H.N. Mayrovitz, L.H. Peterson, and A. Noordergraaf
Bockus Research Institute and Dept. of Biomedical Engineering
University of Pennsylvania
Philadelphia, PA 19104

The existence of rhythmic contractions of microvessels in unanesthetized animal preparations has been demonstrated as early as 1852[1]. Since that time several investigators[2,3,4] have documented their experimental findings relating to some of the intrinsic properties of these dynamic processes. Recently[5], an analytical treatment of venomotion, as observed in muscular venules and small veins of the unanesthetized bat has provided quantitative results suggestive of a local post capillary control mechanism. The analysis included some essential characteristics of the active vessel but did not include the composite hemodynamic effect of the feeding vessel characteristics, mode of active vessel contraction, or the changes in blood suspension associated with these dynamics. It is the objective of the work reported here to describe the hemodynamic consequences of these factors.

General Approach

The active vessel is characterized as a straight, cylindrical vessel which undergoes uniform, periodic diameter change without shape change. The active vessel is bounded by equivalent source and drain vessels which are characterized by their respective fluid impedances as "seen" by the active vessel.

Source and Drain Characteristics

Taking the drain resistance to be small compared to the active vessel resistance, two cases of special interest are distinguished for comparison purposes as upstream conditions P and Q. In condition P the source as seen by the active vessel is taken as one of constant pressure. For this condition the principal quantity of interest is the time average of flow through the active vessel. In condition Q the source is taken as one of constant flow, with the primary quantity of interest being the time average pressure gradient along the vessel.

Mode of Active Contraction

Independent of the upstream and downstream conditions being analyzed, there are two modes of contraction which need to be considered. The physiological mechanism(s) producing contraction amplitude change may occur without substantial change in vessel maximum diameter. On the other hand, different contraction amplitudes can have essentially the same average diameter. Thus we distinguish between a mode I in which the mean diameter of the active vessel remains constant with changing contraction amplitude, and a mode II in which the maximum diameter remains constant with changing contraction amplitude.

Changes in Blood Suspension

The erythrocyte concentration and hence the apparent viscosity within the active vessel is dependent upon the source concentration and the active vessel diameter. Since the active vessel diameter is not constant, differences in vessel fluid resistance between dilated and contracted phases are expected due to this factor. To investigate its hemodynamic significance the approach was to 1) develop a relationship between source concentration and vessel diameter, 2) derive an expression relating fluid viscosity to erythrocyte concentration using theory applicable to heterogeneous media, and 3) to derive an expression for the average pressure gradient.

Method of Analysis

For condition P, expressions were derived for the average flow as a function of contraction amplitudes for both modes. For experimentally determined contraction amplitudes the calculated flows were compared to that flow which would occur in a non-active vessel with a diameter equal to 1) the active vessel's mean value and 2) to the active vessel's maximum diameter. For condition Q the analysis was the same except the hemodynamic quantity was the average pressure gradient. When the viscosity factor was included the average pressure gradient was compared to the average gradient without viscosity effects.

Results

1. The flow through the active vessel is larger for both modes than the corresponding flow through the non-active vessel when the comparison is made on the basis of the same average diameter. For the same contraction amplitude the flow ratio is larger for mode II diameter variation as compared to mode I.
2. The flow through the active vessel is smaller for both modes than the corresponding steady flow when the comparison is made on the basis of the same maximum diameter. For the same contraction amplitude the flow ratio is smaller for mode II diameter variation.
3. In each case the average pressure gradient associated with the active vessel is larger than the corresponding non-active vessels.
4. The derived expression for the viscosity as a function of concentration given by $\eta = \eta_p(1+2.5\frac{H}{1-H})$ in which H = hematocrit, η_p = plasma viscosity agrees very well with experimental data and contains Einstein's[6] result as a special case. This expression has the advantage of being derived rather than empirically fitted.
5. The effect of including the viscosity variations in the active vessel is to reduce the amount by which the active vessel average pressure gradient exceeds the non-active cases. This effect increases with increasing contraction amplitude.

REFERENCES

1. Jones, T.W. Physiol. Trans. Royal Soc., London 142:131, 1852.
2. Wiedeman, M.P. Circ. Res. 19:559 (1966).
3. Wiederhielm, C.A. Bibl. Anat. 9:321 (1967).
4. D'Agrosa, L. Am.J. Physiol. 218:530 (1970).
5. Mayrovitz, H.N., A. Noordergraaf. 24th ACEMB, Las Vegas (1971).
6. Einstein, A. Ann. Phys. 19:289 (1906).

27.4 HEMODYNAMIC CONSEQUENCES OF RHYTHMICALLY ACTIVE MICROVESSELS

H.N. Mayrovitz, L.H. Peterson, and A. Noordergraaf
Bockus Research Institute and Dept. of Biomedical Engineering
University of Pennsylvania
Philadelphia, PA 19104

The existence of rhythmic contractions of microvessels in unanesthetized animal preparations has been demonstrated as early as 1852[1]. Since that time several investigators[2,3,4] have documented their experimental findings relating to some of the intrinsic properties of these dynamic processes. Recently[5], an analytical treatment of venomotion, as observed in muscular venules and small veins of the unanesthetized bat has provided quantitative results suggestive of a local post capillary control mechanism. The analysis included some essential characteristics of the active vessel but did not include the composite hemodynamic effect of the feeding vessel characteristics, mode of active vessel contraction, or the changes in blood suspension associated with these dynamics. It is the objective of the work reported here to describe the hemodynamic consequences of these factors.

General Approach

The active vessel is characterized as a straight, cylindrical vessel which undergoes uniform, periodic diameter change without shape change. The active vessel is bounded by equivalent source and drain vessels which are characterized by their respective fluid impedances as "seen" by the active vessel.

Source and Drain Characteristics

Taking the drain resistance to be small compared to the active vessel resistance, two cases of special interest are distinguished for comparison purposes as upstream conditions P and Q. In condition P the source as seen by the active vessel is taken as one of constant pressure. For this condition the principal quantity of interest is the time average of flow through the active vessel. In condition Q the source is taken as one of constant flow, with the primary quantity of interest being the time average pressure gradient along the vessel.

Mode of Active Contraction

Independent of the upstream and downstream conditions being analyzed, there are two modes of contraction which need to be considered. The physiological mechanism(s) producing contraction amplitude change may occur without substantial change in vessel maximum diameter. On the other hand, different contraction amplitudes can have essentially the same average diameter. Thus we distinguish between a mode I in which the mean diameter of the active vessel remains constant with changing contraction amplitude, and a mode II in which the maximum diameter remains constant with changing contraction amplitude.

Changes in Blood Suspension

The erythrocyte concentration and hence the apparent viscosity within the active vessel is dependent upon the source concentration and the active vessel diameter. Since the active vessel diameter is not constant, differences in vessel fluid resistance between dilated and contracted phases are expected due to this factor. To investigate its hemodynamic significance the approach was to 1) develop a relationship between source concentration and vessel diameter, 2) derive an expression relating fluid viscosity to erythrocyte concentration using theory applicable to heterogeneous media, and 3) to derive an expression for the average pressure gradient.

Method of Analysis

For condition P, expressions were derived for the average flow as a function of contraction amplitudes for both modes. For experimentally determined contraction amplitudes the calculated flows were compared to that flow which would occur in a non-active vessel with a diameter equal to 1) the active vessel's mean value and 2) to the active vessel's maximum diameter. For condition Q the analysis was the same except the hemodynamic quantity was the average pressure gradient. When the viscosity factor was included the average pressure gradient was compared to the average gradient without viscosity effects.

Results

1. The flow through the active vessel is larger for both modes than the corresponding flow through the non-active vessel when the comparison is made on the basis of the same average diameter. For the same contraction amplitude the flow ratio is larger for mode II diameter variation as compared to mode I.
2. The flow through the active vessel is smaller for both modes than the corresponding steady flow when the comparison is made on the basis of the same maximum diameter. For the same contraction amplitude the flow ratio is smaller for mode II diameter variation.
3. In each case the average pressure gradient associated with the active vessel is larger than the corresponding non-active vessels.
4. The derived expression for the viscosity as a function of concentration given by $\eta = \eta_p(1+2.5\frac{H}{1-H})$ in which H = hematocrit, η_p = plasma viscosity agrees very well with experimental data and contains Einstein's[6] result as a special case. This expression has the advantage of being derived rather than empirically fitted.
5. The effect of including the viscosity variations in the active vessel is to reduce the amount by which the active vessel average pressure gradient exceeds the non-active cases. This effect increases with increasing contraction amplitude.

REFERENCES

1. Jones, T.W. Physiol. Trans. Royal Soc., London 142:131, 1852.
2. Wiedeman, M.P. Circ. Res. 19:559 (1966).
3. Wiederhielm, C.A. Bibl. Anat. 9:321 (1967).
4. D'Agrosa, L. Am.J. Physiol. 218:530 (1970).
5. Mayrovitz, H.N., A. Noordergraaf. 24th ACEMB, Las Vegas (1971).
6. Einstein, A. Ann. Phys. 19:289 (1906).

27.4 HEMODYNAMIC CONSEQUENCES OF RHYTHMICALLY ACTIVE MICROVESSELS

H.N. Mayrovitz, L.H. Peterson, and A. Noordergraaf
Bockus Research Institute and Dept. of Biomedical Engineering
University of Pennsylvania
Philadelphia, PA 19104

The existence of rhythmic contractions of microvessels in unanesthetized animal preparations has been demonstrated as early as 1852[1]. Since that time several investigators[2,3,4] have documented their experimental findings relating to some of the intrinsic properties of these dynamic processes. Recently[5], an analytical treatment of venomotion, as observed in muscular venules and small veins of the unanesthetized bat has provided quantitative results suggestive of a local post capillary control mechanism. The analysis included some essential characteristics of the active vessel but did not include the composite hemodynamic effect of the feeding vessel characteristics, mode of active vessel contraction, or the changes in blood suspension associated with these dynamics. It is the objective of the work reported here to describe the hemodynamic consequences of these factors.

General Approach

The active vessel is characterized as a straight, cylindrical vessel which undergoes uniform, periodic diameter change without shape change. The active vessel is bounded by equivalent source and drain vessels which are characterized by their respective fluid impedances as "seen" by the active vessel.

Source and Drain Characteristics

Taking the drain resistance to be small compared to the active vessel resistance, two cases of special interest are distinguished for comparison purposes as upstream conditions P and Q. In condition P the source as seen by the active vessel is taken as one of constant pressure. For this condition the principal quantity of interest is the time average of flow through the active vessel. In condition Q the source is taken as one of constant flow, with the primary quantity of interest being the time average pressure gradient along the vessel.

Mode of Active Contraction

Independent of the upstream and downstream conditions being analyzed, there are two modes of contraction which need to be considered. The physiological mechanism(s) producing contraction amplitude change may occur without substantial change in vessel maximum diameter. On the other hand, different contraction amplitudes can have essentially the same average diameter. Thus we distinguish between a mode I in which the mean diameter of the active vessel remains constant with changing contraction amplitude, and a mode II in which the maximum diameter remains constant with changing contraction amplitude.

Changes in Blood Suspension

The erythrocyte concentration and hence the apparent viscosity within the active vessel is dependent upon the source concentration and the active vessel diameter. Since the active vessel diameter is not constant, differences in vessel fluid resistance between dilated and contracted phases are expected due to this factor. To investigate its hemodynamic significance the approach was to 1) develop a relationship between source concentration and vessel diameter, 2) derive an expression relating fluid viscosity to erythrocyte concentration using theory applicable to heterogeneous media, and 3) to derive an expression for the average pressure gradient.

Method of Analysis

For condition P, expressions were derived for the average flow as a function of contraction amplitudes for both modes. For experimentally determined contraction amplitudes the calculated flows were compared to that flow which would occur in a non-active vessel with a diameter equal to 1) the active vessel's mean value and 2) to the active vessel's maximum diameter. For condition Q the analysis was the same except the hemodynamic quantity was the average pressure gradient. When the viscosity factor was included the average pressure gradient was compared to the average gradient without viscosity effects.

Results

1. The flow through the active vessel is larger for both modes than the corresponding flow through the non-active vessel when the comparison is made on the basis of the same average diameter. For the same contraction amplitude the flow ratio is larger for mode II diameter variation as compared to mode I.
2. The flow through the active vessel is smaller for both modes than the corresponding steady flow when the comparison is made on the basis of the same maximum diameter. For the same contraction amplitude the flow ratio is smaller for mode II diameter variation.
3. In each case the average pressure gradient associated with the active vessel is larger than the corresponding non-active vessels.
4. The derived expression for the viscosity as a function of concentration given by $\eta = \eta_p(1+2.5\frac{H}{1-H})$ in which H = hematocrit, η_p = plasma viscosity agrees very well with experimental data and contains Einstein's[6] result as a special case. This expression has the advantage of being derived rather than empirically fitted.
5. The effect of including the viscosity variations in the active vessel is to reduce the amount by which the active vessel average pressure gradient exceeds the non-active cases. This effect increases with increasing contraction amplitude.

REFERENCES

1. Jones, T.W. Physiol. Trans. Royal Soc., London 142:131, 1852.
2. Wiedeman, M.P. Circ. Res. 19:559 (1966).
3. Wiederhielm, C.A. Bibl. Anat. 9:321 (1967).
4. D'Agrosa, L. Am.J. Physiol. 218:530 (1970).
5. Mayrovitz, H.N., A. Noordergraaf. 24th ACEMB, Las Vegas (1971).
6. Einstein, A. Ann. Phys. 19:289 (1906).