diminished because of the diminution of hemoglobin. In spite of the fact that the difference in carbon dioxid content and tension between arterial and venous blood is comparatively small, there is a very definite difference in P_H. The arterial P_H lies well to the alkaline side of the 7.35 line in those cases where there is a difference, while the venous point lies practically on the 7.35 line.

It is suggested tentatively as an explanation of this phenomenon, that the tissue CO_2-tension and P_H must lie at or above that of the venous blood and not in equilibrium with the arterial blood. As it is presumably the tissue CO_2-tension or hydrogen-ion concentration in the respiratory center which controls the respiratory mechanism, the tendency of the repsirations will be to maintain this constant rather than the hydrogen-ion concentration of the arterial blood. In normal persons arterial and venous P_H are practically identical because of the slope of the dissociation curve and the effect of oxygen. In anemia the effect of these compensating reactions is diminished so that true relations become more evident. It has already been demonstrated by Michaelis,[1] and others that the hydrogen-ion concentration of the venous blood is maintained constant at $P_H = 7.35$ with a variation of ± 0.08, which agrees well with our values for both arterial and venous P_H.

4 (1586)

Precipitin response in the blood of rabbits, following subarachnoid injections of horse serum.

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During the treatment of cases of cerebrospinal meningitis with antimeningococcic serum in a large Army hospital,[2] a curious reaction was repeatedly observed. This appeared in patients who, after having received several intraspinous treatments with serum, were given serum intravenously. While such injec-

[1] Michaelis, Wasserstoffionenkonzentration, Berlin, 1914.

[2] Hospital of the American Embarkation Center, LeMans, France.

tion was made into the blood stream, or immediately thereafter, some of the following signs and symptoms frequently appeared: flushing, sudden feeling of warmth, restlessness,—then, pallor, dyspnea, cyanosis, vomiting, and prostration. Epinephrin and atropin hyperdermatically, induced relief. These manifestations, apparently anaphylactic, occurred only after several days of intraspinous treatments had elapsed before initial intravenous serum therapy, but had no relation to the time of the last intraspinous injection. They were not noted when combined intravenous and intraspinous therapy was applied from the outset. Similar observations are described by Stone and Truit[1] in their report of a large series of cases of meningitis at Camp Funston, and Haden[2] confirms them in one of his case reports of meningitis.

With this experience in mind, horse serum was injected into rabbits intraspinously, and the resulting precipitin formation in the blood was compared with that induced by similar intravenous injections. In a few instances, anaphylactins were studied.

Normal horse serum, without preservative, was used throughout these experiments. This was injected into the subarachnoid space of rabbits by introducing a No. 24 Luer needle attached to a glass syringe, through a sterile field just below the occipital ridge in the mid-line. The needle was carried forward and slightly downward until it punctured the occipito-atlantoid ligament. A yield of from 0.5 c.c. to 1.0 c.c. of spinal fluid was thus readily obtained. Leaving the needle in place, and disconnecting the syringe, a second syringe with a correct amount of serum was then attached. By slightly withdrawing the plunger, freedom from chance puncture of a vessel was assured, and the serum then slowly injected. No anesthetic was needed, the rabbit being securely tied in the prone position to a board and the head flexed and pulled forward by the ears.

Precipitin tests were made by mixing 0.3 c.c. of rabbit serum with 0.3 c.c. of normal horse serum. After incubation at 37° C. for one hour in a water bath, the tubes were placed in the ice chest overnight, and readings made the following morning. As controls, normal rabbit serum and normal sheep serum were

[1] Stone, W. J. and Truitt, R. C. P., *Arch. Int. Med.*, 1919, xxiii, 282.

[2] Haden, R. L., *Arch. Int. Med.*, 1919, xxiv, 514.

used. In all instances, rabbits receiving subarachnoid injections were paralleled by rabbits injected with identical amounts of the same serum intravenously, and the bleedings and precipitin tests of each were done at the same time.

Summary: (1) Rabbits receiving a single dose (0.5 c.c.) of normal horse serum into the subarachnoid space, produce precipitins in the blood in greater abundance, of higher titer, and which persist longer than those in control rabbits receiving a similar injection intravenously.

(2) Repeated subarachnoid injections (0.5 c.c.) of normal horse serum in rabbits, induce precipitins in the blood early. These may appear in high titer as soon as one week after the initial injection, whereas in rabbits similarly treated intravenously, no precipitins were found at this time. They may appear a few days thereafter and reach a high titer.

(3) No anaphylactic manifestations occurred in rabbits treated repeatedly with subarachnoid injections of normal horse serum when the precipitin content of the blood was high.

(4) Anaphylactins, as determined by passive transfer of anaphylaxis, were demonstrated in sera with high precipitin content.

(5) These experiments may explain clinical manifestations of intolerance to horse serum, observed when an initial intravenous injection of antimeningococcic serum followed a series of intraspinous injections of such serum.

5 (1587)

Experimental gigantism produced by feeding pituitary gland.

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Growth of the individual stops, when the size is reached which is specific for the species to which the individual belongs. The causes which lead to the cessation of growth are not fully known. In man it happens sometimes, that growth continues beyond the normal maximum size of the species; this condition is known as gigantism. Clinical evidence points to the conclusion