Rapid Induction of Acute Dyskinesia by Droperidol

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The butyrophenone droperidol (Inapsine), alone or in combination with fentanyl (Sublimaze) as Innovar, enjoys modest use for sedation, premedication, and as an adjuvant to nitrous oxide anesthesia. Extrapyramidal reactions are, however, occasionally induced.

A rapidly developing and quite severe dystonic syndrome following iv administration of droperidol is described.

REPORT OF A CASE

A 41-year-old, 83-kg healthy man was premedicated with diazepam (Valium), 15 mg, im, and an axillary block was performed 45 minutes later with 40 ml of 1 per cent mepivacaine (Carbocaine) for a ganglionectomy. Droperidol, 10 mg, in divided doses was administered iv for additional sedation over a 10-minute period. Approximately 8 minutes later, shortly after surgery had begun, the patient was noted to be wrinkling his forehead and biting his lower lip. Upon questioning, he stated in a strangled voice that he was not in pain. Within one minute, he developed severe perioral spasms, protruded his tongue, and grimaced markedly. His eyes rotated upwards and to the right and seemed fixed in that position. His neck became rigid, his mandible protruded forward, and he bit his tongue. His entire face seemed to be in spasm, and he became polyphonic. He remained fully conscious, able to breathe deeply and move his unanesthetized extremities on command, but was totally unable to speak, uttering only a grunt when questioned. Diphenhydramine (Benadryl), 75 mg, iv, was administered, with resolution of all signs of the extrapyramidal reaction within 1 minute. The entire episode lasted not more than 3–4 minutes from onset until eradication, with no recurrence. The patient later stated he had been completely aware of these changes but had been unable to overcome them.

DISCUSSION

It has been suggested that phenothiazines and butyrophenones act by blocking the dopaminergic receptors within certain areas of the central nervous system, leading to increased production of transmitter substances.†–§

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In the extrapyramidal system, however, this receptor blockade may create a functional dopamine deficiency and cause extrapyramidal symptoms similar to those caused by the actual dopamine deficiency which may be present in Parkinson’s disease within the substantia nigra and corpus striatum.†–§

When drug-induced extrapyramidal symptoms occur, they may take three somatic forms†–§: 1) Akinesia (parkinsonism), difficulty initiating movements, cog-wheel rigidity, mask-like facies, tremors, lethargy, weakness, and lack of initiative. 2) Akinetiasis, characterized by restlessness, inability to sit still, and constant ambulation. Patients localize certain symptoms in their muscles, forcing them to move about. 3) Dyskinesia (dystonic syndrome, dystonia), due to hypertonicity of regional muscle groups, which may be generalized in nature, such as opisthotonus, emprosthotonus (bending forward), scoliosis, and contractures of legs. Frequently this may involve the muscles of the neck, pharynx, and face, leading to speech and swallowing difficulties, perioral spasms, grimacing, protrusion of the tongue, torticollis, masseter spasm with trismus, and oculargic spasms. Consciousness is never impaired.

Although other forms are seen, most extrapyramidal reactions caused by droperidol are of the dystikne type, and they usually occur in the recovery period. Occasionally, as in the present case, they occur more rapidly. Corssen† observed muscle jerking and twitching in three patients shortly after injection of Innovar, and Tornetta† reported 17 patients in whom acute rigidity of facial, mandibular, and pharyngeal muscles developed in the immediate post-Innovar-injection period, occasionally interfering with ventilation. Janis‡ described a patient who sustained rapid development of an acute severe stiffness of neck and back muscles, with facial grimace, without chest-wall rigidity or ventilatory impairment, following premedication with Innovar, iv.
Although it is not possible to rule out other etiologies, including an adverse reaction to fentanyl, some of these cases probably represented acute dyskinnesia of very rapid onset induced by droperidol.

Anticholinergic drugs such as trihexyphenidyl (Artane), benztrpine (Congentin), diphenhydramine, and atropine will usually ameliorate or eliminate extrapyramidal symptoms, apparently indicating an interaction of cholinergic and dopaminergic effects within the extrapyramidal system. Probably related is the observation by Snyder that certain phenothiazines with strong intrinsic anticholinergic activity seem to induce fewer extrapyramidal reactions than do the phenothiazines or butyrophenones, which have weak anticholinergic effects.

REFERENCES


Hepatic Function

AUSTRALIA ANTIGEN AND POST-TRANSFUSION HEPATITIS The authors investigated all 17 cases of reported posttransfusion hepatitis occurring during a two-year period in Copenhagen. In the first year before Au antigen testing was begun, about 21,000 transfusions were performed and nine cases were reported. In the second year, about the same number of transfusions were done, 49 Au antigen-positive units were rejected, and eight cases of hepatitis were reported. Detailed studies confirmed the diagnosis of posttransfusion hepatitis in eight and six cases, respectively. Thus, no real difference in incidence could be shown. Another follow-up investigation showed that in the second year eight patients had received emergency transfusions of blood later found to be Au antigen-positive. Five of these died of their primary disease with no clinical or pathologic evidence of hepatitis. Three survivors were free of clinical hepatitis, increased SGOT, and Au antigen for more than six months, though one developed Au antibody. (Reinicke, V., Banke, O., and Dybkjaer, E.: Australia antigen and posttransfusion hepatitis, Vox Sang, suppl 24:65–71, 1973.) ABSTRACTER'S COMMENT: The evidence suggests that, at least in Copenhagen, there is no correlation between donor Au antigenemia and recipient hepatitis. Many other studies suggest the opposite: for example, Goeke, D. J.: A prospective study of posttransfusion hepatitis, JAMA 219:1165–1169, 1972.