To the Editor—I am perplexed by Lau et al’s article describing circulatory collapse after metoclopramide administration. Although a causative effect was suggested, the dose and rate of administration of the metoclopramide was not mentioned. The proposal that serotonin autoinhibition was a cause of the collapse, based on an animal study done to explain sudden infant death syndrome, is intriguing.

The authors also failed to elaborate on the uncommon but well-documented incidents of intravenous metoclopramide-induced asystole and/or hypotension. Impairment of the autonomic nervous system may be a common predisposing factor. When tested in nine adults with autonomic failure, an intravenous dose of 5 mg of metoclopramide consistently lowered systolic and diastolic blood pressures, starting at a mean of 33 seconds. Withington reported a 54-year-old man who developed complete heart block and asystole that lasted for 25 seconds after receiving 10 mg of metoclopramide post-pancreatectomy. Grenier and Drolet described a 66-year-old woman with diabetes who required external cardiac massage for asystole after receiving 10 mg of metoclopramide post-mastectomy. In a similar case reported by Bentsen and Stubhaug, cardiac arrest was ascribed to the rapid intravenous injection of metoclopramide via the central venous route.

Thus, the report by Lau et al reminds us that intravenous metoclopramide may occasionally cause serious consequences. A slow intravenous infusion over at least 2 minutes may be associated with fewer side-effects.

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References