gest a medulla affection. It is not always well marked. It may suddenly disappear to return as suddenly, and may first occur in consequence of a slight traumatism. Apoplexy with complete coma may occur, followed by hemaplegia, recovery from which may be rapid and complete.

Sometimes there is sudden loss of consciousness, which disappears so rapidly without resulting paralysis as to suggest epileptic states. Vertigo sometimes occurs alone, and sometimes precedes paralysis. Paralytic symptoms occur without loss of consciousness. Hemiplegia may be attended by very bizarre phenomena. In one case a left hemiplegia was accompanied by a monoplegia of the right eyelids. Monoplegias are very frequent in diabetes, and are apt to be extremely transitory. Paralysis of the right arm and face, ptosis, pupil dilatation, strabismus, and hesitancy in speech may follow glycosuria. While speech disorders are generally due to buccal dryness, yet true aphasia occurs, and aphonia from laryngeal paralysis is far from exceptional.

Imperfect muscular co-ordination in the dark attended by formication in the extremities may lead to a suspicion of locomotor ataxia. Cramps of the akinesia algera type frequently attack the lower extremities, especially at night and play an important part in the production of insomnia, being often the first indication of cerebral circulatory disturbance, and may be precursors of serious complications. Convulsions may be associated with coma, or may accompany paralytic phenomena. At times they present the monoplegic epileptic character and alternate with transitory paralysis of the same side.

Diabetic vertigo often assumes an epileptoid character. Asthma, exophthalmic goitre, and other respiratory neuroses are not infrequently temporary expressions of diabetes. Underneath them, and many diabetic neurotic states, lies the " air hunger " of the tissues, which is simply their expression of the need of oxygenation.

Diabetic absorption of oxygen as Voit, Peltenkoffer and others have shown, is much less than the normal, and decreases until toward the end of the disease, when it is hardly half the normal quantity. Carbon dioxide exhaled is proportionally reduced. This oxygen decrease Sajous ascribes, with much plausibility, to suprarenal disorder. Increased suprarenal activity, as Croftan has shown, can so augment the ferment producing power of the pancreas as to greatly increase sugar elimination through increase of the amylolytic ferment supplied by the pancreas which converts the liver glycogen into dextrose.

Herein lies the explanation of neuropathic glycosuria and of