deterioration but not reverse the course of the disease), early genetic counselling is of great importance.

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References


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Recovery from chronic solvent abuse

Sir: The evidence concerning impairment of cognitive functions in solvent abusers is still equivocal: reports have provided evidence both for1–3 and against4–6 cognitive impairment. In studies reporting cognitive impairment the question of whether these effects are reversible has not been satisfactorily answered. Ron7 in a recent review article emphasised that one of the shortcomings of investigations into cognitive dysfunction as a result of solvent abuse is the lack of adequately controlled follow-up studies.

We had the opportunity to conduct a series of examinations on a chronic glue sniffer who had been detained in an institution for young offenders. The institutional setting ensured that he had no access to solvents after his demand and we were able to chart the time course of recovery of intellectual functions over a period of 18 months under unusually controlled conditions.

Inmate AB (fictitious initials) who was 18 years old at the time of the initial interview came to our attention when he reported at the prison’s medical centre complaining of drowsiness, sleeplessness, lack of concentration and memory disorders. He also suffered from poor co-ordination, unsteadiness of gait and general sluggishness. He stated that he had started inhaling solvents at the age of 13 years and continued until the day he was arrested for attempted rape which he had committed while under the influence of solvents. At the age of 16 he habitually consumed 250–500 ml of glue per day 3–4 days a week. Over the year before he was arrested, his consumption had increased to 500–750 ml per day. His experience with other drugs was restricted to isolated experiments with alcohol and cannabis. He almost exclusively used Evostik an adhesive which contains 48.5% toluene and 5.3% n-hexane (information obtained from the manufacturer).

On initial presentation AB had been in custody for 4 months: a time assumed to be well beyond the acute effects of intoxication and immediate withdrawal from solvents. He was assessed neuropsychologically using both traditional measures and followed up on at 2 to 3 monthly intervals using selected tests from a computerised test battery which covers a variety of functions including visual, memory, attention, reaction time, visual search and word recognition. The computerised tests are repeatable, most without a significant training effect, and can be used to monitor recovery. AB was also assessed with

Table Summary of initial and follow-up results for AB and controls

<table>
<thead>
<tr>
<th></th>
<th>Initial assessment</th>
<th>18 months</th>
<th>12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AB</td>
<td>Controls</td>
<td>AB</td>
</tr>
<tr>
<td>Visual memory span (blocks)</td>
<td>13.5</td>
<td>14.7</td>
<td>3.5</td>
</tr>
<tr>
<td>Event detection threshold (ms)</td>
<td>241†</td>
<td>311</td>
<td>8.6</td>
</tr>
<tr>
<td>Movement detection (no. errors: max = 25)</td>
<td>21†</td>
<td>5</td>
<td>3.4</td>
</tr>
<tr>
<td>Visual search (ms)</td>
<td>3772†</td>
<td>2692</td>
<td>282</td>
</tr>
<tr>
<td>Word recognition threshold (ms)</td>
<td>245†</td>
<td>60.2</td>
<td>14.7</td>
</tr>
<tr>
<td>Body sway (deg/min)</td>
<td>17.7†</td>
<td>4.9</td>
<td>1.6</td>
</tr>
<tr>
<td>Reaction time: simple (msec)</td>
<td>319*</td>
<td>254</td>
<td>30.3</td>
</tr>
<tr>
<td>Decision time: Movement time</td>
<td>297*</td>
<td>195</td>
<td>20.7</td>
</tr>
<tr>
<td>Choice (ms)</td>
<td>344</td>
<td>295</td>
<td>27.8</td>
</tr>
<tr>
<td>Decision time</td>
<td>341†</td>
<td>198</td>
<td>24.3</td>
</tr>
</tbody>
</table>

Figures in brackets are standard deviations.

* Differences > two SDs. † Differences > three SDs.
Letters

a Wright-Codoc ataximeter. A detailed description of the tasks is given in. 8

All scores on the WAIS subtests were significantly lower compared with standard norms and to a control group (see table). His score on the Ravens Matrices placed AB at the lower end of the “intellectual average” group: assessment on the Matrices was done 12 months after the initial referral.

The control group consisted of 12 prisoners with a similar socioeconomic and criminal background matched on age and IQ who had no history of solvent abuse. Controls were given four assessments over a period of 1 year. Since they did not show any difference in performance between the third and fourth assessment, it was assumed that no further improvement due to practice would occur and testing was discontinued. AB was followed up at 3 monthly intervals over a period of 18 months. He gradually recovered to normal functioning on seven tasks, but after 18 months was still impaired on two tasks. Scores on word recognition had improved considerably but he still showed a sizeable difference from the control group.

After 18 months AB was given a standard neurological examination, performed by a visiting neurologist, which showed no signs of any residual focal or peripheral abnorm-

The study suggests that neuro-

psychological deficits can be detected in chronic solvent abusers after a period well beyond the acute effects of solvent use or withdrawal. However, even after extensive solvent abuse recovery is possible to a con-

siderable degree, although after 18 months of abstinence normal functioning is still not fully reinstated in the present case. This is consistent with previous studies reporting permanent neurological and psychological damage in solvent abusers. 9-11 The nature of the recovery process and the extent of permanent damage as a consequence of solvent abuse are not yet understood and demand further investigation.

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Acute cerebellar syndrome complicating infectious mononucleosis

Sir: Neurological complications of primary Epstein–Barr virus (EBV) infection occur in up to 1% of cases 1 2 and include meningo-
encephalitis, aseptic meningitis, optic neuritis, facial palsy, transverse myelitis and polyneuropathy. 3-8

We report the occurrence of a recognisable acute cerebellar syndrome due to infectious mononucleosis in two 15 year old teenagers, one of whom suffered permanent, though mild, sequelae.

The pathogenesis of cerebellar involve-
ment, which is infrequent and benign, with complete recovery in all reported cases, 5-9 still requires elucidation. Direct viral replication seems most likely but post-infectious demyelination has been postulated in some reports. 5-9

A fifteen year old schoolgirl presented in May 1982 with a 2 week history of sore throat, malaise, abdominal pain, vomiting and generalised headaches. Four days prior to admission she became unsteady on her feet and noted horizontal diplopia, particularly on left lateral gaze. Examination revealed an alert and orientated girl who was afebrile. The pharynx appeared normal and apart from mild left supravacular fossa lymphadenopathy, general examination was normal. Neurological examination disclosed accadoid ocular pursuit, horizontal nystagmus and horizontal diplopia on lat-
eral gaze to the right and left, and first degree downbeating nystagmus. A mild left lower motor neuron facial palsy and bilat-
eral limb and truncal ataxia were present. The haemoglobin level was 144 g/l and the white cell count was 4.4 x 109/l. The neutro-

phil count was 1.32 x 109/l (normal: 2.0-7.5) and the lymphocyte count was 2.46 x 109/l (normal range 1.5-4.0) with occasional reactive lymphocytes. The differential slide agglutination test (Paul Bunnell–Davidson) for infectious mononu-

cleosis was positive. Cerebrospinal fluid (CSF) examination revealed 18 leuco-

cytes/μL (95% lymphocytes, 5% neutrophils) and 0 red cells/μL. No bacteria were seen. CSF glucose was 3.2 mmol/l (normal 2.8-4.4) and protein 0.22 g/l (normal 0.15-0.45). CSF IgG/albumin ratio was 0.8 (normal 0.04-0.24). Brain stem auditory evoked potentials were normal bilaterally.

She was managed conservatively and at 1 month follow-up had improved symptomat-
ically. She still manifested gaze evoked horizontal nystagmus on lateral gaze bilater-
ally, upbeating nystagmus on upward gaze, downbeating nystagmus on downward gaze, mild limb ataxia and moderate truncal ataxia. At 6 months follow-up her physical signs were unchanged. At 5 years follow-up she was asymptomatic apart from slight horizontal diplopia on horizontal gaze bilaterally and an inability to run effectively. Examination revealed gaze evoked horizon-
tal nystagmus on lateral gaze bilaterally, first degree upbeating nystagmus and first degree downbeating nystagmus. Very mild limb and truncal ataxia persisted.

The second case was a 15 year old school-
boy who presented in January 1987 with a 3