Clinical and other Notes
carried on as usual with no complaints from the teachers or pupils. In conclusion I think this form of prophylaxis is worth further trials in view of its simplicity and small cost.

I wish to thank Assistant Surgeon Aliug of the British Station Hospital, Kasauli, and Assistant Surgeon Raj Singh, of Sanawar, for their valuable assistance in carrying out these experiments.

REFERENCE.


A CASE OF BLACKWATER FEVER OCCURRING IN MESOPOTAMIA.

BY CAPTAIN A. G. HARSANT.
Royal Army Medical Corps.

The 2nd Battalion York and Lancs. Regiment, were heavily infected with malaria whilst stationed at Kasvin (Persia) during autumn of 1920 and spring of 1921.

Lance-Cpl. G., of that Regiment stated that he had sixteen attacks of malaria during that period, for ten of which he was admitted to hospital. He had been having ague attacks every three to four weeks and was discharged from hospital after the last of these, about April 10. He had taken much quinine by mouth and was given daily intramuscular injections of quinine for eight days in December, 1920. He was admitted to 23rd British Station Hospital, Baghdad, on April 25, 1921, apparently suffering from an ordinary malarial relapse. For two days he had an intermittent temperature. His colour and general condition were good, his spleen was enlarged considerably below the costal margin and tender, and he vomited at intervals. He was given a mixture of quinine fifteen grains, three times a day.

On the evening of the third day, he complained of pain in the right side of his chest, apparently superficial; no abnormal physical signs could be detected in his chest.

On the fourth morning patient was very collapsed, was vomiting frequently; his skin was slightly jaundiced and his urine was almost black. He was given an intramuscular injection of quinine bihydrochloride fifteen grains at 7.30 a.m. At 10.30 a.m. he had a severe rigor, temperature 103° in axilla. Pulse 136, very weak. He was vomiting bilious fluid and his jaundice was rapidly increasing. There was tenderness over the spleen and in the right hypochondrium where the lower edge of the liver was just palpable. He was given a further intramuscular injection of quinine at 11 a.m. On the fifth day, the patient was pallid and collapsed, and was still vomiting. One pint of bicarbonate and saline, and ten grains quinine bihydrochloride were given intravenously. The bicarbonate and saline injection was repeated on the seventh day, when the patient's condition was still critical.

There was definite improvement after each of these injections: his temperature however rose to 104° on the seventh day.
Although malarial parasites could not be demonstrated in his blood—he was given an intramuscular injection of quinine on five successive days. This resulted in a steady fall of temperature—and an equally steady improvement in symptoms.

The first few days he was given large quantities of alkaline drinks, with brandy and raisin tea; these for the greater part he retained. He passed from forty to sixty ounces of urine in twenty-four hours; haemoglobin was not seen after the sixth day.

There was a friction rub heard over the spleen on the eleventh day—he complained of severe pain in this region for the following twelve days—and of flatulence and abdominal distension.

There were no abnormal physical signs at any time.

He steadily improved—was brought before a Medical Board and was evacuated to the base for transfer to England on the twenty-fourth day—May 28, 1921.

Four examinations of blood, both by thick and thin film methods, failed to show any malarial parasites (but the patient had been taking quinine before admission to hospital).

**Blood Counts:**

*May 3, 1921... 1-8 million per c.m.m. 45 per cent of control blood*

May 5, (repeated) 2-0 

May 13, 2-55 

May 26, 3-88 

*Hæmoglobin*

55 per cent 

60-65 per cent

**Differential Count: May 5, 1921.**—Polymorphs 50 per cent; lymphocytes 45 per cent; mononuclears 5 per cent (polychromatophilia marked).

The hæmoglobin was estimated, by comparison in a Gowers hæmoglobinometer with a control blood—as the standard tint was obviously unreliable.
Clinical and other Notes

Examination of urine: April 28, 1921.—Very dark brown, strongly alkaline, heavy cloud of albumin. No bile pigments; deposit numerous granular casts.

May 3, 1921.—Clear yellow, very faint trace of albumin; numerous granular casts—some apparently formed by ghosts of red cells.

May 18, 1921.—Alkaline yellow; no albumin; no casts.

My thanks are due to A.D.P. Mesopotamia, Lieutenant-Colonel Hamerton, D.S.O., R.A.M.C., for valuable advice during the progress of the case—and to Lieutenant-Colonel H. Herrick, C.M.G., D.S.O., R.A.M.C., for permission to publish these notes.

THE RELATIVE INCIDENCE OF INJURIES BY GUNSHOT WOUNDS TO PERIPHERAL NERVES.

BY CECIL WORSTER DROUGHT, M.A., M.D.CANTAB., M.R.C.P.LOND.

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The following is a statistical survey of 1,602 cases of gunshot wounds of peripheral nerves, comprising 1,944 nerve lesions personally examined from 1914 to 1920. As the total number of cases is rather larger than most published individual records (e.g., 639 cases by Tinel and 520 by Purves Stewart and Evans) they may, perhaps, give a more correct idea of the proportion in which the different nerves are affected both separately and in association with other nerves. The lists are arranged in order that the frequency with which any individual nerve is injured may be compared with the number of times that nerve is injured in conjunction with other nerves—thus, the figure appearing after the word "alone" refers to the number of cases of isolated injury to that particular nerve; while the figure following the word "total" indicates the total number of cases of injury to the nerve in association with other nerves as well as alone. Multiple injuries are also classified separately from isolated lesions.

Owing to the difficulty of diagnosing a pure nerve injury in the case of the optic and auditory nerves, the first and eighth nerves are intentionally omitted from the list of cranial nerves. To illustrate the relative frequency with which different portions of the brachial plexus are involved, injuries to this important structure are considered as affecting (1) the roots of the nerves forming the plexus; (2) outer cord; (3) posterior cord; (4) inner cord; and (5) two or more cords.

Cranial nerves.

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Total</th>
<th>Alone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor oculi (III)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trigeminal and branches (V)</td>
<td>13</td>
<td>8</td>
</tr>
<tr>
<td>Abducant (VI)</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Facial (VII)</td>
<td>37</td>
<td>27</td>
</tr>
<tr>
<td>Glossopharyngeal (IX)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Vagus and branches (X)</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Spinal accessory (XI)</td>
<td>12</td>
<td>7</td>
</tr>
<tr>
<td>Hypoglossal (XII)</td>
<td>9</td>
<td>3</td>
</tr>
</tbody>
</table>