cheesy material in carcinoma of the body, and at the same time the curettage cures the condition. Senile endometritis is a definite inflammation, and the microscope reveals much small-celled infiltration.

Mucous Polypi.—An atrophic tissue like the endometrium after the menopause would not be expected to produce polypi, but it does so not at all infrequently, especially in the cervix, and I have seen at least half a dozen cases in the last six months. The polypi can usually be detected protruding from the external os, and curettage usually reveals no others in the body of the uterus. Curettage cures the condition.

CONCLUSION.

These are the commonest causes of haemorrhage about and after the menopause, though a number of others, like carcinoma of the vagina, do occasionally occur, but so rarely that we need not keep them constantly in our minds.

The one point we must constantly keep before us is the frequency of malignant disease as a cause of haemorrhage, and the fact that haemorrhage is usually the only early symptom of this disease, and so we must always consider any of these cases as likely to be malignant until we have proved the haemorrhage to be due to some other cause. If we will only do this these malignant cases will be detected much earlier and a far larger number cured of this dread disease.

COMPRESSION OF NERVE FIBRES.

BY

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COMPRESSION OF PERIPHERAL NERVES.

DURING a study of the lesions of peripheral nerves caused by missiles of war observations were made which indicated the possibility in certain circumstances of being able to diagnose compression of a peripheral nerve. The earlier investigations¹ led me to the conclusion that in a case of progressive compression of a nerve, uncomplicated by division of any fibres or serious intraneural changes, three stages could often be recognized.

1. The earliest sign generally found was increasing weakness of the muscles innervated by the affected nerve, demonstrating a loss of conduction in the efferent fibres, which was commonly associated with pain of a neuralgic character radiating along the course of the nerve and induced by movement or stretching of the nerve trunk.

2. The appearance of objective sensory disturbances which exhibited a characteristic type of dissociation. In uncomplicated examples of compression, hypalgesia and thermo-hypaesthesia progressing to analgesia and thermo-anaesthesia have been found to occur almost constantly before any definite tactile anaesthesia, even to the lightest possible tactile stimulus, could be recognized. During the appearance of analgesia the neuralgia frequently diminishes and not uncommonly completely disappears with the development of analgesia in the full area of distribution of the nerve. By this time complete paralysis of all the muscles supplied by the nerve distal to the site of compression is to be expected.

3. The development of tactile anaesthesia, which remains less in extent than the analgesia and thermo-anaesthesia, unless the compression proceeds to complete physiological division and causes an absolute obstruction to the transmission of all impulses.

A knowledge of these three stages in the development of complete strangulation of a nerve, in conjunction with the study of a carefully elicited history, makes it possible to diagnose correctly a large proportion of examples of uncomplicated compression of some part of the peripheral nervous system. In my experience, such complications as partial division or the occurrence of intraneural fibrosis very commonly render diagnosis much more difficult and uncertain. The characteristic manifestations of compression are well illustrated by patients suffering from compression of the lower trunk of the brachial plexus by a cervical or first dorsal rib, and in a study of its production by the latter cause² it was found that in nine out of ten patients the objective sensory disturbance was

distinctive; further observations since the publication of that paper have confirmed this. The dissociated sensory loss is undoubtedly the most striking and the most helpful sign, and possesses the clinical advantages of being rapidly and easily demonstrated and not readily confused with any other neurological condition excepting possibly syringomyelia, an error which ought to be avoided without difficulty. It is important to mention that quite frequently patients are unaware of any sensory loss, and are surprised at the analgesia and thermo-anaesthesia which clinical examination reveals.

COMPRESSION OF THE SPINAL CORD.

During recent years my attention has been directed to the frequent appearance of a similar dissociation of sensation as a result of compression of the spinal cord by such lesions as extramedullary tumours, and this observation has proved on several occasions of considerable diagnostic value. The first occasion on which I noticed this was about eight years ago when investigating a patient suffering from fracture dislocation of the cervical spine and subsequent compression of the spinal cord. The man merely suffered from weakness of the muscles, altered reflexes, and hypalgesia and thermo-hypaesthesia below the level of the lesion, and after relief of the compression all these manifestations almost completely disappeared. Recently I was permitted to examine at the Manchester Royal Infirmary a patient who illustrated particularly well the manifestations of uncomplicated compression of the spinal cord, and exhibited undoubted resemblances to the observations made upon patients suffering from compression of a peripheral nerve. I am obliged to my colleague, Dr. E. Bosdin Leech, under whose care the patient was, not only for the opportunity of making several examinations, but also for permission to publish a brief report of the most significant features.

A man, aged 44, previously very robust and healthy, began to suffer from pain in the region of the shoulders about December, 1924. The pain often travelled down the trunk, especially on the right side, and after a time became very severe, attacks of exceedingly sharp pain occurring frequently and persisting for many hours at a time. He then suffered from stiffness of both legs and attacks of "pins and needles," extending from the level of the umbilicus down the front and back of both lower limbs. The stiffness was succeeded by progressive weakness of both legs until, at the time of his admission to hospital in November, 1925, he was unable to walk without assistance. Shortly before admission to hospital he also complained of difficulty in beginning the act of micturition, but he was able to pass urine normally once he had started the stream.

On examination there was no absolute paralysis of any of the muscle groups of the lower limbs, but very profound paresis and marked spasticity was obvious. The deep reflexes of both legs were greatly exaggerated, and there was an extensor plantar response and ankle clonus on both sides. On testing sensation it was found that there existed, as high as the level of the nipples, complete analgesia and almost complete thermal anaesthesia, although the patient was not aware of any objective sensory disturbance. No defect of any other form of sensation could be discovered, although carefully tested.

A provisional diagnosis of extramedullary spinal tumour at the level of the fifth dorsal segment was made and an injection of lipiodol made by Mr. H. H. Rayner, F.R.C.S., for confirmatory purposes. An x-ray examination after the injection demonstrated the presence of an absolute obstruction at the vertebral level suggested by the clinical investigation.

At the operation, performed by Mr. Rayner, it was found that the spinal cord was compressed by a tumour lying between the dura and bones. The tumour was exerting direct pressure upon the cord both posteriorly and laterally, but had not at any point penetrated the membranes. Microscopic examination of a piece of the tumour, made by Dr. G. E. Loveday, showed it to be an angioma.

A similar dissociated sensory disturbance has been seen in so many cases of spinal compression that I have come to regard it as a valuable aid to diagnosis, and it is primarily for this purpose that the report of this patient has been recorded, since it demonstrates unusually clearly the clinical manifestations of the progress of the compression, and bears rather a startling likeness to what has been found in similar circumstances in the peripheral nervous system. In a recent paper on extradural spinal tumours Stookey³ regards it as possible that the tracts in the white matter are more vulnerable to pressure gradually applied to the cord than the grey matter, and it is interesting to notice that in two of five cases of cervical extradural tumours he mentions that "early evidence of

disturbance in the pyramidal and spino-thalamic tracts was found." It is necessary, however, to point out that later in the paper it is stated that the tumour in one of these cases, a small pointed chondroma, was placed in such a position that it could compress a small circumscribed area of cord in the region of the left spino-thalamic tract, and that in this case the sensory disturbances were confined to the right side.

From personal observation it appears that uncomplicated compression of the spinal cord is commonly manifested by the following symptoms and signs, which as a rule present themselves in the order given:

1. Pain of a severe character, commonly described as of a "burning" or "tearing" nature, in the lower limbs and trunk below the level of the lesion. This may or may not be accompanied by pain in the distribution of the spinal nerves arising at the site of compression and produced by pressure on, or irritation of, nerve roots. In a few cases pain is absent, and the initial symptom is weakness of the legs.

2. Weakness and stiffness of the lower limbs and alteration of reflexes.

3. Objective sensory disturbances below the point of compression, at first affecting conduction in the spino-thalamic tract, but later affecting elements of sensation dependent upon other sensory tracts.

4. Bladder symptoms, accompanied in the limbs by more profound loss of voluntary power and movement and more profound and complete sensory loss.

It is not easy to offer any explanation for the occurrence of this dissociation of sensation, found often in compression of a peripheral nerve or of the spinal cord, as it is difficult to perceive any reason why the fibres conducting impulses concerned with pain and temperature should be more vulnerable than the others. In the case of compression of a peripheral nerve the effect must be upon the peripheral sensory neurones which have their cell station in the posterior root ganglion, whilst in compression of the spinal cord the mechanical effect of the lesion must be exerted upon the secondary sensory neurones, which for the pain and thermal paths have their cell station in the grey matter of the cord. It has been possible to follow the results of release from compression in a large proportion of the examples seen in the peripheral nervous system, and from the rapidity with which recovery of sensation appears in the earlier cases it seems improbable that actual degeneration had occurred during the early stages of the manifestation of the dissociated sensory loss. If the compression persists for some time before it is relieved it seems likely that degeneration subsequently occurs, but from the extremely rapid recovery in the earlier cases (within twenty-four hours of release in a few instances) it does not seem possible to conclude that degeneration of the fibres concerned with pain and thermal sensations is a necessity for the production of the characteristic sensory dissociation. It has not been possible to keep under observation a sufficient number of spinal cases after relief of the compression to express a definite opinion about the possibility of recovery from the objective sensory loss. In early cases where there has been only hypalgesia and thermal hypaesthesia, recovery, as far as one can determine by the customary clinical tests, has occurred. Nevertheless, it is very doubtful if recovery of sensation ever succeeds the establishment of complete analgesia and thermal anaesthesia, and this suggests the early development of degeneration, since there is no possibility of the fibres in the spinal cord undergoing regeneration.

When the analgesia was first observed in compression of peripheral nerves I was attracted for an explanation to the work of Ranson,⁴ which suggested that the afferent fibres concerned with protopathic sensibility are non-medullated, but subsequent researches have failed to support Ranson's view, and there appears to be no reason for believing that the protopathic fibres differ in structure from other sensory fibres.

SUMMARY.

Attention is directed to the frequent occurrence in spinal compression of a dissociated sensory loss, similar to that previously described as a result of compression of peripheral nerves.

Since the first indications of this characteristic sensory disturbance may be discovered at a fairly early stage in

the development of the compression and before the onset of profound paralytic symptoms, they may be regarded as a valuable aid to diagnosis. This often renders it possible to undertake the relief of compression before it has caused irreparable damage to the spinal cord.

In the present state of knowledge it is not possible to advance any explanation of the occurrence of this dissociation, but there is evidence which suggests that hypalgesia and thermo-hypaesthesia may appear before actual degeneration of fibres has occurred in either the peripheral nervous system or the spinal cord.

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THE EFFICACY AND STANDARDIZATION OF HAEMOSTATICS.

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DURING the last two decades the use of organic extracts and of haemostatic serums in the arrest of obstinate haemorrhage has gained some prominence in medical practice, but no unanimity exists as to the value of the preparations in use. On the one hand, the claims of the makers of haemostatics are supported by a considerable amount of clinical evidence; on the other hand, the results of laboratory experiments by different observers are contradictory, the same preparations being described by some as active and by others as inactive (cf. ¹⁻⁴); whilst one well known haemostatic has been included amongst the nostrums described in *The Propaganda for Reform in Pharmacy* (1922).

The work here summarized was undertaken to ascertain the causes of these discrepancies and to devise, if possible, simple and reliable methods of standardization of those haemostatics which decrease haemorrhage by increasing the coagulability of blood.

Previous Experiments.

An examination of the experimental methods used in testing the efficacy of haemostatics reveals the causes of the varying results obtained by different observers, and shows that the conditions imposed differ radically from those existing in blood escaping from wounds. This is apparent from the following summaries of the methods employed.

1. The blood is kept fluid by the addition of either soluble oxalates or citrates which remove or immobilize the calcium ions of the plasma. The haemostatic is then added and coagulant action is noted when present. Blood under these conditions only clots on the addition of thrombin, and this technique merely indicates either the presence or absence of that coagulant. As calcium ions are essential for spontaneous clotting conditions are introduced which are never present either in normal or pathological bloods.

2. A variant of this technique is commonly used by the manufacturers of haemostatics in the standardization of their products. The speed of the clotting of blood after oxalation and recalcification at room temperatures is compared with that of blood similarly treated to which the haemostatic to be tested has been added. The efficacy of the haemostatic is estimated by any increase in the speed of clotting following its addition. Several errors are inherent in this procedure. The acts of oxalation and recalcification modify the stability of the plasma, so that its coagulability differs markedly from that of normal