Parkinson's disease, had selegiline 5 mg q.a.m. added to her previous regime of Parlodel 5 mg t.i.d. and Sinemet 100/25, 8 tablets per day in January of 1990. She was also on amitriptyline, for depression which was discontinued in March of 1990 because of urinary hesitancy. The patient was started on fluoxetine 20 mg q.a.m.

Several days after starting the fluoxetine, the patient started to develop episodes, during which she would shiver and break out into a cold sweat. The episodes would start in midafternoon, and last for several hours. On these occasions, she would feel very clammy, and her hands would be cold. She was seen in the office for assessment one month later. At that time, it was noted that she was very diaphoretic. Her hands were severely vasoconstricted, and the fingers were blue and mottled. Her blood pressure was 200/120.

Previously, the patient had had transient elevations of blood pressure, which were usually induced by stress, but she did not feel under stress on this occasion. The selegiline and fluoxetine were both discontinued, and she recovered over the next few days. Her blood pressure returned to normal (120/90). She did not have any further diaphoretic episodes. She has since restarted fluoxetine with no side-effects.

This patient developed a very unusual reaction, which has not been reported previously with either selegiline or fluoxetine. As she was able to tolerate both medications independently, it would appear to be specifically due to this combination.

Selegiline, a selective MAOB inhibitor at low doses (5-10 mg a day) has been reported to cause hypertension, when taken in high doses, due to loss of selectivity.[4] However, this patient was taking only 5 mg a day and use of deprenyl alone did not cause any elevation of BP on previous visits. Several reports have suggested that administration of tranylcypromine, a non selective MAO inhibitor together with fluoxetine, may result in a "serotonin syndrome" characterized by shivering, diaphoresis, diplopia, nausea and confusion.[5] The temporal sequence of this patient's symptoms, suggests that the episodes of diaphoresis and hypertension may have been due to the combination of fluoxetine and selegiline, and may be similar to the serotonin syndrome reported previously.

No previous reports of a possible interaction between Deprenyl and Prozac have been reported in Canada to this date. However representatives from the manufacturer of Prozac have indicated that they are aware of several reports in the U.S. and that they are recommending that Selegiline and Fluoxetine not be used in combination.

*O. Suchowersky*
*J. deVries*
*Calgary, Alberta*

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## DR. McKENZIE'S YEAR WITH DR. CUSHING

*To the Editor:*

Dr. Kenneth McKenzie and his many contributions to the art of neurosurgery are long remembered even after half a century. After receiving a degree in medicine from the University of Toronto in 1914, he joined the British Royal Medical Corps when World War I was declared. After the war, he returned to the University of Toronto for postgraduate surgical training with Dr. Clarence L. Starr, the Professor and Chief of Surgery there. When Dr. Harvey Cushing was awarded the Charles Mickle Fellowship by the University of Toronto in 1922, he invited Dr. Starr to send a man to Boston to train with him in neurosurgery. Dr. McKenzie was selected for the position and given a $1000 scholarship.[1]

Dr. McKenzie's contributions during his career were numerous and included operative modifications and improvements in the treatment of chronic subdural hematomas and gliomas. He seemed to take particular interest in pathological conditions affecting the cranial nerves. For example he studied and wrote about Menier's disease, acoustic neuromas, and spasmodic torticollis. In reviewing the surgical histories of the Peter Bent Brigham Hospital (PBBH) from 1922 to 1923, we have found that Dr. McKenzie was also interested in the treatment of trigeminal neuralgia. During Dr. McKenzie's training period in Boston, Dr. Harvey Cushing and Dr. Gilbert Horrax performed many operations for the treatment of trigeminal neuralgia. Dr. McKenzie performed admission neurological examinations on most of these patients and recorded both objective and subjective findings for each cranial nerve. He then documented many of his post-operative neurological examinations with diagrams sketched by himself. We report one of the trigeminal neuralgia cases well documented by Dr. McKenzie and show a typical sketch.

AD was a 70-year-old white male who presented with pain involving the entire left side of the face. His problem began 10 years prior to admission; one day while dressing he was seized with a sharp, twisting, shooting, pain in his left forehead lasting for about 30 seconds. He had several more episodes of the same type of pain confined to the left forehead at intervals over 2-3 months. Eight years prior to admission, he developed intermittent paroxysmal pain in the left lower mandibular area which frequently radiated to the left infraorbital region, never crossing the midline. These attacks usually lasted from 15 to 60 seconds and disappeared suddenly. His symptoms gradually worsened both in frequency and severity. A slight jar, chewing, yawning, swallowing, walking without rubber heels or riding over a rough surface could bring on a paroxysm of pain. Neurological examination performed by Dr. McKenzie showed a decreased corneal reflex in left eye but normal sensation in the face.

An avulsion of the third division of the left trigeminal nerve was performed by Dr. Horrax on January 19, 1923. His operative note states: "This case was a very easy and simple one . . . The third division was identified and from this the dura [was] peeled back off the ganglion and sensory root and its fibers avulsed easily and completely as far as could be told. No attempt was made to save the motor division . . . brain [was] allowed to go back to its normal position by lowering the head.

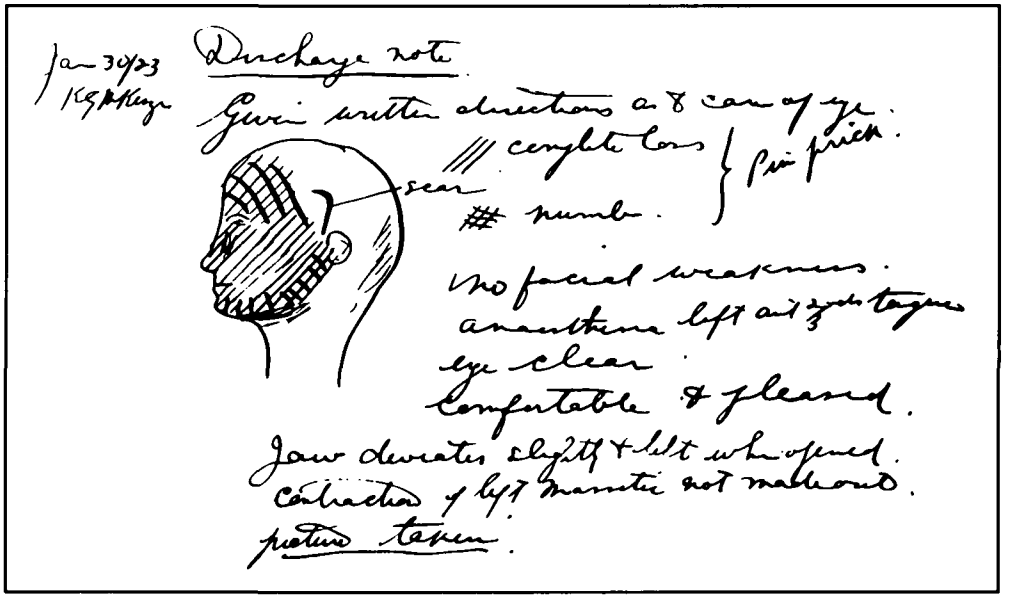


*Figure 1 — Sketch of the left facial sensory examination by Dr. McKenzie dated January 30, 1923.*

Closure [was] then made in layers with fine silk. Patient stood the operation very well."

The patient did well post-operatively and the neurological exam of the cranial nerves at 24 hours by Dr. McKenzie showed complete anesthesia in the areas of division II and III with numbness in the area of division I of the left trigeminal nerve. As expected paralysis of the motor division was noted by left deviation of the jaw, but no facial paralysis was seen. These findings were documented by Dr. McKenzie with a sketch drawn at the margin of his note. AD was discharted on post-operative day 11 in good condition. At that time, the cranial nerve examination findings were again drawn and described by Dr. McKenzie (Figure 1). The patient was found to have complete loss of pin prick sensation in the left cheek, perioral and periorbital area. However, left forehead and mandibular areas showed numbness only. Weakness of the left masseter was noted again. Left facial nerve was intact on discharge.

The above case is typical of the many neurosurgical cases at the PBBH that Dr. McKenzie was involved in. From early in his career, Dr. McKenzie appeared to be interested in cranial nerve pathology. His participation in most trigeminal neuralgia cases during his training and his meticulous cranial nerve examinations seem to reflect this. His lifelong interest in acoustic neuromas may have evolved from this early exposure to cranial nerve cases at the PBBH. Second, like Dr. Cushing, Dr. McKenzie frequently documented his findings with simple, but effective drawings. His use of concise drawings with pertinent descriptions may reflect a subtle influence of Dr. Cushing on the young Canadian neurosurgeon.

*Edward C. Mun*
**Eugene Rossitch Jr.*
*Matthew R. Moore*
*Peter McL. Black*
*Divisions of Neurosurgery*
*Brigham and Women's Hospital*
*Boston, Massachusetts*
**Division of Neurosurgery*
*Duke University Medical Center*
*Durham, North Carolina*

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