Bastianelli says: "The preceding malaria creates the fundamental disposition; the existing malaria the accidental disposition; the quinine the provocative agent."

He divides quinine hemoglobinuria into two forms:
(1) That occurring during the paroxysm—paroxysmal quinine hemoglobinuria.
(2) Post-malarial quinine hemoglobinuria.

In these varieties quinine, through a very considerable length of time, will produce a hemoglobinuria whenever administered. There are, however, instances where the hemoglobinuria due to the taking of quinine occurs only now and then during the paroxysm—that is, is an episode. These cases are rare.

Bastianelli comes to interesting and practical results with regard to treatment. The course pursued depends upon the blood examination.

If hemoglobinuria occurs during a malarial paroxysm and parasites are found in the blood, quinine should always be given.

If, however, no parasites are found, either as a result of previous administration of quinine or on account of the spontaneous disappearance of the organisms, we may remember that the administration of quinine will have no effect upon this attack and that for the time being certainly another attack is not to be expected. In these cases Bastianelli recommends not giving quinine, owing to the possibility that the paroxysm may have been due to its previous administration.

If in an attack occurring in the middle of an ordinary malarial paroxysm there arises doubt as to its origin from quinine, it is well to abstain from the further administration of the remedy, for the quinine already given is usually sufficient to hinder the development of new febrile paroxysms. But if in a hemoglobinuric attack which has come on after the giving of quinine the parasites are still found in the blood, one is justified, despite the danger, in insisting upon the specific treatment; if there be doubt as to the origin from quinine, we may be sure what the result will be if we allow the parasites to go on developing, and it is therefore safer to interfere.

[This careful abstract (for which we are indebted to Dr. Thayer) should prove of great value to those Southern practitioners who are interested in quinine hemoglobinuria. The condition, so far as I know, is not met with in this latitude.—W. O.]

The Direction of Rotation in Cerebellar Affections.—Russell (British Medical Journal, April 10, 1897), in a previous communication to the British Medical Journal, May 18, 1895, published the results of experimental work on animals, in which he had found that animals deprived of one lateral lobe of the cerebellum rotated away from the side of the lesion. Dr. Bruce Ferguson believed that Russell had fallen into an error of expression, and concluded from the latter's description that the rotation was toward and not away from the side of the lesion. Russell, in the present article, endeavors to substantiate his previous views, and states that the discrepancy in views arises from the fact that some describe the direction in terms relating to the observer, while others describe it in terms relating to the observed. He believes that in the case of the human subject, as well as in animals, there
would be less likelihood of a difference of opinion if all physicians observed two rules: (1) That the object should be placed in front of the observer, and that both should be looking in the same direction. (2) That the direction of rotation about a horizontal axis should always be expressed in terms relating to the direction in which that part of the circumference of the object corresponding to its upper quadrant is moving.

Of all the methods of describing rotation, Russell thinks that the simplest is that in which the direction is compared to the rotation observed in a righthanded male screw. This being clear, the direction of rotation either resembles a screw entering or coming out of an object. The next in order of simplicity seems to be the method by which the direction is denoted by the terms "clockwise" and "anti-clockwise." It is necessary, however, in this method to note the relation of the object to the face of the clock.

Pathology and Etiology of Acute Ascending Spinal Paralysis.—Keewer (Zeitschrift f. klin. Med., 1897, Band xxxii.) says that while most writers coincide in the view that Landry's paralysis is a multiple neuritis, every opportunity should be taken to study the changes in such cases. The writer reports four cases. In three of them, ending fatally, the peripheral and central nervous systems were studied microscopically. Changes were found in both. In the former a subacute degenerative neuritis; in the white substance of the cord a similar but more acute change; cloudy swelling of the cells in the gray matter, desquamation of the cells of the central canal. The vessels were distended with blood. No special changes in the cerebrum. No bacteria could be found in the sections. The author thinks that he can conclude that Landry's paralysis is nothing but the second and third stages of a chronic multiple neuritis that affects the cord by continuity. Here it spreads rapidly, generally upward, sometimes downward, causing death by involvement of the vital centres of the medulla. The outbreak usually follows an attack of some infectious disease. Clinically the progressive character of the paralysis is characteristic; the direction of its spread, however, is not. Pathologically, Landry's paralysis is a subacute chronic polyneuritis and an acute diffuse degenerative myelitis.

The Demonstration of Peptone (Albumoses) in the Urine and the Preparation of Urobilin.—Salkowski (Berl. klin. Wochenschr., 1897, No. 17) was struck by the fact that urobilin containing urine gave a violet color upon adding the first drops of copper sulphate of the Trommer test, and made a number of experiments to find if urobilin could give the albumose reactions. The material obtained by precipitating such urine with lead acetate and phosphomolybdic acid gave a biuret reaction and the urobilin absorption-band. The same is true of the residue obtained by shaking out in ether, making it probable that the biuret in the original comes from the urobilin. The desirability of having a preparation of urobilin for experimental purposes is evident. The method of Jaffe, modified by the use of phosphomolybdic acid, yields, the author thinks, a purer product than has yet been obtained. It differs from that obtained by the original Jaffe method in some of its physical characters, and its insolubility in ether. It gave a biuret reaction almost exactly like that of albumose.