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MUHC–MNH

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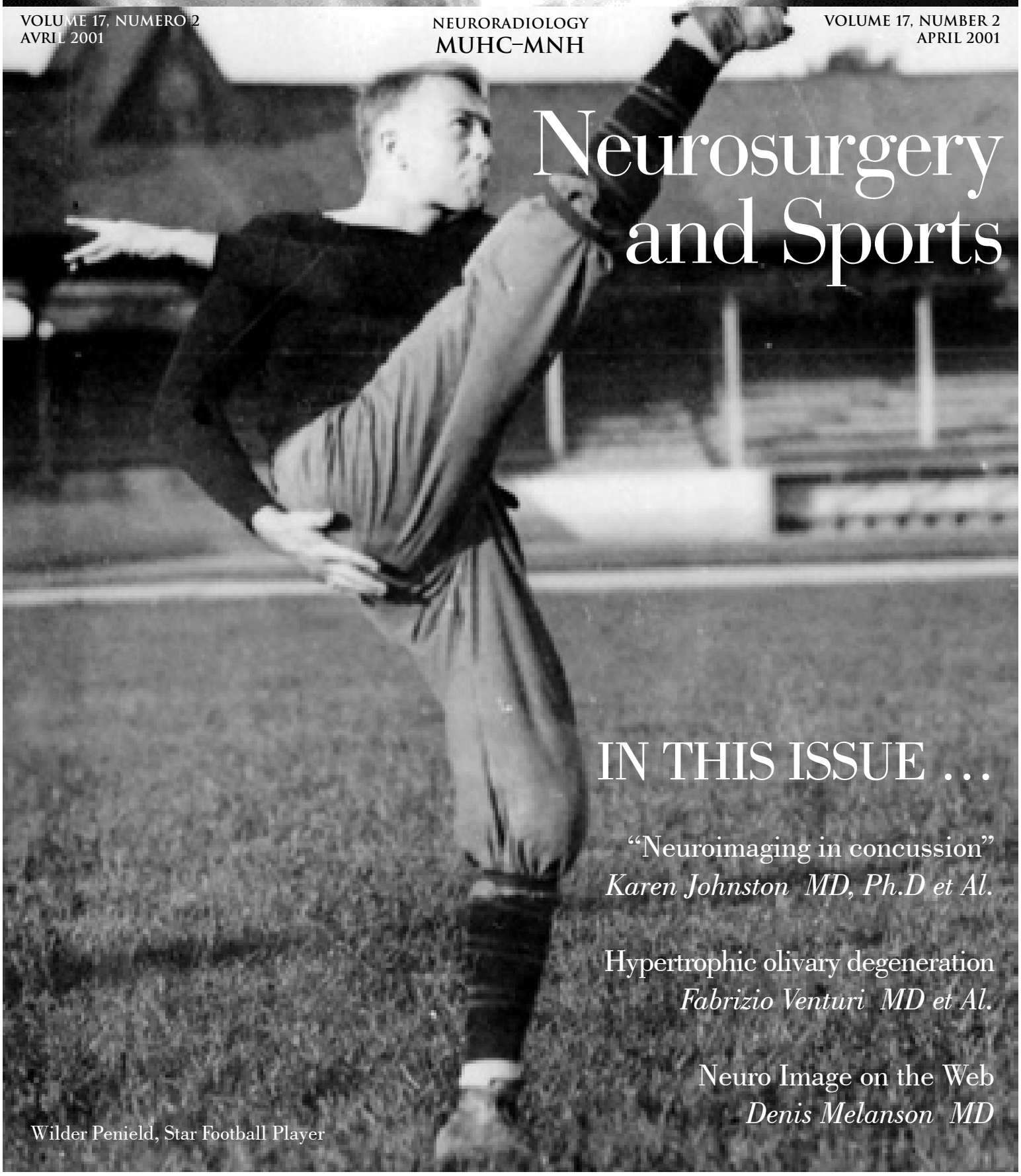

Neurosurgery and Sports

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An Announcement

This is the 5th issue of the revived Neuro Image bulletin. Most of you on the MNI mailing list have received the previous bulletins. But some addresses were incorrect and invoices have been returned. Even with our modern ways of finding people, many of the sendees have not been localised. Please let me know about any of the former MNI fellows you are aware who have not received the bulletin.

This 5th issue will be the last to be printed on a large scale. From now on, Neuro Image will appear on the World Wide Web, linked to the MNI Web page. Those of you who would like to receive the printed copy, please let me know by e-mail denis.melanson@muhc.mcgill.ca or by fax (514)•398•2829.

You will understand that color printing is expensive, as is mailing, and my resources are less than they were. My thanks again to the NeuroPhotography team for their dedicated contribution.

I have received many favorable comments on the last issue. I am very pleased and thank all of those who have written or emailed their comments. However, the answers to the quiz case about the pituitary lesion have not been numerous. There is still time to do it.

The present issue publishes a summary of a paper on " Brain concussion ". Those of you interested in further information can obtain it by writing to Dr. Karen Johnston, at the address appearing in the insert.

Best regards Saluti affettuosi
Respetos Salutations amicales Afectuosamente
Saudações Herzliche gruesse
O Genki De Namaste Cordialmente

Denis Melanson
Neuroradiology
McGill University Health Center
MUHC–MNH

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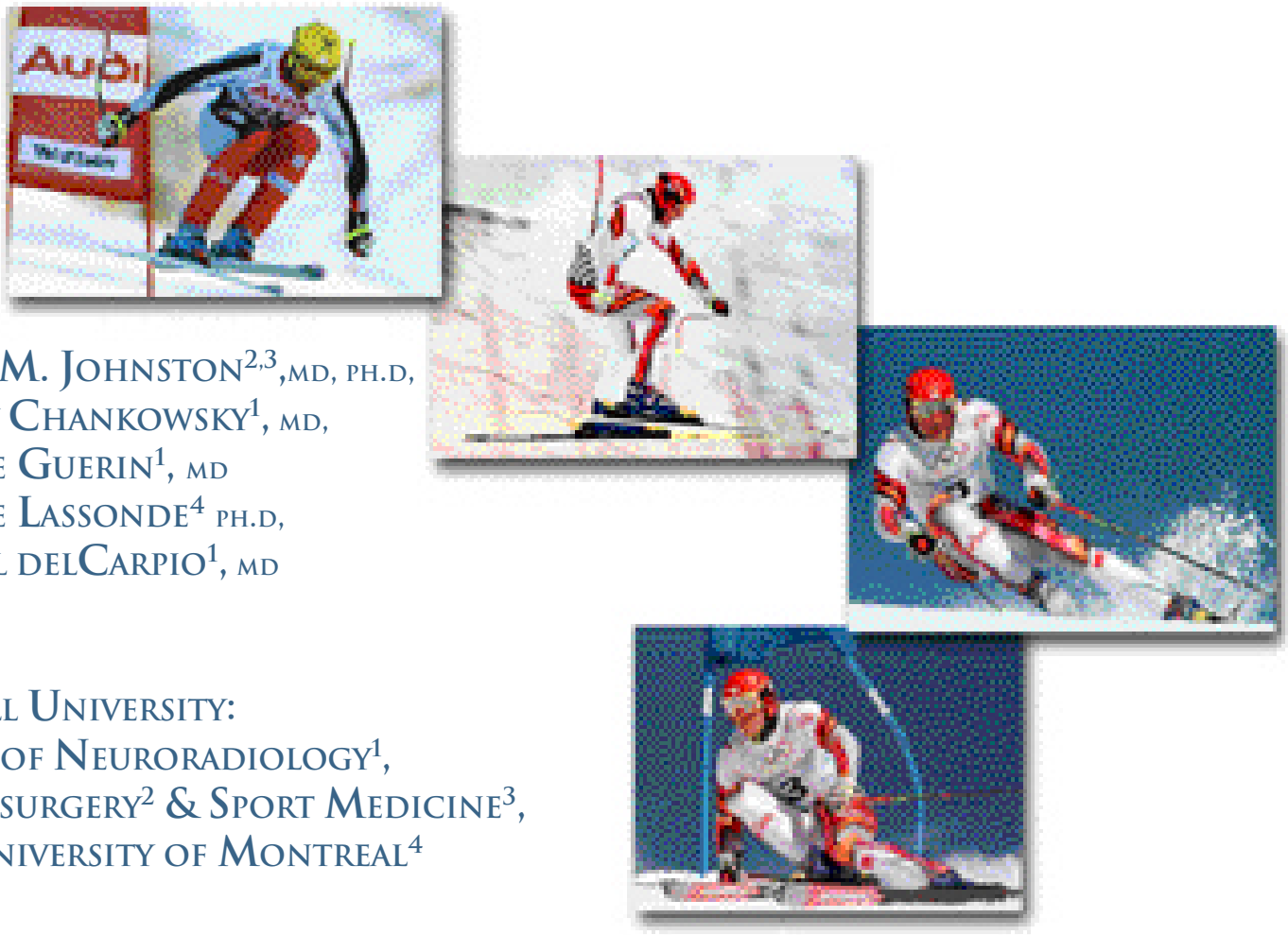
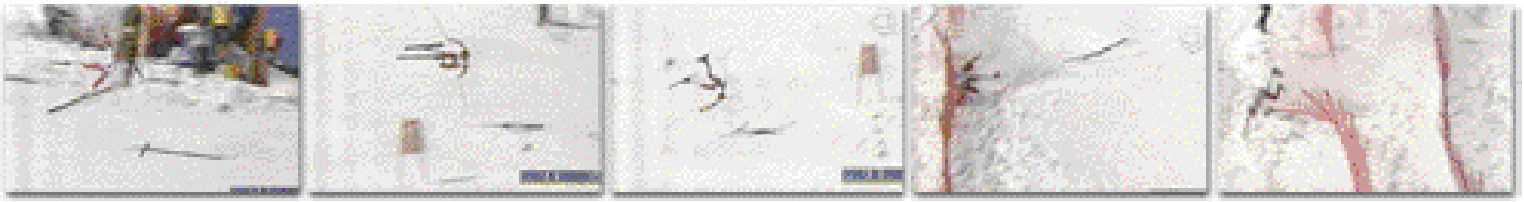
NEUROIMAGING IN CONCUSSION



Karen M. Johnston[2,3], MD, PH.D,
Jeffrey Chankowsky[1], MD,
Maryse Guerin[1], MD
Maryse Lassonde[4] PH.D,
Raquel delCarpio[1], MD

McGill University:
Depts. of Neuroradiology[1],
Neurosurgery[2] & Sport Medicine[3],
and University of Montreal[4]

"One cannot achieve any victory without a few bumps ..."



Austrian Hermann Maier's spectacular crash at the 1998 Winter Olympics in Nagano, Japan; but soon after, he bounced back by winning two gold medals in Giant Slalom and Super Giant Slalom during the same games.

ABSTRACT

Objective:
To evaluate the use of neuroimaging techniques in concussive injury in sport.

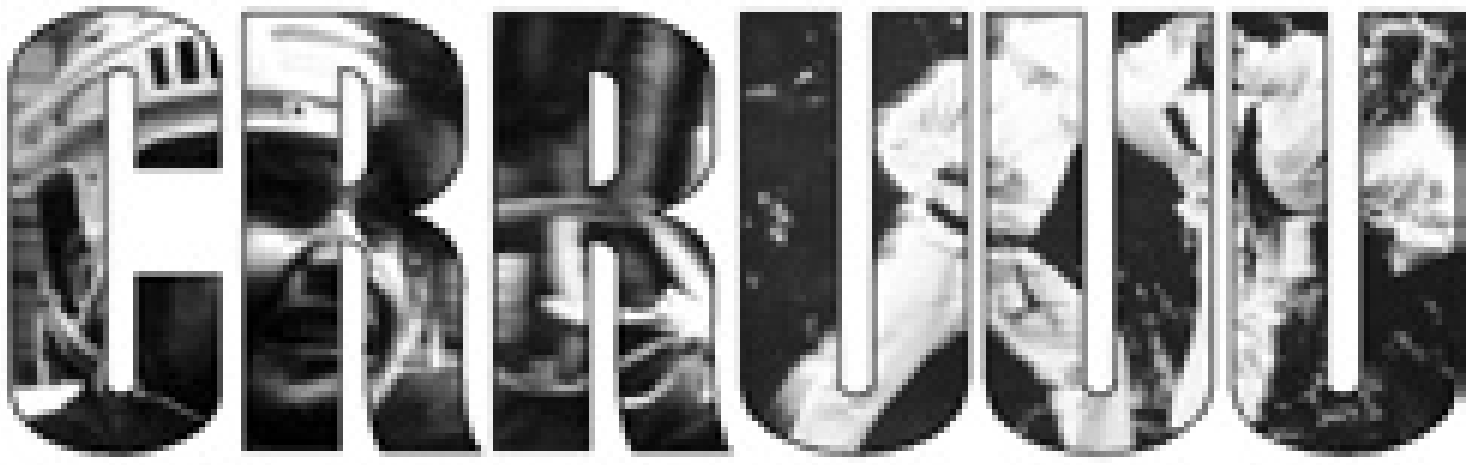
Methods:
15 subjects underwent brain CT, conventional MRI, FLAIR and Diffusion weighted MRI imaging (DWI) after sustaining a concussion during sport activity. Concussions were graded using the severity scale of the American Academy of Neurology (AAN).

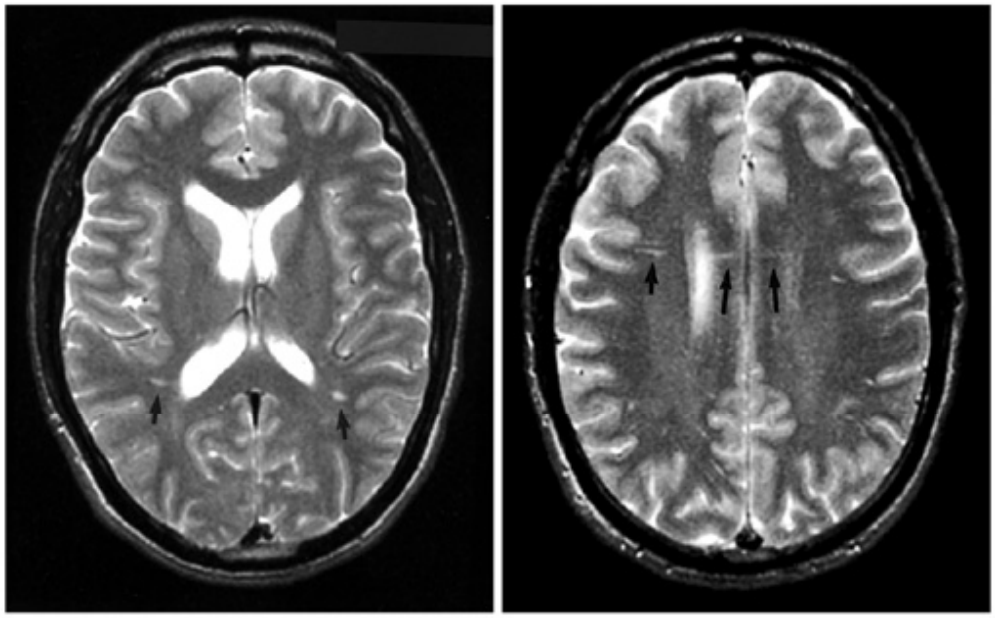
Results:
33% of subjects studied had abnormalities on conventional MR or DWI studies. Findings were seen in low grade injuries and were not associated with initial LOC.

Conclusions:
These findings are among the first objective evaluations described in low grade concussive injury. The addition of DWI to the investigation protocol may increase yield for otherwise occult lesions not detected on routine spin-echo MR images nor CT.

Until now, the neuroimaging of most low grade concussions has revealed few abnormalities. CT has offered little in the way of positive findings and is therefore of limited use in concussion investigation and management issues. In general, this has led to problems objectively evaluating the degree of injury, assessing long term consequences, understanding the anatomy and pathophysiology of the injury and scientifically formulating return to play decisions. The findings described in this paper are among the first descriptions of positive neuroimaging in low grade concussion and identify a potentially important investigative tool. The addition of DWI to concussion evaluation may provide new and valid information to our protocol.

MRI is well known to be superior to CT in morphological assessment of brain lesions. Even so, until now, CT and MRI studies performed on subjects with post concussion syndrome were reported positive in only 9% of cases [25]. Our study identified positive findings

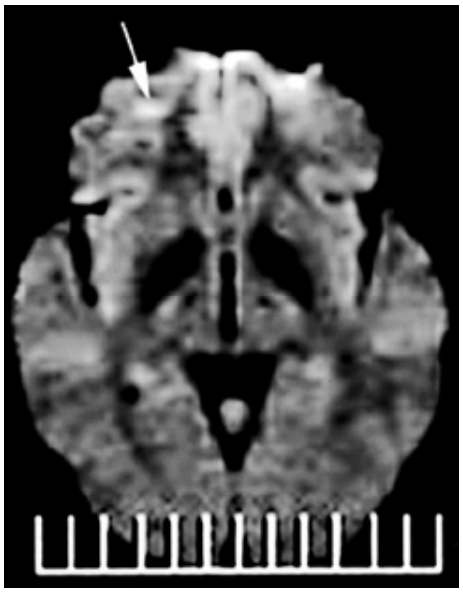




Axial-T2 image of the brain showing abnormal hyperintense foci in the sub-cortical white matter of the frontal and posterior parietal lobes.

by standard MR imaging in as many as 20% of subjects. The addition of diffusion weighted MR sequences to our protocol increased the number of positives to 33% (5/15).

Future questions we wish to pursue are the following; Do these imaging findings predict clinical course? Will the signal changes resolve with resolution of symptoms? What is the exact nature of the lesions detected on delayed DWI but not spin-echo T2? Do these findings correlate with other investigative findings such as baseline and full neuropsychology battery? Is there a correlation with neuropsychological and neurophysiological testing? (Dupuis et al. Neuroreport, 2000, Leclerc 2001 in prep). Do imaging findings reflect repeated or cumulative injury? Our future prospective studies will address these important issues in our attempt to improve the diagnosis and management of sport related head injury.



Diffusion-MR image showing a small subcortical right frontal hyperintense focus. Note that the lesion is distinct from frontal artefact seen adjacent to bone.

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Subject	Sex	Age	Sport	AAN	MRI	MRI Diffusion	Location
1	M	29	index case	3	+		Corpus callosum
2	M	42	trapeze	3			
3	M	29	hockey	2			
4	F	21	soccer	2		+	Left frontal; left partial
5	M	16	hockey	2			
6	M	21	hockey	2			Arachnoid cyst
7	F	36	soccer	2		+	right frontal
8	M	27	rugby	2			
9	M	30	diving	2	+		Corpus callosum (splenium), 1 parietal, right frontal
10	M	24	skiing	3			
11	M	22	hockey	2			
12	M	49	riding	3			
13	F	27	soccer	2			
14	M	15	hockey	2			
15	F	43	bicycling	2	+		right frontal operculum

Summary of study subjects' sport, injury grade and imaging findings.

Scenes from the ice hockey events at the 1998 Winter Olympics in Nagano, Japan

HYPERTROPHIC OLIVARY DEGENERATION (HOD): AN UNUSUAL WAY TO DEGENERATE

Drs. Fabrizio Venturi, Donatella Tampieri, Roland Brassard & Denis Melanson

In the CNS the degeneration of an anatomical structure is usually characterized by neuronal loss replaced by proliferation of glial elements. This change is reflected in the appearance on MRI: a loss of volume associated with a hypersignal on long TR sequences. There is, however, a case in which the degeneration is accompanied by hypertrophy: the degeneration of the inferior olivary nucleus, described for the first time in 1887 by Oppenheim from anatomic specimens. Not seen on CT scan due to artefacts caused by bony structures, the hypertrophic olivary degeneration (HOD) is more recently detected in vivo by MRI and is characterized by an hypersignal of the olive of the medulla oblongata on PD/T2 images with a variable enlargement of the olive itself.

HOD is considered a trans-synaptic degeneration because it occurs following loss of neuronal input to a cell, in this case the neurons of the inferior olivary nucleus. HOD occurs when a lesion, usually a haemorrhage, causes an interruption of the Guillain-Mollaret triangle (Fig1).

The Guillain-Mollaret triangle is a triangular circuit connecting the dentate nucleus of the cerebellum of one side with the red nucleus and the inferior olivary nucleus of the other side, via the superior cerebellar peduncle and the central tegmental tract (CTT) (Figs. 2, 3).

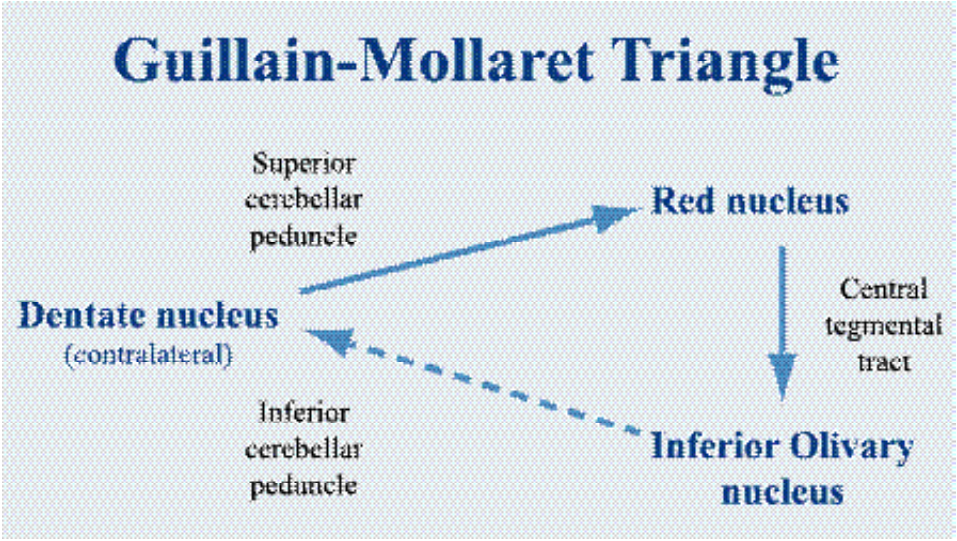


Fig 1

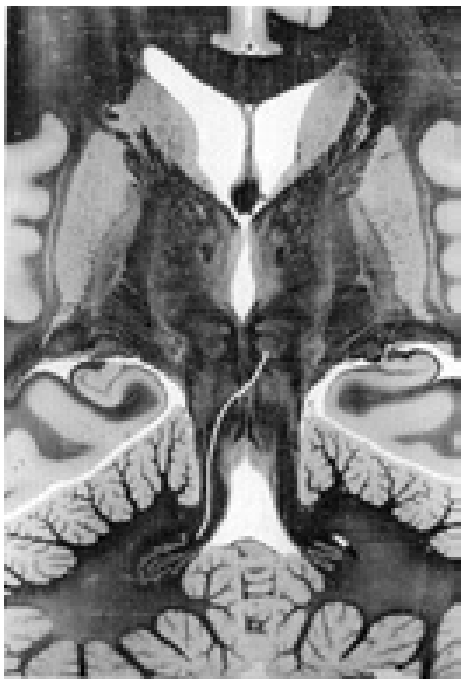


Fig 2
Anatomical oblique section along the superior cerebellar peduncles. The path of the cerebello-rubral tract from dentate nucleus to the contralateral red nucleus is shown.

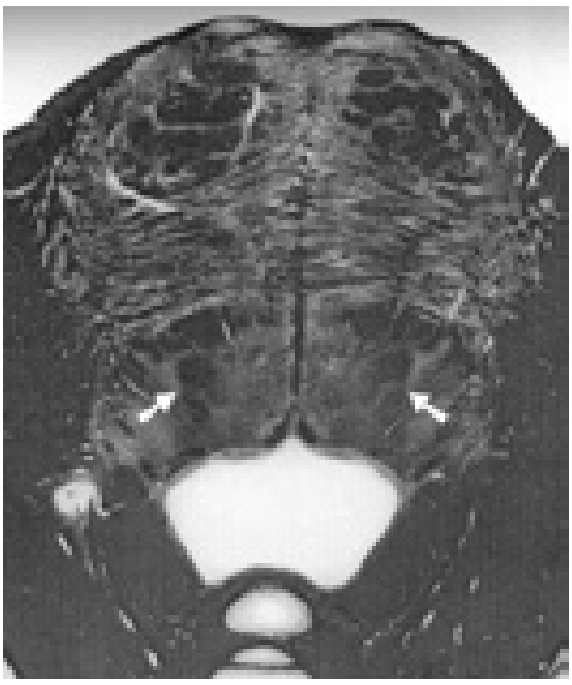


Fig 3
Anatomical, axial section of the brainstem, at the level of the pons, showing the location of the central tegmental tracts.

If the lesion is located in the tegmentum of the brainstem and involves the CTT, the degeneration occurs to the olive ipsilateral to the side of haemorrhage.

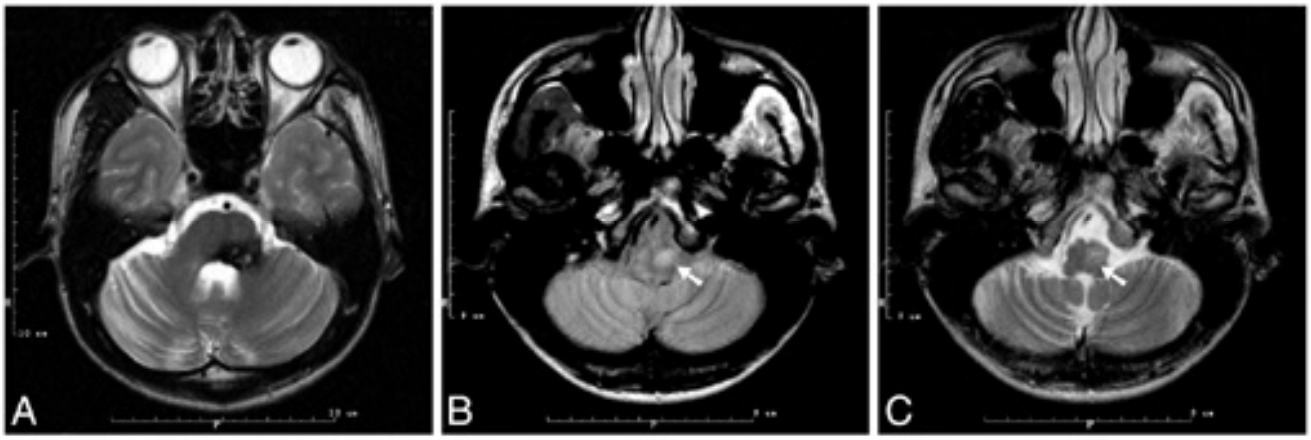


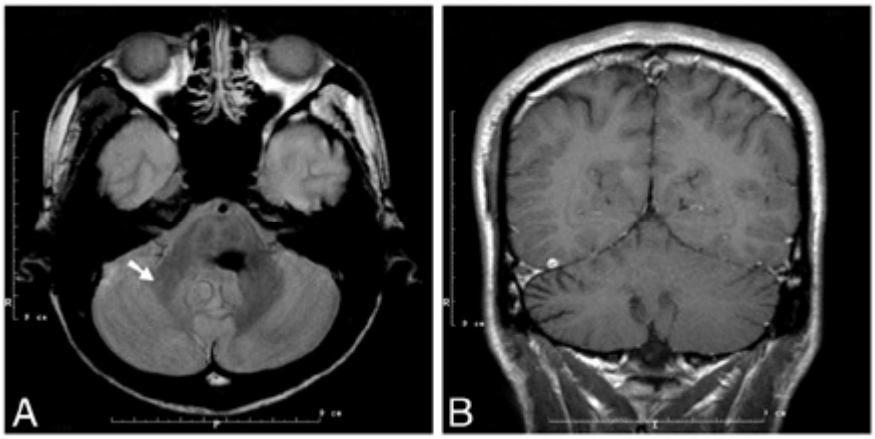
Fig4 *MR images obtained in a patient with brainstem cavernous haemangioma.* ***(a)*** *Axial T2-weighted image shows hyposignal suggesting hemosiderin deposits in the left tegmentum of the pons.* ***(b, c)*** *axial PD/T2-weighted images show hypersignal at the level of the left medullary olive which appears also enlarged in comparison to the right one. The signal change is more apparent on the PD-weighted image* ***(b)****.*

If the lesion is located in a cerebellar hemisphere and involves the dentate nucleus, the olivary degeneration will be contralateral due to the decussation of the dentato-rubral fibers. If the lesion is located in the brainstem and involves the superior cerebellar peduncles at the level of decussation, the degeneration will occur bilaterally.

The microscopic changes underlying HOD are characterized by hypertrophy of degenerated neurons followed by hypertrophy of astrocytes. The presence of proliferation of glial cells (gliosis) is controversial. No change is seen on MRI images or on pathologic specimens within a week after the onset of ictus. A hyperintense olive is demonstrable on PD/T2 weighted images 3 weeks after, corresponding to the pathological stage of neuronal hypertrophy. Maximum hypertrophy of the olive is seen 5-15 months after the onset of ictus and corresponds to an associated neuronal and glial hypertrophy.

Because of projections from the inferior olivary nucleus to the contralateral cerebellum via the inferior cerebellar peduncle (Fig1), contralateral cerebellar changes are associated with the HOD. These changes are characterized by atrophy and increased signal on PD/T2 weighted images of the dentate nucleus and atrophy of the cerebellar cortex.

Fig5 *MR images obtained in a patient with brainstem cavernous haemangioma and left HOD.* ***(a)*** *Axial PD-weighted image shows hyposignal suggesting hemosiderin deposits in the left tegmentum of the pons and increased signal at the level of the right dentate nucleus.* ***(b)*** *Coronal T1-weighted image shows prominence of the right cerebellar cortical sulci.*



HOD is known to be accompanied by palatal myoclonus, a recurrent dysrythmic contraction of the soft palate.

An hyperintense hypertrophic olivary nucleus, associated with a brainstem tegmental or cerebellar lesion, should not be mistaken for a primary medullary lesion, like an infarct, a neoplasm or a plaque of demyelinating disease.