Reflex vasomotor responses in the hands of migrainous subjects

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It has been suggested recently that migraine, rather than being a vasomotor abnormality confined to the cranial arteries, may be a generalized disturbance of arteries throughout the body (Appenzeller, Davison, and Marshall, 1963; British Medical Journal, 1964). The principal evidence for this hypothesis is the study by Appenzeller et al. (1963) of vasomotor reflexes in the hands of migrainous subjects. These workers measured blood flow in the hand in response to heating of the trunk by means of venous occlusion plethysmography in the manner described by Kerslake and Cooper (1950). Observations were made on 10 migrainous patients in whom all medication had been suspended for at least 48 hours before the study. The results were compared with those obtained in 10 non-migrainous controls. All of the controls showed a normal response of rapid vasodilatation, while eight of the 10 migrainous patients failed to demonstrate a significant change. Appenzeller et al. concluded that this provided evidence of an abnormality in the control of blood flow through the hands of some migrainous subjects, which persisted between attacks. Finally, it was postulated that the lack of vasodilatation reflects a generalized abnormality of the blood vessels or their neural control, which is continuously present in some migrainous subjects.

It is well known that the loss of function experienced during a migrainous aura may occasionally become permanent and the patient is left with hemianopia or hemiparesis. If migraine is only one facet of a generalized vasomotor disorder, then the possibility arises that damage to tissues in other parts of the body might also be attributable to this disorder. We felt, therefore, that it was important to repeat the observations on vasomotor control in migrainous subjects, and the present paper reports the results of a similar study on 15 patients suffering from typical migraine.

METHODS

Each of the 15 patients who were studied gave a clear-cut history of migrainous headaches accompanied by an aura. Control observations were made on 11 subjects, of whom five were healthy volunteers, and six patients convalescing from illnesses which were neither cardiovascular nor neurological, apart from no. 10, who had epilepsy. All subjects were studied in a warm, quiet environment, lying on a bed in their underclothing only. A heat cradle carrying six 100 watt lamps was placed over the bare trunk and thighs. The head end of the cradle was closed by blankets, which obscured the light, while the opposite end was left open. Hand blood flow was measured with an air-filled venous occlusion plethysmograph.

Hand blood flow was measured every 10 seconds with the cuff inflated to 60 mm.Hg for five seconds. Resting flow was measured on six successive occasions. The heat cradle was then turned on and a further six flows recorded. Finally, the lights were turned off and six more flows recorded. Thus each run consisted of 18 flows at 10-second intervals over a period of three minutes. A rest period of three minutes was allowed between every run and 10 to 12 runs were performed on each subject. Blood flow was deduced from measurements of the slopes of the inflow curves in the usual fashion and is expressed as ml./100 ml. hand/minute.

RESULTS

A mean flow was calculated for each of the 18 flows recorded at corresponding times during the 10 to 12 runs. We observed, as did Kerslake and Cooper (1950), that the first measurement after turning on the heat frequently showed a fall in flow, which was sometimes considerable. This presumably accounts for the method by which Appenzeller et al. (1963) expressed their results in response to heating as the mean change in hand blood flow observed during the last 32 seconds of a period of 40 seconds’ irradiation. Our method differs slightly from that of Appenzeller et al. in that blood flows were recorded for five rather than for four seconds, in order to increase the length of the slope and thus facilitate accuracy of measurement. Heating was continued for 60 seconds. Our results are presented in a form which is comparable to that of Appenzeller et al.,
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by taking the three measurements over each period between 15 and 40 seconds of irradiation and expressing them as the mean change in hand blood flow observed over this period. These results are recorded in Table I. The total results have also been included in the form of a graph comparing the migrainous patients with the control subjects (Fig. 1). The mean for the six blood flows measured during the first minute is taken as the baseline. Subsequent flows for each individual have been recorded as a rise above or a fall below the baseline and the results averaged for the two groups.

DISCUSSION

It can be seen that both control and migrainous groups behaved in a similar manner. With the exception of no. 7, the migrainous patients showed normal vasodilatation in response to body heating. This subject showed decreased flows during heating over the first three runs but then responded normally for the final seven runs. The effect of the initial three runs was to reduce his mean dilatation to —0.4. He was a nervous subject and the surprise caused by the sudden heat evidently continued to prove a stronger stimulus initially than the heat itself. If the flows are calculated from the final seven runs during heating, there is a mean dilatation of 1.7.

As already mentioned, the initial response to the sudden turning on of the heat results in most cases in transient vasoconstriction. It is well known in

![Graph](image)

**Figure 1.** Results for migrainous patients and controls combined.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Basal Flow</th>
<th>Flow during Heating</th>
<th>Mean Dilatation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Migrainous Patients</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>7.5</td>
<td>10.3</td>
<td>2.8</td>
</tr>
<tr>
<td>2</td>
<td>3.7</td>
<td>6.4</td>
<td>2.7</td>
</tr>
<tr>
<td>3</td>
<td>10.2</td>
<td>14.6</td>
<td>4.4</td>
</tr>
<tr>
<td>4</td>
<td>31.7</td>
<td>33.7</td>
<td>2.0</td>
</tr>
<tr>
<td>5</td>
<td>13.8</td>
<td>15.4</td>
<td>1.6</td>
</tr>
<tr>
<td>6</td>
<td>8.8</td>
<td>10.3</td>
<td>1.5</td>
</tr>
<tr>
<td>7</td>
<td>13.9</td>
<td>13.5</td>
<td>0.4</td>
</tr>
<tr>
<td>8</td>
<td>6.2</td>
<td>10.6</td>
<td>4.4</td>
</tr>
<tr>
<td>9</td>
<td>8.3</td>
<td>10.7</td>
<td>2.4</td>
</tr>
<tr>
<td>10</td>
<td>20.6</td>
<td>27.2</td>
<td>6.6</td>
</tr>
<tr>
<td>11</td>
<td>8.9</td>
<td>12.6</td>
<td>3.7</td>
</tr>
<tr>
<td>12</td>
<td>6.0</td>
<td>8.7</td>
<td>2.7</td>
</tr>
<tr>
<td>13</td>
<td>18.8</td>
<td>21.0</td>
<td>2.2</td>
</tr>
<tr>
<td>14</td>
<td>12.7</td>
<td>17.2</td>
<td>4.5</td>
</tr>
<tr>
<td>15</td>
<td>25.6</td>
<td>27.8</td>
<td>2.2</td>
</tr>
<tr>
<td>Mean = 2.9 (S.D. ± 1.5).</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Control Subjects | | | |
| 1 | 7.8 | 8.6 | 0.8 |
| 2 | 21.5 | 28.6 | 7.1 |
| 3 | 17.9 | 21.3 | 3.4 |
| 4 | 10.0 | 12.4 | 2.4 |
| 5 | 22.1 | 26.0 | 3.9 |
| 6 | 5.8 | 6.4 | 0.6 |
| 7 | 13.6 | 18.1 | 4.5 |
| 8 | 11.5 | 13.2 | 1.7 |
| 9 | 11.3 | 12.1 | 0.8 |
| 10 | 4.8 | 10.9 | 6.1 |
| 11 | 10.8 | 12.7 | 1.9 |
| Mean = 3.0 (S.D. ± 2.1). |

*Results are expressed as ml./100 ml. hand/minute.
such studies that any sudden physical or mental stimulus will have this effect. Turning off the heat is a milder stimulus as the cradle is still warm, but this also tended to cause slight vasoconstriction. The degree of vasodilatation during the heating period varies to some extent between individuals, but it is also dependent upon the strength and the size of the stimulus. Our particular heat cradle was large, the caudal end was left open, and the area heated was slightly reduced by the wearing of pants. The vasodilator response corresponds in magnitude to that found by Kerslake and Cooper (1950) for a 350 watt stimulus. Appenzeller et al. (1963) argued that ergot alkaloids could not account for the abnormal response in their migrainous subjects, since none of their patients had been taking drugs for 48 hours before the experiment. Moreover, the two migrainous subjects in their series who responded normally had both been taking the drug for long periods. Our results bear this out, since subjects nos. 13 and 14 had taken ergotamine within 24 hours of measuring their flows and both behaved normally. Furthermore we repeated the study on one subject, no. 8, after administering 4 mg. ergotamine tartrate in 24 hours, and although the absolute volumes of his flows were reduced, there was still a vasodilator response to heating (Table I).

<table>
<thead>
<tr>
<th>Basal Flow</th>
<th>Flow during Heating</th>
<th>Mean Dilatation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before ergotamine</td>
<td>6.2</td>
<td>10.6</td>
</tr>
<tr>
<td>After ergotamine</td>
<td>2.7</td>
<td>4.4</td>
</tr>
</tbody>
</table>

1Results are expressed in ml./100 ml. hand/minute.

**SUMMARY AND CONCLUSION**

The present study fails to confirm the finding that migrainous subjects have an abnormal reflex hand blood flow response to trunk heating. Both control and migrainous groups behaved in a fashion which corresponds closely to that described in normal subjects. It is concluded that there is at present inadequate evidence to support the conclusion that there is a generalized abnormality of vasomotor control in patients with migraine.

**REFERENCES**


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