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PRESSURE AND SHEAR IN PRESSURE SORES

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Abstract

Pressure and shear are examined in terms of relative significance as causative factors in pressure sore occurrence. Experimental results concerning shear effects are presented. In the case of normal subjects, shear is roughly half as effective as pressure in driving that skin over the thenar eminence toward occlusion. In sitting on a hard, slick seat, paraplegic and geriatric and ill subjects develop roughly three times the median shear load experienced by normal subjects in skin lateral to the ischial tuberosities. The normal (control) group reflects roughly three times the median pulsatile blood volume flow rate developed by paraplegic and geriatric and ill subjects. Thus an association exists between gross state of health, shear, buttocks cutaneous blood flow and proclivity towards pressure sores. Whether this association is causal in nature is unknown.

Introduction

While the precise etiology of pressure sores remains unclear, the one common denominator among all the competing hypotheses is the necessity for excessive, sustained external load in initiating a series of physiological events ultimately leading to necrosis. The prime importance of sustained load is accepted by all workers in this field, for reasons best summed by Guttmann (1), "where there is no pressure, there is no sore".

At least four causal hypotheses are current. These are the ischemic (2), neurotrophic (3), metabolic (4) and the enzyme concepts (5). As our work does not deal with fundamental etiology, we shall not develop these possibilities, except to note that they are not mutually exclusive - some or all may conceivably be acting in a given incident of trauma. Numerous secondary factors pertinent to trauma are believed to exist, including age (6) other infections (7) and local moisture (8) among others. However those workers attributing a major role to secondary factors do not question the primacy of load effects.

There are but two forms of external loading - normal (pressure) and tangential (shear). Pressure effects have been investigated by many workers. Wheelchair cushions and other seat devices have been studied by Souther et al (9), Cochran and Slater (10), Houle (11) and others. Each of these workers employs pressure readings as an index to load and ignores shear.

The emphases upon pressure, as compared to shear, is pervasive. This emphasis does not reflect a lack of interest; for example, in view of Guttmann (1): "As to direction, one must distinguish between purely vertical pressure and shear stress, of which the effect of shear stress is much more disastrous for it cuts off larger areas from their vascular supply". In the opinion of Reichel (12) raising the head of a hospital bed is capable of producing a sufficient shear force over the sacral area to deprive large areas of tissue of a sufficient blood supply. Roaf (13) indicates that "avoidance of shear force is as important as avoidance of direct pressure".

Thus, workers in this area postulate shear force to play a highly significant role in the causation of pressure sores. However, experimental verification of shear significance is lacking.

In this work we are concerned with assessing the relative significance of shear and pressure in pressure sore causation. When employing experimental techniques in connection with human subjects, certain limitations are inherent. For obvious reasons, the experimental procedure must not produce pressure sores. It follows that some index, preferably quantitative, is to be employed as a substitute standard for measuring incipient trauma. One such standard is the load required to produce occlusion in the skin capillaries; the pressure value required for stasis, approximately 32 mm Hg in the normal fingernail bed (14), has been accepted as a threshold pressure value by many workers. Yet other views exist (15) in which critical threshold conditions are believed to vary as a function of the local anatomy.

In this work, we shall consider cutaneous arteriolar occlusion the critical condition which, if sustained, will lead in time to the development of a pressure sore. The means of approach to blood flow occlusion is our standard; those loading conditions capable of producing near occlusion are viewed as correspondingly more severe than those loading conditions offering little blood flow restraint. Within this context both pressure and shear are viewed as forms of potentially obstructive input and the resulting blood flow, the output.

Materials and Methods

A sensor head was prepared (see Fig 1) containing 4 sensors (2 pressure, 1 shear, 1 blood flow plethysmograph) within a 2.5 cm diameter circle and mounted flush with respect to a large aluminum block. The pressure and shear sensors, detailed elsewhere (16) consist of cantilever steel beams whose displacements under load are sensed by strain gauges. Each beam is extremely stiff so that the actual deflection is not apparent to the user, to whom the sensors appear as flush buttons. The blood flow photoplethysmograph is a commercial device suited for the measurement of cutaneous pulsatile blood flow. A miniature light source is positioned to reflect light from the skin onto an adjacent solid state photoreceiver. Light absorbed by the arterial pulsating blood reduces the amount detected at the photocell. Noninvasive and simple to employ, the major drawback of the photoplethysmograph is one of quantitative uncertainty - there is no means of calibration. However linearity of output with instantaneous arteriolar blood flow has been demonstrated by Davis (17). Proper usage is limited to intercomparison among the data given below.

The initial application of the sensor head was made to the thenar eminence of normal subjects, who were encouraged to produce two separate types of runs. In the first, pressure was to be increased continuously by simply pressing vertically upon the sensor head. In the second, maximum shear, short of slip, was to be produced with minimal amounts of pressure. Continuous analog recording of pressure, shear and blood flow permitted, through strain gauge calibration, construction of the instantaneous pressure and shear values developed in these tests.

Subsequent tests employed the same sensor head emplaced within a thick rigid plastic (plexiglas) seat attached to a wheelchair frame so as to simulate a wheelchair seat. All tests were conducted at a location 2 to 3 cm, lateral to the ischial tuberosities. An advantage of this site is the lack of influence over local events of the much higher pressures immediately under the ischial tuberosities. Thus normal young men did not occlude locally at test site pressures less than systolic values.

Normal subjects (N=9) were drawn from hospital staff and research workers. All were in apparent good health. Their average age was less than 30 years.

Geriatric and ill subjects (N=14) consisted of hospitalized veterans. Each was at least 67 years old. While subject to a variety of illness, those evidencing paralysis were excluded.

Paraplegic subjects (N=16) were veterans of average age 46. Diagnosed for lower extremity paralysis, all were able to transfer without assistance from their own wheelchair to the test wheelchair.

After removal of clothing in the area of the buttocks, the subject positioned himself on the test seat in a posture of his own choice. Location of the ischial tuberosities with respect to the sensors was determined in part by inspection through the transparent seat and in part by pressure value measurements obtained at the two pressure sensors. Large differences in pressure values, implying the existance of a severe pressure gradient, resulted in a request to the subject that he shift his body location. In this manner test sites were established close to the ischial tuberosities, yet sufficiently removed to offer small pressure gradients.

Results

Typical raw data resulting from the testing of normal palms (thenar eminence) is given in Figs 2 and 3 for a single subject. In the low shear test condition (Fig 2), despite the large variation in blood flow from run to run, it is apparent that occlusion will occur in the 100-120 mm Hg pressure region. In other words, if we extend the existing curves until they intersect the horizontal axis representing the zero blood flow condition, the intercept will take place at approximately 110 mm Hg. Similarly, if we extend the high shear data (Fig 3) an intercept in the 60 - 80 mm Hg pressure region is to be anticipated. The effect of shear is one of lowering that value of pressure necessary to arrive at occlusion. The same characteristic was noted in every tested subject.

To quantify shear effects, simultaneous equations were established describing those forces capable of halving blood flow from the maximum value observed in each subject. Implicit is the assumption (not proven) that shear and pressure effects upon blood flow inhibition in this domain are linear and "well behaved".

Averaging the results for all tested subjects indicated the application of a unit of pressure (mm Hg) to equal 2.6 units of shear (g/cm^2) SD 0.41 in terms of the tendency to restrict arteriole flow and promote occlusion. Converting to shared units (g/cm^2), a unit of pressure is equal in effect to about 2 units of shear. In brief, within the thenar eminence of normal subjects, with respect to cutaneous arteriolar pulsatile blood flow over the range from maximum flow to half maximum flow, the application of pressure is roughly twice as powerful as shear in reducing flow. Complete procedures and results have been given elsewhere (16).

Seating results (Fig 4,5,6) are presented in a format known as the ogive or "more than curve". Useful for coping with scattered results, the cumulative frequency of occurrence of data above a given value is portrayed. The ordinate or Y value of each curve reflects the percent of all data exceeding the corresponding abscissa or X value.

For example, consider the pressure results, Fig 4. While 100% of all normal (N) data exceeded 40 mm Hg pressure, only 57% of all paraplegic (P) data yielded a value in excess of 40 mm Hg. At the high end of the pressure scale, say 150 mm Hg, no N data exists; P results indicate that 27% of the data reflect pressure values in excess of 150 mm Hg. The median (50% data point) for both P and N groups is approximately 60 mm Hg. In short, P pressure values show a greater variation about the mean than do corresponding N values. Geriatric and ill (G) pressure data yields a slightly lower median value (52 mm Hg) and an extreme value range falling between those of P and N.

Shear results, Fig 5, show N group values to be substantially below those of the P and G groups. Median P and G shear values are roughly three times those of the median N value. A small but significant number (10%) of P and G shear values are large in an absolute sense (greater than 60 gms/cm^2). In short, unlike the pressure results, where a strong centering tendency is apparent at the 52-60 mm Hg level, the shear data demonstrates a considerable departure from the norm for both P and G groups.

Corresponding blood volume flow rates are given in Fig 6. N group flow rates are considerably higher than those evidenced by P and G group members; the median N blood volume flow rate is roughly three time median P or G values. P blood flow is especially low - only 6% of all P data yields a blood flow rate equal to or exceeding N median values.

In summary, when compared to normal males in terms of shear and skin blood flow in the vicinity of the ischial tuberosity, groups of paraplegic and hospitalized geriatric patients displaying roughly equal median pressure values evidence larger shear values and smaller cutaneous pulsatile blood flow volume rates.

Discussion

Before considering the implications of the results, it is useful to review the limitations of the test apparatus and procedures. For example, testing at the thenar eminence location is simple and straightforward- yet this area is known to be atypical anatomically (unusually rich in arteriovenous shunts). Pressure sore incidence in this locale is negligible. While there are pronounced anatomical similarities to the heel pad region, in which 11% of pressure sores are reported to occur (6), we have not tested at the heel pad. Hence while thenar eminence testing may have practical value, such practical application can only be inferred; it would be inappropriate to draw final conclusions from such data.

To test directly in a region with a known high incidence of trauma, the neighborhood of the ischial tuberosities was chosen. However our test procedure employs hard sensors buried flush within a hard seat. Those at risk with respect to pressure sores are unlikely to employ a hard seat. There is some question as to the reality of data gathered in these circumstances.

Hard instrumentation is employed in this work owing to the lack of suitable soft devices. Specifically, there are no known soft shear or soft blood flow instruments. Should one employ hard instruments at the flesh interface in a soft or cushioned environment, serious errors will result from the mismatch of compliance characteristics (18). By mounting the gauges in a hard seat, the sensors simulate a portion of the seat and properly indicate hard seat response to soft tissue loading. While hard seat testing is appropriate for hard sensors, a question arises concerning the practical application of such testing.

Seats, hard or soft, are expected to differ from one another in three respects in so far as load reaction is concerned. These are pressure, pressure gradient and shear. In this work, the reactive pressures encountered are similar to those anticipated in cushion testing. The similarity of pressure results from a test site displaced from the ischial tuberosities and therefore experiencing lower pressures.

Large pressure gradients, typical of hard seats, are rejected in our data assessment process. This is done by comparing the output of the two pressure sensors. Should large differences exist, the data is abandoned.

Shear, as measured in this work, is limited by the slick characteristics of the hard polished seat. Limiting shear values are those of slip between the unclothed subject and seat. It is quite likely that in a clothed and cushioned reality, the ensuing shear values are larger than those given here.

There is also some uncertainty as to the application of cutaneous blood flow characteristics to the issue of soft tissue ulceration. Much pressure sore induced necrosis occurs at levels far below the skin; the literature is unclear and even contentious concerning the relationship between events at the skin and those deep within soft tissue. Kosiak (19) perceives degeneration to be taking place simultaneously at all levels, including the skin. However other opinions exist in which the process of tissue failure is seen as highly sensitive to depth. For example, Rudd (20) concludes that the "subcutaneous tissue and muscle are more easily diamaged by pressure than is the overlying skin".

It is not the function of our work to clarify this issue. We merely assume that there are at least some forms of soft tissue failure that are associated with cutaneous ischemia and proceed to detail those load conditions trending towards dimished pulsatile cutaneous blood flow.

In short the experimental design is limited in terms of practical application. Nontheless, the relative performance of various subject groups in coping with an identical environment is of interest and may have some general value so long as one notes the experimental limitations.

One essential result of this work is that pressure and shear act in an additive fashion in terms of blood flow inhibition. A second result in that in the case of roughly equal median sitting pressures (52 to 60 mm Hg), median shear results for P and G group subjects are nearly three times corresponding normal values; median P and G blood flows are roughly one third normal values.

While it is possible that a causal relationship exists between the enlarged shear and the lessened blood flow characteristic of P and G group members, other possibilities exist. Some combination of seating habits, anatomical distinctions (atrophy) and the reduced systolic pressure experienced by paraplegic subjects (21) may also account for these results. Thus we have no comment on the source of the blood flow- shear relationship, which may be causally related or a matter of simple association.

However the existence of this relationship deserves notice. It follows that the design of a suitable support for those who are paraplegic or geriatric and ill is a more difficult task than designing for normal users.

Load response is sensitive to the precise biomechanical input factors. Given slightly different loading conditions, one can anticipate a markedly different response. Thus, Larsen et al (22) indicates no significant difference to exist in the blood flow cessation pressures between groups of normal and tetra or paraplegic subjects when external pressure was applied through an air pillow to skin capillaries in the sacral area experiencing maximal dilation (histamine). Loading through an air pillow implies the absence of shear; histamine dilatation implies maximizing the flow capabilities of vessels that might otherwise be readily occluded by load application.

In other words, under optimum loading conditions (Larsen et al) the load response of paraplegics can be made equal to that of normals. However under more difficult loading conditions (this study) sitting paraplegics approach ischemia more readily than do normals.

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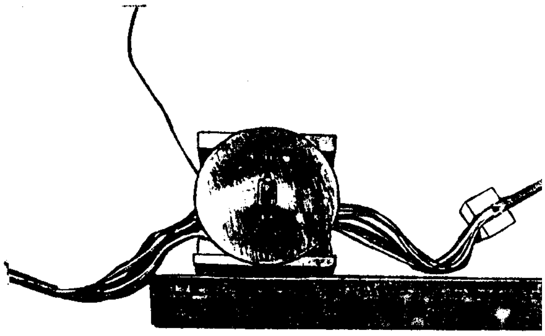
FIG 1. Sensor head containing two pressure gauges, one shear gauge, one blood flow photoplethysmograph



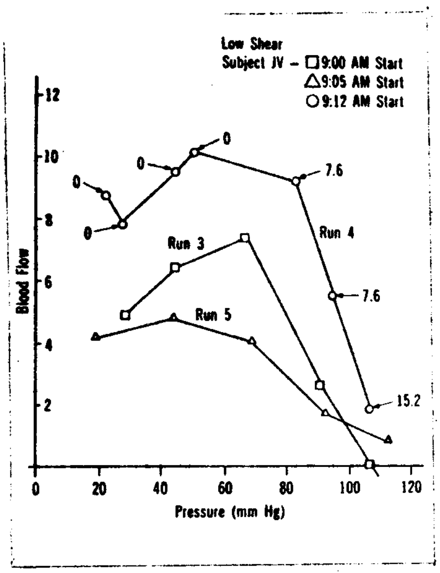
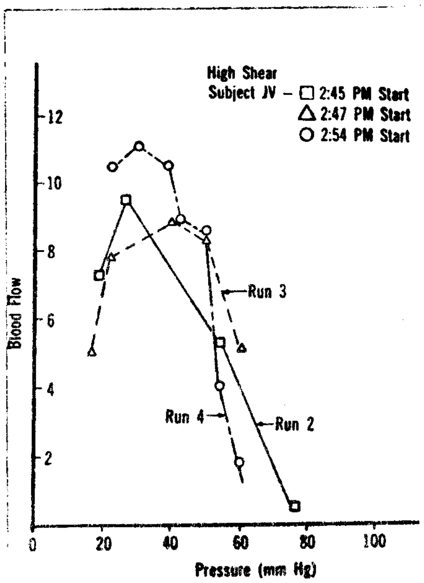
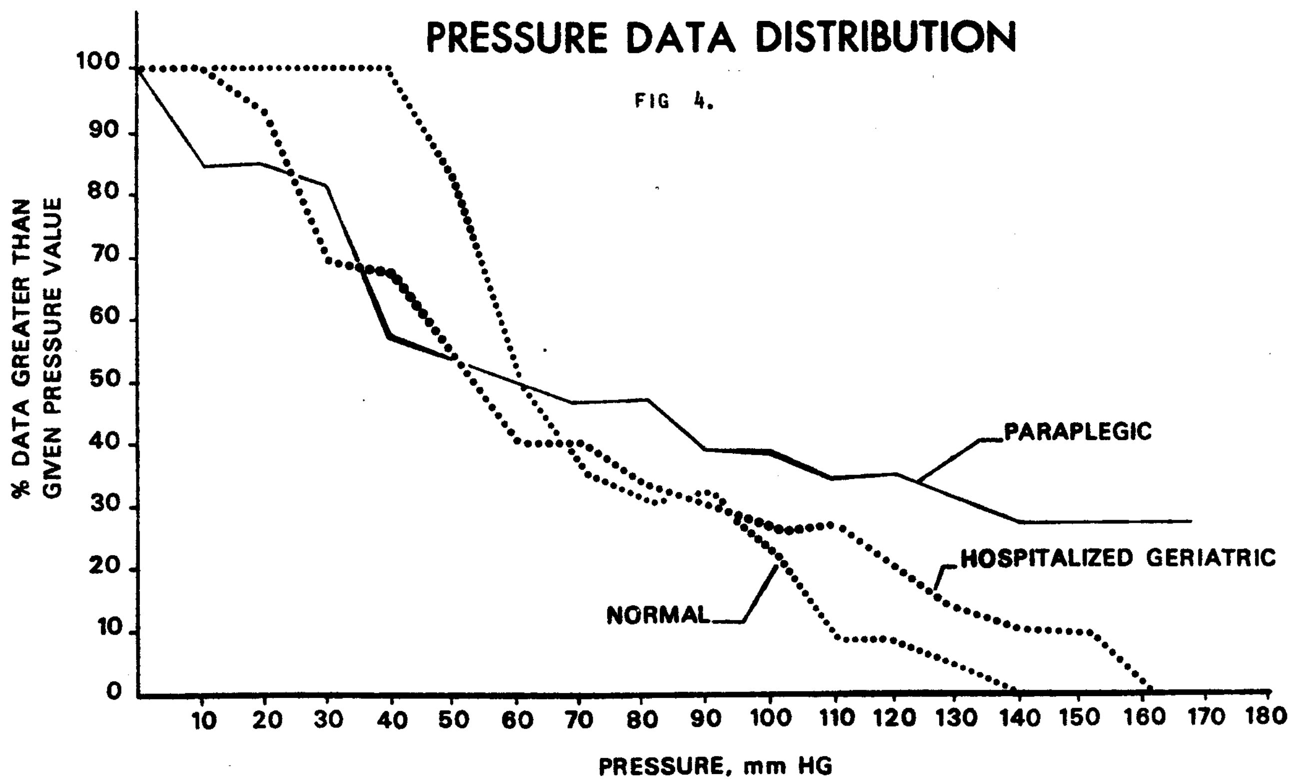


FIG 2. Pulsatile blood flow as a function of applied external pressure. Shear is small. Note trend towards occlusion in the vicinity of 110 mm Hg

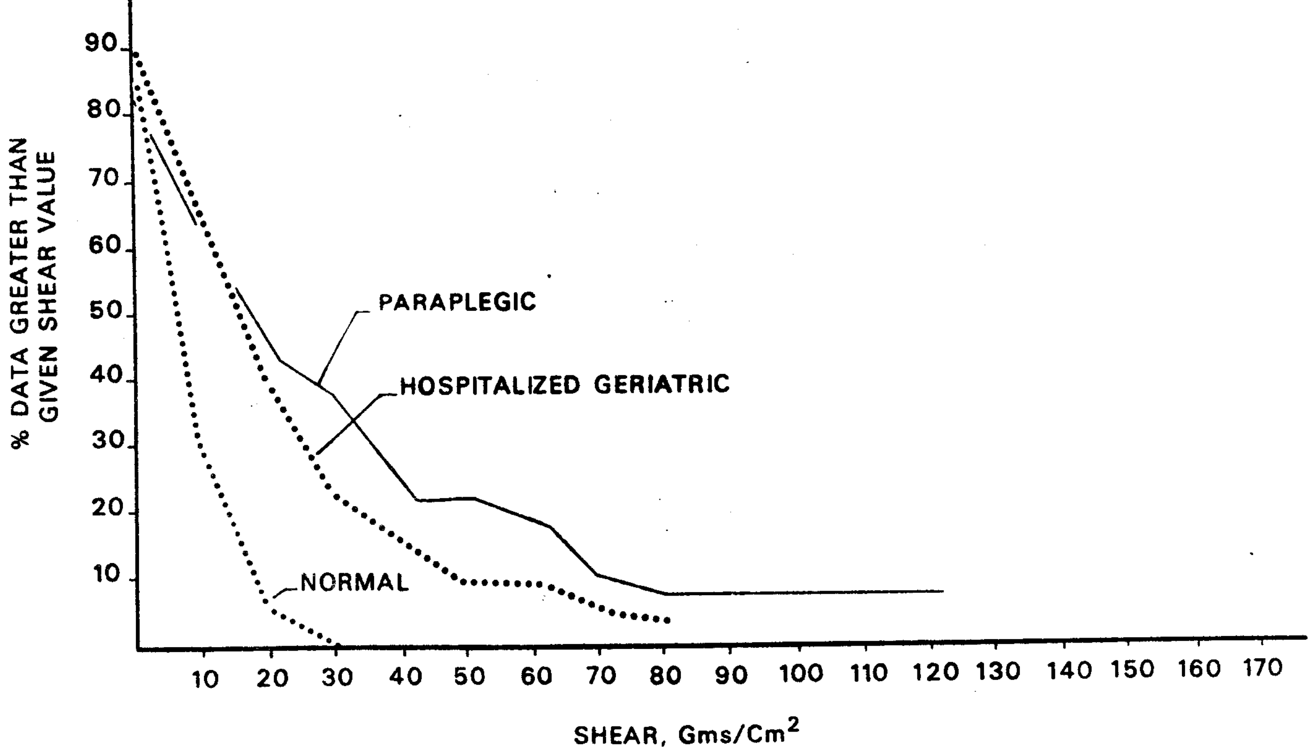
FIG 3. Pulsatile blood flow as a function of applied external pressure. Shear is large. Note trend towards occlusion in the vicinity of 70 mm Hg

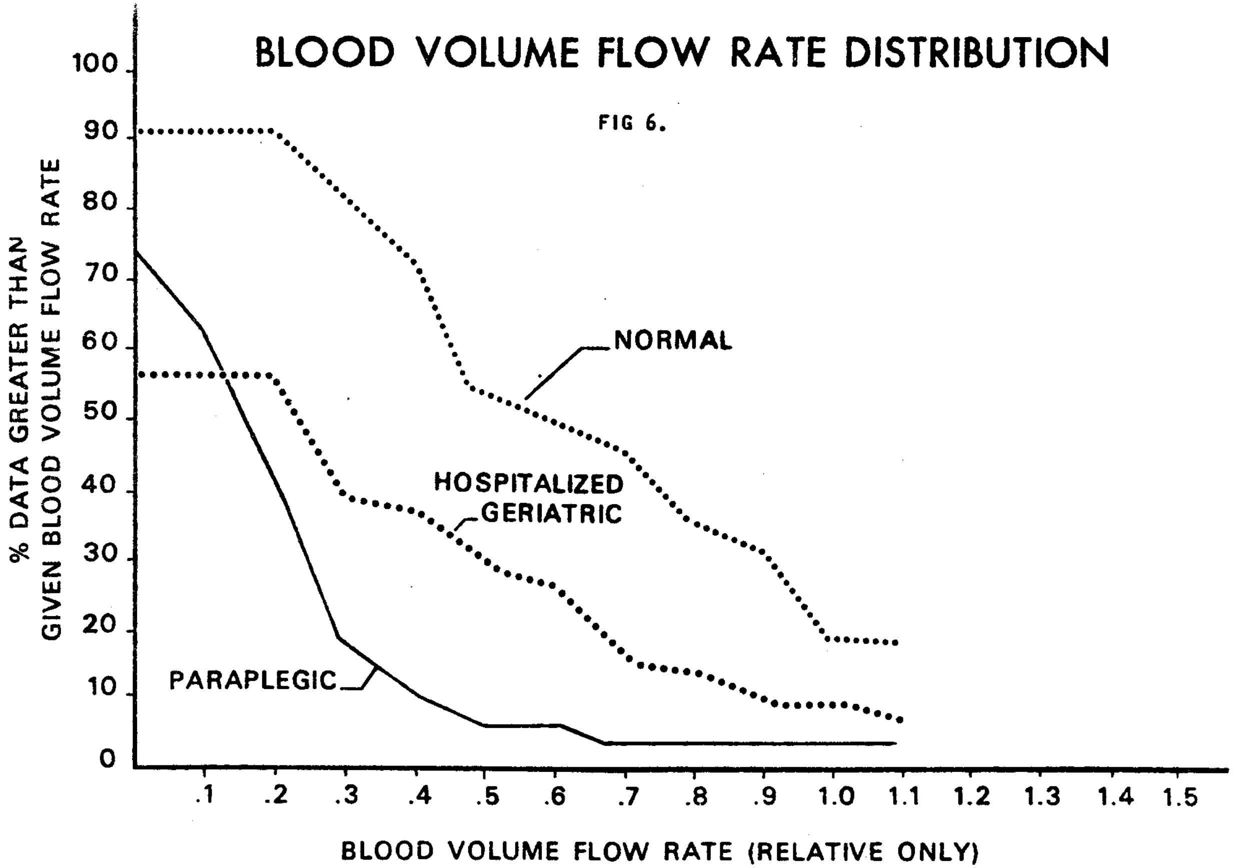



PRESSURE DATA DISTRIBUTION
FIG 4.
% DATA GREATER THAN GIVEN PRESSURE VALUE
0
10
20
30
40
50
60
70
80
90
100
PRESSURE, mm HG
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
PARAPLEGIC
HOSPITALIZED GERIATRIC
NORMAL

SHEAR DATA DISTRIBUTION

FIG 5.




BLOOD VOLUME FLOW RATE DISTRIBUTION
FIG 6.
% DATA GREATER THAN GIVEN BLOOD VOLUME FLOW RATE
100
90
80
70
60
50
40
30
20
10
0
NORMAL
HOSPITALIZED GERIATRIC
PARAPLEGIC
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0
1.1
1.2
1.3
1.4
1.5
BLOOD VOLUME FLOW RATE (RELATIVE ONLY)