rarely during primary HIV illness but they may be underreported; we were unable to identify other reports of transient neurological deficit in this setting. In patients with advanced HIV disease, transient neurological deficit, and cerebrovascular events including cerebral infarction may be associated with non-bacterial endocarditis, CNS infections (including *Cryptococcus* and *Toxoplasma*), and CNS tumours, but they may also occur in the absence of an identifiable underlying cause.¹⁻³ In these patients the mechanism for transient neurological deficit and cerebrovascular events in HIV disease is unknown; anticardiolipin antibodies, which may predispose to cerebrovascular disorders,⁵ were detected in 70% of patients with HIV-related transient neurological deficits in a controlled study.² In our patient, the cause of the transient neurological deficit is unknown; the possibilities include localised ischaemia caused by vascular obstruction or spasm, or a focal inflammatory lesion developing at the time of seroconversion. The preponderance of transient neurological deficit in late stage HIV disease and its occurrence during primary infection in our patient suggest a possible association with high HIV viral load. Primary HIV infection should be considered in at risk patients presenting with unexplained focal neurological symptoms.

T CARD
C G WATHEN
Department of General Medicine

G A LUZZI
Department of Genitourinary/HIV Medicine, South
Buckinghamshire NHS Trust, Wycombe Hospital,
High Wycombe, HP11 2TT, UK

Correspondence to: Dr G A Luzzi, Department of
Genitourinary/HIV Medicine, Wycombe Hospital,
High Wycombe HP11 2TT, UK.

- Engstrom JW, Lowenstein DH, Bredesen DE. Cerebral infarctions and transient neurological deficits associated with acquired immunodeficiency syndrome. *Am J Med* 1989;86:528-32.
- Brew BJ, Miller J. Human immunodeficiency virus type 1-related transient neurological deficits. *Am J Med* 1996;101:257-61.
- Pinto AN. AIDS and cerebrovascular disease. *Stroke* 1996;27:538-43.
- Naranjo IC, Santos JAT, Rodriguez MAA. Ischemic stroke as the sole manifestation of human immunodeficiency virus infection. *Stroke* 1992;23:117-8.
- Gallo P, Sivieri S, Ferrarini AM, et al. Cerebrovascular and neurological disorders associated with antiphospholipid antibodies in CSF and serum. *J Neurol Sci* 1994;122:97-101.

The pallidoreticular pattern of brain damage on MRI in a patient with carbon monoxide poisoning

Neuropathological changes of carbon monoxide (CO) poisoning include the pallidoreticular pattern of brain damage¹ consisting of bilateral necrosis of the globus pallidus and substantia nigra. We here report brain MRI of a CO poisoning victim that showed bilateral lesions of the globus pallidus and substantia nigra. This is the first neuroimaging demonstration of the pallidoreticular pattern of brain damage in a patient with CO poisoning.

A 17 year old previously healthy man was found unconscious in a bathroom. He was transferred to an emergency room and diagnosed as having CO poisoning due to faulty ventilation. Hyperbaric oxygen therapy improved his consciousness from coma to being fully alert. However, he had parkinsonism. Severe akinesia was noted although there was no tremor or rigidity of the limbs. Additional hyperbaric oxygen therapy for six months did not improve his neurological deficit. Brain MRI, a year after CO exposure, disclosed bilateral pallidal lesions: the globus pallidus appeared streaked with a decreased signal intensity on T1 weighted images and an increased signal intensity on T2 weighted images (figure A). Also noted was an increased signal intensity of the substantia nigra on T2 weighted images (figure B). There was otherwise no alteration on brain MRI. A CSF examination showed a decrease in homovanillic acid concentration to 5.9 ng/ml (normal range 28.83-54.28 ng/ml) with normal values of CSF protein and sugar concentrations and normal cell counts.

Auer and Benveniste¹ described the pallidoreticular pattern of brain damage in necropsied cases of CO poisoning. They speculated that the iron rich regions could be affected by binding of CO to iron molecules. In our patient, bilateral pallidal lesions on MRI were consistent with necrosis because of MR signal features. A decreased concentration of homovanillic acid in the CSF² and an increased signal intensity of the substantia nigra on T2 weighted images seem to be due to neuronal loss and gliosis in the substantia nigra.

The pallidoreticular pattern of brain damage¹ to our knowledge has not been reported on MRI in patients with CO poisoning. We emphasise the possible involvement of the substantia nigra in some

victims of CO poisoning. In those patients, parkinsonism would be explained partly by neuronal loss in the substantia nigra in addition to the bilateral pallidal necrosis.³

TORU KAWANAMI
Division of Internal Medicine, Yamagata Prefectural
Shinjo Hospital, Yamagata, Japan

TAKEO KATO
KEIJI KURITA
HIDEO SASAKI
Third Department of Internal Medicine, Yamagata
University School of Medicine, Yamagata, Japan

Correspondence to: Dr T Kato, Third Department of Internal Medicine, Yamagata University School of Medicine, 2-2-2 Iida-Nishi, Yamagata 990-23, Japan. Telephone 0081 236 28 5316; fax 0081 236 28 5318.

- Auer RN, Benveniste H. Carbon monoxide poisoning. In: Graham DI, Lantos PL, eds. *Greenfield's neuropathology*, 6th ed. London: Arnold, 1996;1:275-6.
- Gibson CJ, Logue M, Growdon JH. CSF monoamine metabolite levels in Alzheimer's and Parkinson's disease. *Arch Neurol* 1985;42:489-92.
- Bucher SF, Seelos KC, Dodel RC, et al. Pallidal lesions. Structural and functional magnetic resonance imaging. *Arch Neurol* 1996;53:682-6.

Information searching for multiple sclerosis

The acquisition of information is an important part of scientific work. In recent years, available information sources have multiplied exponentially, and as a result, we have come to find ourselves immersed in an "information overload". Although we feel committed to keeping up to date with the advances in neurology by using our information seeking skills, paradoxically the new, widely expanded information technologies have made it more and more difficult to gain knowledge about relevant literature sources and to retrieve information. Under the pressure of this situation, many of us have resorted to the passiveness of searching only the most commonly used databases such as Medline and Current Contents to acquire the information we need.

In the frequent practice of retrieving information needed for patient assistance, it is accepted that the use of Medline gives good results for European and North American countries. However, in searches directed towards investigation, we suspected that other, perhaps less accessible databases, could provide valuable additional information. To determine to what extent this might be true, we performed a literature search in the field of multiple sclerosis.

The analysis began by connecting to the 179 Science and Technology databases included in the host Dialog (Knight-Ridder Inc), using the commands for multiple database searching. To avoid classification problems due to variations in the internal structure of the databases, we requested that the search term "multiple sclerosis" appear in any part of the document. As the databases cover different periods and have different indexing policies, we restricted the search to the complete year of 1995 and, at the beginning, to the title field. However, we realised that using only the title would lead to poor results (for example, 613 titles in Biosis instead of the 1100 we used later), so we used the entire reference fields instead. Finally, we excluded databases devoted to medical Industries and ones that are contained in more general information sources (for exam-

(A) Axial T2 weighted MRI at the level of the basal ganglia. The globus pallidus shows an increased signal intensity. (B) Axial T2 weighted MRI of the midbrain. The substantia nigra shows an abnormally high signal intensity.

ple, the New England Journal Full Text Database). The first search yielded 24 usable databases with a total of 5350 references.

It was found that most references retrieved (4615) were concentrated within the following databases: Current Contents (1239), Biosis (1100), Embase (892), Medline (739), Pascal (406), and IAC Health and Wellness (378). The titles of the articles recovered were extracted and two main points were developed: duplication of references among the databases and the suitability of the databases for each information requirement. To determine suitability, the documents were classified according to their titles into four groups, representing the major headings in *McAlpine's multiple sclerosis handbook*¹: (1) epidemiology, (2) clinical aspects (signs and symptoms, course, and prognosis, natural history, neuropsychology, diagnosis, laboratory diagnosis, and therapy), (3) pathogenesis (including genetics, immunology, and animal models), and (4) pathology. After carrying out the classification, we found that an important subset of documents did not correspond to any of the groups; these were then categorised into: (5) health promotion (including quality of life and social aspects), (6) general aspects (particularly review articles dealing with several of the former topics), and, (7) noise (documents with no apparent relation with multiple sclerosis). All the documents retrieved were classified by members of the multiple sclerosis unit (clinicians and basic researchers) after training sessions to establish the classification criteria, and the final review was performed by a neurologist expert in multiple sclerosis.

A high percentage of duplication of references was found in five of the databases studied (average overlap 22.4%). Health and Wellness showed no overlapping and, moreover, indexed most of the articles on Social Aspects. Considering only Medline, Embase, and Current Contents, average overlap was 26.6%.

The table shows the number of references retrieved and the percentage of references in relation to the total for each aspect of multiple sclerosis on each database.

For our purposes, quantity was more valuable than percentage, as the larger the number of references obtained the more the information that was gathered.

Thus according to the categories established, we found that the most suitable database for epidemiology, clinical aspects, and pathogenesis was Biosis, whereas the most appropriate for health promotion was Health and Wellness; pathology was best covered by Current Contents, and general aspects by Embase. The highest rate of noise was found in Current Contents.

These results evidence that use of Medline alone for multiple sclerosis information searches will not provide optimum returns and can have economic implications resulting from duplication of scientific efforts.

However, Medline is the best known and most available database around the world and it is unusual to find other information sources integrated within institutional information systems.

A way out of this problem is the use of On-line Search Services, a common element of library services. They can provide access to a wide range of databases and are managed by specialised information professionals.

M JORDÀ

Unidad de Información, Agencia de Investigación y Docencia, Spain

C NOS

I DURÁN

J RÍO

M TINTORÉ

X MONTALBÁN

Unitat de Neuroimmunologia Clínica-Esclerosi Múltiple, Hospitals Vall d'Hebron, Barcelona, Spain

Dr C Nos, Unitat de Neuroimmunologia Clínica-Esclerosi Múltiple, Escola d'Infermeria 5ª P, Hospital General Universitari Vall d'Hebron, Pg Vall d'Hebron 119-129, 08035 Barcelona, Spain. Telephone 0034 3 274 62 02; fax 0034 3 274 60 84; email: xmontal@ar.vhebron.es

1 Matthews WB, ed. *McAlpine's multiple sclerosis*, 2nd ed. New York: Churchill Livingstone, 1991.

Lack of association between hepatitis G virus and multiple sclerosis

The aetiology of multiple sclerosis is still not fully understood. Infectious agents have been postulated as causes of the disease for over a century. A theory proposes that an exogenous stimulus initiates an immune response against endogenous CNS proteins. Supporting this hypothesis, some epidemiological studies strongly implicate an environmental factor in the development of multiple sclerosis.¹ Several common human viruses have been implicated in the pathogenesis of multiple sclerosis. However, despite data obtained from epidemiological, serological, and animal studies, no virus has been consistently isolated, or viral material uniquely identified, from patients with multiple sclerosis.² Hepatitis G virus (HGV), a novel potentially hepatotropic flavivirus, has recently been identified but little is known about the relation of this virus to chronic viral hepatitis and other chronic diseases.³⁻⁴

To investigate the relation between multiple sclerosis and HGV, we have studied the presence of HGV RNA, a marker of ongoing infection, and anti-E2 HGV antibodies, a marker of exposure and recovery of infection, in serum of patients with multiple sclerosis.⁵

We tested serum from 99 consecutive patients (68 females, mean age 35.2 (SD 11.9) years) with definite multiple sclerosis seen at our hospital. Fifty five patients had a relapsing-remitting, 17 a secondary progres-

Number of patients and controls positive for HGV RNA and anti-E2 antibodies

Group	HGV RNA (%)	anti-E2 (%)
RRMS (n=55)	0	8
SPMS (n=17)	0	2
PPMS (n=27)	2	8
Total MS (n=99)	2(2)	18(18)
Blood donors (n=1000)	19(1.9)	140(14)

RRMS=relapsing-remitting multiple sclerosis; SPMS=secondary progressive multiple sclerosis; PPMS=primary progressive multiple sclerosis.

sive, and 27 a primary progressive disease. As controls, we included 1000 consecutive blood donors who had tested negative for HCV, HBV, and HIV markers. HGV RNA was determined by reverse transcription/polymerase chain reaction with specific primers of the 5' and NS5 regions (Boehringer Mannheim) and anti-E2 antibodies were detected from 10 µl serum by µPLATE anti-HGenv (Boehringer Mannheim).

Results in patients with multiple sclerosis did not differ significantly from those in healthy blood donors (table). Two patients with multiple sclerosis had ongoing HGV infection, normal liver tests, and were negative for anti-E2 antibodies. None of the patients with HGV exposure (RNA or anti-E2 positive) had received blood transfusions and were not intravenous drug users or healthcare workers. No differences in age, sex, duration of disease, and clinical forms were found among patients with multiple sclerosis. Although the only two patients positive for HGV RNA were primary progressive patients, this finding must be interpreted with caution.

In conclusion, the prevalence of HGV infection is not higher in our population of patients with multiple sclerosis than in our controls. Our results do not therefore support any causative role for HGV in the pathogenesis of multiple sclerosis.

S SAULEDA

Liver Unit, Department of Medicine

J RÍO

X MONTALBÁN

E MARTINEZ-CACERES

Unit of Clinical Neuroimmunology, Department of Neurology

JJ ESTEBÁN

J GUARDIA

Liver Unit, Department of Medicine, Hospital General, Universitari Vall d'Hebron, Barcelona, Spain

Correspondence to: Dr J Río, Unitat de Neuroimmunologia Clínica, Escola d'Infermeria 5ª planta, Hospital Universitari Vall d'Hebron, Psg. Vall d'Hebron 119-129, 08035 Barcelona, Spain. Fax 0034 3 4274700; email: jrio@ar.vhebron.es

Number (%) of references for each group and database

	Biosis	Embase	Pascal	Health and Wellness	Medline	Current Contents	Total
Epidemiology	52 (29.2)	31 (17.4)	19 (10.6)	4 (2.2)	36 (20.2)	36 (20.2)	178
Clinical aspects	399 (22.9)	372 (21.3)	192 (11)	116 (6.6)	321 (18.4)	341 (19.5)	1741
Pathogenesis	509 (31.1)	319 (19.5)	116 (7)	26 (1.5)	265 (16.2)	400 (24.4)	1635
Pathology	35 (23.8)	26 (17.6)	17 (11.5)	2 (1.3)	31 (2.1)	36 (24.4)	147
Health promotion	20 (11.2)	24 (13.4)	11 (6.1)	82 (46)	21 (11.7)	20 (1.2)	178
General aspects	16 (5.8)	23 (22.7)	8 (7.9)	19 (18.8)	15 (14.8)	20 (19.8)	101
Noise	92 (14.4)	126 (19.8)	49 (7.7)	104 (16.3)	74 (11.6)	190 (29.9)	635
Total	1123	921	412	353	763	1043	4615

1 Kurtzke JF. Epidemiologic contributions to multiple sclerosis: an overview. *Neurology* 1980;30:61-79.

2 Cook SD, Rohowsky-Kochan, Bansil S, et al. Evidence for a viral etiology of multiple sclerosis. In: Cook SD, ed. *Handbook of multiple sclerosis*. New York: Marcel Dekker, 1996:97-118.

3 Linnen J, Wages J, Zhang-Keck Z, et al. Molecular cloning and disease association of hepatitis G virus: a transfusion-transmissible agent. *Science* 1996;271:505-8.

4 Tacke M, Kiyosawa K, Stark K, et al. Detection of antibodies to a putative hepatitis G envelope protein. *Lancet* 1997;349:318-20.

5 Schlueter V, Schmolke S, Stark K, et al. Reverse transcription-PCR detection of hepatitis G virus. *J Clin Microbiol* 1996;34:2660-4.