Thus, if a single site of action is involved in the depressant action of methoxyflurane on the heart, that site would appear to be the glucose phosphate isomerase step. By a similar line of reasoning, this is also the step implicated for halothane.[1, 2, 4]

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Respiration

FAMILIAL DYSAUTONOMIA AND RESPIRATORY CONTROL The authors describe a detailed study of a patient with acquired dysautonomia (*i.e.*, inappropriate response of autonomic function) who had abnormal circulatory control mechanisms with orthostatic hypotension (*i.e.*, on tilting to head-up position, arterial blood pressure fell from 102/60 to 70/35 torr without a change in heart rate; there was no blood-pressure response to cold stimulation nor a post-Valsalva blood-pressure overshoot). Exposure to a mixture of 10 per cent oxygen and 90 per cent nitrogen was not associated with a change of ventilation despite a decrease in Pa_{O_2} from 64 to 30 torr. This response was similar to that observed in the experimental animal with a denervated carotid body or in man following bilateral block of the ninth and tenth cranial nerves. The positive interaction between elevated P_{CO_2} and lowered P_{O_2} was absent; *i.e.*, when the patient was challenged with 5 per cent CO_2 in 95 per cent oxygen there was an increase in ventilation (less than in normal man) which was as large as the response to 5 per cent CO_2 with 15 per cent oxygen. Administration of atropine intravenously in increments of 0.4 mg to a total dose of 2.0 mg did not increase heart rate, although a characteristic increase followed the infusion of isoproterenol. The lack of response to hypoxia demonstrates absent peripheral chemoreceptor activity, while the weak response to CO_2 suggests some central chemoreceptor involvement. (*Eisele, J. H., and others: Abnormal Respiratory Control in Acquired Dysautonomia, New Eng. J. Med. 285: 366, 1971.*)