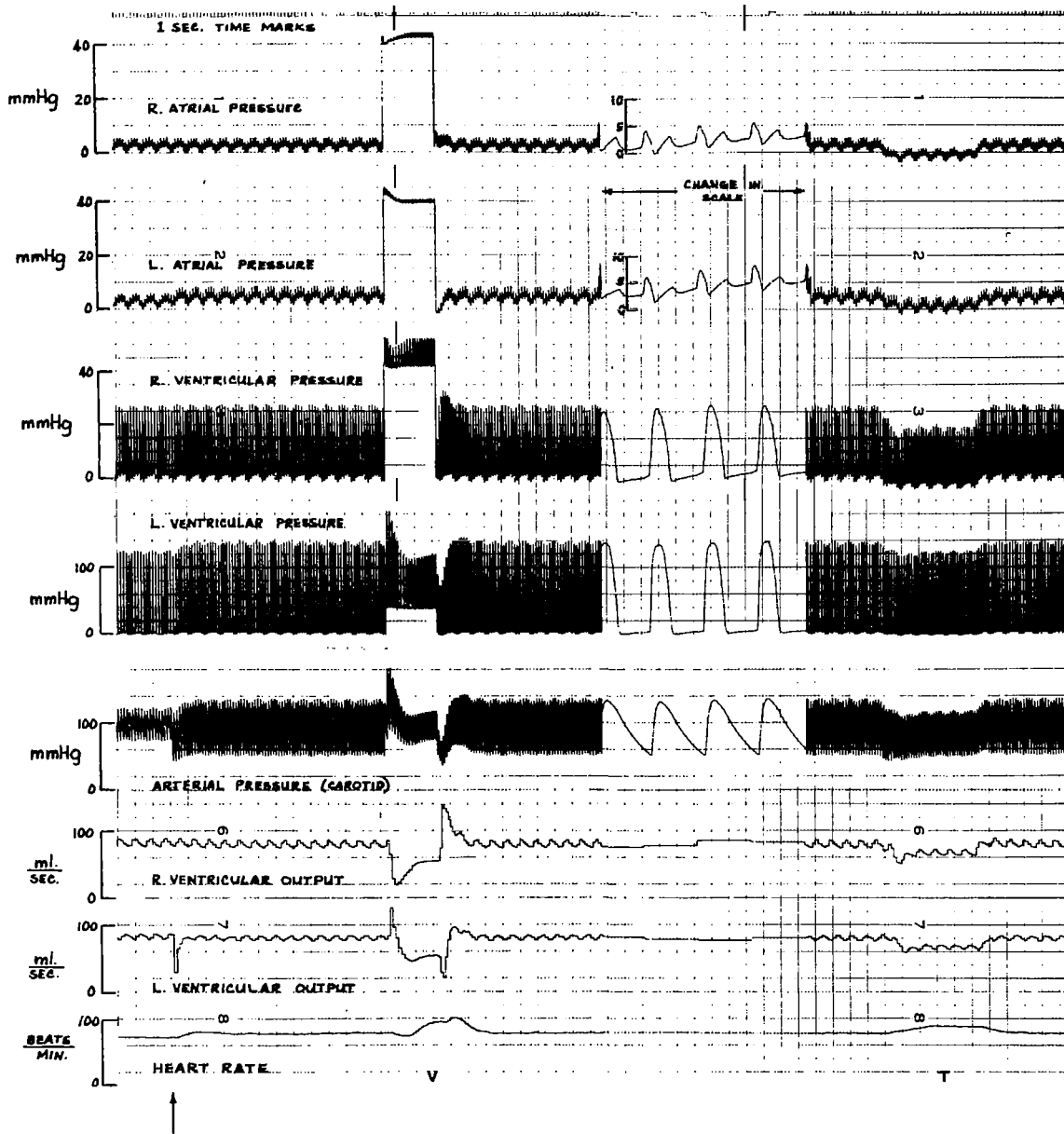
Fig.15. Introduction of an aortic valve insufficiency defect with a reverse
valve resistance of 1000 followed by a Valsalva and tilt.
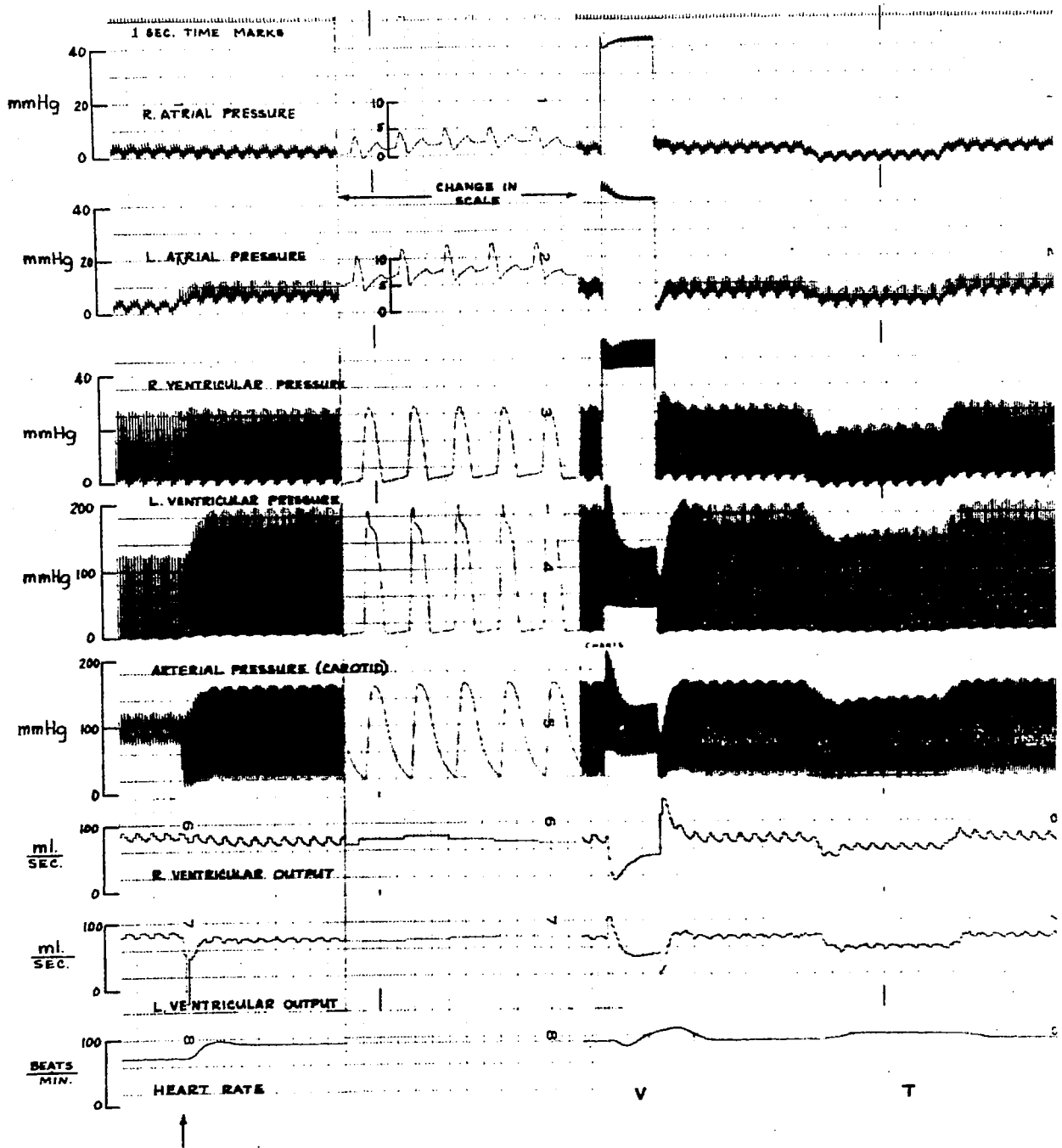
Fig.16. Same defect as in Figure 15, but with a reverse valve resistance
of 200.
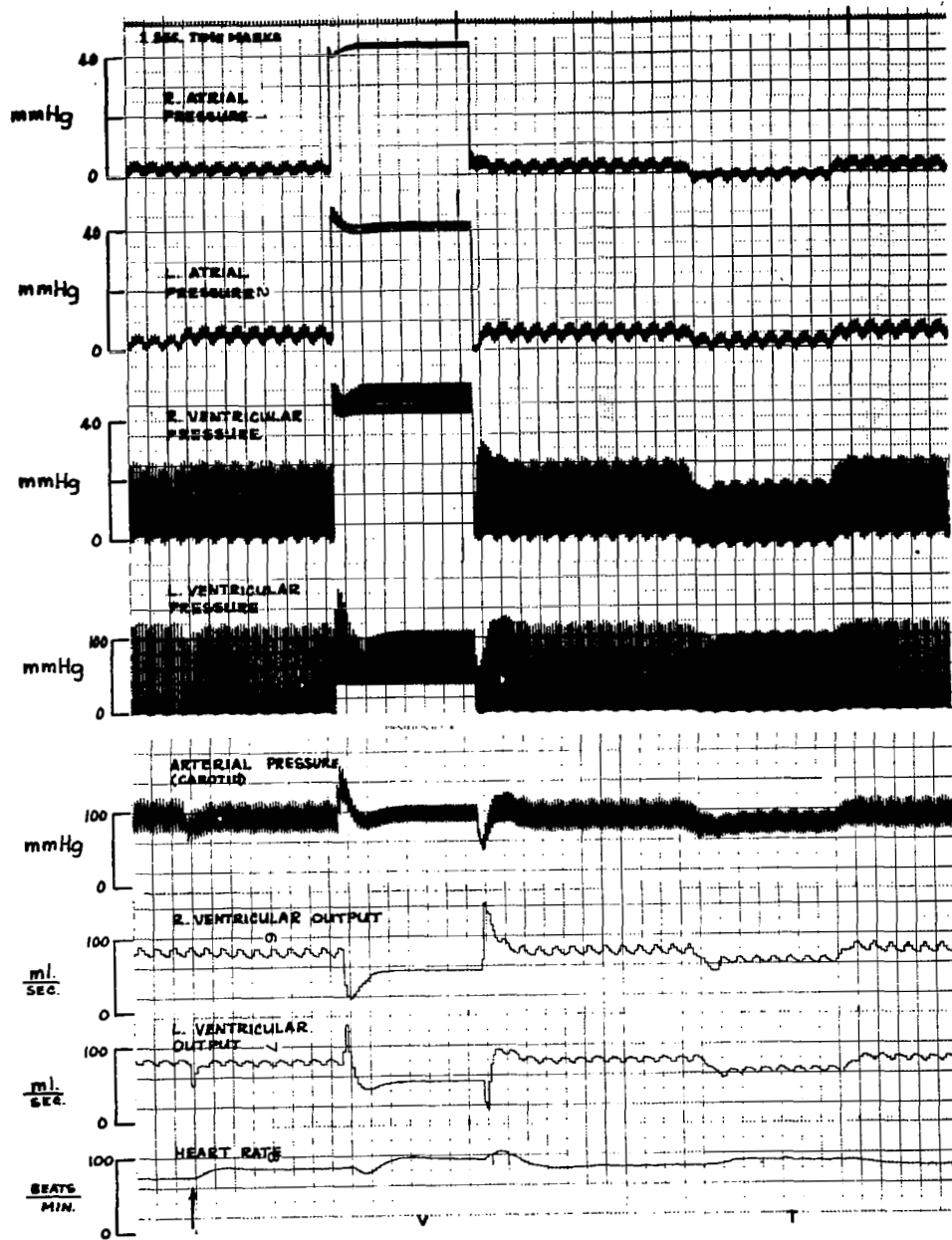
Fig.17. Introduction of a mitral valve insufficiency defect with a reverse
valve resistance of 500 followed by a Valsalva and tilt maneuver.

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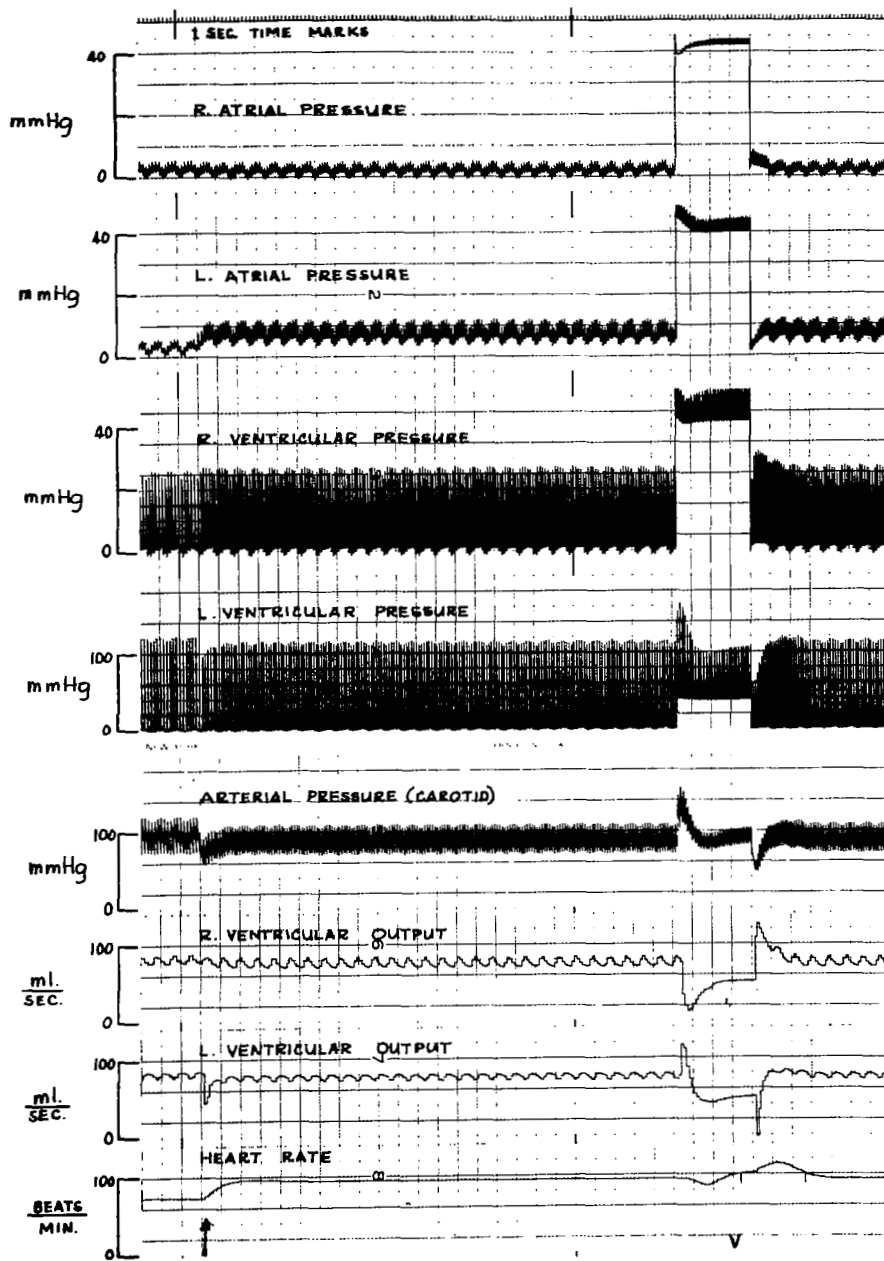
Fig. 18. Same defect as in Figure 17 but with a reverse valve resistance of 200.
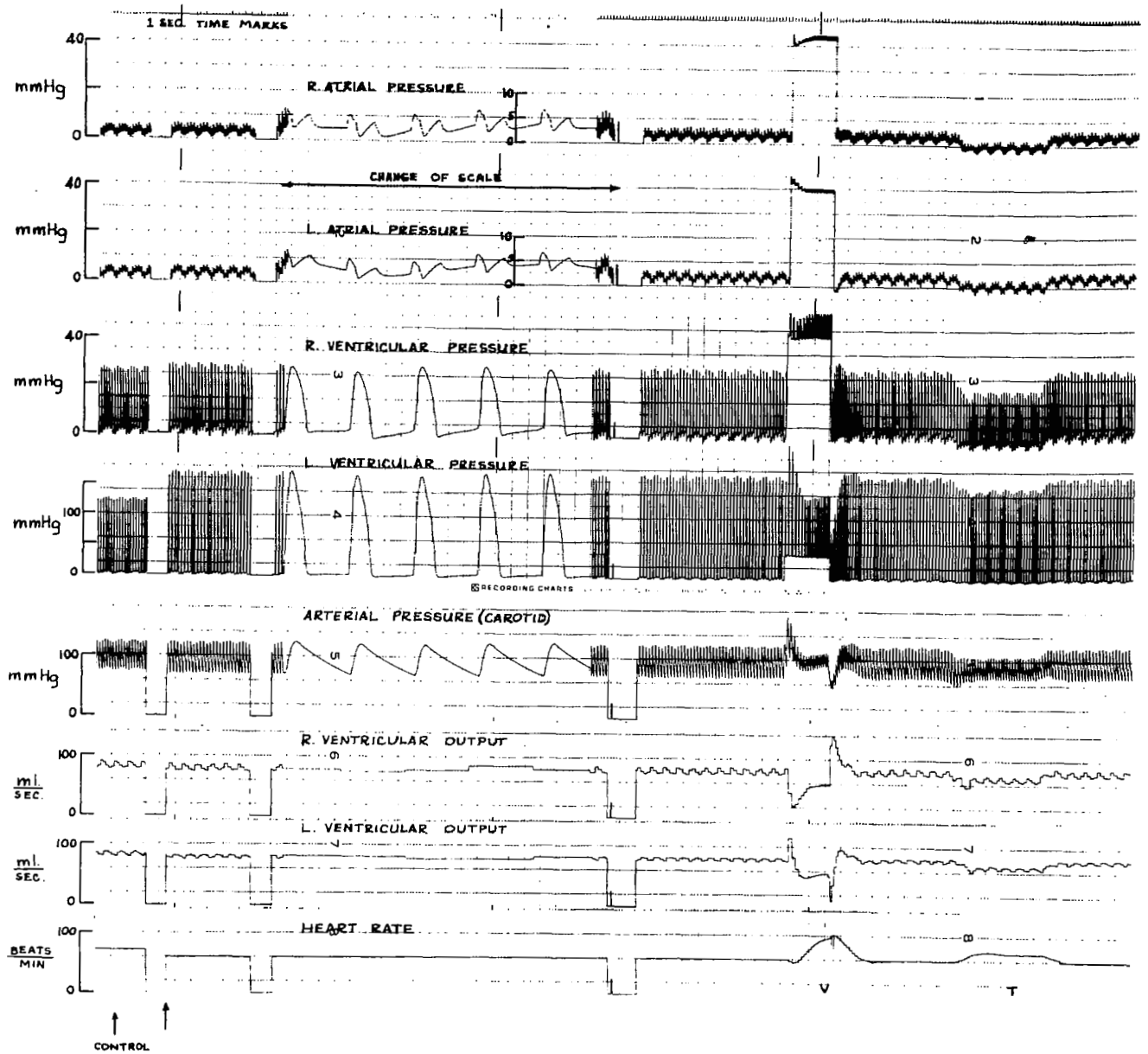
Fig.19. Introduction of an aortic valve stenosis defect where the forward
valve resistance is increased to 133(10 times the normal resistance
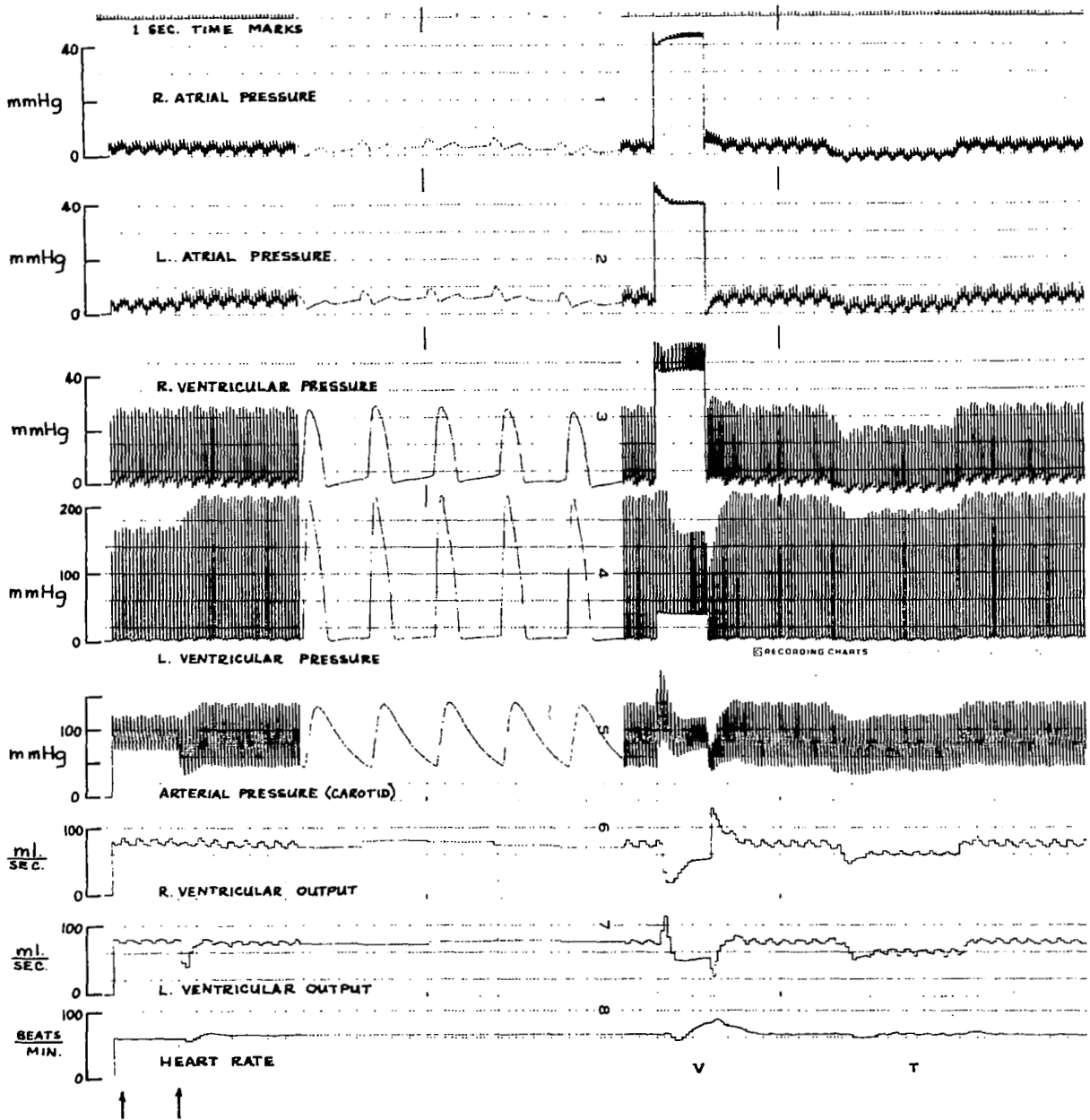of 13.3)followed by a Valsalva and tilt.
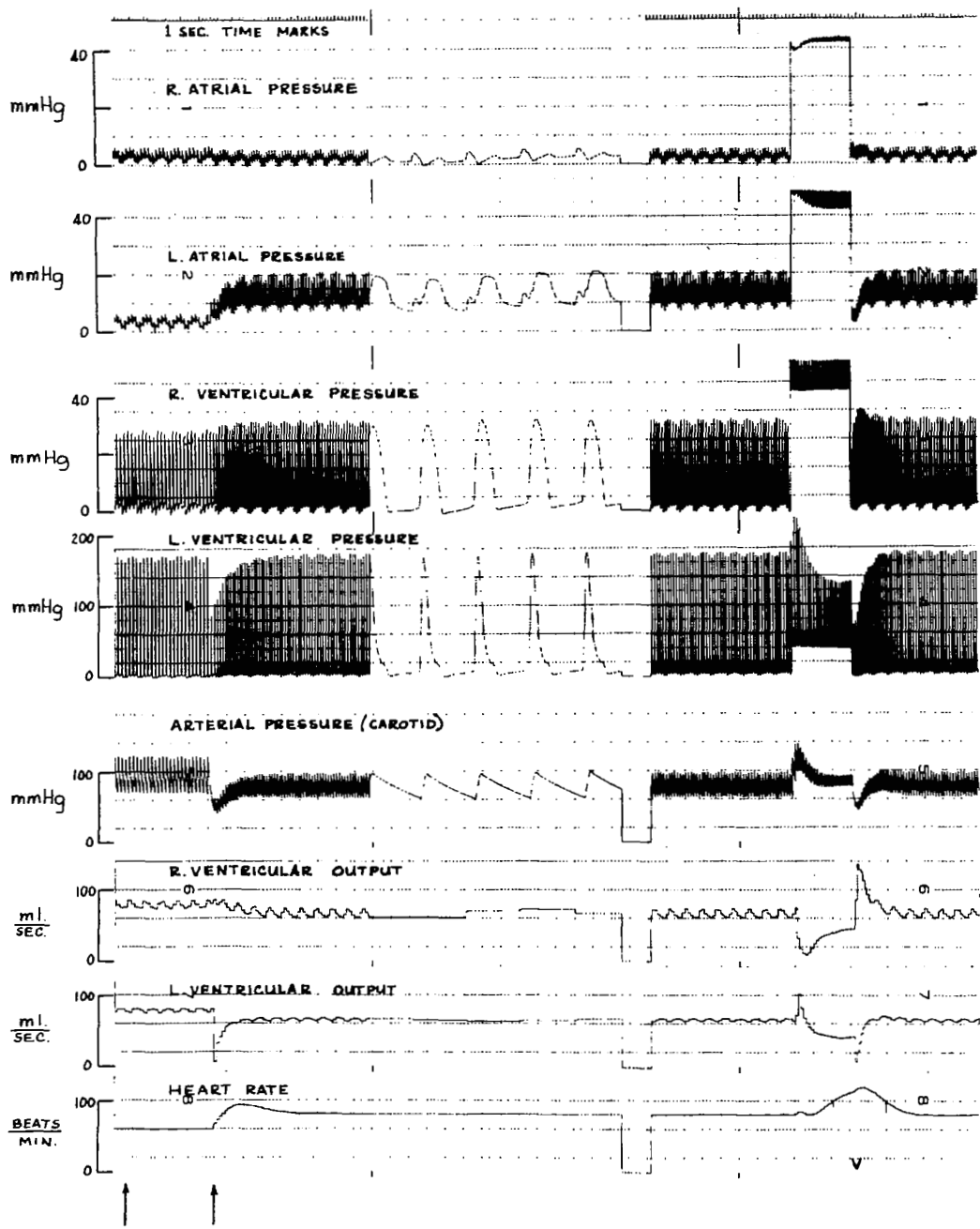
Fig.20. Introductions of two defects simultaneously:aortic valve stenosis
with a forward resistance of 133(1st arrow) and mitral valve insuf-
ficiency with a reverse resistance of 100(2nd arrow) followed by a
Valsalva maneuver.

Fig.21. Introduction of two defects simultaneously: aortic valve stenosis
with a forward resistance of 133(1st arrow) and aortic valve insuf-
ficiency with a forward resistance of 1000(2nd arrow) followed by
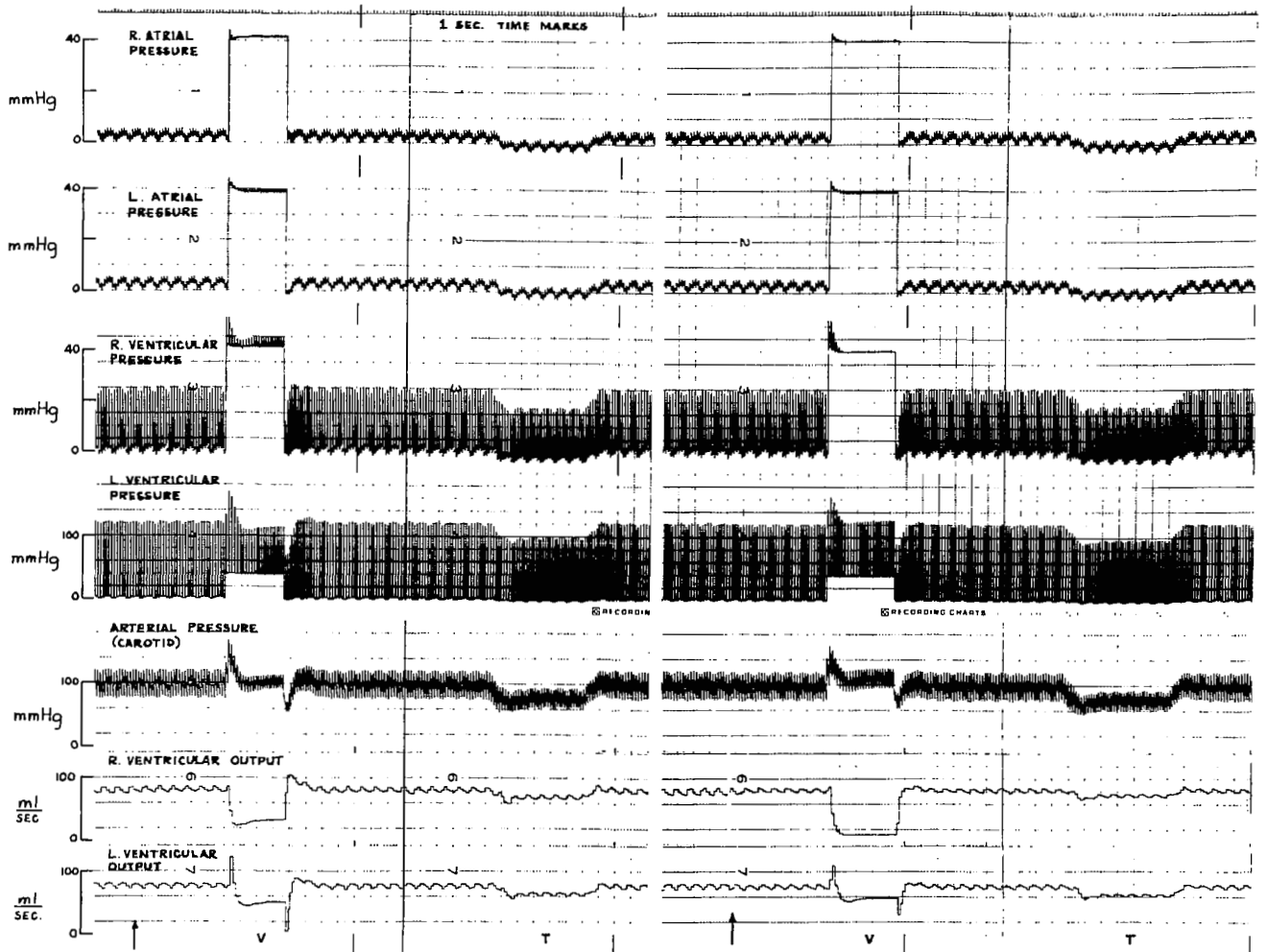a Valsalva and tilt maneuver.

Fig.22. Introduction of an atrial septal defect for two different resis-
tance values thru the septum, namely, 100 (1st arrow) and 10(2nd
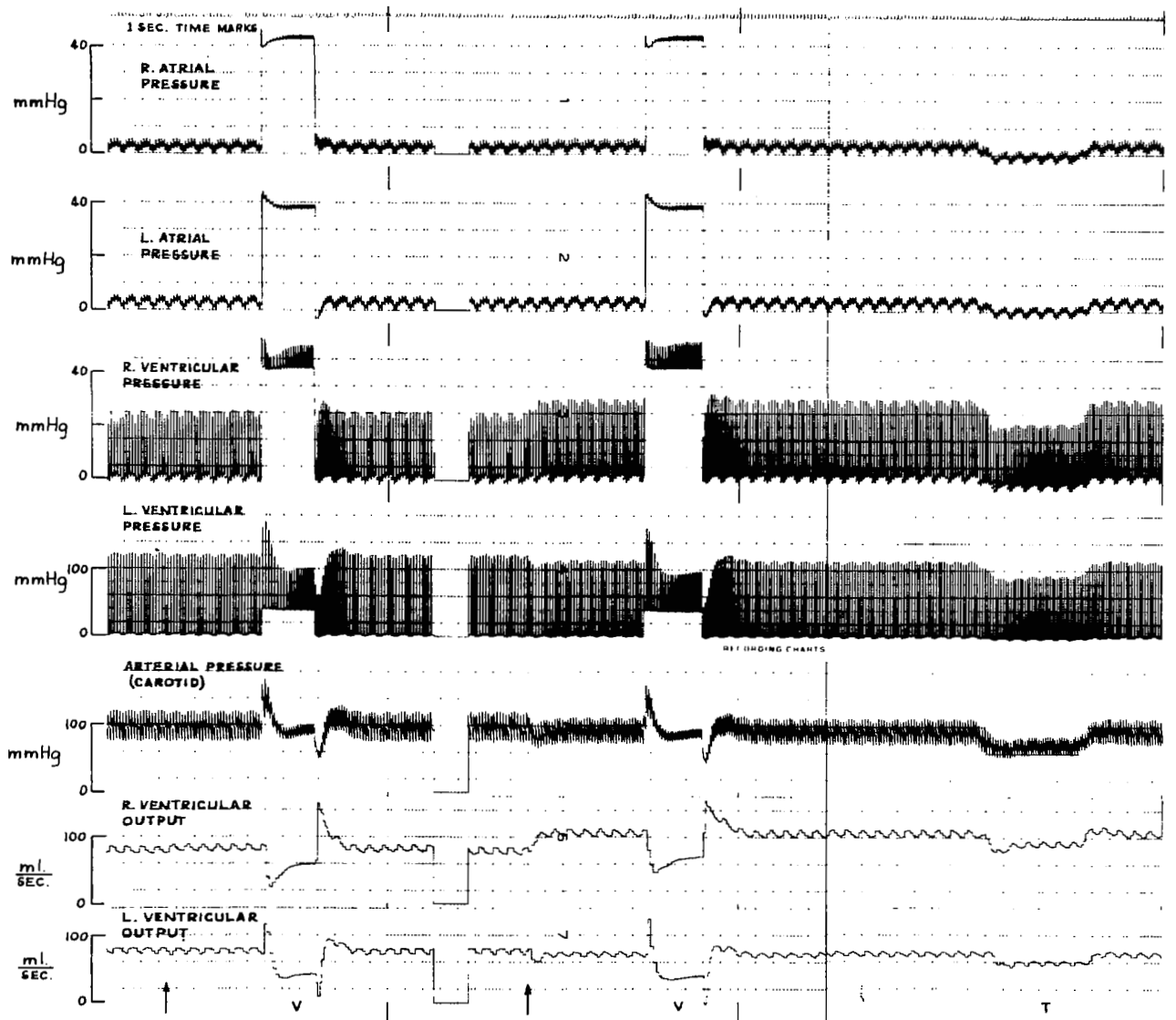arrow) followed by a Valsalva and tilt in both cases.

Fig.23. Introduction of a ventricular septal defect for two resistance
valves thru the septum, namely 10,1000(1st arrow) and 1000 (2nd
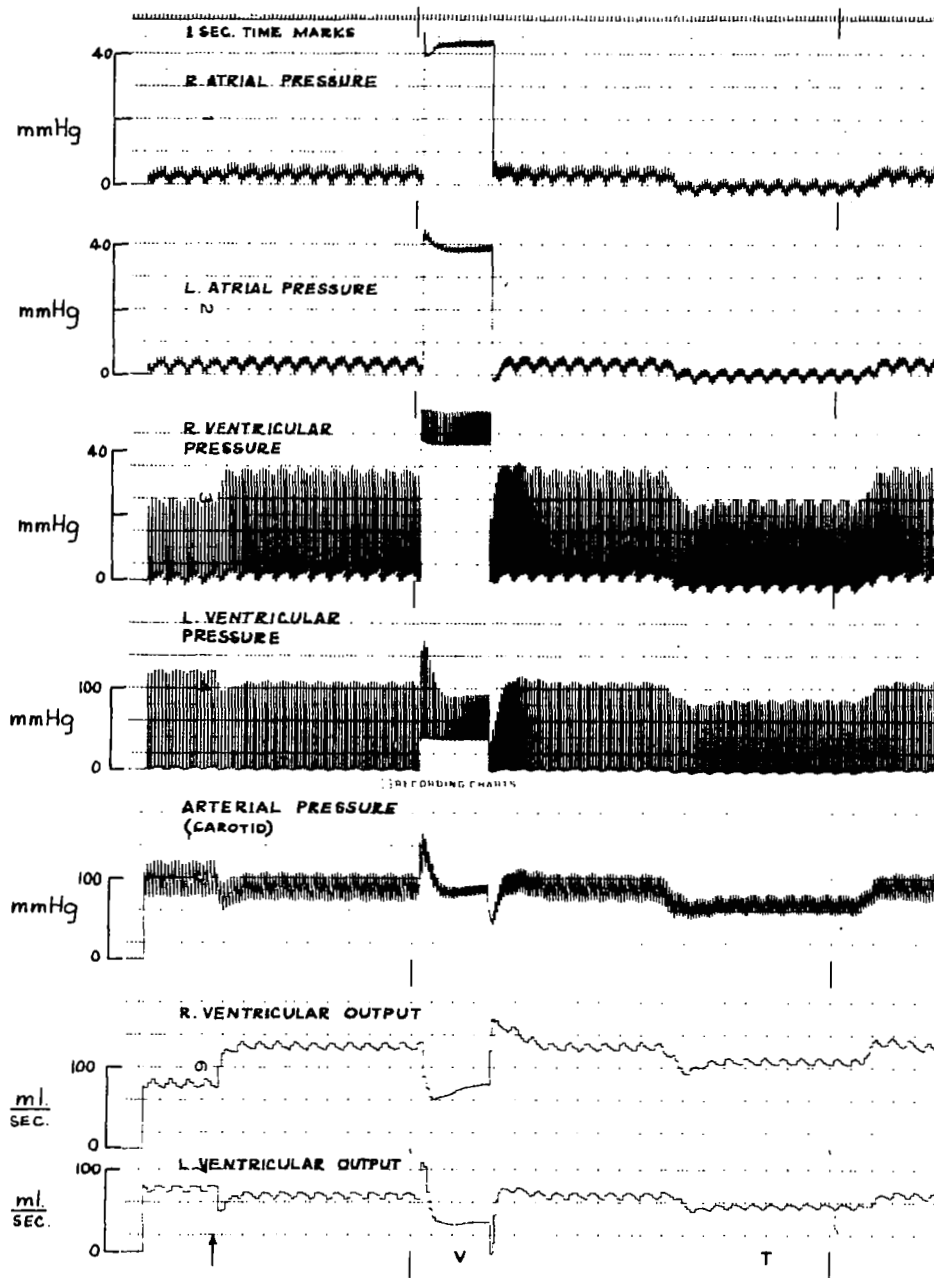arrow) followed by a Valsalva and tilt.

Fig.24. Same ventricular septal defect as in Figure 23, but with a septal resistance of 500 followed by Valsalva and tilt.
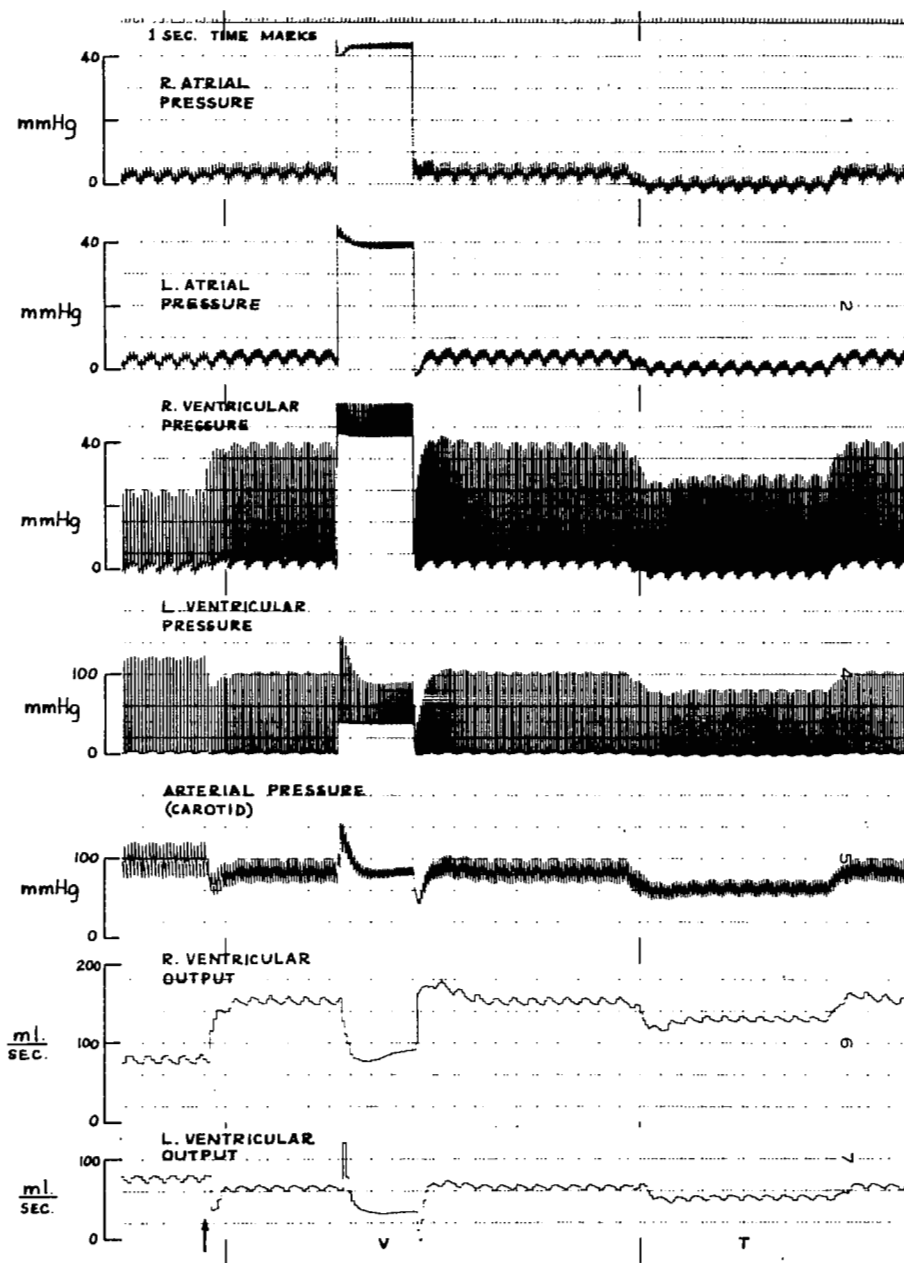
Fig.25. Same ventricular septal defect as in Figure 24, but with a septal resistance of 250 followed by a Valsalva and tilt.