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 Exposure to a blood-pressure overshoot). mixture of 10 per cent oxygen and 90 per cent nitrogen was not associated with a change of ventilation despite a decrease in Pao2 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_{CO2} and lowered P_{O2} was absent; i.e., when the patient was challenged with 5 per cent CO2 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 CO2 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 CO2 suggests some central chemoreceptor involvement. (Eisele, J. H., and others: Abnormal Respiratory Control in Acquired Dusautonomia, New Eng. J. Med. 285: 366, 1971.)