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Regional Differences in Left Ventricular Wall Motion in the Anesthetized Dog

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Data regarding left ventricular function suggest that the extent of shortening may differ between regions. This study was undertaken to determine the effects of negative inotropic drugs used during anesthesia on different areas of the left ventricle. Forty mongrel dogs were anesthetized and instrumented for measurement of global and regional function. Regional function in the short axis of the basal and apical territories of the left ventricle was assessed by subendocardial sonomicrometry. Three different interventions were performed: In the first group 67% N_2O, replacing 67% N_2, was added to opiate anesthesia; in the second group halothane was given by stepwise increases in inspired concentration to 2%; in the third group verapamil (60 $\mu g \cdot kg^{-1} \cdot h^{-1}$) was infused during isoflurane anesthesia. Apical and basal segmental shortening were compared. During baseline conditions and with agents in concentrations that caused minimal myocardial depression (67% N_2O or 1.0% as opposed to 0.5% halothane) differences in systolic shortening between regions were statistically significant. Further myocardial depression affected the apex significantly more than the base: when substantial myocardial depression was induced by halothane (1.5 or 2%) or verapamil, differences in regional function were abolished. Thus, the apical region of the left ventricle is more dynamic and more sensitive to negative inotropic interventions than the basal region. This should be borne in mind when segmental myocardial function is evaluated. (Key words: Biology, physiology: heart, mechanics; regional function. Heart, contractility: regional. Heart, myocardial function: anesthetics; calcium channel inhibitors; opiates; ventricle, regional. Heart, ventricles: left ventricle; regional function.)

INVESTIGATORS INTERESTED in studying the effect of anesthesia on regional ventricular wall function in normal or compromised myocardium use sonomicrometry to measure segment length or wall thickness.[1–4] Uniform contraction of the left ventricle (LV) is usually assumed when LV performance is discussed. Liedtke *et al.*, in humans,[5] and Kong *et al.*, in experimental animals,[6] found evidence suggesting nonuniform contraction of the LV. Le Winter *et al.*[7] confirmed in open-chested dogs that the apical region is more active than the basal region. Transesophageal echocardiography demonstrated this phenomenon recently in humans.[8]

However, the differential effects of anesthesia and cardiovascular drugs on regional performance have not been studied extensively. Recognition of the differences in regional performance is essential to prevent errors in the interpretation of results when regional function is evaluated. Erroneous conclusions may be drawn if results from different segments are treated as if contraction was uniform throughout the left ventricle.

Most previous studies in this laboratory were concerned with myocardial ischemia and involved critical constriction of coronary arteries. In the control stages of these studies, differences in apical and basal segment shortening, before critical coronary constriction was applied, were present. However, these differences were not analyzed in detail because they were not considered relevant to the particular studies at the time. In this study, data selected from several previous studies,[2,9–11] and unpublished data obtained before critical constriction of coronary arteries had been applied, were analyzed to establish whether there are significant differences between the extent of apical and basal LV myocardial shortening under different conditions of general anesthesia and, if this was confirmed, whether negative inotropic interventions have a differential effect on normal apical *versus* basal myocardium.

Methods

INSTRUMENTATION

The studies were undertaken in accordance with the conditions of the Cruelty to Animals Act (1876) and the Animals (Scientific Procedures) Act (1986) of the United Kingdom. Forty mongrel dogs of either sex, weighing between 14 and 30 kg, were premedicated with morphine sulfate (1 $mg \cdot kg^{-1}$). Anesthesia was induced with sodium thiopental (10 $mg \cdot kg^{-1}$). The trachea was intubated, and constant volume intermittent positive pressure ventilation was instituted with 33% oxygen (O_2) in nitrogen (N_2) at a rate of 12 breaths/min, tidal volume 30 $ml \cdot kg^{-1}$. Carbon dioxide (CO_2) was added to the mixture to maintain

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end-tidal CO_2 at 5.3%. Anesthesia was maintained during surgical preparation with halothane 0.7–1.5%, supplied *via* a Fluotec® vaporizer (Cyprane, Keighly, England) calibrated with a refractometer. Temperature, monitored at the midesophagus, was maintained between 37° and 38° C by a heating element incorporated in the operating table.

An intravenous cannula was inserted *via* the femoral vein into the inferior vena cava for infusion of 0.9% saline at 37° C at a constant rate of 4 $ml \cdot kg^{-1} \cdot h^{-1}$. The left common carotid artery was exposed, and a rigid 8-Fr (2.76 mm outside diameter) polyethelene catheter was advanced to within 1 cm of the aortic valve for measurement of systemic arterial pressure with a Statham® pressure transducer and for blood sampling.

A left thoracotomy was performed, the fifth and sixth ribs removed, and the heart exposed and suspended in a pericardial cradle. A rigid 8-Fr cannula was inserted *via* a stab wound in the apical dimple into the left ventricle. LV pressure was recorded with a Stratham® pressure transducer connected to this cannula. An umbilical cannula was inserted *via* the outflow tract of the right ventricle into the pulmonary artery for determination of cardiac output, with the use of indocyanine green dye. The aortic root was dissected free of its fat pad and an appropriately sized electromagnetic flow transducer (Transflow 601®, Skaler Medical, Delft, Holland) was placed around it and connected to a flowmeter (SEM 230®, SE Laboratories, Feltham, United Kingdom).

The left anterior descending coronary artery (LAD) was dissected free distal to the second major diagonal branch. A 3–0 woven Dacron® suture was placed loosely around the artery in 31 dogs. A 2-mm electromagnetic flow probe (see above) was placed around the LAD in 29 dogs. A pneumatic occluder was positioned distal to the flow probe in 29 dogs. Previous observations in this laboratory showed that dissection of the LAD had no significant effect on regional wall function.§

Regional Myocardial Function

Sonomicrometry was used to evaluate regional myocardial performance. The principles behind this technique are well described.[12–14] Two pairs of piezo electric crystals, components ±1 cm apart, were implanted into subendocardial myocardium. One pair was positioned in the anteroapical region (termed "apical" in this article), distal to the second diagonal branch of the LAD. The true apex was avoided, however, because quality of length signals may be poor in that region (fig. 1). The other pair was situated in the basal region, supplied by the left circumflex coronary artery (LC). Of the available studies, we deliberately excluded those with crystal pairs in the intermediate zone (fig. 1). Both pairs of crystals were oriented parallel to the short axis of the LV. Continuous analog output of distances between paired crystals (segment length) was recorded on a Minograf® 81 eight-channel recorder (Elema Scholander, Stockholm, Solna, Sweden).

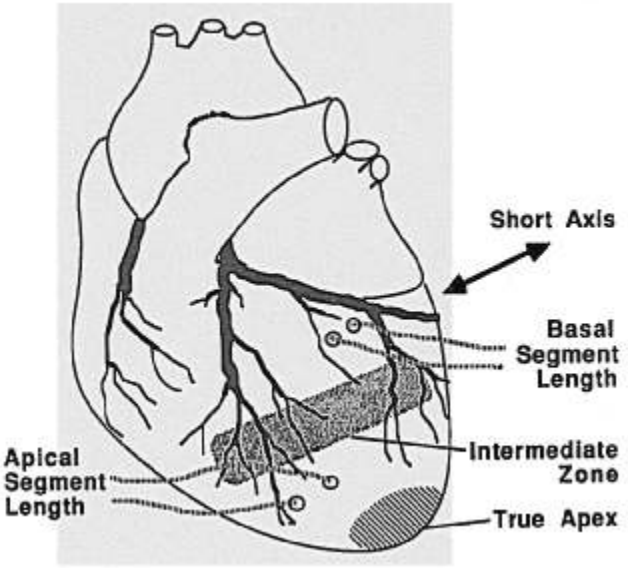

FIG. 1. Apical and basal placement of microcrystal pairs is illustrated. Experimental animals with crystals in the intermediate zone and true apical zone, indicated by the shaded areas, were excluded.

Global Hemodynamics

Aortic and LV pressures were recorded. Left ventricular rate of tension development (LVdP/dt) was obtained by on-line differentiation. Heart rate was calculated from the R-R interval on the ECG. Stroke volume was obtained by integration of the aortic flow signal and was calibrated by simultaneous determination of the cardiac output by dye dilution (indocyanine green).

Experimental Protocol

The effects of three different combinations of drugs on regional function in the apical (LAD) and basal (LC) region were investigated: 1) the effect of nitrous oxide (N_2O) added to an opiate; 2) the effect of stepped increases in halothane concentration; 3) the effect of an infusion of verapamil during isoflurane anesthesia.

Nitrous Oxide

After surgical preparation had been completed, halothane was discontinued. Fifteen minutes later, eight dogs received a loading dose of intravenous fentanyl, 100

§ Francis CM: The interaction between halothane anaesthesia and experimental myocardial ischaemia in the dog. D. Phil. Thesis, University of Oxford, 1982, pp 120–127.

TABLE 1. Hemodynamic Variables in the Fentanyl/Sufentanil Group ($\bar{x} \pm 1$ SD, fentanyl n = 8, sufentanil n = 8, NS = no statistically significant difference)

Condition	Heart Rate (per min)		Mean Arterial Pressure (mmHg)		$LVdP/dt_{max}$ (mmHg/s)		Stroke Volume (ml)		LVEDP (mmHg)		Percentage Systolic Shortening			
											Apex		Base	
67% nitrogen in oxygen (control)														
Fentanyl	111 (±30)	NS	111 (±15)	NS	3,030 (±430)	NS	29 (±7.9)	NS	5.3 (±1.3)	NS	20.2 (±4.2)	NS	16.1 (±3.7)	NS
Sufentanil	119 (±28)		112 (±17)		2,700 (±760)		29.4 (±11.0)		4.9 (±1.1)		23.9 (±7.5)		17.5 (±2.9)	
67% nitrous oxide in oxygen														
Fentanyl	102 (±20)	NS	110 (±11)	NS	2,630 (±430)*	NS	28.8 (±8.9)	NS	5.6 (±1.2)	NS	18.4 (±5.0)	NS	15.3 (±3.5)	NS
Sufentanil	123 (±26)		116 (±19)		2,580 (±810)		28.5 (±10.4)		5.8 (±1.8)		21.4 (±7.6)*		17.1 (±3.1)	
67% nitrogen in oxygen														
Fentanyl	106 (±27)	NS	116 (±15)	NS	3,150 (±650)	NS	29.0 (±8.0)	NS	4.8 (±1.0)	NS	19.9 (±4.0)	NS	16.0 (±2.2)	NS
Sufentanil	129 (±27)*		114 (±19)*		2,960 (±770)*		29.0 (±9.0)		4.3 (±0.7)		23.2 (±7.1)		17.8 (±3.5)	

* $P < 0.05$, compared with control.

$\mu g \cdot kg^{-1}$ over 5 min followed immediately by a continuous infusion of 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Eight dogs received a loading dose of sufentanil, 30 $\mu g \cdot kg^{-1}$ over 5 min followed immediately by a continuous infusion of 0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$. During the following 45 min, instruments were recalibrated and acid–base status corrected where necessary. After the 45-min period of stabilization, baseline measurements were obtained at end expiration. After this, N_2O was substituted for nitrogen (*i.e.*, 67% N_2O in O_2 instead of 67% N_2 in O_2) in all 16 dogs and the effects recorded after 15 min, at end expiration. N_2O was then again replaced by N_2 and recordings repeated after 15 min to determine that recovery had occurred.

Halothane

After surgical preparation had been completed in 15 dogs, a stabilization period of 1 h was allowed to elapse while instruments were recalibrated and acid–base status corrected if necessary. A minimum of 4 h elapsed between premedication and the beginning of the study. Control measurements were made at an inspired halothane concentration of 0.5%. The inspired halothane concentration was then increased in steps to 1.0%, 1.5%, and 2.0% and then decreased to 0.5% again. Each level of inspired halothane was maintained for 10 min before recordings were made. (Preliminary studies showed that circulatory stability was always achieved within 7 min after changing the halothane concentration.¶ All recordings were obtained during a 20-s period of apnea at end expiration.

Verapamil

After completion of surgical preparation in nine dogs, halothane was replaced by isoflurane, 1% inspired concentration, delivered by a Cyprane® vaporizer (Cyprane, Keighly, England), calibrated by mass spectrometry. Blood gases were analyzed and, where necessary, ventilation adjusted to ensure normocarbia and sodium bicarbonate given to correct metabolic acidosis. Control measurements were recorded at end expiration 1 h after introduction of isoflurane. Verapamil was then given intravenously at a loading dose of 250 $\mu g \cdot kg^{-1}$ over 20 min, followed by a maintenance dose of 60 $\mu g \cdot kg^{-1} \cdot h^{-1}$. The effects of verapamil were recorded at end expiration 30 min after the start of the maintenance dose.

COMPUTATIONS

Regional dimensions were measured with the use of the following criteria: end-diastolic length (EDL) was

¶ Francis CM: The interaction between halothane anaesthesia and experimental myocardial ischaemia. D. Phil. Thesis, University of Oxford, 1982, p 112.

TABLE 2. Hemodynamic Variables in the Halothane-treated and Isoflurane/Verapamil-treated Groups. $\bar{x} \pm 1$ SD; halothane n = 15, isoflurane n = 9)

Condition	Heart Rate (per min)	Mean Arterial Pressure (mmHg)	$LVdP/dt_{max}$ (mmHg/s)	LVEDP (mmHg)
0.5% halothane (control)	115 (±23)	81 (±12)	1,420 (±260)	4.0 (±2.1)
1.0% halothane	113 (±25)	71 (±12)*	1,110 (±220)*	4.5 (±2.1)
1.5% halothane	111 (±24)	59 (±12)*	840 (±180)*	5.2 (±2.6)
2.0% halothane	104 (±22)†	46 (±12)*	590 (±180)*	5.3 (±2.0)
0.5% halothane	115 (±31)	79 (±15)	1,250 (±150)†	4.4 (±2.4)
1% isoflurane (control)	113 (±5)	93 (±3)	1,600 (±120)	4.9 (±0.5)
1% isoflurane + verapamil 60 $\mu g \cdot kg^{-1} \cdot h^{-1}$	120 (±5)*	90 (±6)	1,280 (±120)*	5.9 (±0.3)†

* $P < 0.01$, compared with control.

† $P < 0.05$, compared with control.

measured at the time of the beginning of the sharp upslope of the first derivative of LV pressure (LVdP/dt) signal; end-systolic length (ESL) was measured at the time the aortic flow first returned to zero. Systolic shortening (SS) was expressed as percentage of EDL, with the use of the following formula:

$$\%SS = (EDL - ESL)/EDL \times 100 \quad \text{(Theroux } et\ al.^{13}\text{)}$$

The minimum length during systole ($L_{min}S$) was also measured and substituted for ESL when it was shorter than the latter.

Absolute difference between %SS in control (opiate alone; 0.5% halothane; 1.0% isoflurane) and altered conditions (opiate + N_2O; 1.0%, 1.5%, 2.0% halothane; 1.0% isoflurane + verapamil) were calculated and compared in LAD and LC regions. SS at altered conditions was also expressed as percentage of control (normalized %SS), and the change in this index was also compared between apical and basal regions.

Two-way analysis of variance (ANOVA) with a Duncan and Bonferroni option from the SAS® statistical computer package (VMS SAS Production Release 5.16, 1986 SAS Institute, Cary, North Carolina) was used as appropriate. Wilcoxon ranked paired tests were used where data did not follow a normal distribution. Apical and basal shortening were compared with paired t tests at each stage of the studies. The difference between apical and basal regions was also compared over all levels of halothane concentration with a two-way ANOVA, and multiple comparisons between the levels of concentration were obtained with the use of the Tukey test.

Results

Global Hemodynamics

Table 1 summarizes global and regional hemodynamic data in the fentanyl and sufentanil subsets. No statistically significant difference between the subsets could be demonstrated for any variable at any stage. With the withdrawal of N_2O, all hemodynamic values returned to control in the fentanyl subset, whereas heart rate and $LVdP/dt_{max}$ were higher in the sufentanil subset.

Table 2 summarizes systemic hemodynamic data in the halothane- and isoflurane/verapamil-treated groups. Note that left ventricular end-diastolic pressure (LVEDP) remained constant throughout all the stages in the N_2O-treated and halothane-treated groups (tables 1 and 2), whereas a statistically significant but small (1 mmHg) increase occurred with isoflurane plus verapamil (table 2). Mean arterial pressure decreased significantly at higher concentrations of halothane but not when verapamil was added to isoflurane. In both the halothane-treated and isoflurane/verapamil-treated groups, $LVdP/dt_{max}$ decreased significantly compared with control (0.5% halothane and 1.0% isoflurane). In the halothane-treated group, $LVdP/dt_{max}$ did not return to control value at the end of the dose–response study.

Regional Wall Motion

The effect of N_2O on regional function is shown in table 3. Because there were no differences for both global and regional function between the fentanyl- and sufentanil-treated groups (see table 1), the results are presented as pooled data. Under control conditions (33% O_2 in N_2), there was a highly significant difference in percentage systolic shortening (%SS) when apex was compared with base ($P < 0.01$). When 67% N_2O replaced N_2, this difference persisted, the apical region being still more active than the basal region. N_2O caused significantly greater depression of percentage systolic shortening and of normalized percentage systolic shortening in the apex than in the base of the ventricle ($F[1, 5] = 7.3280$, $P < 0.025$).

Figure 2 shows the effect of halothane on %SS. At 0.5% and 1.0% inhaled halothane, statistically significant differences in shortening between LAD and LC regions were noted. Although the LAD region appeared more dynamic at more depressed levels of inotropy (1.5% and 2% halothane), differences in apical and basal shortening no longer

TABLE 3. The Effect of Nitrous Oxide on Shortening in Different Regions of the Left Ventricle*

Region	Percentage Systolic Shortening			Effect of Nitrous Oxide	
	Opiates (control)	Opiates + 67% Nitrous Oxide	Opiates (after nitrous oxide)	Absolute Reduction in %SS	Reduction in %SS, Normalized to Control
Apex	21.9 (±6.2) *	19.9 (±6.4) *	21.6 (±6.1) *	−1.97 (±1.8) †	−10.4 (±2.2)% †
Base	16.8 (±3.3)	16.2 (±3.4)	16.9 (±3.0)	−0.82 (±1.25)	−3.9 (±1.6)%

* Absolute reduction in %SS = absolute change in percentage systolic shortening when nitrous oxide is added. Reduction in %SS, normalized to control = difference in %SS between control and nitrous oxide stages, as percentage of control stage. Pooled data: fentanyl n = 8, sufentanil n = 8; $\bar{x} \pm 1$ SD; NS = not statistically significant.
* $P < 0.01$.
† $P < 0.05$.

reached statistical significance. Absolute differences in %SS between control (0.5% halothane) and the other stages (1.0%, 1.5% and 2.0%) of halothane are shown in figure 3. The decreases in shortening brought about by increasing halothane concentration were significantly greater in the apical than in the basal regions at every increased concentration. Decreases in normalized %SS are shown in figure 4. Each increase above 1% halothane caused a significantly greater decrease in normalized shortening in the apical than basal region ($F[3, 39] = 12.5841$, $P < 0.005$, Tukey critical difference $= 2.0322$, $P < 0.05$).

Table 4 shows the effect of verapamil on regional myocardial shortening. The significant difference between apical and basal shortening under 1.0% isoflurane was abolished by the addition of verapamil. The effect of verapamil on %SS was significantly greater in the apical than in the basal region ($F[1, 8] = 4.7977$, $P < 0.005$).

Discussion

The experimental model used in these studies is well established for assessment of regional wall shortening.[9–11,13–15] Because differences in regional function may become too small to detect when crystal pairs are placed too near each other,[7] only experiments in which apical and basal crystal pairs were placed well apart were included for analysis. The true apical area was also avoided because quality of subendocardial micrometry signals here may be poor because of geometric displacement. Differences in apical and basal function cannot be ascribed to the open-chest dog model because similar differences were demonstrated in awake, closed-chest dogs[16] and have been observed in humans.[5,8] The morphine and thiopental used for premedication and induction of anesthesia could have influenced regional function, although this effect should be negligible[17] because measurements were started 3–4 h after these drugs had been given.

Previous studies have examined regional function when different regions were subjected to different circumstances. Most frequently myocardial ischemia was introduced in either LAD or LC segments and the function in the compromised segment studied and compared with that of the normal segment.[11,18–20] Alternatively, the LAD and the LC were differentially perfused with positive or

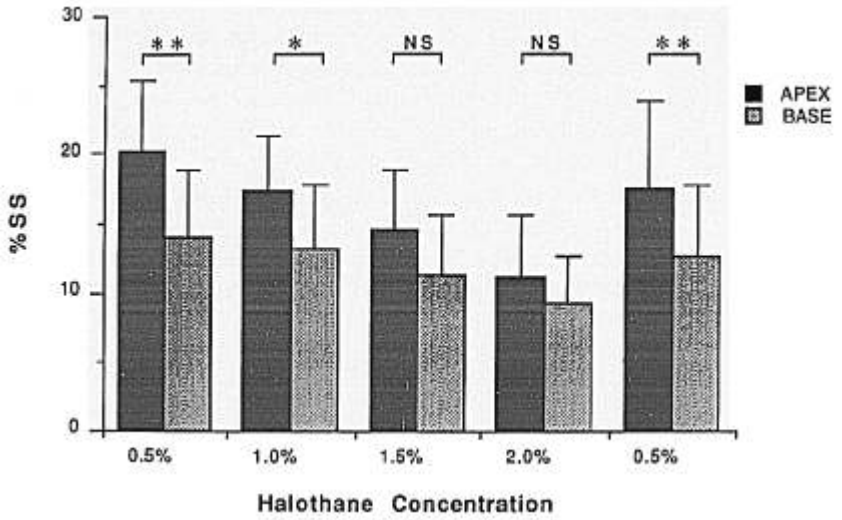

FIG. 2. Percentage systolic shortening (%SS) at each inspiratory halothane concentration for apical and basal regions. Values are mean, bar = 1 SD; N = 15; NS = not statistically significant; *$P < 0.05$; **$P < 0.01$. As halothane-induced depression increases (1.5 and 2.0% halothane), the significant differences in regional performance are suppressed. On returning to control halothane concentration, regional differences reappear.

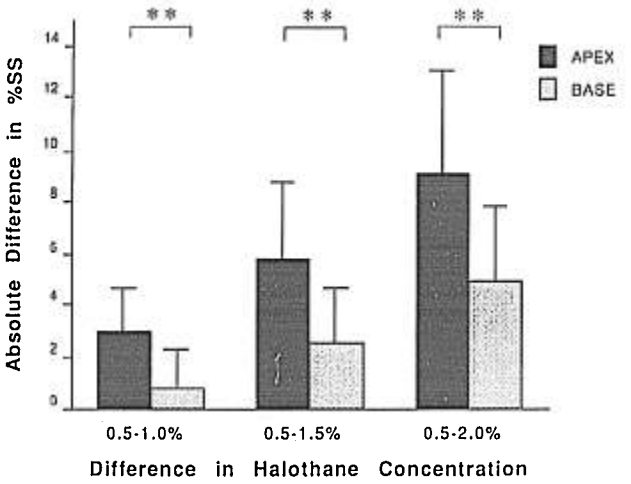

FIG. 3. Absolute reductions in percentage systolic shortening (%SS) as a function of increases in halothane concentration. Values are mean, bar = 1 SD; N = 15; $^{**}P < 0.01$. Reductions in %SS are significantly greater in the apex throughout the dose–response study.

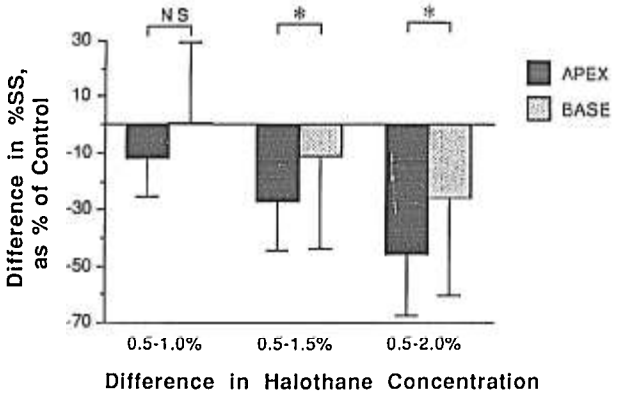

FIG. 4. Difference in percentage systolic shortening (%SS), as percentage of control plotted as a function of increases in halothane concentration. Values are mean, bar = 1 SD; N = 15; $^{*}P < 0.05$; NS = not significant. Because in both segments values were normalized to a control shortening of 100%, differences in effects of halothane on different segments are independent of magnitude of initial shortening.

negative inotropic agents and regional function compared in different segments.[21,22] The findings in these experiments could satisfactorily be explained by an interconnected two-compartment model in which ischemic cardiac muscle in series with normal muscle is stretched during contraction.[23–25] In the present study, there was no interference with the coronary circulation and thus apical and basal regions were subjected to exactly the same interventions. Any difference in performance could thus be ascribed to the inherent characteristics of these regions of the ventricle.

The apical region was always more active than the basal region, exhibiting greater systolic shortening. When depression was increased, the more active apical region was depressed more, both in absolute and relative terms, than the less active basal region (tables 3 and 4, and figs. 3 and 4). Although a linear correlation (r = 0.97) with a slope of 1.45 can be fit to the relationship between average %SS in apical and basal regions for all interventions pooled together, the best fit (r = 0.98) was obtained with a logarithmic function (fig. 5). The fact that all the points lie above the identity line shows that the apex is more active than the base. The good fit of the logarithmic line illustrates that the apex is depressed more than the base with negative inotropic interventions. This tends to abolish differences between apex and base when function is severely depressed.

The differences in regional performance could be explained by a difference in sarcomere length. Laks *et al.* demonstrated longer sarcomeres in the apex than in the base of the LV.[26] In longer sarcomeres, more myofibrillar length is available for contraction before excess overlap of actin and myosin prevents further shortening. Differences between apical and basal shortening may thus be a manifestation of the Frank-Starling law.[27]

However, the greater effect of negative inotropic interventions on the apex is not so readily explained. The effects of volatile anesthetics and calcium channel blocking drugs on the myocardial cell are complex and not fully understood.[28–33] Therefore, a discussion of the mechanism of the differential effects of these drugs on regional

TABLE 4. The Effect of Verapamil on Shortening in Different Regions of the Left Ventricle*

Region	Percentage Systolic Shortening		Effect of Verapamil	
	1% Isofluorane (control)	1% Isoflurane + Verapamil (60 $\mu g \cdot kg^{-1} \cdot h^{-1}$)	Absolute Reduction in %SS	Reduction in %SS, Normalized to Control
Apex	18.3 (±5.8)	10.1 (±6.3)	−8.49 (±2.9)	−48.6 (±20.7)%
Base	13.3 (±2.5)	9.0 (±3.2)	−4.32 (±3.0)	−32.3 (±25.7)%
	*	NS	†	NS

* Absolute reduction in %SS = absolute change in percentage systolic shortening when verapamil is added to isoflurane. Reduction in %SS, normalized to control = difference in %SS between isoflurane and isoflurane plus verapamil, as percentage of control. N = 9; $\bar{x} \pm 1$ SD; NS = not statistically significant.
* $P < 0.05$.
† $P < 0.01$.

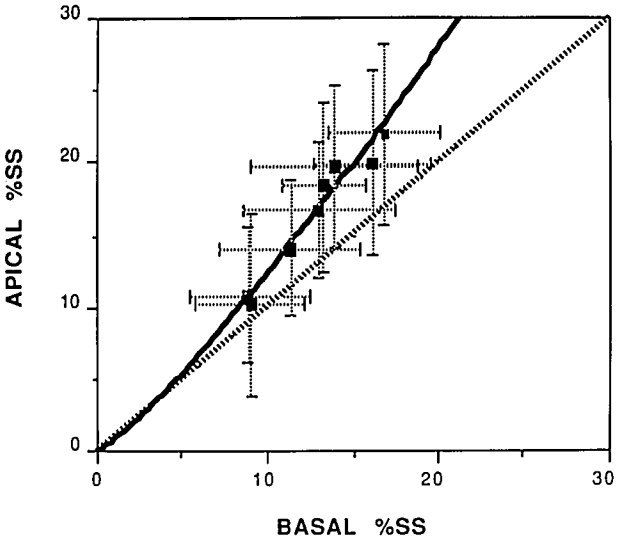

FIG. 5. Relationship between average values for percentage systolic shortening (%SS) in the apical region and the corresponding average %SS values in the basal region. Values are mean, bar = 1 SD, data for all the experiments pooled. A logarithmic curve fit (r = 0.98) fits the distribution of mean values better than a linear fit (r = 0.97). This is to be expected, because negative inotropic interventions suppress the apex more than the base. Note, however, that the line differs from identity line (dotted line), illustrating that apical shortening differs from basal shortening.

function can only be speculative. However, the longer sarcomere lengths in the apex may afford a larger number of binding sites for halothane or verapamil. This may influence the size of or sensitivity to calcium fluxes,[34,35] thus making the region more susceptible to the cardiodepressive effects of these drugs, which are known to modify calcium ion fluxes.[30-33]

If calcium channels are not involved, changes in regional myocardial blood flow may be responsible for the greater effect of halothane, verapamil, and N_2O on apical as opposed to basal function. It has been shown that anesthetics can alter transmural blood flow.[36,37] It has also been shown that myocardial function is very sensitive to blood flow, particularly subendocardial blood flow.[38-40] Although the specificity of regional wall motion abnormalities as an indicator of blood flow is still controversial,** the question arises whether anesthetics may modify regional or transmural blood flow enough to explain the greater depression of the apex. In this respect, hypotension may play a role. With normal blood pressure, apex and base should get the same relative blood supply and oxygen delivery. Severe hypotension may disturb the normal distribution of blood flow to such an extent that one region may exhibit a greater depression of function than the other. Hemodynamic data in this study do not support this hypothesis. Although deeper levels of halothane or administration of verapamil abolished the differences in regional shortening, a significant reduction in blood pressure occurred only with halothane and not with verapamil.

Differences in wall tension may play an important role. According to the law of La Place, wall tension should be less in the apex because of the smaller radius. However, several groups[41-43] have shown that the apical myocardium is thinner: this could partly offset the effect of the smaller radius. Calculations of wall tension have shown wall tension to be higher in the basal region than in the apex.[43,44] Thus, more tension would develop in the basal region but the basal region would shorten less during each contraction, whereas less tension would develop in the apical region and the apical region would shorten more. Moreover, the greater amount of fibrous tissue in the basal region, necessary to anchor the valvular apparatus, may contribute to the reduced shortening of the basal region.[7] Differences in wall tension may explain both initial differences in function and the differential effect of negative inotropic interventions.

Because of differences in regional contraction and differential effects of drugs on segmental function of the normal LV, results from different studies can only be compared if they examined similar segments. The findings of Le Winter *et al.* suggested that regional differences exist even between segments that are not far apart.[7] Some of the confusion and inconsistency of findings regarding regional function could be explained by the fact that the function of different areas of the LV have been examined.[10,11,36,45,46] In studies of myocardial ischemia, the effect of reducing coronary blood flow could be expected, on the strength of regional differences, to be greater in the more dynamic apical region than in the less dynamic basal region. It is clear that the apex cannot be used as a "control" for the base.

The clinical implications of the differences in regional shortening may be that the LV apex is more involved with ejection and the base with firmness of the ventricular outflow tract during ejection, as in the right ventricle.[47] During anesthesia, hemodynamic depression may result largely from exaggerated depression of the more active regions of the LV.

A limitation of this study is that no positive inotropic intervention was examined. However, a recent study by Hittinger *et al.*[16] has demonstrated that, under different loading conditions, as well as increased inotropy, the apex is always more active than the base, and the slope of the relationship of apical to basal myocardial shortening remains relatively constant during these interventions.

** Thys DM: The intraoperative assessment of regional myocardial performance: Is the cart before the horse? (editorial) Journal of Cardiothoracic Anesthesia 1:273-275, 1987.

In summary, there are regional differences in function within the LV, the apical region being more active than the basal region. Negative inotropic interventions, such as increasing anesthetic concentration, adding N_2O, or administering verapamil, cause relatively greater depression of apical contraction and tend to decrease these differences. The nonhomogenous behavior of the LV should be borne in mind when regional LV function is evaluated.

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