

## THE EFFECT OF PRESSURE ON NERVE CONDUCTION AND NERVE-FIBRE SIZE

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*(Received 17 January 1949)*

The purpose of this investigation was to determine whether the effect of circumferential pressure on the size of a nerve could be correlated with its effect on conduction, and in what way these effects were altered by ligation of the vessels supplying the nerve.

Bentley & Schlapp (1943) found that conduction was blocked in the sciatic nerve of the cat by pressures of 130 mm. Hg and over in 3 hr. In their experiments the nerve was compressed between rubber membranes inflated with air. After the application of pressures which completely blocked conduction, they report complete absence of recovery on removal of the pressure, and they conclude that such pressures applied directly to the nerve probably produce a block of conduction by 'deformation of nerve substance at the boundaries of the compressed part' (p. 81). Denny-Brown & Brenner (1944*b*), using metal clips to compress the sciatic nerves of rabbits, found that with small pressures (2-7 g. clips) conduction was unimpaired even after several days, but higher pressures (170-430 g. clips) produced paralysis, lasting 5-18 days, when the clip was left in place for 2 hr. They considered the effect on the nerve to be due entirely to ischaemia. In a previous paper they state 'the compartmented physical structure of a large nerve, like the sciatic, is such as to protect some of the longitudinal vessels from external pressure' (Denny-Brown & Brenner, 1944*a*).

Edwards & Cattell (1928) state that 'the influence of pressure in blocking conduction is entirely dependent upon deformation of the tissue due to its unbalanced action'. Weiss & Hiscoe (1948), in describing acute experiments on the nerves of the rat compressed by arterial sleeves, state: 'The first marked effects appear only after several hours. They consist of a narrowing of the fibres in the constricted stretch'.

Gasser & Erlanger (1929) blocked the A fibres in the sciatic nerve of the frog with pressure of 25 lb./sq.in. The pressure was applied after threading the nerve through a vascular sleeve in the manner used by Meek & Leaper (1911). In a paper dealing with the effects of asphyxia produced by surrounding a nerve with hydrogen, Cooper (1923) suggested that the oxygen was not essential for

the transmission of the impulse, but was required for the oxidation of breakdown products. In a later paper Gerard (1930) did not support this view, and suggests, from the results of his many observations on the effect of an atmosphere of nitrogen on conduction, that 'normal membrane potentials (and other properties) are dependent on oxidations'.

Investigating the sensory changes in the human arm Lewis, Pickering & Rothschild (1931) found that, with their special clamp which compressed the nerves locally but did not occlude the circulation to the limb, they could produce sensory changes with pressures of 60–70 mm. Hg, but that sensory changes were not produced when an encircling cuff was placed round the limb and inflated to a similar pressure. Localized pressure equal to 150 mm. Hg on the ulnar nerve caused a muscular palsy within 30–40 min. which they attributed to a loss of blood supply within the compressed region of the nerve and not to direct pressure on the nerve fibres.

In a previous paper (Causey, 1948) it was shown that circumferential pressure could be applied to a nerve by direct mercury pressure in a glass chamber sealed with a gel of sodium alginate, and that pressures of 80 mm. Hg and over produced a decrease of 28% in the cross-sectional area of the nerve at the site of pressure; part of this decrease was due to a decrease in the mean fibre size, and a larger part to compression of the interstitial area. It seemed that further evidence on some of the points quoted above could be obtained by using the mercury chamber for the application of pressure, and determining the acute effect of localized pressure on nerve size, nerve-fibre size, and conduction.

#### METHOD

All the experiments were carried out on the nerve to the medial head of the gastrocnemius (N.G.M.) of rabbits, anaesthetized with nembutal and ether. All the rabbits were healthy adults; no attempt was made at uniformity of sex, weight or breed. The general method used will be described under two heads: (1) a method of applying circumferential pressure to the nerves, (2) a method of preparation of photographs of sections for measurement and counting.

(1) A small glass chamber (2.5 × 1 cm.), with an open top, was made at the end of a glass tube; the tube was connected to a mercury manometer by rubber tubing with a control clamp between the chamber and the manometer; the whole was filled with mercury. In the top of the sides of the glass chamber were two smooth grooves 1 mm. in depth. The N.G.M. was dissected from the main tibial trunk and the chamber slipped underneath it from the side, so that the nerve lay at the bottom of the grooves. The top of the chamber and the grooves were coated with a gel made of sodium alginate and water. A closely fitting lid was coated on the edges of its undersurface with the same gel and clamped firmly in place on top of the chamber. When the nerve was placed well down in the grooves, release of the control clamp caused the mercury to pass between the nerve and the lid and circumferential compression of a short segment of the nerve was obtained. The height of the mercury head above the level of the nerve was varied from 45 to 670 mm. in different experiments. The stimulating electrodes, consisting of two silver wires embedded in perspex, were placed under the nerve, proximal to the chamber. Muscle response was either observed visually or recorded on smoked paper by a pointer attached to a torsion wire. The tendon of the medial head of gastrocnemius was separated from the calcaneal tendon and attached to the recording lever by a hook and thread.

(2) By placing padding underneath the operated limb a trough was formed between the femur and the biceps muscle, in which the nerve ran longitudinally. At the end of the observational period a modified Flemming's solution, consisting of 15 c.c. 1% chromic acid, 4 c.c. 2% osmic acid and 1 drop of glacial acetic acid, was poured into the trough around the nerve. After maintaining the pressure on the nerve for 5 min. the control clamp was closed and the chamber removed from the nerve. Fixation was continued *in situ* for a further 25 min. The N.G.M. was then removed and fixation continued in a tube of Flemming's solution, with a waxed cork, for 24 hr.

After dehydration in alcohols the nerve was embedded in paraffin, cut at  $5\mu$ . and stained by a modified Weigert technique (Gutmann & Sanders, 1943). Sections were selected from sites above, at, and below, the region of pressure and photographed direct on to bromide paper at  $\times 750$  magnification. On these photographs measurements were made of the total cross-sectional area of the nerve bundle by means of a planimeter, the mean of three readings being used. The nerve fibres were measured by fitting each fibre into a series of circles marked on a thin sheet of perspex; each circle represented the area corresponding to diameters of 2, 4, 6, 8, etc.,  $\mu$  multiplied by 750. Each fibre was pricked off with the needle of a mechanical counter as it was fitted into its size group. A fibre count was thus obtained classifying the fibres into groups at  $2\mu$ . intervals. The root mean square diameter ( $D$ ) was used to give a weighted mean (Sanders & Young, 1946). Only myelinated fibres were measured and the total diameter of the fibre (axon and myelin) was used. In control experiments it was found that the difference in  $D$  at different levels in the same nerve showed a mean of  $0.17\mu$ . with a standard deviation of  $\pm 0.13\mu$ .

*Block of conduction and nerve-fibre diameter.* In a series of fourteen rabbits, pressures varying from 45 to 670 mm. Hg were applied for 10 min. (in some of the cases submitted to higher pressure the time was varied). The nerve was stimulated proximal to the pressure chamber before and at the end of the pressure period, by short bursts from an induction coil, the strength of the stimulus being adjusted, before the application of the pressure, to produce a maximal twitch. The presence or absence of a muscle twitch was noted and fixative solution then poured round the nerve as described above. The N.G.M. of the opposite limb was dissected out and fixed in the same manner, without the application of pressure. The root mean square diameter above, at, and below, the region of pressure and the total nerve-bundle area were determined for the compressed nerve. The root mean square diameter and nerve-bundle area of the control nerve were also determined to show that there was a diminution in size at the site of pressure when compared with the control nerve as well as in relation to the other levels in the same nerve.

## RESULTS

Pressures of 125 mm. Hg and over produce a significant decrease in the mean fibre diameter at the site of pressure, when this diameter is compared with the mean diameter either above or below the site of pressure, or when compared with the mean diameter of the control nerve. In all these cases there was no visible muscle twitch at the end of the period of pressure (Table 1). When the muscle twitch is recorded on a kymograph (see Table 2) the time needed for complete obliteration of the recorded twitch is found to be longer, with an average of 18 min.

In Table 1 the results for all fourteen animals are tabulated. Below 100 mm. Hg pressure there is no blockage of conduction, but there is a small decrease in fibre size, even at 75 mm. pressure. Between 100 and 125 mm. Hg pressure there is variation, both in the effect on fibre size and in the effect of conduction in different animals. The mean of the differences between the root mean square diameters of the fibres at and below the region compressed for those nerves in

which conduction was blocked is  $1.4 \mu.$ , or 12% of the mean of the root mean square diameters of the control nerves. The total area of the nerve bundle, measured with a planimeter, showed for all nerves in which conduction was blocked, a mean decrease in size of the nerve bundle in the compressed region as compared with the area of the nerve bundle below the compressed region. This decrease amounted to 26% of the mean area of the control nerves.

TABLE 1. Root mean square diameter ( $D$ ) measured above, at, and below, the region of pressure, and from a control nerve of the opposite limb. + indicates muscle response on stimulation of the nerve proximal to the pressure.

Rabbit no.	Pressure (mm. Hg)	Time (min.)	$D$ above press	$D$ at press	$D$ below 'press	$D$ control	Conduction
1	45	10	13.2	13.2	13.5	13.2	+
2	50	10	11.2	11.7	11.1	12.2	+
3	75	10	13.0	12.2	12.7	13.0	+
4	100	10	11.7	10.7	11.8	11.7	-
5	100	10	11.7	11.6	11.9	11.8	-
6	105	10	14.2	14.1	14.1	14.1	+
7	125	10	11.7	10.2	11.5	11.1	-
8	150	10	10.8	9.8	11.2	10.5	-
9	250	10	13.1	11.3	13.4	11.4	-
10	370	30	12.5	11.6	13.0	11.9	-
11	470	7	12.0	11.3	12.2	12.3	-
12	500	8	12.4	11.3	12.6	12.1	-
13	560	15	12.0	10.5	12.0	11.6	-
14	670	10	13.7	11.7	14.4	12.7	-

#### *The time required to produce complete block*

To investigate more accurately the time necessary to block conduction a further series of experiments was carried out in which the muscle response was recorded throughout the experiment. The method of application of pressure and the histological technique were identical with the first series, but when the N.G.M. was dissected out, the tendon of the medial head of the gastrocnemius was separated from the calcaneal tendon and the muscle belly of the medial head separated, almost to the knee, from the lateral head. The tendon was then attached by a hook and thread to a lever mounted on a torsion wire. The excursions of the lever were recorded on a slow-moving drum. The stimulating electrodes were held in a clamp and left in place throughout the experiment. The stimuli were square waves of 1 msec. duration, at approximately one every 4 sec., the voltage was adjusted at the beginning of each experiment to give a maximal twitch. The opening of the control clamp between the manometer and the pressure chamber was marked on a base-line by a manually operated switch. Release of pressure was marked on the same base-line.

With pressures of 100 and 150 mm. Hg no failure of muscle response was recorded in 49 and 54 min. respectively. Fig. 1*a* shows the record at the beginning of compression by 100 mm. Hg and 48 min. later in the same preparation. There is only a slight decrease in the height of the twitch recorded. But in another animal (no. 16) block was obtained with 150 mm. Hg pressure in 20 min. With all pressures from 200 to 400 mm. Hg there was failure of conduction. The average time required for the development of block in fifteen experiments was 20 min. All the nerves recovered their ability to conduct an impulse when the pressure was released by removal of the lid of the chamber (see below). The mean time for recovery was 17 sec.

In Table 2 the times of onset of block and of recovery in twenty experiments are shown. The times required to produce block vary from 35.5 min. for a pressure of 300 mm. Hg (rabbit no. 25) to 3.6 min. for a pressure of 400 mm. Hg

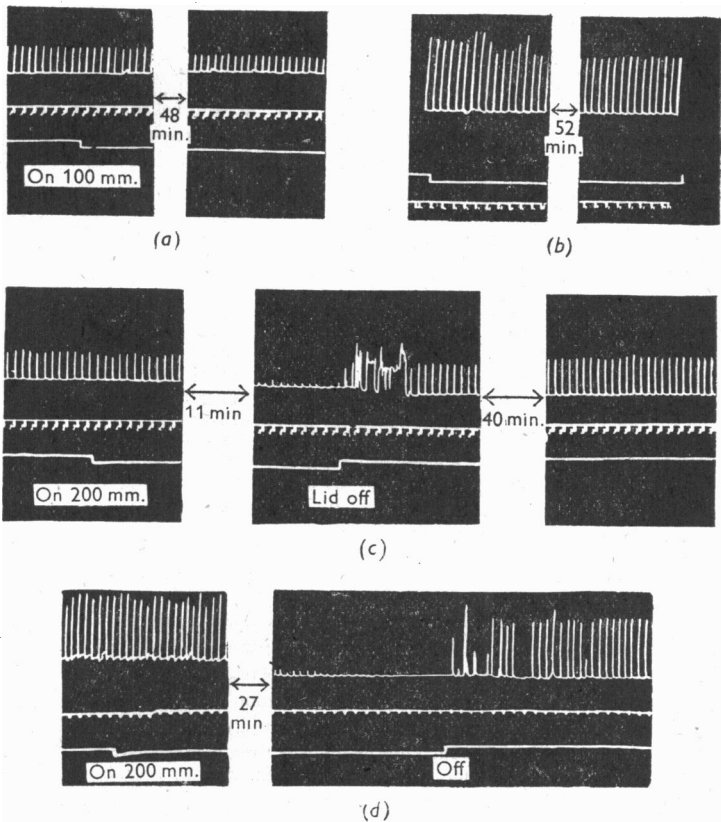


Fig. 1. Kymograph records of twitch from medial head of gastrocnemius of rabbit. Square wave stimulus of 1 msec. duration at intervals of 4 sec. Time marker 5 sec. The figure between the portions of tracings shows the approximate time interval of the deleted portion of the record. (a) At time of application of 100 mm. Hg pressure and 48 min. later. (b) At the beginning and end of local application of air pressure to the N.G.M. under a head of 240 mm. Hg. (c) At time of application of 200 mm. Hg pressure, followed by failure of muscle response after 11 min., recovery of muscle response on removal of lid of chamber and maintenance of response 40 min. later. (d) After application of 200 mm. Hg pressure. Failure of twitch after 27 min. and recovery on removal of lid of chamber.

(rabbit no. 27). In this latter case the nerve had been blocked by the same pressure in 16.5 min. 2 hr. before, and allowed to recover (Barlow & Pochin, 1948). The onset of block was affected by the length of exposure and the amount of manipulation; thus, in cases where the chamber had to be reapplied because of leakages at the seals there was a tendency for a shorter time interval before

block than in the cases where the first application of the chamber was successful. Reapplication of pressure following a previous block and recovery reduced the time preceding onset of block.

TABLE 2. Duration of pressure required to produce block, and time for recovery of conduction

Rabbit no.	Pressure (mm. Hg)	Time for onset of block (min.)	Time for recovery (sec.)	Rabbit no.	Pressure (mm. Hg)	Time for onset of block (min.)	Time for recovery (sec.)
15 L	100	Not blocked in 49 min.		21 L	270	13	20
16 L	120	Not blocked in 50 min.		22 L	270	24	5
15 R	150	Not blocked in 54 min.		23 R	280	18	22
16 R	150	20	10	24 L	290	14	5
17 R	200	24	5	24 R	300	18	22
18 L	200	6	10	25 L	300	36	5
19 L	240	29	40	25 R	300	21	5
19 R	250	21	40	26 L	400	9	20
20 R	250	27	65	27 R	400	17	5
21 R	270	16	5	27 R	400	4	53

(2 hr. later)

Fig. 1 *c, d* show sections of the continuous records taken from preparations with the nerves subjected to 200 mm. Hg pressure. The height of the record slowly diminishes after opening the control clamp and after 12 and 24 min. there is no further twitch. On removal of the lid of the chamber and release of pressure there is a very rapid recovery of the muscle twitch. In Fig. 1 *c* a further record is shown of the muscle twitch 40 min. after the release of pressure, at which time the height of the record is fully maintained.

Experiments were also carried out in which the nerve was surrounded by air under pressure produced by a column of mercury. The glass chamber and distal portion of the manometer tubing were left full of air when the N.G.M. was placed in position in the groove and the lid clamped in position, on opening the control clamp the manometer level first of all fell rapidly as the air was compressed, but equilibrium was quickly attained and by suitable adjustment of the amount of air left in the tubing it was possible to have the nerve completely surrounded by air under pressure. This equilibrium was difficult to maintain, but it was possible to show that there was no block of conduction within the time interval which would cause block with mercury surrounding the nerve. Fig. 1 *b* shows the tracing taken at the beginning of air pressure and 52 min. later, when the mercury head was 240 mm. The height of the twitch has only diminished by 5% and there is no evidence of block at a time well beyond the maximum time for complete block with mercury pressure.

#### *Recovery of nerve-fibre size*

The rapid recovery of conduction, after release of pressure by removing the lid of the chamber, has already been mentioned. In view of this the nerve size and nerve-fibre size after recovery were investigated. In three rabbits the left N.G.M. was dissected out and submitted to pressures of 100, 210 and 440 mm. Hg respectively. After 10 min. compression the trough in the limb was flooded with

Flemming's solution, whilst the pressure was maintained for a further 5 min. On the right side of the same animals the same pressures were applied for the same time, the chamber was then removed, the nerve was left for 10 min. and then flooded with Flemming's solution.

The root mean square diameters above, at, and below, the site of pressure, in these six nerves are shown in Table 3, and the total nerve areas at the same levels in Table 4. These figures show that with a pressure of 100 mm. Hg there is no significant difference either in the nerve size or nerve-fibre size between the three levels on the two sides. With pressures of 210 and 440 mm. Hg there was a decrease both in the nerve size and in the nerve-fibre size in the nerves when fixed with the pressure maintained, but the nerves released before fixation showed the same nerve size and nerve-fibre size at all levels.

TABLE 3. The root mean square diameter ( $D$ ) when pressure was maintained during first 5 min. of fixation and when pressure was released before fixation

Rabbit no.	Pressure (mm. Hg)	Time (min.)	Pressure maintained			Pressure released		
			$D$ above press	$D$ at press	$D$ below press	$D$ above press	$D$ at press	$D$ below press
5	100	10	11.7	11.6	11.9	11.9	11.8	11.8
28	210	10	13.6	12.2	13.4	13.3	13.3	13.6
29	440	10	11.2	10.0	11.1	10.8	10.9	10.8

TABLE 4. The total nerve area at  $\times 750$  magnification when pressure was maintained during first 5 min. of fixation and when pressure was released before fixation

Rabbit no.	Pressure (mm. Hg)	Time (min.)	Pressure maintained			Pressure released		
			Area above press (cm. <sup>2</sup> )	Area at press (cm. <sup>2</sup> )	Area below press (cm. <sup>2</sup> )	Area above press (cm. <sup>2</sup> )	Area at press (cm. <sup>2</sup> )	Area below press (cm. <sup>2</sup> )
5	100	10	460	464	462	529	521	475
28	210	10	484	408	482	535	502	528
29	440	10	451	313	444	413	392	423

### *Recovery of conduction*

The recovery of conduction after removal of the pressure was investigated: (1) On release of pressure and admission of air around the nerve by closing the control clamp and removing the lid of the chamber. (2) On release of pressure by dropping the manometer, so that the head of mercury was at the level of the chamber, and thus with the control clamp open the pressure on the nerve was reduced to zero. The nerve was still surrounded by mercury and by the alginate seals for a distance of 1 cm. (3) The manometer was lowered and the mercury was emptied out of the whole system so that pressure was removed and air admitted to all parts of the nerve except the small areas covered by alginate seals. Continuous records were taken as before.

Release of pressure by removal of the lid involved first closure of the clamp between the manometer and the chamber, then release of the clamp holding the lid of the chamber in place, and finally the removal of the lid. The moment of removal of the lid was recorded as the time of release of pressure. All the nerves recovered their ability to conduct an impulse as soon as the lid of the chamber was removed. The mean recovery time was 17 sec. Table 2, column 4, shows the estimated time of recovery in seventeen preparations. The times vary from less than 5 to 65 sec. This recovery time is so short that it is clear that,

with the record on a slowly moving drum and the lack of a clear-cut end-point in a manipulation which takes a few seconds, they must be approximate. The

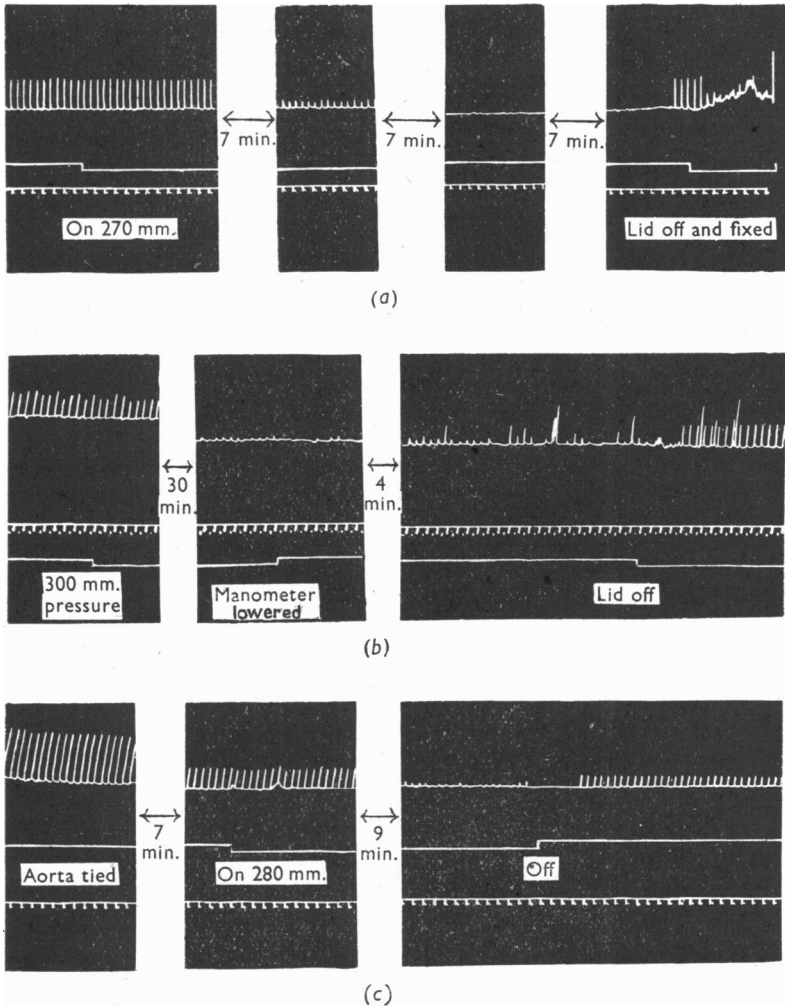


Fig. 2. (a) Effect of application of 270 mm. Hg pressure. No recovery 7 min. after dropping manometer pressure to zero. Recovery on removal of lid of chamber. Fixative solution then poured round the nerve. (b) Failure of muscle twitch 30 min. after application of 300 mm. Hg pressure. Partial recovery 4 min. after lowering of pressure to zero. Complete recovery after removal of lid. (c) Aorta and pulmonary artery tied at the base of the heart. 280 mm. Hg pressure applied locally, 7 min. later. Failure of muscle response after 9 min. and recovery on removal of lid.

average recovery time recorded of 17 sec. is therefore only an indication of the order of the recovery time.



When the manometer was dropped and the pressure reduced to zero recovery was delayed and showed marked irregularity. Fig. 2*a* shows records from a preparation in which, after obtaining block in 16 min., the manometer was dropped. The muscle gave no response for 10 min., at the end of which period removal of the lid was followed by immediate recovery. In Fig. 2*b*, however, after obtaining block in 36 min. the manometer was lowered to zero pressure, and after a period of 6 min. there was an incomplete recovery of the muscle twitch. On removal of the lid the twitch became equal to that obtained at the beginning of the record.

When the mercury was emptied out of the manometer and chamber, the recovery of the twitch was complete. The time and manipulation involved in removing the mercury in this way made it difficult to estimate the time of recovery with any accuracy.

### *Blood supply*

The vascular supply of the sciatic nerve of the rabbit has been fully described by Adams (1942). Large, longitudinal vessels come from the inferior gluteal and popliteal arteries and are reinforced by small lateral vessels from the perforating and muscular branches. In the present experiments the latter small branches were divided in dissecting the N.G.M. from the tibial nerve, but the longitudinal vessels remained. Ligature of the gluteal and popliteal arteries, ligature of the common iliac artery, or ligature of the abdominal aorta, with or without the inferior vena cava, did not appreciably alter the time at which block occurred under mercury pressure and even after these ligatures conduction recovered within seconds on removal of the lid of the chamber. To exclude any possibility of a blood supply by way of the vessels of the abdominal wall or spinal cord the aorta was ligated as it leaves the left ventricle. The muscle and nerve were dissected out first, then the chest was opened in the middle line and a double thread passed through the transverse sinus of the pericardium on an aneurysm needle, and tied firmly round the aorta and pulmonary artery. This method of stopping the circulation seemed most suitable for these experiments because (1) the time of blockage of the circulation could be recorded with accuracy; (2) it avoided the possible poisoning of the nerve by overdose of chloroform or other narcotic; and (3) it avoided local exsanguination.

In trial experiments without localized pressure it was found that muscle twitches of diminishing height were obtained for 30–40 min. after tying the ligature round the aorta. When localized pressure was applied to the N.G.M. after tying these vessels it was found that the twitch was rapidly obliterated, but recovered, though diminished in height, on release of the pressure. The time taken by these phenomena varied considerably, depending on the interval between the placing of the ligature and the successful application of pressure. Table 5 shows the time relation of the events in a typical experiment in which a pressure of 250 mm. Hg was applied 5 min. after the ligature of the aorta. Conduction was blocked in 10 min. and recovery occurred 7 min. after the release of pressure.

In rabbit no. 30, with a pressure of 250 mm. Hg, block was obtained in 10.5 min. and reappearance of twitch within 20 sec. of release of pressure, whilst in rabbit no. 31 a pressure of 220 mm. Hg blocked conduction in

4.5 min. and 4 min. elapsed before the return of a recorded twitch. Fig. 2*c* shows sections from the record of rabbit no. 30. A steady decrease of the height of the twitch is seen between the time of tying the aorta and the beginning of the application of pressure. This decrease in height continues for 10 min. until there is no recorded twitch. Within a few seconds of removal of the lid of the chamber the muscle twitch returns, even though there is no circulation.

TABLE 5. Time-table of the course of events when local pressure was applied to the N.G.M. after ligation of the aorta and pulmonary arteries at the base of the heart

Rabbit no. 32	Nerve to the medial head of gastrocnemius dissected out and threshold determined
12.17 p.m.	Aorta and pulmonary artery tied
12.20 p.m.	Nerve conducting throughout its length
12.22 p.m.	Pressure of 250 mm. Hg applied
12.32 p.m.	No muscle reaction on stimulation <i>proximal</i> to chamber Muscle reaction on stimulation <i>peripheral</i> to chamber
12.33 p.m.	Pressure chamber removed
12.40 p.m.	Nerve conducting throughout its length

#### DISCUSSION

On application of localized circumferential pressure to a small nerve bundle in the rabbit, decrease in the total nerve size and in fibre size is first demonstrable at pressures between 80 and 100 mm. Hg. Block of conduction can be demonstrated, in acute experiments, with pressures of 150 mm. Hg and over, and recovery from such block occurs rapidly in all cases when the pressure is removed and air is admitted to the nerve. These observations clarify certain discrepancies in the findings of previous investigators which are quoted in the introduction to this paper. If a nerve, with a circular or ovoid cross-section, is compressed between two rigid surfaces, its axis parallel to the compressing surfaces will become extended, and the applied pressure will not be evenly distributed throughout the nerve bundle. With the fluid pressure used in the present experiments the transverse section of the compressed region remains circular (Causey, 1948) and the pressure is uniformly distributed around the periphery of the bundle. The pressure gradient is along the radius of the section, causing closer packing at the periphery than at the centre of the nerve. By using the N.G.M. the compartmented nature of the sciatic nerve has been avoided and the protection of longitudinal vessels afforded by this type of structure (Denny-Brown & Brenner, 1944*b*) cannot occur. These factors probably explain the much longer time interval observed by these authors and also by Bentley & Schlapp (1943), before the onset of block, and the absence of rapid recovery in their experiments.

Narrowing of the fibres in the constricted stretch of nerve was found by Weiss & Hiscoe (1948), only after several hours. Our experiments indicate the onset of narrowing very much earlier and are not in accordance with their suggestion that the endoneurial fluid is in a state of very high hydrostatic

pressure. The gradual diminution of the muscle twitch before complete block, shown in the tracings, is presumably due to the peripheral fibres being affected before those in the centre of the nerve.

The reduction in nerve-bundle size and nerve-fibre size which results from the application of pressures sufficient to produce block of conduction, and the recovery after release of pressure show that there is deformation of nerve substance, as suggested by Bentley & Schlapp (1943). But the observations on the greater relative rate of recovery when the compressed region is exposed to air than when the pressure is released without admitting air to the compressed region, indicate that it is the presence of oxygen, and not the recovery of shape, which is essential to the recovery of conduction. This is supported by the observation that pressure on the nerve by air does not produce block within 52 min.

The results of the experiments reported here find their closest parallel in the results obtained by Lewis *et al.* (1931), in experiments on motor and sensory changes in the human arm. With localized pressure of 150 mm. Hg on the ulnar nerve they observed muscular paralysis in 30 min., with recovery in 20–30 sec. Our results suggest that, at least in experimental animals, the primary condition is one of local anoxia caused by local deformation of the nerve bundle, and recovery is rapid when the nerve again obtains access to oxygen.

#### SUMMARY

1. Local application of pressure by mercury to a nerve caused failure to conduct an impulse to the muscle when the stimulus was applied above the site of pressure. All pressures above 150 mm. Hg produced block. The average time required to obliterate the muscle response was 20 min.

2. Recovery of conduction took place within 5–65 sec. after the release of pressure and the admission of air.

3. Nerve-fibre size and nerve-bundle size were diminished at the site of pressure, and there was recovery of nerve-fibre size and nerve-bundle size 10 min. after the release of pressure.

4. Application of pressure by air did not cause failure to conduct an impulse in 52 min.

5. The circulation of the blood was stopped by ligation of the aorta and pulmonary artery at the base of the heart. After this was done it was still possible to block conduction along the nerve by localized pressure, and to obtain recovery on the release of pressure, before the final death of the preparation.

6. It is concluded that, in acute experiments, the failure to conduct an impulse after local pressure, is caused by local anoxia.

We wish to thank Prof. J. Z. Young for his advice, and Mr F. J. Pittock, Mr J. Armstrong and Mr J. E. Marner for technical help.

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