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J. A. MACWILLIAM, M.D.

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"On the Properties of the Arterial and Venous Walls."*

By J. A. MACWILLIAM, M.D., Regius Professor of Physiology in the University of Aberdeen. Communicated by Sir M. FOSTER, Sec. R.S. Received October 24,—Read November 28, 1901.

(From the Physiological Laboratory of the University of Aberdeen.)

Post-mortem *Contraction of Arteries.*

So long ago as 1891 I noticed that the excised carotid artery of a recently-killed ox exhibits a strongly-marked and long-persisting contraction of its muscular wall. The phenomenon is, of course, most easily studied in the arteries of the larger animals. In this investigation I have examined arteries from thirty-five oxen and a considerable number of horses, sheep, cats, and men.

When the carotid of the ox or horse is exposed immediately after death, it is found to be soft and flaccid, and more or less flattened in section, with a large bore, usually 5—6 mm. On exposure to the air, cooling, manipulation, cutting, &c., the artery speedily becomes rigid and contracted; the tube becomes circular in section, and its calibre is greatly reduced, *e.g.*, to an internal diameter of 2—3 mm., or less. The artery becomes so stiff that a piece 7 or 8 cm. long may

* A statement of the chief conclusions arrived at in this investigation was communicated to the Physiological Society (at Edinburgh) on July 20, 1901.

often be held out (by one end) in an almost horizontal position. These changes may be seen 10—15 minutes after death or many hours later; they occur in all systemic and pulmonary arteries that have well-developed muscular coats. An artery excised shortly after death may be strongly and persistently contracted long before *rigor mortis* has appeared in the heart or skeletal muscles, and indeed while these muscles are still obviously living.

Causes of Post-mortem *Contraction.*

(1.) Mechanical stimulation has a powerful influence, *e.g.*, cutting, manipulation, &c. When an artery is cut across, contraction begins at the cut, and thence spreads along the tube more or less completely; an excised piece of artery some inches long often shows very well-marked contraction near both ends, while the middle part may be relatively soft and relaxed; an incision made in this middle part speedily induces marked contraction there also.

Pieces of excised artery show considerable variation in the extent to which the contraction excited at the cut ends involves the intermediate portion of the tube.

(2.) Cooling to a few degrees above zero favours the development of contraction in an exposed artery. Warming a contracted (cold) artery up to about 40° very commonly has a markedly relaxing effect, though the relaxation is usually incomplete; a piece of contracted artery put into defibrinated blood and kept in a warm chamber for an hour or so usually relaxes to a considerable extent—at least, if the experiment is done within a day or so after death, the artery being excised shortly after death; it may again contract very markedly when the temperature falls.

(3.) Exposure to the air also seems to play a part in inducing and favouring *post-mortem* contraction.*

When a piece of artery is excised as speedily as possible after death and at once immersed in olive oil while still flaccid, and kept in the oil, *post-mortem* contraction is, as a rule, much less strongly marked, though it may be extremely long-continued.

Again, when a relaxed artery is exposed immediately after death and covered over with olive oil, cutting into the arterial tube while it is immersed in oil causes, as a rule, a decidedly less pronounced contraction than usual.

Post-mortem contraction is well marked in all arteries, pulmonic as well as systemic.

In the lungs of an ox an hour after death I found, on making deep

* In the case of a living artery *in situ* (posterior tibial), John Hunter observed that the vessel contracted very much on being exposed to the air for some time. Cooling may possibly have played some part here. 'Works' (London, 1837), vol. 3, p. 157.

incisions, branches of the pulmonary artery uncontracted and flaccid, but on excising portions these speedily became contracted, so that a piece of artery 4 cm. or more in length could be held out in a horizontal position by one end. Later—3 hours after death—deep incisions into the lung substance showed the small arteries to be markedly contracted and firm, their cut ends projecting somewhat on the surface of the incision. Branches of the pulmonary artery 3 mm. in diameter when relaxed were found to be less than 2 mm. when contracted.

Small branches arising from the carotid are often apparently more firmly contracted than the carotid itself.

Arteries left undisturbed *in situ* for 24—48 hours after death, and then exposed, may show little sign of contraction at first, but under the influence of manipulation, cutting, exposure to air, &c., may soon enter into strong contraction—persisting for days.

The importance of the presence or absence of *post-mortem* contraction need hardly be insisted on with regard to the measurements of arteries made after death to ascertain the size of lumen, the thickness of wall, tunica media, &c.

Changes in Length of Empty Excised Artery in Contraction and Relaxation.

When an empty excised artery contracts its length becomes increased, and conversely on relaxing the tube shortens. Thus two portions of artery (ox), measuring 25 mm. and 31 mm. in length, while strongly contracted became shortened to 20 mm. and 27 mm. respectively when the muscular contraction had relaxed to a great extent.

Duration of Post-mortem *Contraction.*

In the arteries of the ox and horse *post-mortem* contraction usually lasts for several days; the usual duration of obvious contraction is about 5 or 6 days, when the artery is kept in an ordinary room. In a warm room contraction passes off earlier, and in a cool room its duration is considerably prolonged. When placed in a warm chamber (in defibrinated blood) at about 40° C., the contracted artery is found to become completely relaxed in 24 hours or less; its walls become flaccid, and its lumen enlarges from a diameter of perhaps 2 or 3 mm. to one of 5 or 6 mm. Subsequent stimulation (mechanical, chemical, electrical, &c.), cooling, and exposure to air, entirely fail to induce any contraction. There is no evident change in reaction to litmus paper.

Immersion in olive oil exercises a powerful influence in prolonging the duration of *post-mortem* contraction. In the carotid of the horse so treated I have seen an appreciable amount of contraction present as

long as a fortnight after death; a very slight residuum of contraction has been found even 18 days after death.

Persistence of Excitability in Arteries after Death.

The excised artery of a healthy animal kept at ordinary room temperature either in defibrinated blood or simply in a bottle moistened with salt solution, commonly reacts to certain modes of stimulation for very many hours after death, often for 2 or 3 days. This is very clearly evident when one makes a transverse incision across an artery which has not shown much contraction after death, but has remained with only a slight development of rigidity in its walls and with a relatively large lumen (*e.g.*, 4—5 mm.); soon after the arterial tube is cut across contraction begins—slowly and gradually—to manifest itself, the walls of the vessel become firm, and the lumen diminishes, so that in 5 minutes the diameter may be reduced to about one-half (2—3 mm.). In such cases decided contraction is usually evident in a minute or two after the cut has been made, though it does not attain its full development for some little time.

Again, it is often seen that when an artery has gone into the contracted state soon after death, it may relax to some extent in a day or two, and then a fresh incision, 2 or 3 days after death, may lead to well-marked contraction. This I have observed most frequently in the arteries of the horse.

Gentle manipulation of the artery with the fingers also tends to promote the development of the contraction. Strong galvanic currents induce contraction. Faradic currents, unless very strong, have little or no effect a day or two after death, when other forms of stimulation are quite effective.

Exposure to chloroform vapour is a powerful agency for causing contraction. This is readily demonstrated by putting a piece of artery into a tightly corked or stoppered bottle containing a little chloroform, the artery being supported in such a position as to obviate any contact with the liquid chloroform while it is freely exposed to the vapour. When so treated, an artery which is only slightly and partially contracted a day or two after death shows a very striking change. Its walls soon become firm, while its lumen becomes greatly contracted, *e.g.*, from a diameter of 4—5 mm. to one of 1·5 or 2 mm.; the surface of the artery assumes a whitish appearance.

The contraction so induced may persist for a long time or it may diminish appreciably in an hour or two. In the latter case a fresh exposure to chloroform vapour may again cause contraction. Much depends on the length of time the chloroform vapour is allowed to act; prolonged action of chloroform induces a permanent change in the arterial wall.

Even so long as 4 days after death the carotid of a horse was seen to contract from a diameter of 5 mm. to one of 3 mm.

Suprarenal extract has a marked influence in inducing arterial contraction even at relatively long periods after the death of the animal, *e.g.*, 24—48 hours, or longer. An excised portion of artery placed in a watery decoction (made with normal saline) of suprarenal medulla speedily goes into strong contraction, persisting for a day or two; at length the contraction passes off. As might be expected the contraction is excited more effectively in a relatively thin-walled artery like the carotid of the sheep than in the thick-walled artery of the ox or horse. Decoctions of suprarenal medulla made with tap water, as well as with normal saline, were tried.

It is to be noted that both tap-water and normal saline, by themselves, have a very appreciable effect in inducing contraction in an artery for some time after death. But these effects are much more transient than those produced by suprarenal extract; the contraction following the application of normal saline or water commonly passes off within 2 or 3 hours, while that induced by suprarenal extract is usually well marked till the following day or even later.

When a certain period, varying according to circumstances, has elapsed after death, an excised artery, though still showing marked contraction, fails to respond appreciably to any form of stimulation.

Prevention of Post-mortem *Contraction* (*by Freezing*).

Freezing a piece of artery immediately after excision from a recently-killed animal prevents the development of contraction altogether if the artery be taken out speedily while still flaccid and at once frozen for a sufficient time. When the artery is at length allowed to thaw it remains permanently relaxed, with large bore (5—6 mm). To bring about this result, 4 hours' freezing suffices in all cases; very often much shorter periods ($\frac{1}{2}$, 1, 2, 3 hours) are enough. In these experiments care must be taken to prevent any of the mixture gaining access to the artery. Arteries so treated respond to no form of stimulation. There seems to be no appreciable change in the reaction of the cut surface of an artery (to litmus paper) after freezing.

There is some evidence tending to show that pieces of artery taken very early after death may be prevented from going into contraction by shorter periods of freezing (*e.g.*, 30 min., &c.) than is necessary with pieces taken later.

Freezing one end of a segment of uncontracted artery (excised immediately after death) for some hours causes that end to remain large in calibre and relatively flaccid, while the other end which has not been frozen goes into marked contraction and usually remains contracted for days.

Periods of freezing insufficient to entirely obviate the appearance of *post-mortem* contraction may cause it to be much less pronounced.

When a portion of artery is rapidly excised immediately after death and quickly cooled to about 0° (while still uncontracted) in a suitable metal vessel placed in ice for some hours, the onset of contraction is usually delayed though not ultimately prevented; in some cases its development seems to be very imperfect until the artery is stimulated (manipulation or cutting, &c.).

When contraction has been established for some time (*e.g.*, a day or two) cooling down nearly to 0° for some hours has no appreciable effect.

Removal of Post-mortem *Contraction.*

Freezing.—An artery, however firmly it may be contracted, can be made to relax by freezing it for some hours in the way already mentioned as being effective in preventing the development of contraction. The exact time required varies in different cases, but I have always found 4—5 hours sufficient. On being allowed to thaw, the lumen enlarges to the usual size of the passive artery, while its walls become soft and relaxed. The contractility of the artery is completely and permanently abolished. Its length is less than in the contracted state. There is no apparent change in reaction of the cut surface to litmus paper.

It often seems to require a less prolonged period of freezing to obviate the appearance of contraction in an uncontracted artery than to remove contraction when it has been established for some little time (1 hour, &c.).

Sulphocyanide.—Immersion of a contracted artery in a solution of sulphocyanide of potassium soon leads to complete relaxation. When a 20 per cent. solution is used, relaxation is generally found to have occurred in 20—30 minutes, the time varying with the thickness of the arterial wall, &c. Weaker solutions require longer time. Sulphocyanide of ammonium has effects similar to the potassium salt. The effect may be graphically recorded in the following way:—A strip of arterial wall cut transversely to the long axis of the vessel is suspended in a bath of the solution and made to pull upon a recording lever, which is kept in a horizontal position by a long, feeble, spiral spring. The tracing obtained shows the extensive relaxation which occurs after the sulphocyanide is introduced into the beaker. A skeletal muscle placed alongside the arterial strip in the bath and connected with a similar lever shows a striking contrast, contracting strongly under the influence of potassium sulphocyanide, as Kühne* described many years ago. Here we have the remarkable result that while potassium sulphocyanide induces strong and persistent contraction in

* 'Myologische Untersuchungen,' p. 130.

skeletal muscle, it (or the ammonium salt) causes speedy and complete relaxation of the contracted arterial muscle (fig. 1). This relaxing effect is much more powerful and rapid than that which follows immersion in saline solutions, such as ammonium chloride (13 per cent.), which dissolve out the muscle-proteid, and the mode of action seems to be different, for the sulphocyanide solution seems to extract very little proteid from the arterial wall when it has acted sufficiently long

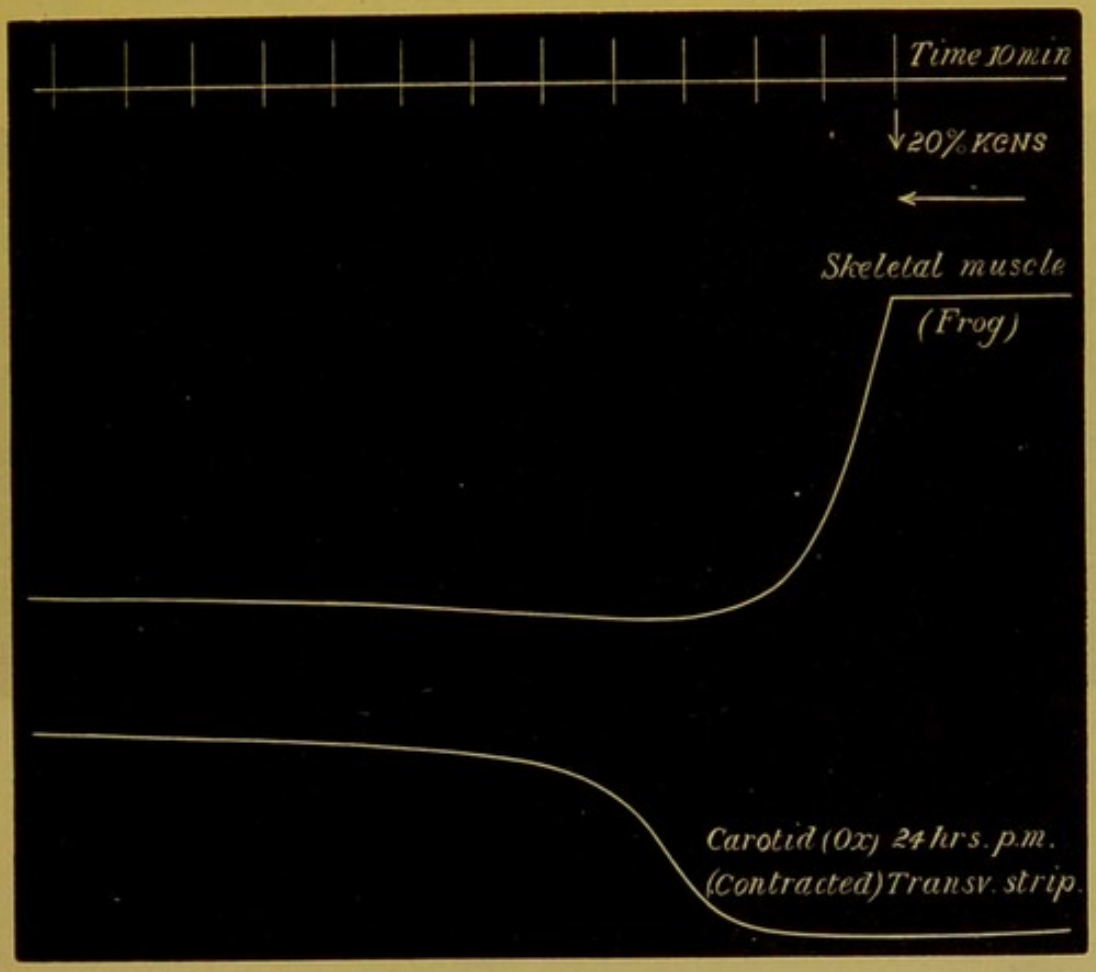

FIG. 1.

to cause complete relaxation; on the other hand, sulphocyanide causes precipitation of proteid in a saline extract of the arterial wall. In contrast with this, saline fluids like the ammonium chloride solution extract a large amount of proteid before they effect complete relaxation, and many hours are necessary for the completion of the effect (*e.g.*, 24 hours). Magnesium sulphate solutions (5 per cent.) require days.

When relaxed by sulphocyanide the artery shows a permanent loss of excitability and contractility.

Ammonia Vapour.—A contracted artery exposed to strong ammonia vapour (in the same way as described in the case of chloroform vapour) speedily begins to relax; in a very few minutes the change is well

marked, and in 10 or 15 minutes relaxation is usually complete, even in a thick-walled tube like the carotid of the ox.

Graphic records were made of the action of ammonia vapour by an arrangement similar to that used with the sulphocyanide solution. Fig. 2 shows the effect upon a transversely cut strip of the wall of a contracted artery, and upon a recently excised skeletal muscle (frog's gastrocnemius). The upward movement of both levers indicates relaxation—very extensive in the case of the artery. The ammonia vapour was applied at the moment of the first time signal.

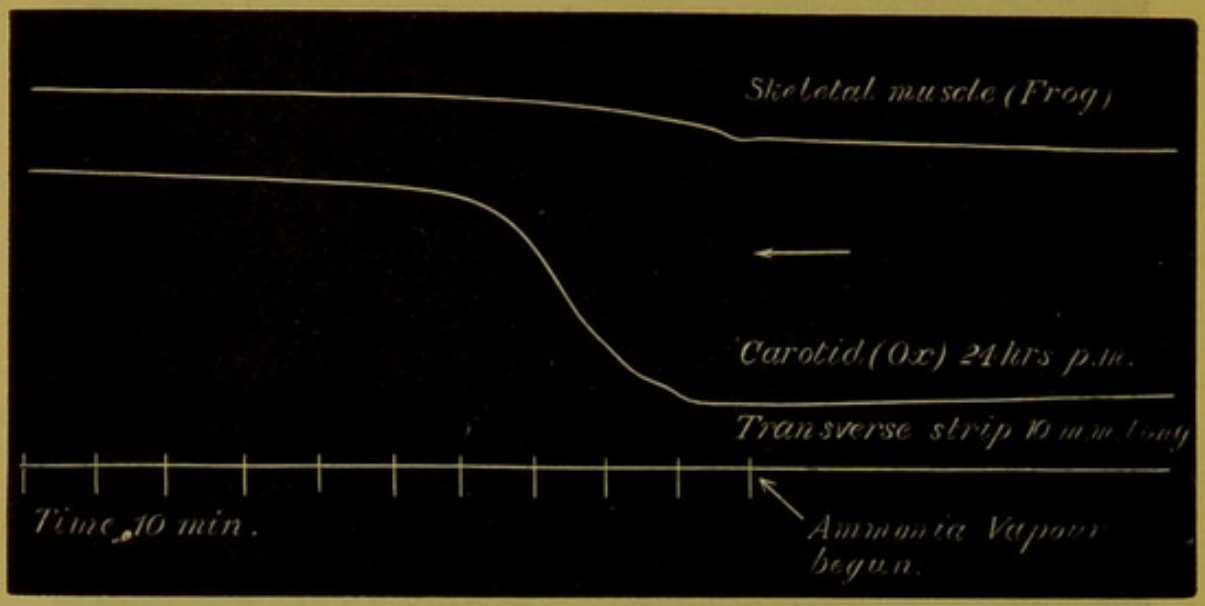

Fig. 2.

It is to be noted that when a strip of artery is suspended in this way it often contracts slightly prior to the application of any reagent —apparently a response to the stimulus of stretching or of compression of its ends by the clamps which hold it, or a combination of these causes.

Heating.—Keeping an artery at about body temperature for a number of hours (*e.g.*, 24) causes the contraction to pass off, as has already been stated.

Heating to about 50—55° C. induces complete relaxation in a few minutes. The effects of heat will be described in detail later.

Kneading, Rubbing, Stretching.—More or less extensive relaxation of a contracted artery can be speedily induced by kneading or rolling the vessel between the fingers for a minute; the wall becomes much less firm, and the lumen opens up markedly—*e.g.*, from a diameter of 1·5 mm. to twice that size or more; later it may again contract to some extent.

Mayo* observed that an artery exposed in a living animal became

* 'Outlines of Physiology,' London, 1837.

markedly relaxed when it was rubbed between the finger and thumb for a minute or two; this relaxation passed off after a time.

Somewhat similar results occur when a contracted artery is forcibly stretched by the introduction of a glass rod 5—6 mm. in diameter. Partial recovery may occur. Probably the arterial wall is injured by the very considerable amount of force required to overcome its contraction in this way.*

Effects of Heat upon Arteries and Veins and upon Saline Extracts.

I have studied the effects of heat upon the arterial wall in different ways—

(1) By direct examination and measurement of portions of excised artery, heated to various temperatures and at various rates while immersed in defibrinated blood (of the same animal) or in olive oil, or simply kept in a bottle or test-tube moistened with normal saline solution.

(2) By obtaining graphic records of the changes in length of strips of the artery, cut either transversely or longitudinally, under the influence of various temperatures. The arterial strip was placed in a bath of olive oil, or defibrinated blood (when relatively low temperatures were all that was necessary); the strip was rigidly fixed at one end, while the other was connected (by means of a fine wire) with a recording lever, suspended in the horizontal position by a long feeble spiral spring, and writing upon a slow drum; the bath was gradually heated up by a spirit lamp or Bunsen burner, usually at about the rate of one degree Centigrade per minute, though the precise rapidity of

* I have very recently found some references in John Hunter's writings, which may be quoted in connection with the subject of *post-mortem* contraction. He describes an experiment on the human umbilical cord, in which he found that when the arteries were cut across 2 days after delivery the lumen was found to be closed 24 hours afterwards; this did not occur when the section was made 3 days after delivery. The experiment was performed on a portion of the cord left attached to the placenta. Hunter also refers to the arteries being contracted after death by hæmorrhage: he speaks of the "stimulus of death," and describes the "contraction by death" as being less in the aorta than in more distant vessels ('Works,' London, 1837, vol. 3, pp. 158 and 168). In his 'Essays and Observations' (London, 1861), vol. 1, p. 133, Hunter mentions an experiment on the uterus and its vessels: "I injected the uterus of a cow that had been separated from the body of the cow about 24 hours; and I found next day that it had contracted very much, and that the vessels had also contracted, for the great trunks were more turgid than when injected, so that the injection had been squeezed back again. This also shows that the small vessels have a greater or longer power of contraction than the large ones."

In a footnote on the same page, Clift comments on the above experiment: "May not this be the effect of elasticity in consequence of the parts having been put into hot water while being injected? I have seen that happen."

heating was varied within pretty wide limits and the temperature was often kept steady at various levels for considerable periods.

In many experiments the oil bath was not heated directly by the lamp; it was immersed in an outer beaker of water, which was placed on a piece of gauze and heated in the way mentioned. In most cases two strips were heated simultaneously in the bath, and made to record on the same drum. The temperature was determined by a thermometer fixed in the oil-bath with the bulb close to the arterial strips. A time record usually showing periods of 5 minutes was simultaneously inscribed. Shortening of the arterial strips is indicated by a downward movement of its lever. The magnification was generally three times.

In many experiments the recording lever was directed at right angles to the circumference of the drum, so that its point moved in a straight line perpendicular to the direction of movement of the recording surface —not in an arc of a circle like a lever used in the ordinary way. Ordinates were drawn through the marks in the time tracing; at one end of each ordinate the exact time was inscribed, at the other end the temperature at that moment.

Instead of spiral springs small weights (*e.g.*, 1—2 grammes) were sometimes employed to maintain tension of the arterial strip during the experiment.

Slight contraction may occur before the heating has begun, and shortly after the strip has been suspended—probably due to the mechanical stimulus supplied by compression of each end of the strip by the clamp which holds it, perhaps assisted by the slight tension of the strip when connected with the recording lever.

Poisseuille* stated long ago that the force of reaction excited by distension of an artery was greater than the force used to distend it. And Bayliss† has recently described a number of experiments upon the reaction of intact arteries (isolated from the central nervous system) to the distending force of a raised blood pressure.

Frequently pieces of excised artery (unopened)‡ 2—3 cm. long were suspended in the bath alongside the arterial strips, and examined from time to time, so as to correlate the changes seen in the unopened tube with those shown graphically by the transverse or longitudinal strips. The results obtained by these two methods were entirely concordant. Experiments were made on arteries with strong muscular coats like the carotid, and also on the aorta and pulmonary artery.

Experiments on the Carotid Artery.—Heating an artery produces very

* 'Journal de Physiologie,' par M. Majendie, vol. 8, p. 272. See note in John Hunter's 'Works' (London, 1837), vol. 3, p. 157.

† 'Journal of Physiology,' vol. 26, "Proceedings of Physiological Society," p. 29.

‡ This means excised segments of artery not laid open longitudinally, though open at the ends.

different effects, according to the state of the artery at the time—whether it is contracted or relaxed; in the case of the contracted artery there are also certain differences in its behaviour at the early and the late stages of contraction.

Relaxed Artery.—An artery in a state of relaxation behaves in essentially the same way when heated, whether its relaxation is due to (*a*) the gradual passing off of *post-mortem* contraction after some days, or (*b*) to being kept at about body temperature for 24 hours, or (*c*) to its having been frozen for some hours, or (*d*) to treatment with potassium or ammonium sulphocyanide solution, or (*e*) exposure to ammonia vapour. When the temperature is gradually raised there is no important change, though slight shortening may gradually occur, until it reaches 60—65° C., when a well-marked and commonly very extensive contraction takes place; this is seen whether a transverse or a longitudinal strip is recorded, and the change is evident in an unopened segment of artery, suspended in the bath—the lumen becomes very markedly diminished, while the arterial wall becomes much firmer to the touch. When the shortening has been completed, and the temperature is allowed to fall, there slowly occurs a certain amount of lengthening, but this is always very incomplete. (Fig. 3.)

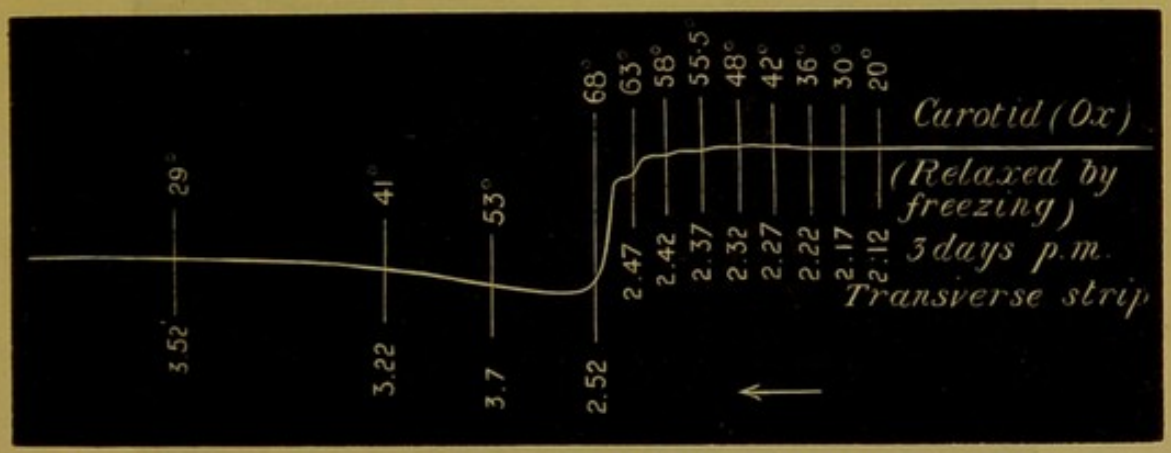

FIG. 3.—Artery (ox) relaxed by freezing. Transverse strip, 10 mm. long; load, 2 grammes. The minor oscillations preceding the extensive descent of the lever, beginning at 61°, are more distinct than usual; they are often entirely absent. Carotid (horse) relaxed by freezing. Transverse strip, 18 mm. long.

This characteristic heat-contraction at 60—65° seems to be due to the elastic and connective tissue elements of the artery, and not to depend essentially on the properties of the muscular coat. For it is well seen in pieces of artery which have had their muscle-proteids almost completely extracted by prolonged maceration in large quantities of saline fluids (ammonium chloride 13 per cent., &c.). It resembles the contraction got from a piece of tendon or of ligamentum nuchæ (ox), placed in the bath and recorded by the same apparatus.

A slender tendon (cat) was found to show extensive shortening at about 63°. Hermann* found that tendon began to contract at 65° and finished at 75°. Brodie and Richardson† describe the contraction as beginning at 60° and being complete at about 64°.

Gottschlich‡ found that a piece of ligamentum nuchæ shortened on heating and lengthened on cooling for all temperatures up to as high as 65°. Another shortening, not removed by cooling, developed between 65° and 75°.

It may be mentioned that a frog's muscle tested with the apparatus I have used for the arterial strips, shows very clearly the three heat-contractions studied by Brodie and Richardson§ with the aid of a photographic method at about 34°, at 45—50°, and at 55°—60°.

Contracted Artery.—When an artery which is in a state of *post-mortem* contraction is gradually heated, it undergoes a characteristic change at a temperature of 50—55° C.—sometimes a little below and sometimes a little above this level. It becomes relaxed, as is shown by the tracing given by a transverse strip; the rise of the lever indicates a marked elongation of the arterial strip (fig. 4); the same change is

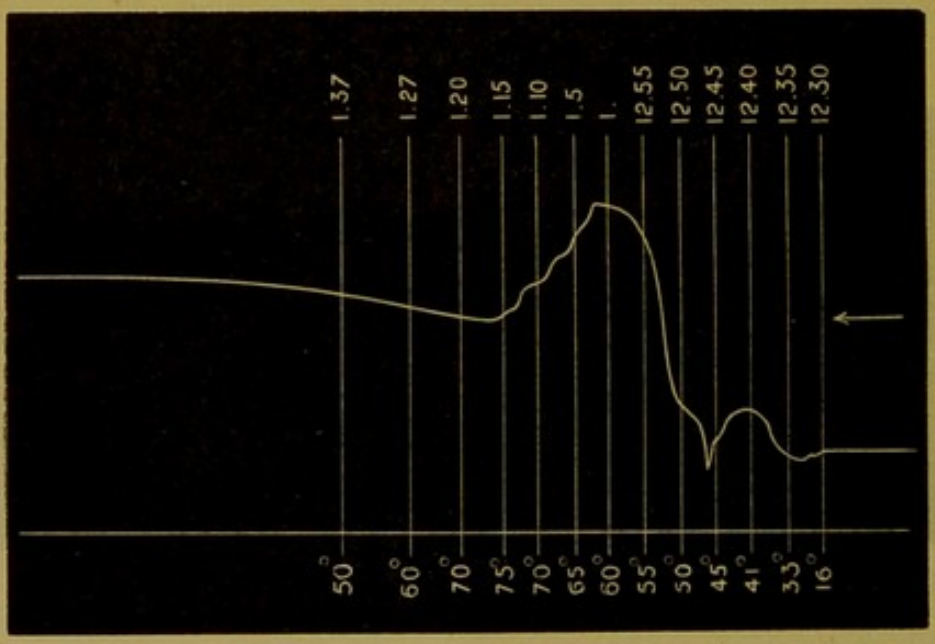

FIG. 4.—Carotid (ox). Transverse strip; 25 hours *p.m.* Load under 1 gramme.

evident in a piece of unopened artery by a notable enlargement of the lumen, while the walls of the tube lose their firmness. When this relaxation has occurred, a subsequent lowering of temperature does not restore the contracted condition. Excitability and contractility are absent after the temperature has been raised to about 50°. It is important

* 'Pflüger's Archiv' (1873), vol. 7, p. 417.
† 'Phil. Trans.,' B, vol. 191, p. 127.
‡ 'Pflüger's Archiv' (1893), vol. 54, p. 109.
§ *Loc. cit.*

to note in regard to the relaxation beginning at 50°, that the arterial strip is subjected to only a very slight amount of tension during the experiment—the spiral springs employed being long and weak; the tension may be regarded as being nearly constant throughout. Instead of springs, weights of small amount (2 grammes) were sometimes used. In the case of the unopened artery where no tension is employed, the only force (outside the muscle fibres) to cause an expansion movement is the elasticity of the arterial wall, which tends to make it assume the position which it occupies in the passive artery.

The extent of the relaxation occurring at 50—55° varies with the amount of contraction present at the time the temperature reaches that level.

When relaxation of contracted artery has begun at about 50° to be completed, perhaps by the time the gradually rising temperature has reached 58—60°, then a further rise to 60° or 60—65° leads to the appearance of the contraction already described in the case of a relaxed artery. (Figs. 6 and 7.) When the process of relaxation has not been completed at the time the changes underlying the 60—65° contraction begin, the latter is often much more slightly marked in the tracing than usual, the tendency to shortening being probably opposed and partially masked by the relaxation which is still going on. (Fig. 11.)

While relaxation at about 50° C. is the most constant and outstanding feature in the behaviour of a contracted artery when heated, there are other important features which vary according as we are dealing with (*a*) the earlier stage of *post-mortem* contraction (a few hours or a day or two after death), or (*b*) its later stage.

(*a*) During the earlier stage—while the artery is still excitable—heating commonly induces important changes long before the level of 50° is reached—often indeed pretty soon after the rise of temperature has begun. The tracing given by a transverse strip may show large curves indicating marked changes of length; there is commonly a curve before 40° is reached, and one after 40° before the extensive relaxation beginning about 50°. Contraction often begins at about 25° to reach its maximum about 35° and then relax; at 40° relaxation is usually well marked. As the temperature rises, a further contraction (sometimes very extensive) takes place, especially between 45° and 50°, and often particularly marked about 47°, to give place about 50° to relaxation. At 60—65° contraction again occurs—to relax slowly and partially when the temperature falls. (Figs. 4 and 8.) Thus when a transversely cut strip of artery from a recently killed animal is heated up to or beyond 65° and then allowed to cool, there are usually three phases of shortening and three phases of lengthening; the first shortening at 25—35°, the second at 45—50°, the third at 60—65°; the first lengthening at 35—45°, the second between 50°

and 60°, and the third during the period of cooling after the temperature of 65° has been reached. These broad features in the behaviour of the arterial strip are remarkably constant in their appearance and behaviour, though there is much variation in detail.

The extent of the first and second shortenings varies much in different arteries and at different periods after death. Commonly they show as quite large curves in the tracing, the shortening in each case being followed by lengthening. The elongation following the first shortening at 25—35° usually reaches its maximum at about 40° or a little higher, when the temperature is steadily raised. Cooling down to 35—25° does not restore the contraction seen at that phase when the temperature was being raised; evidently the 25—35° contraction is not a shortening conditioned simply by the presence of a temperature of 25—35°, but excited by a rise to that level. (Fig. 9.)

When the artery has been kept some time longer (days) before being heated, the contractions (α) at 25—35°, and (β) about 47° gradually become lessened and at a later phase disappear altogether; (β) persists longer than (α) in many arteries. (Figs. 7 and 11.) The tendency to relaxation about 40° is often seen to outlast both the contraction curves (α) and (β), though slight in amount compared to the relaxation at 50—55°, into which it often grades. (Fig. 5.)

(*b*) During the later stage (*e.g.*, several days after death) the artery—still more or less contracted—may show no very striking change till a temperature of about 50° is reached, though there is often slight relaxation beginning at about 40°. The usual 50—55° relaxation leads to a complete abolition of such contraction as is present. (Fig. 5.)

When an artery from a recently-killed animal is placed and kept in olive oil, the duration of its contraction is greatly prolonged, so that very many days after death marked relaxation occurs when a transverse strip is heated to 50—55°. As the contraction gradually diminishes after a number of days, the amount of relaxation obtained on heating lessens.

In the carotid of the horse, distinct relaxation may occur at 50—55° as long as 14 or even 18 days after death—though very much less extensive than on earlier days, when more contraction was present in the artery.

Later, when contraction has quite gone, no sign of elongation is seen at all on heating.

Fig. 5 shows the relaxation that occurs in a transverse strip of horse's cartoid kept in olive oil for 10 days *p.m.* The contraction present in the early days had become greatly diminished by that time. Fig. 6 shows that very slight but still appreciable relaxation occurred 18 days after death in the same artery; there had still been a very small amount of contraction persisting.

Tracings made 14 days and 16 days *p.m.*, showed an amount of relaxation less than is seen in fig. 5, but greater than in fig. 6.

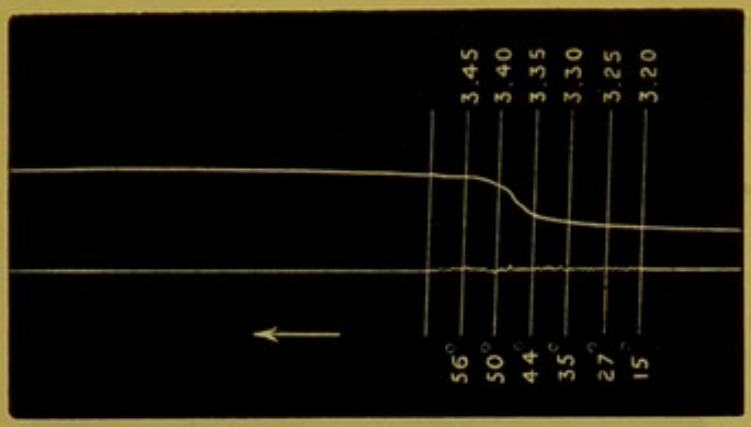

FIG. 5.—Transverse strip, carotid (horse), which has been kept in olive oil for 10 days after death.

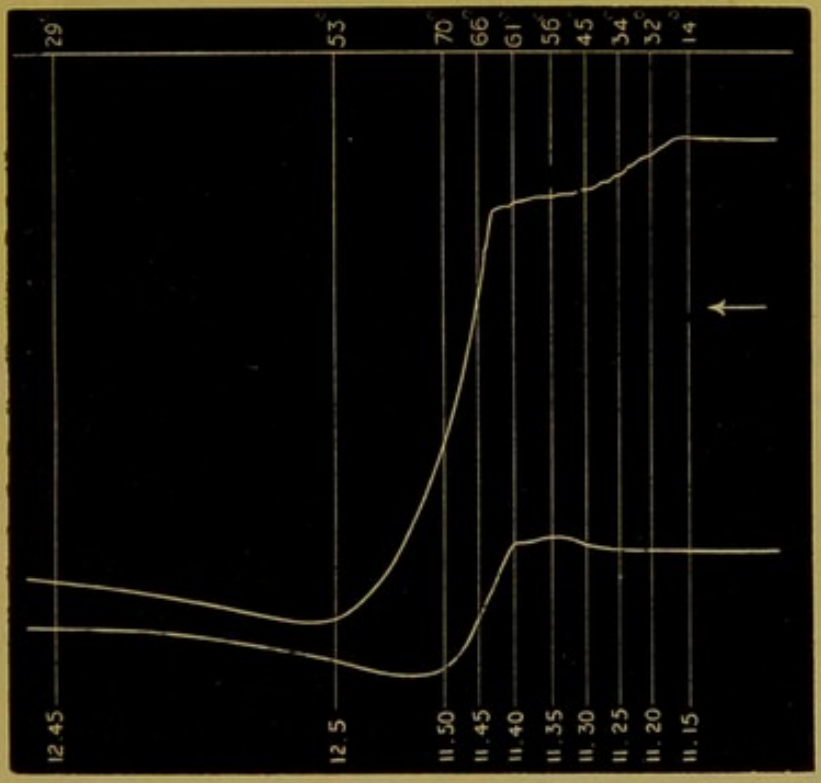

FIG. 6.—Upper tracing from transverse strip of jugular vein. Lower tracing from horse's carotid kept 18 days in olive oil.

Strips cut from a contracted artery parallel to the long axis of the vessel behave, when heated, very differently from transversely cut strips. Indeed, the former show alterations in length which are—up to a certain temperature the converse of those shown by the transverse strip, the contractions of the transverse strip between 20° and 35°, and between 40° and 50° are accompanied by elongations of the longitudinal strip—less in amount it may be, but coinciding pretty accurately in

time. Similarly the lengthening of the transverse strip about 40° and between 50° and 60° is accompanied by a certain amount of shortening of the longitudinal strip. Fig. 7 shows the relative behaviour of the two strips when heated up to about 58°.

But this converse relation ceases as the temperature rises; both strips contract markedly at 60—65°.

(3) Another method of studying the influence of heat upon the arterial wall is to use a segment of artery, one end of which is closed by tying in a wooden plug, while into the other end is fastened one extremity of a long glass tube of small bore. The interior of the artery and the glass tube for part of its length are

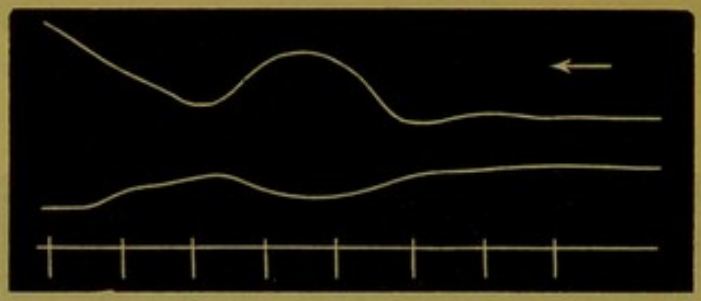

Fig. 7.—Upper tracing, transverse strip of contracted artery (carotid of ox). Lower tracing, longitudinal strip of same artery. The phases of elongation (35°—45° and 50°—58°) in the transverse strip are coincident with phases of shortening in the longitudinal strip and *vice versâ*.

filled with olive oil, and a graduated millimetre scale is placed behind the tube so that the position of the column of oil may be acurately read off. The tube is bent at right angles not far from the artery, so that the main part of the tube (with the scale) may be fixed in the horizontal position while the artery is placed vertically in an oil bath, the temperature of which is then raised in the usual way. Contraction and relaxation of the artery are indicated by the advance or retreat of the oil in the horizontal tube. The results are entirely confirmatory of those obtained with transversely cut strips from the same artery.

Effects of Temperature Changes on some other Unstriped Muscles of Mammals.

The behaviour of the contracted arterial muscle presents a notable resemblance in many respects to what is seen in some other unstriped muscles which have been examined in the living condition.

The retractor penis muscle* retains its vitality long after removal

* Eckhard, 'Beiträge zur Anat. u. Physiol.,' vol. 3, p. 123 (1863); Langley and Anderson, 'Journal of Physiology,' vol. 19, p. 85; Starling, in 'Schäfer's Text-book of Physiology,' vol. 2, p. 349 (1900); Sertoli, 'Archives Italiennes de Biologie,' vol. 3, p. 78; Schultze, 'Arch. für Physiologie,' 1896, p. 54.

from the body. Sertoli found its excitability to persist 5, 6, or even 7 days after excision—especially when kept in blood-serum at a temperature of 5—8° C. The muscle shortens very markedly on cooling and relaxes on warming to about 40°; then remains unchanged in length up to 50°; it is killed between 40 and 50°. Slow rhythmic contractions may appear when cooled from 40 to 35°, even 2 or 3 days after excision.

In a very recent paper, De Zilwa* describes the shortening of the muscle by cooling down to 10 or 15° C., gradual relaxation up to 40° (when it is complete), spontaneous contractions developing at 38° absent at 40°. On further raising the temperature he notes contraction at 47° or 48°; later, relaxation beginning at 52—54° and completed at 58—60°. The muscle is often found to retain some trace of excitability when beginning to relax at 52—54°; when the relaxation is complete the muscle is dead, and its response to excitation or changes of temperature cannot be restored by cooling. There is no evidence of the occurrence of true *rigor mortis* in this muscle.

The unstriped muscle of the cat's bladder also maintains its excitability for relatively long periods.

C. C. Stewart† finds that at ordinary room temperatures, irritability often lasts 24—48 hours after excision; kept in an ice-box at 5—8°, one preparation responded to the Faradic current at the end of 4 days. When cooled a strip of the bladder muscle shortens, the shortening being complete about 10° C. When the temperature is raised from this point it relaxes up to about 40°, above 40° there is shortening (slowly at first, then more rapidly) up to 53—57°, where the muscle apparently loses its excitability and dies. A distinct loss of tone, often of considerable extent, follows; the muscle is comparatively relaxed and very soft. Only when the temperature is raised to 69° does what Stewart describes as the shortening of heat-rigor occur.

The essential correspondence of the main features presented by these living muscles and by a contracted artery under the influence of temperature changes is too obvious to need insisting upon.

Many years ago, Samkowy‡ found that the unstriped sphincter pupillæ muscle of the rabbit contracts at first on warming, but dilates later; when relaxed by warmth, cooling to 28—29° induces contraction. The dilated pupil of a dead cat contracts at moderate temperatures and dilates when the temperature is raised to about 37°. Using also the rectococcygeus muscle of the rabbit and the bladder of the cat, rabbit, &c., he concluded that unstriped muscle relaxes at about 37°.

* 'Journal of Physiology,' vol. 27, p. 200.

† 'American Journal of Physiology,' vol. 4, p. 199 (1900).

‡ 'Pflüger's Archiv,' vol. 9, p. 400 (1874).

Morgen* heated a ring from the œsophagus of the dog and observed abolition of irritability at about 50°.

From the evidence available it is clear that relaxation at 50—55° C. is a phenomenon of widespread occurrence in living mammalian unstriped muscle showing tonic contraction; the muscle is dead when this relaxation is completed.

Experiments on the Aorta and Pulmonary Artery.

Transverse and longitudinal strips were examined soon after death in the same way as the carotid. The changes observed were similar in general character to those described in the carotid at similar periods after death—relaxation about 50—55°, preceded in many cases by contraction, &c.

But these changes are very slight in extent in the aorta and pulmonary artery, as might be expected in vessels provided with muscular tissue in relatively small amount. (Fig. 8.)

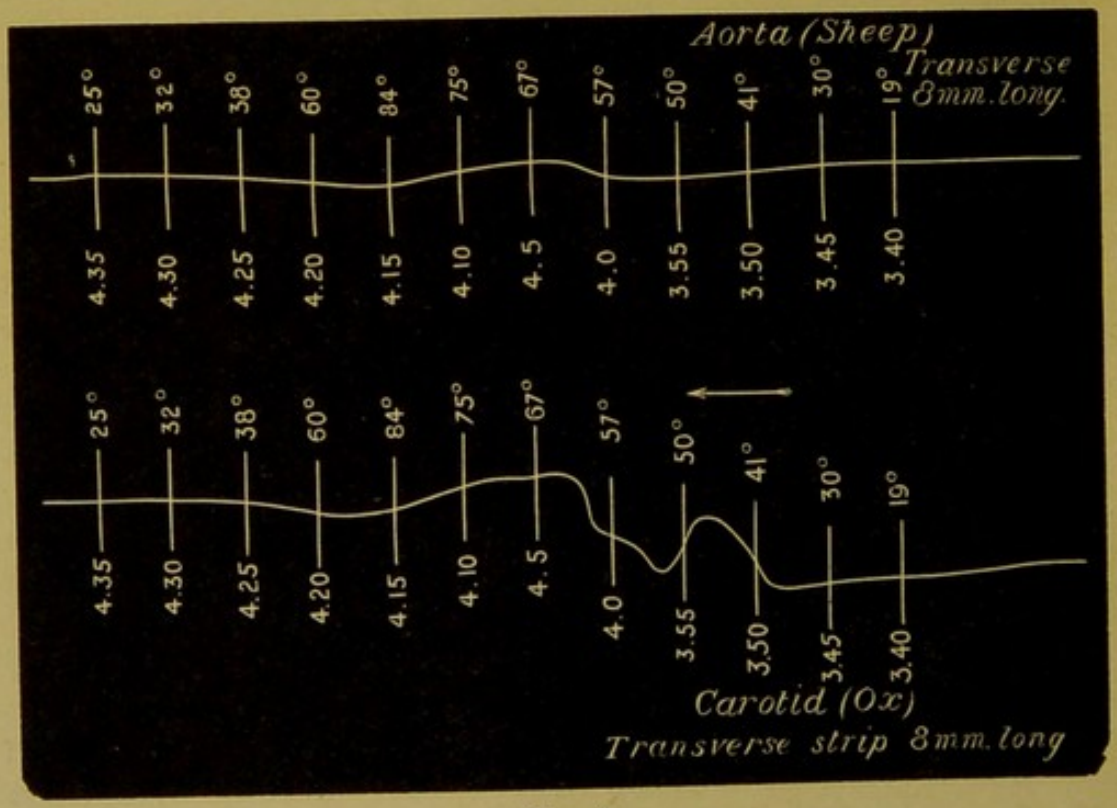

Fig. 8.

At the same time the contraction at 60—65° is hardly so extensive as might have been looked for in strips containing so much elastic and connective tissue.

* 'Untersuch. aus d. Physiol. Inst. zu Halle,' vol. 2, p. 161 (1890).

Experiments on the Jugular Vein.

Strips of vein when heated gave very definite and constant results. Shortening began almost immediately, and went on at first pretty quickly (from the original temperature of 14—16° up till about 30°), then more gradually, till at 60—65° (usually at 62° or 63°), rapid and very extensive shortening took place, going on commonly till the temperature is above 70°. Subsequent cooling is attended by a decided elongation, though the strip still remains very much shorter than it was to begin with. (Figs. 6, 9, and 10.)

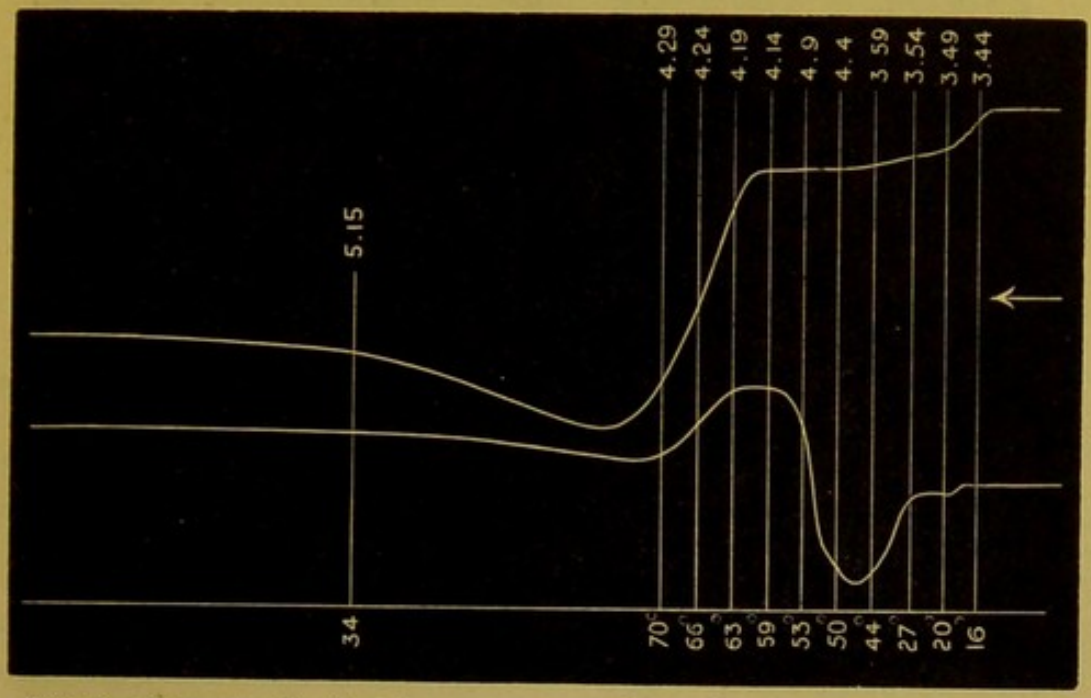

FIG. 9.—Upper tracing from transverse strip of jugular vein, lower from carotid artery of horse.

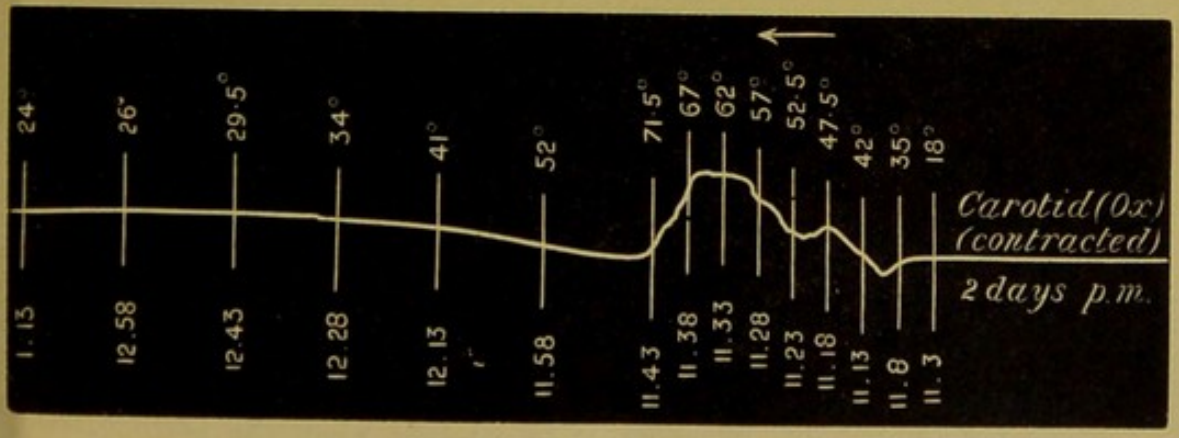

FIG. 10.

When the temperature is only raised to 40° and then allowed to fall, shortening goes on as before up to that point, and the strip

lengthens again during cooling, so that it returns to the same, or almost the same, length as it was at first. (Fig. 11.)

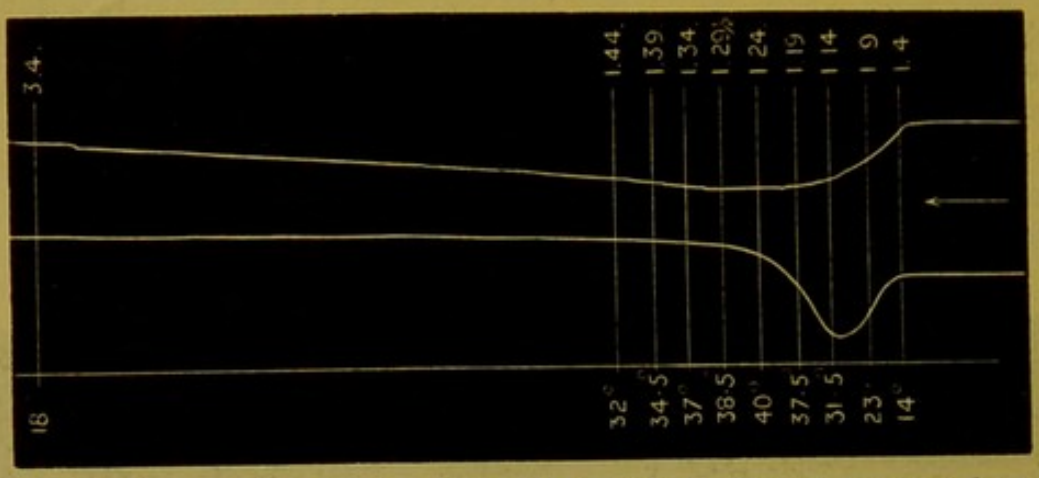

FIG. 11.—Upper tracing, jugular vein; lower, carotid. Transverse strips, heated up to 40° and then allowed to cool.

Saline Extracts of Contracted Artery.

Saline extracts of the fresh arterial wall were made from the carotid, &c., usually with 5 per cent. $MgSO_4$ solution; 10 per cent. NaCl and 13 per cent. NH_4Cl were occasionally employed. The outer and inner coats were often stripped off as completely as possible, so that little more than the tunica media was used.

The 5 per cent. $MgSO_4$ extract is usually neutral.

It was heated up gradually by Halliburton's method, sometimes without acidulation, sometimes faintly acidulated with 2 per cent. acetic acid. The rise of temperature was commonly about the rate of 1° C. per minute, though this was made to vary widely, and the fluid was often kept at certain temperatures for many minutes.

Acidulated Extract.

Coagulation occurred between 45° and 50°, usually about 47°. When this was filtered off and the heating continued, a second coagulation took place at 55—60°—figures corresponding with the coagulation temperatures of Halliburton's paramyosinogen and myosinogen.

(There was some evidence of a further slight coagulation at about 72—75°.) The amount of coagulation occurring at 45—50° and at 55—60° varied very markedly; sometimes the one and sometimes the other predominated. It is to be noted that when the preliminary acidulation is done a certain amount of precipitation occurs—probably nucleo-proteid, in part at least. After filtration, the liquid is treated as above described.

Neutral Extract.

The fluid became strongly opalescent at 45—50°, but as a rule did not become flocculent, and no precipitate could be separated by filtration. (Compare Vincent and Lewis's results with non-striped muscle from the stomach of the calf; these authors remark a similar absence of definite coagulation in neutral extracts.*)

Well-marked coagulation became evident at 60—65° as a rule.

Saturation with $MgSO_4$ appeared to precipitate nearly all the proteid contained in the saline extract. Shaking was not employed in effecting saturation; the extract was allowed to stand for 24 hours with excess of $MgSO_4$ crystals, and inverted from time to time.

Small test-tubes, containing 5 per cent. $MgSO_4$ extract of the arterial wall (neutral or acidulated), were often placed in the oil-bath, in which strips of the same artery were being heated and made to record their changes in length; the changes in the strips and the $MgSO_4$ extract placed side by side under the same conditions were compared.

Contracted Artery.

The early contraction frequently occurring (25—35°) in the strip was attended by no evident change in the $MgSO_4$ extract, nor was the relaxation about 40°. The contraction frequently seen between 45° and 50°, especially about 47°, coincides with the development of marked opalescence in the neutral $MgSO_4$ extract, and with coagulation in the acidulated extract.

The characteristic relaxation at 50—55° follows upon the change in the $MgSO_4$ extract just mentioned, and corresponds with no definite further change in that liquid.

The final shortening of the strip at about 65° takes place pretty much at the same time as coagulation in the extract, but evidently does not essentially depend on that coagulation, since it occurs in strips that have been macerated for many days in large quantities of 13 per cent. NH_4Cl solutions.

When an acidulated $MgSO_4$ extract is used to compare with the changes occurring in the heated strip, the preliminary precipitation caused by the addition of a little 2 per cent. acetic acid has to be borne in mind. For the substance thus precipitated and removed by filtration might possibly have been one which would have shown a change in relation to some of the phases in the behaviour of the arterial strip during heating.

Of course this objection does not apply when a non-acidulated $MgSO_4$ extract is used in making the comparison.

An attempt was made to co-ordinate the changes occurring in a

* 'Journal of Physiology,' vol. 26, p. 445.

$MgSO_4$ or NH_4Cl extract when heated with those occurring within the tissues of the arterial wall in the following way:—Weighed amounts of the arterial wall were taken and heated at the usual rate up to various temperatures (40°, 45°, 50°, 55°, 60°, &c.), immersed in mercury contained in small (corked) test-tubes, which were placed in the oil-bath. The portions of artery so treated were then extracted with equal amounts of 5 per cent. $MgSO_4$ or 13 per cent. NH_4Cl solution for equal periods, for comparison with portions of the same artery which had not been heated at all. The different extracts were tested for the amount of proteid which they contained by heat-coagulation, precipitation by various reagents, &c.

It was found that heating fresh artery up to 40° or 45° made no appreciable difference in the amount of proteid subsequently obtainable from it by 5 per cent. $MgSO_4$ sol., while heating to 49° or 50° markedly diminished the subsequent yield of proteid. Obviously a considerable amount of proteid has been rendered insoluble at 45—50° in the tissue. Heating to 55° did not seem to make any marked difference as compared with heating to 50°. A temperature of 60° caused the subsequent yield of proteid to be greatly lessened; indeed there was little extracted by the $MgSO_4$ sol. after the artery had been heated to 60°. Evidently coagulation had taken place between 55° and 60°.

Comparing these results with what is seen on heating the acidulated $MgSO_4$ extract of an artery that has not been heated, we find that proteid is rendered insoluble in the tissue of the arterial wall at about the same temperatures as in the extract. In the case of the non-acidulated extract, proteid becomes insoluble in the tissue (45—50°) when the extract becomes strongly opalescent, but shows no floccules capable of separation by filtration; later (55—60°) proteid is rendered insoluble at a somewhat lower temperature than is usually required to give a flocculent heat-coagulum (60—65°) in the non-acidulated extract.

Changes in the length of strips of the arterial wall bear a relation to the coagulation of proteid in the tissue similar to what has already been stated in regard to $MgSO_4$ extracts, excepting that the rendering insoluble of proteid at 55—60° in the tissue comes distinctly before the final shortening of the strip.

Saline Extracts of Relaxed Artery.

Some of the methods already described as effective in relaxing a contracted artery would obviously render the subsequent making of saline extracts impracticable (sulphocyanide, &c.).

The freezing method is not open to this objection. Extracts made with 5 per cent. $MgSO_4$ from an artery relaxed in this way show heat

coagulation (when acidulated) at 45—50° and 55—60°; also arteries that have been kept for days until contraction has passed off, and arteries relaxed by keeping at body temperature for 24 hours, &c.

Now strips from such arteries—if completely relaxed—show no important change when heated until the 60—65° shortening takes place; there is no alteration in the tracing coincident with the heat-coagulations at 45—50° and at 55—60°, which are seen in the $MgSO_4$ extract.

Saline Extracts of Aorta and Pulmonary Artery.

These when acidulated and heated show relatively little proteid coagulation, as might be expected from the relatively small amount of muscle in their walls; the temperatures of coagulation seem to be pretty similar to those of extracts of carotid.

Contraction v. *Rigor Mortis.*

Is the *post-mortem* contraction seen in an artery a true contraction of exceedingly long duration, or is it a condition of the same nature as the rigor mortis of skeletal muscle?

There is a variety of evidence in favour of the conclusion that *post-mortem* contraction of arteries is a true persistent contraction, very different in many respects from the rigor mortis of skeletal muscle.

(1) As has been already stated, though the excised artery goes into the contracted state very soon after death, its excitability may be maintained for two or three days. Up to this time then it is obvious that the arterial contraction has been of a nature very different from true rigor mortis. The same is indicated by the partial relaxation of the contracted artery, which may spontaneously develop a day or two after death—to be followed by further contraction of the application of a suitable stimulus. The reaction of the cut surface to litmus paper is alkaline or amphoteric, even after the contraction has lasted for days.

(2) The effect of sulphocyanide of potassium solution applied to a contracted artery is strikingly different from what is seen in the case of a skeletal muscle contracted in rigor mortis. The artery, as has already been described, soon becomes completely relaxed in the solution, whereas the rigor mortis muscle does not relax at all.

(3) The influence of freezing for some hours is very different in the two cases. In the artery freezing quickly after the death of the animal, before *post-mortem* contraction has begun, entirely prevents the appearance of that contraction; in skeletal muscle similar freezing does not obviate the subsequent development of rigor mortis (with acid reaction, &c.). Portions of skeletal muscle from the ox, horse, &c., were frozen for four hours, and showed a marked contrast to arteries

similarly treated; the skeletal muscle went strongly into rigor mortis after thawing.

Again, it has been shown that freezing a contracted artery for some time (*e.g.*, 4—5 hours) completely and permanently abolishes the contraction. In rigid skeletal muscle the result is different; in some cases there is no perceptible diminution in the rigidity after thawing; in other instances, the rigidity seems to be diminished but by no means abolished.

(4) The effect of heating a contracted artery is strikingly different from what occurs when a skeletal muscle is treated in the same manner.

The contracted artery shows a characteristic relaxation at about 50° C., which may or may not be preceded by definite phases of contraction and relaxation, as already described; skeletal muscle in rigor mortis shows no such relaxation. In its behaviour towards changes in temperature the contracted artery shows an unmistakable resemblance to what is seen in non-striped muscle that is unquestionably alive, *e.g.*, the muscle of the cat's bladder and the retractor penis already referred to; there is an essential agreement in the main features evident in each case—increased tonus on cooling, relaxation at about 40°, followed by shortening, relaxation at 50—55° with final abolition of tonus, &c.

(5) When a strip of contracted artery is stretched by the successive addition of equal increments of weight, its behaviour (as will be presently described) is entirely different from that of a rigid skeletal muscle. A strip of relaxed artery, on the other hand, gives results essentially similar in their general character to those yielded by skeletal muscle.

(6) When a contracted artery is distended by internal pressure, it can often completely recover its original volume in the contracted state when the distending force is removed; indeed, in some instances it shows a subsequent increase of the original contraction. This is in sharp contrast to rigid skeletal muscle which, as is well known, fails to return to its former length when it has been stretched by the application of weights.

Elasticity of Strips of the Arterial and Venous Walls.

Wertheim,* and all observers who have worked at the subject since his time, have found that when strips of the arterial wall (aorta commonly used) are stretched by the successive addition of equal increments of weight the amounts of extension produced do not remain constant, but go on diminishing; the coefficient of elasticity increases with increased stretching.

* 'Annales de Chimie et de Phys.,' 3e série, vol. 21, pp. 385—414 (1847).

None of these workers seem to have taken into account the possible conditions of contraction and relaxation, or to have been aware of the existence of *post-mortem* contraction at all.

The method I have employed is an old one with certain modifications. The strip is firmly held by a clamp at one end, and suspended in the vertical position; the other end of the strip is made to pull upon the lever of a Helmholtz myograph, the attachment being made relatively near the fulcrum—the extension of the strip is magnified six times by the movement of the writing point. The lever is directed at right angles to the circumference of the smoked drum and its point, therefore, moves in a straight line perpendicular to the direction of movement of the recording surface. A simple arrangement on Pflüger's plan —somewhat similar to that figured by C. C. Stewart*—is employed to keep the writing point in constant contact with the smoked paper. The successive elongations are represented by vertical lines upon a stationary drum, which was each time moved round a certain distance by hand.

Short strips (commonly 5 × 5 mm.) of the arterial wall were used in order that the angular movement of the lever should not exceed a small limit. The successive weights were allowed to pull upon the strip for equal periods—varying in actual duration in different experiments—most commonly for 1 minute. Increments of 20 grammes were used in all the experiments of which tracings are given, except where otherwise specified. Drying of the strips was prevented by frequent pencilling with defibrinated blood, serum, or salt solution (0·75 per cent.).

Very different results were obtained with (1) strips taken from large arteries like the aorta and pulmonary artery, with their highly elastic walls, and relatively scanty muscular tissue; and (2) strips taken from medium-sized arteries like the carotid, &c., with their strong muscular coat.

Transverse and Longitudinal Strips from the Aorta and Pulmonary Artery.—Both longitudinal and transverse strips were taken from the walls of these vessels (chiefly from the sheep), and the results obtained quite agree in a general sense with those described by Wertheim, Roy, and others. The greatest amount of extension is produced by the first addition of weight, and successive additions of weight cause diminishing increments in length per unit increase of weight. A line joining the bases of the extensions is a curved line concave to the axis or abscissa. (Figs. 12—15.)

Strips from Medium-sized Arteries (Carotid, &c.).—Strips cut longitudinally and transversely to the long axis of the artery were employed, and certain differences were evident in the behaviour of the two kinds. But much more striking and important differences are shown by strips obtained from contracted arteries as compared

* 'American Journal of Physiology,' vol. 4, p. 186.

with those taken from relaxed arteries; transverse strips exhibit the most notable contrasts. As the condition of *post-mortem* contraction does not seem to have been taken into account by previous observers or known to them, it was obviously important to test the arterial wall in the conditions of contraction and relaxation respectively.

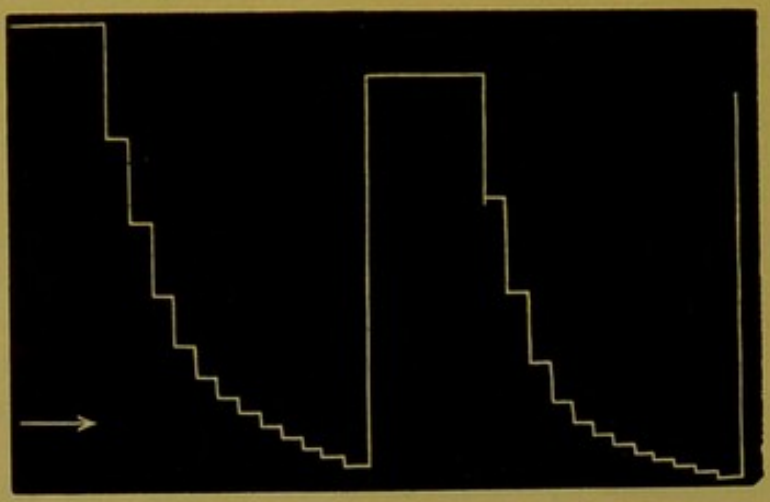

FIG. 12.—Aorta (sheep). Transverse strip. Interval of 12 minutes after unloading; then loaded a second time after the drum had been moved on about 1 cm.

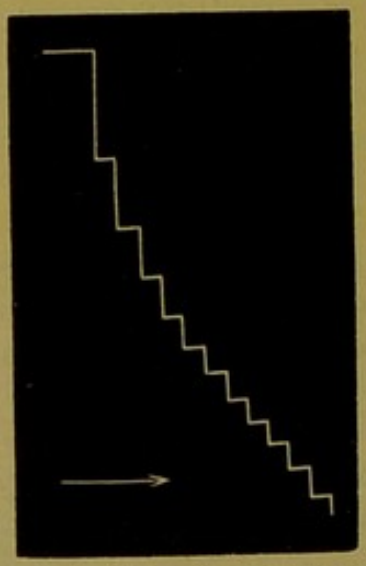

FIG. 13.—Aorta (sheep). Longitudinal strip.

Relaxed Arteries.—Both transverse and longitudinal strips made from a relaxed artery show broad features that are similar in the main; the artery may be relaxed by any of the methods already described.

The result obtained is similar to what has just been described in the case of the aorta and pulmonary artery.

Effects of repeated Stretching.—I have made numerous experiments on the effect of again stretching a strip of arterial wall some little time after it has been stretched by the addition of weights in the usual way and then unloaded.

The general character of the tracing is not altered in the aorta and pulmonary artery or in the relaxed carotid, though the actual amount of stretching is much increased, especially with the first weight or two. (Figs. 12, 14, 15, and 16.)

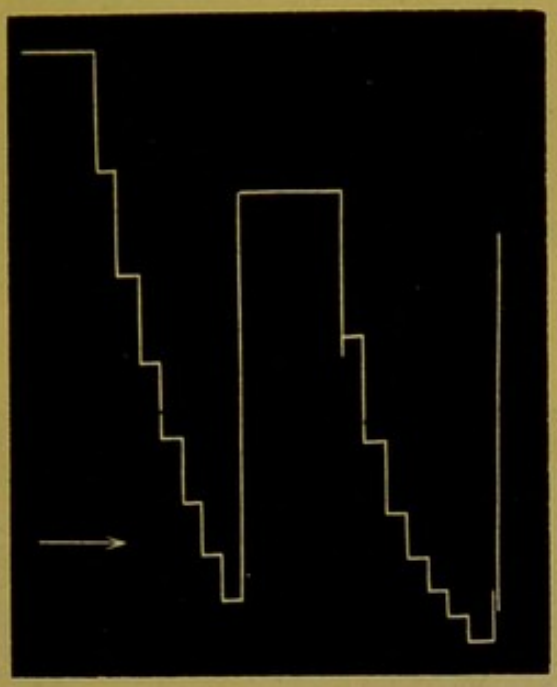

FIG. 14.—Pulmonary artery (sheep). Transverse strip. Loaded a second time after an interval of 7 minutes.

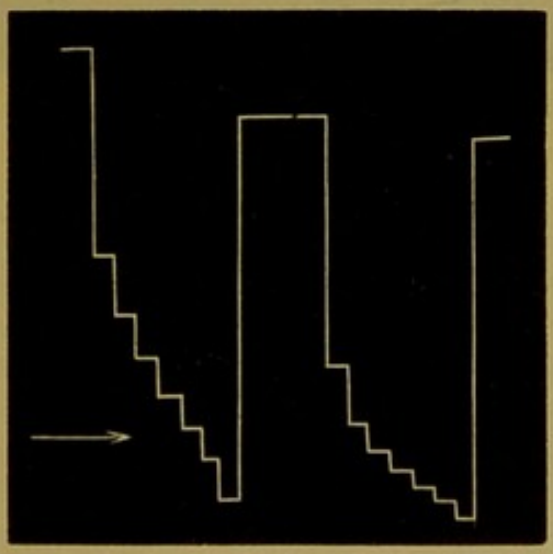

FIG. 15.—Pulmonary artery (sheep). Longitudinal strip. Loaded again after an interval of 7 minutes.

Contracted Arteries.—Transversely cut strips of contracted artery yield remarkable results, entirely different from those just described. Equal increments of weight cause increments of elongation, relatively small at first, and increasing in magnitude up to a maximum, beyond

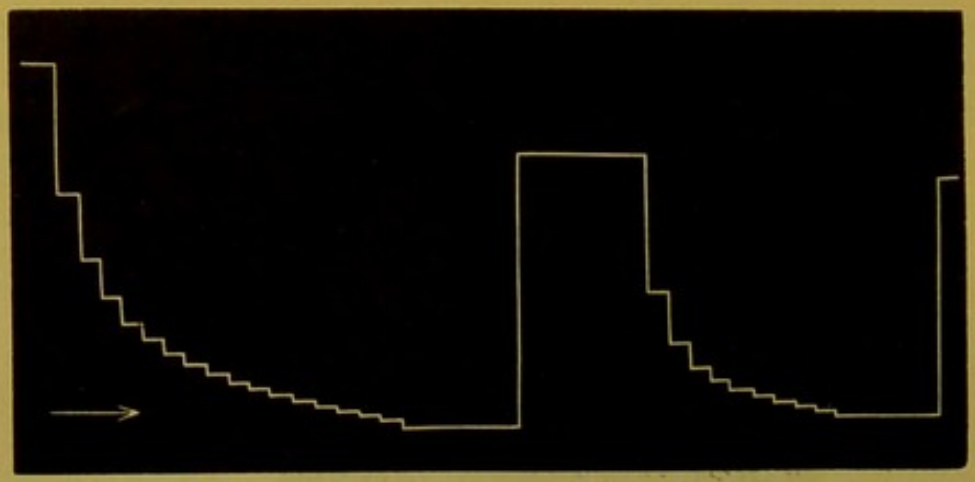

FIG. 16.—Carotid (ox), relaxed. Transverse strip. Loaded again after an interval of 12 minutes.

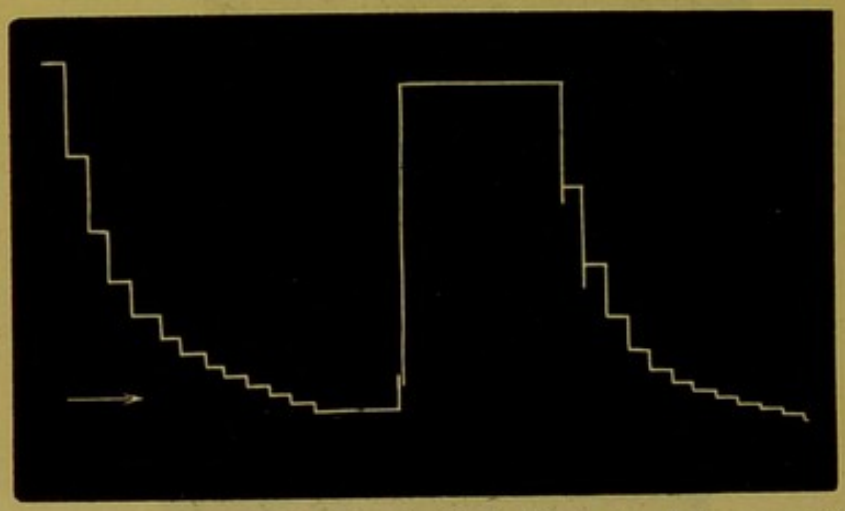

FIG. 17.—Carotid (ox), relaxed. Longitudinal strip. Loaded a second time after an interval of 10 minutes.

which there comes a progressive diminution in the amount of elongation produced by each successive addition of weight. Hence a line joining the lower ends of the vertical lines representing extension is a curved line at first convex towards the axis and then concave. (Fig. 18.)

In the early part of the process of stretching a contracted artery the resistance is solely muscular; later, when the muscular resistance has been so far overcome that the strip is stretched to what would be its normal length in a passive or relaxed artery, further stretching brings into play the resistance of the elastic and other elements in the arterial walls, and, as we have seen, a strip from such an artery resists elongation more and more, with successive increments of stretching force.

The behaviour of a transverse strip from a contracted artery is so constant and characteristic that it is always easy to tell from the tracing obtained whether the artery was contracted or relaxed. I am

not aware that any other tissue or structure has been shown to give a tracing of this sort.

These results are strikingly different from those of Wertheim,* Roy,† and others; these workers employed (chiefly or exclusively) strips of aorta in which little muscular tissue is present, frequently taken from the human subject some considerable time after death (from various diseases). Their tracings show a diminishing amount of elongation for unit weight, the same result that I have obtained in

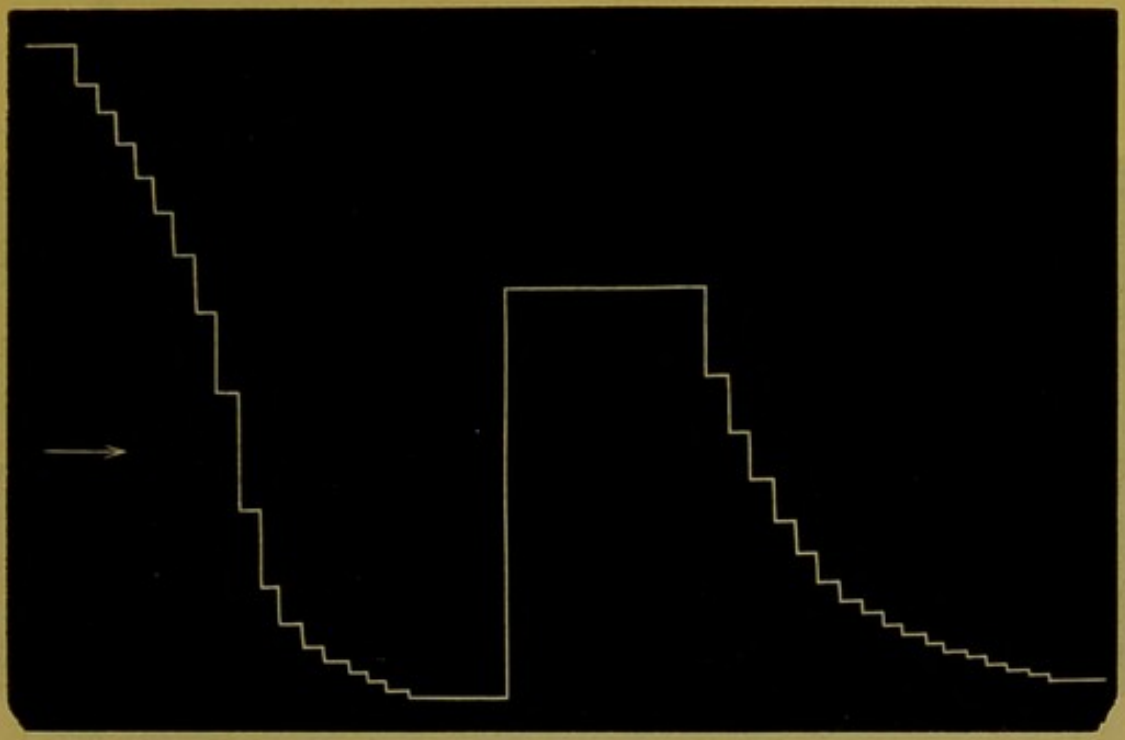

FIG. 18.—Carotid (ox), contracted. Transverse strip; 8 hours *p.m.* Second loading after interval of $1\frac{3}{4}$ hour.

the aorta and pulmonary artery. Some of Roy's curves are at first nearly, though not quite, straight in the early part of their course, *i.e.*, the elongation was during that part nearly, though not quite, proportional to the stretching weight: then a more marked and progressive diminution in extensibility becomes evident.

In some of my tracings the increment of length caused by the application of the first weight is very small—the smallest of an increasing series; in other cases the first extension is more considerable, and is followed by a very small one, to which others succeed in progressively increasing series. As regards the extent of the first elongation much seems to depend on the exact arrangement of the strip before the first weight is applied, and on the degree of its curvature, &c.

In some instances when a strip of artery taken not very long after

* *Loc. cit.*

† 'Journal of Physiology,' vol. 3, p. 125.

death is used, the series of increasing extensions is broken by a number of smaller extensions to give place again to larger increments. This is probably due to a contractile reaction of the muscular tissue of the strip against the stretching force, or is a result of mechanical stimulation caused by the clamps grasping each end of the strip. (Fig. 19.)

Repeated Stretching.—When a transverse strip is weighted in the way described and then unloaded, and after a time again weighted in the same way, a second tracing is obtained differing very strikingly from the first. It is in fact essentially similar to that obtained from a strip of relaxed artery. The extension caused by the first weight is very great. A third experiment of the same sort gives results essentially similar to those of the second experiment. (Figs. 18 and 19.)

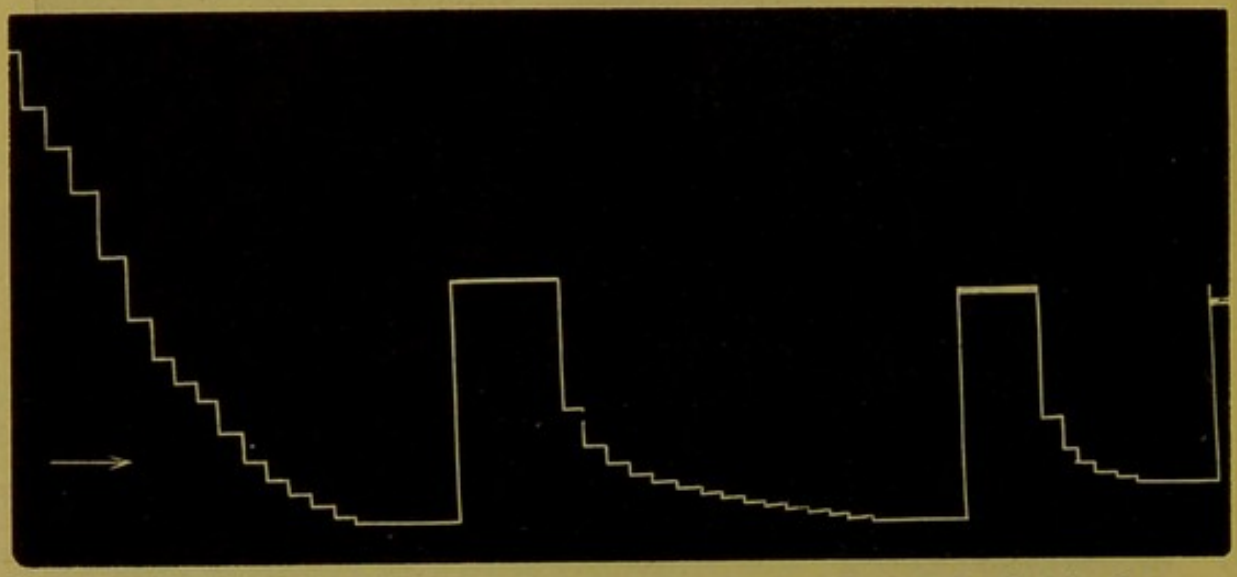

FIG. 19.—Carotid (ox), contracted. Transverse strip. During the first loading the largest extensions were followed by two or three small ones; these were succeeded by decidedly larger ones which gradually diminished in size.

Longitudinal Strip of Contracted Artery.—With strips of similar dimensions cut longitudinally from the arterial wall, the total elongation produced by a total load similar to that used with transverse strips (*e.g.*, 240—340 grammes) is very much less than with transverse strips —commonly between a half and a third.

The character of the tracing differs in the two cases. In the longitudinal strip there is only a slight increase, if any, in the amount of elongation produced as the earlier weights are added, then a progressive diminution.

After unloading, the longitudinal strip recovers much more readily and completely than the transverse strip; the former shortens quite to its original length. (Fig. 20.)

Further, a repetition of the process of loading does not cause the striking alteration in the tracing seen with the transverse strip; with

the longitudinal strip the second loading gives a result essentially similar to the first.

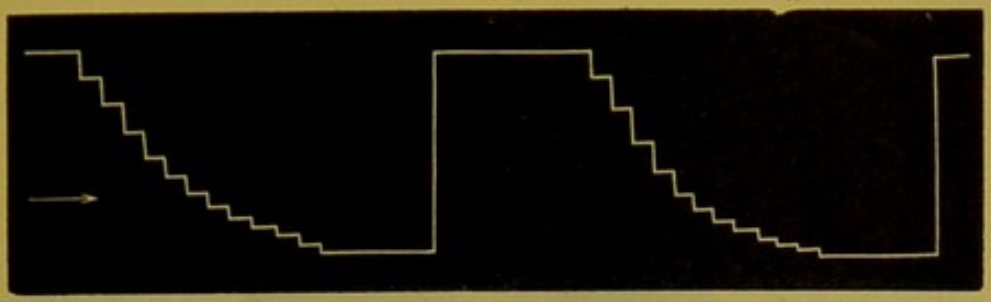

FIG. 20.—Carotid (ox), contracted. Longitudinal strip. Second loading after interval of 12 minutes.

Jugular Vein.—Transverse* and longitudinal strips give results very similar to relaxed arteries, though of course much smaller weights are required to stretch them; increments of 5 grammes were used instead of 20 grammes.

When stretched a second time, the difference in elongation is much slighter than in arteries; a longitudinal strip of vein may indeed show no appreciable difference. (Figs. 21 and 22.)

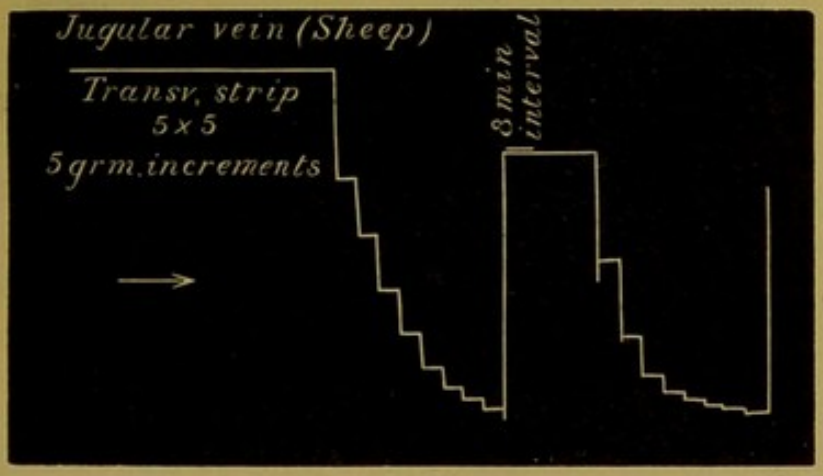

FIG. 21.

Contracted Artery treated with Salicyl-sulphonic Acid.—A contracted artery which was left in 2 per cent. watery solution of salicyl-sulphonic acid for 24 hours with the result that its proteid constituents were precipitated, gave a series of elongations almost proportional to the weights employed, recalling the results got with iron wire or india-rubber. This is well seen in fig. 23.

A second application of the weights—if the first stretching was not

* *Cf.* Braune's 'Beiträge z. Anat. u. Phys.,' 1874 p. 7, and Bardeleben's 'Jenaische Zeitschrift,' vol. 12, p. 40, 1878, on the changes in length of veins when weighted.

extensive—may give a tracing differing relatively little from the first loading—in marked contrast with what happens in a contracted artery. The elongation caused by the first weight (in the second loading) is

FIG. 22.

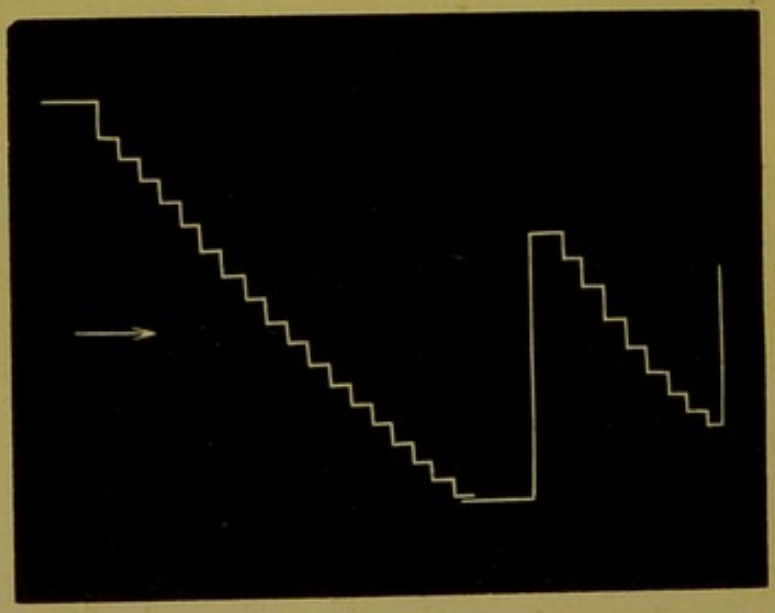

FIG. 23.—Carotid (ox), in contracted state, a few hours after death was put into 2 per cent. solution of salicyl-sulphonic acid and kept in the solution for 24 hours. Transverse strip then loaded. Second loading after interval of 15 minutes.

small; the subsequent ones are somewhat larger than before—to diminish after a time. When the first stretching is very extensive, the second weighting may give results more resembling those got with a contracted artery.

Relation of Cubic Capacity of an Artery to Internal Pressure.

Various observers have investigated this subject, but their results are very discordant.

Marey,* working with the aorta of man and different animals, and using a water-plethysmograph, found that the expansion (resembling the extension of strips of the vascular wall which Wertheim† had first described), follows the rule that the higher the absolute pressure the less the artery expands with equal rises of pressure.

Roy‡ found that in healthy arteries taken immediately after death the increase of capacity with unit increase of pressure became progressively augmented up to a certain point, beyond which the increase of capacity declined. The turning point he found to be about the normal level of blood pressure in the animal from which the artery was taken; he formulated the conclusion that the arteries are most elastic and distensible at pressures corresponding more or less exactly to the normal blood pressures to which they were exposed during life (dog, cat, rabbit, &c.).

In cases where there had been marked marasmus before death, Roy found the arteries more distensible than normal arteries; they expanded most readily at lower pressures, and he sometimes found the maximum distensibility to be immediately above zero pressure—as he found to be normally the case with veins.

Zwaardemaker§ found the augmentation of cubic capacity to be at its maximum in the excised arteries of the horse and dog at 32—50 mm. Hg. (very different figures from Roy's), in the ox at 100—150 mm. In an experiment on a living artery *in situ* (dog) he concluded that the maximum distensibility was at 75—100 mm.‖

Thoma and Kaefer¶ examined the increase of the diameter of certain arteries (external iliac and common carotid of man). They found that as the pressure was elevated the diameter increased rapidly at first, then more and more slowly.

Tigerstedt** calls attention to the discordant nature of these results, and the need of further investigation.

The relation of the cubic capacity of an artery to internal pressure I have studied by the following method:—

A portion of the artery to be examined was closed at one end by a wooden plug firmly tied in; into the other end a cannula of suitable size was made fast, through which the artery could be subjected to any

* 'Travaux de Laboratoire,' vol. 4, p. 178 (1880).

† *Loc. cit.*

‡ *Loc. cit.*

§ 'Nederlandsch. Tijdschrift voor Geneeskunde,' 2 Reeks, vol. 24, 1, pp. 61—76 (1888).

‖ Quoted in Tigerstedt's 'Lehrbuch des Kreislaufes' (1893), p. 319.

¶ 'Arch. f. Path. Anatomie,' vol. 116, p. 9 (1889). *Cf.* Luck, 'Ueber Elasticitätsverhältnisse gesunder u. kranken Arterienwand,' Inaug. Dissert., Dorpat, 1889; Kaefer, 'Zur Methodik der Elasticitätsmessungen au der Gefässwand,' Inaug. Dissert., Dorpat, 1891.

** *Loc. cit.*

desired pressure of air by means of a movable mercury reservoir communicating with a pressure bottle, from which in turn a tube leads to the arterial cannula; this tube has a lateral connection with a mercury manometer to show the amount of pressure acting upon the internal surface of the artery. The artery thus prepared is inclosed in a sort of plethysmograph consisting of a glass vessel filled with olive oil, and provided with a long graduated tube of small bore projecting horizontally at one side; the size of the bore employed varied in different experiments—commonly 1 mm. for contracted arteries, and 3 mm. in experiments on relaxed arteries (ox). Variations in the volume of the artery were accurately indicated by the movement of oil in the horizontal tube. In the figures the rises of pressure are taken as abscissæ and the expansions in volume (indicated by the number of millimetres the oil moves in the graduated tube) as ordinates. The length of the portion of artery used was measured between the ligatures fastening the cannula and the wooden plug *in situ* at either end of the artery.

The pressure was usually raised by increments of 20 mm. Hg, and kept steady at each level for 1 minute.

As might be anticipated, great differences were observable in the behaviour of relaxed and of contracted arteries.

Relaxed Artery.—Completely relaxed arteries when subjected to equal increments of internal pressure do not respond by equal increments of cubic capacity; the increase in volume is greatest at first and successively diminishes as the pressure is raised—the distensibility of the vessel is greatest at the first rise above zero. This result is similar to what has already been described as holding good in the case of veins; it is in entire opposition to what Roy found in healthy arteries examined soon after death. The expansion of volume caused in the relaxed artery by each successive elevation of pressure goes on pretty quickly at first each time the pressure is raised, then proceeds much more slowly during the latter portion of the period during which the pressure is kept steady at any particular level. (Figs. 24 and 26.)

When the pressure is raised a second time after an interval, the distensibility of the artery is found to be greatly increased. (Fig. 25.)

I have tested the elasticity of arteries (chiefly of ox and sheep) in which relaxation has been induced in the different ways already described. No doubt these various modes of treatment induce changes in the arterial wall very different in their nature in some respects. But they have one feature in common, inasmuch as they all abolish contraction, and concurrently they lead to a characteristic change in the behaviour of the tube towards internal pressure, in virtue of which the artery becomes most distensible at low pressures—immediately above zero pressure, &c.

While an idea of the behaviour of arteries relaxed by keeping and by

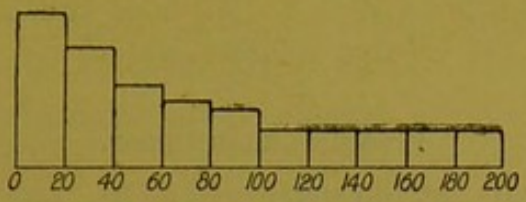

FIG. 24.—Carotid (ox), relaxed, having been kept in defibrinated blood for several days. Length (between ligatures) 14 mm. Diameter of lumen 5 mm.

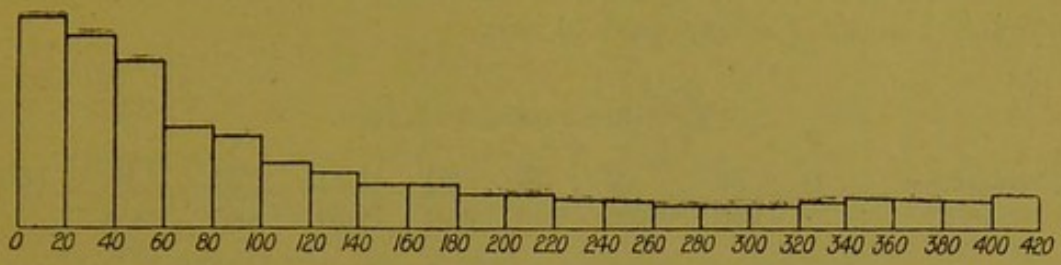

FIG. 25.—Second elevation of pressure in same portion of artery as in fig. 24, after an interval of 30 minutes. Great increase in distensibility, with comparatively low pressures. After the expansion has declined very greatly (pressure of 300 mm., &c.) there is again a slight increase of expansion between 320 mm. and 420 mm.

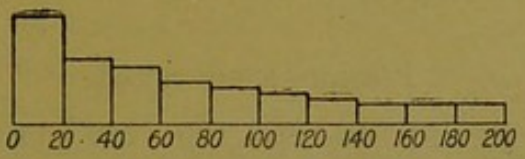

FIG. 26.—Carotid (ox), relaxed by freezing for 5—6 hours. Lumen 5—6 mm. Length of artery 19 mm.

freezing is given by figs. 24 and 26, the nature of the results obtained with arteries relaxed by other methods will be seen from the following instances. The amount of expansion is indicated by the movement of oil in the graduated tube stated in millimetres :—

Carotid (Ox) relaxed by Potassium Sulphocyanide.—A contracted artery 37 mm. long, with lumen of 3 mm., and thickness of wall nearly 2 mm., was put into 20 per cent. solution of potassium sulphocyanide for an hour or two. It was then found to be 34 mm. in length, with lumen about 5 mm., and thickness of wall 1·5—1·75 mm. Rigidity gone. Length of portion used (measured between ligatures) 17 mm. Pressure raised in usual way.

Pressure.—	0	20	40	60	80	100	120	140	160	180
		200	220	240	260	280	300	320	340	360
		380	400	420.						
Expansion.—		14	13	9	8	6	6	5·5	4·5	4·5
		4·5	5	5	55	5	5	5	4·5	4·5
		4·5	4·5.							

Pressure was then lowered to 0. An hour later the artery had not yet diminished to its original volume—the oil was 28 mm. from the starting-point.

Pressure again raised.

Pressure.—	0	20	40	60	80	100	120	140	160	180	200.
Expansion.—		18	14	12	10	6·5	7	4·5	4·5	3·5	4·5.

Carotid (Ox) relaxed by keeping it in blood at 39° *C. for* 24 *hours.* Lumen 6 mm. Length of artery used 16 mm.

Pressure raised as before.

Pressure.—	0	20	40	60	80	100	120	140	160
		180	200	220	240	260	280	300.	
Expansion.—		14	11	10	10	10	10	9	8
		10	10	8	9	8	6	6.	

Pressure lowered to 0.

An hour and three-quarters later oil was 20 mm. from starting-point.

Pressure again raised.

Pressure.—	0	20	40	60	80	100	120	140	160	180	200.
Expansion.—		27	21	16	11	7	6	6	5	3	4.

Carotid (Ox) relaxed by Ammonia Vapour.—A strongly-contracted artery exposed to ammonia vapour changed as follows:—Length diminished from 28 to 24 mm.; lumen enlarged from 2 mm. to 5—6 mm.; wall thinned from 2 mm. to about 1 mm. Length of artery used in plethysmograph, 13 mm.

Pressure.—	0	20	40	60	80	100	120	140	160.
Expansion.—		12	9	6	5	5	5	4	4.

Contracted Artery.—When a portion of artery is excised from a recently killed animal and prepared for experiment upon its distensibility in the way described above, it is usually in a state of pretty strong contraction, the contraction present on excision being as a rule markedly augmented by the necessary manipulation, tying in the cannula and wooden plug, &c. When the internal pressure is raised step by step by equal increments (*e.g.*, of 20 mm. Hg.), the artery at first yields relatively little and the augmentation of its volume is slight. Successive rises of pressure cause increasing amounts of expansion, and in the case of a thick-walled and strongly-contracted artery (*e.g.*, carotid of ox the increasing expansion may go on up to very high

pressures (*e.g.*, 420 mm.), the muscular resistance being more and more overborne by the successive increments of internal pressure. The expansion caused by each rise of pressure is very gradual—in marked contrast to what is seen in a fully-relaxed artery; the oil in the horizontal graduated tube keeps progressing along the tube during the period of raised pressure. The increase in capacity with unit increase of internal pressure may go on increasing up to pressures vastly higher than the normal blood-pressure of the animal. (Fig. 27.) Beyond a

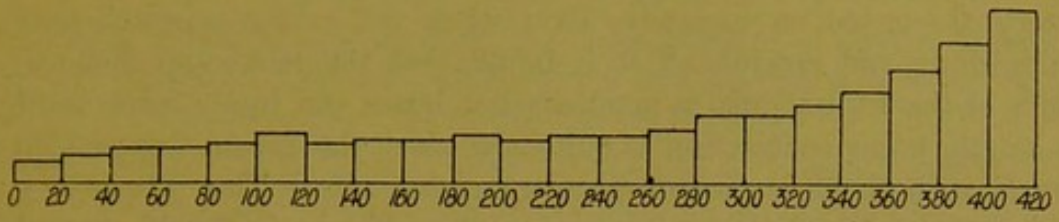

FIG. 27.—Carotid (ox), strongly contracted (48 hours *p.m.*).

certain pressure, varying in different cases, diminished expansion occurs, though in a strongly-contracted artery an enormous pressure is required to reach this point—at least when the pressure is raised at the rate adopted in the experiments now being described.

When an artery is taken which presents a slighter muscular resistance to distension—on account of its muscular coat being relatively thin, or its contraction being less strongly developed, the increase of capacity with unit increase of internal pressure becomes progessively augmented up to a certain point—which is more easily reached—and then declines. (Fig. 28.) The turning-point—indicating the maximum

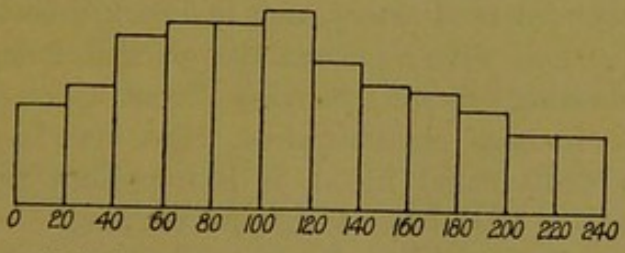

FIG. 28.—Carotid (ox), weak contraction (5 days *p.m.*). Length 12 mm.

distensibility of the tube—may come at various levels of pressure according to circumstances; it may or may not correspond to the normal height of the blood pressure in the animal from which the artery was taken. The change seems to take place some time after the muscular resistance is so far overcome, that the artery has become distended to its normal size in the passive condition; the elastic resistance of the arterial wall (now becoming stretched beyond its size in the passive state) comes into play to resist further distension. In the process of distension, when the arterial wall is being moved from the position which it occupies in a contracted artery to its position in

a passive condition, the resistance must evidently be entirely muscular, since the purely elastic property of the arterial wall tends to make it take up the position seen in a passive artery. Increased opposition to further distension will be offered by the elasticity of the arterial wall resisting distension beyond its position in the passive (relaxed) artery.

In arteries with relatively slight contraction present, the maximum distensibility is found to occur at a relatively low level—which may be about the normal blood pressure or much below the normal blood pressure of the animal. (Fig. 28.) When portions of the same artery are tested on successive days when *post-mortem* contraction is diminishing and passing off, it is found that the maximum distensibility of the arterial tube is manifested at lower and lower levels until at length, when contraction is quite abolished, the greatest expansion occurs with the first rise of pressure above zero. Thus the artery which showed increasing expansion up to 420 mm. in fig. 27, showed some days later (when its contraction was weak) maximum expansion at 40—60 mm., and later still immediately above 0.

Here it is necessary to remark upon the extraordinary persistence of a residuum of contraction in the arterial wall—in some cases even after signs of putrefaction are evident. Putrefaction begins in adherent blood, periarterial tissue, &c., much earlier than in the tunica media, and the survival of the latter is favoured by cleansing the artery after excision from blood and serum, and by removing connective tissue, &c., from around the vessel; still more by keeping the artery so prepared immersed in olive oil. Roy remarked that the elasticity curve of any given artery remains the same until putrefaction is far advanced—which is intelligible in view of what has just been stated.

In warm weather pieces of artery kept in a corked bottle, moistened with normal saline, may have a putrefactive smell in 2 days, while still contracted and showing a striking increase of contraction on stimulation by cutting, &c. In such circumstances, when arteries are kept for several days in defibrinated blood, it is important that the blood should be replaced by fresh blood from time to time.

Effects of Repeated Distension.—When a strongly-contracted artery has once been distended by high pressures, it is more easily stretched by a repetition of the rise of pressure, while the general character of the expansion remains the same—unless the first distension was very great. This is seen on comparing figs. 29 and 30.

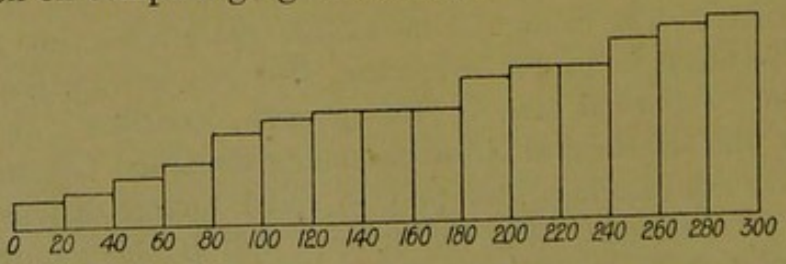

Fig. 29.—Carotid (ox), strongly contracted; 48 hours *p.m.* Length 14 mm.

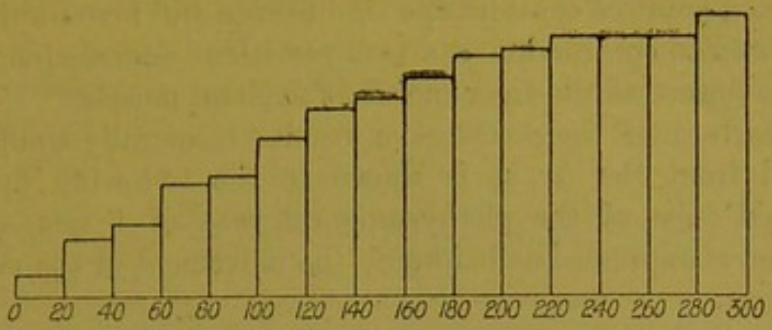

FIG. 30.—Same artery as in fig. 29; second elevation of pressure 24 hours after the first. The distensibility is seen to be much increased, though the general behaviour in giving enlarging increments of volume per unit rise of pressure is not changed.

In other cases where the artery is only slightly contracted, and the maximum distensibility is found at, say, 100—120 mm., a second rise of pressure causes the maximum distension at a much lower level—*e.g.*, at 40—60 mm., or, it may be, immediately above zero. (Compare figs. 28 and 31.)

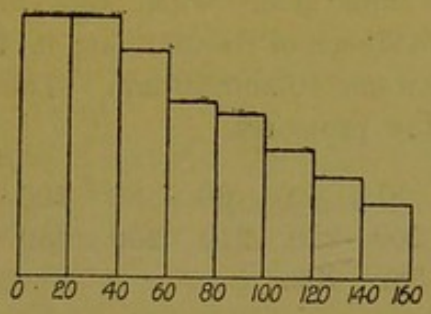

FIG. 31.—Second elevation of pressure in same portion of artery as in fig. 28, half an hour after completion of first experiment.

Such effects of repetition of the rise of internal pressure are manifested whether the second rise is induced very shortly after the first one (a few minutes) or after an interval of 24 hours or more. In the latter case the change may be more extensive than in the former—owing no doubt to the lapse of time leading to a diminution of the contraction present in the artery.

As regards the recovery of a contracted artery after it has been distended by internal pressure, this is often quite complete—at least when the artery is tested at a relatively early stage of *post-mortem* contraction, *e.g.*, within a day or two after death. The oil which has been driven out along the horizontal graduated tube when the artery expands under the increase of internal pressure returns quite to the starting-point; indeed it often moves back beyond its starting-point—showing that the artery has not only completely recovered from distension, but has contracted somewhat in volume from what it was at the beginning of the experiment.

This is a point of considerable significance and favours the view that the *post-mortem* contraction is a true persistent contraction, and not of the same nature as the rigor mortis of skeletal muscle.

The carotid of the sheep gave results essentially similar to those obtained from the ox, as is shown in the following figures. The graduated tube of the plethysmograph was of 1 mm. calibre; the expansion is expressed as before by the movement of the oil—stated in millimetres.

Pressure.—	0	20	40	60	80	100	120	140	160	180	200.		
	220	240	260.										
Expansion.—	4	5	10	14	21	26	28	30	42	45	55	65	80.

Another sheep's carotid with less strongly-marked contraction showed the maximum expansion at 100—120.

Pressure.—	0	20	40	60	80	100	120	140	160	180.
Expansion.—		4	4	7	10	13	16	14	14	12.

A portion of the same artery when kept for some days in blood showed only slight evidence of contraction; its maximum distensibility was found to be between 40 and 60 mm. The artery was very much more distensible at low pressures.

Pressure.—	0	20	40	60	80	100	120	140	160	180
		200	220	240	260	280	300	320	340	360
		380	400.							
Expansion.—		17	32	50	42	29	20	12	10	8	7	6
		5	5	5	4	4	3	4	3·5	3·5.		

Roy's well-known conclusion that the maximum distensibility of an artery was found at pressures corresponding, more or less exactly, to their normal blood-pressure, was probably based on the fact that he was dealing (unwittingly) with arteries in *post-mortem* contraction.

As regards the arteries in diseased animals and persons, Roy found that when defective nutrition, marasmus, &c., had been well-marked before death the arteries were found to be wider than normal, and their maximum distensibility was reached at pressures below normal—in some instances coming immediately above zero pressure.

Here there is good reason to believe that *post-mortem* contraction was small in amount or absent. I find that in emaciated worn-out old horses, *post-mortem* contraction is commonly very much less strongly developed than in more vigorous horses; and notably less so than in healthy oxen. In cases of exhausting disease in man there is reason to believe that *post-mortem* contraction is very slight in amount or absent.

It is to be noted that arteries show a striking general resemblance in (*a*) the expansion of the arterial tube when distended by internal pressure in each case, and (*b*) the elasticity of strips from the arterial wall when stretched by weights. This applies to both contracted and relaxed arteries.

Changes in the Length of Arteries during Variations in Internal Pressure.

We cannot at once predict the exact behaviour of an artery as regards changes in its length when subjected to variations in internal pressure from a study of the behaviour of longitudinally-cut strips of the arterial wall stretched by weights. For the relations of the tissues of the arterial wall are very different when they are in the form of a strip to what they are while forming a tube which is distended by internal pressure.

We have already seen that in an artery containing air at atmospheric pressure, the occurrence of contraction is accompanied by a lengthening of the tube, while relaxation is attended by a considerable amount of shortening.

An artery subjected to a considerable rise of pressure in its interior undergoes expansion in both its transverse and longitudinal diameters—in contrast to an excised artery at atmospheric pressure, which shortens when it widens from relaxation.

In the artery distended by internal pressure it is evident that the tendency to shortening which is associated with a marked increase in the transverse diameter of the tube is overborne by the stretching effect of the internal pressure.

The method I have employed for studying the changes in the length of an artery is as follows :—

A segment of artery was connected with a system of pressure bottles as already described. The portion of artery so prepared was placed in the vertical position, and the brass cannula tied into its upper end was rigidly fixed. The part of the wooden plug projecting from the lower end of the artery was then grasped by a small clamp attached to the lever of a Helmholtz myograph; the lever was directed at right angles to the smoked surface so that the writing point traced a vertical line. The pressure was usually raised 50 mm. Hg. at a time, beginning at zero, and it was usually kept at each level for 1 minute. The elongation is magnified 6 times by the lever. The pressure was raised to 300—400 mm. Hg.

Contracted Artery.—The total elongation in a strongly contracted artery is relatively small in amount; there is progressively increasing augmentation as the pressure is raised.

When the pressure is lowered the artery speedily returns to its original length—commonly indeed it becomes shorter than before.

A repetition of the process of raising the pressure sometimes causes the maximum elongation to occur much earlier than before, *e.g.*, with the rise from 50 to 100 mm., or from 100 to 150 mm. At other times there is simply a general increase in extensibility without the maximum elongation coming early (0—300 mm.). (See Fig. 32.)

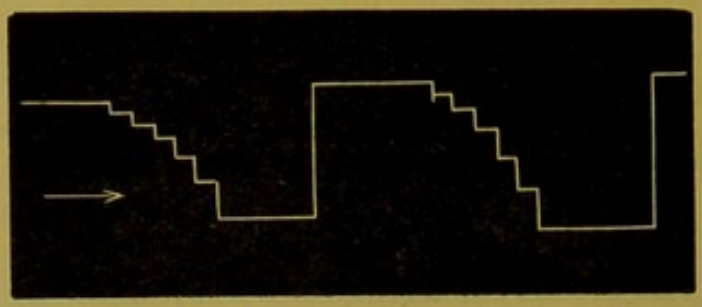

Fig. 32.—Elongation of contracted artery with rise of internal pressure, 0—300 mm. Length 16 mm.

Relaxed Artery.—A relaxed artery tested in the same way shows a vastly increased elongation when the internal pressure is raised. The maximum elongation occurs with the first or second rise; sometimes the second and third are equal in extent. (Fig. 33.)

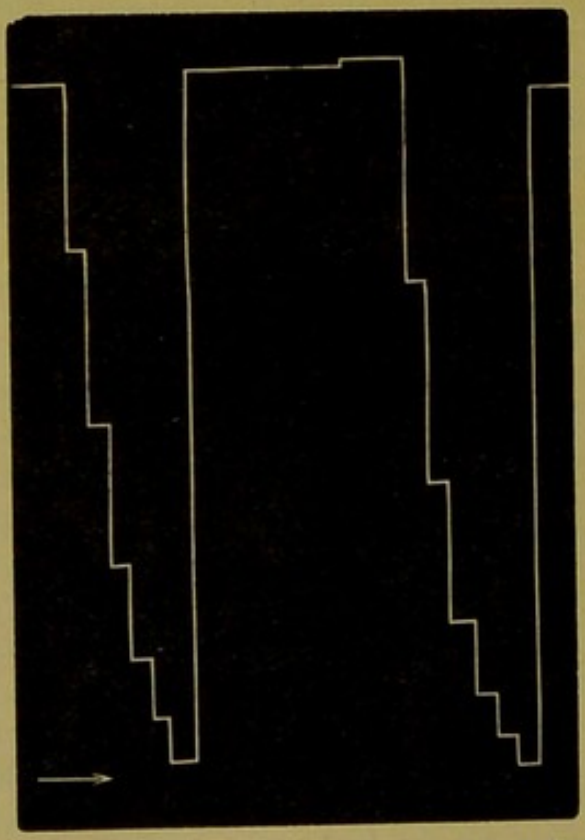

Fig. 33.—Relaxed artery which has been kept in blood for some days—till contraction had passed off. Length 21 mm.

A repetition of the rise of pressure shows an increased extensibility —especially for low pressures.

The artery was usually first tested in its contracted state, then relaxed by one or other of the methods mentioned, without any disturbance of the cannula or plug, and again tested in the same way as before. (Compare figs. 32 and 34.)

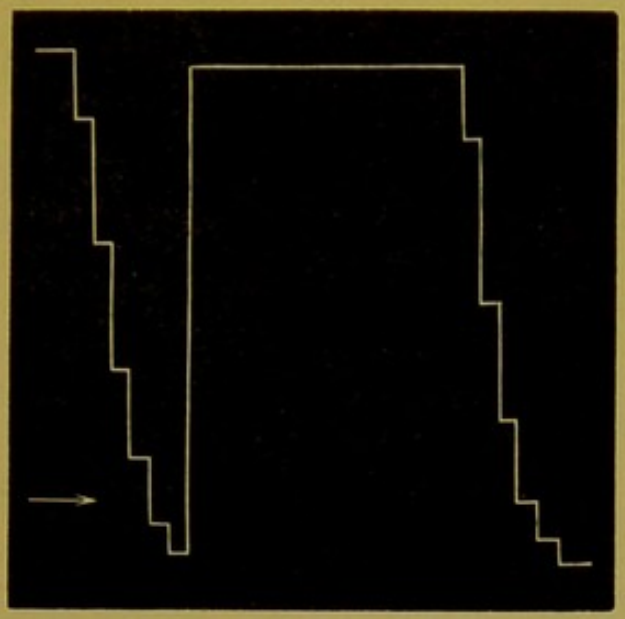

FIG. 34.—Artery, relaxed by keeping in warm chamber at 39° C. for 24 hours. Same portion of artery as was used (in contracted state) in fig. 32.

The relatively great tendency to elongation, even under low or moderate internal pressures, in a relaxed artery is important with regard to the tendency of certain arteries in the human body to become elongated and tortuous, apart from any recognisable structural change in the vascular wall. Elongation of the healthy temporal artery when relaxed is easily discernible even in young subjects; prolonged and frequently-recurring periods of relaxation tend to induce a more or less distinctly tortuous condition. Prolonged relaxation of the arteries of the uterus and mamma during pregnancy leads to similar changes in them.

Pulsatile Expansion of Relaxed and Contracted Arteries.—I have examined the pulsatile expansion of arteries when a rhythmical series of elevations and depressions of internal pressure is mechanically produced—in imitation of the rhythmical changes of pressure caused in a normal artery by the pumping action of the heart. This was tried when the pressure within the artery was at zero to begin with, or when a certain height of pressure had first been established; the apparatus for testing distensibility already described was used. Rhythmical compression of a rubber tube containing air in connection with that filling the interior of the artery was used to produce the pulsatile variation of pressure. The changes in the volume of the artery in response to the variations of pressure in its interior were shown by the to and fro movement of the oil in the horizontal graduated tube.

Relaxed Artery.—When there is zero pressure within the artery, to begin with a pulsatile rise of pressure of a certain amount causes an extensive pulsatile change in volume; *e.g.*, the oil in the graduated tube may move 15 mm. at each pulsation. (The tube is 3 mm. in calibre.)

When the pressure within the artery is originally 50 mm. Hg., the same rhythmical injection of air causes a much smaller expansion of volume, *e.g.*, 10 mm.

With higher pressures there is a smaller and smaller pulsatile expansion, *e.g.*, with an original pressure of 100 mm. the movement is 6 mm., with an original pressure of 200 mm. the movement is 3 mm.

Contracted Artery.—When a contracted artery is tested in the same way, the pulsatile expansion is very small in amount, and there is no very evident difference in the amount of pulsatile expansion at different (pre-existing) pressures. From the fact that (as has been described) a contracted artery is most distensible at a high pressure, one might expect that with a tolerably high pressure a further pulsatile rise would give larger expansion than when the pressure is at or near zero. But the rapid oscillations of pressure such as are now under consideration are much too brief in duration to have much effect; for the expansion of a contracted artery is slowly and gradually effected, and a rise of pressure, from whatever level it starts, has to last for some time if it is to exercise its full effect upon the arterial wall.

In accordance with this, in the intact arteries of men and animals there would be much less pulsatile expansion in a contracted artery than in a relaxed one; and in a relaxed artery expansion would be very much more extensive when the mean blood-pressure is low. Further, elongation would occur markedly in the relaxed artery as compared with the contracted one. And when a long stretch of artery is concerned the increase in length is very much greater than the increase in transverse diameter.

Illustrations of the above conclusions may be seen in healthy human arteries, the pulsatile changes in the relaxed temporal artery, also the extensive pulsation of arteries in inflamed parts, &c.

Various observers have described an apparent absence of transverse expansion at each heart-beat in arteries *in situ*. A. W. Volkmann* remarks that when an artery is laid bare in the living animal the only perceptible evidence of expansion of the vessel during pulsation is the tortuous form assumed by it at every pulse, and that the transverse expansion which (on other grounds) he believes to take place is inappreciable.

Lister† states that the arteries are not increased in diameter by the strokes of the powerful cardiac pump—"The surgeon when tying a

* 'Haemodynamik,' 1850.

† 'Brit. Med. Journ.,' 1879, vol. 1, p. 924.

large arterial trunk in its continuity does not find, on clearing the vessel of its sheath with the point of his knife, that he is dealing with a body that swells at every pulse, but with one of unvarying dimensions." Experimenting on the metacarpal artery (horse) with the circulation going on, he found that transverse measurements with suitable callipers showed no change as long as the limb was kept in any one position.

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