SOME OBSERVATIONS OF CRITICAL CLOSING PRESSURES IN THE PERIPHERAL CIRCULATION OF ANAESTHETIZED PATIENTS

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SUMMARY
Critical closing pressures have been measured in the hand vessels of anaesthetized patients undergoing elective surgery by a method as close as possible to that used in previous studies on conscious subjects. The critical closing pressure has been found to vary considerably during anaesthesia. In general, the variations are parallel to those of the systolic blood pressure. No correlation has been found between the critical closing pressure and the pre-operative blood pressure. It is therefore suggested that findings in conscious volunteers cannot be used to assess the possibility of critical closure during anaesthesia; it appears necessary to measure the critical closing pressure during anaesthesia.

The possibility of critical closure in arteries during hypotension has been put forward as an objection to the induction of hypotension during anaesthesia. Previous work in conscious volunteers has shown that the higher a subject's blood pressure, the higher his critical closing pressure is likely to be. This might seem to support the belief that induced hypotensive anaesthesia is especially dangerous in hypertensive patients. In 1951 Burton suggested, on theoretical grounds, that a certain minimum pressure across the wall—the transmural pressure—is required in an artery to maintain patency when the wall is in a state of tension (Burton, 1951). This minimum pressure he called the critical closing pressure. The reasoning behind this suggestion is summarized in the appendix. Since 1951 several workers have reported evidence of critical closure in vascular beds. They include Burton and Yamada (1951), Yamada (1954), Roddie and Shepherd (1956), Gaskell and Krisman (1958), Coles and Gough (1960) and Ashton (1962a, b, c), in humans; and Nichol et al. (1951) and Howell and Richards (1955), in animals.

Most of the work in humans has been done by enclosing the forearm, hand or finger in a plethysmograph whose internal pressure can be varied. The easiest way to do this, and the method used in this present study, is to use a water-filled plethysmograph connected to a column of water the height of which can be varied. As the height is increased, there is an increased hydrostatic pressure in the plethysmograph which is assumed to be transmitted throughout the tissues of the enclosed member. If the pressure inside the arteries is taken to be equal to the blood pressure, the pressure across the artery wall can be found by subtraction thus:

\[
\text{Transmural pressure} = \text{Blood pressure} - \text{Hydrostatic pressure}.
\]

Provided that a means of varying the height of the water column is available, the transmural pressure can be altered at will by the experimenter without any need to alter the blood pressure.

Bloodflows with this apparatus are measured in the usual way, by suddenly occluding venous return from the part by inflating a pneumatic cuff just proximal to the plethysmograph. Volume changes are transmitted through the water column and then through air. A side arm is necessary to ensure that the top of the water column is at atmospheric pressure at the beginning of each observation. Provided that the cross sectional area of the top of the column is large in comparison to the volume of blood flowing, the increase in hydrostatic pressure due to the displacement caused by the bloodflow can be neglected.

A series of kymograph tracings with increasing pressure within the plethysmograph, i.e. decreas-
The occluding pressure (OP) measured by noting the height of a column of water, taking 13.6 cm water pressure as equivalent to 10 mm Hg pressure. The systolic blood pressure throughout was 100 mm Hg. The transmural pressures have been obtained by subtracting the occluding pressures from the systolic pressures.

FIG. 1A

The flow rates measured as the gradients of a trace, plotted against the systolic transmural pressure calculated as above.
CRITICAL CLOSING PRESSURES IN THE PERIPHERAL CIRCULATION

ing transmural pressure, is shown in figure 1A. If the bloodflow is plotted against transmural pressure the graph is approximately a straight line with a positive intercept on the pressure axis, as shown in figure 1B. These results are typical of those found by previous workers using the method of pressure plethysmography.

The evidence has been reviewed by Ashton (1963) who prefers to use the term "flow cessation pressure" rather than critical closing pressure in view of the uncertainty of the precise mechanism involved.

The possibility of critical closure has been quoted in discussions of the safety of hypotensive anaesthesia. Little (1957) said: "The clinical anesthesiologist must recognize the possibility that even moderate reductions of blood pressure may achieve the critical closing pressures of the small vessels of a vital organ in the individual patient." Armstrong Davison (1958), in a paper on the disadvantages of controlled hypotension in surgery, uses Burton's concept as part of his argument against this technique. Rollason and Hough (1960) writing on the safety of deliberate hypotension in the elderly suggest that the possibility of critical closure must be considered in the discussion of this question. Brierley and Cooper (1962) report the case of a previously healthy woman of 43 who developed Korsakow's psychosis following induced hypotension for mammoplasty. They give detailed histological findings of brain damage found on postmortem examination and, in the course of a general discussion on the causes of tissue ischaemia during hypotension, mention the possibility of critical closure in cerebral vessels, although suggesting that other factors might have been of greater importance in the case described.

There is a general impression that hypotensive anaesthesia is particularly hazardous in hypertensive patients. In view of this it is interesting that several workers have found raised values of critical closing pressure in hypertension (Burton and Yamada, 1951; Gaskell and Krisman, 1958; Coles and Gough, 1960; Ashton, 1962b). If critical closing pressure is plotted against the subject's arterial pressure the relationship appears to be linear. All these workers found that the high critical closing pressures in subjects with high blood pressure could be reduced by such procedures as nerve blocking, warming and reflex vasodilatation, although generally they remained higher than in normal subjects undergoing the same procedures. In the forearm, Ashton (1962b) found critical closing pressures between 10 and 68 mm Hg (mean 33.4) in the controls, and a range of 50 to 101 mm Hg (mean 66.8) in the hypertensive group. These results seem to support the commonly held view that induced hypotension is more dangerous in hypertensive patients.

All the work on critical closure in human vascular beds, however, appears to have been done on conscious volunteers. Since this concept has been used as an argument against hypotension during anaesthesia, it seemed desirable to measure critical closing pressures in anaesthetized patients by a method as close as possible to that used in previous work. By this means it was hoped to discover to what extent it seems justifiable to use results obtained during consciousness in discussions about anaesthetized patients.

METHOD

Apparatus.

The apparatus, shown in figure 2, is based on that of Burton and Yamada (1951). The plethysmograph is of perspex, with an outer air jacket to keep the temperature of the water constant. This it does within acceptable limits. The hand was enclosed in a surgical rubber glove cemented on to a rubber washer. Bulging of the glove out of the plethysmograph was prevented by a split ring made of bakelite. Severa}
Diagram of apparatus used. The hand is in a rubber glove cemented on to the rubber washer (A, shaded). A perspex plate (B), fixed with eight wingnuts, holds a bakelite split ring to stop the glove bulging. The venous occlusion cuff (C) lies just proximal to the plethysmograph, which is water-filled and surrounded by an air jacket.

The drip bottle is suspended by a graduated chain over a pulley (D). The side arm (E) on the righthand tube can be closed.

The float recorder (F) is of about 4 ml capacity and writes directly in ink.

0.5 ml/100 ml tissue/min. Flows of less than half this were easily recognizable. Ashton's apparatus was slightly more sensitive than this, flows of 0.2 ml/100 ml tissue/min being measurable (Ashton, 1962a). In preliminary trials on conscious volunteers this apparatus gave results similar to those of Ashton (1962a).

Procedure.

The plethysmograph was filled with water at 34° to 35°C. Since the investigation was concerned with closure of the vessels, in order to save time the first readings were taken at a fairly low transmural pressure. This was done by adjusting the height of the water column initially to the equivalent of 30 or 40 mm Hg. The side arm was open so that the pressure at the surface of the water in the drip bottle was atmospheric. The side arm was closed and the venous occlusion cuff inflated rapidly to 5 to 10 mm Hg above the plethysmograph pressure. The record was labelled and the side arm opened. The column was raised 13.6 cm, equivalent to 10 mm Hg, and another record taken as before. The column was raised by increments, decreasing as suggested by the records and previous experience until no flow could be detected. As soon as this happened the blood pressure was taken as quickly and accurately as possible and noted together with the height of the water column. The plethysmograph pressure was reduced to atmospheric between each set of readings. Each determination took about 5 minutes to perform.

Patients studied.

Forty determinations of critical closing pres-
sure have been made on twelve adult patients anaesthetized for elective surgery. The ages and operations are recorded in Table I. The range of pre-operative blood pressure was 120/80 mm Hg to 270/105 mm Hg. None was receiving hypotensive therapy. In order to reduce the number of variable factors estimations of critical closing pressure were only made in the earlier stages of anaesthesia in those cases where blood loss occurred or where long-acting relaxants and controlled respiration was used. At the time of investigation all patients were breathing spontaneously, none was showing clinical evidence of cardiovascular disturbance due to blood loss, and none was receiving a blood transfusion.

**Anaesthetic technique.**
All patients were anaesthetized by colleagues

<table>
<thead>
<tr>
<th>Age</th>
<th>Operation</th>
<th>Pre-operative blood pressure (mm Hg)</th>
<th>Systolic blood pressure at time (mm Hg)</th>
<th>Systolic critical closing pressure (mm Hg)</th>
<th>Anaesthetic agents in use</th>
</tr>
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<tr>
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<td>180</td>
<td>105</td>
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<td>90</td>
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<td>Sigmoidoscopy and piles</td>
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<td>155</td>
<td>60</td>
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T = Trichloroethylene.  
H = Halothane.
who were not concerned with the plethysmography. The patients were premedicated according to the standard practice of the anaesthetist administering the anaesthetic. Anaesthesia was induced with thiopentone followed by suxamethonium then, after inflation of the lungs with oxygen and topical analgesia to the larynx, intubation was performed. It was thought possible that there might be some difference between halothane and trichloroethylene with respect to alterations in critical closing pressure, so whenever it was felt to be clinically justifiable these agents were interchanged during the course of the anaesthetic.

There was no attempt to standardize the percentages of these agents. The vaporizer settings were determined by the response of the patient, the aim being to maintain smooth anaesthesia throughout. Halothane was generally used at 1 to 2 per cent. Trichloroethylene was used with the plunger of the Boyle's bottle fully up and the lever usually at about the halfway position.

RESULTS

The results are shown in table I. Applied pressures in excess of the diastolic blood pressure were required to stop flow in several cases. Yamada (1954) suggested that when this was so the transmural pressure should be calculated as

Transmural pressure =
Systolic arterial pressure – Applied pressure

The transmural pressure thus calculated at which flow ceased he called the systolic critical closing pressure, distinguishing it from the more usual calculation using the mean arterial pressure, the mean critical closing pressure. Systolic critical closing pressures have been used throughout here.

In every case the systolic critical closing pressure varied during anaesthesia. Broadly speaking, these variations were parallel to variations in systolic blood pressure. An alteration of systolic blood pressure was generally accompanied by an alteration of systolic critical closing pressure in the same direction, and approximately equal to the blood pressure change. Some examples are to be seen in figures 3 and 4.

In individual cases halothane gave the impression of lowering the systolic critical closing pressure, apart from its effect on the blood pressure. Similarly, in circumstances where vasoconstriction

![Graph showing approximately parallel variations of systolic critical closing pressure with blood pressure.](url)
Systolic b.p.  
Diastolic b.p.  
Systolic c.c.p.  

Time (min)

T - trichloroethylene  
H - halothane

Fig. 4
Showing the lowering of systolic critical closing pressure which seems to accompany halothane anaesthesia.

Fig. 5
Graph of systolic critical closing pressure against systolic blood pressure at the time of measurement. The forty points represent data from twelve different patients.
was to be expected, a rise in systolic critical closing pressure was generally seen.

Since repeated observations on the same patient during the same anaesthetic are unlikely to be completely independent, it may not be entirely accurate to combine the data. However, because the investigation is largely concerned with variations during the course of a single anaesthetic, it would seem reasonable to do this in spite of this theoretical objection. If this is done, a significant correlation is found between the systolic critical closing pressure and the systolic blood pressure at the time of measurement. There is a less obvious but still significant correlation between the systolic critical closing pressure and the pre-operative systolic blood pressure. Patients with high pre-operative blood pressure, however, are likely to have higher blood pressures during anaesthesia than patients with lower pre-operative blood pressure; and to disentangle these factors it is necessary to calculate partial regression coefficients.

Calculation shows that in the cases studied the best estimate of the systolic critical closing pressure is given by

\[
\text{Systolic C.C.P.} = 0.76 \times \text{(systolic B.P. at time)} + 0.091 \times \text{(pre-operative systolic B.P.)} - 62.8
\]

The partial correlation coefficient between the systolic critical closing pressure and the blood pressure at the time of measurement is 0.66 (highly significant, \(10^{-5} > P > 10^{-4}\)); and between systolic critical closing pressure and pre-operative blood pressure is 0.12 (not significant). In other words the systolic critical closing pressure depends on the blood pressure at the time, and not on the pre-operative level. Any apparent dependence on the pre-operative blood pressure in these present cases derives entirely from there being a correlation between pre-operative blood pressure and blood pressure during anaesthesia.

Figure 5 shows the relationship between systolic critical closing pressure and blood pressure at the time of measurement. A wider scattering of the readings during or within 15 minutes of halothane anaesthesia appears to be present.

**DISCUSSION**

The systolic critical closing pressure has been taken as the systolic blood pressure minus the plethysmograph pressure when flow was no longer detectable.

The apparatus used for flow rate measurement was about as sensitive as that used by Ashton (1962a) and gave similar results on conscious volunteers. The question of very small flow detection has been thoroughly discussed by Ashton (1962a). When the critical closing pressure is very low the plethysmograph pressure must be high. This means that the venous occlusion pressure approaches the systolic blood pressure. Landowne and Katz (1942) found that in both normal and hypertensive subjects the apparent flow with increasing cuff pressures did not diminish until a pressure above the systolic blood pressure was reached. Above this pressure (sometimes well above the systolic blood pressure) the apparent flow started to decrease, until eventually arterial obstruction was complete.

The pressure in the plethysmograph was easy to measure and the error was not more than a centimetre or so of water. Not all this pressure can be assumed to be transmitted to the tissues, however (Landowne and Katz, 1942). In the case of the hand, where the blood flow is mainly in the skin and the surface area large, this is not likely to be an important source of error.

Most previous workers, studying conscious subjects, have used the mean arterial pressure to calculate the critical closing pressure. It has been usual to calculate the mean arterial pressure from the formula

\[
\text{Mean arterial pressure} = \text{Diastolic pressure} + \frac{1}{4} \times \text{Pulse pressure.}
\]

If the mean critical closing pressure is thus calculated for the anaesthetized cases described here, however, eleven out of the forty readings give "negative critical closing pressures". This suggests that this method of calculation is not valid under the conditions of anaesthesia. Most previous workers have used the mean arterial pressure because it is the best estimate of the pressure in the arterioles, believed to be the site of critical closure. However, in vasodilatation, as Greene (1958) has pointed out, the arteriolar pressure is more nearly the same as the arterial pressure than is normally the case. Since hand flow is mainly in the skin, and vasodilatation of the skin a feature of any anaesthetic, it would seem more sensible to
use the systolic pressure, as was done by Yamada (1954) when the applied pressure exceeded the diastolic.

Systolic pressures have been obtained by auscultation. Steele's comparison of indirect readings and direct readings taken in the radial artery, quoted by Pickering (1955) showed that indirect readings underestimated the systolic and overestimated the diastolic pressures. This would tend to give too high values of the mean, and too low values of the systolic critical closing pressures in the work described here.

The correlation between systolic critical closing pressure and blood pressure at the time of measurement might be expected on theoretical grounds. The critical closing pressure will be determined by the tension in the arteriolar walls. The resistance to flow is determined by the same factor. Yamada (1954) has shown good correlation between the resistance of the peripheral vessels being studied and their critical closing pressures. Therefore, if the other factors determining blood pressure remain roughly constant, one would expect a correlation between critical closing pressure and blood pressure, close or otherwise according to the extent that the resistance of the hand vessels is representative of the total peripheral resistance. These theoretical considerations suggest that when hypotension is associated with vasodilatation, i.e. a relaxation of tension in the arteriolar walls, there might be a "built-in safety mechanism" in the form of a simultaneous lowering of critical closing pressure. The results presented in this preliminary study support rather than contradict this.

It is therefore suggested that in order to assess the possibility of critical closure during anaesthesia the critical closing pressure should be measured in anaesthesitized subjects, and the factors associated with its variations during anaesthesia should be studied.

**Acknowledgments**

I should like to express my thanks to Prof. H. D. Ritchie for permission to make these observations on his patients. I am grateful to Drs. A. I. Parry Brown, B. R. J. Simpson and J. V. I. Young for encouragement and helpful criticism. The apparatus was constructed by the staff of the research laboratories and physiology department of the London Hospital Medical College, whose help and advice were invaluable. The statistical calculations were performed by Dr. J. F. Scott, of the Unit of Biometry, Oxford.

**Appendix**

**Theoretical Considerations**

Burton considered the consequences of Laplace's law applied to arteries considered as elastic cylindrical tubes. Laplace's law states that in the case of a cylinder, if \( P \) is the transmural pressure (pressure inside the tube minus pressure outside), \( T \) the circumferential tension in the wall of the cylinder, and \( R \) the radius, then:

\[
P = \frac{T}{R}
\]

In the case of an artery Burton postulates three components of circumferential tension, \( T \):

1. The elastic tension, \( T_e \).
2. The active tension, depending on vasomotor tone, \( T_a \).
3. Surface tensions at interfaces, which he suggests can be neglected.

The tension in the wall of an artery will depend on how much it is stretched. The radius is a measure of the stretching. Arteries become increasingly difficult to stretch so that the graph of tension against radius shows an increasing slope, as in figure 6.

![Fig. 6](image)

The total tension, \( T \), is obtained from the elastic tension, \( T_e \), by adding the active tension, \( T_a \). On the graph this will appear as a shift up the tension axis a distance equivalent to \( T_a \); the shape of the curve will remain more or less the same since it is an expression of the mechanical properties of the artery. This is shown in figure 7.

![Fig. 7](image)

Laplace's law can also be represented as a graph of \( T \) against \( R \). This is done by rewriting it as \( T = PR \). This will give a family of straight lines through the origin whose slope is \( P \), whatever value of \( P \) is being considered.
If we wish to know at what radius an artery is in equilibrium for a given pressure and active tension, we can discover this, in principle at any rate, by considering the conditions of equilibrium. The condition imposed by Laplace's law is expressed by the line $T = PR$. The condition imposed by the tension in the vessel is expressed by the curve of figure 4. Equilibrium is only possible if both conditions are satisfied, that is at the points where the curve and the line intersect. There are three cases to consider.

1. There is no intersection, as at pressure $P$, in figure 8. No equilibrium is possible and the artery will be collapsed.

2. The line is a tangent to the curve, as $P$, in figure 8. The equilibrium is unstable, the least drop in pressure causing collapse of the artery.

3. The line cuts the curve at two points, as $P$, in figure 8. At point $A$ the slope of the curve is greater than that of the line. This means that slight variations of $P$ will be compensated for by greater variations of $T$, therefore the equilibrium is stable. At point $B$ the line has the greater slope and a slight fall in $P$ will lead to collapse of the artery, and a slight rise will cause it to expand to a stable point in the region of $A$.

Therefore only such points as $A$ represent possible states of the artery, and there is a minimum pressure below which the vessel will be collapsed. This pressure Burton called the critical closing pressure.

**REFERENCES**


**QUELQUES OBSERVATIONS DE PRESSIONS D'OCLUSION CRITIQUES DANS LA CIRCULATION PERIPHERIQUE DE MALADES ANESTHESIES**

Les pressions d’occlusion critiques ont été mesurées au niveau des vaisseaux de la main de malades anesthésiés pour intervention chirurgicale élective. La méthode utilisée était aussi semblable que possible à la méthode utilisée précédemment chez des sujets conscients. Dans ces circonstances les pressions d’occlusion critiques varient considérablement au cours de l’anesthésie. En général les variations de pression sont parallèles à celles de la pression sanguine systolique. On n’a trouvé aucun rapport entre la pression d’occlusion critique et la pression sanguine pré-opératoire. Voilà pourquoi on pense que les constatations recueillies sur des volontaires conscients...
ne peuvent être utilisées pour définir les possibilités d’occlusion critique en cours d’anesthésie; il paraît nécessaire de mesurer la pression d’occlusion critique au cours de l’anesthésie.

EINIGE BEOBACHTUNGEN ÜBER DEN KRITISCHEN VERSCHLUSSDRUCK IM PERIPHEREN KREISLAUF BEIM ANÄSTHESIERTEN PATIENTEN

ZUSAMMENFASSUNG


BOOK REVIEWS


Anaesthetists cannot but be interested in hypnosis for it is a procedure which is, at its least, a related, and at its most, a possible alternative to anaesthesia. These two books will be informative to those whose interest in hypnosis is more than peripheral. The first one demonstrates the complexity of the terminology now, in use and the extent to which this field is occupied by non-medical therapists. The book will make the browser, for this is hardly one for us to read, aware that, as the preface says:

"Contemporary findings and particularly the increasing emphasis upon motivational elements in hypnosis and their relationship to the psycho-physiological aspects of consciousness provide a basis for the recognition, description and eventual integration of the multidimensional variables that are grouped in the broad undifferentiated concept of hypnosis."

The second book, edited by Marcus, outlines the progress of hypnosis in the major countries of the world. As in the first, many of the authors are non-medical, including the one who deals with Great Britain. This section is naturally of particular interest dealing as it does among other things with the legal aspects of hypnosis including an account of the Hypnotism Act, and the activities of popular hypnotists such as Messrs. Casson and Slater which led to its introduction. The attitude of the churches is also discussed, as well as the directions in which new developments in hypnosis are taking place. Neither of these books can be other than spare time reading for the average anaesthetist.

W. W. Mushin

International Anaesthesiology Clinics. Cardiovascular Problems. Edited by John B. Stetson. Vol. 2, No. 1, 1963. Little, Brown and Company, Boston. Pp. 156. This volume is composed of the published lectures of the Greater Boston Anesthesia Teaching Conferences, the legitimate successors of a programme begun at the crest of World-War II interest in anaesthesiology. The earlier concentrations on the practice of anaesthesiology steadily gave way to a broad, graduate, continuing course which added a basic science foundation to the clinical features. The papers presented do not all come from the pens of anaesthesiologists, and the bright patchwork provided is well in keeping with the present-day variety in leading anaesthetic publications. The theme of this volume for 1963 is "Circulation" under the title of Cardiovascular Problems and the nine chapters follow this objective pretty closely though "Respiration" is never easily shouldered aside. Indeed one of the best chapters combines these functions and deals with the importance of controlled ventilation following open heart surgery. All the chapters cater for a mixed audience and there is a good blend of science and practical application, as might be expected. This makes this volume very informative and very readable to all members of an anaesthetic department or group, containing among others, chapters on Temperature Regulation and Anaesthesia; The Development of Cardiac Resuscitation; The Effects of Minute Transfusions of Blood and Blood Preservatives. For those anxious to read further most chapters conclude with a bibliography, one of which contains no less than 128 references.

It is noteworthy that in the article on Cardiac Resuscitation where the use of carbon dioxide mixtures in the treatment of carbon monoxide poisoning is described, no mention is made of the value of hyperbaric oxygen in these circumstances. On pages 106 and 107, the captions under the figures have been transposed. The book is well produced, with a helpful index and in itself provides easy and yet informative reading.

H. H. Pinkerton