veins of the legs) and found that there was increased radioactivity in the thrombosed limb. We have similarly found such increased activity and gamma scans of the legs show that this activity is confined to hot spots, probably at valve pockets. It may be that such local vascular accumulations are associated with venous thrombosis, but they are not diagnostic.

Brownell and colleagues studied a large number of patients without venous thrombosis, but since many patients were excluded from their survey with conditions often confused with deep vein thrombosis—for example, superficial phlebitis, haematomata, arthritis, cellulitis, and gross oedema of the legs—the control group of venographically negative legs cannot be considered representative. In a series of 32 patients including such conditions, using the 115I fibrinogen test as in the original method of Flanc, Kakkar, and Clarke,1 we have not found correlation of increased 115I fibrinogen uptake with deep venous thrombosis. However, there was often a higher initial count over the affected limb, possibly owing to pooling of the isotope in the deep veins.

There is a great danger of overdiagnosis of epidemic keratoconjunctivitis. In the case of Schütz's tonometer I suppose that readings could be taken in such a way, though their reliability would seem to be open to question. However, I have always sterilized the Schütz's instrument before use by holding the footplate in the flame of a spirit lamp for about five seconds, and I am assured by my bacteriologist friends that this will effectively destroy the adenovirus and any other infective agents.

The application of the tonometer presents a more difficult problem. I cannot imagine that it could be used through any rubber membrane, however fine, and the application will not withstand flame sterilization, while soaking in antiseptic solutions is an unreliable method of removing virus contamination. Application tonometers and the various diagnostic contact lenses are now in regular use in eye clinics, and the usual methods of cleaning these instruments scarcely deserve the name of sterilization. I am sure that the choice of a sympathetic person will feel unenthusiastic about this problem, and would welcome guidance on it.—I am, etc.,

IAN W. PAYNE

Plymouth

Obesity and Smoking Habits

Sir.—Dr. G. Pincherle (30 October, p. 298) has called for details of obesity and smoking habits by social class. The following data were analysed in the recent paper stage of reports on dietary sugar, smoking, and obesity,1,2 but were not published in detail. The figures are not directly comparable with those of Dr. Pincherle or those of Dr. T. Khosa and Professor C. R. Lowe (2 October, p. 10) since they show weight gain in men (aged between 40 and 54 at the time of the survey) since the age of 25. One might well expect that weight gain in this span of 15 and 29 years would show the effect, if any, of social class. The figures show that current cigarette smokers (most of whom have smoked for over 20 years) have gained less, on average, than ex-smokers, those currently smoking pipes and cigars only, and those who have never smoked. This is in line with the findings of Dr. Khosa and Professor Lowe and of Keys et al.3 though Dr. Pincherle might well think that the variations between the smoking categories in this present series do not show the "considerable differences" demonstrated by Dr. Khosa and Professor Lowe. The means of weight gain are standardized for social class and it is clear from the Table that social class had little effect in this sample on smoking habits and obesity.

Mean Weight Gain by Smoking Habits in Males Aged 40-54 Years

<table>
<thead>
<tr>
<th>Smoking Habits</th>
<th>Never smoked</th>
<th>Ex-smokers</th>
<th>Cigars/pipes only</th>
<th>Cigarettes</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of men</td>
<td>34</td>
<td>17-7</td>
<td>18-8</td>
<td>18-8</td>
<td>18-9</td>
</tr>
<tr>
<td>Mean weight gain (lbs)</td>
<td>17-8</td>
<td>17-8</td>
<td>18-8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean weight gain after social class standardization</td>
<td>18-8</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than</td>
<td>20</td>
<td>cigarettes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 or more</td>
<td>—</td>
<td></td>
<td>—</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The data on which this Table is based confirm the findings of an earlier study4 in which male manual workers (lower social classes) were heavier at all heights and ages than non-manual workers (higher social classes). Weight gain and social class are related in these present data when smoking habits are disregarded. Weight gain was lowest in those smoking 1-19 cigarettes a day; both Dr. Pincherle and Dr. Khosa and Professor Lowe found that this class of smoker had the lowest incidence of overweight. This analysis was prepared during the course of employment with the U.K. Atomic Energy Authority.—I am, etc.,

R. W. HOWELL

Tilehurst, Reading, Berks


Cot Deaths

Sir.—With reference to your leading article on "Cot deaths" (30 October, p. 250) I propose, as a hypothesis to be tested, that non-traumatic atlanto-axial dislocation which occurs in children in association with throat infection is the cause of some of the cot deaths. Non-traumatic atlanto-axial and also occipito-atlanto dislocation in children following sometimes trivial throat infection have been described by many authors since 1875.1 In children such dislocation manifests itself by vomiting and cervical stiffness.

In the infant the consequence of dislocation may be instant death owing to pressure on the spinal cord; spontaneous reduction may occur with damage to the cord. Reduction of the dislocation may occur even after death. In the adult dislocation of the atlas occurs only as a result of severe trauma and is associated with fracture of the odontoid process.

The anatomical features of the atlanto-axial joints of the infant differ greatly from those of adults or of older children and the different anatomical features in each age group explain the different sequence of events. In the adult the articular facets are large, concave, inclined, and divergent. They form a cup for the reception of the condyle; this strong ligaments prevent dislocation. In the infant the articular facets are flat, there is no inclination of the facets, and the condyles are small. Therefore dislocation occurs more easily in adults. In infants various forms of the anomaly are associated with its development are known to occur. Some variants may be more liable to dislocation than others. Such variants may be similar in brothers and sisters and this similarity of the anatomy may explain that more than one cot death occurred in some families. Genetical factors appear to be implicated.

Cowdry brought forward the theory that the atlanto-axial joints, also the transverse ligament, are affected by inflammatory processes of the throat and of the neck and he supported his view by the finding that the lymph channels which connect the lymphnodes of the neck with the atlanto-axial joints. Atlantoaxial dislocation in the infant may cause instant death due to damage of the spinal cord; this is more likely to occur during sleep when muscles are relaxed and reflexes fail or cause harmful spasm. In such event the cause of death will be difficult to detect unless suspected causes are looked for. Dislocation of the atlas may be reduced by movement of the head even after death and therefore the head and neck should be well supported especially during transport.

I suggest that following examinations should be carried out in sudden infant death in addition to those that are done at present. External examination; the tip of the transverse process should be located by palpation. Normally this tip can be felt just in front of the mastoid process. In occipito-atlanto dislocation, which is probably very rare, the tip can be felt behind the mastoid. In atlanto-axial dislocation, however, this tip will be in front of the mastoid. The posterior surface of the oropharynx should be examined by inspection and by palpation; x-ray examination of the atlanto-axial region: lateral and