Anatomy and Steady Flow Characteristics of the Arterial System with an Introduction to Its Pulsatile Characteristics*

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ABSTRACT

For the past thirty years Green's table in Glasser's Medical Physics [6] has been used as the common quantitative source for data on the arterial tree in mammals. The present paper updates that table by coordinating additional anatomical data from man and dog into a unified model of branching levels. The model and data appear to be consistent as to geometry, topology, and fluid resistance, as well as in number, size, and volume, of tubes. Application of such quantitative modeling to two problems is sketchily presented. The first application concerns the way the quantitative modeling information is involved in the treatment of pulsatile flow in the arterial system. The second application adduces some evidence that the wide resistive range of real arterial systems (as opposed to the average model developed) should be interpreted as mean power regulation by the system, rather than mean pressure regulation.

INTRODUCTION TO THE RELATION BETWEEN THE STEADY FLOW AND PULSATILE CHARACTERISTICS

A complete technical description of the cardiovascular system involves its *physics*: general anatomy and geometry of the arterial tree, mean pressure and flow relations, transmission line theory, pulsatile flow, flow characteristics of the microcirculation; *physiological physics*: arterial control functions of the heart, determinants of stroke volume and heart rate, characteristics of the independent circulations, chemoelectric exchanges in the capillary (i.e., exchange of metabolites and electrolytes),

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electrophysiological control; and *medical aspects*: preventive maintenance, diagnosis, management, and repair.

To provide a physical foundation for such a technical description, this report treats three topics that relate to the physics of the arterial system and touches on the actual regulated state of the terminal resistance system that is found in clinical observation. The three topics are the quantitative anatomy, geometry, and topology of the arterial system; the calculation of average peripheral resistance; and some introductory remarks to the pulsatile flow characteristics of the system.

The anatomy of the human arterial tree is pictured in atlases—typically, those of Gray, Morris, Grant, Cunningham, and Adachi. References with quantitative data are, however, quite sparse. Most sources (e.g., [27] or [2]) cite Green [6], who based his arterial model on Mall's [17] 1888 measurements of branch sizes and numbers in the mesenteric artery of a 6-kg dog. Green's table is shown as Table I.

An elementary physical model that may serve as a background for this table is an elongated central chamber, the aorta, representing a storage chamber for blood and having elastic walls that provide sufficient tension to support a high central pressure; and a peristaltic heart pump, capable of producing pulses of flow against the high central back pressure. The stroke volume and rate of this pump are jointly modified by regulatory action to provide a fairly constant mean pressure and saturated oxygen tension for the blood in the aorta. Physically, the heart pump may be regarded essentially as a constant pressure source (100 mm Hg). Its pulsatile nature (80–120 mm Hg) is analogous to a fluctuating dC voltage generator.

Starting from the single aorta, the arterial tree branches and distributes blood to the order of a billion capillary subscribers to the central service. They are served by two major system parameters, a high central pressure for mechanical purposes and a high oxygen tension for chemical purposes.

The capillary subscribers are not fixed in number. There is a dynamic twinkling, both in the local microcirculation and the systemic circulations (i.e., the capillaries open and close, in time), knowledge of which dates back at least to Krogh [15]. In a study of temperature regulation [11] a spectrum of effects was uncovered in the energetics of the whole body system, with large-amplitude metabolic cycles demonstrating periods of the order of 100 sec, 400 sec, 1500 sec, 5000 sec, and 12,000 sec. It was hypothesized and is in process of demonstration that the 100-sec cycle represents local dynamics in the microcirculation (at the level of the

TABLE I

ESTIMATE OF THE GEOMETRIC CHARACTERISTICS OF THE ARTERIAL SYSTEM IN A 13-KG DOG[a]

Name	Level	Bore (mm)	No. of tubes	Total area (cm^2)	Length (cm)	Velocity (cm/sec)	Pressure drop (mm Hg)	Cumulative drop (mm Hg)
Aorta	1	10	1	0.8	40	50	1.1	1.1
Large arteries	2	3	40	3.0	20	13.4	1.7	2.8
Main branches	3	1	600	5.0	10	8	4.6	7.4
Secondary	4	0.6	1800	5.0	4	8	4.8	12.2
Tertiary	5	0.14	76,000	11.7	1.4	3.4	13.4	25.6
Terminal arteries	6	0.05	10^6	19.6	0.1	2	4.5	30.1
Terminal branches	7	0.03	13×10^6	91	0.15	0.44	4.0	34.1
Arterioles	8	0.02	40×10^6	125	0.2	0.32	8.6	42.7
Capillaries	9	0.008	1.2×10^9	600	0.1	0.07	5.6	48.3
								60 mm[b]

[a] From Green [6], based on Mall [17].

[b] Up to the venous arterioles.

capillaries), whereas the 400-sec cycle indicates vasomotor dynamics of the blood flow among several of the systemic circulations (at the level of the arterioles). Thus, subject to these dynamics, the acting resistance bed, especially the capillary bed, is really represented by an average geometry that conforms to particular operative states of the entire animal.

A dominant anatomical impression appears to be a subdivision into levels, approximately associated with size, and a reduction in average blood velocity or proliferation in area as we descend from aorta to capillary. McDonald [19] states that the velocity in a branch is regarded as being perhaps 0.8 that of the velocity in the parent trunk. Rushmer [25] portrays a common view, that the mean velocity drops from a value in the tens of centimeters per second in the aorta to a few hundredths of a centimeter per second in the capillaries. Physically, diminution in velocity implies proliferation in the cross-sectional area. All of this Green indicates in his table.

Such gross facts—the number of tubes from anatomical casts, geometric subdivision of tubes into levels, proliferation of cross section indicated by the association of velocity with level, pressure, or pressure drop at various levels—appear to be available experimentally and are generally regarded as adequate descriptions of anatomical and hematological characteristics. They do not furnish a satisfactory description for many substantial physical questions, particularly when quantitative modeling is required. The following pulsating (aC) flow problem is sketched as an illustration. (A second problem, the actual operative system resistance, clinically found, is discussed later.)

(1) Observations indicate that the high mean pressure does not fall until anatomical subdivisions near the terminal levels in the arterial tree are reached. The measurements in Landis [16], Rappaport *et al.* [22], Wiederhielm *et al.* [28], and Intaglietta and Zweifach [12] indicate that major drops in the mean pressure only take place in tubes below the 100-μ diameter level (1000 μ = 1 mm), with significant contribution from tubes below the 30-μ level. Furthermore (as a relation of length and diameter of vessels suggests), the length associated with these last levels is quite small, a few millimeters. Since there is a pulse velocity, there are transit times in the pressure pulse getting out to these levels. However, they are not large. Remington and Wood [23] furnished direct experimental data on the pressure pulse in man from the aorta extending peripherally out more than 2 feet into the radial artery. Thus the arrival of pressure and flow are little delayed in time or magnitude of pulsation.

(2) From the aortic "windchamber," or *windkessel,* however (the foregoing are terms used by Hales and by Weber for the central storage

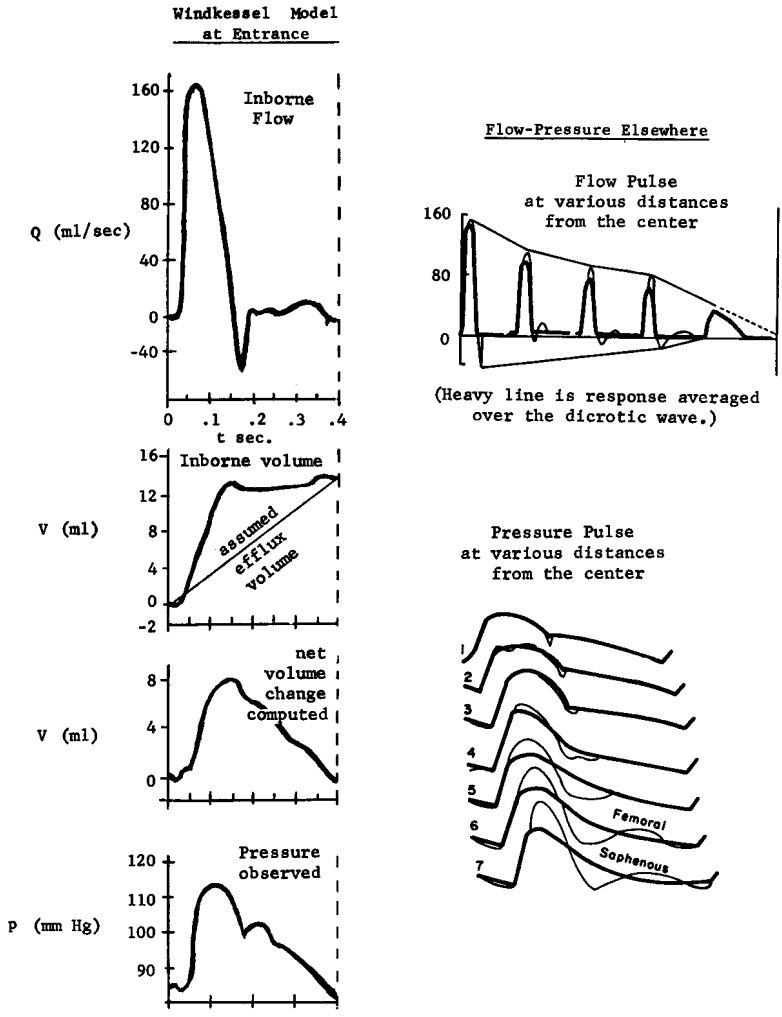

FIG. 1. Correlation between the pulse of flow and the sawtooth of pressure.

system with elastic walls and remote terminal resistances), we might expect a resonant response, namely, that the transformation into pressure of the pulselike flow from the heart would take place with a considerable oscillatory character. Thus, unless a source for sufficient damping loss

can be proposed, we would expect a highly throbbing character in the response of the aorta to the periodic pulsing. The system response is not, however, highly throbbing; it is the characteristic lub-a-dub response that is traditionally taught. An elementary possible source of damping, viscoelastic loss in the walls, is ruled out by excellent simultaneous measurements of Patel *et al.* [20], which showed the essential in-phase nature of pressure and diameter throughout the entire length of the aorta. (Earlier, Womersley [29] discussed the data of Lawton.)

(3) We can estimate the wave characteristics in the elastic wall (this has been known for a hundred years and is now commonly referred to as the Moens-Kortweg wave) from its elastic characteristics, and, thus, the resonant frequency that might be associated with this wave in the aorta. It is about 4 cps and, since the study of Hamilton and Dow [9], some evidence is considered to exist for near-resonant or standing waves in the dicrotic wave that appears as one goes downstream in the system.

(4) Since the time of Otto Frank [5], however, it appears clear that the system response (the driving point characteristic) is very nearly resistance-capacitative in nature. The source of this resistance must be accounted for in order to trust any further estimate of line impedances. In Fig. 1 it can be noted that the flow pulse from the heart, if summed in time, charges up the central aortic volume, which decays as a near-constant efflux through the many subscriber arterioles. The resultant volume change into the aorta forms a triangular wave. The wall elasticity transforms the net input volume change into pressure. Further, an earlier report [10] showed that the characteristic response of a pulse of flow transforming into a triangular sawtooth of pressure held at every point in the arterial tree for which experimental data could be found. This is also illustrated in Fig. 1.

(5) At most, the pulsatile character is a mild oscillation around the flow pulse and pressure sawtooth at values near the dicrotic wave frequency. A dynamic physical analysis of the pulse wave, accounting for this response characteristic of nonresonance without significant loss in mean pressure, was presented in [10]. (An earlier study by Karreman [14] illustrated, with a few lumped elements, the type of network that might lead to the dicrotic wave response.) Its general features are of interest. To simplify the analysis, while maintaining the fundamental characteristic of no damping loss of the pulse with no resonant throbbing, a constant cross-sectional area from level to level was assumed, except at the capillary level. (A high mean velocity of 50 cm/sec was assumed throughout the

system, dropping precipitously to a mean of 0.07 cm/sec in capillaries.) This is crudely consistent with data in the physiology texts of Rushmer [25], Best and Taylor [3], and Guyton [8]. The levels in the system, which may be viewed as a sequence of diminishing steps in diameter, were replaced by a continuous equivalent branching. This represented the system as an ever-branching tapered tube model. The wave that spreads out and reflects through such a system can then be treated.

Fortunately, this model in its mathematical details seemed to have the right characteristics and the apparent discrepancies fell into place. There is little or no damping throughout most of the system. There is little or no drop in mean pressure. There is the appearance of a dicrotic wave (not the incisura in the input pressure, which has other causality). There is rational damping of the higher-frequency components (of which the incisura is an example), yet there is no resonant throbbing. The dicrotic wave is formed from out-of-phase lags of the harmonic components higher than the fundamental. The basic ingredient from which these properties emerge, put somewhat paradoxically, is that although there is little damping in the outgoing or incoming wave, there is no reinforcing amplitude left in the returning wave. It has been damped at the remote terminal resistance end. The description is similar to Rayleigh's explanation of acoustic damping near a porous wall and, in fact, the model may be referred to as a tapered porous wall model of the arterial system.

In subsequent discussion with research workers, various modeling questions were raised involving the physics, physiology, and pathology of the system. To insure that the background for these questions was adequate, it was necessary to clarify various details about the real system. For example, a question of the choice of mean entrance velocity and its subsequent near constancy indicated that projecting a model of the hemodynamic events required that it be as faithful as possible to all known anatomical details and based on the best possible model of anatomy and peripheral dc resistance. Only then could any pulsating or varying results be seriously considered. This report is designed to provide a representative anatomical description.

THE ANATOMY AND STEADY FLOW CHARACTERISTICS

From literature research it was found that apparently only Green [1] had attempted to assemble an anatomical description into a coordinated whole, that his data simply scaled Mall's [17] 1888 measurements on one

mesentric artery, and that very few other investigators had ever been similarly involved. Based on considerable search, data have been assembled for a more certain anatomical summary from the work of Patel *et al.* [21], Mall (his post-1888 studies [18]), and Suwa [26].

Patel *et al.* reported on geometric data in the dog aorta and large arteries. Most significantly, the data were derived from the averages of many dogs, both living and dead, corrected to the living state, whereas data in atlases are generally based on postmortem sources. Averaged for 23-kg dogs, they indicated an aorta entrance area of 3.2 cm^2 and about 29 "major" arteries whose total entrance area is about 3.4 cm^2. It may be surmised that the cross-sectional area of the cardiovascular system does not change much from the aorta to the major arteries. The same conclusion was stressed by Mall [18, p. 238]. "Thoma made many measurements of arteries and their branches and tabulated Bencke's measurements of the aorta with its branches. These measurements show that the area of all of the branches of the aorta equals about the area of the ascending aorta, being a little less before the thirtieth year of age and a little greater thereafter. . . ."

From Altman [1], the cardiac output for a 23-kg dog may be estimated as nearly 50 ml/sec at rest (or 200 ml/sec at high activity). The mean velocity in the aorta is thus about 15–20 cm/sec at rest (or about 60–70 cm/sec at high activity). This also seems to be the correct magnitude of mean velocity in the ascending aorta in other species, including humans. (For example, in the human at rest, a cardiac output of 120 ml/sec with an internal diameter of 2.7 cm for the aorta gives about 20 cm/sec.) The present geometric concern is with cross-sectional area, not velocity, which depends on activity level. However, it is necessary to point out the need for consistency.

Thus it appears that the first two lines of Green's table are inconsistent regarding velocity and area. The burden exists to propose a more nearly correct table. The source data assembled in Fig. 2 are based on: (1) The Patel data, aorta and main artery sizes in living 23-kg dogs; (2) the post-1888 Mall data, based on cast counts from five circulations in a 6-kg dog, intestine, stomach, adrenal, spleen, liver; (3) the 1963 Suwa data, based on cast counts from seven circulations in humans, kidney, intestine, femoral, pancreas, heart, cortex, basal ganglia. (It is noteworthy that Suwa's geometric studies stem from his interests as a pathologist of the cardiovascular system.)

Figure 2 exhibits level area versus level diameter range. From Patel's data on the aorta, the values chosen were a maximum entrance diameter of 20 mm, a minimum exit diameter of 7 mm, and an entrance area of 3.2 cm^2. For major arteries, a maximum diameter of 11 mm was estimated, a probable minimum exit of 2 mm, and a similar entrance area of 3.4 cm^2.

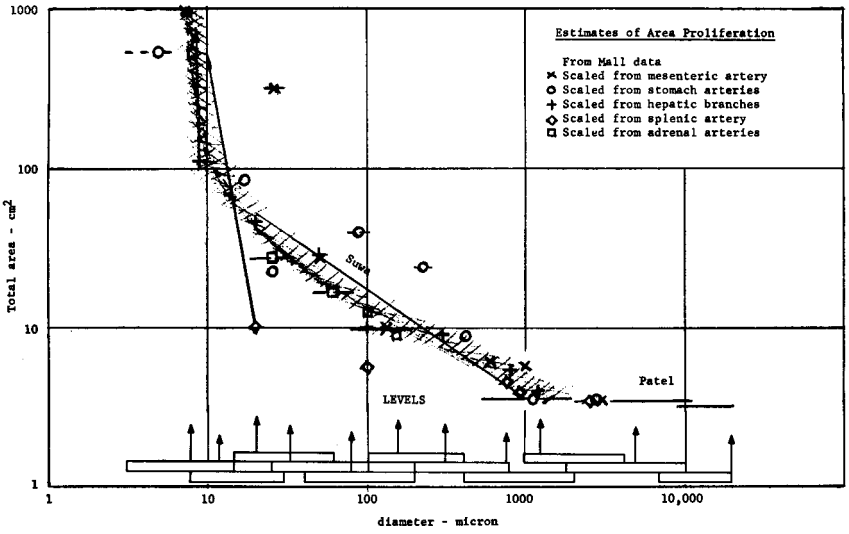

FIG. 2. Estimate of cross-sectional area, levels, and mean diameter for a 23-kg dog's arterial tree, from Patel [20], Mall [18], and Suwa [26].

Mall's data, taken on a 6-kg dog, can be scaled up to Patel's 23-kg dog data by increasing cross-sectional area by a factor of 4. This may be specifically justified by comparing cross-sectional area data on those arteries that are reported in common.

Unfortunately, although Mall's data provide the most extensive arterial count found, they only covered 30% of the high-pressure arterial circulation. In utilizing his data, it was preferred to regard each circulation (including the one Green used) as an independent fractional estimate of the entire circulation. This would be valid if mean velocity and diameter were uniformly associated throughout the system. Thus Mall's data are scaled, for each major artery that he lists, to fit the Patel data. The scatter is large, but we can reasonably infer that the cross-sectional area does not change much until arterial diameters of the order of $\frac{1}{2}$ mm are reached; then an approximately uniform increase in area per level for

arterial sizes down to about 20–30 μ occurs, after which a large increase in area down to capillary sizes of the order of 8 μ takes place.

Suwa's results can be summarized algebraically. Let d_0 = an entrance diameter and $d_{1,2,\ldots}$ = branching diameters. If the area after every branching were constant,

$$d_0^2 = d_1^2 + d_2^2 + \cdots .$$

Instead Suwa showed that at every branching

$$d_0^{2.7} = d_1^{2.7} + d_2^{2.7} + \cdots .$$

This result was also obtained earlier by Groat [7]. We can then estimate that the area at every level is nearly proportional to $1/d^{0.7}$. This line is plotted in Fig. 2; the approximate agreement may be noted. Thus, Patel's, Mall's, and Suwa's data are consistent and they now permit the necessary amendment of Green's table. The Suwa 2.7 power law derived from humans in the 25- to 1000-μ diameter range seems to agree with data derived by Mall from a dog. For larger size tubes, the area does not change much. For the one or two levels below 25 μ, the area proliferates more rapidly. Thus we find three anatomical regions.

A substantive question is, What constitutes a level? In the present view, Suwa's data destroy the idea that levels intrinsically exist as specifically sized entities, since he showed an essentially continuous distribution of diameters. Although the basis for anatomical levels of subdivision in the tree has not been reviewed here, clearly they have been associated with size, distinctness, uniqueness, and function. The aorta and major arterial branches form two distinct levels. The system ends in capillaries that are said to be fairly uniform in diameter, near 10 μ in mammals. Yet we may note that Mall lists capillaries down to 3 μ, as does Wiedeman [27], and in the microcirculation literature it is common to find descriptions of capillaries that include diameters as great as 20 μ.

These three levels appear certain. Obviously, the anatomist successfully follows secondary branches from the major arterial branches. However, as one aorta becomes twenty main branches, with many variants even in a given species, and becomes hundreds of branches in the next few levels, the difficulty increases. At the microcirculation end, the distinction among arterioles, terminal arterioles, and small arteries also becomes difficult. Thus five levels of division appear certain, and as many as thirteen levels have been discussed in the literature.

Yet Suwa shows, in plots of segment length versus diameter in different circulations, that the diameter range is essentially continuously distributed over his experimental range of 20–4000 μ, and Patel's data covering diameters from 2 to 20 mm are also consistent with an essentially continuous diameter variable. What, then, is a level?

It is the present opinion that the concept of level is an intrinsic concomitant of the act of branching. A level may be associated with a tube as long as we can retain the view of a "main" tube. As smaller tubes are shed from view, we can continue to pursue the main tube. Ultimately, however, there arises a bifurcation into nearly equal tubes. Then the concept of that level ends. What seems really intrinsic to the concept of level is the number of significant branchings that take place before an equal bifurcation occurs.

On this basis we can assign some kind of estimate to the number of levels. Taking some common numbers regarding capillaries, say one capillary segment of about 100-μ length associated with a region of about 30-μ diameter, this represents about 3×10^{11} capillary segments in a 23-kg dog. (That is, we compute 3×10^{11} segments of tissue 30 μ in diameter and 100 μ long in a 23-kg dog if near water density is assumed. One way to assess whether the capillary number is reasonable is to compute the internal volume as 8-μ capillaries. A common view is that in complete dilation—extreme shock—the open capillaries will hold substantially all the blood. According to Altman [1], who presents an average of about 95 ml/kg for dogs, a 23-kg dog has about 2200 ml of blood. Multiplying 3×10^{11} capillaries of 8-μ diameter and 100-μ length gives a volume of about 1600 ml.)

$$N^m = 3 \times 10^{11}$$

where N = number of branchings per level and m = number of levels. Solution of the foregoing equation leads to the estimate in column A of Table II.

The results in Table II overestimate the number of levels because the terminal arteriolar resistor serves more than one capillary. We can estimate a lesser number of first-branch capillaries from the cardiac output and the nominal velocity. Assuming total flows in the range 50–200 ml/sec (resting to active), average capillary velocity of about 0.07 cm/sec, and an average capillary diameter of 8 μ, the number of series-parallel capillary systems that are served range from about 1.4 to 5.6×10^9 (i.e., numbers agreeing with Green's estimate). These differ from the geometric number

TABLE II

Branchings per level	Estimated number of levels		
	A	B	C
2	38	32	30
4	19	16	15
6	15	12½	12
8	13	11	10
10	11½	10	9
15	10	8½	8
20	9	7½	7
30	8	6½	6
40	7	6	6

by about a factor of 60–240. The discrepancy is accounted for by the fact that on the average not all of the capillaries are open at any one time and, in fact, only a small percentage are open. (An estimate of the number open varies in different circulations from about a factor of 2, in rest to maximum activity in muscles, to about a factor of 200 in the skin circulation from highly vasoconstricted to highly vasodilated. The present average estimate is valid to within an order of magnitude.) Then, approximately,

$$N^m = 1.4 - 5.6 \times 10^9.$$

These lead to the estimates in columns C and B in Table II.

Suwa's length-to-diameter data provided assurance that the number of branchings is certainly at least five (for the same diameter, he shows a scatter of length to diameter on the average of about 5 to 1). More to the point, he showed that eight branchings are common and twenty to thirty are not excluded. Thus, the number of levels is probably in the 7–12 range. (Therefore, in addition to any anatomical reasons, geometry also helps create a "reason" for finding anatomical levels.)

We may choose, arbitrarily at this time, either a branching or a level number. At present it appears that the greatest coherence lies near 10–11 levels, in which the diameter range associated with each level is divided in approximately equal ratios. From Patel's data it is estimated that a diameter range of about 2 or 3 to 1 is appropriate to a level. (This type of subdivision has also quite plausibly been derived from Mall's data.) With these data, we may now attempt to construct a model of the total resistance in the system.

For each level, Poiseuille flow gives

$$\Delta p_i = \frac{128}{\pi}\mu q \frac{l}{d^4}\left[\left(1+\frac{6}{d}\right)^2\right]^{-1}.$$

The factor $1/(1+6/d)^2$, the Fahraeus-Lindqvist correction, is a viscosity correction for red blood cell flow in small tubes (d in microns). References and discussion justifying this correction are given in [6], [26], and [24].

$$q = \frac{Q}{N}; \qquad \Delta p_i = 32\mu \frac{4Q}{\pi N d^2}\frac{l}{d^2}\left[\left(1+\frac{6}{d}\right)^2\right]^{-1};$$

$$A = \frac{\pi}{4}Nd^2; \qquad \Delta p_i = 32\mu Q \frac{l}{Ad^2}\left[\left(1+\frac{6}{d}\right)^2\right]^{-1}.$$

Summed over levels,

$$\Delta p = 32\mu Q\left[\frac{l_1}{A_1 d_1^2}\left[\left(1+\frac{6}{d_1}\right)^2\right]^{-1} + \frac{l_2}{A_2 d_2^2}\left[\left(1+\frac{6}{d_2}\right)^2\right]^{-1}\cdots\right],$$

$$\frac{\Delta p}{Q} = R = 32\mu\left[\frac{l_1}{A_1 d_1^2}\left[\left(1+\frac{6}{d_1}\right)^2\right]^{-1}\cdots\right],$$

$$\frac{R}{32\mu} = \frac{l_1}{A_1 d_1^2}\left[\left(1+\frac{6}{d_1}\right)^2\right]^{-1}+\cdots,$$

where Δp = pressure drop, q = flow in a single tube, Q = total flow, N = number of parallel tubes in a level, l = tube length at a level, d = tube diameter at a level, μ = viscosity, R = resistance ($= \Delta p/Q$).

Suwa also indicated that for any level in the 20- to 4000-μ range the ratio l/d tends to be constant. Actually, he showed that an arterial segment will likely branch within three diameters and probably will have had appreciable side branchings within fifteen diameters. Patel's data on the aorta indicated a 20-to-1 ratio of length to entrance diameter. From this, the lack of much tapering in small tube sizes below the 100-μ level (as seen in Bloch's data [4]), and the extremes of Suwa's data, we may estimate that a level may extend to the order of 20–30 diameters before a nearly equal bifurcation. This is consistent with the estimate, concerning number of branchings per level, that about 7–10 branchings would indicate about 9–12 levels, and it checks reasonably well with the findings by Mall of 10–11 levels. Thus $l/d = 25 \pm 5$ is not an unreasonable guess.

Then a resistance function may be constructed solely from geometric factors as follows.

$$\frac{R}{32\,\mu\,(l/d)} = \left[A_1 d_1\left(1+\frac{6}{d_1}\right)^2\right]^{-1} + \left[A_2 d_2\left(1+\frac{6}{d_2}\right)^2\right]^{-1} + \cdots .$$

If we assume, for the 23-kg dog, $\Delta p = 110$ mm Hg ($= 0.15 \times 10^6$ dynes/cm^2), $Q = 50$ ml/sec (at rest), $l/d = 25$, $\mu = 0.035$ poise (gm/cm sec)

$$105/\text{ml} = \left[A_1 d_1\left(1+\frac{6}{d_1}\right)^2\right]^{-1} + \left[A_2 d_2\left(1+\frac{6}{d_1}\right)^2\right]^{-1} + \cdots .$$

In Table III, derived from Fig. 2, an estimate is made of the $1/Ad(1 + 6/d)^2$ sums of 109/ml, which is thus of the correct magnitude.

In Table III: (a) the number of levels, 11, was assigned a priori.

(b) The entrance and exit diameter and area for the aorta, level 1, were taken from [21], as were the entrance diameter range and total area for level 2.

(c) The average entrance area for all levels, 2 and beyond, were estimated from the prior exit area by the Groat-Suwa bifurcation rule $d_0^n = 2d_1^n$, from which $A_1 = 2^{-2/n}A_2$. If $n = 2$, $A_1 = A_2/2$; $n = 2.7$, $A_1 = A_2/1.67$; $n =$ large, $A_1 = A_2$. The average entrance diameter was then computed from the average entrance area.

(d) The total diameter range in Fig. 2 was divided into 11 overlapping ranges, taking into account the known ranges of levels 1 and 2 from [21] and the likely range of the last capillary level (i.e., diameter ranges of about 4 to 1 were selected). Each diameter range was assumed to provide both an estimate for the entrance diameter range and the lower bound for the exit diameter. The exit area was computed from this diameter.

(e) The total area was then estimated from Fig. 2, essentially for the value of average entrance diameter. (From Suwa, we estimate $A_0 \alpha 1/D_0^{0.7}$).

(f) For the viscosity correction $6/D_0$ in the resistance column, D_0 is in microns. This column represents the contributions at each level to the resistance function $R/32\,\mu(l/d)$; $\mu = 0.035$ poise, $l/d = 25$.

(g) The capillary area is simply a fictitious value.

(h) Meaningful completion of the table with some consistency for capillaries would require the use of the experimental data for small-sized tubes, which is limited. At present it can be based on consistent correlation among data on the frog and mammals in [12, 16, 22, 28]. (The frog data are not pertinent; they are simply given as a parallel guide to qualify the mammalian data.)

FROG MESENTERY[a]

Reference		Capillary		
		Arterial	Middle	Venous
[16] (Summary table)				
Pressure		11		9
[28] Size	260[b] 126 21	26 (ent.)	24	22 (ex.)
Pressure	30 22 14	14	9	7
[12] Size	17[b] (ent.)	17 ($\frac{1}{3}$ way down)	19 ($\frac{2}{3}$ way down)	23 (ex.)
Pressure	18	15	13	6

[a] Summary: Central pressure 30 mm Hg; cap. ent. 16 ± 2, ex. 7 ± 2, drop 9 ± 3.
[b] Tube size is expressed in microns, mean pressure in millimeters of Hg.

MAMMAL[a]

Reference	Modal value in capillaries		Average limits for deviation around mode	
[16] (Summary table, human)	13		8–22	
[16] (Rat, guinea pig, human, summary)	27[b]		12½[c]	
[12]	16 (ent.)	7[b]	7[c]	13 (ex.)
Rat	24	24	23	20

[a] Summary: Central pressure 70–100 mm Hg; cap. ent. 24 ± 4, ex. 16 ± 4, drop 8 ± 4. Thus, an apt estimate (quiescent) is 8 mm Hg drop across capillaries and 16 mm Hg pressure at the capillary exit into the venous side.
[b] Arterial side.
[c] Venous side.

(i) The estimate may be compared with the following standard: Gregg, in [3], p. 274, states "The carotid blood pressure (mean) of the unanesthetized, basal dog approximates 110 mm Hg." Altman [1] gives the basal flow as about 50 ml/sec. The computed resistance function was 105 ml/sec.

(j) A more consistent view of the capillaries may be had by assuming approximate parameters, such as $Q = 50$ ml/sec, $V = 0.07$ cm/sec, $l/d =$

100/8, $d = 0.0008$ cm, $\Delta p = 8$ mm Hg, N = number of capillary channels open, m = number of layers of capillary resistances in the series-parallel bed, then,

$$A = \frac{Q}{V} = 710\,\text{cm}^2, \qquad N = \frac{4A}{\pi d^2} = \frac{710}{0.785 \times 0.0008^2} = 1.4 \times 10^9,$$

$$m = \frac{\Delta p}{32\,\mu V}\frac{d}{1}d\left[1 + \frac{6}{d}\right]^2 = \frac{(8/750) \times 10^6 \times 0.0008 \times 8 \times 1.75^2}{32 \times 0.035 \times 0.07 \times 100} = 27,$$

$$mN = 3.7 \times 10^{10}, \qquad \text{Total (est.)} = 33 \times 10^{10}.$$

Thus 10% (i.e., 3.7/33) of the capillaries are normally open (accuracy of calculation is indeterminate).

(k) A final consistency check is from the probable volumes in the arterial tree. From [1], the blood volume for dogs averages about 95 ml/kg or 2200 ml for a 23-kg dog. From Gregg [3], pp. 152–153 the aorta volume is about 2% of the blood volume (50 ml), the arterial volume 8% (175 ml), the arterioles 1% (20 ml), and the capillaries (quiescent operation) about 5% (100 ml), giving a total of 16% (350 ml).

The volume may be computed as follows: In the $n = 2$ regime, the volume is pyramidal, $V = \frac{1}{3}(A_{\text{ent}} + A_{\text{ex}})L = \frac{1}{3}(L/D)D_{\text{ent}}(A_{\text{ent}} + A_{\text{ex}}) = 8.3D_{\text{ent}}(A_{\text{ent}} + A_{\text{ex}}) \approx 9.2D_{\text{ent}}A_{\text{ent}}$ (if exit not certain). In the $n = 2.7$ regime (really n approximately 3 regime), there is a near tendency to preserve volume from level to level. Thus $V = A_{\text{ent}}L = 25D_{\text{ent}}A_{\text{ent}}$.

For the capillaries, the volume $= (mN)(\pi/4)D^2L$. Based on the $mN = 3.7 \times 10^{10}$ segments, 100 μ long, 8 μ diameter, the volume = 185 ml.

If the long logical process by which the estimate is made is followed, we can only marvel at the nominal validity of the estimate. All that is proposed is that an estimate or adjustment of peripheral resistance can be made with the available data on a purely geometric-anatomical basis.

In summary, the effective cross-sectional area in the tree has not changed much out to internal diameters of the order of 1 mm. In the size range of from human to medium-sized dog, this may be out as far as the first four levels in the tree, namely, the aorta, main arteries, and main and secondary branches.

The approximate "length" of every level is about 25 times the diameter. Thus there has been little change in cross-sectional area out to within a

TABLE III

ESTIMATED ARTERIAL TREE GEOMETRY AND RESISTANCE FOR A 23-KG DOG[a]

Level[(a)]	Diameter (cm)			Area (cm^2)			No. of tubes	Groat-Suwa exp. (n)[(c)]	$1/A_0\overline{D}_0$ $(1 + 6/D_0)^2$ (1/ml)[(f)]	Level volume[(k)] (cm^3)
	Ent. (D_0)	Exit (D_1)	Av. ent. ($\overline{D}_0$)	Total (A_0)	Av. ent. (A_1)	Exit (A_2)				
1	2.0[(b)]	0.7[(b)]	2.0	3.2	3.2[(b)]	0.35[(b)]	1	2	0.2	47
2	1.0–0.18[(b)]	0.18[(d)]	0.47[(c)]	3.4[(b)]	0.17[(c)]	0.026[(d)]	20	2	0.6	31
3	0.4–0.1[(d)]	0.1	0.13	3.4[(e)]	0.013	0.008	260	2	2.3	34
4	0.2–0.04	0.04	0.08	4.0	0.005	1.3×10^{-3}	800	2.7	3.1	20
5	0.08–0.02	0.02	0.031	5	7.6×10^{-4}	3.0×10^{-4}	7×10^3	2.7	6.4	10
6	0.04–0.01	0.01	0.016	6	1.9×10^{-4}	8.0×10^{-5}	3×10^4	2.7	10.1	6
7	0.02–0.004	0.004	0.008	10	4.8×10^{-5}	1.3×10^{-5}	2×10^5	2.7	11.8	5
8	0.01–0.0025	0.0025	0.0032	16	7.6×10^{-6}	5.0×10^{-6}	2×10^6	2.7	17.3	4
9	0.006–0.0015	0.0015	0.0020	25	3.0×10^{-6}	1.8×10^{-6}	8×10^6	2.7	16.5	4
10	0.003–0.0008	0.0008	0.0012	35	1.1×10^{-6}	5.0×10^{-7}	3×10^7	large	16.5	3
11[(j)]	0.0015–0.0004	0.0008	0.0008	80[(g)]	5.0×10^{-7}	5.0×10^{-7}	2×10^8		8.0[(h)]	— 185
									93	165 350
									16[(h)]	
									109 ml^{-1} [(i)]	

[a] Superscript parenthetical letters generally refer to the entire column following; each letter indicates the relevant item that appears earlier in the text.

few centimeters of the arterial termini. Furthermore, there is little mean pressure change out to that level.

The highest resistance region is the last, less than 30μ in diameter, approximately 0.6 mm in length.

The concept of an anatomical level has a strong geometric foundation, based on the average number of branchings before an equal bifurcation takes place and on the ratio of length to diameter before branching. All of these concepts are self-consistent with the number of subscriber elements and internal volumes.

There is thus reason to believe that the anatomy, geometry, topology, and flow resistance of the arterial tree can be validly modeled, albeit approximately, and that no single level dominates the resistance picture. To provide some idea of how this summary paper may be used, two applications will be briefly commented upon.

SOME FURTHER COMMENTS ON THE PULSATILE (AC) FLOW

This more precise modeling of the dc characteristics permits a review of the previous modeling of pulsatile events in the tree [10]. It appears that the model is reasonably valid for the following reasons.

Since the cross-sectional area does not change until well out near the periphery of the system, up to that point the system may be viewed as a tapered porous tube model (i.e., through any running length, there is an approximately uniform number of branchings that can be replaced by an effective tapering area with an effective equivalent branching area that distributes into side tubes). This region may be regarded as the long transmission line or wave propagation zone, which, by hydrodynamic reasoning, is fairly undamped at the higher frequencies.

The region in which the area changes markedly is no longer concerned with such traveling waves. It was treated [10] as if it consisted only of "terminal resistors" of the order of 30μ in diameter. It now appears that there really is a modest terminal impedance region below a diameter of a few hundred microns. That is, in addition to flow resistance, there remains some wall elasticity and some storage capacity. This will make some moderate changes in the frequency response of the long line, but not much. Thus the earlier result presented much of the dominant character of the transmission line. These terminal impedances are where the resonant energy is soaked up in an out-of-phase return of the higher-frequency components.

SOME COMMENTS ON PERIPHERAL RESISTANCE FOUND CLINICALLY

Whereas a table such as Table III may give the impression that resistance is determined by a fixed geometry for the system, experimental data show that there is considerable variability in both the pressure and flow that go to make up the resistance in both clinically normal and abnormal individuals. This raises significant questions about the regulation of pressure and flow in the arterial system.

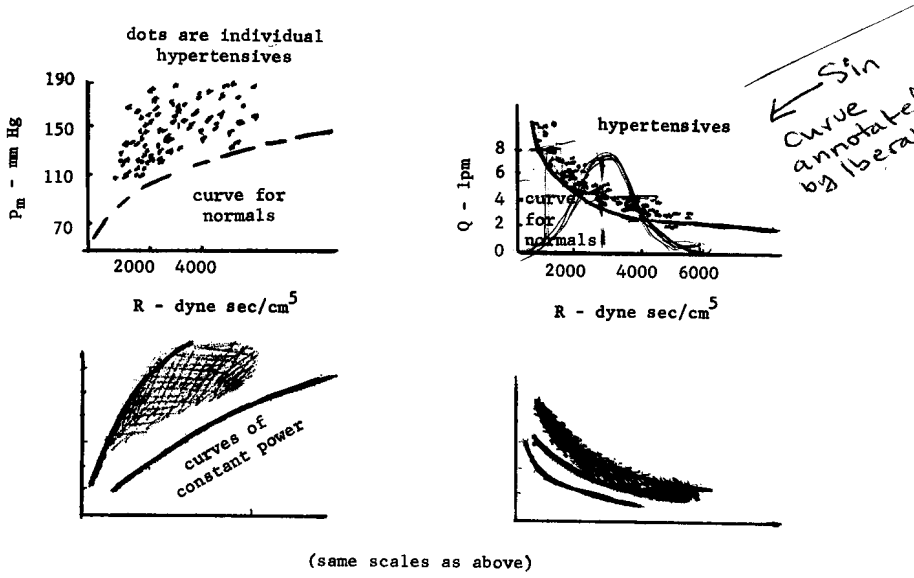

FIG. 3. Correlation of mean pressure and flow in normal and hypertensive (quiescent) humans by power levels (data from [13]).

Two sources of data have been examined. A Russian paper ([13], p. 212) states, "It is now the accepted belief ... that the main hydrodynamic mechanism causing a rise in arterial pressure in hypertension is increased resistance to the blood flow in the arterioles, resulting from spasm of the latter. An indication of this mechanism in hypertension is the increased peripheral resistance accompanying the disease". Data on 111 hypertensive patients (see Fig. 3) are presented as mean pressure versus resistance and mean flow versus resistance, as well as a curve based on normals. They state that normal healthy patients show a mean pressure that varies with the one-fourth root of resistance or flow that varies inversely as the three-fourths power of resistance.

Such a result for normal patients was somewhat surprising, although not based on any real knowledge about clinical hypertension. A casual impression, confirmed by inquiry among some cardiologists and cardiovascular physiologists, was that the basis for identifying "hypertension" was a loss in pressure regulation to values higher than normal. Yet these data indicated (as do the studies of Master, in Rushmer [25]) normal individuals with mean pressure in a range 80–150 mm Hg, which is a wide range compared to an elementary idea of a near-constant mean pressure. Also, invoking a law as exotic as the fourth root of resistance is hardly indicative of understanding of a process.

It is clear that hypertension was indicated as pressure above this normal level, but equally surprising was the wide resistance range covered by normal and still-functioning systems, "from 900 to 7900 dyne sec/cm^5, averaging 2745 ± 1220.." ([25], p. 212). After considering these data carefully, we formulated a simple orienting hypothesis.

Power from its electrical analogue is proportional in the flow case to Pm^2/R, or Q^2R.

Consider plots of lines of constant power consumption shown in the lower graphs of Fig. 3. What emerges is that the normal curve is probably associated with a given power level requirement from the heart, rather than with pressure. A hypertensive must work at a higher basal power rate. From these data, we would infer that hypertension is not determined by pressure but by power. In fact, in an extremely hypertensive patient, the power level appears to be about 2–3 times that in the normal one, and apparently about a 20% higher power level than normal is a discriminable level of hypertension.

Thus, it appears that the characteristic "homeostatic" regulation of the normal system (at rest) is a near-constant power level. These systems that are imperfectly regulated deviated from this power level. However, the actual functioning resistance range among humans may be quite large, as large as 1000–5000 dyne sec/cm^5 (i.e., in the units of the table, 30–170 ml^{-1}).

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