been taking the drug for a sufficient length of time to allow me to eliminate the effect of change of weather, etc. It will be better for me to defer any account of these till I have more definite information.

I have brought these results before the Association at this time in the hope that some others who have more ample opportunities than myself may be perhaps induced to make trial of helenine in early phthisis. I do not think I am justified in saying that any one of the constituents of ebecampare root possesses greater value than the mixed product; probably this would meet all the requirements of clinical experiment.

The above research was carried out under the direction and at the cost of the Therapeutic Committee of this Association. I owe my very best thanks to Dr. Lauder Brunton, F.R.S., who has throughout given me very valuable suggestions and help, and in whose laboratory at St. Bartholomew's Hospital the experiments were mostly carried out.

THE PATHOLOGY OF TUBERCULOUS MENINGITIS WITH REFERENCE TO ITS TREATMENT BY TAPPING THE SUBARACHNOID SPACE OF THE SPINAL CORD.

Read in the Section of Medicine at the Annual Meeting of the British Medical Association, held in Bournemouth, July, 1891.

By CHARLES A. MORTON, F.R.C.S.,

Registrar Bristol General Hospital; Pathologist Bristol Children's Hospital.

In view of the recent procedure of tapping the subarachnoid space of the spinal cord in cases of tuberculous meningitis, the pathology of the disease derives quite a new importance. The prospect of relief by surgical interference in these cases depends on the relation which increased intraventricular pressure bears to other pathological changes and to the symptoms, and whether, from the condition found after death, there seems a fair chance of our being able to relieve this pressure by tapping the subarachnoid space of the cord.

In trying to answer these questions I have used my notes of fourteen cases of meningitis observed during the last few years, and I have prepared a table showing the symptoms and corresponding pathological changes present in ten of the cases. In one case I did not make the *post-mortem* examination, but as I observed the case during life and carefully examined the brain after death, I have included it among the ten. Nearly all the cases are tuberculous, only three are non-tuberculous; these I have added for comparison. For most valuable information which will aid us in this inquiry we are indebted to Dr. Gee's thorough observations on the pathology of the disease, made many years ago.

First, then, we must consider the pathological effects of the increased intraventricular effusion, and we must satisfactorily answer the question as to the patency or otherwise of the communication between the fourth ventricle and the subarachnoid space. Pathologically we find that together with the excess of fluid in the ventricles there is marked softening of the white matter in and around them. Is this softening due to the pressure of the excess of fluid or not? In considering this question we may, I think, exclude the theory that it is merely due to soaking of the fluid into the brain tissue after death. The fact that we find the whole of the corpus callosum so often softened when its anterior part must lie above the ventricles, even in the horizontal position of the body, seems to me to disprove this theory.¹ And, further, the observations of Dr. Bastian have shown that this softened tissue has a diminished specific gravity, and contains pathological cells. Putting on one side this theory then, we have to consider whether the softening is due to the pressure of excessive intraventricular fluid, or to inflammatory changes in the brain tissue.

That the tuberculous disease penetrates much farther into

¹ In one case of tuberculous meningitis in which I made the *post-mortem* examination, the only part softened was the corpus callosum; the parts at the base and posterior portion of the brain were not at all softened.

the brain than might be supposed we know from an observation made by Dr. Gee. He mentions a case where the capillary vessels throughout one hemisphere were everywhere studded by miliary tubercles. They remained after the softened cerebral tissue had been washed away. If, then, tubercles and granulation cells, as described by Dr. Bastian, are found in this softened tissue, and we remember the intimate relation existing between the vessels of the pia mater and the brain, I think we shall be right in considering that in this softening we have some inflammatory action and not a mere pressure effect. Dr. Gee thinks inflammation of the lining membrane of the ventricle is enough to cause serous effusion into the brain tissue, the softening being rendered complete, as he expresses it, "by the malnutrition which ensues in consequence of the more or less arrested capillary circulation."

But in all the cases of tuberculous meningitis which I have examined there has not been the slightest increase in vascularity in the softened tissue, which seems to me to show that inflammatory changes are not sufficiently marked in themselves to cause the softening, but that the pressure of the excessive fluid has much to do with it. In the chronic condition of excess of fluid—chronic hydrocephalus—it is true we have expansion and thinning rather than softening; but the gradual effect of the pressure in these cases may, I think, explain the difference, just as we have impaired mental power rather than coma.

Then there is Dr. Bastian's theory of thrombosis in the veins of Galen as a cause of this softening. I have often carefully examined for this thrombosis and have never found it. Dr. Gee says: "The blood may be made to flow from the veins of Galen backwards into the straight veins with perfect ease, and the plexuses are often quite pale and nearly empty."

If, then, the excess of fluid causes softening around, one would expect tapping the subarachnoid space to relieve the symptoms, supposing the ventricles still communicate with it. In making *post-mortem* examinations in cases of tuberculous meningitis, I have again and again tried to find out the condition of the openings in the pia mater closing in the fourth ventricle, but invariably have I found it so torn in removal of the brain that my observations were of no value. The foramen of Magendie is not the only opening that must be blocked in order to shut off the subarachnoid space from the ventricles. There are two others, one on each side, behind the upper roots of the glossopharyngeal nerve in the pouch-like extension of the membrane beneath the flocculus.

In only three out of ten cases is there a note that the membrane closing in the ventricle was thickened, but I should say not so altered as to block all the openings.

Dr. Gee says: "I have several times examined the cerebrospinal opening *in situ*, and have always found the membrane about it perfectly natural."

Again, on this point, which is a pathological one, we have distinct clinical evidence. In one of the four cases in which the subarachnoid space was tapped, reported by Dr. Wynter, the immediate change in the condition of the pupils was so marked as to lead me to think the pressure in the ventricles must have been relieved by tapping the subarachnoid space of the cord.

There are, however, some cases which seem especially to favour the view that the excess of fluid is a mere obstructive accumulation. I refer to cases where we find the ventricles very distended with fluid, and very little evidence of meningitis, cases which gave rise to the old name of acute hydrocephalus. The fluid is quite clear, and its excess is the one thing we notice *post mortem*; but with so little lymph (and in these cases we find rather opacity of membrane than lymph), it is quite unlikely the foramen of Magendie and other openings into the fourth ventricle are blocked, but rather that there is an inflammatory change in the ependyma of the ventricles (hardly recognisable except by the microscope), which has caused the excess of intraventricular fluid; and it is this inflammatory condition of the pia mater forming the choroid plexus that is doubtless the cause of the excess of fluid in all cases. It may be clear, as in a pleurisy with effusion, but often it is turbid and sometimes distinctly reddened—conditions which show its inflammatory origin.

Now we come to a very important part of the subject, and

that is the question as to the relation of the increased pressure and softening to the symptoms and tendency to death. In order to try and answer this question I have prepared a table² of the *post-mortem* changes and the more serious symptoms associated with these in ten cases of meningitis—seven of tuberculous meningitis, three of non-tuberculous to compare with them. With regard to the relation between the amount of softening and the duration of the coma, out of the ten cases in five the softening was not marked, and in one of these the duration of both the drowsiness and the actual coma exceeded their duration in cases where the softening was very marked; in the other cases the drowsiness and coma were both of short duration; so that we find in cases where the coma is not marked little softening, but it must be observed that two of the cases were non-tuberculous, and therefore free from infiltration of miliary tubercles and inflammatory products in the brain tissue. But still I think the fact that half the cases in which there was little or no softening were cases in which there was no marked coma shows a certain relationship between the softening and the coma.

It seems to me very difficult to understand why we get unilateral spasm or convulsion or paralysis or conjugate deviation in a bilateral affection like tuberculous meningitis. In those cases in which such conditions occurred there was no difference in the pathological appearances on the two sides and no evidence of thrombosis, and a hemiplegia, or hemispasm in arm and leg could not be caused, as a squint might, by a little more matting together of the parts about the base on one side than the other. We cannot at any rate put these unilateral affections down to increased intraventricular pressure.

Sometimes the convulsions were bilateral, but one would hardly think even bilateral convulsions were caused by pressure. Convulsions of any kind are not common in surgical compression.

In surgical compression we generally have slowing of the pulse. In these cases, as a rule, there was marked slowing, with often extreme irregularity, but this was chiefly during the early stage of vomiting and headache, and, when drowsiness and coma had set in, rapidity of pulse was rather to be noted.

As a rule the pupils were dilated to enable a frequent examination for miliary tubercles to be made; but in the six cases in which their condition is noted they were widely dilated in five, a condition common but by no means invariably present in surgical compression.

Although the relation between the softening and coma is such as to faintly encourage us, the nature of some of the more serious symptoms would not lead us to hope that they are merely due to compression, and that by diminishing the intraventricular pressure we could relieve them. But we understand so little of the intimate connection of brain changes and symptoms that it is rather to the pathological changes which seem to be largely the result of pressure than to symptoms as indicating compression, that we must look for guidance in trying to determine the probable value of this new method of treating tuberculous meningitis, by tapping the subarachnoid space of the cord.

What symptoms can be relieved by it only the practice of the operation can show; that out of four cases operated on in the Middlesex Hospital, in one there should have been contraction of the unduly dilated pupils, and in another slight improvement in the general condition, is encouraging when we remember that drainage was not maintained in all the cases. The operation does no harm, and as the patient is already comatose no anæsthetic is required.

But in any efforts we may make to remove the more serious symptoms of tuberculous meningitis by draining the intraventricular fluid we must remember it is nearly always only part of general tuberculosis, which may, and probably will, prove fatal in other ways, though if in this rapidly fatal meningitis we can prolong life it may be some time longer before the general tuberculosis does its deadly work.

² Not here reproduced.

PROFESSOR NOTHNAGEL celebrated his 50th birthday on September 28th. He succeeded Duchek in the Chair of Medicine in the University of Vienna in 1882.

A CORONER'S NOTES ON SUDDEN DEATHS.

Read in the Section of Medicine at the Annual Meeting of the British Medical Association held in Bournemouth, July, 1891.

By W. WYNN WESTCOTT, M.B.LOND.,
Deputy Coroner for Central Middlesex.

THESE observations are founded on the records of deaths which have taken place during the last three years in London, and which have been investigated by me. Broad principles alone can be sought out, and strictly accurate minute pathology or exact inference is not procurable in these inquiries. But even with such limitations these notes, I believe, give indications which may not safely be neglected.

The latest 1,000 inquests have been selected from my register, and have been tabulated. I have collected from this 1,000 all the cases that could be described as "sudden deaths," excluding, however, violent deaths, murders, manslaughters, and suicides, and also accidental deaths; I have excluded also all deaths under the age of 12 years, so as to avoid the great number of infantile deaths in which the cause may be doubtful. For example, when a baby is found dead in bed, although the death is probably sudden, and no doubt unexpected, yet there is very great difficulty in deciding whether such a death is from accidental suffocation, or from spasm of the glottis.

Of the 1,000 deaths upon which inquests were held, there were 303 which might be properly called sudden, unexpected, and natural in cause. Of these 303 cases, there were 185 males and 118 females; and of these 303, the medical witnesses affirmed that in 88 of them, or in 29 per cent., the death was either directly or indirectly attributed to excessive use of alcoholic liquors; of the 185 males, 57 were alcoholic; of the 118 females, 31 alcoholic; that is, alcoholic excess caused almost one-third of the male deaths and more than one-fourth of the female deaths, or, more accurately, 30.8 per cent. of the men, and 26.27 per cent. of the women died suddenly, unexpectedly, and without warning from the effects of intemperance, in addition to the very large number who died during the same period of slower but no less surely fatal alcoholic poisoning.

I was somewhat in doubt as to how I should classify these cases, as to cause of death, in order to provide a useful result. Only a few of the deaths had occurred in hospitals, or had been examined and inquired into by pathologists, so that any attempt at highly scientific methods was useless. *Post-mortem* examinations had been made in a great majority of the cases, but not in all; but even when made, the examinations were, of course, conducted by ordinary practitioners, and made no pretence to being exhaustive. Yet, inasmuch as the same or very similar conditions will be maintained for many years to come, the results of even these superficial examinations may give us much valuable and real information, and may supply the profession (and could, indeed, enlighten the public) with some details concerning ever-present dangers, and afford some hints of necessary and desirable precautions tending to the preservation of life. My purpose is not specially to carry on the crusade against alcohol; but the immense importance of the drink question forces itself on our notice, and cannot be set aside, so I will add the figures relating to this cause of mortality, after the specification of the pathological causes of death.

Bearing in mind that the majority of the above-mentioned medical practitioners would be likely to be most familiar with the old-fashioned classification of the causes of death into the three classes of death commencing at the heart, lungs, or brain, and believing that they had in cases of doubt been influenced by this method in assigning causes of death, I decided to divide these 303 deaths into (1) those assigned to syncope, or failure of the heart; (2) those marked by coma; and (3) those apparently due to asphyxia, or suffocation—although it may be true, as Stevenson teaches, that coma itself ends in either syncope or asphyxia.

This division showed:

1. Syncope	210 cases.
2. Coma	64 "
3. Asphyxia	29 "