Hence the mosquitoes responsible for the new infections in the locality which occur in May must be in their larval stage of development at least some five or six weeks before this yearly rise. This fact becomes quite evident when it is kept in view that:—"When anyone develops an attack of malarial fever, for the first time in any season, it may be assumed (*i*) that he was bitten by an infected anopheles at least ten days or possibly a fortnight previously, (*ii*) that the infected anopheles that bit him lived as an adult insect for a period of at least 12 or 14 days, for the development of parasites in its stomach and salivary glands, and (*iii*) prior to that it had spent another 10 or 12 days in passing successively from egg to larval stage and eventually from pupal to adult form."

The place was visited in the month of May and most of the railway borrow pits were dry, with the exception of a few which had been breeding *A. rossi* and *A. vagus* only. Hence the source of mosquitoes responsible for the malaria at Madarihat must have been some place other than these borrow pits.

The actual findings showed that *the small stream in the abandoned bed* of the Tursa and *the pools either natural or artificially* made in the bed were the chief breeding places of at least three dangerous species of anopheline mosquitoes, viz.—

(*i*) *A. culicifacies,*
(*ii*) *A. listoni,*
(*iii*) *A. maculatus.*

Thus in the opinion of the authors these breeding places are mainly responsible for the malaria at Madarihat, and until and unless the stream and pools in the old bed of the Tursa are carefully dealt with, no amount of attention to the borrow pits only will help matters much. If anything is intended to be done in the way of dealing with the breeding places, scrub jungle should first be cleared to bring into view all the hidden pools where mosquitoes are breeding. It is not the intention of this article to suggest the solution of the problem.

PATHOLOGICAL LAUGHING AND CRYING.

By J. NORMAN PACHECO, M.R.C.S., L.R.C.P.,
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Ranchi European Mental Hospital.

In few textbooks on medicine is pathological laughing and crying described as a sequela of certain cerebral lesions, and the condition is so rarely met with that a description of the following case is not without interest. A European, aged 38, was admitted on the 4th February, 1929, with paresis of the left arm and leg. His previous history showed that he contracted syphilis in 1915, for which he was treated with a full course of mercury and salvarsan. In 1919 his Wassermann reaction was still positive and he had a further course of salvarsan. Up till 1927 he was quite fit and active. Since May 1927 he suffered from severe and continuous headache which ceased only at night and was accompanied by vomiting. The area of pain was just over the right eyebrow. Three days before admission he felt dizzy and reeled to the right side. Vertigo was constant and at the same time he noticed that his left eye grew weaker, and when reading he experienced a crescentic halo to his left. The day previous to his admission while typing letters he noticed that his left arm was cold, numb and weak. When he awoke the next morning he found his left arm and leg were paralysed. He was admitted soon after and on examination it was found that in addition to the arm and leg the left side of his face was paralysed. He could however smile on both sides equally on emotion.

The left hand lost its grip and all the deep reflexes of the arm were exaggerated, but there was no loss of tactile or postural sensibility.

The leg was less paralysed and could be raised off the bed for six inches. The deep reflexes were exaggerated. Babinski's sign (plantar extensor) was present, plus ankle and patellar clonus and no anæsthesia to pain, pressure or temperature.

The right arm and leg and sphincters were unaffected. Jaw clonus was present and the speech was slightly dysarthric.

On examination of the optic discs the right side was normal, but the left in addition to being "choked" had a small dark crescent in the lower and outer quadrant. The pupils were equal and active to light and accommodation. All other cranial nerves were unaffected. He was treated as a case of simple hemiplegia, and on 16th February, 1929, as his Wassermann reaction was positive, 0.45 gm. novarsenobillon was injected intravenously. The next morning he was found to be completely paralysed on both sides and had developed signs of acute bulbar paralysis. He was completely aphasic, had great difficulty in breathing and was unable to swallow, but no fluid regurgitated through the nose. The palate was also paralysed.

The plantar response was extensor on both sides. Throughout his illness the patient never had a rise of temperature.

The most marked feature of the case at this stage was the appearance of uncontrollable fits of laughter. He laughed heartily, incessantly, without cause, and without feeling any emotion whatever, and this in spite of the serious condition he realized he was in. Any stimulus, even the most serious topic, was sufficient to evoke peals of laughter which he could not control even when alone. His mental state was quite clear, and he experienced no pleasure or elation, but rather felt annoyed and expressed the opinion that it was

a positive nuisance. He cried on two occasions without cause and without experiencing any appropriate emotion. The next day (18-2-29) he regained some power in his left arm and left side of his face, and though there was considerable incoordination, he was able to raise his left hand and touch his nose. The power in both limbs gradually returned and in a fortnight he was able to use them, though weak, quite freely.

As he was still aphasic, he was only able to communicate by writing, which he did a good deal most of the day. The arms were the first to regain power. The legs followed gradually and then the face. A second injection of novarsenobillon was given and in a week's time he was able to pronounce a few labial consonants like P. B. M., but not linguals. Two days after the second novarsenobillon injection he complained of an uncontrollable impulse towards crying. He used to cry silently, and again without cause or any accompanying emotion. This however did not last long, and laughter reappeared. He was discharged on 20th February, 1929, by which time he had completely regained the power of his hands and was able to pronounce a few words and name common objects, though with a nasal twang, and could walk several steps but with some ataxia. He is now able to walk quite steadily and can talk a great deal but with a slight slurring and a nasal intonation. The laughter, however, still persists but is decidedly less noticeable.

Apart from its clinical interest the case affords an excellent proof of the fallacy of the James-Lange theory of the emotions, for here we have the exact opposite, i.e., vasomotor disturbance with the consciousness of the characteristic bodily changes, yet without experiencing the appropriate emotion. Numerous examples of this syndrome associated with pseudo-bulbar paralysis have been described by continental writers—the *Zwangslachen* and *Zwangsweinen* of German writers and the *rire et pleurer spasmodiques* of the French.

Kinner Wilson in an article in the *Journal of Neurology and Psychopathology* quotes a patient who "went off in a torrent of laughter on the slightest provocation or on none" and others who laughed on the receipt of grave news.

The question that is important in this problem is where is the lesion? According to Head and Holmes the thalamus is the seat of the emotions, but thalamic lesions are not always found at post-mortem in these cases, and, again, lesions of the thalamic and subthalamic regions do not always produce pathological laughing or crying. According to Brissaud the integrity of the thalamus is essential to life and the causative lesion is one involving the anterior limb of the internal capsule, where he places his *faisceau phychique* or fronto-thalamic tract of control over thalamic centres, but for pseudo-bulbar palsy he places the centres for involuntary expressions of the emotions in the basal ganglia. Several other authorities place the lesions in different areas but no precise information is given as to how these act. Kinner Wilson suggests that "lesions of the geniculate bundle anywhere in its course will impair volitional control over the musculatures concerned with expression of the emotions and as pseudo-bulbar paralysis is a disease of the geniculate bundles, lesions affecting it are particularly prone to be accompanied by the phenomenon of *rire et pleurer spasmodique*." He also lays stress on the fact that "corticifugal paths to the facio-respiratory centres in the pons and medulla are independent of the voluntary cortico-ponto-bulbar tracts to the same nuclei," that excitation will "modify the facio-respiratory synkinesis in the direction of either laughter or the reverse."

Now it is a well known fact that if a patient with a lesion, say of the right optic thalamus, is told a joke he smiles only on the right side, but when he assumes a smile both sides of the face act equally. But in a lesion of the right Rolandic area he smiles equally on both sides in response to a joke and an assumed smile occurs only on the right side, showing therefore that the motor element of emotion crosses to the opposite side. Now the only bundle which crosses from the mesencephalon to the opposite side is Monakow's rubro-spinal bundle. I suggest that this bundle subserves the function of the motor part of emotion that is involuntary. In addition there is a cortical portion of the system of motor neurons subserving the function of emotion connecting the cortex with the nucleus rubra. M. and Mme. Dejerine describe such a system, the fibres of which they state originate from all parts of the cortex, especially the parietal lobe. They skirt the thalamus just above the radiations of the internal geniculate body, enter into the formation of the tegmentum and reach the red nucleus at its antero-supero-external part, forming an upper segment of the emotional motor system.

I venture to suggest that the cause of pathological laughing and crying in cases of pseudo-bulbar palsy is to be found in the vicinity of the red nucleus, the lesion stimulating the rubro-spinal tract of Monakow, which may be considered the involuntary tract for emotion, and thus producing involuntary laughter or crying, and at the same time cutting off the cortico-rubral tract described by Dejerine, which tract may be considered to exercise a voluntary control over Monakow's rubro-spinal tract.

The result is that when the voluntary control of Dejerine's cortico-rubral fibres is cut off, stimulation of the lower rubro-spinal

tract produces involuntary laughing and crying without the registration in the association areas of the affective tone of emotion.

CHAULMOOGRA OIL IN THE TREATMENT OF TRACHOMA.

By R. GUBBAY,
Superintendent, Zenana Hospital, Jammu, Kashmir.

Of the very large number of patients who attend our out-patient eye clinic, by far the majority are suffering from trachoma or granular lids.

I have tried many different remedies for this condition, expression, various operations, silver nitrate, and other drugs. Two years ago, however, I commenced to use chaulmoogra oil (obtained from Messrs. Smith, Stanistreet & Co., Calcutta), and the results have been so encouraging that I now use it almost to the exclusion of other applications. With the very first application there is immense relief, the lids feel lighter, the gritty sensation is less troublesome, the sticky discharge decreases, and vision is clearer.

Mode of Application.—A swab stick is soaked with the oil, and, the eyelids being everted, this is well rubbed into the surface, taking care to go as far back as possible into the fornix, at the same time protecting the cornea with the lower lid. The tears combining with the oil form a soapy substance. There is generally intense irritation for a few minutes and much watering of the eyes, but no more than occurs with copper sulphate or silver nitrate. This passes off very quickly, and I have never observed any injury to the cornea or conjunctiva, and have used the method successfully in hundreds of cases.

The application may be made daily, or—if this is found too irritant—every alternate day. About ten to fifteen applications suffice to clear up the condition; but I have given a longer series of applications with no cauterising effects. Scarring is much less with chaulmoogra oil than with other remedies. As to how the oil acts, I cannot say. Early cases react best to treatment, and in these three or four applications are enough to check the progress of the disease.

In acute cases after the first application there may be much swelling and œdema of the lids. This, however, is very rarely the case and does not occur if ordinary measures are used, such as hot fomentations, protargol drops, etc., until the acute symptoms subside.

One case treated was a child one year old. The lids were everted with huge granulations and could not be closed naturally. After four applications there was voluntary closure of the lids. The patient was brought to the hospital for a month, and thereafter was not seen again, so I conclude that there was no further trouble.

Chronic cases do very well indeed. In cases of affection of the margin of the lid with redness, the oil is rubbed well into the margin of the lids—about two or three applications are sufficient. I have treated babies a few weeks old with this remedy and the results are excellent, with no ill effects.

(*Note.*—We have shown the above article to the Superintendent of the Eye Infirmary, Medical College Hospital, Calcutta, who gives it as his opinion that the chief trouble about the treatment of trachoma and granular lids in India is the almost universal application of too irritant remedies, with bad results. Possibly chaulmoogra oil may represent a happy mean. The microbic cause of trachoma is almost certainly a filter-passing organism, and until something more is known about the ætiology of the disease than is at present the case, treatment is bound to remain empirical. Many of the acute cases are complicated by secondary infections, a Koch-Weeks' bacillus infection, gonorrhœal conjunctivitis, etc., and the treatment of these should not be neglected.—Ed., *I. M. G.*)

CHEMOTHERAPEUTIC INVESTIGATIONS WITH ANTIMONY PREPARATIONS IN THE EXPERIMENTAL KALA-AZAR OF THE HAMSTER.

By the late WILHELM ROEHL, M.D.,
*Elberfeld.**

The production of a general kala-azar infection in different laboratory animals, especially in dogs and mice, was a task which succeeded very rarely, until Smyly and Young (1924) discovered that in the Chinese hamster (*Cricetulus griseus*) infection occurred in practically 100 per cent. Thereafter M. Mayer (1926) showed that also in the European variety of the hamster (*Cricetus frumentarius*) a positive result could always be obtained.

The present writer examined the chemotherapeutic properties of different antimony compounds in infected hamsters. The leishmania strain used was obtained through the kindness of the Institut. f. Schiffs-u. Tropen-hygiene, Hamburg.

First of all the toxicity of each compound per kilo body-weight was ascertained. The injections were given subcutaneously. For treatment a single weekly injection was found to be the best possible method. The efficiency of the different compounds was estimated by puncturing the liver of the hamster once weekly and examining a smear for leishmania after staining with Giemsa's stain.

(* *Note by the translator:*—Dr. Wilhelm Roehl was an assistant of Professor Paul Ehrlich from 1907 to 1909 and took part in the investigations concerning the new preparations 606 and 904. From those days he devoted his life to chemotherapeutical research, the methods of which he had been taught by the originator himself. In 1911 he became Superintendent of the Chemotherapeutical Research Laboratory of Messrs. F. Bayer in Elberfeld. An important part in the history of the discovery of "Bayer 205" is due to his research and only lately he carried out all the numerous trials connected with the discovery of "Plasmoquine." The method of administering certain quantities of the chemical compound to a bird infected with *Plasmodium relictum* by means of the stomach tube is due to him. And only with this method it was possible to work out the exact chemotherapeutic effects of the different compounds of the Plasmoquine group.

His last work was connected with antimony research, but was not finished. He died suddenly of septicæmia, at only 48 years of age.)