1. When a person assumes the erect position without muscular movements of his legs. If a person is tilted on a board or table with his feet down and maintained there long enough he will usually faint and collapse (after ten to fifteen minutes).

2. Extensive sympathectomy deprives a patient of many of his compensatory mechanisms.

3. Any drug which produces marked peripheral vasodilatation, such as nitrites or morphine. Drew showed that patients who had received morphine were four times as apt to faint when placed with their feet down. When heat is applied to the whole body or when the body temperature is increased, widespread vasodilatation also occurs.

4. The compensatory mechanisms are decreased by trauma, deep anesthesia, or prolonged anoxia.

5. The effect of postural change is increased by a decreased blood volume as in hemorrhage or shock—the venous return is already deficient, and further gravitational factors may overwhelm the compensatory mechanisms.

Early ambulation postoperatively is very important in preventing postural hypotension by not permitting skeletal muscle atrophy to develop.

E. E.

ULMER, JACK L., AND MAYFIELD, FRANK H.: *Causalgia.* Surg. Gynec. & Obstet. **83**: 789–796 (Dec.) 1946.

The term causalgia was first used by Mitchell, Morehouse, and Keen in 1864 for a bizarre symptom complex following injury to peripheral nerves. The symptoms include severe burning pain and hyperesthesia in association with trophic and vasomotor changes. Following an injury certain people set us a reflex mechanism accompanied by abnormal vasomotor activity so that there exists varying degrees of vasodilatation or vasoconstriction. The true cause of this is not known.

A series of 75 army cases was presented. These patients had incomplete nerve injuries from shell fragments or bullet wounds. In 44 of these burning pain developed immediately after injury; in 14 within forty-eight hours; and the other 17 from seventy-two hours to two months. In all the patients the symptoms had been present from three and a half weeks to fifteen months at the beginning of the treatment. There seemed to be no correlation between the severity of the lesion and the severity of the symptoms. There was either vasodilatation (skin red, dry, scaly, and warm) or vasoconstriction (skin cold, glistening, and perspiring).

Seventy of the 75 cases were treated by surgical interruption of the sympathetic chain. This included preganglionic ramisectomy of the second and third dorsal ganglia for the upper extremity, and the second and third lumbar ganglia for the lower extremity.

In every case procaine blocks of the appropriate sympathetic ganglia were done before surgery. This invariably gave immediate and dramatic relief for one to three hours. A few had partial relief for several days. Some were injected 4–8 times hoping for a cure from the injection alone, but this was not accomplished. The procaine block accomplishes two things: (1) it relieves the pain temporarily so that the extent of the nerve injury can be evaluated more accurately, and (2) it establishes the indication for sympathectomy.

E. E.

WHITTIER, LA MONT, AND DOMBROWSKY, EDWARD F.: *Mucous Cyst at Base of Tongue as a Cause of Sudden Death in an Infant.* J. Pediat. **92**: 774–776 (Dec.) 1946.

A case of sudden death due to a mucous retention cyst at the base of the tongue is reported.

Although uncommon, a mucous retention cyst at the base of the tongue

should be considered in the differential diagnosis of asphyxia. Whenever unexplained choking, dyspnea, or cyanosis occurs in an infant, a cyst of this nature should be ruled out by careful visualization of the base of the tongue. The importance of this becomes apparent when it is realized that this condition is readily amenable to treatment. Since these cysts are usually near the midline, the ordinary large wooden tongue depressor may cover and flatten them, obscuring them from the examiner. A small, infant size tongue blade therefore should be used in this examination.

D. S. H.

HOUSTON, CHAS. S.: *Operation Everest: A Study of Acclimatization to Anoxia.* U. S. Nav. M. Bul. **46**: 1783–1792 (Dec.) 1946.

During a month's period, four volunteer subjects were confined continuously in an elaborate low pressure chamber which was equipped as adequately as possible for comfort, recreation and exercise, and into which attendants entered through a communicating lock. "Ascent was made at the rate of 2,000 feet per day to 9,000 feet, at 1,000 feet per day to 15,000 feet, and at 500 feet per day thereafter, all made gradually during a two hour period in the evening. . . . On the last three days of the study, altitude tolerance was evaluated by an eight hour ascent to 29,000 feet without supplementary oxygen, and a five hour ascent to 50,000 feet using only the non-pressurized diluter demand oxygen regulator. . . . None of the subjects were seriously affected by altitude below 20,000 feet, and at no time during the study was the clinical picture of 'mountain sickness' observed. There was a general tendency to laziness and loss of strength; the subjects were unable to exercise as much above 18,000 feet as below. . . . The changes in (arterial) blood and alveolar gases may be summarized as follows: Arterial carbon dioxide pressure fell progressively to as low as 17 mm. at rest with corresponding oxygen pressure of 29 mm. Several resting arterial oxygen saturations below 60 per cent were found, and in most cases both the arterial oxygen pressure and saturation fell during work. Above 10,000 feet, alkaline reserve decreased from a sea level of 46 vols, % to an average of 34 vols. %. All four men became alkalotic with arterial pH values of 7.45 to 7.60; despite this, and the low carbon dioxide pressures, no signs of tetany were seen. Although the alveolar oxygen pressures followed the mean curve described by Boothby, the alveolar carbon dioxide pressures fell below the Boothby curve by 5 mm. at 18,000 feet and 10 mm. at 25,000 feet. The reticulocyte count, as well as the hemoglobin content and red cell count increased moderately in all four subjects. The polycythemia, however, was not great (no counts exceeded 6.7 million) and bore no constant relation to the completeness of acclimatization." . . . In the electrocardiograms "most subjects developed progressive lowering of the T-wave with increasing altitude up to 16,000 feet, but the trend was reversed above this altitude and records taken at the peak altitudes show T-waves actually higher than at sea level. Premature beats, slight P-R prolongation and a tendency to sinus arrhythmia with Cheyne-Stokes breathing occurred in some records. Six-foot films showed no change in either size or shape of the heart at increasing altitude. . . . Exercise tolerance decreased in varying degrees in all subjects. . . . The limiting factors were dyspnea or fatigue or both. Changes noted with increasing altitude in the pre-exercise resting pulse, the peak rate attained during exercise and the final rate after recovery. . . . One hundred per cent of oxygen at 20,000 feet markedly in-