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The Effect of Bending on Canine and Human Arterial Walls and on Blood Flow

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SUMMARY The femoral and popliteal arteries are least affected by atherosclerosis where they cross the hip and knee joint. We evaluated possible reasons for this by studying the changes in arterial length and diameter, and the patterns of blood flow in these arteries. The changes of length due to bending of human and canine popliteal arteries were determined radiographically and changes in arterial resistance and diameter were deduced from simultaneous measurements of pressure and flow in the artery above and below the canine knee joint in different degrees of flexion. Flow patterns were assessed in the canine popliteal artery during bending by radiographic screening of a streamline. Equivalent observations were made in a mechanical model. During flexion of the knee joint, the adjacent artery shortens by as much as 20%, but the arterial diameter remains effectively unchanged. The results of the flow pattern experiments suggest that turbulent flow is generated in the distal position of an artery crossing a bending joint. We suggest that these changes may be responsible for the lack of atherosclerosis in the segments of arteries that cross joints.


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THE BEHAVIOR of arteries under a variety of physical stresses has been investigated extensively in man and in animals (MacDonald, 1974). The majority of these studies examine the effects of pressure and pulse waves on the arterial wall, and the effects of arterial geometry on the pattern of blood flow. Most of the experiments have been performed on the aorta and the iliac arteries. Only a few studies have investigated the properties of the femoral and popliteal arteries, and all consider the arteries as if they were a set of immobile tubes. To our knowledge there are no published investigations of the changes that occur in an artery that...
crosses a joint during movement of the joint. Our clinical experience suggests that those parts of an artery that lie across a joint are less affected by occlusive atherosclerosis than adjacent sections of the artery not subjected to bending. Figure 1 shows the results of analysis of the distribution of atheroma in 50 translumbar arteriographs. The external iliac/common femoral artery and the popliteal artery, the vessels which cross the hip and knee joint, respectively, are the segments least affected by occlusive atherosclerosis. We considered, therefore, that an investigation of the effects of bending on the artery wall and the blood flow within it might be of relevance to the study of atherogenesis in man. This paper presents the results of a series of in situ experiments on man and on dogs of the effects of joint movement on arterial length, resistance, and diameter, and a study of the effects of bending on streamlines introduced into models.

Methods

The Measurement of Arterial Length

**Man**

Simultaneous biplane arteriographs were taken of the popliteal artery with the knee extended and then flexed in 10 male patients who were being investigated for renal or abdominal disease by perfemoral aortography.

Institutional rules for the protection of human subjects were observed by obtaining informed consent from the patients to take these additional films, after ethical approval by the hospital's research committee.

**Dog**

All experiments were performed on adult greyhounds, anesthetized with sodium pentobarbital (20 mg/kg, iv).

Arteriograms were performed through a catheter tied into a branch of the femoral artery just below the groin with the hindlimb in different degrees of flexion, using the same contrast medium as for man. AP and lateral films were taken, and the distances between the origins of identifiable branches above and below the knee joint were measured on the lateral projection.

To avoid the vasoactive effects of the radiopaque dye, the length of the popliteal artery was measured directly in three dogs. The superficial femoral and posterior tibial arteries were exposed through 2-cm incisions, 10 cm above and 5 cm below the knee joint, with a minimum of dissection. Small pieces of platinum wire were clipped to the adventitia of the arteries in a way that did not tether or damage the vessels. A 0.5 mm in diameter flexible radiopaque nylon catheter obturator, narrow enough not to cause any obstruction to blood flow, then was inserted into the femoral artery via a side branch high in the thigh and passed down inside the artery until the tip was below the lower marker. Radiographs were taken with the knee in various degrees of flexion. The distance between the arterial wall markers was measured along the line of the intraluminal obturator.

The Measurement of Popliteal Artery Resistance

In two greyhounds, short segments of the superficial femoral and posterior tibial arteries were exposed, and polythene catheters (external diameter, 0.6 mm) were inserted into the lumen of each artery through the side branches. These catheters were connected to Statham 23 DE transducers used for measuring mean arterial pressure. The catheter tips were kept in the same horizontal plane, which was also the zero reference level. A Statham electromagnetic flow gauge was fitted around the superficial femoral artery, just above the point of entry of
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Figure 2 The preparation for hindlimb nonpulsatile perfusion.

The measurements were repeated after division of the lumbar sympathetic chain.

The degree of movement was measured with a protractor. The speed of movement was kept as constant as possible but not timed. Flexion from 160° to 50° usually took 1 second. Measurements were taken 2 minutes after movement because preliminary studies showed that they were stable by this time.

The Assessment of Changes in Flow Pattern during Bending

In Pulsatile Flow

In three greyhounds the right femoral artery and the left femoral vein were cannulated and connected by a shunt of Silastic tubing 50 cm long and 4 mm i.d. Sodium diatrozoate (45%) (Hypaque) was injected through an 18-gauge needle inserted through the wall at the arterial end of the shunt at a rate which produced a radiopaque streamline in the shunt. The effect of a static bend on this "streamline" in normal pulsatile blood flow and the changes during passive repeated bending were recorded by standard cineangiography.

In Steady Flow

An apparatus was constructed of stiff plastic and Silastic (Fig. 3) to watch a fluorescent streamline in a column of water passing through a flexible tube (1 cm in diameter) which could be mechanically bent and straightened. The flow rate was maintained just below that at which turbulence occurred, approximately 15 ml/sec per cm². A stiff entrance tube 20 cm long ensured laminar flow in the tube entering the bending section. The behavior of the fluorescent streamline illuminated by ultraviolet light was recorded photographically.

The flexible Silastic tube was bent to angles up to 120°. The radius of the bend was never less than...
20 times the internal radius of the tube. Bending and straightening was repeated at rates of between 40 and 100 bends/min (rate of bending commonly occurring at the knee during walking).

Statistical Analysis

All results were analysed by Student's t-test or linear regression analyses. Probability values of 5% or less were interpreted as being statistically significant.

Results

Change in Artery Length during Knee Flexion

Man

Measurements were obtained from the arteriograms of 10 males whose ages ranged from 45 to 65 years (Fig. 4). Note that the straight leg has an angle at the knee joint of 180°. The fully flexed knee has an angle of 30-45°.

The absolute lengths and the percentage changes seen in the lateral views are presented in Table 1. The average amount of flexion was 100° (i.e., flexed from 180° to 80°), and this caused a mean shortening of 4.5 cm, 20% of the initial mean length. If the relation between angle and length is linear, the change was 0.5 mm per degree of flexion, i.e., a shortening of 0.22% per degree of flexion. The length of the vessels could not be measured in the AP films because the vessels were foreshortened. This view was taken to see whether there was any kinking or tortuosity. In every AP film the artery remained straight so that the changes in length on the lateral films were real, not apparent.

We also measured the diameter of the popliteal artery immediately opposite the knee joint when the joint was flexed and straight but could detect no change in diameter. Thus the changes in the vessel wall during flexion maintained the diameter, yet altered the length. The only noticeable change was the appearance of crinkling of the internal surface of the vessel when the artery was fully bent.

Dog

Table 2 gives the lengths of the popliteal artery of six dogs determined from arteriograms at different degrees of flexion, and gives the measurements remained straight so that the changes in length on the lateral films were real, not apparent.

Table 1: Changes in Length of the Human Popliteal Artery Caused by Knee Flexion

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Degrees of knee flexion</th>
<th>Before flexion</th>
<th>After flexion</th>
<th>% Shortening</th>
<th>% Shortening/1° of flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>102</td>
<td>230</td>
<td>178</td>
<td>23</td>
<td>0.23</td>
</tr>
<tr>
<td>2</td>
<td>146</td>
<td>209</td>
<td>171</td>
<td>18</td>
<td>0.12</td>
</tr>
<tr>
<td>3</td>
<td>90</td>
<td>269</td>
<td>227</td>
<td>16</td>
<td>0.18</td>
</tr>
<tr>
<td>4</td>
<td>115</td>
<td>217</td>
<td>167</td>
<td>23</td>
<td>0.20</td>
</tr>
<tr>
<td>5</td>
<td>130</td>
<td>215</td>
<td>170</td>
<td>21</td>
<td>0.16</td>
</tr>
<tr>
<td>6</td>
<td>75</td>
<td>200</td>
<td>160</td>
<td>20</td>
<td>0.26</td>
</tr>
<tr>
<td>7</td>
<td>140</td>
<td>204</td>
<td>173</td>
<td>15</td>
<td>0.11</td>
</tr>
<tr>
<td>8</td>
<td>70</td>
<td>245</td>
<td>188</td>
<td>23</td>
<td>0.33</td>
</tr>
<tr>
<td>9</td>
<td>55</td>
<td>231</td>
<td>184</td>
<td>20</td>
<td>0.36</td>
</tr>
<tr>
<td>10</td>
<td>85</td>
<td>231</td>
<td>180</td>
<td>22</td>
<td>0.26</td>
</tr>
</tbody>
</table>

Mean 100.8 225 180 20 0.22

All measured from arteriograms.

Figure 4: Examples of the changes in the human popliteal artery during flexion of the knee. The change in length between the arrows in a and b is 4 cm (19%), and between the arrows in c and d is 5.2 cm (21%). The crinkling of the inner surface of the artery when flexed is visible in b.
Table 2 Changes in Length of the Canine Popliteal Artery Caused by Knee Flexion

<table>
<thead>
<tr>
<th>Dog no</th>
<th>Degrees of knee flexion</th>
<th>Artery length (mm)</th>
<th>% Shortening of flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before flexion</td>
<td>After flexion</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>121 115</td>
<td>5.0</td>
</tr>
<tr>
<td>3</td>
<td>80</td>
<td>123 114</td>
<td>7.3</td>
</tr>
<tr>
<td>4</td>
<td>46</td>
<td>133 115</td>
<td>6.5</td>
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<tr>
<td>5</td>
<td>46</td>
<td>137 125</td>
<td>9.0</td>
</tr>
<tr>
<td>6</td>
<td>96</td>
<td>137 113</td>
<td>17.5</td>
</tr>
<tr>
<td>7</td>
<td>62</td>
<td>100 88</td>
<td>12.0</td>
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<tr>
<td>8</td>
<td>67</td>
<td>100 85</td>
<td>15.0</td>
</tr>
<tr>
<td>9</td>
<td>64</td>
<td>99 92</td>
<td>7.0</td>
</tr>
<tr>
<td>10</td>
<td>77</td>
<td>99 88</td>
<td>11.0</td>
</tr>
<tr>
<td>Mean</td>
<td>115</td>
<td>99 83</td>
<td>16.0</td>
</tr>
</tbody>
</table>

Measurements for dogs 2-7 were made from arteriographs; measurements for dogs 8-10 were made along intraluminal obturator, between the external markers.

The average flexion of 70° (from 150° to 80°) caused a mean shortening of 12 mm, 9.5% of the length when straight. Figure 5 shows that the relationship between the change in length, and flexion was linear over the range of flexion studied. The mean shortening was 0.17 mm per degree of flexion (i.e., 0.25% per degree of flexion, a figure similar to that found in the human measurements). The AP films showed no tortuosity or kinking, so the shortening in the lateral films was real.

The internal diameter of the arteries showed no measurable change. Crinkling of the inner surface of the vessel was not visible, but the maximum flexion was less than that studied in the men.

Changes in Artery Resistance during Knee Flexion (Dog)

From the measurements of arterial blood flow and the pressures above and below the knee, it was possible to calculate the resistance of the popliteal artery between the sites of pressure measurement in units of mm Hg/ml per min.

The resistance at different degrees of flexion during normal pulsatile flow, before and after lumbar sympathectomy, in two dogs is shown in Figure 6. Preliminary analyses showed that the changes in resistance were complete in 30 seconds and that the measurement at 2 minutes was representative of the new steady state. The regression analyses between the angle of flexion and the resistance for each dog showed a significant but very slight decrease in popliteal artery resistance with flexion. The response was abolished by the sympathectomy.

The maximum decrease in resistance caused by 100° of flexion was 0.033 mm Hg/ml per min.

In the two experiments in which flow was controlled and depulsed, the popliteal arteries also showed a significant decrease in resistance when the knee was bent, but lumbar sympathectomy...
Figure 6 The relationship between popliteal artery resistance and the angle of knee flexion, before and after sympathectomy. Normal pulsatile blood flow. K.J. = knee joint.

abolished this response in only one of these animals (Fig. 7).

Changes of the Internal Diameter of the Popliteal Artery

Attempts to measure the internal diameter of the popliteal artery from the arteriographs failed because of the lack of precise definition of the vessel wall caused by the grain of the film. Measurements to the nearest 0.2 mm showed no change. We therefore attempted to calculate any change in internal diameter in arbitrary units by using Poiseuille’s equation, although it was appreciated that, in the strict sense, the equation applies only to laminar flow in a straight tube. Poiseuille’s equation states

\[
\Delta P = \frac{8L \mu Q}{\pi r^4}
\]

where \(\Delta P\) = pressure change, \(L\) = length, \(r\) = radius, \(\mu\) = viscosity, and \(Q\) = flow.

Values for \(Q\) and \(P\) were derived from results obtained during the dog experiments to determine arterial resistance. The change in length between various degrees of flexion was derived from Figure 5, using an arbitrary figure of 100 for the length when the knee was straight; viscosity was assumed to remain unchanged. Using these approximations and assumptions, the calculations suggest that each animal had a small increase in popliteal arterial radius following knee bending (Table 3).

The Effect of Bending on Flow Characteristics

The characteristics of flow around a static bend as visualized in the pulsatile blood stream and steadily flowing water were similar. Laminar flow was maintained around static bends of up to 70° in the steady flow, water-filled model and in the pulsatile flow, blood-filled, Silastic shunt. Above this angle, the flow in the second half of the bend sometimes became disturbed and the streamline spread out.

During the bending the streamline of fluorescein or contrast material showed a consistent change. As the tube bent, the streamline, when seen in one plane, appeared to continue in a straight line and then reflected back, to and fro across the distal half of the bend. This probably represented a helical flow disturbance, but by the end of the bend there was frank turbulence with complete dispersal of the streamline marker across the whole column of fluid.

Thus, the effect of bending a tube, whether the flow was steady or pulsatile, was to cause turbulent flow in the distal half of the bend during movement, which returned to laminar flow once the movement had ceased.

Discussion

The experiments reported in this paper were stimulated by our observations of the distribution

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>Angle of knee joint</th>
<th>Calculated radius (arbitrary units)</th>
<th>% Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>18</td>
<td>150</td>
<td>4.11</td>
<td>+11.2</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>4.57</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>180</td>
<td>3.72</td>
<td>+18.5</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>4.41</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>150</td>
<td>6.69</td>
<td>+35.1</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>9.04</td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>150</td>
<td>5.90</td>
<td>+9.3</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>6.45</td>
<td></td>
</tr>
</tbody>
</table>

Table 3 Calculated Change in Popliteal Artery Radius during Knee Flexion
of human atherosclerosis. We can find no record of any studies on the effects of bending on arteries in the medical and physiological literature and no equivalent analysis in the engineering field.

The most striking observation has been the simultaneous marked (10–20%) shortening of the artery during flexion with the maintenance of an almost constant lumen. The shortening clearly prevents the vessel from becoming tortuous or kinked. This suggests that the length and diameter of an artery may be adjusted independently. If the popliteal artery does indeed shorten by 5 mm for every 10° of flexion without the lumen changing, it will have the particular value of preventing any gross change of resistance.

This response may be partially under sympathetic control via the muscle coats of the artery because the correlation between resistance and flexion was abolished by sympathectomy in three of the four dogs studied. Although the muscle and elastic layers of the artery can contract, it appears that the endothelium cannot, hence in human radiographs the arterial wall adjacent to the lumen appears to be crinkled when the joint is fully flexed.

The observation that an artery crossing a joint becomes less stretched when the joint is flexed is relevant to the consideration of why such an artery may be spared from atherosclerosis. The flux of lipoproteins across the arterial wall is considered to be an important controlling factor in the development of atherosclerosis, and much research has been directed toward identifying the factors affecting this flux. Duncan et al. (1965), Carew and Patel (1973), and Fry (1973) all have shown that flux of lipoproteins into the arterial wall is influenced by stretching. The more the wall is stretched, the larger the amount of lipoprotein that enters the vessel wall. It could be postulated, therefore, that the common femoral and popliteal arteries may be spared from atherosclerosis because they spend less total time stretched than, for example, the superficial femoral artery which is held in a constantly stretched state (Fry 1973).

The flow pattern within an artery may be as important a factor in the production of atherosclerosis as the behavior of the vessel wall. Caro et al. (1971, 1977) and Fry (1973) both have stressed the influence of wall shear rate on lipoprotein transport, and Mustard and Packham (1975) have indicated the relevance of flow patterns at all stages of the atheromatous process through its influence on thrombosis and platelet behavior at the endothelium. We have assumed that flow into the normal femoral and popliteal arteries is fundamentally laminar, and suggest that laminar flow may not be maintained in a vessel which is bending and straightening. Such flow changes may affect lipoprotein flux into the arterial wall by altering shear, but the details of any shear changes resulting from bending cannot be deduced from the studies reported here.

It may be that the combinations of turbulent flow and stretching and crinkling of the intima may reduce the deposition of platelets and fibrin by a mechanical "washing and cleaning" action.

Hypotheses based on the supposition that turbulence develops during bending must, however, be viewed with caution because we know of no published work that confirms that there is either laminar flow into the human superficial femoral artery and/or turbulent flow in the distal popliteal artery during bending. Spectral analysis of Doppler ultrasound recordings of femoral flow during exercise may solve this question but, so far, we have been unable to obtain "clean" enough recordings for useful analysis.

The joints of the upper limb have a wider range of movement than those of the lower limb. Measurements on a human brachial artery showed that, at the elbow, this vessel shortened by 2.3 cm (16.3 cm at 180°-straight, to 14 cm at 105°), i.e., 3 mm/10° of movement with no measurable change in diameter. It may be that the greater mobility of the shoulder and elbow joints protects the vessels of the arms from atheroma.

Although the bifurcation-turbulence theory may explain the deposition of atheroma in many sites it does not, for example, explain the extreme predilection for atheroma to appear at the adductor hiatus, in the middle of a straight tube. We suggest that atheroma is a generalized disease and that all arteries would be equally affected if it were not for factors that prevent its development. Movement appears to be one of these factors, and we suggest that movement of the arteries in some parts of the lower limb vessels and all of the upper limb vessels keeps them free of disease. Conversely, it may be that fixed points such as bifurcations and the adductor hiatus become severely diseased not only because of their geometry but because they are fixed.

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