INTERRELATIONSHIP BETWEEN CORONARY VENOUS PRESSURE AND VENTRICULAR CONTRACTION*

By

S. SRIVASTAVA,** D. LAURENT AND S. SEROUSSI

Centre d'etudes des technique chirugicales, Broussais Hospital, Paris.

The existence of reflexes arising from the coronary venous bed and acting on the heart, (Szentivanyi and Juhasznagy, 1963) has been postulated. Recently their patterns have been analysed. Nevertheless their physiological role has not been clearly demonstrated but the origin of reflexes from pressoreceptors localized in the wall of the coronary veins has been postulated. Instantaneous pressure in any part of the coronary venous system depends on left ventricular and right auricular pressures; in order to analyse both these factors it seemed worthwhile, to separately control the auricular and ventricular rates and eventually obtain summation of auricular and ventricular borne coronary venous pressure waves.

MATERIALS AND METHODS

Experiments were performed on six mongrel dogs weighing between 17-25 *kg*. They were prepared in the manner described earlier by Srivastava *et al.* (4). Two stimulation catheters were then guided under fluroscopy, one into the right auricle placing its tip as close as possible to the sinus node, the other into the left ventricle placing its tip on the left inflow ventricular tract. (*Fig.* 1). In every instance their positions, were secured in such a way that the interval between the stimulation artefact and the evoked auricular and/or ventricular depolarisation, were as short as possible i.e. as a rule less than 20 ms.

As soon as a steady state was obtained, 2.5 ms rectangular pulses, 1-3 volts strength were delivered to the right atrium and left ventricle by two separate stimulators at rates slightly higher than the spontaneous sinus rate. In some experiments, the left ventricular stimulation was superimposed on the spontaneous uncontrolled sinus rate. Auricular stimulation was achieved at a rate very near to the spontaneous sinus rate of the animal and was controlled in such a way that it actually captured the auricular activity. The rate of the ventricular stimulation was chosen very close to the auricular stimulation rate (or the sinus spontaneous rate if the auricles were not controlled). Thus by varying the periods of auricular and ventricular stimuli an auriculo-ventricular asynchrony with a preset A:V ratio was produced. The periods of auricular and ventricular stimuli were very close to each other in order to obtain cycles of long duration.

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** Present address : Department of Physiology, King George's Medical College, Lucknow.

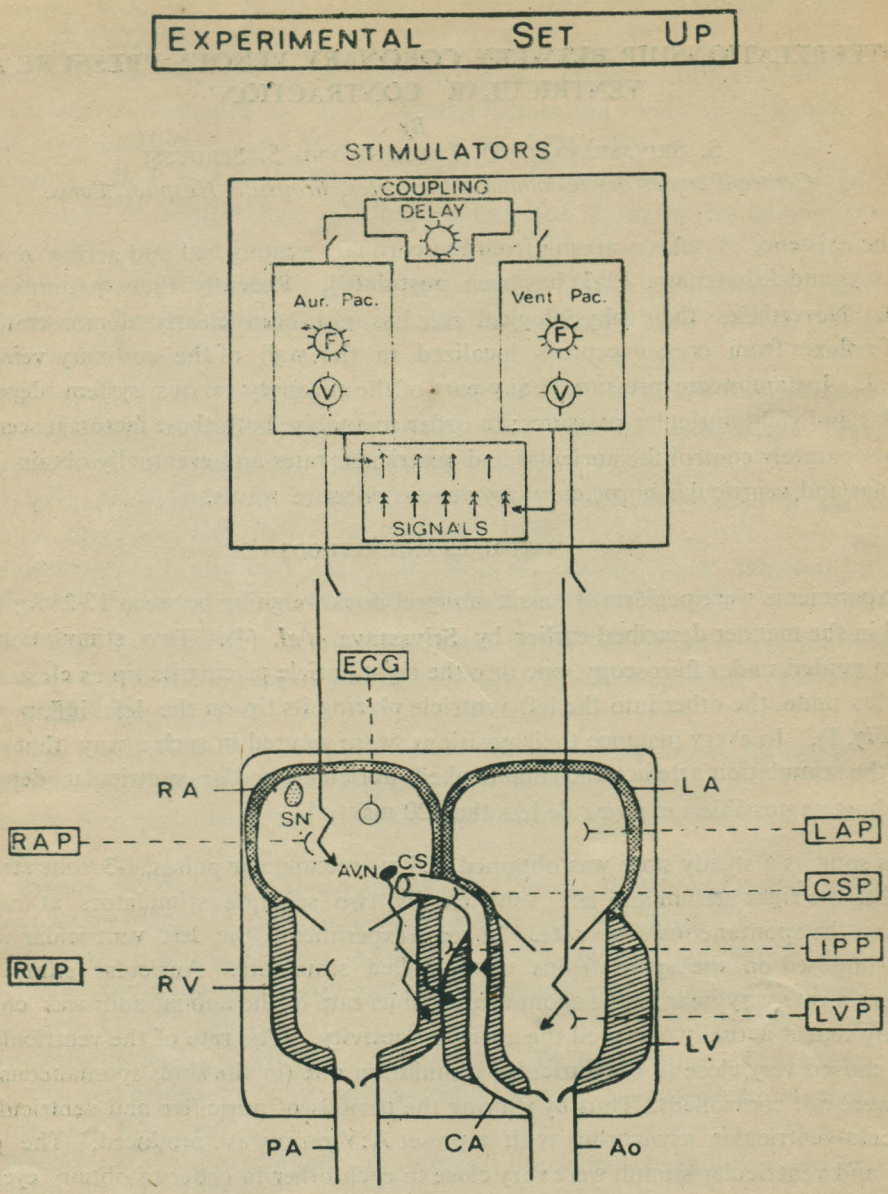

Fig. 1.

Experimental set up.

***RA, LA, RV, LV**, right auricle, left auricle, right ventricle, left ventricle. CS-coronary sinus. SN Sinus-node, AVN atrioventricular node. PA-pulmonary artery. Ao-aorta. CA-coronary arterial bed. CSP-Coronary sinus pressure. IPP-intrapericardial pressure.*

RESULTS

The effects of AV asynchrony on the various pressure tracings were studied in all the 6 experiments. It was noticed that both, right and left auricles, and right and left ventricles behaved in the same way therefore the right auricular and left ventricular pressure recordings were done. For each successive beat the maximal coronary venous pressure, maximal right atrial pressure and maximal left ventricular pressure were tabulated. The intervals between the P waves of the E.C.G. and the "a" waves of the right atrial pressure and the peak sinus pressure were measured. The interval between the ventricular stimulation (artefact S_2) and the auricular event (which was present before or after the ventricular event) was also measured for the successive beats and was represented as S_2P interval. It was not always possible to obtain periodic conditions regularly because of the occurrence of cardiac irregularities due to ventricular captures or even retograde ventriculo-auricular conditions, therefore only 25 sequences were selected for study (Tables I and II). When the ventricular rate was faster than the auricular, the ventricular stimulation ran all through the cardiac cycle. Conversely, when the auricular rate was faster only a part of the cycle was swept over by the ventricular stimulus, a PR pattern of progressively varying duration being predominantly obtained. This fact is due to the temporal relationship between the refractory period of the A-V junction and the difference in the atrial and ventricular periods.

TABLE I

Dog number	*Period (ms)*		*Cycle's period (ms)*	*Coronary venous pressure in (mm. Hg.)*		*Change in C.V.P.*	*Peak L.V.P. (mm. Hg.)*		*Fall in peak L.V.P.*	*R.A.P. (mm. Hg.)*		*Absolute rise in R.A.P.*
	Aur.	*Ven.*		*Max.*	*Min.*	%	*Max.*	*Min.*	%	*Max.*	*Min.*	*mm. Hg.*
4506	343	344	26400	16.0	2.0	—87.5	114.0	76.6	33.3	14.0	1.5	12.5
	287	325	4320	5.0	0.0	—100	120.0	100.0	16.6	16.0	6.0	10.0
	318	306	9420	7.0	0.0	—100	150.0	80.4	16.6	17.0	4.0	13.0
	347	344	40200	10.0	2.0	—80	113.4	84.1	25.6	16.0	2.0	14.0
4525	300	305	10200	9.0	—2.0	—122	120.0	76.0	36.6	15.5	1.5	14.0
4463	325	332	18000	30.0	12.0	—60	120.0	74.0	38.3	9.5	1.0	8.5
	336	327	13800	29.0	11.5	—60.3	726.0	72.0	42.8	12.0	2.0	10.0
3299	406	384	7080	6.0	4.0	—33.0	170.0	120.0	29.4	N.A.	N.A.	N.A.
4525	315	300	6020	19.0	—2.0	—90.5	100.0	80.0	20.0	5.5	2.0	3.5
	310	300	8400	13.0	—2.0	—86.6	100.0	80.4	20.0	9.0	1.0	8.0
	306	360	12900	10.0	—2.0	—83.3	100.0	80.4	20.0	16.0	2.0	14.0

Data concerning 11 sequences of the *inphase* cyclic pattern induced in coronary venous pressure, by an atrio-ventricular asynchrony.

CVP: Coronary venous pressure. LVP: Left ventricular pressure.

RAP: Right auricular pressure. NA: Not available.

TABLE II

Dog number	*Period (ms)*		*Cycle's period (ms)*	*Coronary venous pressure in (mm. Hg.)*		*Change in C.V.P.*	*Peak L.V.P. (mm. Hg.)*		*Fall in peak L.V.P.*	*R.A.P. (mm. Hg.)*		*Absolute rise in R.A.P.*
	Aur.	*Ven.*		*Max.*	*Min.*	%	*Max.*	*Min.*	%	*Max.*	*Min.*	*mm. Hg.*
4506	311	321	8880	23.5	7.0	+ 220	120.0	100.0	16.6	12.0	2.5	9.5
	316	321	1680	18.5	8.0	+ 130	130.0	48.0	35.4	11.5	3.0	8.5
4525	286	297	6840	16.0	2.0	+ 100	114.0	82.0	28.0	16.0	1.0	15.0
	336	330	21600	20.0	12.0	+ 150	120.0	80.4	33.3	18.0	4.0	14.0
3299	392	383	16220	18.0	26.0	+ 200	200.0	128.0	36.0	N.A.	N.A.	N.A.
	359	378	7230	26.5	6.5	+ 307	190.0	110.0	42.1	8.5	2.5	6.0
	400	381	6000	31.5	5.5	+ 470	210.0	120.0	42.8	6.6	0.8	5.8
	385	368	8330	57.5	5.0	+1050	220.0	135.0	38.6	4.3	0.3	4.0
	385	397	18000	61.2	2.5	+ 242	195.0	130.0	33.3	N.A.	N.A.	N.A.
	376	399	13800	39.9	11.2	+ 25	190.0	125.0	29.0	N.A.	N.A.	N.A.
4442	350	312	2870	26.5	14.0	+ 89	172.0	132.0	23.0	N.A.	N.A.	N.A.
	340	332	14110	27.0	11.0	+ 145	178.0	124.0	30.3	N.A.	N.A.	N.A.
	330	326	26900	26.0	10.0	+ 160	194.0	130.0	33.0	N.A.	N.A.	N.A.
	316	296	4670	29.5	10.0	+ 195	194.0	137.0	34.5	N.A.	N.A.	N.A.

Data concerning 14 sequences of the *out of phase* cyclic pattern induced in coronary venous pressure, by an atrio-ventricular asynchrony.
(same symbols as in Table I)

The period of the cycles depended on the difference between the auricular and ventricular rates, whether the auricular rate was controlled or it was spontaneous. When the auricular rate was not controlled, the variations in the spontaneous sinus rats made this check difficult, as the period of the cycle created by atrio-ventricular asnchrony was not constant. The auricular periods ranged from 287 to 406 ms, and the ventricular from 297 to 399 ms. The rhythm of the cyclic changes varied from 1.680 to 40.3 ms, which corresponds to a frequency varying between 35.7 to 1.5 per mm. The fall in the left ventricular pressure varied between 20 to 90 mm *Hg*, which represented a drop of 16.6 to 42.1 per cent of the initial value (average 30%). Higher the initial level of ventricular pressure, more pronounced was the decrease.

The right auricular pressure rose when the left ventricular pressure fell, thus the two being out of phase with each other (Fig. 2). The analysis of the tracings showed that this rise was due to an increase in the amplitude of the "a" wave of the auricular pressure, On the other hand the "v" wave of the auricular pressure showed an insignificant change (Fig. 3). The spontaneous maximal right auricular pressures ranged from 0.3 to 6 mm Hg rising to 4.3 to 18.0 mm Hg during the course of the cycle.

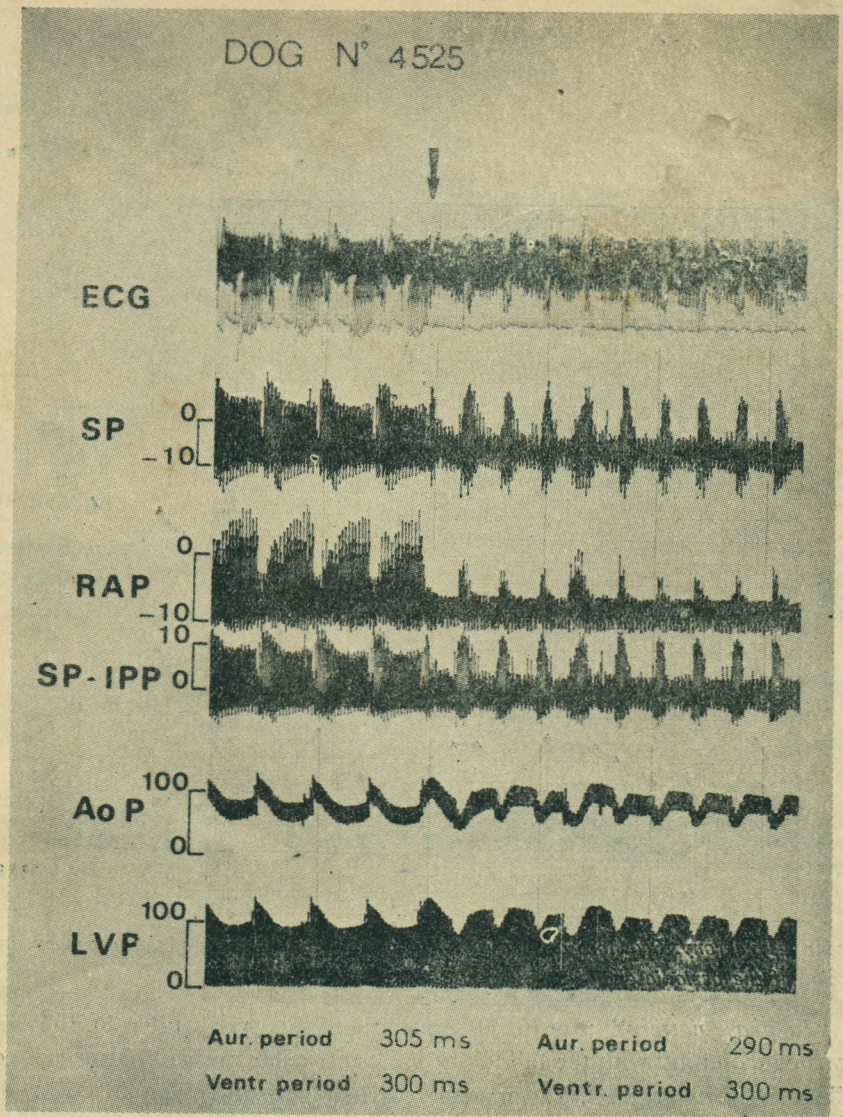

Fig. 2

Cyclic changes produced by atrioventricular asynchrony at two different atrioventricular ratios. In the part to the right of the arrow the auricles are driven at a higher rate than the ventricles, corresponds to an in-phase pattern of the coronary sinus pressure. The left part corresponds to the establishment of a new cycle due to a slower auricular rate and is demonstrative of an out of phase pattern of the coronary sinus pressures.

Whatever the site of registration of the coronary venous pressure the result was separable into two groups depending on whether the variation of the coronary venus pressure was *in* or *out* of phase with the left ventricular pressure changes.

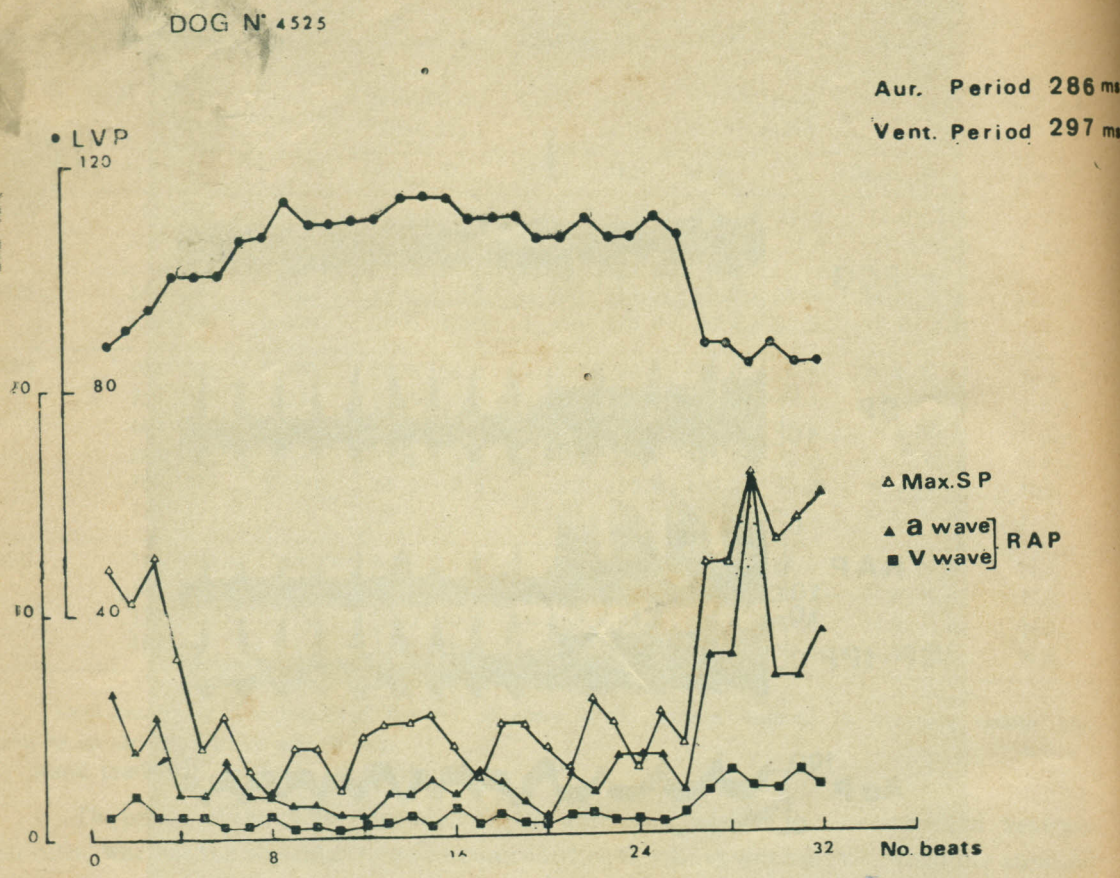

Fig. 3

Atrioventricular asynchrony. Beat to beat analysis of an out of phase pattern of the coronary sinus pressure. (SP) for one cycle of the periodic change.

a) "*In phase pattern*" : In the 11 sequences of this type (table 1), the fall in the sinus pressure was concomitant with the fall in the left ventricular pressure (first part of fig. 2 and fig. 4). The fall in the C. V. pressure varied between 5 to 21 mm Hg which represents a fall of—33 to—122%. This fall in the coronary venous pressure was observed in its ventricular borne "v" waves.

b) "*Out of phase pattern*" : In the 14 sequences of this type (table II) it was found that the sinus pressure rose when the left ventricular pressure fell (second part of fig. 2 and fig. 3). The rise in the coronary venous pressure was between 8 to 58.7 mm Hg (+25 to +1050%). In these records the rise in coronary venous pressure corresponded to the fusion of the auricular and ventricular borne pressure waves (Fig. 5) though the maximal amplitude of change was sometimes much higher than the arithmetic sum of the two waves. The coronary venous pre-

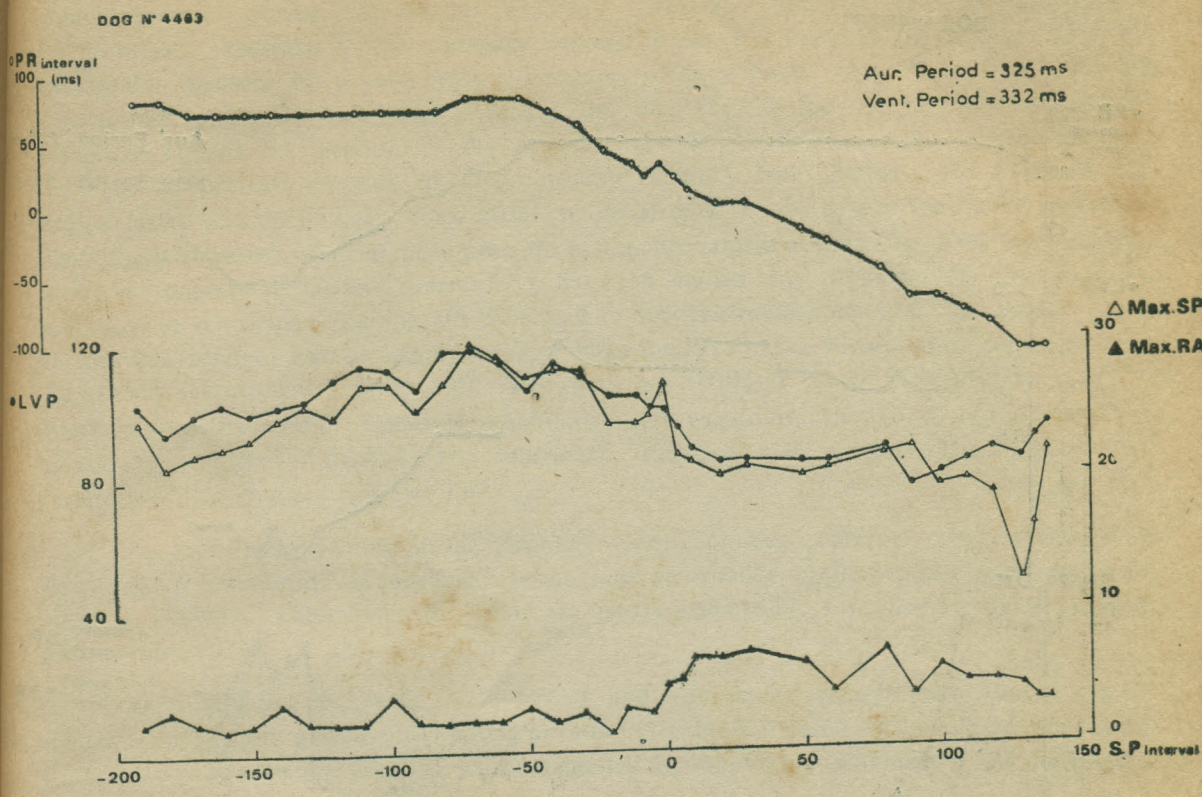

Fig. 4

Atrioventricular asynchrony. Beat to beat analysis of an inphase pattern of the coronary sinus pressure (SP).

ssure started rising progressively from the moment the E.C.G. showed shortening of the P R interval, the maximum occurring when the ventricular event preceded the auricular. The relationship between the auricular and ventricular complexes and the respective waves of the coronary venous pressure ("a and "v" waves) remained constant throughout the full sequence (Fig. 5).

It is noteworthy that when an *out of phase* relationship was found, the change in venous pressure preceded (by a few beats) the change in the ventricular and auricular pressure which were concomitant with each other. This was in striking contrast to the simultaneous changes in the coronary venous, auricular and ventricular pressures when an *in phase* relationship was obtained.

DISCUSSION

The amplitude of the fall in the left ventricular pressure during AV-asynchrony was very marked. The fact that this fall was regular and progressive for a given instance and was present in every experiment tends to rule out abnormal ventricular depolarization as a major

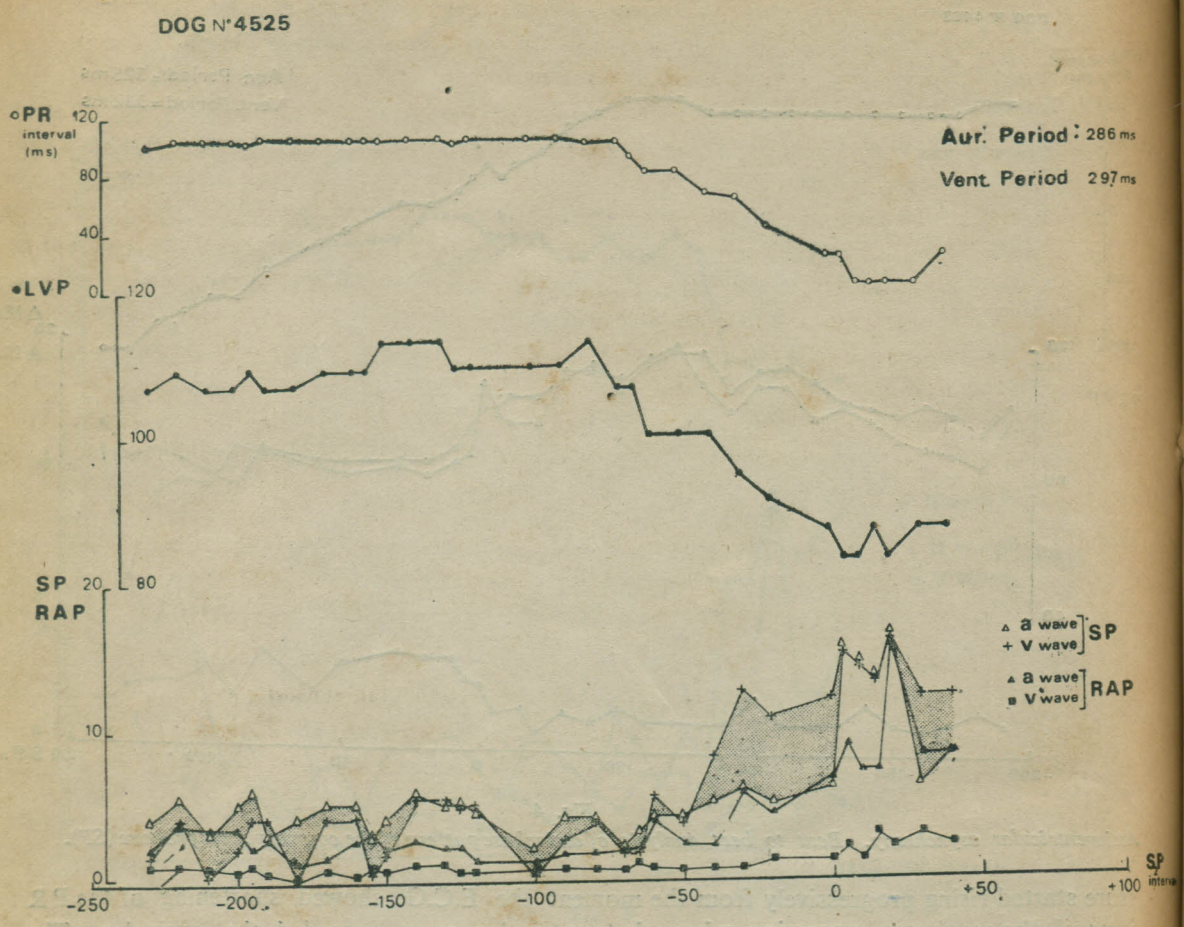

Fig. 5

Atrioventricular asynchrony. Beat to beat analysis of a cycle with an out of phase pattern. Maximal sinus pressure was obtained when "a" and "v" waves of the tracing were summed. In abscissa the S_2P intervals are figured.

cause for this change. Moreover it was noticed that the systolic left ventricular pressure during the part of the cycle with a PR interval within normal limits remained at the same level as it was during the control period.

The inphase pattern or drop in the coronary venous pressure together with the fall in the ventricular pressure could be simply understood as being due to dimunition of left ventricular contraction during ventricular depression.

The *out of phase* pattern of the coronary venous pressure has to be explained. The possibility of an atrial regurgitation when the PR interval is decreased or when a RP pattern is obtained is to be discussed. As there was no significant increase in the "v" waves of the auri-

cular pressure during the rise in coronary venous pressure this possibility can be excluded. Increase in the coronary venous pressure preceded the fall in the left ventricular pressure by a few beats, therefore, it is unlikely that dimunition in the myocardial contractile force and a consequent decrease in the intramyocardial vascular resistance caused an increase in the coronary blood out flow and was responsible for the observed pressure increase in the coronary venous bed. Samet and Bernstein (3) demonstrated that the properly timed atrial systole permits an increase in ventricular contractility without elevation of atrial mean pressure by the mechanism of the atrial 'Kick' contribution to the ventricular end-diastolic pressure. It has been shown by Brockman (1) that in dogs submitted to ventricular coupling, the difference in atrioventricular pressure was greatest at a normal PR interval and decreased rapidly when the PR interval decreased or increased. In the present experimental setup with AV-asyschrony (without A—V block) at various PR intervals (positive and/or negative), a similar pattern was found, though during simultaneous measurement of the end-diastolic left ventricular and auricular pressures the amplitude of variation of this gradient was very low.

The *out of phase* changes in the coronary venous pressure,therefore, suggest that they were due to summation of auricular borne and ventricular brone pressure waves and were primary in type. These changes could be partly responsible for changes in myocardial contraction.

It is to be noted that when the auricular and ventricular periods were almost strictly identical resulting in a cycle of very long duration (viz. more than 30 s.) the fall in ventricular pressure was not sustained (Fig. 6), which suggested an adaptable mechanism of the postulated receptors of the coronary venous bed.

It is conceivable that the relationship between coronary venous pressure at sites corresponding to the out of phase pattern observed in AV dissociation and cardiac contraction, is due to a refex mechanism, the receptors of which could be of an adaptable type, their normal adequate stimulus being directly related to the contraction of the different parts ef the myocardium and thus originating a feed-back regulatory loop. Their site is probably in or near the wall of the coronary veins and their threshold could be relatively high. The different portions of the coronary venous bed along the whole myocardium have not been extensively studied, but the present data suggest that the baro or tensioreceptors could be spread over a long portion of the coronary venous bed.

SUMMARY

The coronary sinus, right auricle and left ventricle of mongrel dogs were catheterised under fluoroscopy. Two stimulation catheters were then guided, one close to the sinus node and the second at the left ventricular inflow tract. AV-asynchrony was induced with the auricular and ventricular periods very close to each other and cyclic changes were observed in the left ventricular pressure when simultaneous recording of left ventricular, coronary venous, and right auricular pressures were done. A some

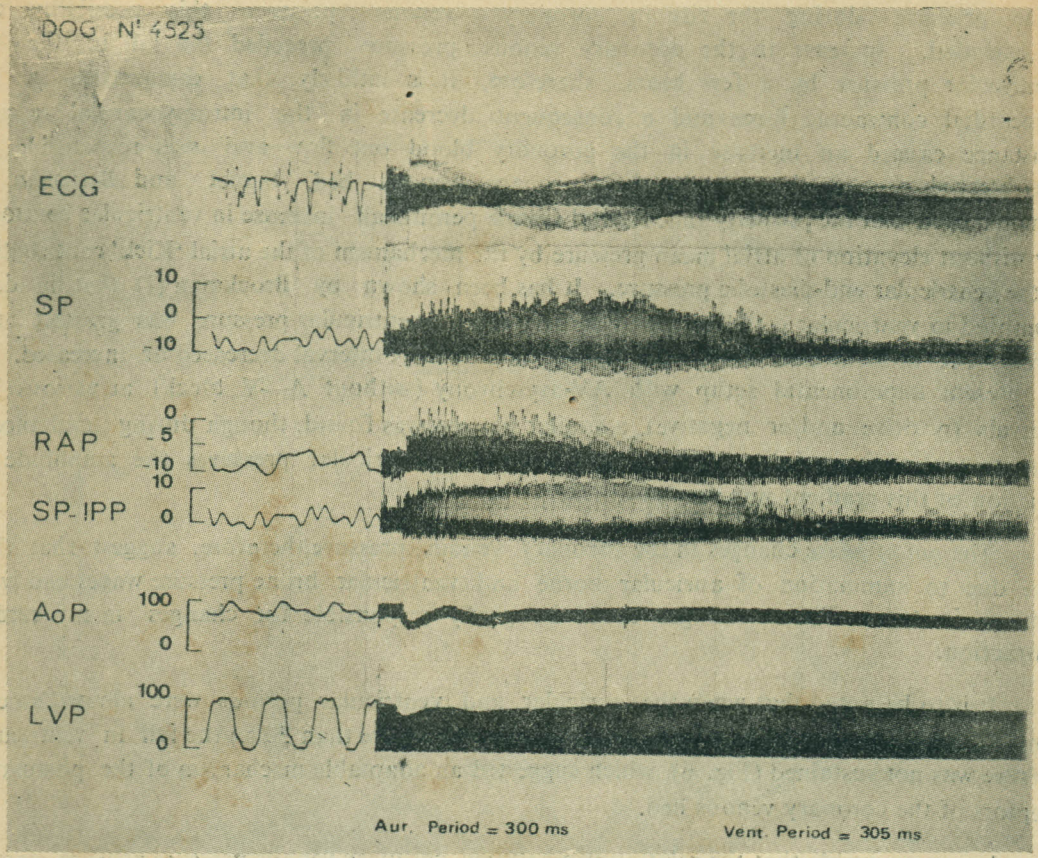

Fig. 6

Atrio-ventricular dsynchrony. Phenomenon suggesting an adaptation of the postulated receptors of the coronary sinus. The fall in ventricular pressure was not sustained.

sites of registration the coronary venous pressure changes were preceeding and were *out of* phase with the left ventricular pressure changes. The former were not accounted for by the concomitant changes in the right atrial pressure. The results suggest the possible role of a reflex mediated control mechanism of myocardial contractility, originating from presso-receptors of an adaptable type which are localized in the venous part of the coronary bed.

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