Regional Dynamic Behavior of the Total Right Ventricle[1] (37098)

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The structure-functional dynamics of the right ventricle differ importantly from the left ventricle. By cauterizing the right ventricular free wall, Kagan concluded that the right ventricular pressure generation was a function primarily of the septum (1). This view is compatible with the concept that the right ventricle is primarily a volume pump. Recently however, significant pressure gradients have been demonstrated from inflow to outflow tracts (from sinus to conus) (2) and these differences have been related to the regional dynamic behavior (3) as determined by architectural differences between the sinus and conus (4). However, the close dynamic relationships between the free wall and septum have been completely neglected when considering right ventricular function. Recent measurements of intramyocardial pressures within the interventricular septum demonstrate significant regional contractile changes under neural and humoral control. This report describes the behavior of both the free wall and the septal components of the right ventricle, and the analysis of the relative independence of this behavior with respect to left ventricular function.

Methods. Sixteen open-chest dogs, weighing 18–24 kg, under phencyclidine hydrochloride (2 mg/kg im) and α-chloralose (80 mg/kg iv) anesthesia were studied during positive pressure respiration. A standard limb lead electrocardiogram was utilized and two button catheters (2) placed in the lateral wall of the right ventricular sinus and conus connected to P23Db Statham transducers. Two Model SA-SA-M-7BW Scientific Advances subminiature pressure transducers were placed halfway through the right ventricular myocardium, one in mid-sinus and the other in mid-conal areas. These intramyocardial pressure (IMP) recorders have a pressure range of −100 to +500 mm Hg and a frequency response to 1000 Hz; the frequency response of the entire recording system was linear to 50 Hz. An epicardial slit allowed the device to be inserted into the mid-wall region so that its thin axis was at right angles (long axis parallel) to the epicardium; the dimensions of the transducer were 2.5 × 3 × 1 mm. The linear calibration properties of the Scientific Advances pressure transducer compared to a P23Db Statham transducer has been detailed elsewhere (4).

In 11 other open-chest dogs, under inflow occlusions two Walton-Brodie strain gauge arches were applied to the right side of the interventricular septum. The right basal septal force gauge was placed anterior to the conal papillary muscle parallel to underlying fibers, and the apical gauge was sutured to the right ventricular sinus and left ventricular anterior epicardial surfaces. A model SA-SA-M-7BW Scientific Advances subminiature pressure transducer was placed in the midwall of the septum and two Statham P23Db pressure transducers were employed to measure left and right ventricular cavitary pressures (IVP). In the last group of experiments comprising 10 dogs, two SA-SA-M-7BW Scientific Advances subminiature pressure transducers were employed to monitor the intramyocardial pressures (IMP) on the right and left sides of the septum simultaneously. Via a tunnel made from the anterior septal epicardium the transducers were placed subendocardially in the right and left sides of the septum. The position of these devices, checked on autopsy, was 10–15% and 75–85% through the septal wall proceeding from the right to left side. Concomitant pressures

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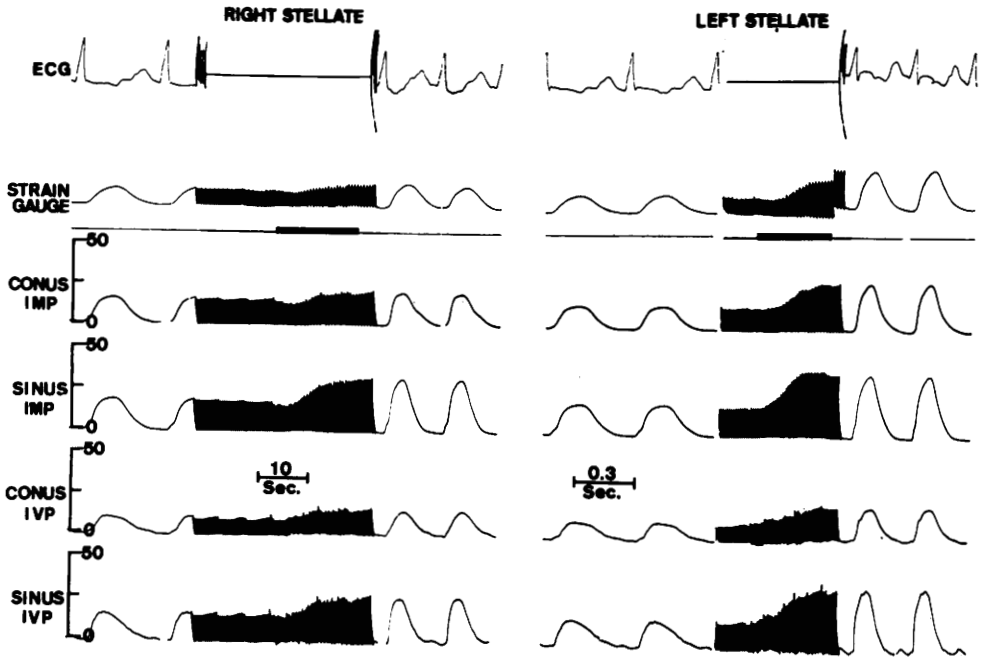

FIG. 1. The effects of right stellate stimulation (left panel) and left stellate stimulation (right panel) is shown as recorded via a strain gauge arch on the sinus epicardium, intramyocardial pressures (IMP) in the conus and sinus, and intraventricular pressures (IVP) in the conus and sinus regions of the right ventricle. The sudden shift in base line in strain gauge trace immediately following left stellate stimulation is a technical artifact.

were recorded from the right and left ventricular cavities via Statham P23Db pressure transducers; Walton-Brodie gauges were sutured to the epicardial surfaces of the right ventricular sinus and the anterior left ventricle. Both stellate ganglia were isolated, and stimulation was achieved with a Grass square-wave stimulator (Model S5) at parameters of 4 V, 5 msec, and 10 Hz. Augmentation of contraction was also achieved by infusion of isoproterenol (1 μg/kg), norepinephrine (0.5 μg/kg) or phenylephrine (0.05 mg/kg); digital coarctation of the pulmonary artery or aorta were performed on all groups as was the rapid infusion of 50 ml of saline into the right ventricle in group 4 animals. Stimulation of the peripheral end of the severed cervical vagosympathetic (4 V, 5 msec, and 10 Hz) was also performed. At the termination of the experiment, the locations of all recording devices were checked upon autopsy; all records were obtained via a type R dynograph. In two experiments the function of the septal IMP devices was checked in an intact preparation, after right ventricular decompression while on a bypass oxygenator, or after left and right ventricular chamber decompression while on bypass, in order to determine the effect of chamber pressure on the septal IMP recordings.

Results. Figure 1 represents the effects of right and left stellate stimulation upon a lead II ECG, a sinus epicardial strain gauge (strain gauge), conal and sinus intramyocardial pressures (conus IMP, sinus IMP) as well as right ventricular chamber pressures in the conus (conus IVP) and sinus (sinus IVP) regions. Right stellate ganglion stimulation increased the sinus IVP from 15 to 28 mm Hg and the conus IVP from 12 to 15 mm Hg; the sinus IMP rose from 18 to 32 mm Hg and the conal IMP from 15 to 23 mm Hg. During left stellate ganglion stimulation the sinus IVP rose to 35 mm Hg and the conal IVP to 20 mm Hg; concomi-

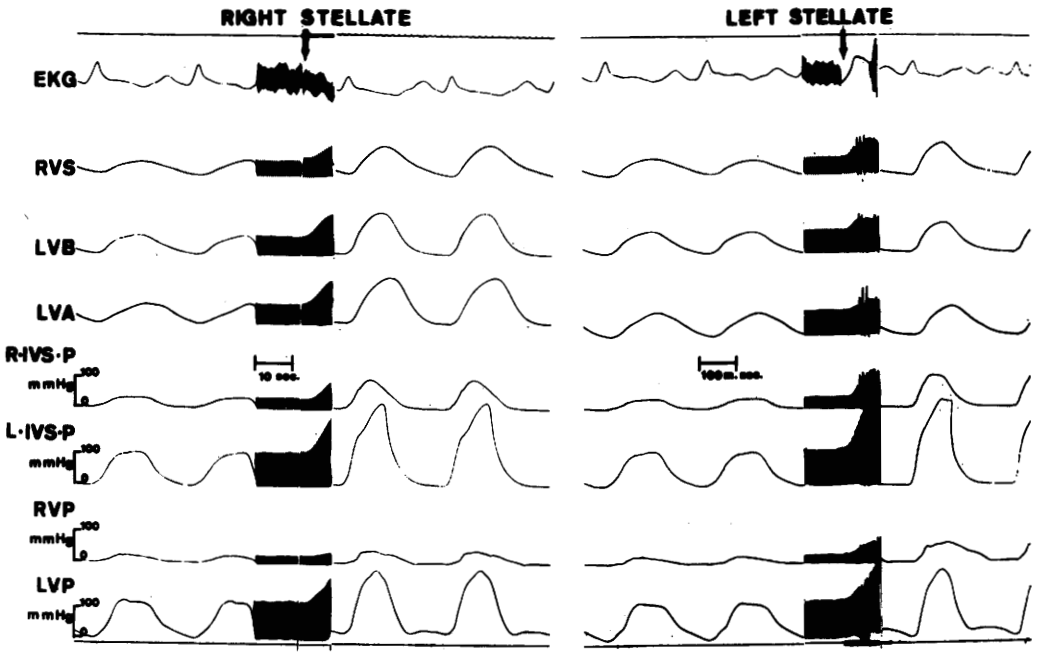

FIG. 2. Right and left stellate ganglia stimulation increases in right sinus force (RVS), left ventricular epicardial anterior basal force (LVB), left ventricular epicardial apical force (LVA), the septal intramyocardial pressures of the right side (R-IVS-P) and the left side (L-IVS-P), as well as right (RVP) and left (LVP) chamber pressures are shown.

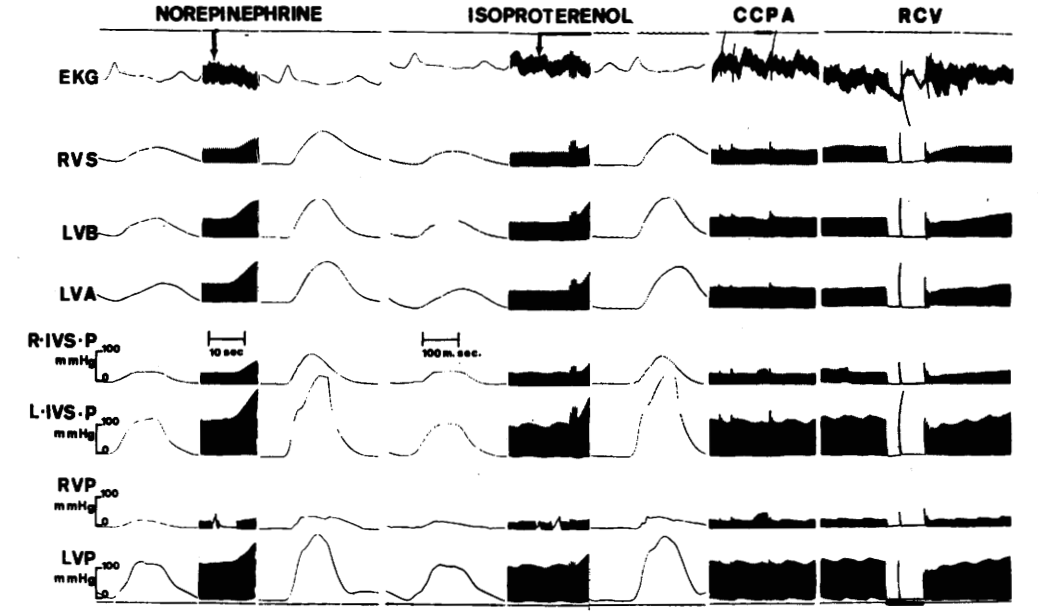

FIG. 3. In the same animal as in Fig. 2, with the same recorded areas of the heart, the effects of norepinephrine and isoproterenol infusions are compared with cross clamping of the pulmonary artery as well as stimulation of the efferent right cervical vagus. Note the augmentation in the R-IVS-P during coarctation of the pulmonary artery.

TABLE I. Myocardial and Chamber Pressures in the Right Ventricle.[a]

Group	No. of expts	Heart rate	Peak intraventricular pressure (mm Hg)			Peak intramyocardial pressure (mm Hg)			Peak intraventricular dP/dt (mm Hg/msec)			Peak intramyocardial dP/dt (mm Hg/msec)		
			Sinus	Conus	$p<$	Sinus	Conus	$p<$	Sinus	Conus	$p<$	Sinus	Conus	$p<$
Control	16	102 ± 6	19 ± 2	16 ± 2	NS	22 ± 3	26 ± 5	NS	2.2 ± 0.6	1.7 ± 0.4	NS	0.5 ± 0.1	0.6 ± 0.1	NS
Right stellate stimulation	16	135 ± 6	42 ± 7	21 ± 5	0.001	41 ± 4	49 ± 8	NS	11.2 ± 3.2	3.5 ± 0.8	0.01	3.3 ± 0.7	6.3 ± 0.8	NS
Left stellate stimulation	16	127 ± 9	43 ± 7	16 ± 2	0.001	46 ± 6	61 ± 7	0.05	68.3 ± 5.2	35.3 ± 2.9	0.05	3.0 ± 0.2	7.8 ± 1.6	NS
Norpinephrine	16	138 ± 10	59 ± 6	20 ± 3	0.001	73 ± 12	79 ± 11	NS	37.1 ± 6.2	33.9 ± 5.8	NS	8.5 ± 2.4	11.6 ± 4.8	NS
Isoproterenol	8	132 ± 3	52 ± 1	16 ± 2	0.001	76 ± 6	78 ± 10	NS	87.5 ± 8.9	74.7 ± 15.6	NS	24.3 ± 0.5	40.0 ± 2.1	NS
Cross clamp pulmonary artery	6	105 ± 2	40 ± 6	38 ± 4	NS	40 ± 4	54 ± 1	0.01	6.2 ± 0.8	2.5 ± 0.7	NS	0.9 ± 0.4	1.2 ± 0.5	NS

[a] ± = SEM.

tantly the sinus IMP was elevated to a peak pressure of 37 mm Hg, while the conal IMP rose to 29 mm Hg, demonstrating a considerable pressure gradient from wall to chamber in the conus region.

Figures 2 and 3 illustrate the simultaneous alterations in intramyocardial pressure immediately beneath the right and left endocardial surfaces of the interventricular septum. Figure 2 demonstrates responses to both right and left stellate stimulation upon standard EKG, right ventricular sinus epicardial contractile force (RVS), left ventricular epicardial base (LVB) and apex (LVA) forces, right interventricular septal muscular pressure (R-IVS-P), left septal muscular pressures (L-IVS-P), right (RVP) and left chamber ventricular pressures (LVP). Right septal intramyocardial pressure in control states was 28 mm Hg while that of the left side of the septum was 105 mm Hg. Concomitant right ventricular pressure was 20 mm Hg while left ventricular chamber pressure was 120 mm Hg. Right stellate stimulation induced a more nearly synchronous onset of rises in force and pressures; the right septal IMP increased to 95 mm Hg and left side IMP to 268 mm Hg, while RVP rose to 40 mm Hg and LVP to 230 mm Hg. Left stellate stimulation had even more profound effect on the pressures; the right septal IMP rose to 120 mm Hg, left septal IMP to 305 mm Hg, RVP to 65 mm Hg, and LVP to 245 mm Hg.

Figure 3 represents the effects of norepinephrine, isoproterenol, cross clamping of the pulmonary artery, and right cervical vagal stimulation in the same experiment as illustrated in Fig. 2. Norepinephrine augmented right septal intramyocardial pressure from 30 to 100 mm Hg, and the left side of the septum from 125 to 285 mm Hg. Force gauges demonstrated a fairly uniform augmentation. The same type of responses were elicited by infusion of isoproterenol. Right septal IMP rose to 96 mm Hg while left septal pressure rose to 290 mm Hg. Cross clamping of the pulmonary artery resulted in minimal force changes, however the right septal IMP rose from 30 to 50 mm Hg, the left sided IMP dropping from 125 to 115 mm Hg; right

TABLE II. Onset of Rise in Intramuscular and Chamber Pressures (msec) After the Q Wave.[a]

Group	No. of expts	Q-Sinus IMP	Q-Conus IMP	Q-Sinus IVP	Q-Conus IVP
Control	16	47.0 ± 4.4	57.6 ± 3.9	79.2 ± 4.7	80.0 ± 5.1
Right stellate stimulation	16	39.0 ± 4.0	42.9 ± 2.1	57.1 ± 3.2	56.5 ± 3.3
Left stellate stimulation	16	39.5 ± 4.0	37.6 ± 4.0	59.3 ± 3.9	58.2 ± 2.0
Norepinephrine	16	37.5 ± 2.8	34.8 ± 3.3	56.5 ± 4.8	52.5 ± 3.8

[a] ± = SEM; IMP = intramyocardial pressure; IVP = intraventricular pressure.

ventricular chamber pressure rose from 18 to 50 mm Hg, without change in the left cavitary pressures. Stimulation of the right distal cervical vagus (RCV) resulted in depression in force and pressure in all recorded regions as evidenced immediately after the stimulation ceased. Right septal systolic IMP fell from 22 to 20 mm Hg, and left sided IMP from 90 to 80 mm Hg.

Table I presents data collected from 16 animal experiments. Control sinus intraventricular systolic pressure averaged 19 ± 2 mm Hg, while that in the conus was 16 ± 2 mm Hg; peak sinus IMP was 22 ± 3 mm Hg and conus IMP was 26 ± 5 mm Hg. The differences between peak intramyocardial and peak chamber pressures were not significant ($p < 0.2$). The rates of development of the regional pressures are given in the four right columns. The rate of pressure development within the myocardial wall and the cavity were statistically similar. Stimulation of the right stellate ganglion augmented intramyocardial pressures in both regions (p value of differences < 0.05), the rate of pressure generation being greater in the conus; whereas the sinus wall and chamber pressures were statistically similar, they were statistically different in the conus. Left stellate ganglion stimulation augmented conus IMP considerably more than that in the sinus region, and rate of pressure development was augmented even more profoundly than during right stellate stimulation. During the latter manipulation the conus and sinus wall pressures were similar, whereas intraventriculur pressures were statistically different. Norepinephrine and isoproterenol augmented intramyocardial pressure uniformly in both regions, in sharp contrast to chamber pressures, where they were again statistically different; the rate of pressure development was more augmented by norepinephrine, particularly in the conus. Increase in IMP generation via pulmonary artery cross clamping was uniform, statistically, for both IVP and IMP; however the rate of IMP development was minimally augmented. Note that during pulmonary artery occlusion the myocardial pressures were similar and the right ventricular cavitary pressure gradient was abolished. Although the comparison of sinus myocardial and chamber pressures showed no significant difference during all inotropic interventions, the conal intracavitary pressures were always significantly lower than those in the sinus. Intramyocardial pressures, on the contrary, were invariably higher in the conus musculature.

Table II represents the time sequence of onsets of pressure generation in the two regions of the free wall of the right ventricle as related to the Q wave of a standard EKG. In the control group the sinal IMP developed pressure first followed by conal IMP; cavity pressures developed later. Each of the positive inotropic interventions resulted in shortening of the interval between initial deflection of the Q wave and the onset of pressure rise within the muscular walls or cavity compared to control values. A statistical difference ($p < .001$) existed between Q and intramyocardial pressures in sinus and conus regions under control circumstances; this difference disappeared with positive inotropic intervention when pressures rose nearly synchronously in all instances.

Table III contains the results of experimental interventions upon the force generation by the right side of the septum, along with septal intramyocardial pressure, right and left

TABLE III. Changes in Contractile Force, Intramyocardial Pressure (IMP), and Chamber (Right and Left Ventricles) Pressures from Control Values.[a]

Group	No. of expts	Heart rate	Contractile force (%)				Septal IMP (mm Hg)	Right vent. press. (mm Hg)	Left vent. press. (mm Hg)
			Right vent. sinus	Right septal base	Right septal apex	Ant. left vent. epi.			
Control	11	164 ± 9	100	100	100	100	86 ± 10	21 ± 2	111 ± 6
Right Stellate	11	232 ± 10	140 ± 13	169 ± 15	180 ± 20	145 ± 27	181 ± 25	53 ± 9	150 ± 17
Left Stellate	11	190 ± 10	190 ± 20	192 ± 23	183 ± 25	200 ± 36	184 ± 26	69 ± 8	208 ± 21
Isoproterenol	11	221 ± 7	230 ± 30	246 ± 22	208 ± 25	245 ± 45	236 ± 20	73 ± 7	223 ± 26
Norepinephrine	11	183 ± 21	210 ± 30	215 ± 23	208 ± 25	203 ± 63	213 ± 22	63 ± 10	202 ± 20
Phenylephrine Cross-clamp	10	173 ± 7	180 ± 30	138 ± 15	167 ± 33	145 ± 36	174 ± 18	46 ± 5	192 ± 21
Pulmonary artery	10	149 ± 11	120 ± 20	84 ± 8	125 ± 25	100 ± 20	89 ± 14	43 ± 5	93 ± 7
Cross-clamp Aorta	10	161 ± 8	110 ± 10	107 ± 15	116 ± 23	82 ± 8	137 ± 16	27 ± 4	179 ± 11
Right vagus	8	44 ± 11	60 ± 10	69 ± 7	83 ± 16	90 ± 9	66 ± 9	18 ± 3	76 ± 7

[a] ± = SEM.

chamber pressures, and right and left ventricular epicardial forces. Right stellate stimulation augmented force most in the apical right septum, as opposed to left stellate stimulation when the augmentation was rather uniform in the right septum as well as epicardial segments. Norepinephrine augmented the septal forces uniformly and similarly to the right ventricular sinus and the left ventricular epicardium, whereas isoproterenol caused augmentation in the right septal base more than in the apex. Phenylephrine augmented the apical septum more than the basal region as did cross clamping of the pulmonary artery, although in the latter instance minimal augmentation of force in the apex of the right septum was accompanied by a decrease in force in the basal area. Aortic coarctation caused little change in right sided septal force and vagal stimulation was accompanied by generalized depression of force, again primarily on the basal portion of the right septum. Table IV depicts the onset time of force or pressure generation after the Q wave of a standard EKG. Statistically all the onset times during control periods were synchronous, excepting septal intramyocardial pressure; however, with right stellate stimulation the right septum contracted before the sinus region ($p < .001$), and before the left ventricle epicardium ($p < .01$). With left stellate stimulation the right septal apex contracted before the base ($p < .001$), and at the same time as the anterior left ventricle. Similar patterns of contraction occurred with isoproterenol and norepinephrine augmentation, in as much as the apical right septum contracted before the basal region, the latter contracting synchronously with the right ventricular sinus epicardium. Vagal stimulation was followed by the basal region contracting before the apical right septum, the latter area contracting before the right ventricular sinus. In all experimental procedures the left ventricular pressure began to rise before that in the right ventricle.

Table V shows the results of intramyocardial pressures recorded simultaneously from the left and right sides of the septum, as well as right and left chamber pressures dur-

TABLE IV. Onset of Change in Contractile Forces and Pressures After the Q Wave (msec).[a]

Group	Right vent. sinus	Right septal base	Right septal apex	Ant. left vent. epi.	Septal IMP	Right vent. press.	Left vent. press.
Control	38.5 ± 3.7	35.0 ± 3.2	28.0 ± 3.2	35.0 ± 4.6	18.5 ± 1.8	52.0 ± 4.2	22.5 ± 3.1
Right stellate	33.5 ± 2.7	25.5 ± 2.5	23.6 ± 3.0	30.1 ± 3.3	15.5 ± 3.0	38.5 ± 4.1	15.0 ± 3.7
Left stellate	31.0 ± 3.1	33.5 ± 5.3	24.0 ± 4.6	26.3 ± 6.9	19.0 ± 3.6	32.0 ± 2.8	20.0 ± 6.0
Isoproterenol	33.5 ± 2.9	34.5 ± 4.6	26.2 ± 2.4	30.0 ± 7.4	18.5 ± 3.3	37.5 ± 2.7	18.7 ± 3.5
Norepinephrine	35.0 ± 1.0	32.5 ± 4.0	24.0 ± 3.3	33.8 ± 8.5	17.5 ± 4.0	39.0 ± 4.7	19.5 ± 4.0
Right vagus	56.3 ± 7.5	41.3 ± 4.3	47.5 ± 11.1	45.4 ± 8.6	27.5 ± 5.2	60.0 ± 11.6	36.3 ± 11.4

[a] ± = SEM.

ing a variety of interventions. Epicardial forces on the left and right ventricles were also recorded in order to correlate the data with the previous groups of experiments. Right septal IMP was much less than that recorded from the left ($p < .001$), although it was higher than the pressure recorded in the right ventricular cavity. With right stellate stimulation the septal IMP increased, the right side to an average of 91 mm Hg and the left septal IMP rose to 184 mm Hg; the right septal IMP rose to 91 mm Hg and the left septal IMP to 211 mm Hg with left sided stimulation. Chamber pressures increased and once again the septal IMP was greater than that of the corresponding ventricular pressures. Augmentation of contraction via isoproterenol and norepinephrine developed similar transseptal IMP gradients. With cross clamping of the pulmonary artery the right septal IMP, not the left, was increased; with aortic occlusion both septal IMPS were elevated, particularly the left side. Vagal stimulation diminished both septal intramyocardial pressures, although infusion of 50 ml of fluid into the right ventricle minimally elevated right chamber pressures, it had little effect upon systolic right septal IMP.

Table VI contains the times after the Q wave of onsets of contraction and pressure of the same experiments as in Table V. Note that the right septal IMP started at the same time as right sinal force, and the left septal IMP at the time of left ventricular force generation.

Discussion. The right ventricle consists of a free wall and a septal wall, the sinus region constituting the bulk of the musculature (5); the conus region is relatively small and becomes very scanty where it fuses with the septum. The right ventricular septal musculature, although frequently considered to be quite separate from the left ventricle (6), actually consists of muscle fascicles which are intimately related to those of the left ventricle. These right ventricular septal fascicles are generally oriented in an apex to base direction (5). It is presumably this septal muscular mass which developed right ventricular pressures after extensive cauterization of the right ventricular free wall in Kagan's experiments (1). The sinus and conus intramyocardial pressures in control states are similar, and slightly above those in the underlying ventricular cavity. When inotropic interventions increase ventricular contractility, sinal cavity pressures are elevated more than those in the conus; however, intramyocardial pressures demonstrate an equal augmentation in both regions (Table I). Inotropic intervention augments all muscular elements of the right ventricular free wall similarly, as has been previously reported when epicardial forces were measured (7). Note that when the cavity pressures were augmented during coarctation of the pulmonary artery (Table I) the increase in these pressures was similar for both the sinus and conus; intramyocardial pressures in these two free wall regions were similarly elevated.

A majority of the sinus free wall muscular fascicles have an oblique direction from apex to base with a major average radius of curvature approximating 4 cm, whereas the muscle fascicles of the conus are predominantly parallel and circumconal with an average radius of curvature of 0.8 cm (5). Assuming these radii to represent the major

TABLE V. Simultaneous Measurements of Contractile Force on Epicardial Segments of Right and Left Ventricles, Together with Intramyocardial Pressures in Both Right and Left Interventricular Septal, as Contrasted with Chamber Pressures.[a]

Group	No. of expts	Heart rate	Right sinus force (%)	Ant. left vent. force (%)	mm Hg			
					Right septal IMP	Left septal IMP	Right vent. press.	Left vent. press.
Control	10	151 ± 8	100	100	33.3 ± 5.1	98.3 ± 11.9	19.6 ± 1.6	117.8 ± 4.9
Right stellate	10	207 ± 11	213 ± 25	149 ± 9	90.5 ± 15.6	183.8 ± 25.1	41.7 ± 70	183.6 ± 14.5
Left stellate	10	176 ± 9	246 ± 21	178 ± 14	90.6 ± 13.5	210.6 ± 33.7	54.3 ± 9.2	200.6 ± 19.1
Isoproterenol	10	179 ± 12	264 ± 28	208 ± 21	109.3 ± 7.9	280.7 ± 25.1	54.3 ± 7.8	203.6 ± 16.0
Norepinephrine	10	187 ± 10	219 ± 19	203 ± 19	90.6 ± 9.9	218.9 ± 28.4	46.3 ± 9.1	192.8 ± 16.3
Cross-clamp pulmonary art.	10	152 ± 11	188 ± 19	116 ± 10	86.1 ± 9.9	98.3 ± 14.5	66.3 ± 6.8	103.9 ± 6.9
Cross-clamp aorta	10	155 ± 12	139 ± 23	137 ± 13	59.6 ± 17.8	168.9 ± 25.6	22.5 ± 1.9	189.4 ± 20.5
Right vagus	6	52 ± 6	60 ± 11	58 ± 3	26.7 ± 9.3	73.3 ± 1.7	19.0 ± 3.6	82.3 ± 9.1
Infuse right vent.	6	146 ± 17	130 ± 7	108 ± 10	35.0 ± 2.5	67.5 ± 12.5	22.5 ± 2.5	110.0 ± 5.0

[a] ± = SEM.

direction of muscle contraction and that the IMP recording device is sensing pressure at one depth, the muscle layers generating the pressure can be considered to be approximated by partial cylinders (4). Hoop tension, which can be estimated by a simple LaPlacian analysis calculated in peak systole (4, 8, 9), demonstrates an average sinal stress of 1.1×10^5 dyn/cm and conal stress of 2.7×10^4 dyn/cm during control states. During left stellate stimulation the sinal stress reaches a mean of 2.4×10^5 dyn/cm and conal stress 6.3×10^4 dyn/cm; isoproterenol infusion caused the regional stresses to become more evenly distributed as the sinus developed a stress of 4.0×10^5 dyn/cm and the conus a stress of 8.1×10^4 dyn/cm. It is apparent, utilizing these gross approximations, that the sinal muscular fascicles generate a greater stress during systole than the conal musculature although local intramyocardial pressures are quite similar in the two regions. The configuration of the muscular fascicles permits the conus to produce similar intramuscular pressures to those in the sinus with development of lesser regional stress; this may account in part for the ability of the conal fibers to overcome high pressures in the sinus chamber by interposing a large resistive element within the outflow tract so that pulmonary vascular pressure does not become excessively elevated during inotropism (2). The onset of IMP pressure development begins in the sinus and finishes in the conus during control states ($p < .001$) (7, 10–12). However, this sequence is abolished and occasionally reversed during positive inotropism (Table II). Right ventricular intramyocardial diastolic pressures also demonstrated differences in the sinus and conus portions of the free wall. In 10 experiments, right stellate stimulation and norepinephrine infusion resulted in reduction of end-diastolic pressure within the sinus musculature (-2.9 ± 0.56 mm Hg with p value $< .01$ for stellate stimulation and -3.05 ± 0.50 mm Hg with p value .001 for norepinephrine) while comparable pressures within the conus musculature increased ($+2.7 \pm 0.53$ mm Hg with p value .001 for stellate stimulation and $+2.8 \pm 0.4$ mm Hg

TABLE VI. Onset of Change in Contractile Force, Intramyocardial (IMP) and Intraventricular Pressures Following the Q Wave (msec).[a]

Group	Right sinus force	Left ant. vent. force	Right septal IMP	Left septal IMP	Right vent. press.	Left vent. press.
Control	39 ± 8	43 ± 5	36 ± 4	40 ± 5	56 ± 4	41 ± 6
Right stellate	23 ± 4	22 ± 3	22 ± 3	18 ± 3	39 ± 4	28 ± 4
Left stellate	31 ± 4	22 ± 3	25 ± 5	17 ± 3	35 ± 4	22 ± 3
Isoproterenol	24 ± 3	21 ± 3	17 ± 3	12 ± 1	37 ± 3	25 ± 3
Norepinephrine	31 ± 5	26 ± 4	28 ± 5	18 ± 2	42 ± 5	24 ± 3

[a] ± = SEM.

with p value of .01 for norepinephrine). Thus diastolic tone in the sinus is reduced while that in the conus region is elevated.

Contractile forces developed by the right ventricular septum (Table III) demonstrate a sequence of contraction originating in the apex and progressing to the base. This sequence is exaggerated during positive inotropism. Augmentations in septal force are equal in the base and apex during positive inotropism, but cross clamping of the pulmonary artery induced an increase at the apex and a decrease at the base. The right septal IMP during control states is considerably lower than that found near the epicardium of the anterior left ventricle (4).

Both sides of the septum show greatly increased IMP during sympathetic stimulation, the left side considerably more than the right. However, when the right ventricular pressure was increased by coarctation of the pulmonary artery the right septal pressure alone was elevated; aortic coarctation on the other hand elevated the intramyocardial pressures on both sides of the septum. When both ventricles were empty (total cardiopulmonary bypass), septal intramyocardial pressures were unaltered from control values, the gradient remaining from left to right. It appears that the right septal musculature only minimally effects the septal pressure, but the left exerts a distinct influence on the right.

Thus, the right ventricle can be thought of as contracting free and septal walls, both of which have a large sinal area and a smaller conal zone; the conal zone is particularly small where it attaches to the septum (5). The contractile behavior of these regions, associated with a unique architectural configuration, determines total right ventricular function—the generation of flow within a low pressure system (2, 13). The bulk of the right ventricle, the sinus, contracts to generate pressure, and the outflow musculature—the conus—acts as a pressure regulator.

Summary. Right ventricular regional contractile behavior demonstrated that the sinus free wall and interventricular septum contract synchronously and in similar fashions in response to physiological interventions. The conus musculature, being very scanty in the septal region, has its regulatory behavior confined primarily to the free wall. Conal intramyocardial pressures, unlike the sinus region, are always greater than underlying cavity pressures particularly during inotropic states, thus behaving as a pressure regulator to the outflow of the right ventricle.

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