Experimental Study on Mechanism of Establishing Collateral Circulation

Part I: Comparative Investigation on Pulsatile Flow and Steady Flow

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Introduction

Successful development and growth of collateral circulatory systems usually have great influence upon prognosis in obstructive arterial disease, and failure to establish collateral circulation may result in death of tissues peripheral to the obstruction. It has been widely believed that collateral circulation may be established secondarily if blood circulation is temporarily or permanently interrupted for various reasons. The first study on rise of collateral circulation, as far as I know, was made by John Hunter,¹ in 1785, who reported his observation on growth of stagantler after unilateral ligation of the external carotid artery. Many papers have been reported since then on mechanism of development and growth of collateral circulation, but to date no consensual interpretation has been established. In fact, too many problems have remained obscure in regard to circulatory dynamics and pathological physiology under circumstances of obstructed blood circulation. Resolution of those problems may contribute greatly to the treatment of patients with obstructive vascular diseases, especially now that more and more people are suffering from peripheral circulatory disturbances such as hypertension, arteriosclerosis, etc.

Many surgeons have agreed that marked development of collateral circulation is seen peripherally if one or more arterio-venous fistulae were established beforehand, whether congenital or acquired. Clinical application of this idea also has been attempted by making artificial arterio-venous fistulae hoping that peripheral blood circulation might be improved. In cases of extracorporeal circulation by means of cardiopulmonary bypass, it is reported²⁻⁵) that pulsatile blood flow, in other words amplitude of pulse pressure, has marked influence on peripheral blood circulation, which is especially true
when bypass time is prolonged. This fact suggests close relationship between peripheral blood circulation and pulsatile blood flow.

This series of experiments has been focused on the role of pulsatile blood flow, especially its influence upon development and growth of collateral circulation bypassing obstructed areas in cases of acute arterial obstructive disease.

**Experimental Methods**

Experimental animals & Experimental conditions:

Mongrels of approximately 10 Kgm. body weight were used for this experiment. Under intravenous Sod. Nembutal anesthesia (30mg per Kgm, body weight), and intratracheal airway tube, the abdominal cavity was explored through a median incision. Each end of two synthetic teflon tube grafts of 0.8cm in diameter, was anastomosed to the abdominal aorta peripheral to the junction of the renal arteries with an interval of approximately 3cm between upper & lower graft. The other ends of the teflon grafts were connected through a depulsator made with compressible air chamber to dampen pulse. (Fig. 1.). This procedure was performed successfully without interrupting aortic blood flow at all. Blood flowed distally through the depulsator when the abdominal aorta was clamped between the anastomoses, and thus non-pulsatile blood flow was obtained.

Every mongrel used for this experiment was premedicated with intravenous Heparin, 1mgm per kilogram of body weight.

Under the experimental conditions described above, development and growth of peripheral collateral circulation was compared in two major groups, one with pulsatile blood flow and the other with steady flow through the depulsator.

**Measurements**

a) Blood Pressure in Aorta:

A 2mm inside diameter polyethylene tube was inserted into the aorta from a branch of the subclavian artery to the level of the renal artery for measuring aortic pressure.
b) Peripheral Arterial Blood Pressure:

A thin vinyl tube of 0.7mm outside diameter and 0.5mm inside diameter was inserted from a branch of the anterior tibial artery to the site of obstruction in order to measure the blood pressure at the level of the femoral artery before and after interruption.

Throughout the entire procedure measurement of the pressure was done with strain guage (Statham) electric manometer and recorded by polygraph (made by San-yei Denki Co., Ltd.).

c) Amount of Blood Flow:

Peripheral blood flow immediately after occlusion by clamping was so little that it was very difficult to measure accurately. In this experiment, therefore, amount of back-flowing blood, collected from the femoral artery distal to the occlusion was measured 15 minutes after the occlusion.

d) Venous Pressure:

Venous pressure was measured through a small diameter catheter inserted into the femoral vein via a branch of the great saphenous vein.

Results of Experiment

a) Blood Pressure in Aorta:

Average blood pressure of 150-120mm Hg was always obtained and very little change

![Diagram showing recovery pattern of arterial blood pressure below femoral artery block](image)
Table 1 Change of blood pressure in aortic, femoral artery and vein during the experimental procedure

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B : Before connection with depulsator  
A : After connection with depulsator  
BFo : Before femoral artery interception  
1.3.5.15. : each numerical number express the time (minutes) after femoral artery occlusion.  
AP : Aortic pressure  
FP : Femoral artery pressure (peripheral side of occlusion)

of pressure was noted when the depulsator was connected. Temporary change of blood pressure at the time of switching the blood flow from original pulsatile flow to steady flow through the depulsator was technically avoided by prior regulation of the average pressure in the depulsator to that of the aorta as shown in Fig 2, Table 1.

Blood pressure at the level of the aorta was seldom influenced by interruption of the femoral artery on one side.

b) Peripheral Arterial Pressure:

1) Group with pulsatile Flow:

Average pressure of 150-120 mm Hg was recorded before interruption. The pressure dropped promptly to near 30 mm Hg upon interrupting the femoral artery on the same side. It began to recover, however, approximately 30 seconds after interruption, and almost 80% recovery was assured in 15 minutes. Rate of recovery was very slow.
Table 2 Change of blood pressure in aortic, femoral artery and vein during the experimental procedure

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B : Before connection with depulsator. A : after connection with depulsator
Bfo : Before femoral artery interception
1, 3, 5, 15 : each numerical number express the time (minutes) after femoral artery occlusion
A P : Aortic blood pressure
F P : Femoral artery pressure (peripheral side of occlusion)

thereafter. Very weak as it was, recovery of pulsation was noted in 3 minutes of interruption (Fig 2.).

2) Group with Steady Flow:

With this group of steady flow, rapid decrease in peripheral arterial pressure down to approx. 20mm Hg was noted immediately after interruption, which was somewhat lower than the pressure recorded with the pulsatile flow group. Tendency of recovery was also noticed 30 seconds afterwards with this group, but the speed of recovery was definitely slower than the pulsatile flow group. Approx. 50% recovery was accomplished in 3 to 5 minutes but recovery thereafter was extremely slow. Fig. 3 explains comparative degree of recovery 15 minutes later. In the pulsatile flow group average pressure of 90mm Hg was obtained, which was about 80% recovery, but in the steady flow group an average pressure of 50mm Hg (40% recovery) was barely
Table 3 Comparison of hemodynamic changes in hind limb of dog by femoral artery occlusion between pulsatile and non-pulsatile

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<th>No. of Experimental Dog</th>
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*For the calculation of the statistical significance between pulsatile group and non-pulsatile group. N.S. not significant (0.05<p)

Figure 3

Figure 4

Supplement to J. Kansai Med. Sch., Vol. 25, March 1970
obtained.

c) Venous Pressure;

Temporary lowering of the venous pressure was noted at the time of femoral inter-
ruption, but after dropping, the venous pressure tended to immediate recovery and no
significant difference was noticed between the two groups.

d) Amount of Blood Flow;

Amount of blood flow was measured by collecting back-flow blood dripping from the
femoral artery below the occlusion. As shown in Fig. 4, Table 3, a maximum amount
of 1.65 ml/sec minimum amount of 0.71 ml/sec and average amount of 1.09 ml/sec
were obtained with the pulsatile flow group. On the other hand, max. 0.81 ml/sec
min. 0.15 ml/sec and average amount of 0.54 ml/sec were recorded with the steady
flow group, only a half that of the pulsatile group.

Discussion

The following is an outline of theories discussed to date in connection with develop-
ment and growth mechanism of collateral circulatory systems in cases of arterial
obstruction:

1) Mechanical Pressure Elevation Theory; 6) 7)

Because of obstruction to blood flow, pressure on the proximal arterial wall elevates
secondarily and this results in formation of collateral circulation.

2) Hydrodynamic Theory; 8) -11)

Because of obstruction, difference in pressure proximal and distal to the site of
obstruction becomes suddenly larger and this encourages blood flow into very small
blood vessels which are otherwise usually empty. Thus collateral vessels which have
existed but not utilized grow larger because of hydrodynamic power, and take the role
of active collateral vessels.

3) Biochemical Stimulation Theory;

Ischemic tissues peripheral to obstruction produce so-called vasodilation materials
(VDM) as a result of anomalous metabolism, which help development and growth of
collateral blood vessels.

4) Neurological Reflex Theory; 6)

Reflexes of the autonomic nervous system are emphasized as important in development
of collateral circulation.

The first theory, in which only mechanical stimulation caused by elevated blood
pressure is taken into consideration, is easy to understand grossly, but is not enough
to explain even such a simple question as why collaterals are not built in cases of hypertensive disease when blood pressure is abnormally high without obstruction, and this becomes the ground for denial of this theory. Many scholars agree with the second theory of hydrodynamic origin which rationally explains the semipermanent organic dilatation of vessels. The theory, however, fails to explain satisfactorily the genesis so-called non-utilized small blood vessels. The third, biochemical growth theory, is not widely accepted because VDM themselves are obscure. And in addition, those who believe in the second theory are not persuaded. The fourth theory of nerve reflex origin is acknowledged by many scholars but is no more than hypothesis at this stage of study.

Seki and others reported an interesting paper lately on formation of collateral circulation at early on stage after obstruction in dogs, in which they emphasized that elasticity of the wall of blood vessels newly utilizes as collaterals was markedly decreased and local elevation of pressure proximal to the obstructing lesion was not a necessary condition for the formation of collateral circulatory pathways. They also reported that strong hydrodynamic power caused by increased pressure differential increasing the blood flow has almost no direct effect on initial formation of collaterals. As the cause of the marked decrease in elasticity of vascular walls they suggested neurological factors but came to no final conclusion. They denied indirectly the accumulation of so-called VDM by proving that oxygen content of the peripheral tissues had not been compromised at any stage of the experiment.

Investigation from the viewpoint of pressure theory in the blood vessels, quality and quantity of pulse pressure and pulsatile flow which may have direct influence upon the vascular wall becomes quite important when elasticity of vascular wall and state of peripheral blood flow are discussed.

It was believed that pulsation caused slight change in the speed of blood flow at the level of peripheral capillary vessels, but had almost no influence on vascular diameter. The reason pulsation is not noticed in blood flow in small veins has been explained as due to pulsatile flow in capillary vessels of different length, resulting in cancelled pulsation.

Recent study, however, has proved that blood flow in pulmonary capillary vessels does pulsate and is never steady flow at all. This fact must not be neglected if one tries to discuss the mechanism of oxygen exchange in lungs etc.

R. J. Parsons and others reported that pulsatile blood flow was essential for lymph to circulate smoothly and steady blood flow might cause disturbance of lymphatic
circulation resulting finally in tissue edema. Ogata et al made⁴, ¹⁵ a comprehensive comparative study on mechanism of pulsatile flow reaction of the whole organism when extracorporeal circulation by artificial heart-lung machine was attempted. According to their report peripheral circulation was strongly influenced when circulating blood was switched from pulsatile flow to steady flow showing the following changes; 1) Difference of oxygen content between artery and vein became smaller, 2) Metabolic acidosis took place because of insufficient utilization of oxygen in peripheral tissues, and 3) Blood flow in the true capillary finally stopped circulation after presenting sludging phenomena for a while secondary to the opening of arterio-venous shortcut pathways, and thus edema in the peripheral tissues developed after all. They pointed out that increase in absolute volume of circulating blood was essential in order to improve these unfavorable phenomena. It is true, however, that recent advanced knowledge in the rational dilution method of circulating blood such as with low molecular weight dextran has contributed greatly to prevention of sludging phenomena of blood flow which might have occurred otherwise in the true capillaries. ¹⁶, ¹⁷

Thus the state of blood flow, whether pulsatile or not, has much to do with peripheral circulation, and naturally it influences greatly the mechanisms of establishing collateral circulation in cases of arterial obstruction or interruption.

When the results of this experiment are carefully analyzed it can be said that the prompt recovery in 30 seconds of the rapid drop in blood pressure (measured at the level of a branch of the anterior tibial artery after interruption of the femoral artery) means successful opening of collateral circuits, and moreover, close observation of changes in blood pressure suggests that mechanism of development and growth of collaterals can be divided into two phases; the first phase, 30 seconds to one minute, in which prompt opening and rapid establishment of collateral circuits are observed, and the second phase in which slow but steady development of collaterals is expected.

This experiment, although it was limited to observation of the first phase alone, showed that beginning of the collateral vascular formation was first noted in 30 seconds to one minute of interruption and then actively functioning collateral pathways were completed in 15 to 20 minutes. So far as the first phase is concerned it may be said that pulsatile flow contributes more to the formation and growth of collateral circuits than does steady flow. In order to investigate this proposition quantitatively further study from the viewpoint of theory is required.

The relation between pressure and flow through the vessel from point A to B is given by the following equation.⁴, ¹⁸

\[ P = \frac{1}{C} \frac{dQ}{dt} \]
\[ PA - PB = KQR \]  
\[ R = \frac{(PA - PB)}{KQ} \]  

where  
\( PA \) = Blood pressure at the point of A  
\( PB \) = Blood pressure at the point of B  
\( Q \) = Volume flow rate  
\( R \) = Resistance of the vessel between A & B  

When no occlusion exist and distance between A & B is short, 
\( R \) is practically nearly zero, then,  
\( PA = PB \)  

If artery is interrupted between A & B, blood flows from A to B via only collateral vessels. The \( R \) means total resistance of collateral pathways. 

Then, following expression exist. 
\[ R = K \frac{(PA_1 - PB_1)}{q} \]  

where \( q \) means amount of blood flow via collaterals, \( PA_1 \) & \( PB_1 \) are blood pressure at A & B respectively. \( K \) is constant  

In this case \( q \) may be replaced by amount of backflowing blood collected from the femoral artery without any significant error. Collateral vascular resistance \( R \) is then calculated by substituting the amount of blood volume measured 15 minutes after interruption for \( q \) as show in Fig. 5. 

In the pulsatile flow group, \( R \) varied from a minimum of \( 18.5 \times 1332 \) dyne sec/cm\(^5\) to a maximum of \( 63.5 \times 1332 \) dyne sec/cm\(^5\). with the steady flow group on the other hand, it varied from a minimum of \( 92.4 \times 1332 \) dyne sec/cm\(^5\) to a maximum of \( 600 \times 1332 \) dyne sec/cm. Thus obvious decrease in resitance of collateral vascular systems is proved with the pulsatile group, which means better development and growth of collateral vessels for pulsatile flow over steady flow. In order to obtain correct value of \( R \), other factors such as temperature, reactive hypermia caused by operative procedures and so forth naturally should be taken into consideration, but in this experiment, where care was taken to keep these factors similar throughout the procedure,
even simple comparison of $R$ thus obtained between the two groups is quite significant.

From the experimental results mentioned above in detail it can be said that in the pulsatile flow group dropping peripheral blood pressure recovers more rapidly, and more back-flowing blood and less resistance of the collateral vascular walls are recorded. These data indicate that faster and more satisfactory development and growth of collateral vessels are secured with pulsatile flow than with steady flow when the femoral artery is interrupted.

When evaluating this experiment, it is important to remember that total extracorporeal circulation was not used, but blood circulation was accomplished by the heart itself, keeping the so-called autoregulatory mechanisms of the body, such as aortic arch and carotid sinus untouched. Further studies are required at this point in the future. In 1932 D. W. Bronk\(^\text{19}\) succeeded in recording centripetal impulse from the carotid sinus of rabbits which corresponded exactly to the pulsation. It is easily presumed that the centripetal impulse thus recorded may travel to the periphery via the vasomotor centre which is believed to exist in the diencephalon, and may regulate the tone of the vessels. In fact some kinds of Raynaud Phenomena are distinctly controlled with tranquilizers such as meprobamate. In addition, pulsatile flow influences vascular walls not only as hydrodynamic power but also transmits pulsation to vascular wall, which is immediately conducted to the periphery. Blood circulation in the periphery is thus closely effected by pulsatile flow.

Clinically, transmission of pulsatile flow is compromised if sclerotic changes of the vascular wall such as atherosclerosis or other pathological changes like Bürgers disease are encountered. Other factors such as viscosity of the blood, contents of fat, protein, metabolic products and metabolic mechanism itself in the peripheral tissue, etc., should not be neglected.

I believe that the important and favorable role of so-called pulsatile flow in the development and growth of collateral circulation immediately after acute arterial obstruction of the lower extremities is successfully explained here.

**Summary**

1) Establishment of collateral vessels after artificial interruption of one femoral artery was carefully observed and compared between two groups, one with pulsatile flow and the other with steady flow. A depulsator was connected to the aorta without blocking it in order to obtain steady flow.
2) Peripheral blood pressure which dropped rapidly upon clamping the femoral artery began to recover in 30 seconds and kept rising rather rapidly for the next 15 minutes, but rather slowly thereafter. More rapid and more satisfactory recovery was observed with pulsatile flow.

3) Quantitatively more back-flowing blood was collected from the pulsatile flow group 15 minutes after interruption.

4) Significantly less collateral vascular resistance was calculated in the pulsatile group, which indicated that pulsatile flow was superior to steady flow in ability to establish collateral circulation.

References

16) Thorsen, G., and Hint, H.: Aggregation, sedimentation and intravascular sludging of

