Regional myocardial shortening in relation to graft-reactive hyperemia and flow after coronary bypass surgery

Extent of regional shortening of myocardium in areas newly perfused by bypass grafting was determined in 56 patients by a new technique employing four to six radiopaque markers sutured in pairs to the epicardium near the coronary anastomosis. Paradoxical systolic expansion (PSE) was manifest in 16 regions (a 12 percent incidence) during the follow-up period, and six of these showed spontaneous remission. All cases of PSE were in the region of the left anterior descending artery. Correlation between graft flow measured during operation and regional shortening during the postoperative period revealed that the development of PSE could not be predicted from the hemodynamic measurements. In the majority of cases postoperative myocardial infarction could also be excluded as an explanation. At 1 year after operation most grafts were patent in PSE regions but collaterals, apparent preoperatively, could not be visualized. Excluding PSE, shortening fraction (ratio of shortening to maximum marker separation) for all graft regions at 1 week was 9.8 percent; 1 month, 12.8 percent; 3 months, 13.3 percent; and six months, 13.9 percent. Average graft flow was 56 ml. per minute and average reactive hyperemia was 25 percent with 37 percent of grafts showing no response. For those regions that did not develop PSE there was a positive correlation between shortening fraction and flow that became significant (null hypothesis: $r = 0$) when reactive hyperemia exceeded 20 percent. Correlation was greatest at 1 week and 1 month, but became nonsignificant at 6 months. These results are consistent with a simple interpretation of reactive hyperemia: Graft-reactive hyperemia is related to the dependence of viable tissue on the functioning of the graft.

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Flow via coronary artery bypass graft has been shown to be a relevant factor in bypass occlusion in the early postoperative period,[1–4] but the significance of reactive hyperemia (as measured by a temporary occlusion of the graft) has remained unclear.[5–7] In dogs, bypass-reactive hyperemia is not as great in the presence of collateral circulation and/or submaximal parent artery occlusion.[8] In man it has been suggested that the magnitude of the bypass-reactive hyperemia is related to the dependence of the viable tissue on the functioning of the graft.[9] That is, if reactive hyperemia is absent following graft occlusion-release, there is either little viable tissue being perfused or there are other sources of perfusion, such as the parent artery or collaterals, while a strong reactive hyperemia response suggests that the graft is contributing significantly to the total perfusion.

Wiggers,[10] and then Tenant and Wiggers,[11] originally demonstrated that regional shortening correlates with coronary perfusion and this has been verified in man both during operation and in the postoperative period.[12, 13] To the extent that these observations confirm that regional shortening correlates with total perfusion, an indirect test of the reactive hyperemia hypothesis can be devised. In those regions perfused variably by the parent (diseased) artery, collaterals, and bypass graft, the correlation between regional shortening and graft flow alone would be nearly random when reactive hyperemia is absent, whereas with an increas-

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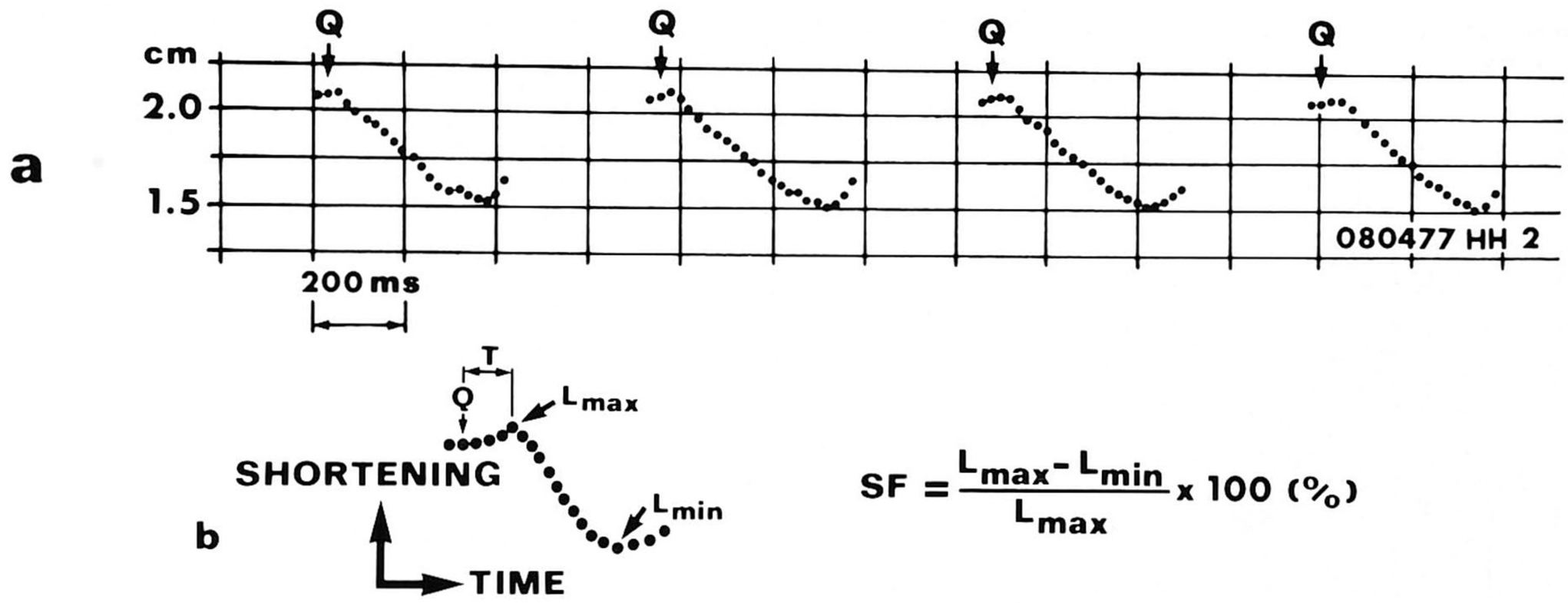

Fig. 1. *a*, The marker pair shortening for a normal contraction is shown. The simultaneously recorded ECG is indicated *(arrows)* at the Q wave of the ECG. The markers are 2 cm. apart and 0 to 3 cm. distal to the coronary anastomosis. *b*, By use of the recording in *a*, shortening fraction *(SF)* and time to onset of shortening *(T)* are determined beat by beat and averaged over four beats.

ing reactive hyperemia response, the correlation should improve. This does not imply that the correlation coefficient should approach unity, only that it should be significantly greater than zero. The explanation for the nearly random relationship for absent reactive hyperemia is that the total perfusion is unknown and only a part of this, the bypass flow, is measured. For a strong reactive hyperemia response, the bypass flow would presumably more closely approximate the total perfusion.

However, testing this hypothesis requires accurately measuring myocardial shortening in the region perfused by the graft early in the postoperative period. We have developed a new technique employing radiopaque markers[14] for this purpose. This has proved to be sufficiently accurate and specific to demonstrate the basic validity of the hypothesis.

Patients and methods

Patients. The group consisted of 52 men and four women, ranging in age from 41 to 61 years, who were consecutively scheduled for elective coronary artery bypass grafting. Of these, four patients underwent aneurysmectomy.

Measurement of regional shortening. Pairs of radiopaque markers were sutured to the epicardium 0 to 3 cm. distal to the coronary anastomosis, 2 cm. apart, and in transverse alignment with the major axis of the heart. As a rule, markers were not placed in aneurysmatic or infarcted tissue as observed at operation. A total of 120 pairs were placed in bypass regions: 51 pairs near the proximal and mid–left anterior descending artery including the first and second diagonal branches; 35 pairs near the posterior portion of the right coronary artery; and 34 pairs near the circumflex artery including the marginal and posterolateral branches.

Marker motion was recorded with a conventional monoplane cineangiographic apparatus at 50 frames per second. The C-arm was positioned perpendicular to the shortening axis of the marker pair being recorded (minimal foreshortening), and this was repeated for each marker pair. For follow-up recordings, the previously recorded C-arm position for each marker pair was used. Patients were examined at 1 week, 1 month, 3 months, and 6 months, and coronary angiography was performed at 1 year. Five heartbeats were filmed at end expiration and the electrocardiogram (ECG) and frame markers were recorded simultaneously on paper. The films were projected and marker pair separation was measured with a conventional vernier caliper. Marker pair separation for at least four heartbeats was measured starting from the frame prior to onset of the Q wave of the ECG to the minimum separation. Shortening fraction *(SF)* and time to onset of shortening (T = time from onset of the Q wave of the ECG to maximum marker separation) were determined as illustrated in Fig. 1. The greatest source of measurement variability in SF is the physiological beat-to-beat variability (±0.9 percent for an SF of 10 percent) followed by intra- and interobserver variability (±0.6 percent and ±0.8 percent, respectively). This presents no serious limitation to the method. Accuracy of time to onset of shortening is limited by the interval between frames (20 msec.). A change of 40 msec. between follow-ups can be detected with 95 percent confidence.

Paradoxical systolic expansion (PSE) was identified

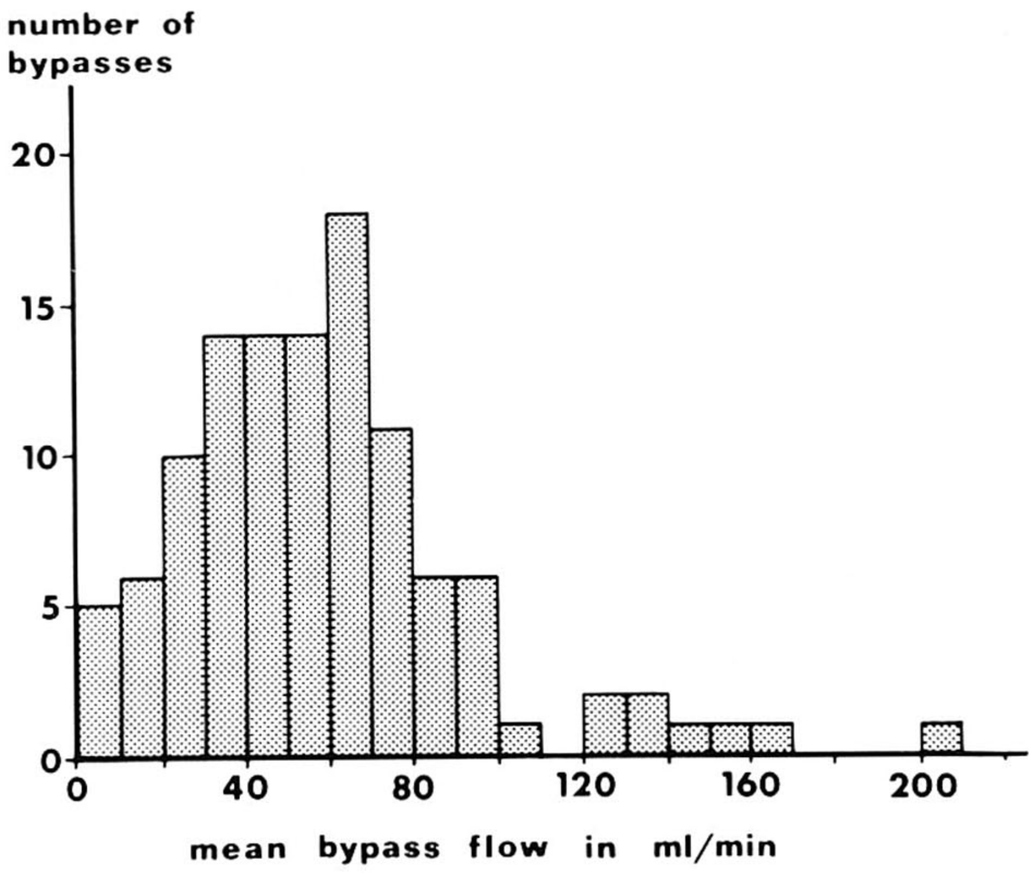

Fig. 2. The bypass flow histogram in increments of 10 ml. per minute shows a nearly gaussian distribution of flow in the bypass grafts. The mean flow is 56 ml. per minute, with a standard deviation of 34 ml. per minute.

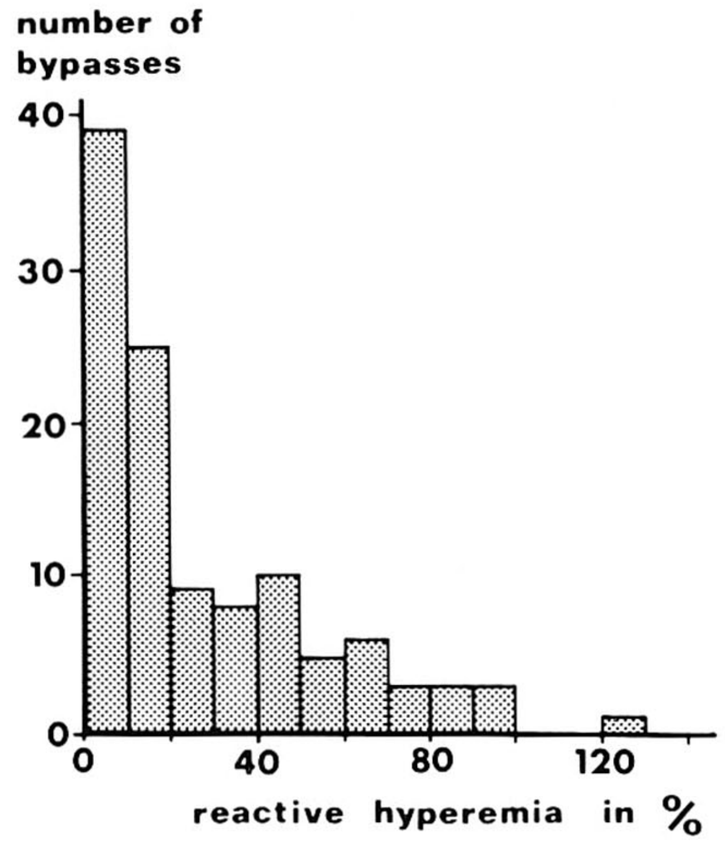

Fig. 3. The reactive-hyperemia histogram shows a nongaussian distribution with the majority of grafts showing a reactive hyperemia of less than 20 percent.

by inspection of the graph of marker separation versus time in relation to the QRS complex or with the left ventricular pressure recording when available. PSE usually appeared as an early systolic expansion with a delayed (>200 msec.) onset of apparent shortening at the moment expected for isovolumetric relaxation. The significance of SF in PSE is different than that in normally contracting regions; in PSE, SF is measured from the end of systole to early diastole which can be interpreted as the fractional reduction in length as a result of removal of the peak systolic pressure loading. Thus, in the extreme, it is a passive phenomenon and cannot be thought of in the same way as active shortening.

Bypass flow and reactive hyperemia. Graft flow was measured prior to thorax closure, about 30 minutes after ending extracorporeal circulation, using an electromagnetic flowmeter* with a perivascular probe (2, 2.5, 3, 3.5, 4, or 5 mm.) providing a snug fit. Each probe was calibrated in vitro as described previously.[3, 4] Reactive hyperemia (RH) tests were performed by occluding the graft distal to the flow probe for 10 seconds. The peak diastolic flow (PkF) just after release (max PkF) was compared to steady state values measured 45 to 60 seconds later:

$$RH = \frac{100\% \text{ (max PkF} - \text{PkF)}}{\text{PkF}}.$$

Data analysis and subgroups. A correlation analysis between SF and mean graft flow was performed for subgroups of regions, based on the RH response: 1, those that showed no response, i.e., RH = 0; 2, RH > 0; 3, RH > 10 percent; 4, RH > 20 percent; 5, RH > 30 percent; and 6, RH > 40 percent. The correlation coefficient, r, and the probability that r was different from zero were computed for each follow-up examination: 1 week, 1 month, 3 months, and 6 months. For those regions that eventually developed paradoxical systolic expansion, all measurements of SF were used up to, but no including, the moment that PSE was first detected. Those regions developing PSE at any time during the first 6 months' follow-up were treated as an ancillary group. Of those shortening patterns identified as not showing PSE, which included those with a delayed onset of shortening, a subgroup was defined including only those regions with a normal onset of shortening, T < 100 msec.

Results

Graft hemodynamics. The bypass flow (Fig. 2) ranged from 3 to 200 ml. per minute, the mean flow was 56 ml. per minute (S.D. = 34, n = 112). Of the 139 bypass grafts implanted in the 56 patients, there were 112 regions that met the inclusion criteria: bypass flow and reactive hyperemia measured and marker motion adequately recorded in the follow-up period. The mean bypass flow to the region of left anterior descending artery was 57 ml. per minute (n = 48); the circumflex artery, 49 ml. per minute (n = 34); and the right coronary artery, 62 ml. per minute (n = 30).

*Transflow 600, Skalar bv, Delft, The Netherlands.

Table I. *Occurrence of paradoxical systolic expansion in bypass graft regions from 1 week to 6 months after surgery and presence of paradoxical systolic expansion at time of recatheterization, 1 year after operation*

Patient No.	*PSE*				*Myocardial infarction*		
	Onset by	*Remission by*	*At 1 yr.*	*Region*	*Preoperative*	*Perioperative*	*Postoperative*
19	1 wk.	–	Yes	LAD	No	Yes	No
22	1 wk.	–	Yes	LAD	Yes	No	No
25	1 wk.	–	Yes	LAD	Yes	No	No
40	1 wk.	–	Yes	LAD	Yes	No	No
4	1 mo.	6 mo.	No	LAD	Yes	No	No
17	1 mo.	–	Yes	LAD	Yes	No	No
20	1 mo.	6 mo.	No	LAD	Yes	No	No
35	1 mo.	3 mo.	No	LAD	Yes	No	No
41	1 mo.	–	Yes	LAD	Yes	No	No
44	1 mo.	–	†	LAD	Yes	No	No
46	1 mo.	6 mo.	No	LAD	No	No	No
52	3 mo.	–	Yes	LAD	Yes	No	No
11	6 mo.	1 yr.	No	LAD	Yes	No	No
21	6 mo.	1 yr.	No	LAD	Yes	No	No
29	6 mo.	–	Yes	LAD	No	No	No
49	6 mo.	–	Yes	LAD	No	Yes	No

Legend: PSE, Paradoxical systolic expansion. LAD, Left anterior descending coronary artery.
†Died at 3 months.

There was no significant difference between flows in these regions.

The RH ranged from 0 to 120 percent. The distribution is nongaussian with 33 percent of the measurements showing an RH of less than 20 percent (Fig. 3). Of those RH measurements greater than zero, the mean is 37 percent (n = 76). For an RH of less than 20 percent, the mean flow is 52 ml. per minute (S.D. = 34, n = 64), and for an RH greater than or equal to 20 percent, the mean flow is 61 ml. per minute (S.D. = 32, n = 48); the apparently greater flow for reactive hyperemia greater than 20 percent is not significant at the 0.05 level. Linear regression analysis between flow and RH revealed no significant correlation for the entire group or for any subgroup defined by bypass region or RH range.

Marker motion. The marker motion was recorded at 1 week, 1 month, 3 months, and 6 months postoperatively. The mean heart rates for the entire group were 95 beats per minute at 1 week, 92 beats per minute at 1 month, 83 beats per minute at 3 months, and 77 beats per minute at 6 months; the pooled standard deviation was 15 beats per minute. The mean shortening fraction (SF), excluding those marker motion patterns identified as paradoxical systolic expansion (PSE), was 9.8 percent at 1 week, 12.8 percent at 1 month, 13.3 percent at 3 months, and 13.9 percent at 6 months; the pooled standard deviation was 4.7 percent. The mean SF in apparently normally perfused control regions on the anterior surface at the base of the heart was 11.5 percent, 12.0 percent, 13.1 percent, and 13.7 percent for the respective follow-up periods.

Paradoxical systolic expansion. The inventory (Table I) of every occurrence of PSE in bypass-perfused regions shows the first occurrence of PSE during the first 6 months after operation, whether PSE was still present at 1 year, the bypass region involved, and the history of myocardial infarction. There are 16 entries out of a total group of 139 bypass regions with implanted marker pairs, or an incidence of 12 percent. Of these 16 entries, nine still showed PSE at 1 year, and six showed a normal or delayed onset of shortening, that is, spontaneous remission of PSE. One patient died at 3 months after operation. In every case the region of the left anterior descending bypass was involved, resulting in a significantly nonuniform ($p < 0.0001$) regional distribution of PSE. Twelve were found to have had a myocardial infarction preoperatively and only two patients were completely free of myocardial infarction both preoperatively and during the 1 year of follow-up. No marker pairs were placed in infarcted tissue as visually identified during operation, but it is possible that markers were placed over areas of subendocardial infarcted myocardium. However, the occurrence of preoperative or perioperative infarction cannot be the explanation for the 12 instances of PSE

Table II. *Regional wall motion scored visually (right anterior oblique projection regions 1 to 5 and left anterior oblique, 6 and 7) preoperatively and 1 year postoperatively in patients with marker pairs showing paradoxical systolic expansion at 1 year*

Patient No.	*Visual scoring or regional wall motion*							*Graft anastomosis (Open/Closed)*	*Obstruction of proximal artery (%)*	*Collaterals*
	1	*2*	*3*	*4*	*5*	*6*	*7*			
19: Preop.	Nor	Hypo	Hypo	Nor	Nor	Unk	Unk	—	50-90, 100	2 to 8
Postop.	Nor	Hypo	Hypo	Hypo	Hypo	Nor	Nor	3(O), 8(O)	91-99, 100	None
22: Preop.	Nor	Nor	Nor	Nor	Nor	Unk	Unk	—	50-90, 100, 91-99	4 to 8, 10
Postop.	Nor	Nor	Nor	Nor	Nor	Nor	Nor	3(C), 8(O), 12(C)	91-99, 100, 91-99	None
25: Preop.	Nor	Nor	Nor	Hypo	Nor	Unk	Unk	—	100, 50-90, 50-90	1 to 3; 4 to 8, 13
Postop.	Nor	Nor	Nor	Hypo	Nor	Nor	Nor	3(O), 8(O), 13 (O)	100, 50-90, 100	None
40: Preop.	Nor	Nor	Nor	Nor	Hypo	Nor	Nor	—	91-99, 50-90, —	None
Postop.	Nor	Nor	Nor	Nor	Hypo	Nor	Nor	3(O), 8(O), 12(O)	100, 50-90, 100	None
17: Preop.	Nor	Nor	Nor	Nor	Nor	Unk	Unk	—	50-90	None
Postop.	Nor	Nor	Nor	Nor	Nor	Nor	Nor	7(O)	100	None
41: Preop.	Hypo	Akin	Hypo	Nor	Hypo	Hypo	Nor	—	50-90, 100, —	? to 7, 8
Postop.	Hypo	Akin	Hypo	Nor	Hypo	Akin	Nor	3(O), 8(O), 12(O)	50-90, 100, 91-99	None
52: Preop.	Nor	Hypo	Nor	Nor	Nor	Unk	Unk	—	50-90, 100	1 to 7, 8
Postop.	Nor	Hypo	Nor	Nor	Nor	Hypo	Hypo	12(O), 7(O)	91-99, 100	None
29: Preop.	Nor	Hypo	Nor	Nor	Nor	Unk	Unk	—	51-90, 100, 91-99	? to 8
Postop.	Nor	Nor	Hypo	Nor	Nor	Nor	Nor	3(O), 7(O), 12(O)	91-99, 100, 100	4 to 13, 14
49: Preop.	Nor	Nor	Akin	Hypo	Nor	Unk	Unk	—	91-99, 100, 91-99, 50-90	4, 6, 9 to 8
Postop.	Nor	Nor	Nor	Nor	Nor	Nor	Nor	3(O), 8(O), 9(O), 12(C)	100, 100, 100, 50-90	None

Legend: Region: 1, Anterobasal. 2, Anterolateral. 3, Apical. 4, Diaphragmatic. 5, Posterobasal. 6, Septal wall. 7, Posterolateral. Nor, Normal. Hypo, Hypokinetic. Akin, Akinetic. Unk, Unknown. Coronary artery code. 1, Proximal right coronary artery. 2, Midright coronary. 3, Distal right. 4, Right posterior descending. 5, Main stem. 6, Proximal left anterior descending. 7, Midleft anterior descending. 8, Proximal left anterior descending. 9, First diagonal. 10, Second diagonal. 11, Proximal left circumflex. 12, Obtuse marginal. 13, Midcircumflex. 14, Posterolateral. 15, Posterior descending.

not present at 1 week, but occurring later.

The preoperative and 1 year postoperative results of regional shortening (by visual scoring of the cineventriculograms) as well as the perfusion status are summarized in Table II for those regions still showing PSE at 1 year. In no instance in the postoperative period was regional wall motion scored as dyskinetic from the cineventriculograms in spite of simultaneously recorded PSE from the epicardial markers. Of 49 preoperative-postoperative measurements of regional shortening the majority showed no change over 1 year, but four regions did deteriorate while three improved. This group did not show any particular pattern in graft closure which could explain the PSE; there were only two patients with one or more occluded bypasses. However, with one exception, collaterals identified preoperatively could not be seen during the postoperative investigation. In the only exception, Patient 29, all bypasses were open, but there was significant progression of disease in all proximal arteries. Of those bypass regions identified as showing PSE for two or more follow-up examinations, the mean graft flow measured at operation was 67 ml. per minute (S.D. = 44), 27 percent had zero RH, and the average RH for the remainder was 38 percent. In summary, PSE of epicardial marker pairs has been found to occur at random times during the follow-up period, and in 38 percent of cases spontaneous remission occurred. For those patients with PSE still present at 1 year after operation, the cineventriculograms usually showed an abnormal contraction pattern, but frank dyskinesia was not found. Neither the occurrence of new infarction nor bypass closure could explain these results. However, in the majority of cases PSE was coincident with either the disappearance of or the inability to visualize the collateral circulation.

Shortening and graft hemodynamics. The relationship between postoperative shortening fraction and perioperative graft flow is summarized in Table III, which excludes regions that showed PSE as described in the previous section. Table 3 summarizes the results for increasing values of RH. The first column includes only those regions showing no RH response (RH = 0), and the second column includes all regions with RH greater than zero. The third and subsequent columns include all regions with an RH greater than the value

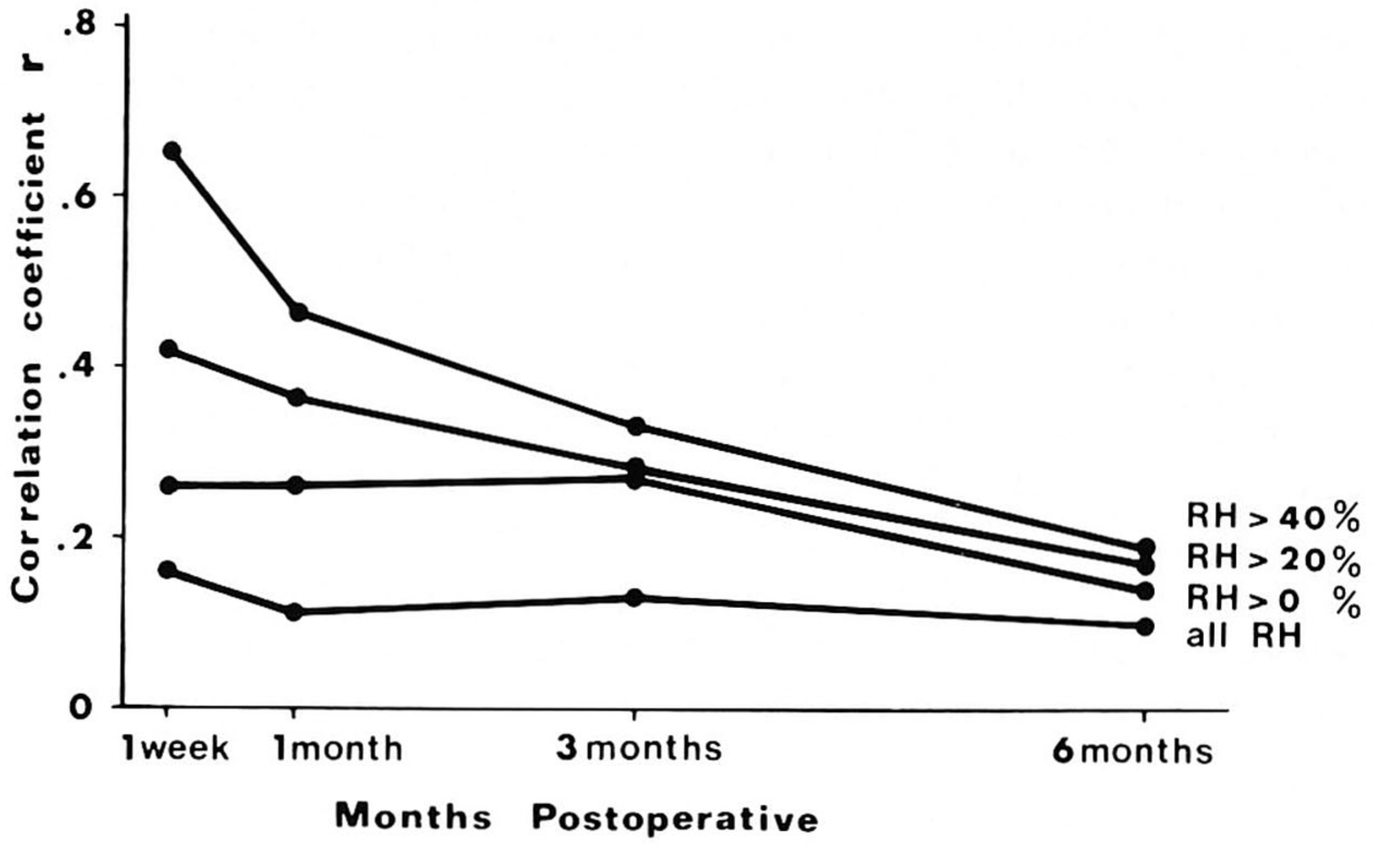

Fig. 4. The correlation coefficient resulting from linear regression of shortening fraction against graft flow is plotted over the entire follow-up period. The lowest line includes all measurements of reactive hyperemia, while the other lines include only those measurements of reactive hyperemia greater than the indicated value.

Table III. *Correlation coefficient (r) for linear regression of shortening fraction against graft flow, and the probability (p) that such a correlation could occur by chance if r = 0**

	RH					
	0	*>0%*	*>10%*	*>20%*	*>30%*	*>40%*
1 week:						
$0 < T < 200$ r	−0.07	0.17	0.18	0.33	0.30	0.40
p	N.S.	N.S.	0.10	0.05	0.10	0.05
$0 < T < 100$ r	−0.05	0.26	0.27	0.42	0.45	0.65
p	N.S.	0.10	0.10	0.05	0.05	0.01
1 mo.:						
$0 < T < 200$ r	−0.14	0.22	0.26	0.36	0.31	0.44
p	N.S.	N.S.	0.10	0.05	0.10	0.05
$0 < T < 100$ r	−0.14	0.26	0.31	0.36	0.32	0.46
p	N.S.	0.10	0.05	0.05	0.10	0.05
3 mo.:						
$0 < T < 200$ r	−0.04	0.28	0.32	0.26	0.17	0.28
p	N.S.	0.05	0.05	N.S.	N.S.	N.S.
$0 < T < 100$ r	−0.10	0.28	0.32	0.28	0.18	0.33
p	N.S.	0.10	0.05	0.10	N.S.	N.S.
6 mo.:						
$0 < T < 200$ r	0.16	0.18	0.17	0.18	0.12	0.20
p	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
$0 < T < 100$ r	0.19	0.14	0.17	0.17	0.11	0.19
p	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.

*The measurements are separated into columns by the magnitude of graft-reactive hyperemia: 0, only those grafts with zero reactive hyperemia; >0%, all those grafts associated with graft-reactive hyperemia greater than 0%; and so on. First, the correlation coefficient for all measurements is given with only paradoxical systolic expansion excluded, followed by those regions showing a normal onset of shortening ($T < 100$ msec).

indicated. For each follow-up, first the correlation coefficient (r) and the probability that r is different from zero are shown for all measurements in the respective hyperemia class, and the results for the subgroup having a normal onset of shortening ($T < 100$ msec.) are shown immediately below. A delayed onset of shortening ($T \geq 100$ msec.) is associated with later development of PSE.[14] The correlation coefficient between shortening fraction (SF) and bypass flow for those regions with a normal onset of shortening is illustrated in Fig. 4 for the entire follow-up period. At 1 week the correlation coefficient between SF and mean flow be-

comes significant (at the 0.05 level) for RH > 20 percent. As the RH level is increased to 40 percent, the significance level becomes 0.01. A similar pattern is manifest at 1 month. However, by 6 months the correlation coefficient is not significantly different from zero at any level of RH. The pattern is similar even when the measurements with a delayed onset of shortening are included. However, the correlation coefficients are reduced and the measurements with delayed onset of shortening have a randomizing influence. In summary, the correlation between shortening fraction and mean flow in the early postoperative period becomes more certain with increasingly stringent criteria for a positive RH test.

Discussion

The mean bypass flow of 56 ml. per minute is well within the range previously reported.[3, 5, 7, 15, 16] We found no significant difference in flow to different regions, nor does a positive result to the reactive hyperemia test imply any significant difference in mean bypass flow. This is in agreement with Kreulen and colleagues[7] who reported a flow of 53 ml. per minute for a positive reactive hyperemia (RH) test and 42 ml. per minute for a negative test (no significant difference). We find that in 63 percent of bypasses there is a positive RH test (i.e. RH > 10 percent). Of those cases of a nonzero RH, the mean RH is 37 percent. Kreulen and associates[7] found a positive test (RH > 10 percent) rate of 43 percent, while Greenfield and his colleagues[5] found a positive result (RH > 20 percent) in 84 percent of grafts in 32 patients. Bittar[6] reported a positive test rate of 76 percent. We find no relation between the RH and bypass flow, in agreement with most other reports. Thus our group is not atypical in that the flow and RH measurements are similar to those reported previously in several independent studies.

Of the studies using myocardial markers to measure shortening of the epicardial minor axis,[17–23] the work of McDonald[18] is most directly comparable by reason of placement and orientation of the markers. However, in order to compare minor axis shortening to localized shortening, it must be assumed that shortening does not vary greatly over the perimeter so defined. McDonald reported a mean SF in the basal area of 15.8 percent (S.D. = 2.0 percent), while the equator SF equaled 12.0 percent (S.D. = 1.6 percent) in patients undergoing operation for mitral valve disease. Our group average SF was 9.8 percent at 1 week, 12.8 percent at 1 month, 13.3 percent at 3 months, and 13.9 percent at 6 months (pooled S.D. = 4.7 percent). These values are similar to results obtained by McDonald at the equator, but our standard deviation is greater, representing the greater variation in SF due to regional disease in our patients.

Those regions showing a paradoxical systolic expansion (PSE) pattern represent a clinically important subgroup. Mean graft flow for this group was 67 ml. per minute compared to 57 ml. per minute for normal onset of shortening (less than 100 msec. from the Q wave); the flows for these two groups are not significantly different. Of the PSE group, 27 percent had an RH of zero versus 32 percent for normal onset of shortening. The intraoperative graft flow and RH measurements for the regions that subsequently developed PSE are, if anything, greater than mean values for the entire group. These results are difficult to explain by a simple graft flow–reactive hyperemia–regional shortening relationship and strongly suggest that other factors not examined in this study are operating here. In our group of 16 patients showing PSE, it is clear that myocardial infarction cannot be the primary cause: Twelve regions developed PSE after 1 week postoperatively, and coincident myocardial infarction could be excluded. Also PSE could not be attributed to bypass closure which is consistent with the findings of Bulkley and Hutchins[24] who found regional necrosis in the presence of patent grafts. At this point we can only speculate about the significance of the apparently absent collaterals 1 year after operation. They were clearly present preoperatively in the majority as a result of high-grade lesions in the diseased artery. However, their absence at 1 year may just as well be due to renewed perfusion preventing their visualization as to some unexplained trauma. It is possible that the transient episodes of PSE represent a temporary imbalance between supply and demand, but the prognostic significance of this finding remains to be established.

The results show that the correlation coefficient between SF and mean bypass flow is greatest for bypasses with a positive RH test (see Table III). For example, the correlation coefficient is 0.65 ($p < 0.01$ against $r = 0$) at postoperative week 1 provided RH is greater than 40 percent, but becomes 0.26 ($p < 0.10$) when the group is extended to all bypasses with RH of more than 0 percent. The fact that a region of myocardium can receive blood to a variable extent, from both the native proximal coronary circulation and the graft, along with collateral blood flow from other coronary vessels, underscores the difficulty in relating postoperative regional epicardial dynamics to measured intraoperative graft flow alone. Arterial blood pressure, temperature, cardiac index, and heart rate at the time of graft flow measurement significantly affect graft flow

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rates and must contribute to any associated variability. Furthermore, even when graft flow can be estimated at the time regional shortening is recorded, the relationship is influenced by the functional status of the myocardium, status of the native circulation, and regional function elsewhere.[13] Thus it is remarkable that a statistically significant correlation can be found in view of the number of other determinants of perfusion and shortening. Nevertheless, the test of the hypothesis requires only that the correlation coefficient be significantly different from zero, not necessarily close to unity. That the correlation between shortening and flow improves by excluding those flow measurements which show no RH or an insufficient response is taken as indirect evidence that bypass-reactive hyperemia relates to the dependence of viable tissue in the bypass region on the functioning of the graft.

The fact that the correlation between SF and intraoperative graft flow becomes nonsignificant before 6 months suggests that events in the intervening period result in significant changes in myocardial shortening: Recovery from operation and effects of extracorporeal circulation, new collateral formation or closure, bypass closure or progression of disease, and altered medical therapy all must play a role.[7, 25] As myocardial shortening shows an increase from 1 week to 6 months (42 percent on average), it would seem that muscle performance increases in spite of the development of pericardial adhesions. Markers placed in normally perfused control regions also showed an increase during the same period, but only one half as great.

Excluding those patients who eventually developed PSE, our results support the hypothesis that graft-reactive hyperemia is related to the dependence of the viable tissue on the functioning of the graft. Although the correlation is sufficiently great to support the hypothesis in the early postoperative period, the correlation is not so close to unity to suggest a direct graft flow–reactive hyperemia–regional shortening relationship of sufficient magnitude to be of prognostic value. Certainly, after 3 months, there is little prognostic information in the flow measurements vis-á-vis regional shortening, and the development of PSE remains unpredictable.

Summary

We explored the hypothesis that RH relates to the dependence of the grafted region on graft flow, and determined to what extent graft flow measurements at operation are predictive of regional shortening in the postoperative period.

In 56 patients 120 bypass regions were studied. Flow was measured with an electromagnetic flowmeter, and the reactive hyperemia test was performed by a 10 second bypass occlusion 30 minutes after termination of extracorporeal support. Epicardial marker pairs were implanted in the region perfused by the bypass and their motion was filmed at 1 week, 1 month, 3 months, and 6 months postoperatively.

Bypass graft–reactive hyperemia and flow measurements were similar to those reported previously in independent studies. There was a positive correlation between SF and mean bypass flow when graft-reactive hyperemia exceeded 20 percent. The correlation was greatest at 1 week and became nonsignificant by 6 postoperative months. This is taken as indirect evidence that RH relates to the dependence of viable tissue on the functioning of the graft, i.e., the absence of alternative perfusion pathways. On the other hand, graft flow and reactive hyperemia in those regions eventually developing a pattern of PSE, as measured during operation, did not differ from these measurements in regions where a normal pattern of shortening was observed. Thus, while our results support the hypothesis in the early postoperative period, graft hemodynamics is not a useful predictor of eventual onset or severity of disturbances in regional wall motion per se.

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