Now I am well" (figure D, the first and second lines). In figure D, the seventh character right of the comma on the second line (we defined as L2R7) and L3R6 should be L4R7, which is an orthographic paragraphia. Then, she began to rewrite sentences from L3, although not exactly identical. These repetitive sentences were divided into two clauses by a comma. The first clause said, "My physical condition is not very good", which she repeated 17 times (L3 to L19). The second clause said, "(I) was sent to this hospital by a doctor of this hospital". There were several minor errors in the second clauses, including character omissions (between L3R4 and L3R5, L10R6 and L10R7, and L12R3 and L12R4) and redundant characters (at L7R6 and L23R10 to R12). She changed "friend" (L3R2 and L3R3) to "doctor" (L8R4 and L8R5) and unnecessarily repeated the modifier "this hospital" twice (L14R2 and L14R3 as well as L14R8 and L14R9). She wrote five identical copies of a long sentence (L14 to L18 of figure D). She stopped writing when the space on the paper was used up.

Our patient was discharged on the 14th day. Her husband reported that on the 17th day she watered flowers, but played with the water at the same time. She rapped kitchen utensils with a spatula when cooking, and rapped bowls with a spoon when eating. Three days later, she began to repeat phrases, and followed events in the stock market on television. However, she became angry with her husband without reason on the 21st day, which was unprecedented. On the 26th day, her verbal output seemed normal. The clonic motor perseveration subsided gradually and eventually stopped.

Most of this patient's symptoms had been described elsewhere.[1] The repetitive writing had been reported only for letters, characters, and phrases.[2] To our knowledge, repetition of a long meaningful sentence has never been reported. Our patient seemed totally involved when writing the sentences, and would not be distracted. Furthermore, the clonic motor perseveration and the repetitive sentence writing showed similarities in that both were repetitive and elicited by a stimulus. The clonic motor perseveration was elicited by nearby objects attracting her, and the repetitive sentence writing by an idea to describe her illness.

Regarding our patient's ability to write while she was mute, we suggest two points. Firstly, her inner language was intact. This resembles "dynamic aphasia" with which patients seem almost mute in conversational speech, but show a dramatic preservation of the ability to name objects, to read, and to repeat sentences.[3] Kleist thought that patients with dynamic aphasia had intact verbal propositional thought and intact "sentence schema" but these were disconnected.[4] Our patient wrote sentences with correct grammar and word selection to describe a meaningful idea, which supports this notion. Secondly, the mechanism for writing was intact. The neural substrates accounting for writing were unaffected in our patient, as shown by the proper sentence syntax and legible ideograms.

Dominant mesial frontal lesion may impair the processes of modification and monitoring of the motoric components of writing, resulting in disinhibition and loss of monitoring. Moreover, Shallice[5] proposed an idea of "supervisory attentional system" which controls and modulates the lower level processes. This system is activated when a person is dealing with a non-habitual condition and is thought to be a frontal lobe function. This explains why our patient rewrote the sentences so many times, yet did not detect the errors in the writing.

We thank Professor Atsushi Yamadori of Tohoku University, Japan for his helpful comments in the preparation of this manuscript.

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Peripheral neuropathies among patients with HIV infection

Peripheral neuropathies often affect patients with HIV disease,[1] and various peripheral neuropathies have been described at different stages of HIV infection, from seroconversion to AIDS. Acute inflammatory demyelinating polyneuropathies have been found in association with primary HIV infection; chronic inflammatory demyelinating polyneuropathies, isolated mononeuritis, and multiplex mononeuritis have more commonly been described in those in advanced stages of HIV disease, and distal symmetric polyneuropathy has been found to be the most common neuropathy among persons with AIDS. In most cases, the aetiopathogenesis of these neuropathies is still unclear, although various factors have been implicated (infectious, metabolic, immunological, inflammatory, nutritional, and toxic).[2] To date, relatively few studies have estimated the incidence of peripheral neuropathies among HIV infected patients belonging to different population groups. Data derived from the Multicenter AIDS Cohort Study showed annual rates slightly higher than 1.5/100 person-years for sensory neuropathy.[3] Another study found a roughly 1% annual incidence of neuropathies in symptomatic patients without AIDS.[4] To estimate the incidence of peripheral neuropathies during the different stages of HIV disease, we analysed data from a cohort study of patients with known dates of seroconversion (a documented HIV seronegative test followed by a confirmed positive test within two years). The seroconversion date was estimated as the midpoint in time between the last negative and the first positive HIV test. After seroconversion, patients were followed up to obtain clinical information and data on laboratory indices about every six months. The study design and methodology have been described in detail elsewhere.[5] The present analysis included four clinical centres, which performed a detailed neurological examination of the participants of the study. These centres were contacted to provide complete information on peripheral neuropathies by completing a standardised data collection form for each episode, providing detailed information on diagnostic criteria (neurological signs and symptoms, instrumental diagnosis), AIDS prophylaxis, and antiretroviral drug therapy. The study population consisted of 621 HIV seroconverters. The median age of the participants was 27 (range 14–66) years, 29 for males and 24 for females. Of these patients; 267 (42.9%) were intravenous drug users, 217 (34.9%) were homosexual men, and 127 (20.5%) were heterosexual contacts; 10 (1.6%) patients had unknown or undetermined risk factors.During a median follow up time of 5.7 years, 19 (3.1%) patients developed symptoms suggestive of peripheral neuropathies. The estimated incidence of peripheral neuropathies was 5.5/1000 person-years of follow up. The median age at the time of diagnosis was 35.2 years (range 24.8–53.6 years). Thirteen events (68.4%) occurred among males and six (31.6%) among females. For the exposure category, peripheral neuropathy was found among seven (2.6%) intravenous drug users, five (2.3%) homosexual men, and seven (5.51%) heterosexual contacts. The incidence rate of peripheral neuropathies was 4.4/1000 person-years in intravenous drug users, 4.3/1000 person-years in homosexual men, and 11.4/1000 person-years in heterosexual contacts. Acute infection was observed in 43 of 621 (7%) patients. One episode of peripheral neuropathy occurred during acute infection (the incidence rate of peripheral neuropathies was 42.4/100 person-years), six episodes during the asymptomatic phase (0.2/100 person-years), eight in the pre-AIDS symptomatic phase (2.4/100 person-years), and four episodes in patients who had already presented with diseases indicative of AIDS (3.8/100 person-years). The CD4 cell count at diagnosis of peripheral neuropathies was 266.2 for the patients with acute infection; the median was 324.6 (range 149.7–655.2) for those in the asymptomatic phase, 216.5 (range 14.9–1440.0) among those with pre-AIDS symptoms, and 38.0 (range 7.2–124.8) among persons with AIDS. Of the 19 patients with peripheral neuropathies, 15 (78.9%) had distal symmetric polyneuropathy, two (10.5%) had multiplex mononeuritis, and two (10.5%) acute inflammatory demyelinating polyneuropathies. The median CD4 cell count at diagnosis of peripheral neuropathy was 31.2 (range 16.3–47.0) for persons with multiplex mononeuritis, 184.1 (range 7.5–655.4) for those with distal symmetric polyneuropathy, and 304.3 (range 342.1–266.3) for those with multiplex mononeuritis All 19 patients had sensory neuropathy, and 14 (73.7%) also had motor impairment. Of these 19 patients, 13 (68.4%) underwent electrophysiological investigations, showing abnormalities of nerve conduction. Another patient presenting with a mononucleosis-like syndrome and meningoradiculoneuritis had a raised number of cells (184/μl) in the CSF. The CSF of the other participants was normal. No participant reported the use of known neurotoxic drugs such as DDC; 10 patients had received AZT and two had received DDI (both patients who were treated with DDI had discontinued treatment six months before the onset of peripheral neuropathies; one had multiplex mononeuritis and one distal sym-

metric polyneuropathy). Only one participant reported alcohol misuse. In conclusion, the incidence of peripheral neuropathies seems to be high among patients with acute HIV disease, although in our study the number of patients with acute disease was not very high. In the asymptomatic phase, the incidence of peripheral neuropathies was low, with a tendency to increase in the advanced stages of HIV infection. Our findings are in accord with the results of other studies[4] which showed that the development and progression of neuropathy was correlated with the progression of HIV disease. The issue of peripheral nerve disorders in HIV infection should not be neglected, as the symptoms of peripheral neuropathy may be extremely painful and debilitating and may complicate the management of HIV positive patients, negatively influencing the quality of their life.

This work was supported by the Ministero della Sanità-Istituto Superiore di Sanità, Project grant 940R, 1996.

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Focal vertebral artery dissection causing Brown-Séquard's syndrome

Vertebral artery dissection typically causes an ischaemic brain stem stroke. We report a man who presented with left sided neck pain and Brown-Séquard's syndrome localised to C1, and in whom a focal C1 ischaemic cord lesion secondary to a localised vertebral artery dissection was found using MRI.

A 47 year old machine operator was admitted as an emergency with neck pain, headache, and gait disturbance. Three weeks before admission he developed minor stiffness of his upper cervical region while decorating his home, which was relieved by ibuprofen. There was no history of neck manipulation or trauma. Two days before admission he awoke with a severe left sided pounding headache and neck ache with an associated cold sensation on the left side of his neck. He was nauseated but did not vomit. His symptoms again resolved within an hour with ibuprofen, but two hours later there followed a tingling sensation in his left index finger, thumb, and forearm spreading up to his elbow. He slept for a few hours but awoke with more generalised paraesthesiae in the left arm which spread over the next 36 hours to involve the left trunk and leg. He noticed minor gait disturbance as well as left eye pain described as "like an ice cream headache". The remainder of the history was non-contributory.

Apart from xanthelasma the general examination was entirely normal. His blood pressure was 120/80. There was minor limitation of movement of his cervical spine. Cranial nerve examination was normal. Slight spasticity was evident in the left arm and leg. Both plantars were extensor. There was decreased sensation in the distribution of C2 on the left and decreased sensation to pinprick and temperature on the right and to vibration and joint position sense on the left below this. Thus the examination was consistent with Brown-Séquard's syndrome on the left at the level of C1–2.

Biochemical, haematological, and immunological investigations were normal. Fasting cholesterol was 8 mmol/l, triglycerides 3.80 mmol/l. ECG was normal. CSF protein was 0.63 g/l, glucose 3.1 mmol/l (blood 4.3 mmol/l). There was no xanthochromia. There were three white cells and 57 red cells and no oligoclonal bands. Visual and auditory evoked potentials were normal but there were abnormal somatosensory evoked potentials from the left upper limb. A sagittal T2 weighted MRI showed an area of high signal, consistent with an ischaemic lesion, in the posterior aspect of the cervical cord at the level of C1 (figure). This was shown to involve the left posterior column and left corticospinal tract on corresponding axial cuts. T1 weighted spin echo images through the C1 region showed the typical features of an arterial dissection[1] with an eccentric rim of high signal adjacent to a residual flow void in the left vertebral artery. This periarterial collar was distinguished from adjacent fat tissue by a fat suppression sequence. He was anticoagulated with heparin and then warfarin and started on lipid lowering agents. Three months later his original symptoms and signs had resolved completely.

Vertebral artery dissection has generally been associated with head and neck pain and brain stem stroke. There have, however, been some case reports of vertebral dissection with symptoms initially suggesting cervical involvement. Giroud *et al*[2] described a woman who had intermittent pain in her right arm for five days before presenting with sudden onset cerebellar dysarthria and diplopia. A few cases seem to remain localised to the cervical cord. Dubard *et al*[3] described a cervical myelopathy secondary to a vertebral

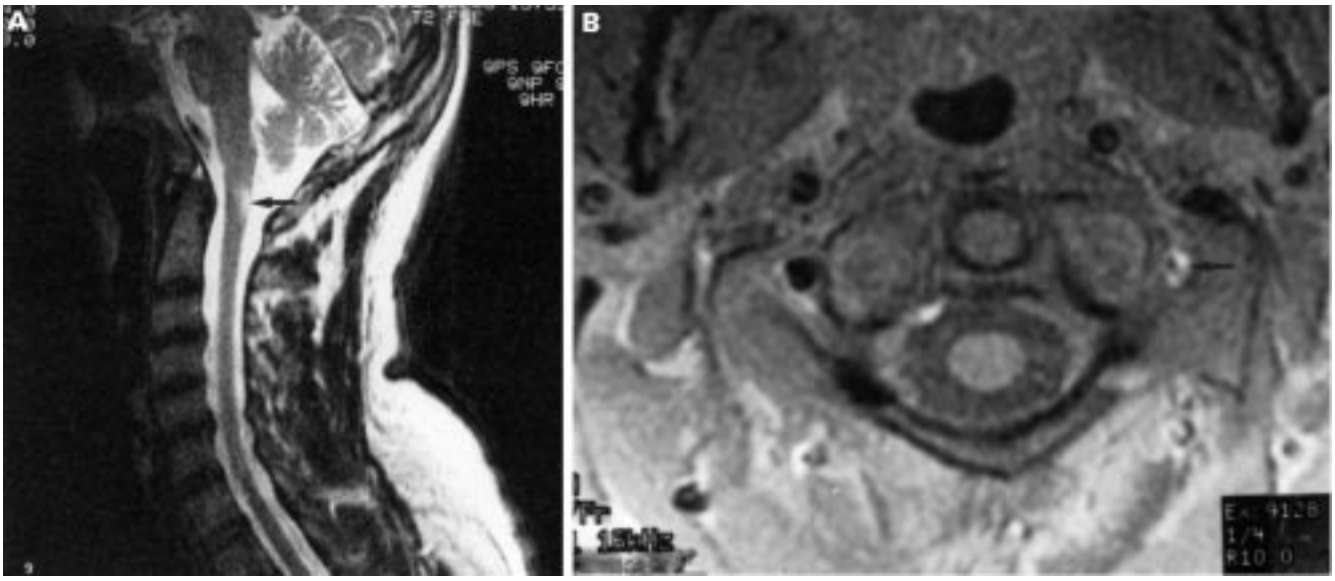

Figure (A) Sagittal T2 weighted fast spin echo sequence (TR/TE 5000/130 ms) through the cervical cord showing an area of high signal (arrow) in the posterior aspect of the cord at C1 level not associated with cord swelling. (B) Axial fat saturated T1 weighted spin echo (TR/TE 480/14 ms) through C1 shows a crescentic high signal rim (arrow) lateral to the narrowed residual lumen of the left vertebral artery as it runs though the C1 transverse foramen.