There are two generally proposed causes of dissection of the aorta: (1) cleavage caused by blood entering the tear; and (2) haemorrhage that dissects the media and tear secondary to the cleavage. Using analysis of pressures and forces, this article shows that, on some occasions, these mechanisms alone cannot be responsible for causing aortic dissection.

This article addresses a possible cause of dissection of the aorta. Let us start with a variation of dissection which is not so common—that is, the dissection that has an entry tear located just beyond the left subclavian artery, but which extends proximally into the ascending aorta (in other words, the dissection cleavage propagates from the tear towards the aortic root).

To simplify matters, let us assume that the body is in the supine position. In other positions of the body the calculation is a bit more complicated but the result is the same.

**ANALYSIS OF PRESSURES AND FORCES**

**Forces acting on the flap proximally from the tear**

It is thought that cleavage from blood entering through the tear causes the propagation of dissection towards the aortic root. This is represented in fig 1. Points denoted with “F” are in the false channel and those denoted as “T” are in the true channel. If we compare pressure in the true channel at the tear to pressure at the aortic root it is obvious that pressure in the true channel always gradually rises from the tear to the aortic root (so pressure at T1 is greater than pressure at the tear, pressure at T2 is greater than pressure at T1, and pressure at T3 is greater than at T2).

If there is no flow in the false channel the pressure at any point within the false channel must be the same as the pressure at the tear (so pressure at F0, F1, F2, and F3 is the same as at the tear). When applying these facts to fig 1 it is obvious that pressure at point T1 is greater than pressure at the tear, pressure at T2 is greater than pressure at T1, and pressure at T3 is greater than at T2).

If there is no flow in the false channel the pressure at any point within the false channel must be the same as the pressure at the tear (so pressure at F0, F1, F2, and F3 is the same as at the tear).

If blood enters from the true channel through the tear into the false channel (that is, flows towards point F3) then pressure at F1 is lower than pressure at the tear (because liquid can flow only from higher pressure towards lower pressure), pressure at F2 is lower than at F1, and pressure at F3 is lower than at F2. Therefore, the pressure difference that pushes the flap towards the adventitia is greater than in the case in which there is no flow in the false channel.

**Pressure drop in the aorta**

To calculate maximum forces acting on the flap, the maximum pressure difference between a point in the false channel and its most adjacent point in the true channel (for instance, between the points F0 and T0) must be determined. The maximum pressure difference is when there is maximum pressure drop along the aorta. Since the maximum pressure at the aortic root is systolic and minimum pressure at the distal end of the aorta is diastolic, the maximum pressure difference between the aortic root and distal end cannot be greater than systolic pressure minus diastolic pressure. It can also be assumed that pressure linearly falls from the aortic root to the distal end.

So if, for example, systolic pressure is 135 mm Hg, diastolic pressure is 90 mm Hg, and the length of the aorta is 45 cm, then the maximum pressure drop is (135 – 90)/45 = 1 mm Hg/cm (this pressure drop will be used in further calculations).

**Forces acting on the flap distally from tear**

If the distance between the tear and T0 (fig 1) is 1 cm then pressure at T0 is 1 mm Hg lower than at the tear. When there is no flow in the false channel the pressure at any point within the false channel must be the same as the pressure at the tear (so pressure at F0, F1, F2, and F3 is the same as at the tear). When applying these facts to fig 1 it is obvious that pressure at point T1 is greater than pressure at the tear, pressure at T2 is greater than pressure at T1, and pressure at T3 is greater than at T2).

If no flow in the false channel the pressure at any point within the false channel must be the same as the pressure at the tear (so pressure at F0, F1, F2, and F3 is the same as at the tear).

If blood enters from the true channel through the tear into the false channel (that is, flows towards point F3) then pressure at F1 is lower than pressure at the tear (because liquid can flow only from higher pressure towards lower pressure), pressure at F2 is lower than at F1, and pressure at F3 is lower than at F2. Therefore, the pressure difference that pushes the flap towards the adventitia is greater than in the case in which there is no flow in the false channel.

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channel, the pressure at F0 (point F0 is just at the distal end of the false channel) is equal to the pressure at the tear, so the pressure at F0 is 1 mm Hg higher than at T0. Since the pressure difference at the beginning of the flap (just at the tear) is zero, the mean pressure difference on the flap distal from the tear is \((0 + 1)/2 = 0.5\) mm Hg. If we suppose, for the purpose of this discussion, that the transverse length of the flap is 5 cm (longitudinal length is equal to distance between tear and F0 or T0—that is, 1 cm) then the area of the flap distal to the tear is \(5 \times 1\) cm\(^2\) which, when multiplied by the mean pressure difference on the flap, gives a force by which the flap distal to the tear is pushed towards the true channel.

Converting 0.5 mm Hg to N/m\(^2\) gives \(1 \text{ mmHg} = 132.88 \text{ N/m}^2\) at 20°C): \(0.5 \times 132.88 = 66.4 \text{ N/m}^2\), and converting centimetres to metres, 1 cm = 0.01 m and 5 cm = 0.05 m. Thus, the force is:

\[
66.4 \times 0.01 \times 0.05 = 0.0332 \text{ N}.
\]

This force is too small to cause any further increase of the dissection in the distal direction. If blood enters the false channel then the pressure at F0 is lower than in the case when there is no flow in the false channel, therefore forces on the flap are lower too.

**Forces from haemorrhage**

It is also thought that haemorrhage dissects the media and that tear is secondary to it. Figure 2 shows a haemorrhage in the media. Point T is the point in the lumen of the aorta which is nearest to the haematoma. For the purpose of this discussion, let us say that the haemorrhage is situated just beyond the left subclavian artery, about 15 cm from the aortic root. If we use the same pressure drop as in the previous example, then pressure at point T is \(15 \text{ cm} \times 1 \text{ mm Hg/cm} = 15 \text{ mm Hg}\) lower than at the aortic root. The maximum possible pressure in the haematoma is pressure at the aortic root. This means that pressure in the haematoma cannot be more than 15 mm Hg higher than at point T and from that the maximum force produced by the pressure difference between the haematoma and point T can be calculated.

Let us suppose that the haematoma has an area of \(2 \times 2\) mm. Converting 15 mm Hg to N/m\(^2\) gives: \(15 \times 132.88 = 1993 \text{ N/m}^2\), and converting millimetres to metres, 2 mm = 0.002 m. Thus, the maximum force that is produced by the pressure difference between the haematoma and T is:

\[
1993 \times 0.002 \times 0.002 = 0.00797 \text{ N}.
\]

That force is too small to make any cleavage in the media or to tear the intima. If there is haemorrhage at the aortic root then the pressure difference between the haematoma and the most adjacent point to it in the lumen of the aorta is zero; therefore it is obvious that there is no force at all and that blood from that haematoma cannot undermine the aortic valve.

**THE REAL CAUSE OF DISSECTION**

**Formation of the dissection**

If the aortic smooth muscles contract the adventitia will follow that contraction. But as seen in fig 3, at the area of the aorta where the left subclavian, left carotid, and brachio-cephalic artery originate, downward movement of the aortic wall is very limited by those arteries. Upward movement of area A of the aorta is very limited by area D of the aorta. So at the segment of the aorta which comprises the origin of the left subclavian artery and area A, the aortic wall is fixed at the upper and the lower part, so at the upper and lower part of the segment the adventitia can only partially follow the contraction of smooth muscles (fig 4). As a consequence high stresses...
blockers and/or calcium antagonists will reduce this strong
cystic degeneration in the media, and haemorrhage takes
of the smooth muscles, especially when there are areas of
degeneration in the media and/or if there is any other
weakness in the media.

Why does dissection take place just beyond the origin of the
left subclavian artery and not at area A? It is because the
smooth muscles of the aorta (and thus also forces from the
smooth muscles) are not arranged around the origin of the left
subclavian artery as evenly as they are at area A. This uneven
distribution causes the concentration of forces in the aortic
media at the origin of the left subclavian artery which then
causes dissection.

Eventually, if the forces from the smooth muscles continue
to increase, the intima and part of the media on the smooth
muscle will break and a tear will result.

**Propagation of dissection**

Figure 5 shows a transverse cross section of dissection through
the flap. It can be seen that blood pressure forces act on all the
inside surfaces of the false and true channels. Assuming, for
simplicity's sake, that pressures both in the true and in the
false channel are the same, the forces acting on the flap from
the false channel and from the true channel are also equal
(and so cancel each other) and therefore they are not shown.

The forces shown stretch the aortic wall. While stretching,
the aortic wall stretches the flap in the direction that is
perpendicular to the long axis of the aorta and parallel with
the flap. Because each force must have a counter force of the
same intensity and opposite direction it means that the flap
pulls the aortic wall by force FF (fig 5, right). That force is
caused by elasticity of the flap and/or the presence of forces
from smooth muscles in the flap. This force FF by which the
flap pulls the aortic wall tends to damage the media further
and to increase dissection in the transverse direction. The
increasing dissection in the transverse direction is accompa-
nied by movement of the flap towards the centre of the aorta.
This movement pulls the rest of the flap which is located dis-
tally and/or proximally from the part of the flap shown in cross
section, and that causes further dissection in the distal and/or
proximal direction.

If blood pressure rises the stretching of the flap increases;
consequently the forces that tend to damage the media and to
increase dissection also rise. Once the flap, which is
perpendicular to the long axis of the aorta, reaches a certain
length (the length depending on pressure in the aorta), the
elastic forces from the flap alone are sufficient to increase dis-
section further.

If a tear does not occur, blood from haemorrhage could
slowly fill the false channel, thus establishing in the false
channel the same pressure as in the aorta, as occurs when
there is a tear. But, because of low inflow of blood from haem-
orrhage in this case, dissection can propagate only very slowly.

**CONCLUSION**

Dissection takes place as a consequence of major contraction
of the smooth muscles, especially when there are areas of
cystic degeneration in the media, and haemorrhage takes
place because of dissection of the media. Administration of β
blockers and/or calcium antagonists will reduce this strong
contraction of the smooth muscles, thus reducing dissection.1
On the other hand, discontinuation of long term therapy with
β blockers may provoke dissection.4

Fibrous tissue at the aortic root also fixes the aortic wall,
which makes this part of the aorta also suitable for initiating
dissection. This fixing of the aortic wall explains why more
than 95% of dissections begin at two places: either just above
the aortic valve or just beyond the origin of the left subclavian
artery.5

In hypertensives there is an increase in smooth muscle
mass and that also produces a stronger force.6 7 That, together
with acquired cystic degeneration in hypertensives, may
explain why dissection takes place mostly in hypertensives.

Smooth muscles in the media in large, elastic arteries are
generally helically oriented.4 Therefore it is readily possible
that dissection propagates in a direction perpendicular to the
orientation of these helically oriented smooth muscles
(anonymous to “normal” propagation which is perpendicular
to circumferentially oriented smooth muscles). As a result spiral
dissection develops, known as “spiral barber pole” dissection.1

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