TABLE I.
Desensitization of Rabbits by Intravenous Injections of Human Serum.

| | Skin s'tivity | Serum p'pitin | Serum p'pitinogen |
|---|---|---|---|
| Rabbit 173 injected 0.5 cc. per kg; total 1.44 cc. | | | |
| Before injection | 1,000 | — | — |
| 1 hr. after injection | und.* | und.* | 100 |
| 6 " " " | und. | und. | 100 |
| 24 " " " | | 10 | 10 |
| 48 " " " | 100 | 10 | 10 |
| 120 " " " | 1,000 | 10,000 | und.* |
| Rabbit 213 injected 0.5 cc. per kg.; total 1.41 cc. | | | |
| Before injection | 1,000 | 10,000 | — |
| 1 hr. after injection | — | — | 100 |
| 6 " " " | und. | — | 100 |
| 24 " " " | | 100 | 100 |
| 48 " " " | 100 | 1,000 | 10 |
| 120 " " " | 1,000 | 10,000 | und. |

* Reactions obtained with undiluted serum only.

normal rabbits. Forty-eight hours after the desensitizing injection, the skin response was 100; in 120 hours, it reached the original level. Of interest is the fact that the precipitin titer which was negative at the time of the desensitizing injection, reached 10,000 120 hours after this injection.

Rabbit 213 which showed a skin sensitivity titer of 1,000 and a serum precipitin titer of 10,000, was completely desensitized after an intravenous administration of 0.5 cc. human serum per kg. of body weight. One noted a return to the sensitized state 48 hours after the desensitizing injection.

It is evident also that the precipitinogen content of the serum is at its height during the desensitized state, disappearing as the titers of skin sensitivity and serum precipitins reach a high level.

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## Dizziness, Fainting and Convulsions Due to Hyperactivity of the Carotid Sinus Reflex.

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A study has been made of the circulatory and the nervous systems of 12 subjects who complained of dizziness and fainting and in whom pressures of graded intensity on the right or left carot-

id sinus promptly and regularly induced asystole, fall in blood pressure (9 cases), dizziness (10 cases), fainting (8 cases), unconsciousness (7 cases), and convulsive movements (8 cases). The unconsciousness was preceded by typical and characteristic visual or auditory aurae. The convulsive movements were entirely contralateral on mild stimulation of the sinus, while on stronger stimulation they became generalized. After the pressure was relieved they ceased almost immediately. Dizziness and fainting were induced spontaneously by sudden turning of the head in 2 cases. The ages of the patients varied from 14 to 71 years; 10 were above 55 years.

A definite aneurysmal dilatation of one or both carotid sinuses was noted in 5 cases. A small tumor pressing on the sinus was found in 3 cases. No gross pathology of the sinus was detected in the remaining 4. Aneurysmal dilatation does not necessarily cause hyperirritability of the sinus reflex, for this lesion was observed in cases which failed to show hyperactivity of the sinus. Hyperactivity of the carotid sinus reflex is not always permanent, as was demonstrated by 2 cases which exhibited periodic hyperactivity of the reflex. The observations so far indicate that the hyperactivity of the reflex depends on the following factors, singly or combined: the excitability of the afferent nerve endings within the sinus, the state of the medullary center, and the excitability of the efferent cardiovascular nerve endings.

In some cases the convulsions depended on the duration of the asystole or the degree in fall of the arterial blood pressure. An asystole of 8 to 10 seconds was usually followed by convulsions. In one case fainting was observed without slowing of the heart or fall of the arterial blood pressure.

Comparative measurements of the volume and velocity of the blood flow indicate that the circulation time is considerably prolonged, and the cardiac output diminished, during the convulsive state. Internal jugular blood, taken during a convulsion, showed a decidedly lower oxygen and higher carbon dioxide content than when the patient felt well.

Instantaneous changes following pressure and release of pressure on the carotid sinus were observed in the cardiac rhythm and intracardiac conductive system. These changes in different cases consisted in the development and cessation of complete or partial A-V block, ventricular escape, complete temporary arrest of all chambers of the heart, bundle-branch block, and alterations characteristic of coronary artery disease.

In one case in which pressure of a small tumor on the sinus was

responsible for fainting and convulsions, surgical removal of the tumor and unilateral denervation of the sinus by Dr. Tracy J. Putnam abolished all sinus responses. Following section of the carotid nerve, this patient developed an acute arterial hypertension and auricular fibrillation; the hypertension disappeared within 48 hours, the fibrillation after 7 days. In this case complete relief followed the surgical treatment.

All the abnormal responses of the carotid sinus were abolished after unilateral novocainization of the sinus; while the reactivity of the opposite sinus remained unaltered or was increased. Intracarotid administration of a minute amount of atropine abolished ipsolaterally the effect of stimulation. Peripheral paralysis of the vagus endings by atropine abolished the cardiac effect but not the vasomotor. Epinephrine given parenterally abolished both the vasomotor effect and the cardiac slowing, presumably by overcoming the vagal inhibition.

Continuous intravenous administration of large amounts of histamine, acetylcholine, and carbon dioxide, which are dilators of the cerebral and peripheral blood vessels, failed to influence the effect of stimulation of the sinus.

The hyperactive state of the carotid sinus reflex is specific and is usually independent of the condition of other reflexes. In the cases studied, the somatic and other autonomic reflexes were normal. Conversely, in control subjects with sensitive and irritable vasomotor systems, the carotid sinus reflex was normal. In 150 cases of idiopathic epilepsy pressure on the carotid sinus failed to induce seizures.[1] Convulsions may be due to many causes, and hyperactivity of the carotid sinus reflex is seldom found when convulsions occur, but it can produce them.

[1] Lennox, W. G., personal communication.