AN INVESTIGATION OF CHARACTERISTICS OF BLOOD FLOW AT A BIFURCATION WITH REFERENCE TO "STEAL"

by

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ABSTRACT

Many factors affect the relative division of blood at a bifurcation. Clinically significant conditions sometimes result in a flow reduction to one downstream branch of a major bifurcation as the result of a reduction in the flow resistance of the contralateral branch. This condition is called "steal" and has been demonstrated at several major bifurcations in man. This behavior has been successfully modeled using the parameters of flow, pressure, and resistance in the upstream and two downstream vessels. The model was confirmed experimentally in mongrel dogs by a pressure versus flow study at the external iliac trifurcation. Flows were measured electromagnetically, and pressures by a fluid-filled catheter and diaphram LVDT transducer. Resistances were manually controlled. As a result of this study a simple test was devised to determine if a site selected for vascular surgery has a potential for developing steal.

ACKNOWLEDGEMENTS

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INTRODUCTION

The systemic vascular system incorporates branching of blood vessels at all levels, from the aorta to the capillaries. Blood flow usually divides at each branch without adverse consequences. The pressure in the major vessels of the body is pulsatile, varying from the high, systolic pressure pulse that is caused by the ventricular contraction, to the low diastolic value that occurs as a result of the relaxation of the ventricles. These highs and lows, however, fluctuate about a rather constant "mean" arterial pressure. This pressure is maintained through a variety of biological control mechanisms including the baroreceptors and stretch receptors associated with the aorta and larger vessels. This mean pressure is essentially the driving force for the movement of blood to the capillaries and back to the heart. Thus, flow to each of the first few branches is determined by the peripheral resistances of the vascular beds supplied by each respective branch. This is expressed mathematically by the phenomenological relationship:

$$\Delta P = QR \qquad (1)$$

Where ΔP is the pressure loss or drop between two points, Q is the flow and R is the associated resistance. This relationship is true for any portion of the circulatory system, but, as it will be shown, the pressure must be the actual value at a point and should not be assumed to be equal to the commonly-used clinically-measured blood pressure.

Blood flow at a bifurcation (where one vessel divides into two daughter vessels) is governed by the same factors and relationships that determine flow in the other parts of the system. It has been clinically observed, however, that when one daughter branch requires a high flow rate, the other daughter branch or branches can be deprived of their needed blood flow and ischemia in the area perfused by these branches can develop. This phenomenon has been named the "steal" syndrome and is of clinical importance in many situations involved with flow in the vicinity of a stenosis or surgical revascularization. Little quantitative research has been done in this area of hemodynamics, although the qualitative aspects of a variety of "steal syndromes" have been described. This study presents an analysis of the mean pressure-flow characteristics of dividing blood flow. This analysis is incorporated in a model of flow at a bifurcation. This study presents the methodology used to experimentally confirm the model and presents some of the results of the experiment. In addition, some of the related published literature is discussed.

LITERATURE REVIEW

"Steals" and related arterial phenomena have been reported since Cortornie described the clinical case of "subclavian steal" in 1960 (Isch, 1975). This condition was discovered because there was a retrograde or reverse flow of blood in a patient's left vertebral artery. Although this would at first seem impossible it is easily explained. Four major arteries supply the head and brain, these being the left and right carotid and left and right vertebral arteries. The two vertebral arteries latter join to form the basilar artery. The basilar artery is indirectly joined with the two carotids by the circle of Willis (the circle is not always complete). The left proximal subclavian artery was occluded, therefore the only available blood supply to the left arm was via the basilar artery and retrograde flow down the left vertebral. The basilar artery supplied this retrograde flow from the normal blood flow to the brain in the other three arteries (Callow 1964, Sammartino and Toole, 1964). The total flow in the three "normal" vessels often equalled that in all four arteries in normal patients. Under these conditions there is no cerebral blood insuffiency and patients are often asymptomatic. Most patients have additional occlusive disease and show classical symptoms of cerebral insufficiency including dizziness, loss of memory, and fainting.. (Killen, et.al., 1966, Siekert, er.al., 1964) The same retrograde flow occurs in the right vertebral artery following occlusion of the

innominate artery. (Killen, et.al., 1966)

It was, however, soon determined that total occlusion of the involved proximal artery was not necessary to observe reversed vertebral artery flow. It was also found that the reversed flow could be intermittant, only occuring following exercise of the involved arm. This situation is more complex. In this situation two possible paths of blood flow exist. When the flow requirements of the vascular beds supplied by the occluded artery are increased, the pressure drop across the obstruction increases directly with flow (according to equation 1). When the pressure drop becomes sufficiently large, the gradient from the vertebral-subclavian bifurcation to the basilar artery becomes negative and retrograde vertebral artery flow will result. (Heyman, et.al., 1964). It is important to note that this alternate retrograde flow possibility is the exception rather than the rule in the systemic circulation.

The "subclavian steal", as shown, does not truely represent the spectrum of other steal syndromes. In most of the body the daughter vessels of the larger bifurcations are isolated, linked only by the generally insignificant collaterals. It has been well documented that aorta-illiac revascularization (re-establishment of the blood supply to the legs) can cause a stealing of blood from the mesenteric artery with resultant intestinal gangrene (Connally and Stemmer, 1973). "Steals" have been investigated in the coronary arteries (Meyer, et.al., 1974).and in sketetal muscle arteries (Hirche and Bovenkamp, 1974). The emphasis of these later studies was the clinical nature of the "steal". Steal syndromes involving the pulmonary and celiac arteries

have been described recently. (Isch, 1975).

As previously stated, "steals" have been reported even in the abscence of alternate pathways of blood flow such as exists in the subclavian steal syndrome. The focus of this study has been to develop a quantitative analysis of flow division, including steal, at an idealized bifurcation. Two clinical studies concerning steal at a bifurcation were found in the literature. Dogs were used to investigate the aorta-iliac artery trifurcation, with the median sacral artery tied off to form a bifurcation. In this configuration the blood supply to each leg is largely independent. One study, by Grimes, et.al., recorded steal subsequent to a period of occlusion. In their case the reported steal was the result of a transient reactive hypermia (Grimes, et.al., 1972). The other study by Ehrenfeld, et.al., reported that "steal" was a misnomer when the proximal pressure was normal. Their study claimed that the flow to one side could be increased ten-fold without a significant drop in contralateral flow (Ehrenfeld, et.al., 1968). The model and results in this study show how these apparently contradictory results are indeed correct. The contradiction is an indication of a lack of real understanding on the division of blood flow at a bifurcation.

MODEL

It is generally accepted that for the purposes of describing the systemic circulatory system the heart is a constant pressure source, delivering pulsatile pressure about a constant mean. This mean pressure value is for the most part independent of flow, being regulated by the baroreceptors in the aortic arch and stretch receptors in the aorta and great vessels. It is also well known that blood flow follows a phenomenological parallel to Ohm's law, namely the pressure drop, ΔP, is equal to the product of flow, Q, and resistance, R. This is expressed:

$$\Delta P = QR \qquad (1)$$

The arterial pressure which drives the flow is disipated in the arterial system, capillaries, and venous system until the pressure of blood returning to the heart is essentially zero. This model assumes a venous pressure of a constant zero value. The arterioles and capillaries dissipate most of the pressure and therefore are responsible for most of the resistance to the flow of blood. Thus the overall system meets its variable flow requirements primarily by changing the resistance in the arterioles and capillaries while maintaining the constant pressure as discussed above.

Using the relationship in equation (1) for a system with a constant pressure source of pressure P (representing the heart) which delivers a flow of Q_T to a single vessel of resistance R , which subsequently

divides into two downstream vessels, one of resistance R_1, the other of resistance R_2, the following results can be obtained. (This situation exactly parallels the major bifurcations of the arterial tree. It is also analogus to the experimental phase of this study but is more universally applicable.) The pressure at the division, P_B, is:

$$P_B = P - (Q_T)(R) \tag{2}$$

The flow, Q_T, is given by:

$$Q_T = P/(R + (R_1R_2/R_1 + R_2)) \tag{3}$$

The two branch flows, Q_1 and Q_2 are given by:

$$Q_1 = P_B/R_1 \text{ and } Q_2 = P_B/R_2 \tag{4}$$

Combining equations (2) and (3) and (4) results in:

$$Q_1 = PR_1R_2/R_1(R(R_1 + R_2) + R_1R_2)) \tag{5}$$

$$Q_2 = PR_1R_2/R_2(R(R_1 + R_2) + R_1R_2)) \tag{6}$$

Note that if R is very small Q_1 and Q_2 reduce to:

$$Q_1 = P/R_1 \text{ and } Q_2 = P/R_2 \tag{7}$$

And:

$$P_B = P \tag{8}$$

These results closely approximate the normal hemodynamics at the major divisions of large vessels because R is much less than the total resistance of the arterial system. These results are not unexpected and under these conditions the downstream flows, Q_1 and Q_2, are completely independent of one another being governed by R_1 and R_2 respectively. The total flow is, of course, constrained by the flow balance:

$$Q_T = Q_1 + Q_2 \tag{9}$$

"Steal" has previously been described as a reduction in flow to the region supplied by one downstream branch of a bifurcation in response to a reduction in resistance in the downstream contralateral branch. This situation is in contradiction to equation (7) in which the flows are independent. Thus the assumption that R is very small will not be imposed on the model. From the definition of steal a dimensionless "steal coefficient" may be defined. The "steal coefficient", q, is defined as the ratio of flow in branch 1, Q_1, when the resistance of the contralateral side, R_2, is equal to some fractional portion, α, of the resistance of side 1, R_1, to the flow in branch 1 when $R_1 = R_2$. This fractional proportion of R_1, is thus defined as α such that the relationship of resistances described above is:

$$R_2 = \alpha R_1 \tag{10}$$

Applying these conditions and substituting from equation (5) results in:

$$q = \frac{\alpha/(R\ (R_1 + \alpha R_1) + \alpha R_1^2)}{1/(R\ (2R_1) + R_1^2)} \tag{11}$$

As previously explained the small upstream resistance approximation is not appropriate for simulating the steal phenomenon. It is, however, desirable to include the resistances, R and R_1, in some dimensionless form. This is accomplished defining a dimensionless quantity, $r = R/R_1$. Substituting this into equation (11) the expression for q becomes:

$$q = \frac{\alpha(2r + 1)}{r + \alpha(r + 1)} \tag{12}$$

From equation (12) it is clear that as α approaches 1 the value of q approaches 1 also, which is the required result. It is also

clear that as r approaches 0 (the small upstream resistance approximation) q approaches 1. This indicates that for a small upstream resistance q is relatively independent of α. This, agrees with equation (7) which states that for these conditions Q_1 is not a function of R_2.

The "steal coefficient" is now written in terms of α, the resistance ratio of R_2 to R_1 and r, the resistance ratio between R and R_1. A plot of q as a function of α for different values of r is shown in figure 1. This plot is illustrative of several significant points. When the upstream resistance is small the ratio r is also small. This is a very good approximation of the situation in a healthy arterial tree. As already discussed under these conditions the flow, Q_1, is largely independent of R_2 and therefore α. The curve representing r = .01 shows that R_2 would have to be less than one-tenth that of R_1 (α = .1) before the flow, Q_1, would fall to 90% of its value when R_1 equals R_2. It is obvious that some effect severely lowering R_2 would normally be required to significantly effect the blood flow in side 1. At the other extreme, one observes that, for larger values of r (r = 10), q (and thus the flow to side 1) is almost a linear function of α (and thus a function of R_2). The dependence of Q_1 on R_2 is the definition of the "steal" phenomenon. The conclusion must be that a significant upstream resistance is needed in order to have the linking of downstream flows and a "steal" syndrome.

By similar manipulations it can be shown that the dimensionless ratio of pressure drop at the bifurcation to the system pressure drop

is expressed by:

$$(P - P_B)/P = \frac{r(1 + \alpha)}{r(1 + \alpha) + \alpha} \qquad (13)$$

Once again as r approaches 0 the ratios in equation (13) approach 0. This indicates that $P_B = P$ as predicted in equation (8). The pressure drop ratio, $(P - P_B)/P$, is shown as a function of α for various values of r in figure 2. This figure shows that the pressure drop ratio (which is indicative of the upstream pressure loss) is dependant on α, the ratio of the two downstream resistances. It also shows the expected dependance on the upstream to downstream resistance ratio (expressed in the variable r).

It will be shown that this model closely approximates the behavior of the external iliac bifurcation in the dog. This model is of key importance to the data analysis in the study.

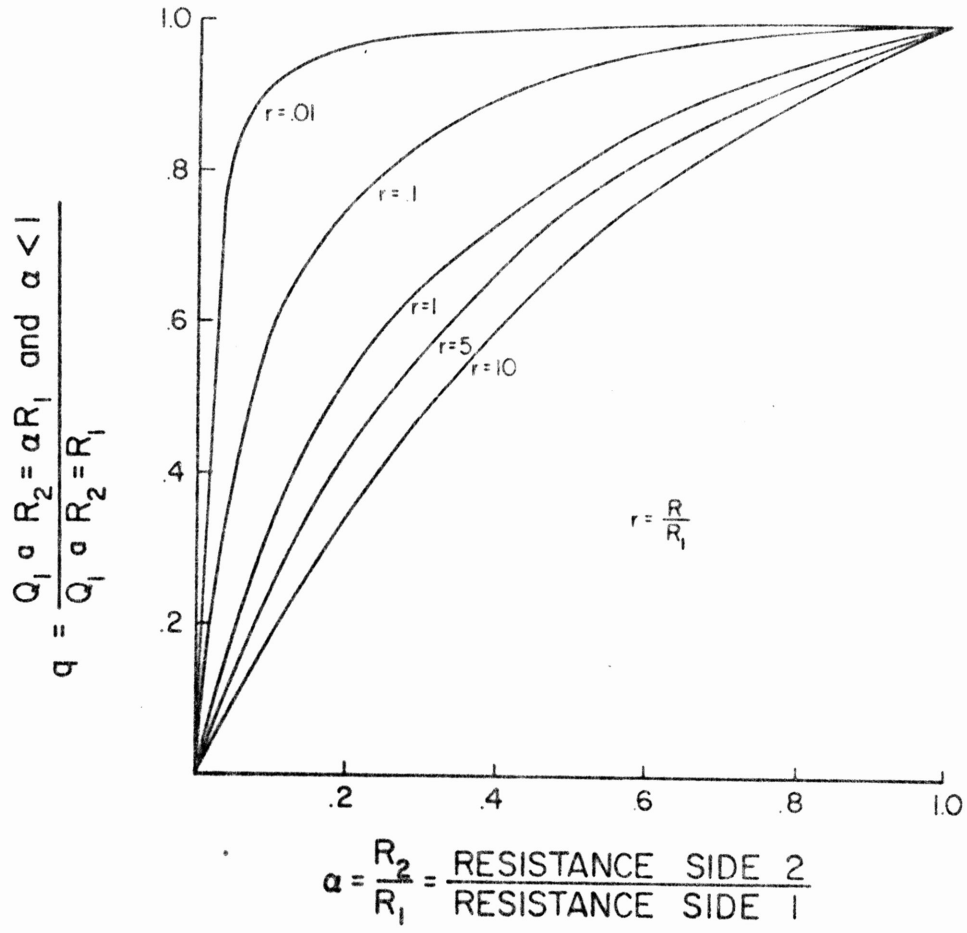


FIGURE 1

Dimensionless Flow as a Function
of Dimensionless Resistance

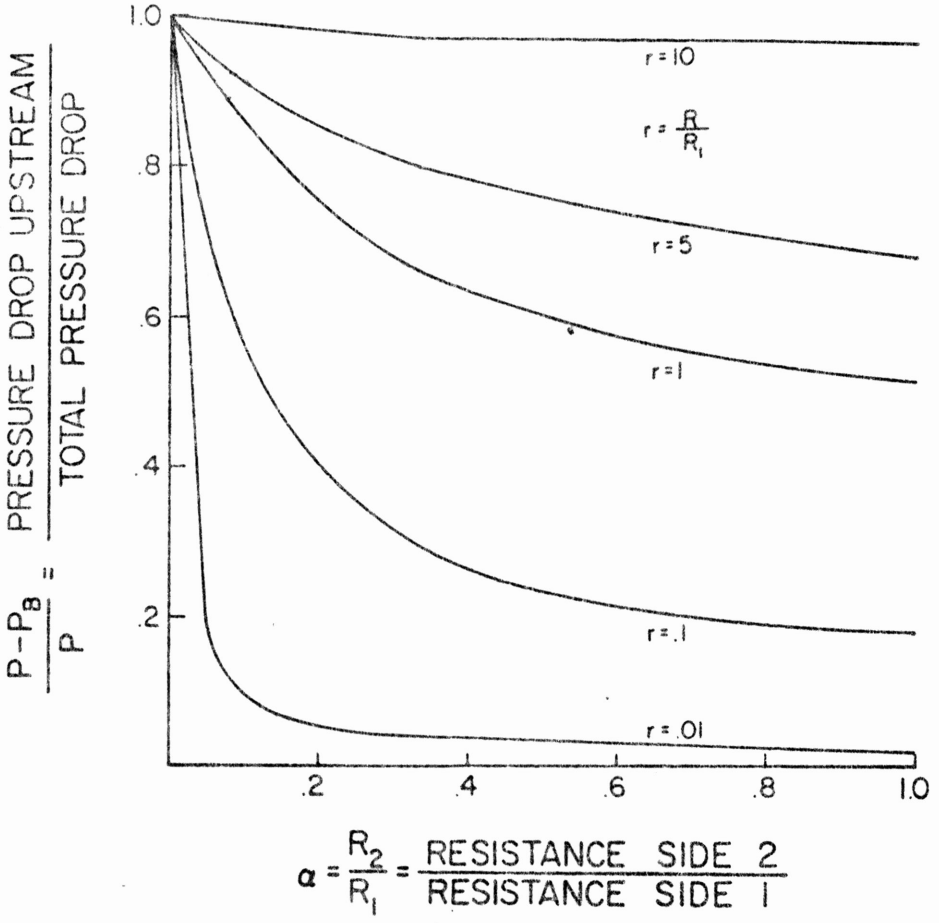


FIGURE 2

Dimensionless Pressure as a Function
of Dimensionless Resistance

METHODS AND MATERIALS

The experimental phase of this experiment was performed on mongrel dogs. Dogs were chosen for their size, suitability, cost, availability, and consideration of the facilities for confinement and surgery. The experimental apparatus consisted of a Biotronex Laboratory, Inc. (BLI) 630 pressure amplifier and a BLI 9630(X)-C9LVDT (linear variable displacement transformer) pressure transducer. The transducer was calibrated with a mercury manometer and used with heparinized saline-filled plastic catheters. Becton and Dickenson metal stopcocks were connected to form a manifold for flushing the catheter. The flows were measured with a BLI 630 pulsed logic electromagnetic flowmeter. It was found that the external iliac arteries of dogs of 15.9-20.4 Kg. (35-45 lbs.) were best fit by a probe with a 3.5 mm. lumen. The probes used were precalibrated and the calibration was checked by in-vitro methods.

Laparotomies were performed on mongrel dogs anesthetized with sodium pentobarbital (30 mg/Kg. initially, additional as needed). The intestines were retracted to expose the aortic trifurcation. Two electromagnetic flow probes were placed around the external illiac arteries. Effort was made to place each probe an adequate distance from the trifurcation. The probe wires were sutured to the body to help prevent cable torsion and the resulting signal artifact. The ground wires were sutured to the abdominal wall muscles.

A balloon type occluder was placed around the abdominal aorta as far upstream as reasonably possible. In dogs where the circumflex iliac arteries and/or the caudal mesenteric artery had origins between the occluder and the trifurcation, these arteries were tied off. The occluder was left open initially.

The internal iliac arteries were next ligated as far distal to the trifurcation as possible. A loose tie of umbilical tape was next placed around the median sacral artery. A small cut was made between these ligatures and the pressure measuring catheter was placed in the system with the open tip protruding up into the aorta. The trifurcation was in effect changed into a bifurcation by tightening the umbilical tape until hemostasis was achieved. The catheter was sutured to the body to help prevent it from becoming accidentally dislodged.

A cutdown was performed on each medial upper thigh to expose the femoral artery and vein. The distal portion of both arteries and both veins was tied off. Loose ligatures were placed around the proximal portion of all four vessels. Two mechanical arterio-venous fistulas were made by cannulating the artery and vein on each side with sections of "treated" vinyl tubing. The proximal venous ligatures were tightened and arterial cannulas were threaded up the arteries until the ends could be felt in the external iliac arteries just distal to the flowmeters. The proximal arterial ligatures were then tightened and additional ties were placed around the cannulated external iliac arteries distal to the open cannula ends. The resistance of these mechanical A-V fistula were independantly regulated by means of screw clamps. The vinyl tubes had been treated

by soaking for 12 hours in solution of Clay Adams "Siliclad" (a concentrated mixture of high molecular weight silicones). The purpose of this treatment was to delay the clotting of the tubes. The fistula resistance was periodically reduced causing the flows to typically exceed 400 ml/min. in order to "flush out" any clotting products.

The mean pressure and flow data were recorded manually and on a strip chart recorder for subsequent analysis. The basic test performed involved several steps. First, the flow on each side was adjusted by the screw clamps to approximately the same flow as was observed before the mechanical A-V fistula was inserted. The pressure at the bifurcation was measured and noted. By sliding the pressure catheter farther up the aorta the mean pressure was observed to be virtually constant. This indicates that no major stenosis or obstruction was present in the terminal section of the aorta. Then the flow being measured on one side was observed while the flow on the other was increased or decreased. Pressure was also observed. The flow meter was switched to measure the opposite side and the procedure was repeated.

These results were similar to those observed in dogs without A-V fistulas whose downstream resistances were altered with other mechanical or pharmacological methods. The advantage of the mechanically controlled A-V fistulas was two-fold. When no fistulas were present and one downstream vessel was occluded no pressure change was noted but a 20% increase in flow to the opposite side was observed. Investigation revealed that the dog is noted for its collateral circulatory system. In all likelihood the flowing side was diverting blood to

the occluded side by newly opened collaterals. This communication was not desirable and not thought to be particularly related to "steal". The increase in flow when the opposite side was occluded is the reverse of "steal", and is due to a different phenomenon. The A-V fistula described will prevent this communication between the two legs. The second advantage was related to the equipment used. The BLI flow probes have a problem of slow baseline drift. This necessitates rebalancing of the flowmeter before a measurement is taken. The in-vivo balancing is done by occluding the involved artery to cause a zero flow. This would normally result in a period of reactive hyperemia being superimposed on the steady-state flow one is attempting to measure. The mechanical A-V fistula allowed the experiment to be conducted without this problem.

After this documentation of the "normal" state, the balloon occluder was used to simulate a diseased, stenotic lower aorta. First, the two downstream resistances were adjusted to obtain flows that approximate the normal physiological flows. The pre-occlusion pressure at the bifurcation was noted. This had already been shown to be very good approximation of the upstream pressure. The balloon occluder around the aorta was inflated. The pressure transducer was used to monitor the degree of occlusion. One flowmeter probe was balanced and the opposite side was occluded. The flow to the side being measured was now varied and the pressure drop across and flow through the occlusion were recorded. The pressure-flow data was then recorded with the flow through the opposite side to minimize variation. The two downstream sides were again adjusted to

the "normal" flow rate. By measuring one side the resistance on the opposite side could be decreased to now demonstrate "steal". The knowledge of the pressure at the bifurcation and the pressure-flow relationship of the stenosis allowed the computation of the upstream flow. The knowledge of the measured downstream flow allows the subtractive computation of the other downstream flow.

Through these techniques the examination of the effect of uneven side to side resistances and of an upstream resistance were examined in-vivo. The results parallel the prediction of the model presented in this study (see Results). The experimental set-up described above is also shown in figure 3.

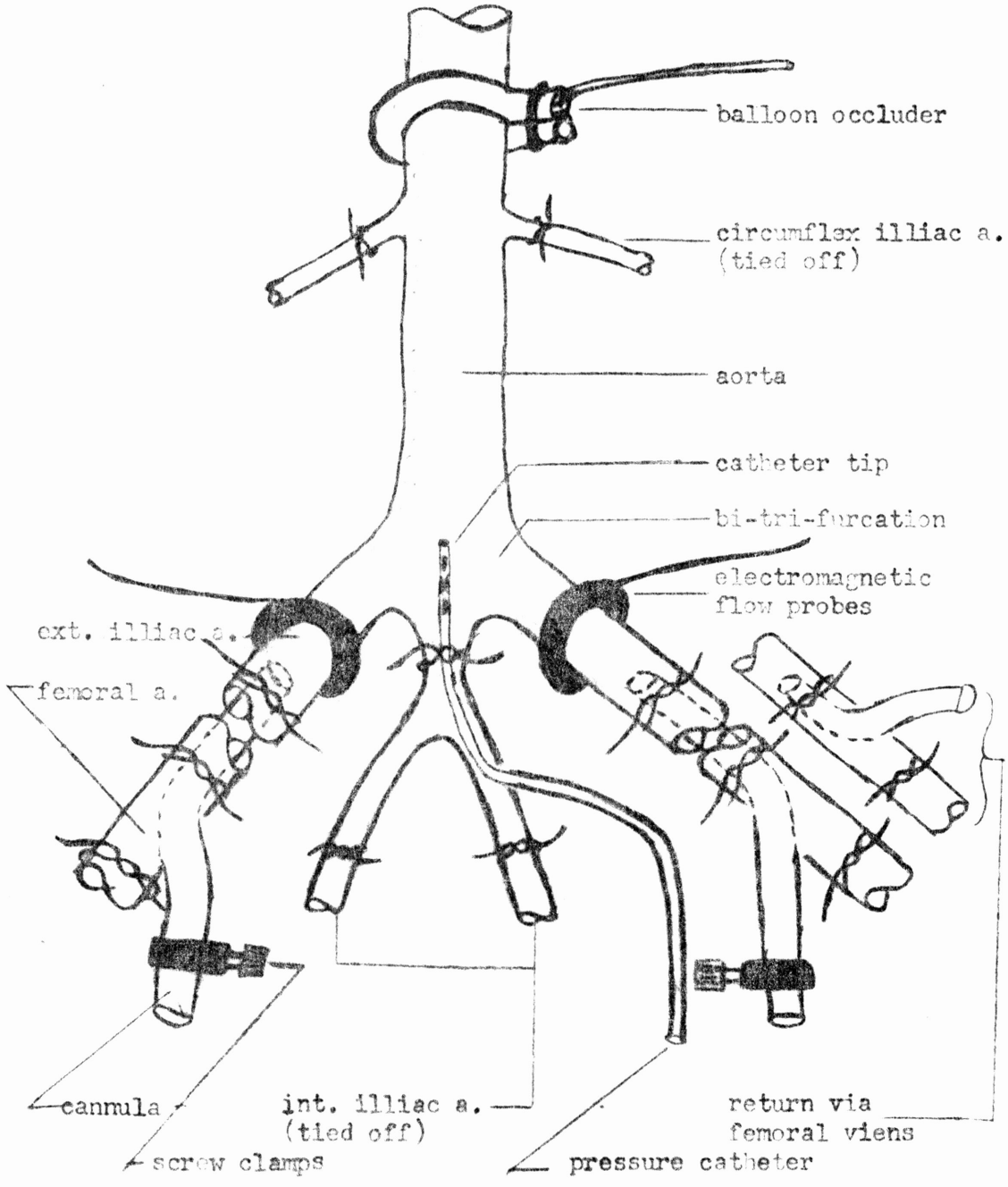


Figure #3

Design of Experiment

RESULTS

The resluts of the experiment show good agreement with the predictions of the model. The results of the test performed without any significant aortic (upstream) resistance were very uniform. First, extremely high total flows, generally 5 liter/min or more, were required to observe a significant pressure drop at the bifurcation. This result is consistent with model predictions for a very small upstream resistance, R, as expressed in equation (8). Secondly, the two downstream flows were almost completely independent of each other. Ten-fold increases in a downstream flow were required to significantly reduce the contralateral flow. This also agrees with the model predictions for a small upstream resistance as given in equation (7). The results for this "normal" hemodynamic state are in precise agreement with the results published by Ehrenfeld, et al. (1968). The results were similar in all dogs tested and were very reproducible. Due to the unchanging nature of the values measured the data is of little quantitative interest. It is not reported in this study.

The results of tests performed with a stenotic (balloon occluder inflated) aorta were very interesting. The results closely followed the predictions of the model given by equations (12) and (13). The steal phenomenon is shown in figure 4. This figure, taken from a strip chart recording, demonstrates the interaction of the downstream flows when an upstream resistance is present.

Data is presented from four stenoses. The data for each stenosis consists of two parts. The first part consists of the pressure drop versus flow data for the occlusion. This is presented in tables 1 and 2 and figures 5 through 8. The figures show a pressure drop versus flow plot for each of the four stenoses analyzed. These graphs have a characteristic shape. The initial (lower pressure drop) section is linear but the portion representing the higher flow rates falls away from the linear relationship. At least two explanations are possible for this deviation. First is the pressure component due to the velocity of the fluid. As the flow increases, the velocity of the fluid entering the end port of the pressure measuring catheter increases. The Bernoulli equation predicts the pressure artifact due to this effect will be proportional to the square of the velocity. Thus the deviation due to this effect is expected to be more significant at higher flows. The second mechanism might be due to high Reynold's number effects in the immediate region of the stenosis. At high Reynold's numbers pressure drops for restrictions characteristically are proportional to the square of the velocity. In all four plots the deviation is in the expected direction.

The second part of the data is presented in table 3. This data was taken following the recording of the data for the pressure-flow curve of each stenosis. The data actually taken were the downstream pressure, P_B, and the flow on one side, Q_1, as the resistance on side two, R_2, was changed incrementally. From the ΔP vs. Q plot for each stenosis, the total flow, Q_T, was found. Q_2 was found subtractively from equation (9). From equation (1) resistance can be found by taking

the quotient of pressure drop and flow. Thus r was found by the ratio:

$$r = \frac{(P - P_B)/Q_T}{P_B/Q_1} \qquad (14)$$

The calculation of α was equally simple. The expression used was:

$$\alpha = \frac{P_B/Q_2}{P_B/Q_1} = \frac{Q_1}{Q_2} \qquad (15)$$

The data shows that the downstream flows were set to be equal, or nearly so, for each stenosis. This was done once for each stenosis by first adjusting for equal flows and then applying the stenosis. The downstream resistance ratio was unchanged by this action. Therefore the two downstream flows were still approximately equal. This was checked manually. These values allow the calculation of the steal coefficient, q, by the following expression:

$$q = \frac{Q_1 \text{ @ given rate}}{Q_1 \text{ @ } Q_1 = Q_2} \qquad (16)$$

The calculation of $\Delta P/P$ was straightforward. It was evaluated by its definition:

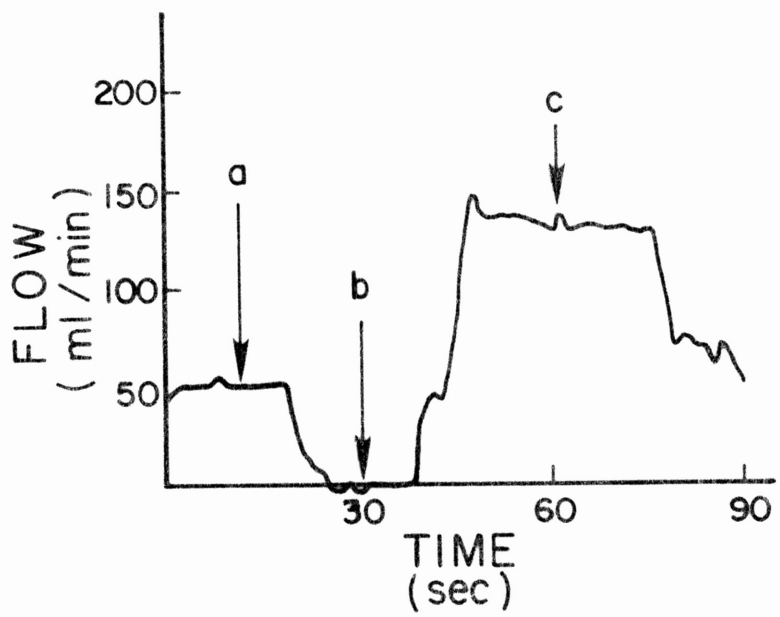
$$\Delta P/P = (P - P_B)/P \qquad (17)$$

The numerical values found in equations (14) through (17) are dimensionless.

An interesting observation was the consistancy of the calculated r value for each stenosis. Neither the upstream resistance, R, or the resistance of one downstreams side R_1, were changed during the experiment. Since r is the ratio of R to R_1, this consistancy was expected. The relatively low values of r are indicative of an important concept. When the balloon occluder was inflated too far

the artery would collapse. The value of pressure that causes complete occlusion is called the critical closing pressure. Higher upstream resistances can be formed by using more than one balloon occluder. The balloons would of course be used in series along the aorta. This was not done in this experiment. Even so, the largest r (r = .19) is quite sufficient to conclusively demonstrate the steal syndrome.

The correlation of the experimental data with the model's predictions are shown in figures 9 through 12. Each figure presents the q versus α and $\Delta P/P$ versus α plot for one stenosis (one r value). The discrete data points from the experiment are shown on each plot. Small deviations between the theory and practice are observed. This small "error" does not invalidate the model. Conversely, the small size of the deviation adds credibility to the model presented. The results not only validate the model but point to several conclusions that can be drawn. These will be explained in the last section.



Demonstration of Steal
FIGURE 4

Flow in left external iliac artery of mongrel dog (20Kg). Abdominal aorta occluded with balloon occluder. Mean blood pressure= 111 mmHg. Three parts represent a) right external iliac flow of 70 ml/min. b) right side flow fully open - 400+ ml/min. c) right side closed - 0 ml/min. Both downstream resistances controlled by mechanical shunts.

TABLE #1

Pressure Drop Versus Flow

Stenosis #1 — Upstream Pressure = 110mmHg

Downstream Pressure mmHg	Downstream Flows ml/min	
	Right	Left
91	60	65
90	125	0
84	170	0
76	210	0
51	275	0
100	65	0

Stenosis #2 — Upstream Pressure = 127mmHg

Downstream Pressure mmHg	Downstream Flows ml/min	
	Right	Left
93	70	75
116	45	0
109	87	0
99	120	0
77	180	0
49	250	0

TABLE #2

Pressure Drop Versus Flow

Stenosis #3 — Upstream Pressure = 111mmHg

Downstream Pressure mmHg	Downstream Flows ml/min	
	Right	Left
94	77	70
108	0	110
96	0	125
91	185	0
70	240	0
48	0	250

Stenosis #4 — Upstream Pressure = 131mmHg

Downstream Pressure mmHg	Downstream Flows ml/min	
	Right	Left
105	60	60
122	0	40
111	0	70
106	0	135
58	0	240

TABLE #3
MODEL CONFIRMATION DATA

	P_T	Q_1	Q_2	Q_T	r	α	q	$\Delta P/P$
	mmHg	ml/min.	ml/min.	ml/min.				
Stenosis #1	91	60	65	125	.10	.92	.95	.17
Pressure	82	55	130	185	.10	.42	.88	.25
Upstream 110 mmHg	60	35	215	250	.12	.16	.56	.45
Stenosis #2	93	70	75	145	.18	.93	.96	.26
Pressure	82	60	115	175	.19	.52	.83	.35
Upstream 127 mmHg	70	45	155	200	.19	.29	.62	.45
Stenosis #3	94	70	70	140	.09	1.0	1.0	.15
Upstream	87	65	145	210	.08	.45	.93	.22
Pressure 111 mmHg	78	45	180	225	.08	.25	.65	.30
Stenosis #4	105	60	60	120	.12	1.0	1.0	.20
Upstream	88	53	147	200	.13	.36	.88	.33
Pressure 131 mmHg	80	43	177	220	.12	.24	.72	.39

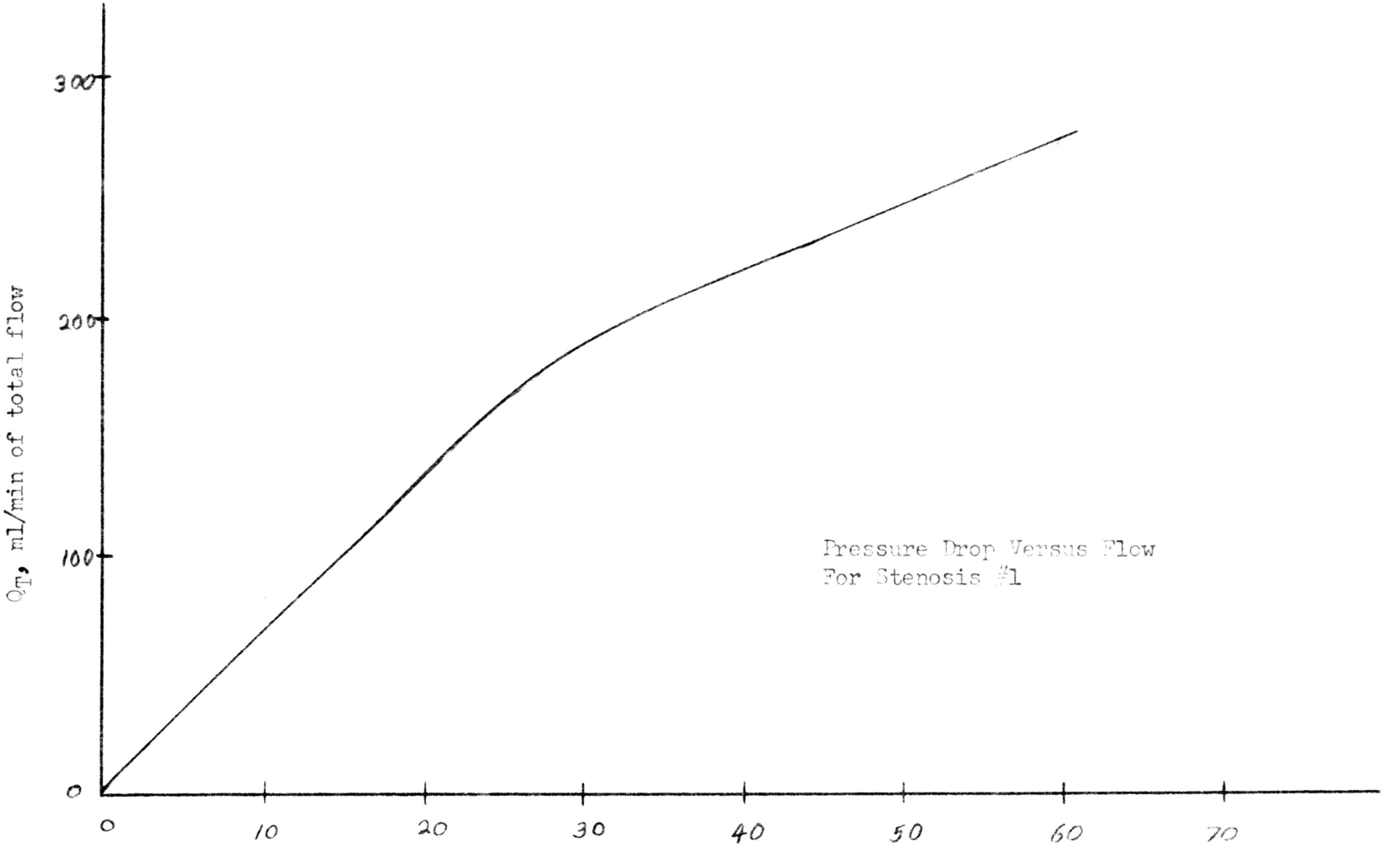
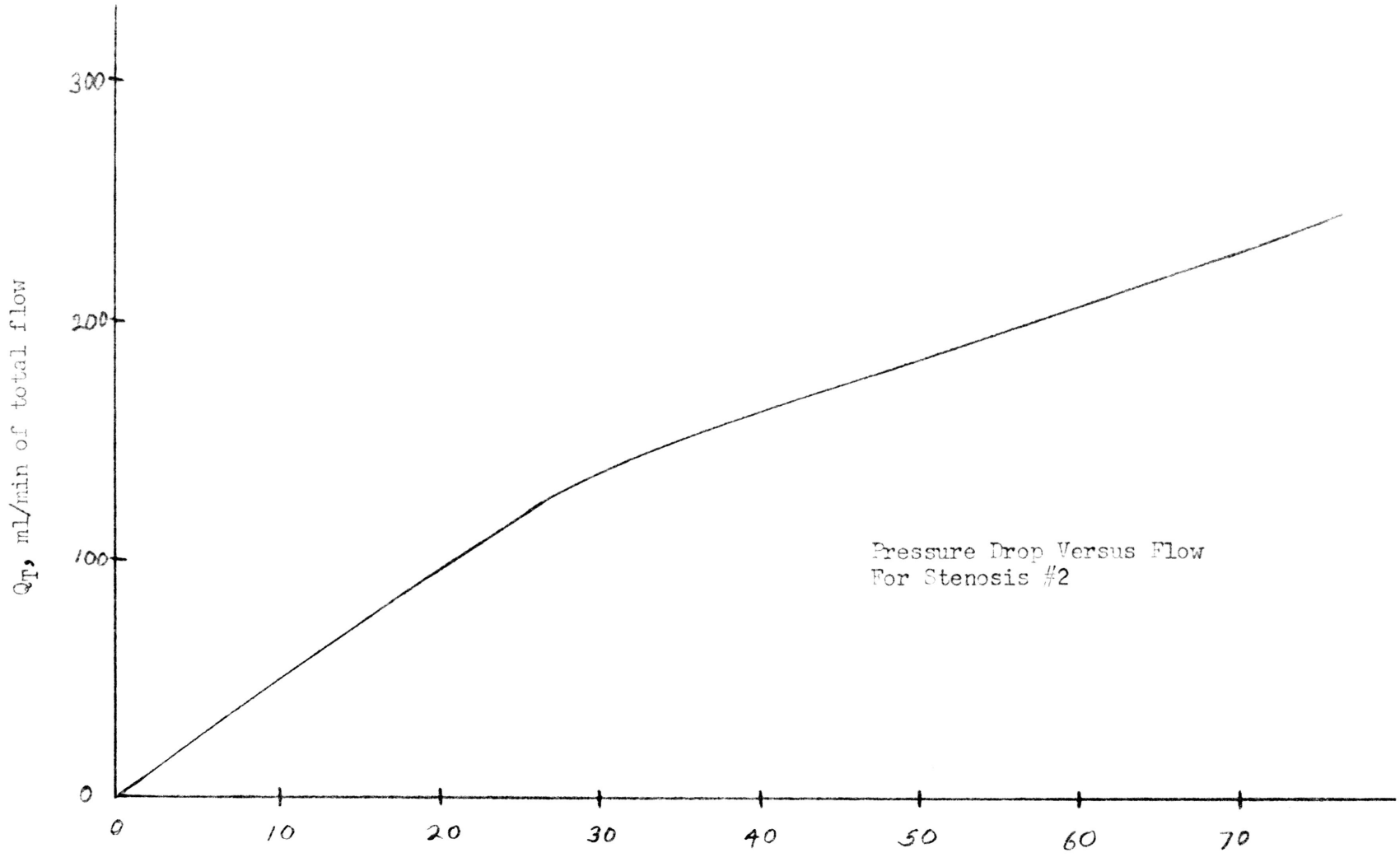

300
200
100
0
0
10
20
30
40
50
60
70
Qp, ml/min of total flow
Pressure Drop Versus Flow
For Stenosis #1
ΔP, mmHg Pressure Drop Across Bifurcation

Figure 5


300
200
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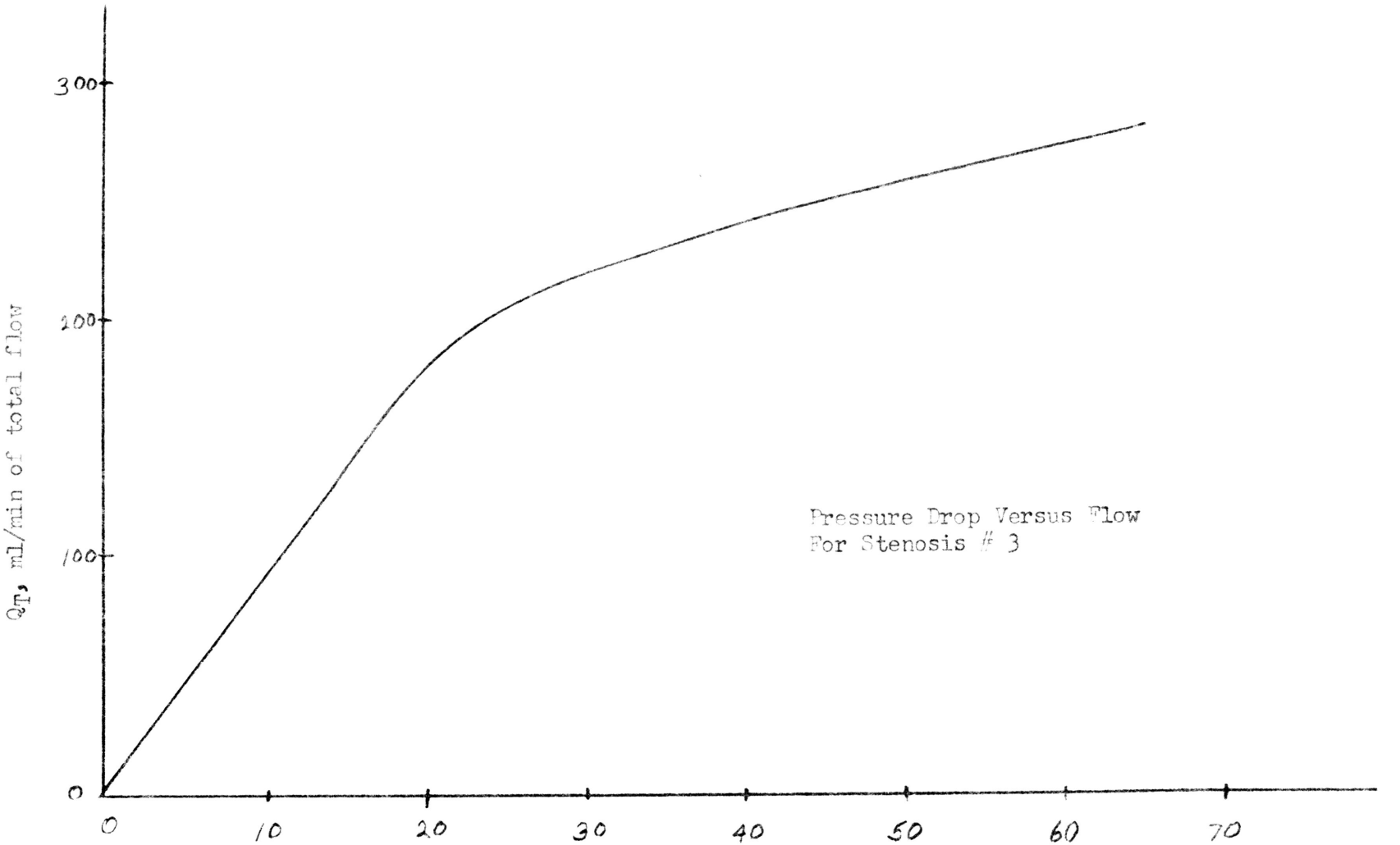
40
50
60
70
Qp, ml/min of total flow
Pressure Drop Versus Flow
For Stenosis #2
ΔP, mmHg Pressure Drop Across Bifurcation

Figure 6



ΔP, mmHg Pressure Drop Across Bifurcation
Figure 7

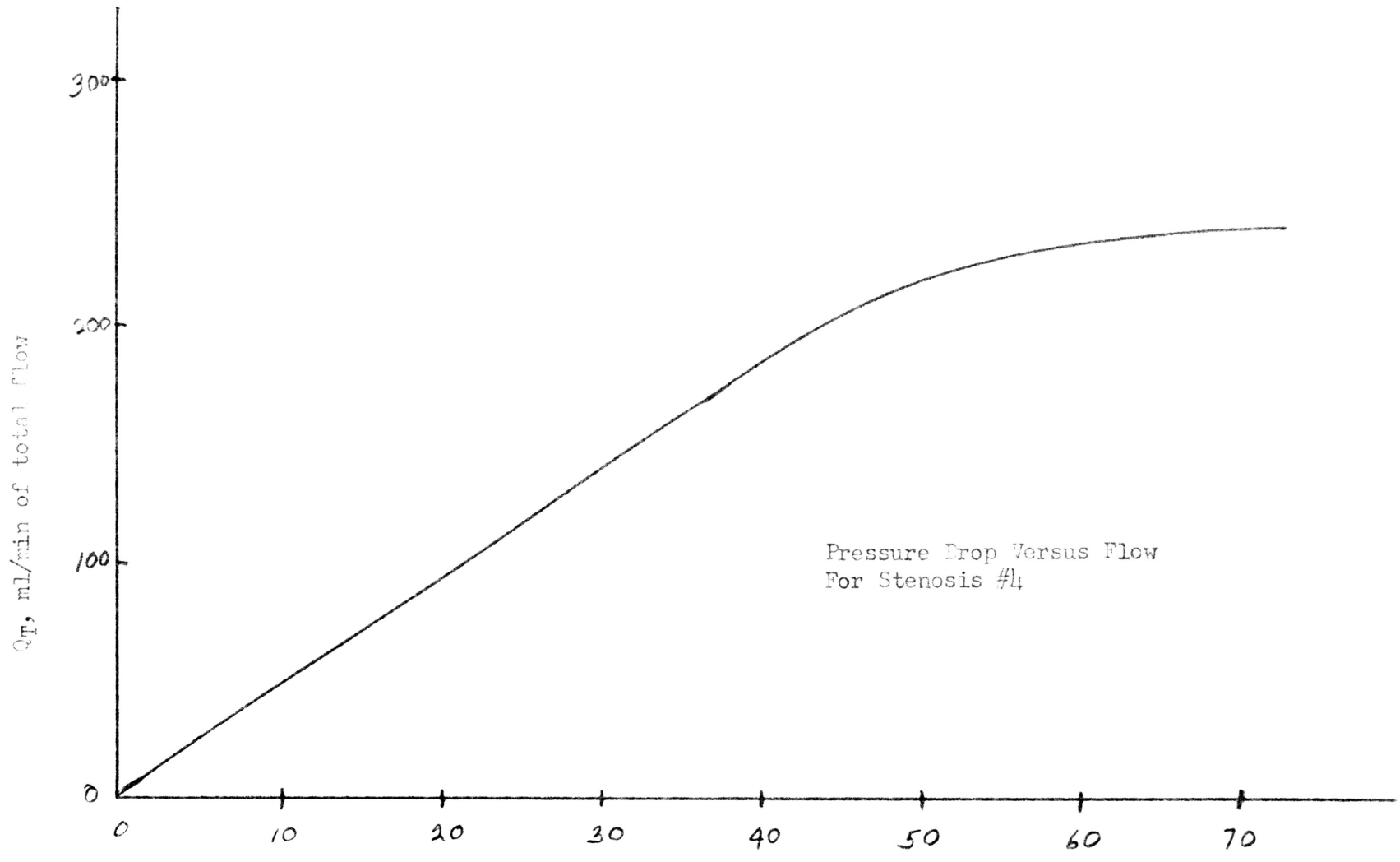
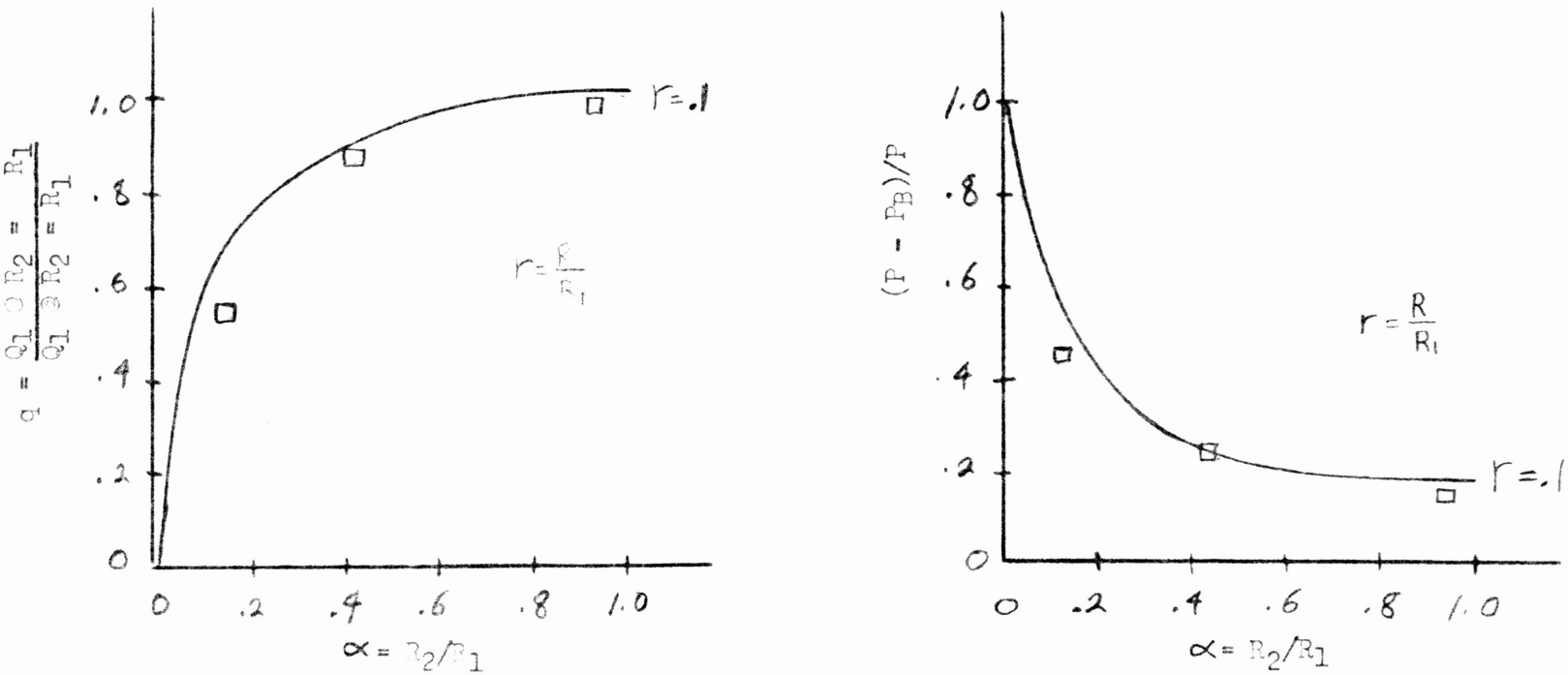

300
200
100
0
0
10
20
30
40
50
60
70
Q_T, ml/min of total flow
Pressure Drop Versus Flow
For Stenosis #4
ΔP, mmHg Pressure Drop Across Bifurcation

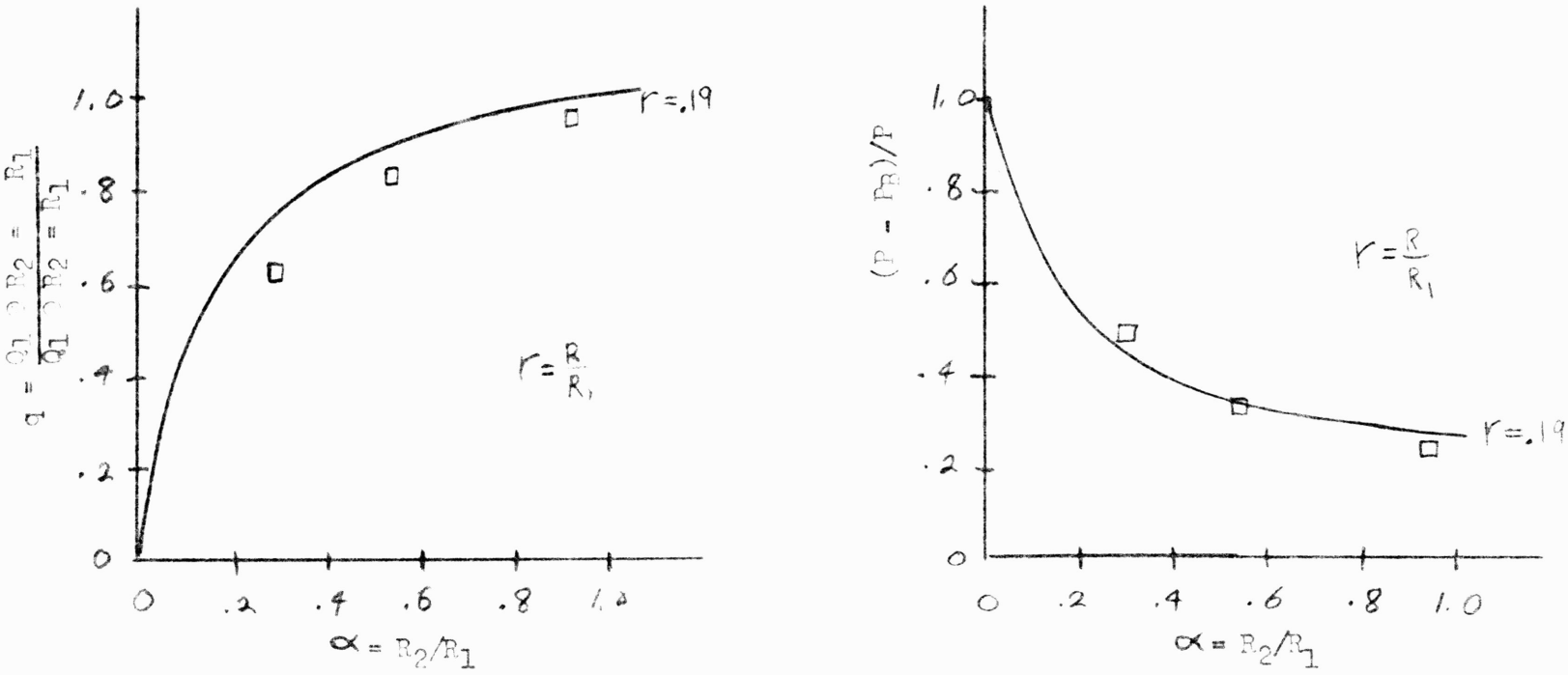
Figure 8

STENOSIS #1



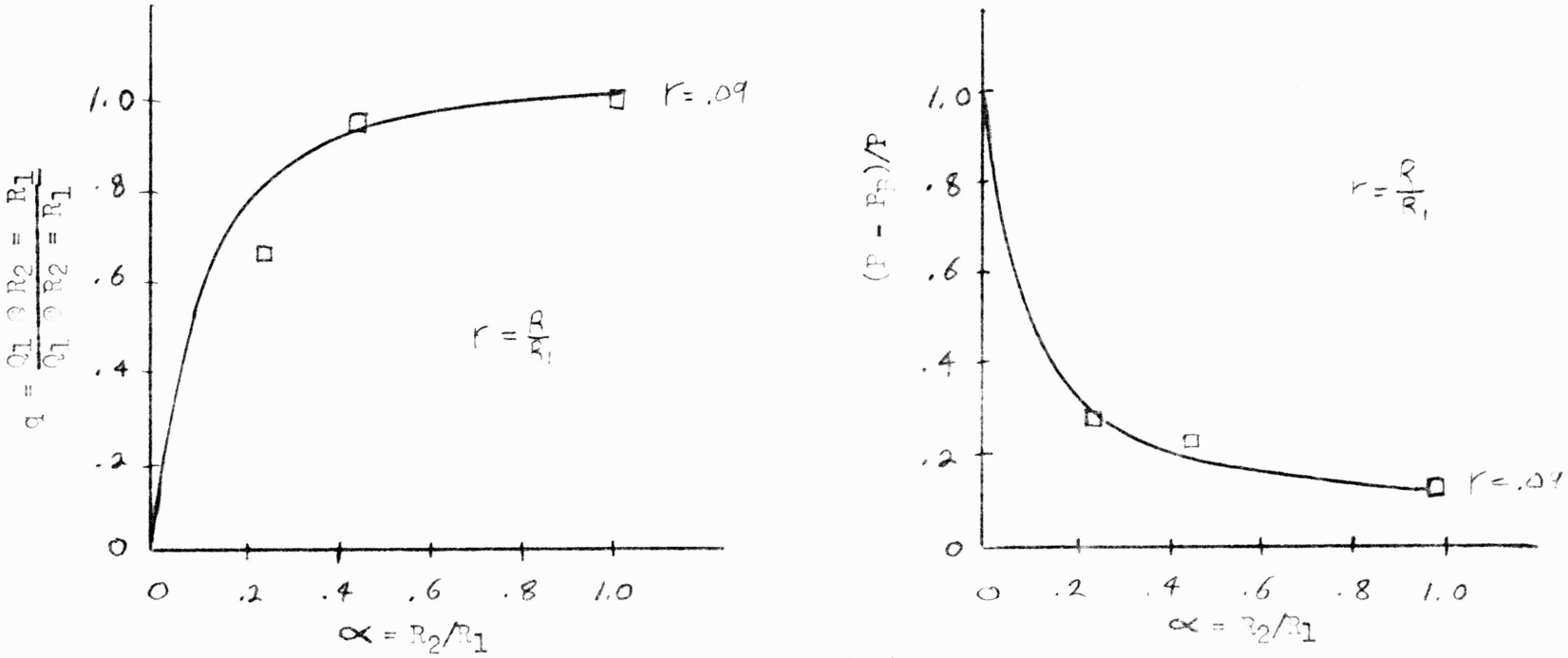
Comparison of Model Prediction (—) to Experimental Values (□) For Selected Dimensionless Parameters

Figure 9



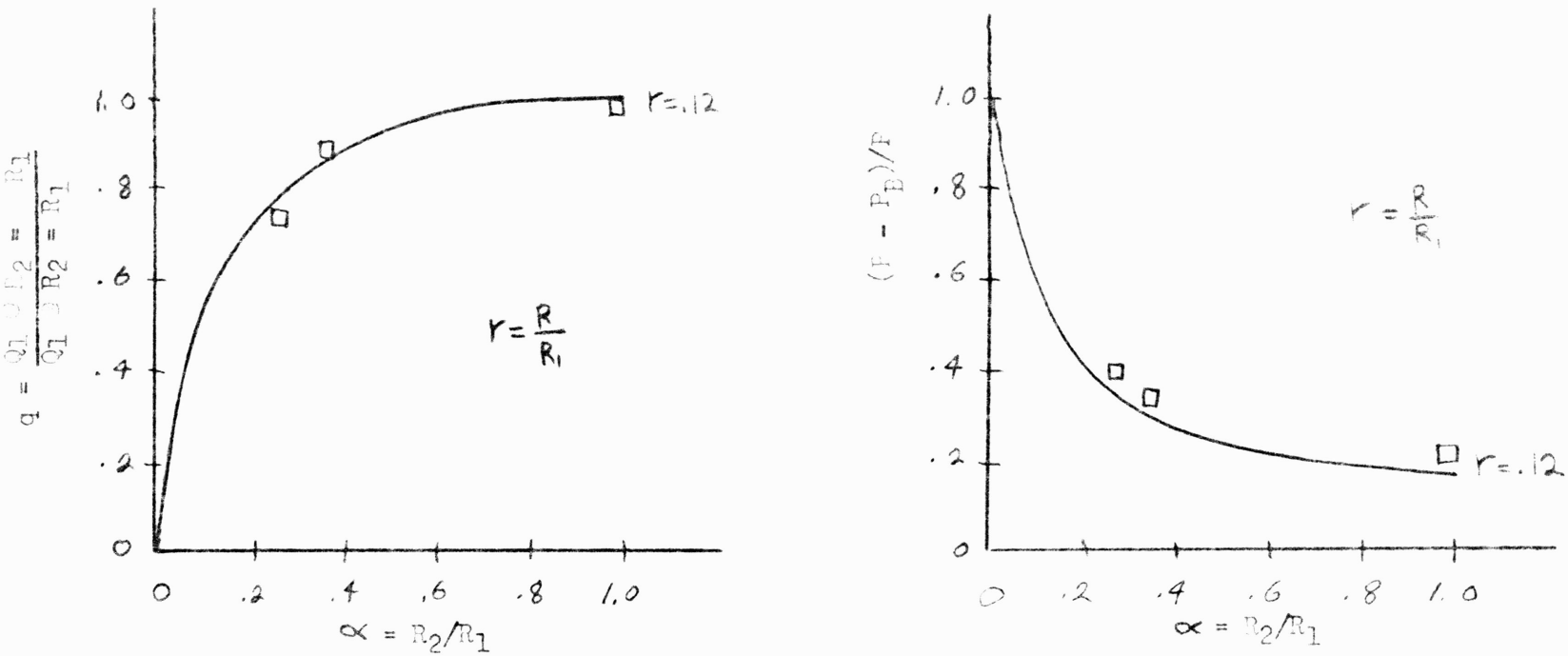
Comparison of Model Prediction (—) to Experimental Values (□) For Selected Dimensionless Parameters

Figure 10



Comparison of Model Prediction (—) to Experimental Values (□) For Selected Dimensionless Parameters

Figure 11



Comparison of Model Prediction (—) to Experimental Values (□) For Selected Dimensionless Parameters

Figure 12

CONCLUSION

The apparent contradiction between the Grimes, et al, (1972) study and the Ehrenfeld, et al., (1968) study can now be resolved. Ehrenfeld's conclusions were made for the condition of normal pressure at the bifurcation. Both the experiments and the model indicate that no steal will occur when there is no significant upstream resistance. If no upstream resistance is present then the pressure at the bifurcation will indeed be normal. Under these conditions no steal is expected and none occurred. The Grimes study stated that steal is "accentuated" by the presence of simulated occlusive disease. However, their data also showed "steal" without an aortic stenosis. This contradicts the experiments and model of this study, and no information was found in their paper to clarify this point.

The application of this study to the field of vascular surgery is important. A hypothetical example will best illustrate the importance.

> A patient has an undiagnosed aortic stenosis. He also has a clinically significant obstruction in the right external iliac artery. The right leg has thus been subject to chronic hypotension and ischemia. The normal autoregulation becomes ineffective under these conditions. Even after normal pressure is supplied, the downstream resistance will remain smaller than actually required.

This effect will last for weeks (Martin and Conrad, 1974). The left leg would not be affected by this mechanism. When the surgery to revascularize the right external iliac is performed a steal situation develops. An upstream stenosis is present. The right leg resistance will be lower than the left due to the hypotension reaction. The left leg now is "stolen" from and becomes ischemic. If no upstream stenosis was present, this would not occur. If no downstream hypotension mechanism is in play, then the two downstream resistances should be approximately equal. (The downstream mechanism would not be as important in bifurcations with normally unequal flows and resistances.) The physiology and fluid dynamics must both be considered.

One possible test that can be applied to a potential site for revascularization would be to occlude the downstream flows while observing pressure. If the pressure at the site changes with flow then it is a potential "steal" site.

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