failures) than would be expected by its 70% share of the market.

In the UK, vaccination against Hib disease was begun in October, 1992, and had achieved 90% coverage in children aged 12 months by November, 1993.1 Children receive three doses of PRP-T vaccine at 2, 3, and 4 months as part of the standard vaccination programme. One dose of HbOC was used for catch-up immunisation in children who were less than 4 years but more than 12 months at the start of the programme. We have compared the incidence of Hib meningitis in England and Wales for the 12 months before the vaccination programme began (Aug 1, 1991, to July 31, 1992) with the 12 months after vaccination (Aug 1, 1993, to July 31, 1994) to see whether the effect of vaccination more closely resembles the experience in the Netherlands or that in Germany. In addition, we have stratified the incidence by age to determine if the incidence of disease in younger children (<2 months) who have not yet been vaccinated has been affected by vaccination of older children.

![Graph showing incidence of Hib meningitis](image)

Figure: Hib meningitis in England and Wales by age
Source: Laboratory reports to Public Health Laboratory Service, Communicable Disease Surveillance Service.

The annual incidence of Hib meningitis fell in children under 5 years from 10.4 per 100,000 before vaccination to 0.6 per 100,000 after vaccination (358 and 19 cases, respectively). The drop more closely resembles the fall seen in the Netherlands (from 22 to 0.6 per 100,000) than that recorded in Germany (from 23 to 1.9 per 100,000). In addition, we noted a large fall in the incidence in children under 2 months who had not yet received the vaccine (figure).

These findings show that the PRP-T and HbOC vaccines have achieved a 95% reduction in disease among vaccinated children and a 75% reduction in children under 2 months of age who have not received the vaccine. The latter observation presumably related to decreased carriage among vaccinated children.

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Acute epiglottitis after Hib vaccination

SIR—Booy and colleagues (Aug 6, p 362) report 100% efficacy of Haemophilus influenzae type b (Hib) conjugate vaccine PRP-T in the prevention of Hib infection. We report a case of acute epiglottitis after Hib in a fully vaccinated child.

The child was aged 22 months and presented to her local hospital with a 6 h history of pyrexia and respiratory distress. She was pale, deeply cyanosed, and tachycardic. Gas induction anaesthesia was undertaken and she was orally intubated. She was referred to our paediatric intensive care unit with a provisional diagnosis of acute laryngotracheobronchitis. Laryngoscopy, for nasal intubation, revealed a swollen oedematous epiglottis, characteristic of acute epiglottitis. A swab of the epiglottis was taken and blood culture was done. High-dose intravenous cefotaxime was started. The mother was confident that all the child's vaccinations, including Hib, had been completed. Over the following 18 h the child's temperature fell from 39.5° to 37.3°. She was then successfully extubated with only slight intermittent stridor. Hib was identified from both the blood culture and the epiglottic swab. At this point we confirmed that the child had been vaccinated at the recommended ages of 2, 3, and 4 months with the same vaccine (ActHIB, Merieux) on each occasion. There was no evidence of immune deficiency, with normal white cell numbers and immunoglobulins.

Previously meningitis due to Hib has been reported when vaccination has been incomplete.1,2 This case shows that life-threatening infection from Hib may still arise after vaccination according to the British accelerated immunisation schedule. Hence invasive Hib infections should still be considered in all vaccinated children.

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Syncope and near-death experience

SIR—The existence of near-death experiences is no longer debatable. Their origin, however, is still a matter of controversy: physiological, psychological, and transcendental explanations have been offered.1 Whilst studying motor phenomena of syncope2 we were impressed by similarities between syncopal hallucinations and near-death experiences.

Syncope lasting up to 22 s was induced in 42 healthy young adults by hyperventilation and Valsalva manoeuvre. Subjects reported visual hallucinations—perception of colours and lights which could intensify to a glaring brightness, or landscapes and familiar people, in some cases with no discernible faces; out-of-body experiences—scenes in which they were involved yet they observed them from above; and auditory hallucinations that ranged from roaring noises to screaming or unintelligible human voices.

Most subjects described the emotional experience of syncope as pleasant, detached, and peaceful, making them unwilling to return. Some compared it to drug or meditation experiences. 2 were reminded of an earlier post-traumatic near-death experience. One participant disclosed: "I thought..."
that if I had to die in this very moment I would willingly agree."
The frequency of hallucinatory elements was similar to
that in near-death experiences:

<table>
<thead>
<tr>
<th>Near-death experience</th>
<th>Syncope %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Out-of-body experience</td>
<td>36</td>
</tr>
<tr>
<td>Visual perceptions</td>
<td>38</td>
</tr>
<tr>
<td>Audible noise or voices</td>
<td>40</td>
</tr>
<tr>
<td>Feeling of peace and painlessness</td>
<td>42</td>
</tr>
<tr>
<td>Appearance of light</td>
<td>44</td>
</tr>
<tr>
<td>Life review</td>
<td>46</td>
</tr>
<tr>
<td>Entering another world</td>
<td>48</td>
</tr>
<tr>
<td>Encountering preternatural beings</td>
<td>50</td>
</tr>
<tr>
<td>Tunnel experience</td>
<td>52</td>
</tr>
<tr>
<td>Knowledge of the future</td>
<td>54</td>
</tr>
</tbody>
</table>

Near-death experience during syncope has been reported
anecdotally. Our experiment confirms the possibility of
cerebral hypoxia to induce near-death experiences which
may represent an agonal limbic syndrome rather than a
glimpse into another life after life.

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Idiopathic thrombocytopenic purpura after human herpesvirus 6 infection

Sir—Yamanishi and his colleagues revealed the causative
agent of exanthema subitum as human herpesvirus 6
(HHV-6). Many complications of exanthema subitum have
been reported: febrile convulsion, liver dysfunction,
meningitis, encephalitis, intussusception, and pneumonia.
Before the discovery of HHV-6, there was one report of
thrombocytopenic purpura associated with exanthema
subitum and recently a case of exacerbation of idiopathic
thrombocytopenic purpura by primary HHV-6 infection was
reported. We report the first case of idiopathic
thrombocytopenic purpura-associated exanthema subitum.

On July 8, 1994, a 6-month-old Japanese girl had a
temperature of 37.6°C. Next day fever rose to 38.0°C and
she was seen by a paediatrician and given josamycin and
paracetamol (400 mg per day). 2 days later petechiae
appeared on her lower limbs, she was sent to Mie National
Hospital.

On admission, platelet count was 68 000/mm³. On day 5
of the illness, bone marrow examination showed
megakaryocyte count was 106/mm³ and 97% of
megakaryocytes were non-platelet forming type. Platelet-
associated IgG was 139-2 ng/10⁹ cells (normal range, 9 to
25). Idiopathic thrombocytopenic purpura was diagnosed.
Fever decreased the same day and a rubella-like eruption
appeared on her face and trunk. On day 6, petechiae faded,
and by day 7 the rash had faded and petechiae disappeared.
Platelet count was 141 000/mm³. Next day the rash disappeared. She was discharged on day 10 with a platelet count of 632 000/mm³.

Mononuclear cells of peripheral blood and bone marrow
from day 5 were cultured with RPMI 1640 containing fetal
calf serum (10%), interleukin 2 (0.1 U/mL), and PHA
(5 µg/mL). After 10 days, cytopathic effect was observed.
Co-cultivation was done with cord-blood mononuclear cells
and cytopathic effect also observed. Infected cells were fixed
by cold acetone and indirect immunofluorescence was done
with anti-HHV-6 and anti-HHV-7 monoclonal antibodies,
healthy adult serum (HHV-6 type 160, HHV-7 type 640),
and phosphate-buffered saline. Fluorescence was positive
with anti-HHV-6 monoclonal antibody and healthy adult
serum, and negative with anti-HHV-7 monoclonal antibody
and saline.

The case we report may show that HHV-6 can cause
idiopathic thrombocytopenic purpura. HHV-6 infection of
megakaryocytes or cytokines released from HHV-6-infected
cells may have suppressed thrombocyte production. Knox et
al reported that HHV-6-infected normal bone-marrow
mononuclear cells inhibited, on average, 71% of the growth
of megakaryocyte linages in vitro.

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CORRECTION

Comparison of chorionic villus sampling and amniocentesis for fetal karyotyping at 10-13 weeks' gestation—In this article by Nicolaides and colleagues (Aug 13, p 435), table 3 was incorrect. The correct version is given below.

<table>
<thead>
<tr>
<th>Total n=1301</th>
<th>Amniocentesis</th>
<th>Chorionic vilus sampling</th>
</tr>
</thead>
<tbody>
<tr>
<td>n=493</td>
<td>Randomised n=238</td>
<td>Choice n=320</td>
</tr>
<tr>
<td>Survival</td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>1211</td>
<td>93-08</td>
<td>457 (92.7)</td>
</tr>
<tr>
<td>Total loss</td>
<td>89 (6-84)</td>
<td>35 (7-1)</td>
</tr>
<tr>
<td>Spontaneous death</td>
<td>52 (4-00)</td>
<td>25 (5-1)</td>
</tr>
<tr>
<td>Termination</td>
<td>37 (2-84)</td>
<td>10 (2-0)</td>
</tr>
<tr>
<td>Chromosomal defect</td>
<td>31 (2-38)</td>
<td>9 (1-8)</td>
</tr>
<tr>
<td>Normal karyotype</td>
<td>6 (0-46)</td>
<td>1 (0-2)</td>
</tr>
</tbody>
</table>

Table 3: Pregnancy outcome