conformation, according to race, age, sex, and even stature, not to mention pathological variations. In no two individuals of the same race, age, sex, and stature are the skulls ever precisely alike; nor, under such circumstances, are the fissures and convolutions of the brain ever identical in pattern. The relations of the brain's convolutions and fissures to definitely chosen anatomical points on the surface of the skull or scalp vary in individuals of the same race, etc. Lastly, as the convexity or surface area of the brain is always less than the outer surface area or convexity of the skull, and still less than the surface of the scalp, lines drawn on the scalp cannot always correspond exactly to the fissures and convolutions of the brain.

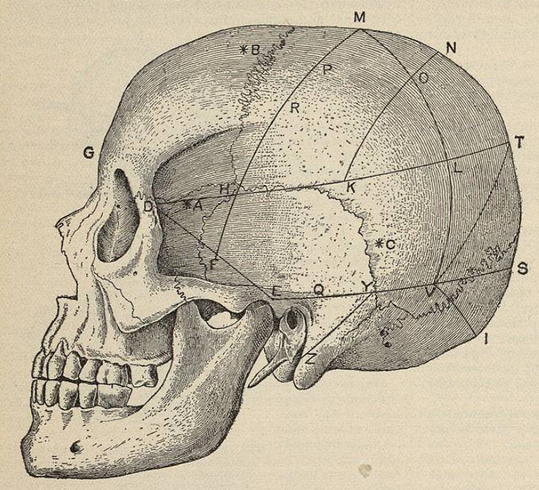

FIG. 4319.—Chiene's Guiding Lines in Cranio-Encephalic Topography. *G*, glabella; *I*, inion and indicates site of torcular Herophili; *M*, mid-point between *G* and *I*; *T*, three-quarter point between *G* and *I* and overlies parieto-occipital fissure; *S*, seven-eighth point between *G* and *I*; *E*, pre-auricular point on root of zygoma; *Q*, post-auricular point on supramastoid crest; *Y*, a finger's breadth behind *Q*; *V*, mid-point of line *E S* and marks highest convexity of lateral sinus; *Z*, near tip of anterior border of mastoid process; *D*, external angular process of frontal bone; *F*, mid-point on line *D E* and overlies point of bifurcation of middle meningeal artery; *H*, Sylvian point located by intersection of the Sylvian line *D T* and the line *F M*; this Sylvian point overlies the Sylvian point of the fissure of Sylvius and also the anterior division of the middle meningeal artery; *L* indicates angular gyrus and extremity of parallel fissure and is located by the intersection of line *D T* and line *V M*; *K*, mid-point of line *H L*; *H M*, precentral line and overlies precentral fissures; *K N*, line drawn from *K* to median sagittal plane parallel with *H M* and is the postcentral line; *H A*, anterior horizontal limb of the Sylvian fissure; *H K*, posterior horizontal limb of the Sylvian fissure; *O K L*, triangular area indicating parietal eminence, supramarginal convolution, and terminal part of Sylvian fissure; *R*, at the level of the temporal crest and on line of inferior frontal fissure; *P*, on line of superior frontal fissure; *T V* courses a little in front of the lambdoid suture; *T V I*, triangular area indicating occipital lobe of cerebrum; *I V Y Z*, guiding lines for lateral sinus; below *I V Y Z* is the region of the cerebellar hemisphere; *F E V L H*, pentagon indicating temporal lobe of the cerebrum except the apex which projects a finger's breadth downward, forward, and inward from *F*; *H K N M*, parallelogram indicating cortical motor area (Rolandic area); **A*, site for opening subarachnoid space; **B*, Kocher's point for tapping the lateral ventricle; **C*, Keen's point for tapping the lateral ventricle; *Y Z*, indicating anterior limit of lateral sinus.

In view of the preceding facts the well-informed anatomist knows that the anatomical lines used in cranio-encephalic topography to locate fissures, convolutions, and areas of the brain are simply approximations—approximations that have been correlated from examinations of many brains and skulls. Fortunately, these approximations are found to be exceedingly useful as reliable guides in surgical anatomy. Practically, the surgeon uses the lines and points correlated by the anatomist, and overcomes the difficulties due to variations in individuals, by removing a greater area of the cranial wall than that indicated to him by the anatomist's lines and points as corresponding to the portion of the encephalon which he desires to expose. The brilliant results of modern cerebral surgery clearly proclaim the practical utility of the anatomist's approximations in cranio-encephalic topography.

Many methods have been devised for mapping out the relations of the scalp to the cranial contents. From a clinical point of view, that introduced by Professor John Chiene, of Edinburgh, is a very simple and useful one (Fig. 4319). His method is as follows, viz.: The head being shaved, the *mid-point* (*M*) is located in the sagittal plane of the vertex from the glabella (*G*) to the inion (*I*); then the *three-quarter point* (*T*); and then the *seven-eighth point* (*S*). Next locate the *pre-auricular point* (*E*) on the root of the zygoma, and the *external angular process* (*D*) of the frontal bone. Having located these points join *D T*, *E S*, and *D E*. Bisect *E S* at *V* and *D E* at *F*. Draw the line *F M* and also the line *V M*. Bisect *H L* at *K* and draw a line from *K* to *N* parallel to *H M*. Trisect *H M* at the points *R* and *P*. Draw a line from *T* to *V* and another from *V* to *I*. A finger's breadth (18 mm.) posterior to the *post-auricular point* (*Q*) is the point *Y*; at the tip of the anterior border of the mastoid process is the point *Z*. A line drawn from *Y* to *Z* indicates the anterior border of the mastoid portion of the *lateral sinus*. The line *H M* corresponds to the *precentral fissures*, and may be called the *precentral line*. The points of its trisection correspond to the posterior extremities of the *superior* (*P*) and *inferior* (*R*) *frontal fissures*. The line *D T* is called the *Sylvian line*; it intersects the precentral line at the point *H*, which corresponds to the *Sylvian point* of the fissure of Sylvius, and also to the *anterior division* of the middle meningeal artery. *H A* corresponds to the anterior horizontal limb of the Sylvian fissure, and *H K* indicates the posterior horizontal limb of the fissure of Sylvius. This posterior limb of the Sylvian fissure terminates at the parietal eminence in the triangle *K O L* at the level of the temporal crest, indicating the position of the supramarginal convolution. The point *T* is the termination of the Sylvian line, and is somewhat in advance of the *parieto-occipital fissure*. The triangle *T V I* corresponds to the outer surface of the occipital lobe of the cerebrum. The line *I V* about corresponds to the tentorium cerebelli and the upper margin of the *lateral sinus*, while the line *V T* is a little above the *lambdoid suture*. The upper portion of the line *K N* corresponds to the *superior postcentral fissure*, but the lower part of the line is somewhat behind the *inferior postcentral fissure*; the line *K N* may be called the *postcentral line*.

The parallelogram *H K N M* overlies the ascending parietal and ascending frontal convolutions separated by the *central fissure* (fissure of Rolando), and hence corresponds to the cortical motor area (*area of Rolando*). The pentagon *F E V L H* outlines the *temporal lobe* of the cerebrum, except the apex; the latter is directed forward, downward, and inward about 18 mm. (a finger's breadth). About 18 mm. below the line *H L* is the superior temporal fissure (*parallel fissure*), which turns upward and terminates at *L*, thus indicating the position of the *angular gyrus*.

By drawing a line on the scalp from a point 12 mm. behind the mid-sagittal point (*M*) downward and forward for 8.5 cm. and at an angle of 67° to the sagittal line, the *fissure of Rolando* will be mapped out.

The inion *I* corresponds to the *torcular Herophili* and thus overlies the point of junction of the superior longitudinal, straight, lateral, and occipital sinuses. The groove for the lateral sinus may be mapped out by drawing a line from a point 1 or 2 mm. above the inion, in a slightly curvilinear direction with the convexity upward, to the point *V* at the postero-inferior angle of the parietal bone. The point *V* forms the highest part of the convexity of the sinus. From this point the upper margin of the sinus follows the line *E V* for 25 mm.; it then curves forward and downward to a point 18 mm. below and behind the centre of the external auditory canal. The anterior border of the mastoid portion is indicated by the line *Y Z*.

The groove for the superior longitudinal sinus extends from the glabella to the inion. It increases in size from before backward and usually becomes continuous with the right lateral sinus. The edge of the trephine should be maintained at least 18 mm. from the mesial plane when the skull is opened over the posterior part of the vertex.

After the *middle meningeal artery* enters the middle cranial fossa through the foramen spinosum it runs outward and forward for about 37 mm. to the point *F*, which is located about a finger's breadth (18 mm.) above the mid-point of the zygomatic arch. It here divides into anterior and posterior divisions. The anterior division passes, with a slight convexity forward, across the pterion upward and slightly backward behind the coronal suture. It gives off branches which ascend over the Rolandic area corresponding to the parallelogram *H K N M*. This anterior branch corresponds to the lower two-thirds of the precentral line *H M*. In trephining over the lower part of the Rolandic area, especially over the cortical motor centres for the face and tongue, this anterior division will be encountered.

The *posterior division* of the middle meningeal artery passes backward almost horizontally toward the postero-inferior angle of the parietal bone. It may be indicated on the exterior of the cranial wall by drawing a line backward from the point *F* (which is 18 mm. above the mid-point of the zygomatic arch) parallel to the line *E V*.

When the calvarium is removed in the recent state the meningeal arteries will be found intimately adherent to the dura mater. The middle meningeal artery is the only one of them that is of surgical importance. In fractures of the skull the artery is frequently ruptured; the extravasated blood will generally be found between the cranial wall and the dura mater, and beneath the clot will be found the bleeding point of the artery.

To expose the anterior division of the artery the point of the trephine should be applied over the Sylvian point *H*.

To expose the posterior division of the artery the point of the trephine may be applied a finger's breadth (18 mm.) above the zygomatic arch and the supramastoid crest between the points *E* and *V*.

To expose the main trunk of the meningeal artery between the foramen spinosum and the point of bifurcation at *F*, the trephine is applied immediately above the mid-point of the zygomatic arch. When the anterior division of the meningeal vessel is ruptured the extravasated clot presses upon the temporo-parietal portion of the cerebrum and induces motor symptoms through pressure upon the centres for the face and arm; when the *frontal* branch of the meningeal's anterior division is wounded, the clot of blood is in the temporo-frontal region of the cerebrum and produces pressure symptoms in the motor area for the face, and in addition, on the left side, involves Broca's convolution. Wounds of the meningeal's posterior division involves the occipito-parietal region and the pressure symptoms are sensory.

To reach the lateral hemisphere of the cerebellum the point of the trephine is placed over the mid-point of a line drawn from the inion to the tip of the mastoid process. In turning down the flap for this operation the mastoid emissary vein and the occipital vessels will be divided.

To tap the lateral ventricle at the commencement of its descending horn the point of the trephine should be placed a finger's breadth (18 mm.) below the mid-point of the line *K L*; in this operation only 1 cm. of brain tissue is penetrated through the posterior half of the first temporal convolution.

The asterisk at *C* overlies the site chosen by Keen for tapping the lateral ventricle; he makes an opening through the cranial wall 31 mm. above Reid's base line and the same distance behind the external auditory canal; he then passes the instrument into the brain tissue toward the summit of the auricle on the opposite side. The undistended ventricle will be reached at a depth of 5 cm. from the surface.

The asterisk at *B* indicates the site selected by Kocher for draining the lateral ventricle. He makes an opening two fingers' breadth (31 mm.) in front of the point *P*, the instrument being directed backward and downward through the superior frontal fissure for a depth of 4 or 5 cm.

The asterisk at *A* overlies the site at which the point of the trephine is entered for draining the subarachnoid space; a small trephine should be used and the operator should be careful to keep in front of the middle meningeal artery.

The cisterna magna is reached by trephining a little above the foramen magnum and a little to one side of the external occipital crest so as to avoid the occipital sinuses.

Daniel Kerfoot Shute.

SKULLCAP.—(*Scutellaria*, U. S. P.). The dried herb, *Scutellaria lateriflora* L. (fam. *Labiatæ*). This genus consists of bitter perennials, nearly destitute of the aromatic properties found in most species of this large family, and further distinguished by a peculiar helmet-like development of the upper sepal, to which it owes its name. Calyx two-lipped, persistent; closed-in fruit until maturity, when it splits and opens widely. Corolla bilabiate, ascending; stamens four, also ascending, and under the upper lip of the corolla; lower anthers one-celled. Leaves opposite, petiolate; flowers axillary, usually solitary, sometimes in apparent spikes or racemes. There are nearly two hundred species, forming a very distinct and natural genus, distributed over nearly the whole north temperate zone. There are about a dozen in the United States, and several of them are used like the official, from which they are distinguished in trade by the appellation "Western skullcap."

The official drug is thus described:

Dark green, smooth, or slightly puberulent on the younger portions, the branches elongated, slender, sharply quadrangular; leaves opposite, exstipulate, shortly petioled, the blades rarely exceeding 8 cm. (3 in.) long and about a third as broad, ovate, rounded or truncate at the base, acuminate, obtusish or acutish, shortly and obtusely serrate, thin, veiny; flowers in axillary, peduncled, mostly simple and secund racemes, nearly sessile, about 6 mm. (¼ in.) long, the calyx bilabiate, in fruit becoming closed and developing a large helmet-shaped crest, the corolla deep blue, bilabiate, the stamens didynamous; fruit of four depressed, globose, papillose nutlets.

So-called "Western scutellaria," more or less rough gray-hairy and with much larger flowers, in terminal panicles, is not of this species.

Skullcap contains a very little volatile oil, and its bitter principle has been called *scutellarin*.

The Scutellarias have had from time to time some transient or popular reputation in medicine for the cure of mad-dog bites, chorea, epilepsy, or other nervous diseases; also as tonics, antiperiodics, etc. The official species is considerably used by the eclectic school of prac-

titioners and in home medication. It is reputed to be antispasmodic, anticonvulsant, etc., and is given for restlessness and wakefulness. It certainly possesses bitter tonic and mildly laxative properties.

W. P. Bolles.

SLEEP. See *Drowsiness.*

SLEEP, DISORDERS OF. See *Insomnia.*

SLEEPING SICKNESS.—Definition.—Sleeping sickness, or negro lethargy, is a disease of the central nervous system—a meningo-encephalomyelitis—endemic in Western equatorial Africa; characterized by gradually increasing drowsiness and lethargy, both mental and physical; and, after a period of about a year, terminating in death.

Synonyms.—Negro Lethargy, Sleeping Sickness of West Africa, Sleeping Sickness of the Congo, Congo Sickness, African Lethargy, Sleeping Dropsy, Lalaregolo, Lalangolo, Manungina, Nélavane, Ntolo, Dâdane, Somnolenza, Hypnose, Hypnosie, Maladie du Sommeil, Maladie des Dormeurs, Die Schlafkrankheit der Neger, Enfermedad del Sueno.

Geographical Distribution.—This disease is endemic on the western coast of Africa, between the River Senegal in the north and Loando in the south, a distance of about fifteen hundred miles. It is found on the coast, inland, and on the islands; how far inland it spreads has not been ascertained, but it may be safely said to be endemic in the whole of the region drained by the Senegal, the Niger, and the Congo. Outside of this region it has never been known to originate. Those who have resided within this area, and have left it, are liable to be attacked by the sleeping sickness at any time within six or seven years, so long is the latent period that has been assigned to this disease. Those cases of sleeping sickness that have been reported from other parts, as the Bahamas, Brazil, British Guiana, the French West Indies, etc., either have been imported from the endemic region or else are not sleeping sickness at all, but most probably ankylostomiasis. Thus "Guérin in an experience of one hundred and forty-eight cases at Martinique never saw the disease in a negro who had not been imported from Africa, nor in one who had been resident on the island for more than ten years" (Manson, in "Allbutt's System of Medicine"). Within the area of its endemicity sleeping sickness rages in certain places and at certain times as an epidemic. The spots thus unfortunately visited are selected with a capriciousness that defies all explanation. Settlements, villages, or garrisons are suddenly attacked with the epidemic; all who can, leave the infected place; the others remain—and die. Other spots in the neighborhood are passed by, at any rate for the present; later on their turn comes, and another district is desolated and decimated. Further, some districts are constantly attacked; and others, in fairly close proximity, as constantly and inexplicably escape. Epidemics are very frequent in the Lower Congo region, while at Stanley Pool and at the mouth of the Congo they are almost unknown. From recent accounts it would seem as if the disease was spreading. Dr. Cook (*Jour. of Trop. Med.*, iv., p. 236) reports cases occurring at Uganda on the Upper Nile; and Dr. Castellani has witnessed it raging among the natives in the Victoria Nyanza district. According to this observer, fifty thousand natives died of this disease in one year (see also the London *Hospital* of December 27th, 1902). Sleeping sickness is almost entirely confined to the negro race; the mulatto and the Moor have been attacked, but it has not yet been observed in a European. Whether this is due to hygienic precautions and conditions, or to something inherent in the disease, is at present unknown, and is likely to remain so until the etiology of sleeping sickness is settled.

Etiology.—The cause of sleeping sickness is not known. The disease has, in turn, been attributed to a variety of causes, some of which need only be mentioned to be rejected. For example: 1. *Sunstroke* has been put forward as the cause; but whether the sun was hotter or the negro's skull thinner in the endemic region was never explained; nor were the peculiar epidemic outbreaks so characteristic of the disease. If sunstroke had been the exciting cause, sleeping sickness would have had a much wider area of distribution. 2. Various *foods* and *intoxications*, as fungus, hemp, maize, the bitter manioc or cassava. When properly cooked these are said to be non-injurious; but when eaten raw it is claimed that they are liable to produce the disease. But these plants are found outside of this region, and are just as liable to be eaten uncooked in other districts; hence as an etiological factor they have not much value, although some analogy may be claimed to pellagra and lathyrism. 3. *Emotional causes*, such as nostalgia, pining under ill-treatment when away from home; and yet it has never been asserted, much less proved, that the negro from western equatorial Africa was either worse treated than others, or that he was more tender-hearted than his brother from other parts. 4. Various *microbes* have, of course, been suggested, but, unfortunately, the observers do not agree among themselves.

Bettencourt thinks that the cause of the sleeping sickness is a micrococcus; Cagigal and Lepierre did not find the micrococcus, but report a bacillus; Dr. Broden also found a bacillus. Marchand thinks that the disease is due to the pneumococcus; Drs. Mott and Bulloch found no micro-organisms at all; but, of course, this does not preclude the possibility of the disease being due to a specific organism, for, as in the case of syphilis, the organism may be there, but so far may have eluded the bacteriologists.* 5. Among the *parasites* that have been put forward as the probable cause are *Filaria perstans* (Manson), *Rhabdonema strongyloides* (Le Dantec), and *Ankylostomum duodenale* (Ferguson). Of these, Manson's suggestion of the Filaria perstans is by far the most probable. Manson has shown that the geographical distribution of the Filaria perstans is the same as that of the sleeping sickness; and further, the Filaria perstans has been found in an exceedingly large proportion of the cases in which any proper examination has been made; the latent period and the peculiar endemicity are both capable of explanation on this theory. Probably there is something else requisite besides the Filaria perstans, for many negroes have this latter and yet escape the sleeping sickness; so far, Manson only puts it forward as a hypothesis, but it is quite the most reasonable that has appeared. 6. "Like all tropical pathological puzzles, sleeping sickness has been attributed to *malaria*—that blessed cloak for ignorance—but there are none of the clinical or pathological marks of malaria about the disease" (Manson, *Jour. of Trop. Med.*, i., 125). 7. Dr. Andriezen has pointed out how very similar the symptoms of sleeping sickness are to those produced by destruction of the pituitary gland. Whether the pituitary gland is a causative factor in sleeping sickness is not known. 8. Crombie suggests that the deep cervical glands, which receive the lymph from the brain, may, by becoming obstructed by the Filaria perstans or its ova, cause sleeping sickness. 9. The virus, whatever it may be, is said to be conveyed by the saliva, the negroes using their fingers to eat with out of a common dish.

Symptoms and Course of Disease.—Before describing the symptoms of sleeping sickness it may be well to remember that the negro is naturally prone to sleep. To those who have lived for any length of time in the tropics, the following quotation from Scheube's "Diseases of Warm Climates" will appear to be a remarkably mild statement of fact: "The negro works—when forced to—but with frequent interruption; he eats and drinks, sings, laughs, perspires; he capers and dances with extreme gayety—or he sleeps. When unemployed he cannot keep himself awake without a noise. It is therefore customary in the negro schools of the West Indies to oblige the unemployed half of the scholars to sing hymns while the other half receive instruction. If this were not done half of the scholars would be asleep" (Junker von Langegg). Neither *sex* nor *occupation* has any influence either as regards liability to the disease or immunity from it; all *ages* are susceptible to it, chiefly the young and vigorous adult from about fifteen to twenty years; in infants, and in those over forty years of age, the disease has not been noted.

Sleeping sickness begins very gradually with general weakness and languor, enfeebled powers of endurance, debility both muscular and intellectual, a more than ordinary disinclination to work coupled with an almost irresistible longing to doze or sleep, and this latter shows itself not only during the hours of labor, but even at meal-times. With this muscular fatigue and weakness there is also a corresponding mental dulness and vacuity, so that in place of his usual vivacious disposition the afflicted negro gets morose and silent, does not initiate conversation and talk for the mere love of talking as is his wont; but at first he will only answer questions, then only in monosyllables and when pointedly and repeatedly interrogated; later on, it is only after considerable lapse of time that an answer can be extracted from him, thereby showing the slowness and impairment of the mental processes. Headache is often one of the prominent early symptoms, though it is by no means an invariable one; when present, it is generally occipital.

Vertigo is often present. The patient has a shuffling, staggering gait, as if half asleep. The face is puffy; the upper eyelid droops, though whether as the result of drowsiness or from paralysis of the levator palpebræ superioris is not known. The lower lip also droops, showing the lower teeth and allowing the saliva to dribble and almost flow out of the mouth. The state of torpor increases, sometimes punctuated by brief snatches of seeming improvement which are not only short-lived, but generally lead to worse relapses, so that work of any kind becomes absolutely impossible and the patient does little but sleep; food is taken if brought to the patient, and if he is kept awake while eating it; otherwise he is very liable to fall asleep while a bolus is making its way from his tongue to the œsophagus. He lies around anywhere and in any position, even the most uncomfortable, and sleeps. If left alone now he would starve. Even now under proper care and attention it is more than probable that nutrition will fail, bedsores form, and diarrhœa and epileptiform seizures come to add to his generally miserable condition. The skin is generally cold, though the temperature may rise to 101° or 102° F., or even higher. There is enlargement of the lymphatic glands, chiefly the cervical; but the supraclavicular and occipital glands are often noticeable too. These enlarged glands vary in size from a small bean to a walnut, are tender on pressure, are discrete, do not adhere to the skin, and have no tendency to suppurate. The skin suffers from loss of the peculiar gloss and lustre which is noticed in health; it is also liable to be covered with an eruption, either papular, vesicular, or pustular; sometimes it may have the appearance of an exanthem. Such eruption is most common on the chest and abdomen. There is a very pronounced pruritus, and the consequent scratching leaves whitish lines of abraded epithelium on all accessible parts of the skin. Muscular tremors and light fibrillar twitchings are noticed.

Sensation is not much affected, except toward the end of the disease, when it may be diminished. The secretions are fairly normal, except the saliva, which constantly flows or dribbles out of the mouth; but whether it is a hypersecretion or inability of the patient to swallow, is not settled. Insanity is apt to supervene before the end. The superficial reflexes are normal and the knee-jerk is normal or increased. Choreic attacks have been observed, with tonic or clonic convulsions, and in severe cases epileptiform attacks. Muscular spasms are most apt to affect the flexors of the limbs and the sterno-cleido-mastoid. The above are the chief symptoms that have been noted, but it must be understood that no case presents *all* the symptoms, and there is considerable variation in their presentation.

Duration.—The disease may last anywhere from three months to three years; about one year is the average time.

Diagnosis.—There ought to be but little difficulty in diagnosing sleeping sickness; the geographical distribution, the progressive somnolence and bodily weakness, and the pruritus present a complex that is not afforded by any other disease. It has been confounded with beriberi, but the following table, compiled from Manson (Allbutt's "System of Medicine"), will show the differences:

Sleeping Sickness.	Beriberi.
1. Diseases of the central nervous system.	1. Disease of the peripheral nerves.
2. Progresses slowly.	2. Progresses rapidly.
3. Nearly always fatal.	3. Rarely fatal.
4. Limited endemic distribution.	4. Extensive distribution.
5. Not a peripheral neuritis, and not associated with marked œdema, although in the earlier stages there may be puffiness of the face, and in the later stages puffiness of the feet.	5. Has all the signs of a peripheral neuritis, and generally is accompanied with more or less œdema.
6. Muscular atrophy of rapid development, palpitations, abolished knee-jerk, and hyperæsthesia of muscles not found.	6. Torpor, tremor, insanity, and itching papular eruption not found.

Prognosis.—The disease is almost invariably fatal. Guérin reports 148 cases with 147 deaths; Gore 179 cases with 132 deaths; Forbes 13 cases with 11 deaths (and the termination of the other two not known).

Pathology and Pathological Anatomy.—Although sleeping sickness has been known for a little over a century, it is only in the last few years that anything has been learned about its pathology. Among the few notices that make any direct reference to the pathological anatomy, there is one that stands quite alone for its completeness and utility; this is a report of two cases (including autopsy) by Dr. F. W. Mott, director of the Pathological Laboratory of the London County Asylums. We here quote from his report in the *British Medical Journal* for 1899, i., p. 1108:

"The brain and spinal cord, pituitary body, and spinal ganglia were examined. To the naked eye the tissues in Case I. presented but little change beyond some slight thickening of the pia-arachnoid. The cerebral convolutions were complex and not atrophied; the brain weighed fifty-four ounces. The two hemispheres were of equal weight and there was no excess of fluid. In Case II. (the younger patient) the dura mater was found adherent to the calvaria. A considerable quantity of cerebrospinal fluid was present. The pia-arachnoid was somewhat thickened and opaque over the convolutions. The base of the brain likewise showed thickening and opacities of the pia-arachnoid. The weight of the brain was thirty-six ounces. Neither of the brains showed flattening of the convolutions, erosions on stripping the membranes, or dilated ventricles with granular ependyma. The nervous tissues before mentioned were removed so soon after death as to avoid post-mortem fallacies. Portions of different parts of the hemispheres, cerebellum, pons, medulla, and cord, as also the spinal ganglia, were stained by the Nissl, Marchi, and Marchi-Pal methods after suitable fixation. The microscopical examination of these sections exhibited in both instances similar conditions. There was a leptomeningitis and an encephalomyelitis. Throughout the whole central nervous system, but especially in the medulla and base of the brain, sections showed all the perivascular limits distended with mononuclear leucocytes. . . . Sections were also stained for micro-organisms by Gram's, Pfeiffer's, and other methods, but with negative results. . . . The general and special appearance of the nerve cells was as follows: In Case I. the outline of the nerve cells and their arrangement appeared fairly normal; neither was it considered that the

* As this goes to press, Dr. Castellani, an Italian physician residing at Uganda, claims to have discovered the microbe of the disease, viz., a new variety of streptococcus. This same observer has since reported the finding of a species of Trypanosome in the blood and cerebro-spinal fluid of patients suffering from sleeping sickness. This has also been verified by Colonel Bruce, who found a trypanosome in the blood of twelve out of thirteen cases of sleeping sickness, and in the fluid obtained by lumbar puncture in every one of thirty-eight cases. ("Brit. Med. Jour.," 1903, i., 617 and 1218.)

neuroglia cells were markedly increased. The columns of Meynert in the cortex cerebri were distinctly evident, thus contrasting with the appearance of the brain in general paralysis. The cells themselves throughout the whole nervous system showed a uniformly dull, diffuse, staining reaction, and in none of the cells were the Nissl granules evident. This change was undoubtedly due to the hyperpyrexia during the last hours of life. In Case II. the cells for the most part presented a normal outline and exhibited Nissl granules on the dendrons and in the body of the cell. In the medulla, however, a considerable number of cells showed chromolytic changes, and to a less degree changes were found in the motor cells of the anterior cornua. The cells in the left hemisphere showed degenerative changes in sections of the motor area, Meynert's columns were not distinctly visible, and many of the cells seemed atrophied and broken up. Fibres: Sections of the brain and cord were stained by Marchi and Marchi-Pal methods. Nothing abnormal was found in Case I. except perhaps that the tangential fibres were not so numerous as in the normal cortex cerebri. In Case II. there was obvious wasting of the tangential fibres in both hemispheres, but especially of the left. There was slight sclerosis of the crossed pyramidal tracts of the cord, more marked on the right side, and also a number of recently degenerated fibres were exhibited by the Marchi method. The arteries of the central nervous system exhibited no trace of endarteritis. In the choroid plexus there were numerous microscopical psammomata. The central canal of the spinal cord was filled up with proliferated glia tissue. The posterior spinal ganglia showed the same appearances around the vessels, but the ganglion cells in Case I. only showed the diffuse staining of hyperpyrexia, and in Case II. exhibited a fairly normal appearance. . . . The symptoms which were, however, present in both patients, and characteristic of the disease—namely, progressive drowsiness and lethargy, and the progressive weakness in body and mind, without any distinct paralysis or mental disability—could best be accounted for by supposing that the metabolism or functional activity of the neurons as a whole was affected injuriously by some toxic product either circulating in the blood or existing in the cerebrospinal fluid; that this toxic agent, whatever it might be, occasioned great proliferation of mononuclear leucocytes beneath the pia-arachnoid and in the perivascular lymphatics. It might, however, be supposed that the functions of the nervous system were affected by an interference with their nutrient lymph supply, owing to the perivascular lymphatics being filled with leucocytes. The liver, kidneys, lungs, pituitary body, spleen, lymphatic glands, and duodenum were also examined. The results were for the most part, with the exception of the duodenum and lymphatic glands, negative. The lymphatic glands were much enlarged owing to great increase of lymphocytes. Sections of duodenum showed a large number of lymphocytes, and a proliferation of the same in the lymphoid nodules."

In addition to the above, Dr. Mott furnished a much fuller report, with illustrations, to the *British Medical Journal* for December 16th, 1899. To this the reader, in search of further details, is referred.

Treatment.—This has, so far, been most unsatisfactory. Purgatives, tonics, quinine, and arsenic have all been tried, but without much effect on the death rate. Among the newer suggestions are: (1) Hypodermic injections of testicular extract; (2) venesection of about one-fourth of a litre per week, followed by injection of an artificial serum, the object being gradually to free the blood from the Filaria perstans; (3) thyroid extract; (4) electricity over the spine. Probably the best that can be done will be found along the lines of hygiene, nursing, and feeding. *R. J. E. Scott.*

SMALLPOX.—Definition.—Smallpox is an acute, contagious, febrile, exanthematous malady, characterized by an eruption which passes through four stages of development, *i.e.*, macule, papule, vesicle, and pustule. The initial symptoms are, ordinarily, chill, fever, headache, lumbar pain, and vomiting. The fever disappears when the eruption appears, and recurs when the latter has reached the pustular stage. The period of incubation varies from seven to twenty-one days.

History.—Though an ancient disease, its origin is unknown. DeHaen, Willan, Moore, and Barron contend that it was known to the ancient Greeks and Romans; Friend, Mead, Good, and Adams deny this assertion. Rhazes, an Arabian physician, who practised medicine about the year 910 A.D., was one of the most celebrated of the earlier writers on smallpox. When we consider the date of his writings and the state of medical knowledge at that time, it will be seen that he delineated the natural history of the disease with remarkable accuracy. Rhazes contends that Galen was familiar with the malady, and cites extracts from his first, fourth, ninth, and fourteenth books as evidence of the fact. He also mentions Ahrún, of Alexandria, and Mesue, of Bagdad, among the other early writers on variola. It is, however, commonly agreed among historians of medicine that this disease cannot with certainty be traced to a period anterior to the Christian era. Since smallpox first attacked mankind it has never wholly disappeared, and from Europe and Asia has been carried all over the world. It appeared in England in the first part of the thirteenth century, and in Germany in the latter part of the fifteenth. It was imported into the United States soon after the discovery of America. It reached Mexico in 1527. Smallpox is a malady to which the human race is wellnigh universally susceptible, probably not more than one in a thousand persons being naturally immune. In consequence of the universal susceptibility of unvaccinated persons, and the neglect of vaccination in almost all communities, smallpox smoulders in different localities, and at varying periods breaks loose from these endemic foci and assumes epidemic proportions in municipalities, States, and nations.

Like all other contagious maladies epidemics of smallpox differ markedly one from another, not only in the amount and character of the eruption, but in the character and intensity of symptoms, and in mortality as well. For centuries prior to Jenner's discovery of vaccination (1796) smallpox was regarded as the king of fatal diseases. M. de La Condamine, writing of this malady, says that it was the cause of one-tenth of all the deaths among mankind. He also says: "Among those who outlive it, many, either partially or totally, lose their sight or hearing; many are left consumptive, weakly, sickly, or maimed; many are disfigured for life by horrid scars, and become shocking objects to those who approach them. Immense numbers lose their eyesight by it." Black, Frank, and other reputable writers on this disease state that it caused half a million deaths annually in Europe prior to general resort to vaccination. The great English historian Macaulay, in speaking of this malady in England, says: "The havoc of the plague had been far more rapid, but the plague visited our shores only once within living memory, but the smallpox was always present, filling the churchyards with corpses, leaving on those whose lives it spared the hideous traces of its power, turning the babe into a changeling at which the mother shuddered, and making the eyes and cheeks of the betrothed maiden objects of horror to her lover."

Rosen says that one-tenth of the deaths in Sweden resulted from smallpox. Simon says that this disease concurred with fire, and sword, and famine to depopulate St. Domingo.

In an epidemic of smallpox in Mexico in the sixteenth century 3,500,000 of the inhabitants died in a few years, leaving in some places scarcely enough people alive to bury the dead. Prescott ("Conquests of Mexico," vol. vi.) describes this epidemic as "sweeping over the land like fire over the prairies, smiting down prince and peasant, leaving its path strewn with the dead bodies of natives who perished in heaps like cattle stricken with the murrain." Smallpox invaded Brazil in 1653, and in some instances whole races of men died of the malady.

In a few years the Province of Quito lost 100,000 of her Indian population by this disease. Iceland had been invaded by smallpox seventeen times prior to 1707, and desolation and ruin followed in its wake for years, causing in the year 1707 the death of 18,000 out of a total population of 50,000. The terrible fatality of the disease in Greenland in 1707 is evidenced by the following extract from Crantz's "History of Greenland": "Empty, depopulated houses and unburied corpses, some within and some without the houses, were commonly encountered. In one island they found one girl with the smallpox on her, and her three little brothers; the father, having first buried all the people in the place, had laid himself and his smallest sick child in the grave, raised with stones, and ordered the girl to cover him." In 1734 Greenland lost two-thirds of her population from smallpox. From authentic sources it is learned that in one epidemic of this malady one-sixth part of the inhabitants of Ceylon died. Siberia had a similar experience, and Kamchatka has suffered in like manner. Captain Cook ("Voyages to Pacific Ocean," 1785), speaking of the first appearance of smallpox (1767) in Kamchatka, describes it as "marking its progress with ravages not less dreadful than the plague, and seeming to threaten their extinction." M. de La Condamine says that prior to vaccination one-tenth of all the deaths in France were from smallpox. In 1805 M. Laborde says: "I had been a witness of the variolous epidemic which had, in 1792, swept off one-fourth of the population of the Isle of France." North America has been fearfully scourged by smallpox; whole tribes of our Indian population were almost literally extinguished by it. McKenzie says of the disease among the Indians: "It was as a fire consuming the dry grass of the field. The infection spread with a rapidity which no flight could escape, and with a fatal effect which nothing could resist." Godfrey says that 2,000,000 of the inhabitants of the Russian empire died of smallpox in a single year. Sir Gilbert Blaine says: "When there was no vaccination in our navy, one-fifth of all the men enlisted died of smallpox." Mr. Makena describes an epidemic of this disease in the Argentine Confederation from 1846–48, as "sweeping with the wings of death over that enormous tract of country from the seaboard of the Atlantic on the East to the cordillera of the Andes on the West." Alexander Wheeler, of England, an ardent antivaccinist, says: "In 1875 smallpox carried off 101,397 inhabitants of India." Hirsch says that between 1866 and 1869 smallpox killed 140,000 natives in Bombay and Bengal. In the whole of India (1873–74) 500,000 inhabitants died of this disease.

An exhaustive account of the various epidemics of smallpox is, of course, not to be expected in a limited article. The instances above quoted are presented in order to show how malignant smallpox commonly was in prevaccinal times, as well as the present, in a population unprotected by vaccination. The instances cited are entirely authentic, and justified Macaulay in styling smallpox "the most terrible of all the ministers of death." It, however, would be wholly erroneous to contend that all epidemics of smallpox had been as pernicious as the ones above cited. It is an incontestable fact that throughout the entire written history of this malady individual epidemics have differed markedly one from another, not only in mortality, but in the quality and quantity of the eruption and the other attendant symptoms. In some epidemics a large percentage, and in others the largest percentage, of cases of smallpox were of the mildest type—varioloid as it is called—and this resulted, not, as some text-writers assert, in consequence of previous vaccination or a prior attack of smallpox, but from an inexplicable partial natural immunity of populations more general at one period than at another, for this disparity of different epidemics as to mortality, quality, and quantity of eruption, and other symptoms was noted prior to vaccination. I cite three instances from a number at hand. George Cleghorn, M.D., in his book on the "Epidemic Diseases of Minorca," says: "The smallpox were twice epidemical in Minorca while I resided there, viz., in 1742 and 1746. About the middle of March, 1742, the smallpox broke out in Minorca, to the great consternation of the natives who had not seen them since the year 1725, but well remembered the destruction which they then occasioned. During the first six or eight weeks (1742) the distemper was favorable and seldom proved fatal; but its virulence increased with the heat of the weather, so that in June and July it was not uncommon, both at Mahon and Ciudadella, to bury ten or twelve a day. Nevertheless, in proportion to the numbers not many died, and what mortality there was happened chiefly among children at the breast and the common soldiers." Of the epidemic of 1745–46 he says: "Then they (the smallpox) travelled northward to Ciudadella, and disappeared in the spring, having carried off almost all of the children who survived the chincough and the summer fevers. It was, however, very remarkable that the longer the infection continued in the island the milder it became, so that there was much less mortality in the northern part than in the southern, where it first broke out." He then cites the fact that in 1846 at St. Phillip's Castle three-fourths of the smallpox patients died.

In the early part of 1898 smallpox appeared in several of the Southern States. The first case in this city (Augusta, Ga.) was detected May 5th, 1898, the patient having contracted the disease in South Carolina. The malady spread from one State to another until it invaded all sections of the United States. The disease is said to have been brought to this country from Cuba, it having been prevalent there during the Spanish-Cuban war. In a large number of communities it assumed epidemic proportions by reason of the fact that physicians failed to recognize its nature until it had attacked a large number of the inhabitants. Cases of the disease were observed by me in citizens of twelve counties in Georgia and four counties in South Carolina. In each and all of the communities in which I encountered the disease it was, in my experience, unprecedentedly mild and irregular both as to constitutional symptoms and quantity and quality of eruption, and equally strange was the further fact that while perhaps one-sixth of the cases were either confluent or semiconfluent in type, the vast majority of discrete and semiconfluent cases went on to and through the purulent stage without secondary or maturation fever. This fact was ascertained by repeated daily observations with the thermometer. Again, not more than five per cent. of confluent cases terminated fatally. In the confluent and semiconfluent cases pitting of the face was in about the percentage usually attendant on severer epidemics. The disease was confined almost exclusively to the unvaccinated. No one of the four hundred and twenty-nine cases coming under my observation during the last four years had a typical vaccine scar. Ten of them had a scar, and claimed to have been vaccinated from ten to twenty years previously, but in no one of them could I say from the cicatrix that the patient had undergone vaccinia.

The initial symptoms of the malady were headache, lumbar pain, chill, fever, and vomiting, and these symptoms were as commonly complained of by those having the disease mildly as by those who had the severer types. In those who had the disease mildly, *i.e.*, about two-thirds of the cases, the constitutional symptoms disappeared wholly when the eruption appeared, and these patients then went to work or visited their friends. I found a large per cent. of them at work in the fields, shops, and other industrial pursuits while the eruption of smallpox was in full bloom on them. Two of these patients after remaining at home three days resumed their work of delivering ice to our citizens. Others were apprehended on the public thoroughfares or in houses where they were visiting. These individuals claimed that their disease was chickenpox, and in not a few instances had been so informed by their physicians. In one case the total number of pustules on the patient's body was six, yet this patient gave rise to an epidemic of smallpox in the jail in our city. Another patient