J. Physiol. (1951) 114, 56–70

OBSERVATIONS ON THE BLOOD FLOW IN THE SPINAL CORD OF THE RABBIT

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(*Received* 18 *August* 1950)

Cobb & Talbot (1927) presented evidence that in the experimental animal heightened activity of a portion of the brain resulted in an increased vascularity of that region. A year later Fulton claimed to have evidence of a similar occurrence in man. The introduction by Gibbs (1933) of the heated thermocouple method of measuring blood flow has facilitated the investigation of the problem in the surviving animal. As a result it is now known that regions of the brain entering upon a temporary increase of activity acquire an augmented blood flow for the duration of such activity, and moreover that the phenomenon may be precisely localized (Gerard & Serota, 1936; Santha & Cipriani, 1938; Schmidt & Hendrix, 1938).

No similar investigation relating to the spinal cord appears to have been made, and it was to this problem that attention has been directed in the present work. Increased activity of the spinal cord was brought about, for example, by pinching the heel, or stimulating the central end of the cut sciatic nerve, and the temperature changes in the active spinal cord segments accompanying the reflex activity were recorded. Such stimulation of the sciatic nerve is known to release adrenaline (Tournade & Chabrol, 1926). The effect of intravenous infusions of adrenaline has therefore been investigated. Inhalations of CO_2 were also given as Schmidt & Hendrix believed CO_2 to be the factor regulating local cerebral blood flow. To show that the observed temperature changes were not due to heat production in contracting muscles or due to movement of the animal, further experiments were done on anaesthetized animals when fully curarized. A brief report of the work has already been given (Field, Grayson & Rogers, 1949).

MATERIAL AND METHODS

Variations in blood flow were measured by thermocouples of the heated type (Gibbs, 1933) or a modification of the cooled couple of Schmidt & Pierson (1934). The principle in both methods is to maintain the recording thermocouple embedded in the tissue under investigation at a temperature

either above that of the afferent blood or below it. With the former an increased blood flow results in cooling of the recording thermocouple producing a fall in recorded temperature, whilst with a thermocouple below the temperature of the blood an accession of relatively hot blood produces a rise in the temperature record. In both cases sensitivity depends upon the difference in temperature between the recording point and the afferent blood. In the present investigation a certain cooling of the exposed spinal cord to below the temperature of the aortic blood (0·2–0·5° C.) was inevitable and sometimes this difference provided sufficient sensitivity for blood flow variations to be recordable. Much more striking records were, however, obtained when the temperature difference between cord and blood was increased by sucking a uniform stream of air through the wound as described below.

Thermocouples were of 36 S.W.G. (0·193 mm.) copper and constantan wire, and carefully shielded by 'cystoflex' sheathing. Attention to detail (e.g. the avoidance of ammonium salts in soldering flux) was found to be of prime importance in obviating artifacts. The cold junction was immersed in oil in a Dewar flask at a temperature a few degrees below that of the animal. Each circuit consisted of a d'Arsonval galvanometer, a resistance box, a cold junction, a recording couple and a control box. The latter made it possible to switch different recording couples into the circuit, to change the cold junction (e.g. to record spinal against abdominal temperature) or to short-circuit the galvanometer.

All long leads were of co-axial cable; other sheathed connexions were suspended in air as far as possible. The various components of the circuit stood on earthed plates and the animal table was also earthed. Temperature sensitivity could be varied by means of the resistance box and was maintained at about 15 cm. scale deflexion to 1° C.

Heating of the Gibbs type thermocouple was carried out by means of a low voltage alternating current from a step-down transformer (P. 210 V., S. 6 V.) the primary of which was fed from a 'Variac' transformer. A milliammeter checked constancy of the heating current.

Galvanometric deflexions were recorded kymographically by a modification of the method proposed by Schmidt & Pierson (1934). A more detailed consideration of the technique will be presented by one of us (A.F.R.) elsewhere.

All observations were made on adult rabbits (1·8–2·5 kg.), anaesthetized by pentobarbitone sodium ('nembutal') given intravenously. The usual initial dose was 40–50 mg./kg. body weight. Altogether 60 animals were employed. A tracheal cannula was inserted and the blood pressure recorded from the carotid artery. A thermocouple was placed in the posterior part of the abdominal cavity close up against the aorta. In some cases a balloon was inserted to record the intra-abdominal pressure. Lumbar and thoracic laminectomies were performed, the former just above the level of the iliac crests, each exposing about 5–8 mm. of spinal cord. The recording couple was inserted in such a way that its point might be expected to lie in or near the anterior horn of grey matter. It was repeatedly found that if a brisk movement of the leg did not occur when the couple was inserted the preparation would be unsatisfactory. Stimulation after reinsertion of the couple at the correct level usually gave a good response. At the opposite end of the wound a small thick-walled rubber tube was stitched into place so that the open end lay in the centre of the wound a few mm. above the cord surface, which was protected by a thin layer of cotton wool. Both thermocouple and tube were rigidly fixed in place by stitches previously inserted through the muscles and their fascial sheaths on either side of the spine, so that even gross movements of the animal could not result in their displacement relative to the cord. The wound was loosely packed with cotton wool and the skin edges brought together except at the centre where a glass ring was sewn in place to leave an opening of constant size for the admission of air. These procedures took 1½–2 hr. The animal was left undisturbed for about an hour and if found to be reacting satisfactorily heparin was administered (3 mg./kg.), the blood pressure manometer connected up and recording commenced.

Where records from both heated and cooled thermocouples were desired, a thermocouple with attached heating element was inserted at the beginning of the experiment. The same thermocouple could thus be cooled by suction, or heated, as required.

RESULTS

The relation between intra-peritoneal and cord temperatures

The body temperature of the experimental animal could be varied at will by means of heaters fitted to the operating table. The heaters were adjusted to maintain the body temperature between 38° and 40° C.

When the laminectomy wound was closed, the resting temperature of the spinal cord was 0·2–0·5° C. lower than the intraperitoneal temperature. Except where the heated thermocouple was used, additional cooling was usually necessary, and was obtained by drawing air through the wound at a constant rate by means of the rubber tube and glass ring described above. Most of the experiments were thus performed with the local cord temperature 2–3° C. below that of the peritoneal cavity.

It was usually found difficult to maintain an absolutely steady body temperature. Using the table heaters, however, the range of variation was limited to 0·5° C. In a typical experiment the body temperature rose and fell in a series of long, slow undulations where the periods of increase and decrease each occupied about 25 min. The cord temperature varied similarly, but to a less extent, and lagged some 5 min. behind body temperature.

It seemed likely that these effects were the direct result of alterations in blood temperature. There was no evidence that changes in body temperature had any direct effect on the blood flow in the spinal cord, although evidence will be presented later to show that changing body temperature may influence the sensitivity of response to peripheral stimulation.

The effect of pentobarbitone sodium anaesthesia

Pentobarbitone sodium ('nembutal') (40–50 mg./kg.) administered intravenously, was the anaesthetic used in all experiments. Maintenance doses produced changes in blood temperature which were accompanied by changes in the spinal cord temperature. Two types of blood pressure response were noted in different animals: (*a*) In the first the blood pressure fell abruptly, then slowly mounted to a level higher than the former level. (*b*) In the second group the blood pressure fell abruptly, then climbed slowly, but never beyond the former level.

Two types of reaction were recorded from the spinal cord: (i) Where, after an initial fall, the blood pressure never rose above former levels, a transient fall in cord temperature occurred (Fig. 1A). When heated thermocouples were used, the cord temperature rose slightly. The return to resting levels was usually complete within 3 min. In some cases the effect on the cord was very slight, even absent. In a typical instance, where the blood pressure fell from 100 to 60 mm. Hg. the cord showed no change whatever (Fig. 1C). (ii) In seven experiments, where after its initial fall the blood pressure rose beyond its

original resting level, the cord temperature also fell, remaining low for long periods or very slowly returning towards its resting level (Fig. 1 B).

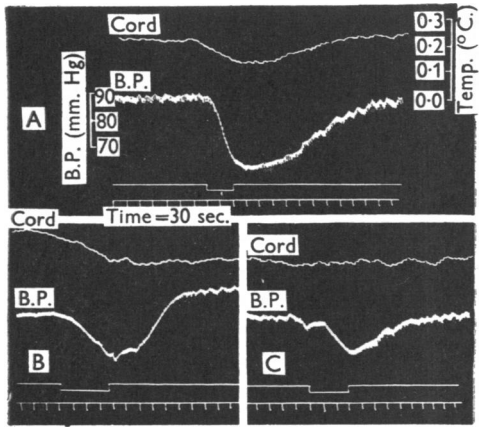

Fig. 1. The effect of intravenous pentobarbitone sodium (nembutal) on the spinal cord circulation. Unheated thermocouples in the lumbar cord.

The evidence thus indicated that intravenous pentobarbitone sodium usually produced a temporary diminution in cord blood flow, associated with the initial drop in blood pressure. Where the blood pressure was ultimately raised, however, the diminution in blood flow was more prolonged and must be assumed to have been the result of an active vasoconstriction.

Blood flow in the cord in relation to gross blood pressure changes

Observations were made which suggested that under certain conditions the spinal cord circulation might passively follow changes in blood pressure.

The tracings shown in Fig. 2, for example, are from an animal in poor condition exhibiting periodic breathing of Cheyne-Stokes type. Cord temperature changes reflected those of the blood pressure, rising when the blood pressure rose, falling when the blood pressure fell, and showed the same time relationship.

In other instances of spontaneous blood pressure change such as occasionally occurred just before death, there was a similar apparently passive relation between spinal cord temperatures and blood pressure, with a time lag of up to 30 sec.

In 6 animals temperature and circulatory changes were recorded at the moment of death. Where the unheated thermocouple was used (4 animals) a rapid fall in cord temperature accompanied the cessation of circulation without any comparable sudden change in the intraperitoneal temperature which continued to fall slowly. (Fig. 3). In two experiments where the heated

thermocouple was used, the cord temperature rose sharply when the terminal fall of blood pressure occurred. There can be no doubt, therefore, that the recorded effects were due to blood flow change.

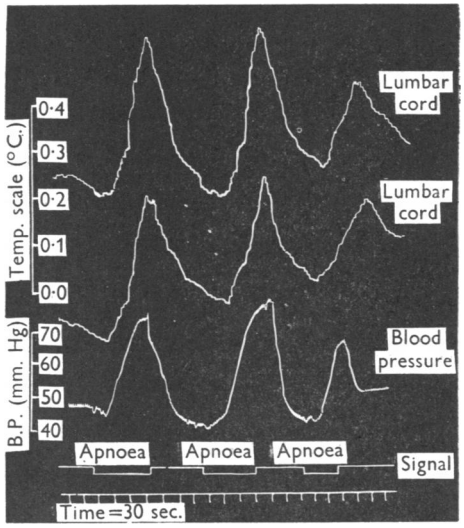

Fig. 2. Reactions in the lumbar cord of the rabbit during periodic breathing. Unheated thermocouples, 0·5 cm. apart.

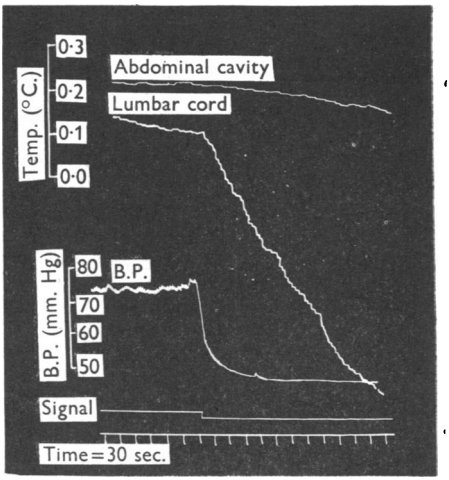

Fig. 3. The effect of pentobarbitone sodium overdose on the circulation in the spinal cord. Unheated thermocouples. Signal=60 mg. pentobarbitone sodium (intravenous).

The effect of D-*tubocurarine chloride on the spinal cord circulation*

Curarized animals were used in some of the investigations to be described. It is necessary, therefore, to consider the direct action of the drug on the spinal cord circulation.

In 6 animals where it was used, the blood pressure, the body temperature and the cord temperature fell simultaneously immediately after the administration. The drop in cord temperature was always greater and quicker than the drop in body temperature (Fig. 4). In another example, the body temperature continued to fall for over 5 min. after the beginning of artificial respiration, dropping from 38·6° to 38·4° C. The cord temperature fell by 0·40° C., the fall being complete 1 min. after the start of artificial respiration; it returned to base-line 5 min. later when the blood pressure had recovered. Similar results were obtained in all cases.

The effect of CO_2 *inhalation*

The effects of CO_2 inhalation were variable. Four experiments were performed. 10% CO_2 in O_2 was administered through the airway from a Douglas bag. In one animal inhalations of 1–2 min. caused diminutions in lumbar cord

temperature of 0·3° C. In a curarized animal, similar, but less pronounced, diminutions in cord temperature were recorded in response to CO_2. In the other two animals short inhalations of CO_2 had no effect, whereas inhalations of 2–3 min. produced slight increases in cord temperature.

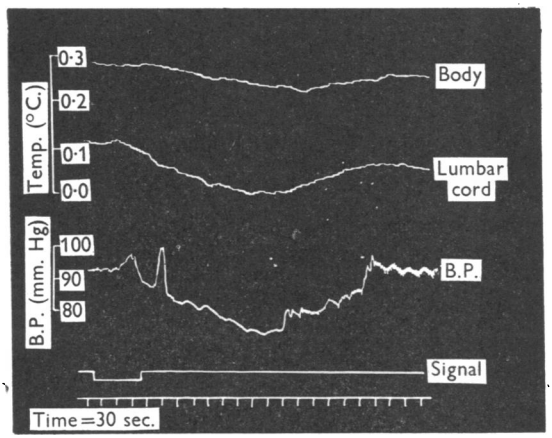

Fig. 4. The effect of curare on the circulation in the lumbar cord. Signal, intravenous D-tubocurarine chloride. Artificial respiration. Unheated thermocouples.

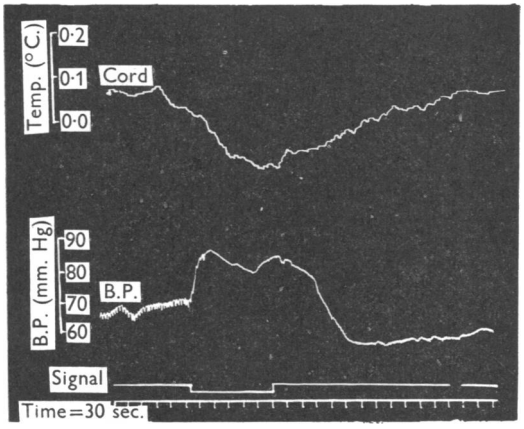

Fig. 5. The effect of intravenous adrenaline (2 μg./min.) on the lumbar spinal cord circulation. Unheated thermocouple.

The effect of intravenous adrenaline

Intravenous infusion of adrenaline was performed in three animals. The effect depended to some extent on the dose. Thus, in one rabbit 10 μg./min. given into an ear vein produced a definite increase in cord temperature. In the other two rabbits doses of 1–2 μg./min. produced marked falls in the cord

temperature, accompanying the rise in blood pressure. These latter results can only be interpreted as evidence of active vasoconstriction (Fig. 5). Unfortunately blood pressure records were not obtained from animals which showed evidence of increasing blood flow.

The effect of asphyxia

In the non-curarized animal

Blocking the airways of non-curarized rabbits for periods of 45 sec. produced varying effects in 10 experiments. The blood pressure responses were variable, but depressor and pressor effects usually occurred in succession (Fig. 6B). The cord temperature responses were more stereotyped. The usual reaction is shown in Fig. 6B where a preliminary slight rise in thoracic or lumbar cord temperature (unheated thermocouples) occurred at the beginning of asphyxia, followed by a fall, with a slow return to resting level.

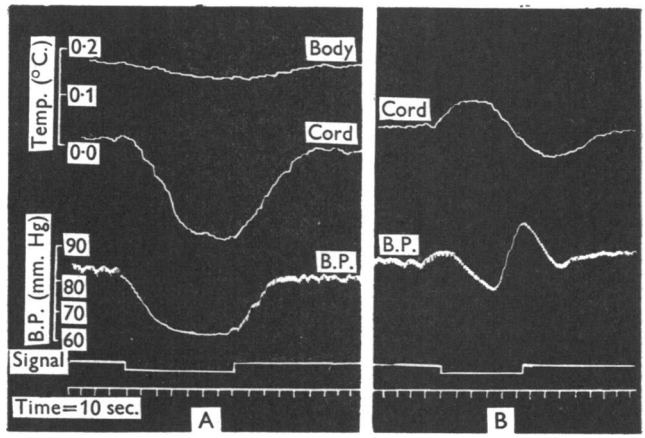

Fig. 6. The effect of asphyxia: A, in the curarized animal, pump stopped; B, in the non-curarized animal, airway blocked. Unheated thermocouples.

In three experiments the preliminary rise was absent. In two experiments where the blood pressure rose more than in the other cases, the preliminary rise was succeeded by a return to base-line as the blood pressure recovered, with no significant fall below.

In the curarized animal

Asphyxia was induced in curarized animals by stopping the respiration pump. A drop in blood pressure always occurred together with a drop in cord temperature. On restarting the pump, both the cord temperature and blood pressure usually returned rapidly to base-line (Fig. 6A).

In two experiments, however, after artificial respiration had been restarted,

there was a slight recovery which was followed by a further prolonged fall in cord temperature. The body temperature showed a similar drop during the same period, but the drop in cord temperature was always more rapid and much greater (Fig. 7).

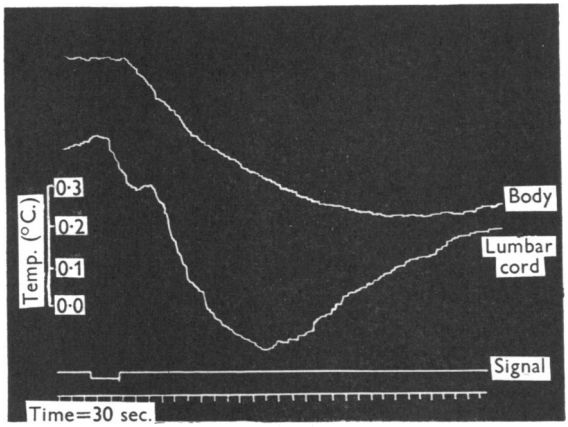

Fig. 7. Vasoconstriction in the spinal cord following asphyxia (signal) in the curarized animal. Unheated thermocouples.

The effect of peripheral nerve stimulation

A total of 50 rabbits was used in order to study the effects of peripheral stimulation on the circulation in the spinal cord. Records were made of temperature in the lumbar cord and thoracic cord. In some experiments heated thermocouples were used. The following methods were used to produce peripheral stimulation:

(*a*) Pinching the heel. Continuous or intermittent pinching sufficient to produce slight limb withdrawal in the non-curarized animal was the method most frequently used.

(*b*) Squeezing the limb. This was done manually or by using a tourniquet.

(*c*) Stroking the skin or fur.

(*d*) Electrical stimulation of the limb by needles inserted into the thigh muscles.

(*e*) Electrical stimulation of the intact sciatic nerve (shielded electrodes).

(*f*) Electrical stimulation of the central end of the cut sciatic nerve (shielded electrodes).

A Ritchie-B.N.I. square wave stimulator was used for all electrical stimulations.

In general one or both of two clear-cut effects was produced in the lumbar cord by these procedures, namely: (1) vasodilatation during stimulation (the usual effect), or (2) vasoconstriction following stimulation (an occasional finding).

Vasodilatation produced in the spinal cord by peripheral stimulation

Increased blood flow was manifested by an increase in the temperature of the spinal cord using the cooled or unheated thermocouple, and by a decrease in the temperature of the heated thermocouple.

Heel pinching or electrical stimulation of the central end of the cut sciatic nerve were found to give the most consistent results. Fig. 8A shows a typical result of heel pinching, viz. a rise in lumbar cord temperature of 0·2° C. during a stimulation of 45 sec. duration, followed by a return to base-line. In successful experiments similar effects followed all types of leg stimulation, which could be repeated with similar results over considerable periods. On a few occasions observations were made for periods of 10 hr.; in one experiment 30 separate stimulations were carried out, each causing an increase in cord temperature with subsequent return to base-line.

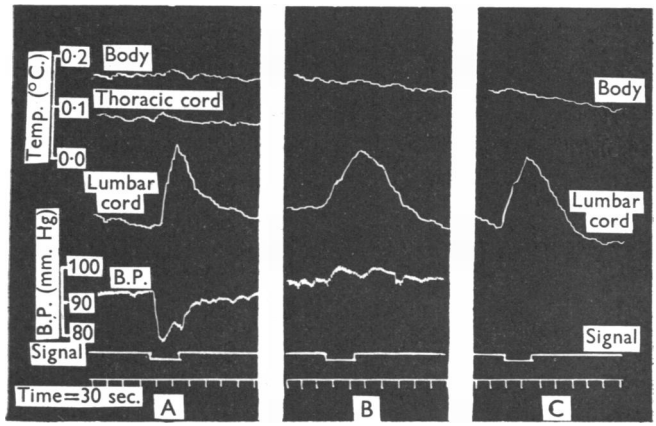

Fig. 8. The effect of heel pinching on the lumbar spinal cord temperature: A, non-curarized animal; B, curarized animal; C, curarized animal, after section of cervical cord. Unheated thermocouples.

In the experiment shown in Fig. 9 (thermocouple cooled), stimulating the central end of the cut sciatic nerve produced the usual rise in the lumbar cord temperature. After a number of stimulations had been carried out to confirm the consistency of the effect (Fig. 9A), the procedure was repeated with the thermocouple heated to 2·0° C. above the intra-peritoneal temperature. Small diminutions in cord temperature then accompanied each stimulation (Fig. 9B). Finally the thermocouple heater was switched off, suction turned on, and further stimulations performed, giving results similar to that shown in Fig. 9A.

Similar findings were obtained in all experiments of this kind. It was concluded, therefore, that the changes in temperature which took place in the lumbar cord following peripheral stimulation were the result of an increase in blood flow.

The blood pressure changes during stimulation were found to be variable. Both pressor and depressor effects were obtained even in the same animal without any apparent difference in prodecure, and there was no apparent correlation between the blood pressure changes and the spinal cord reactions. In those cases where the blood pressure fell during the period of rising cord temperature (e.g. Fig. 8A) it can only be concluded that the cord blood flow changes were the result of an active vasodilatation.

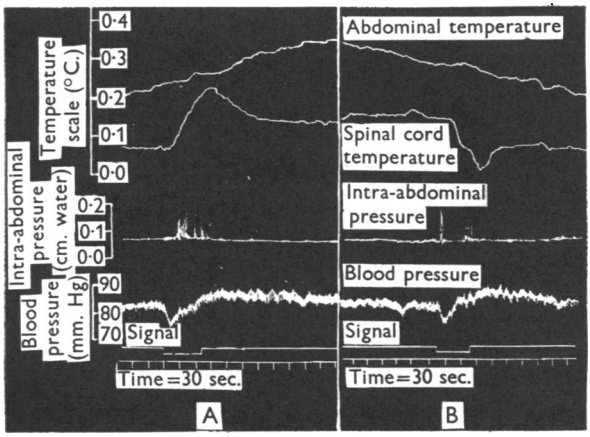

Fig. 9. The effect of stimulation of the central end of the cut sciatic nerve on the lumbar spinal cord circulation in the rabbit: A, cooled thermocouple; B, heated thermocouple.

The type of stimulus did not greatly affect the result obtained. In the non-curarized animal, heel pinching or electrical stimulation of the central end of the cut sciatic was most frequently used. Squeezing the limb and stroking the skin produced less certain responses. Electrical stimulation of the limb of an intensity sufficient to produce slight twitches was usually effective, and electrical stimulation of the intact sciatic was as effective as electrical stimulation of the central end of the cut sciatic.

Localization of vasodilatation in the spinal cord. The dilator effect described applies to the lumbar cord. In 18 experiments, records were taken simultaneously from the lumbar and thoracic cords. In every case responses to peripheral leg stimulation were only obtained in the lumbar cord (Fig. 9A). Accurate implantation of the thermocouples in the segments giving origin to the sciatic nerve was essential in order to record positive dilator effects. In three experiments where negative results were first obtained, reinsertion of the couple in a different segment enabled the usual dilator response to be obtained on sciatic stimulation. In three other cases where no positive results could be obtained even after reinsertion, post-mortem examination of the animal

showed a post-fixed lumbar plexus. In two experiments simultaneous recordings were made from adjacent segments. Vasodilatation occurred only in the segments contributing to the sciatic nerve.

In most experiments the thermocouple was implanted near the anterior horn, not far from the mid-line, and it was usually found possible to obtain positive dilator results by stimulating either limb. In some cases, however, the localization was such that positive dilator effects could only be obtained by stimulating the leg on the same side as that in which the thermocouple was implanted.

Stimulation of the forelimbs and other parts of the body usually had no effect on the lumbar cord.

The influence of body temperature on the sensitivity of the responses. In most experiments body temperature was not found to affect the results. In three experiments, however, evidence of vasodilatation following peripheral stimulation could not be obtained during phases of rising body temperature. When the body temperature was falling, however, dilator results were obtained. The explanation of this observation is not clear.

Vasodilatation in the spinal cord of the curarized animal. The peripheral stimulation experiments of all kinds were repeated in anaesthetized curarized rabbits. There was no movement of the animal during stimulation. In two cases the central end of the cut sciatic nerve was stimulated electrically. In all instances the characteristic dilator responses were obtained (Fig. 8B).

Vasodilatation in the spinal cord following cervical section. In one animal the spinal cord was completely transected in the cervical region. The usual vasodilator responses were obtained on stimulation before and after the transection (Fig. 8C).

Vasoconstriction in the spinal cord following peripheral nerve stimulation

In seven of the 50 animals in which peripheral stimulation was employed, this was occasionally followed by a period during which the cord temperature fell rapidly—even precipitously—thereafter returning slowly to base-line (Fig. 10). Usually these events were preceded by vasodilatation. The fall varied from 0·3° to 0·8° C. and was complete in 2–5 min.; the return to base-line occupied from 5 to 30 min. according to the extent of the fall.

Blood pressure changes during the phase of dropping cord temperature were usually slight, the most frequent being an increase in blood pressure. Body temperature remained constant. It is considered that these effects were the result of a diminution in cord blood flow, which, in view of the absence of a significant fall in blood pressure, must have been brought about by active vasoconstriction.

No evidence was obtained to suggest the conditions which predisposed to these vasoconstrictor effects. In most experiments their occurrence could not

be related to anaesthetic, body temperature or gross blood pressure change. They were obtained after every kind of stimulation, and in curarized and non-curarized animals. In one animal peripheral stimulation was always followed by vasoconstriction; in the other six, where similar vasoconstriction was observed, the effect could not be regularly produced.

DISCUSSION

Under the experimental conditions described, the temperature of the spinal cord was always somewhat lower than that observed in the posterior part of the abdominal cavity. It may be safely assumed therefore, that cord tempera-

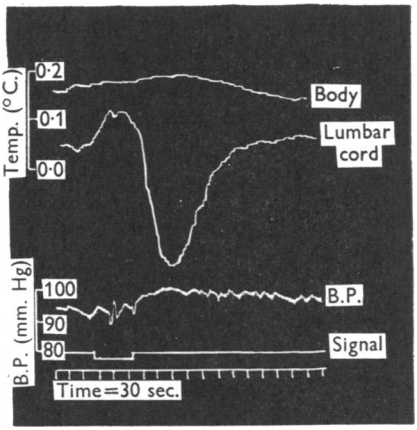

Fig. 10. Vasoconstriction following vasodilatation in the lumbar cord—sciatic nerve stimulation. Unheated thermocouples.

ture is lower than that of its afferent blood supply. Feitelberg & Lampl (1935) observed that in the *conscious* animal the temperature of the brain is above that of the carotid blood, and that exhibition of hypnotics and narcotics in general lowered cortical temperature *below* that of the afferent blood. In view of these findings it must be emphasized that all recordings in the present work were made under established anaesthesia.

In protracted experiments, when the general condition of the animal had deteriorated, it was often found that changes in blood pressure were passively reflected in the cord temperature (Fig. 2). Under these conditions, too, the animal was frequently insensitive to stimuli which, under more favourable conditions, had evoked alterations in cord temperature. The records shown in Fig. 1, however, and the results of peripheral nerve stimulation demonstrate that in animals in good condition, under the usual experimental conditions, the circulation in the spinal cord possesses a certain degree of autonomy *vis-à-vis* changes in blood pressure and can react independently (see Fig. 10).

The methods of measurement used were in no way quantitative for flow, and

it was impossible to estimate the resting blood flow in the spinal cord. The above considerations, however, and the fact that it is possible to produce dilator effects independently of blood pressure change suggest that there may be some degree of resting tone in the spinal cord vessels.

Schmidt & Pierson (1934) found that CO_2 was the most powerful of a number of substances tested for influence on medullary blood flow. They concluded that medullary blood vessels normally have a high intrinsic tonus which may be antagonized to varying degrees by the dilator action of CO_2. It must be remembered that the medulla may well represent a special case with regard to sensitivity to CO_2. However, Schmidt & Hendrix (1938, pp. 232–3) state: 'We believe that the vasodilator effect of CO_2 is the mechanism by which all of the various parts of the brain receive a blood supply proportionate to their needs'. The few observations made in the present work do not suggest that CO_2 has a consistent dilator action in the spinal cord and it seems unlikely, therefore, that the mechanisms suggested by Schmidt & Hendrix can be extended to this region.

Adrenaline, however, appears to have a definite vasoconstrictor action on the blood vessels of the spinal cord. In this respect the responses of the blood vessels in the spinal cord are similar to cutaneous and visceral vascular reactions (Grayson & Swan, 1950). It is tempting to assume, therefore, despite failure to demonstrate autonomic nerves in relation to the smaller cerebral vessels (Penfield, 1932), that the autonomic nervous system may nevertheless play a part in the maintenance of resting vasomotor tone in the central nervous system.

In the experiments described, pinching the heel sufficiently to elicit a sustained withdrawal reflex was almost always associated with an increased blood flow in the lumbar cord. The clearest records were obtained when the thermocouple recording point was thought to be resting in the anterior horn grey matter—as might be expected from its considerably richer blood supply.

The experimental evidence indicated that the vasodilatation accompanying muscular activity was localized to the active neural segments.

In the brain, localized vascular responses have been frequently observed to accompany nervous activity. Cobb & Talbot (1927) found that the olfactory bulb of the rabbit showed a considerably increased vascularity if the animal were allowed to inhale ammonia or ether for a few minutes before death. They concluded: 'If this be a true local increase of capillary bed in response to nerve stimulus, the physiological and psychological implications may be important.'

Fulton (1928) brought forward an interesting clinical case supporting the results of Cobb & Talbot. He observed a case of ganglioma arteriale racemosum of the occipital cortex in which the bruit was found to increase in intensity during visual effort. The increase came on in 20–30 sec. and was maintained

for nearly 1 min. after cessation of visual effort. No concomitant changes in blood pressure were found, nor was the bruit affected by activity of other sense organs, e.g. hearing, smell. Fulton concluded that 'localized increase in vascularity of the brain occurs under appropriate sensory stimulation'. Schmidt & Hendrix (1938, p. 267) show an elegant record from the occipital cortex of the cat supporting Fulton's suggestion. Gerard & Serota (1936) also observed localization of vascular changes to active neural structures, but the most detailed investigation was made by Santha & Cipriani (1938). Using a heated thermocouple of the Gibbs type introduced into different parts of the basal ganglia they were able to establish a strict relationship between localized cortical areas and definite parts of the thalamus and caudate nucleus. The increase in blood flow resulting from stimulation of a given cortical point was found to be sufficiently localized to enable the method to be used as a means of establishing subcortical-cortical relationships.

Although in the experiments described here the degree of localization was not so extreme, it appears that localization of vascular responses similar to that observed in the brain also occurs in the lumbar region of the spinal cord.

Santha & Cipriani thought that localized vasodilatation in the brain was mediated by the local liberation of metabolites such as CO_2 and acetylcholine rather than by nervous reflex, pointing out that Penfield (1932) had been unable to demonstrate a nerve supply to vessels less than 30 μ. in diameter entering the base of the brain.

In the present experiments where CO_2 was administered, there was no evidence to indicate a specific vasodilator role for CO_2 in the cord. Penfield emphasized the technical difficulties in the way of successful impregnation of cerebral vascular nerves and it is at least possible that improvements in technique may show their presence in association with smaller vessels in the depth of the nervous tissue. Unpolished histological preparations made by one of us (E.J.F.) have shown that there are fine nerve fibres accompanying the vessels entering the cord substance through the anteromedian furrow in man, though no fibres were demonstrable around the smaller vessels. The question of mechanism in this 'reflex' vasodilatation must therefore be left open for the moment.

SUMMARY

1. A technique is described for the detection of blood flow changes in the spinal cord of the rabbit.

2. In debilitated animals blood pressure changes may produce passive responses in the blood flow in the spinal cord.

3. In fresh preparations, however, the blood flow in the spinal cord is largely independent of general blood pressure changes.

4. The effects on spinal blood flow of anaesthesia, CO_2, adrenaline, asphyxia and curare are described.

5. Peripheral stimulation usually causes vasodilatation localized to the activated cord segment which still occurs after curarization or cervical cord section.

6. Occasionally, under conditions which are not understood, peripheral stimulation leads to a pronounced vasoconstriction in the spinal cord.

The authors are indebted to Professors R. J. Brocklehurst and J. M. Yoffey for their continued interest and encouragement. The expenses of this work were covered in part by a grant from the Colston Research Fund. The pentobarbitone sodium was supplied by Messrs Abbots Ltd., the curare by Burroughs, Wellcome and Co. Ltd., and the authors wish to express their thanks for this help.

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