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VASOCONSTRICTION IN THE HAND DURING ELECTRICAL STIMULATION OF THE LUMBAR SYMPATHETIC CHAIN IN MAN

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Vasodilatation in the hand has been shown to occur when the legs are heated by means of a radiant heat cradle (Kerslake & Cooper, 1950). This increase in hand blood flow is dependent on afferent nervous impulses arising in the heated area. The response is abolished by lumbar sympathectomy, and there is presumptive evidence that the afferent fibres concerned in nervous reflex vasodilatation are anatomically located, for part of their course, in the sympathetic chain (Cooper & Kerslake, 1953). It has also been suggested that the rate of discharge of impulses along these afferents is a function of the rate of flow of heat through the area of skin in which they arise (Kerslake & Cooper, 1954). It was thought worthwhile, therefore, to attempt to stimulate the lumbar sympathetic chain electrically, and to observe any concomitant change in the hand blood flow. If the supposition as to the origin of the afferent impulses were correct, electrical stimulation of the fibres would produce an effect analogous to cooling the skin, and reflex constriction would occur in the hand.

METHODS

Experiments were carried out on patients during the performance of lumbar sympathectomy for peripheral circulatory disorders. The patients were anaesthetized with pentothal (thiopentone sodium) and the anaesthesia was maintained with nitrous oxide. Scoline (succinyl choline chloride) was given intravenously to give adequate relaxation of the abdominal musculature for the surgical procedures.

Hand blood flows were measured with a venous occlusion plethysmograph using an electrical volume recorder (Cooper & Kerslake, 1951). The flows were measured on the side opposite to that on which the sympathectomy was performed, a site appointed solely as being most convenient to the surgeon.

An electronic stimulator was used enabling square wave shocks to be delivered to the stimulating electrodes. The pulse duration could be varied between 1·0 and 35 msec, and the frequency of stimulation between 0·5 and 30 impulses/sec. Various electrodes were devised, and the most suitable

were found to be two stainless-steel laminectomy hooks 30 cm long insulated with 'Araldite' resin (Aero Research Ltd.) and bared on the inner curvature of the hooks. The radius of curvature of the hooks was 2 mm. The polarity of the electrodes with respect to each other was controlled by a commutator on the stimulator.

Since the anaesthetic risk to the patient might be increased with the duration of the operation, and the risk of infection is greater the longer the abdomen is open, experiments were limited in time to about 20 min. This meant that a number of desirable investigations had to be left undone. The approach to the sympathetic chain was retroperitoneal via an anterior abdominal incision; and the state of the nerve during stimulation depended on the amount of trauma which it had suffered during its dissection and identification.

RESULTS

Nine experiments have been performed in which the lumbar sympathetic chain was stimulated and the blood flow in the opposite hand was measured. In six experiments no change occurred in the hand blood flow, and in three, hand vasoconstriction occurred during stimulation. The possible reasons for the failure of stimulation to produce a response in the six unsuccessful experiments will be examined in the discussion, and the changes in hand blood flow in the three successful experiments will be presented in this section.

Subject A.M. In Fig. 1 the left-hand arterial inflow curves during stimulation of the intact right lumbar sympathetic chain, between L_1 and L_2 ganglia, are shown. In the upper trace it can be seen that the slope of the arterial inflow trace is reduced during stimulation. The stimulus used was a 20 msec pulse at a frequency of 25/sec and of 10 V amplitude. The vasoconstriction was apparent less than 8 sec after the stimulus was applied. On switching off the current the blood flow returned to its resting level. The slope of the inflow curve after cessation of stimulation increased abruptly 11–12 sec after the stimulus was removed. The middle trace shows a repetition of this experiment in which there was again a vasoconstriction in the left hand when the right lumbar sympathetic chain was stimulated. In the lower trace the effect of stimulating a nearby piece of connective and fatty tissue is seen.

Subject A.G. The right lumbar sympathetic chain was stimulated, between L_2 and L_3 ganglia, with 20 msec pulses at a frequency of 25/sec. The left-hand blood flows were measured. Fig. 2 shows the trace obtained of the arterial inflows in the left hand. There is a diminution of blood flow in the hand during stimulation. The onset of this vasoconstriction is less than 6 sec from the time of application of the stimulus. Fig. 3 shows a similar experiment in which the stimulus was removed at the beginning of an inflow tracing. From this, it can be seen that the return of the blood flow in the hand to its normal level commences in less than 10 sec after cessation of stimulation (i.e. between the end of stimulation and the point Y in Fig. 3), but there is no evidence of this occurring during the first inflow curve after stimulation ceases. It will also be noted that the hand volume has decreased during stimulation.

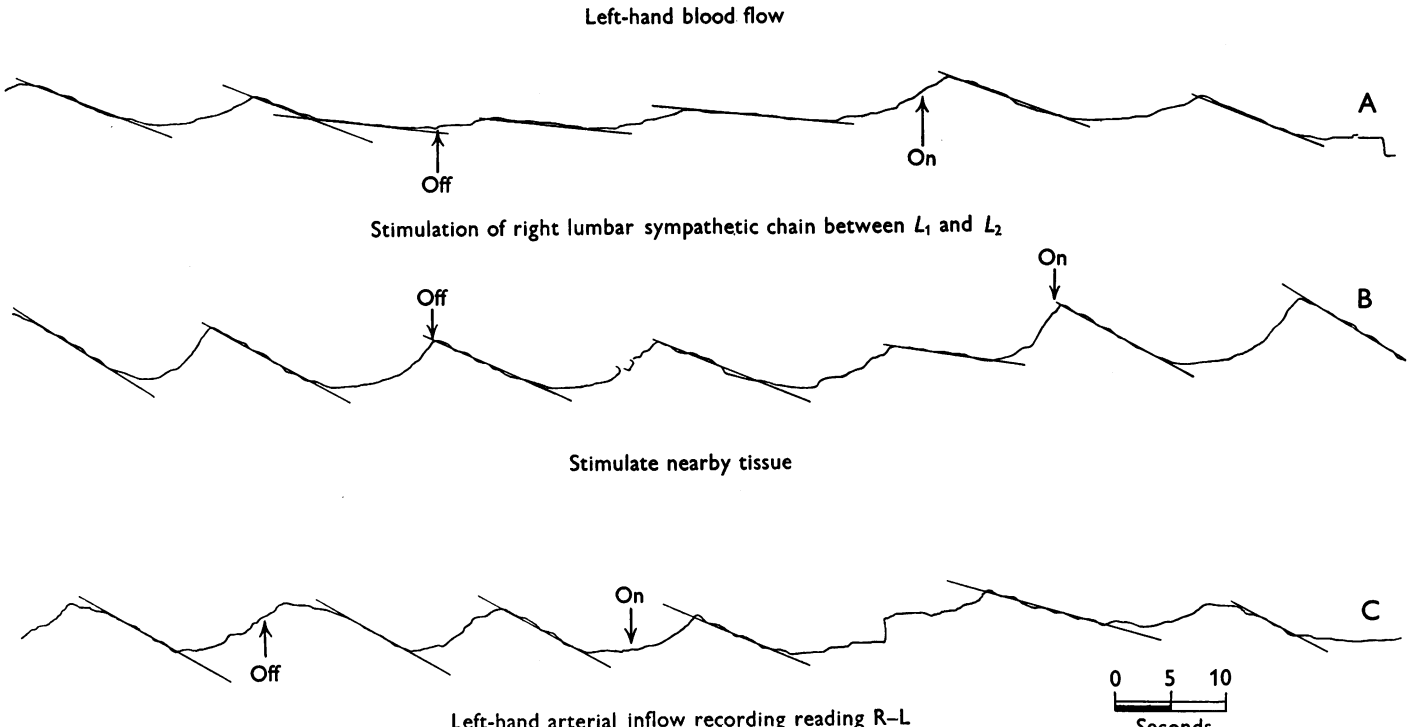

Fig. 1. The upper and middle traces illustrate the blood-flow curves in the left hand when the right lumbar sympathetic chain was stimulated. The lower trace was taken when the stimulus was applied to an adjacent piece of fatty and connective tissue. The traces read from right to left.

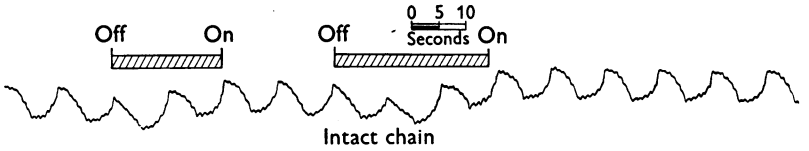

Fig. 2. Blood flows in the left hand when the intact right lumbar sympathetic chain was stimulated between L_2 and L_3 ganglia. The traces read from right to left.

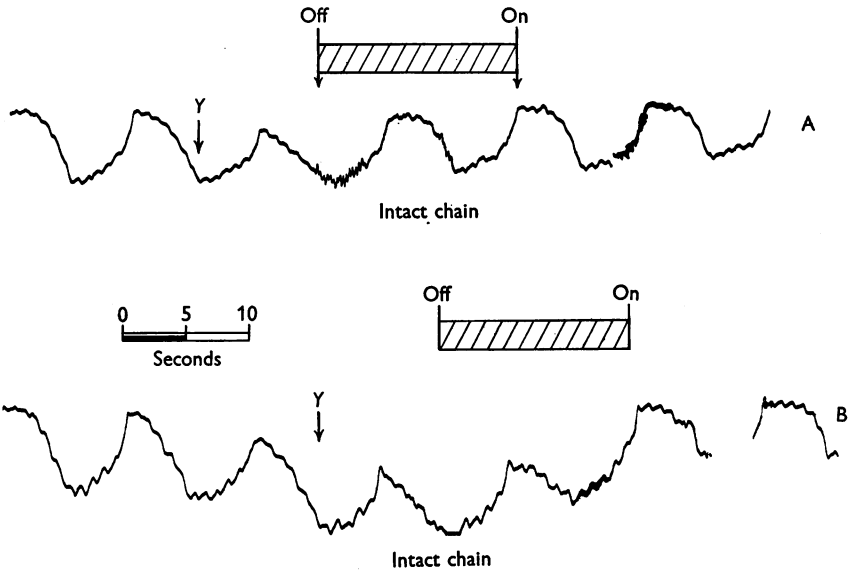

Fig. 3. Hand blood-flow records during stimulation of the lumbar sympathetic chain. The arrow Y is placed 10 sec after the end of stimulation. The traces read from right to left.

Subject E.B. During the operation on this patient, it was possible to stimulate the intact right lumbar sympathetic chain between L_2 and L_3 ganglia, and then, having divided the chain between these ganglia, to stimulate the cut central end and the cut peripheral end. The left-hand blood flow was measured. The intact sympathetic chain was stimulated first with the electrodes about 2 mm apart and no change occurred in the left-hand blood flow. A wide range of shock durations and frequencies were tried but no evidence of vasoconstriction or vasodilatation in the hand was found. The electrodes were then separated further so that there was more than 1 cm between them. Fig. 4 A, B show the changes in the hand blood flow which occurred when 28 msec pulses were applied at a frequency of 25 and 20/sec to the sympathetic chain. There was a significant vasoconstriction in the left hand. The sympathetic chain was then cut between the 2nd and 3rd lumbar ganglia. The left-hand arterial inflows were recorded at this time and are shown in Fig. 5 A. The mechanical stimulus of nerve section produced a diminution in hand blood flow. (A similar though

not so dramatic response is shown in Fig. 5B, when later the chain was again cut, between the 1st and 2nd lumbar ganglia, after the stimulation experiments described below were completed.) The cut central end of the sympathetic chain was stimulated with the electrodes more than 1 cm apart, below the 2nd lumbar ganglion. Hand arterial inflow traces during this stimulation are shown in

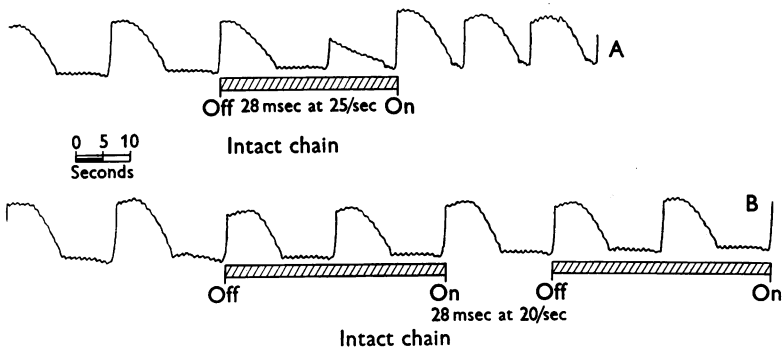

Fig. 4. Hand blood-flow records during stimulation of the intact lumbar sympathetic chain: (A) with 28 msec pulses at 25/sec, and (B) with 28 msec pulses at 20/sec. The traces read from right to left.

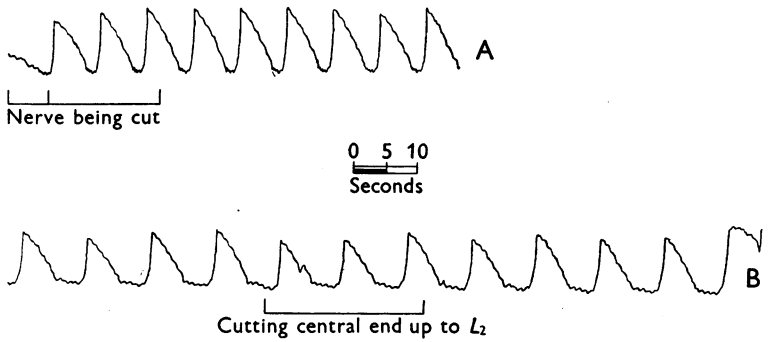

Fig. 5. Subject E.B. Hand blood-flow records taken while the sympathetic trunk was cut: (A) between L_2 and L_3, and (B) between L_1 and L_2. The traces read from right to left.

Fig. 6A, B. In both cases hand vasoconstriction occurred during stimulation, being more intense in the experiment shown in Fig. 6A than in Fig. 6B. Finally, in Fig. 7A, B the hand arterial inflows during stimulation of the cut peripheral and above the 3rd lumbar ganglion of the chain can be seen. Comparing Fig. 7 with Fig. 6, it will be seen that there was no response in the left-hand blood flow. Between the points indicated in Fig. 7A a twitch occurred owing to spread of current to the muscles of the abdominal wall during a stimulation, but no change in hand blood flow was caused.

General observations. Analysis of the tracings reveals no change in heart rate during the period of stimulation of the lumbar sympathetic chain. No measure-

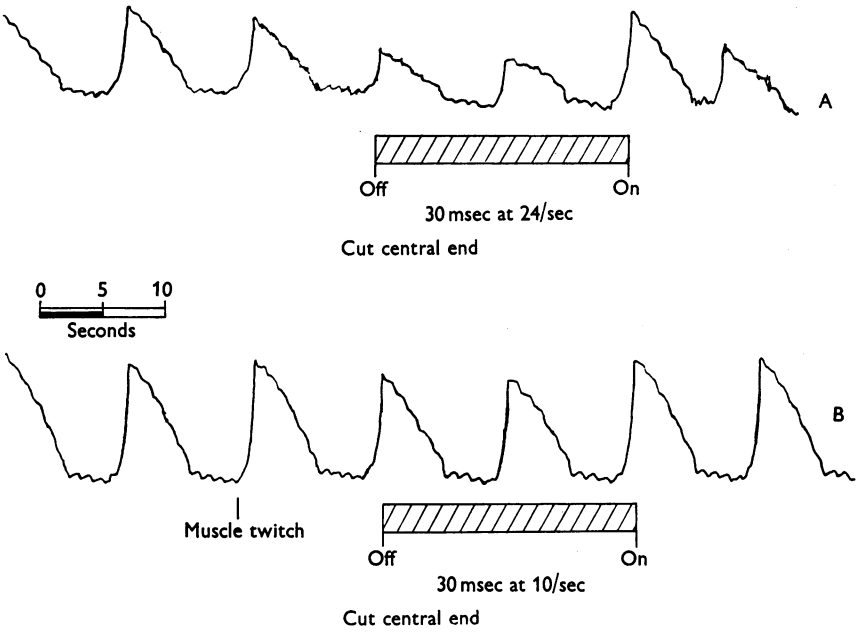

Fig. 6. Hand blood-flow records during stimulation of the cut central end of the lumbar sympathetic chain, with 30 msec pulses: (A) at 24/sec and (B) at 10/sec. The traces read from right to left.

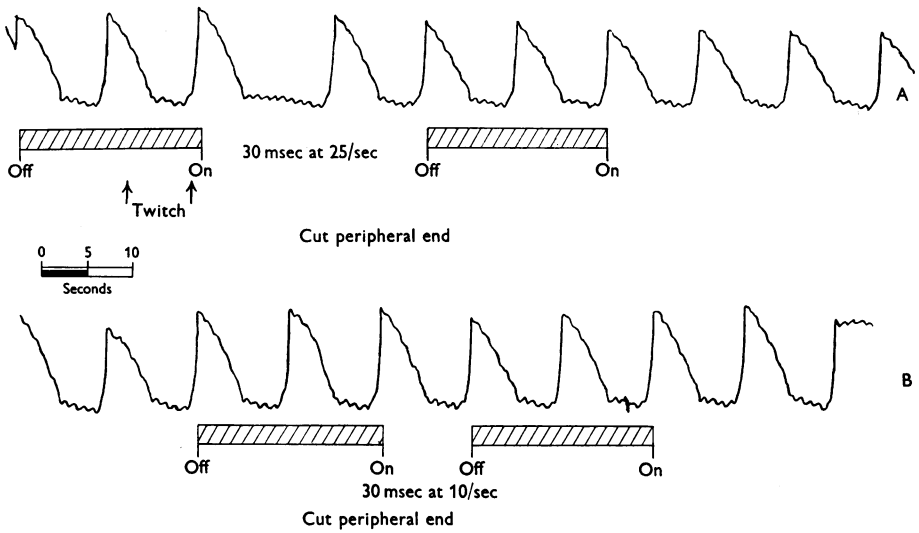

Fig. 7. A, B, hand blood flows taken during stimulation of the peripheral cut end of the lumbar sympathetic chain. The traces read from right to left.

ment has been made of blood pressure partly owing to the short duration of stimulation, and partly owing to shortage of man-power. No measurement was made of respiration, but the anaesthetist who was watching for marked respiratory responses could report no visible gasp or variation in respiration during stimulation. The results of stimulation of subject E. B. are displayed in graph form in Fig. 8. No response other than vasoconstriction has been elicited so far by any of the range of pulse durations, frequencies and amplitudes used during stimulation.

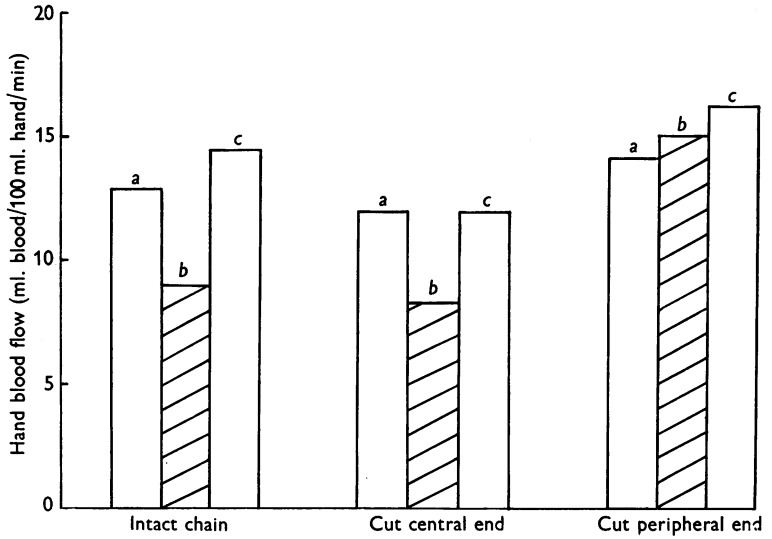

Fig. 8. The mean hand blood flows of subject E. B.: (*a*) during the period prior to stimulation, (*b*) during stimulation, and (*c*) immediately after stimulation of the lumbar sympathetic chain.

DISCUSSION

Four factors are thought to have played a part in the failure to elicit a response in six experiments. First, it was not realized until late in the series that the inter-electrode distance was critical. In the experiments where failure occurred the inter-electrode distance was of the order of 3 mm, whereas the optimum spacing appears to be greater than 1 cm. Secondly, the depth of anaesthesia at the time of stimulation varied. Thirdly, with some designs of electrodes, there was considerable pulling on the nerve in order to lift it from the body wall on to the electrodes. Such trauma may well have been adequate to damage or destroy the nerve fibres which it was hoped to stimulate. Fourthly, no precautions could be taken to prevent drying of the nerve after its exposure, and the degree of this dehydration may have varied from experiment to experiment. It is felt, therefore, that no significance can be attached to the absence of a response under the experimental conditions used.

From the evidence adduced, it is most probable that the nerve fibres stimulated were running in the sympathetic chain and were not ordinary somatic nerve fibres excited by spread of current to the body wall. Stimulation of nearby tissue on the body wall (Fig. 1) did not elicit the hand blood-flow response which was obtained by the same stimulus to the sympathetic chain. Again, on stimulating the peripheral cut end of the chain, there was no hand vasoconstriction, even when a twitch occurred in the muscles of the posterior body wall during stimulation (Fig. 7 A).

The onset of vasoconstriction in the hand when the lumbar sympathetic chain is stimulated occurs with a latency of less than 6 sec. This is consistent with the latency between switching off a radiant heat cradle over the trunk or legs and the fall in hand blood flow which then occurs (Kerslake & Cooper, 1950). Similarly, the time which elapses between switching on the heat cradle and the appearance of a hand vasodilatation (< 12 sec, > 6 sec) is of the same order as the latent period between the cessation of stimulation and the increase in hand blood flow which then occurs. Some evidence suggests (Kerslake & Cooper, 1954) that the skin receptors responsible for nervous reflex vasodilatation discharge at a frequency which is correlated positively with the rate of heat flow (normally outward) through the skin. If the nerve fibres arising from these endings were those stimulated in the work described here, then the response to be expected in the hand would be vasoconstriction. Folkow (1952) has shown that the latent period of relaxation of the small blood vessels is a function of the rate at which their motor nerves have been stimulated, and may lie between 6 and 18 sec. He considers that the latent period between the cessation of stimulation and the vascular relaxation is probably mainly due to delay in the destruction of the transmitter substance at the vasomotor nerve endings. If this is the case, then the delay in vasodilatation after lowering the frequency of the afferent sympathetic discharge will be similar whatever the mechanisms whereby that discharge frequency is lowered.

Previous evidence (Cooper & Kerslake, 1953) suggests that part of the afferent arc of reflex vasodilatation due to warming the legs with radiant heat is anatomically located in the lumbar sympathetic chain. The fibres subserving the afferent side of this reflex may therefore be those excited during stimulation of the sympathetic chain. It is also known that some visceral afferent fibres travel in the sympathetic chain (Bain, Irving & McSwiney, 1935; McSwiney, 1945), but the anatomical levels at which such fibres enter the sympathetic chain in man are not clearly defined. That distension of some viscera, especially the bladder, can lead to vasoconstriction in the hand was demonstrated by Guttmann & Whitteridge (1947). It is possible that afferents from such a viscus were involved in the responses described in this paper. The absence of any noticeable change in the pattern of respiration makes it unlikely that the vasoconstriction was secondarily due to a gasp response from

pain fibre stimulation. This view is also reinforced by the absence of any heart rate change during stimulation of the sympathetic chain.

It is concluded that fibres exist in the lumbar sympathetic chain, stimulation of which causes vasoconstriction in the opposite hand. These fibres may arise in the viscera, or may be the afferent fibres from the legs concerned in the reflex vasodilator response to heating the skin.

SUMMARY

1. Electrical stimulation of the intact and the cut central end of the lumbar sympathetic chain between L_1 and L_3 ganglia in man, under nitrous oxide anaesthesia, caused vasoconstriction in the opposite hand in three of nine experiments.

2. Similar stimulation of the cut peripheral end of the lumbar sympathetic chain and of nearby connective tissue did not cause hand vasoconstriction.

3. It is concluded that nerve fibres in the sympathetic chain were stimulated, and that afferent impulses set up in these fibres were responsible for the hand vasoconstriction. The effectiveness of stimulation appeared to depend in part on the inter-electrode distance.

4. The latency of the vasoconstriction after the onset of stimulation was less than 6 sec. The latent period between cessation of stimulation and the release of vasoconstriction in the hand was between 5 and 12 sec. The significance of this latency is discussed.

5. The view that these fibres are associated with afferent impulses from the legs which mediate hand vasomotor responses to heating the legs is discussed. Other possible origins of these fibres are also discussed.

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