VAGINAL MOVEMENTS — Dr. Jastreboff has made a series of experiments upon the vagina of rabbits, and arrived at the following conclusions :

I. In the wall of the vagina are automatic nerve-centres which cause rhythmic contraction of the separate segments.

2. A direct electric tetanization of the vagina causes peristaltic contractions.

3. Weak irritation of the central end of the sciatic causes antiperistaltic contractions.

4. Extreme anæmia causes an increase of the vaginal peristalsis. —Archiv für Physiologie, von Du Bois-Reymond, 1884, Heft 6.

IS THE NERVOUS IMPULSE DELAYED IN THE MOTOR-NERVE TERMINATIONS ?- Dr. Hoisholt has made a series of experiments upon this subject, using the nerves of frogs. His results were as follows : A preponderance of the maximum contraction by direct stimulation of the muscle. A great difference in the latent period by stimulation of the nerve-trunk near the hilus, and of the muscle-substance rich in nerves. This is ascribed by Bernstein to a delay in the process of stimulation at the motor-nerve terminations, where the amount of latent power set free is to accumulate until it equals the stimulus necessary for the muscle. More probably, however, it is dependent upon the summation of stimulus. A latent period by stimulation of nerveless musclesubstance, which, in some cases, was the same as that resulting from nerve stimulation, more frequently, instead of being shorter, was longer than the latent period obtained by stimulation of the nerve. Although this last paradoxical result remains inexplicable, the related experiments render at least an affirmative answer to the question heading this article highly improbable .- Fournal of Physiology, vol. vi., Nos. 1 and 2. ISAAC OTT, M.D.

## C.-GENERAL PATHOLOGY OF THE NERVOUS SYSTEM.

DOUBLE INFANTILE SPASTIC HEMIPLEGIA.—In the January number of the American Jour. of the Med. Sciences, Dr. S. J. McNutt reports a case of double infantile spastic hemiplegia, with carefully recorded notes of the post-mortem appearances, illustrated with seven cuts, exhibiting the lesions found. This is believed to be the third, or, at the most, the fourth, case of its kind upon record. Dr. McNutt has collected and tabulated thirty-four cases in which autopsies have been made, and each of them presented atrophy of the cerebral cortex, near the fissure of Rolando. The main features of the cases having autopsies are presented in the following table :

## PERISCOPE.

No.	Physician.	Sex and Age.	Seizure.	Symptoms.	Autopsy.	By whor reported a where.
r	Heschl,	M. 26 yrs	From birth left extremi- ties weak.	gia; contrac-	Central segment centrum ovale with convolutions pertain- ing to it absent on right side from the convexity to the fis- sure of Sylvius.	Die Poren
2	Heschl,	F. 7 yrs		Right hemi- plegia, with contractures.		Die Poren
3	Brechet,	F. 3 <sup>1</sup> yrs		Rigbt hemi- plegia, atrophy.	Lefthemisphere de-	Kundra Die Poren phalie, 18
4	Maschede,	F. 27 yrs		Left hemiple- gia.	Right parietal bone defective $\frac{1}{2}$ in. behind coronal suture, the opening $_3$ in. by $\frac{1}{2}$ to r in. Right hemi- sphere presents cav- ity in posterior half, Walls of connective tissue of neighboring convolutions rusty brown.	Kundra Die Porer
.5	Rogers,	M. 41 yrs	When 15 yrs unconscious 3 weeks; recov- ered with par- alysis of left leg and arm.	Left hemiple- gia with atro- phy.	Auterior half of	Kundra Die Porer phalie, 18
6	Rogers,	M. 49 yrs	Dates from convulsions in youth.	Right hemi- plegia; weak- minded.	has a cavity connect- ed with ventricle.	Die Porei
7	Hugel,	F. 5 yrs	Asphyxiated at birth.	Left hemiple- gia.	Right hemisphere presents an excava- tion in anterior half	Kundra Die Pore
8	Brodowski,	F. 12 yrs		Right hemi- plegia; strabis- mus, nystag- mus.	1½ x 2 x 3 in. Communication be- tween fissure of Syl- vius and ventricle, Surrounding convo- lutions converge into this cavity.	Die Porer phalie, 18
'9	Kundrat,	M. 15 mc		Left hemiple- gia, contrac- tures.	Right hemisphere atrophied, especially about fissure of Ro-	Die Pore
10	Kundrat,	F. 5 yrs	1 year be- fore death.	Double hemi- plegia ; idiocy.	er § fissure of Rolan- do; right more af- fected than left. Sul- cus communicates with lateral ventricle Septum pellucidum labsent.	Die Porer phalie, 18
11	Sperling,	F. 29 yrs	Difficult la	birth; shorten ing of the arm and leg, con	Depression behind fissure of Rolando including the ascend- ing parietal convolu- ftion; cicatricial tissue and pigment.	Die Pore phalie, 1
12	Cazauvieilh	59 yr:	5	Left hemiple gia, contrac- tures; sensibil- ity and intellec not impaired.	rigbt hemisphere less developed than left.	<i>Méd.</i> , 18 xiv., p.
13	Cazauvieill	F. 51 yr:	5	Right hemi- plegia, includ- ing face; righ	Convolutions lef hemisphere less de veloped than right intellect obtuse.	Cazauvie Arch. Gér Mêd., 18 xiv., p.

No.	Physician.	Sex and Age.	Seizure.	Symptoms.	Autopsy.	By whom reported and where.
14	Cazauvieilh	F. 42 yrs		Left side par- alyzed and un- developed.	Right hemisphere atrophied.	Cazauvieilh, Arch. Gén. de Méd., 1827,
15	<sup>i</sup> Cazauvieilh	F. 30 yrs		Left half of bodyemaciated, especially leg; mouth drawn to right side. Epi- leptic; vora- cious appetite; intellect ob-	defective.	xiv., p. 5. Cazauvieilh, Arch. Gén. de Méd., 1827, xiv., p. 5.
16	Cazauvieilh	F. 68 yrs	From birth.	mouth drawn to	In posterior part of left frontal lobe, a cavity having an "ac- cidental" opening in- to the ventricle.	Cazauvieilh, Arch. Gén. de Méd., 1827, xiv., p. 5.
17	Cazauvieilh	F. 27 yrs		Right hemi- plegia with atrophy.	less prominent than right.	Med., 1827,
18	Morgagni,	•••		Hemiplegia.	base, in frontal lobe, most marked in me-	Cazauvieilh, Arch. Gén. de
19	Little,	18 yrs	Instrumen- tal delivery; mother died.	plegia with atrophy y and contractures.	sphere atrophied; surface of right hemi- sphere cicatrized with	Little, Trans. Obst. Soc., London, 1862.
20	Gihb,	Still- born.	an accidental blow on ab- domen by a	Rigid contrac- tures of joints of limbs of left side, without breaking ten- dons could not	ecchymosed; remains of old clot in right hemispheres above ventricle.	NOV TO.
21	Pullain,	F. 8 yrs	board.	be extended. Atrophy right extremities ; in- t e 11 i g e n c e small.	Left hemisphere smaller than right.	Gerhardt's Hdb. Kinder- krankheiten,
22	Bourne- ville,	F. 16 yrs	At 16 mos, with spasms of right ex- tremities.	plegia, atrophy.	Atrophy of left hemisphere, especial- ly of ascending fron- tal, ascending parie- tal, paracentral lob- ule, and 1st frontal convolution.	Gerhardt's
23	Henoch,	F. 19 yrs	At 3 mos. convulsions, followed by paralysis.	lect pocr.	Middle upper part of left hemisphere occupied by cyst; right pyramid ½ nor- mal size; hematoidin crystals in wall of	Henoch, Hd. f. d. Kin- derkrankh., 1883, p. 231.
24	Henoch,	F. 12 yrs		Right hemi- plegia, atrophy,	cyst. All convolutions of left hemisphere small, rusty-brown color;	derkrankh.
25	Henoch,		Healthy to 1 <sup>1</sup> / <sub>2</sub> yrs. After carriage acci- dent suddenly paralyzed.	gia; destruc- tive tempera- ment speech	phied posteriorly; dense and white.	10031 p. 2311
26	Henoch,		paralyzed. Healthy to 6 months, then had measles with convul- sions 8 days, followed by general mus- cular rigidity.	tering, imbe- cility.	rst frontal convolu- tion on both sides atrophied, also zd, but in less degree. Corpus callosum, for- nix, and septum luci- dum atrophied.	Henoch, Hd. f. d. Kin- derkrankh., 1883, p. 231.

## PERISCOPE.

No.	Physician.	Sex and Age.	Seizure.	Symptoms.	Autopsy.	By whom reported and where.
27	Rénoy,	F. 4 yrs	From con- vulsions at birth.	gia, contrac- tures, stra- bismus; spoke badly, intelli-	hemisphere, especial- ly about fissure of Ro- lando, with secondary degeneration of pyra-	Rénoy, Progrès Méd 1879, p. 769.
28	Huebner,	2¥ yrs	When 15 months had fever with convulsions, followed by complete par- alysis.	Double hemi- plegia, contrac- tures of ex- tremities; little intelligence, no	midal tracts. Atrophy of both ascending convolu- tions of left hemi- sphere, with anterior parit of right inferior pariteal lobule; right lenticular nucleus. Anterior half of pons, with pyramidal tracts in it destroyed. Em- bolus found in right middle cerebral ar- tery from root to bi- furcation.	Huebner, Berl. klin. Wochenschr. 1882, p. 737.
29	Suckling,	•••	• • • •	Hemiplegia, contractures, atrophy, epi-	Atrophy about fis- sure of Rolando.	Suckling, <i>Bir. Med.</i> <i>Rev.</i> ,1883,p.55
30	Ross,	F. 2½ yrs	Congenital.	plegia · mono-	Sulcus occupying central convolutions on both sides; bot- tom sulcus opened into lateral ventride; pyramids and lateral columns small. No cicatriciat tissue, Giant cells of third layer absent in atro- phied part.	Ross, Dis. Nerv. Syst. 1883, vol. ii. p. 480.
31	Lambl,	F. 12 yrs		mus, nystag- mus; intelli- gence good;	Depression in left hemisphere occupy, ing fissure of Sylvius; lower part of ascend- ing convolution of island of Reil and an- terior part of first temporal convolution	d. Psych. u. Nervenheil- kunde.
32	Lachi,	F. 44 yrs	• • • •	weakness of left leg; atro-	terior part of fissure of Sylvius on left side; bottom of fissure opens into ventricle; septum lucidum ab-	Clinical, Feb
33	Blanchi,	M. 73 yrs	Convulsions in early in- fancy.	plegia, with weakness of left leg; atrophy	pying central convo- lutions of left hemi- sphere, communicat- ing with ventricle. Similar sulcus occu- pying superior third of central convolu- tions of right hemi- sphere. Paracentral lobule not affected. No descending de-	Bianchi, Abst. Am. 3 Neurol. ana Psychiatry.
34	McNutt,	F. 21 yrs	sented; in- strumental delivery; con- vulsions for 9	tures, dyspha- gia, and dysp-	generation. Atrophy about the fissure of Rolando on both sides. Atrophy affects most the base of convolution, giv- ing mushroom shape. Descending degen- eration of both pyra- midal tracts. Atro- phy of larynx with puckering of mucous membrane over vocal cords.	Amer. Jour Med. Sci., Jan., 1885.

A CONTRIBUTION TO JACKSONIAN EPILEPSY AND THE SITUA-TION OF THE LEG CENTRE .- Dr. William Osler, of the University of Pennsylvania, records, in the January issue of The American Fournal of the Medical Sciences, the history of an instructive case of Jacksonian epilepsy. His case lasted over fourteen years, the convulsions beginning in the left hand, at first monobrachial, then extending to the leg, afterwards becoming unilateral, and finally general; at first without loss of consciousness. For the last nine years of the illness there were remarkable intermissions, lasting for six or seven months, once an entire year. Six months after the onset the left leg got weak and stiff. For four years, the tenth, eleventh, twelfth, and thirteenth of the illness, the seizures were frequent. During this period there were six weeks of unconsciousness in which the spasms were very frequent, fifty to eighty in the day. Ten months prior to the final attacks there was freedom from convulsions. The intellectual faculties were unimpaired. The case is unusual in the limitation of the lesion to the ascending frontal convolution, and to its fasciculus of white matter, scarcely involving the gray substance, which is commonly affected in cortical epilepsy. The accurate localization, and the remarkable absence of tissue-changes in the immediate vicinity, give the case the nature of an exact physiological experiment. With this limited lesion of the motor area, there was permanent paralysis with contracture of one extremity and epileptiform convulsions. Another feature of interest in the case is the light it throws on the situation of the leg centre. The fibrous mass was situated entirely within the anterior part of the paracentral lobule. limited in extent, confined chiefly to the medullary fibres of the superior frontal fasciculus, and only touched the gray matter in places. A point to be referred to is the absence of the paralysis of the leg for the first six years, for, if the convulsions and monoplegia were caused by the same lesion, how explain the late onset of the latter ? From the fibroid state of the tumor it might reasonably be inferred that it was originally larger and had shrunk, but the absence of puckering on the surface, and the way in which the margins merged with the contiguous parts, make it probable that the growth was always small-so small, in fact, that at one period of its development, it may have caused sufficient irritation to induce the convulsions, and yet at the same time not involve the special fasciculi of white fibres to the extent of producing weakness of the leg or monoplegia.

A CASE OF LODGMENT OF A BREECH-PIN IN THE BRAIN; RE-COVERY.—Dr. G. W. H. Kemper, of Muncie, Indiana, reports in the January number of *The American Fournal of the Medical Sciences*, a very instructive case in which a lad received a compound fracture of the frontal bone, immediately above the right frontal sinus, by a bursting gun. The breech-pin was found imbedded in the brain at a distance of one-half inch, and was withdrawn by the aid