

hard against the testicles, in a longitudinal direction, and as much subcutaneous tissue as possible pressed out before cutting. This lessens the number of vessels that will be cut. After severing the scrotum with knife or scissors the clamp is taken off and all bleeding points are carefully caught and tied with fine catgut.

The varicose pampiniform plexus is now exposed as far away from the testicle as possible and separated from the vas deferens with its artery and veins. The plexus lies with the spermatic artery in a separate sheath which need not be split. Sometimes the separation is difficult on account of adhesions which exist as a result of inflammation. Ligation of the spermatic artery does not impair the nutrition of the testicle.

The vas deferens is easily recognized by its cord-like feel. It must not be handled unnecessarily, and is held aside with its vessels while a double chromicized catgut ligature is passed around the veins. This is separated and the veins finally tied in two places two inches apart, the lower ligature being tied first. The intervening section is now removed, care being observed to leave the stumps long enough to prevent slipping. The ends of the ligatures may be left long and the cord shortened by tying them together. This saves time but leaves a lump which will persist for several weeks to vex a nervous patient. It is better to approximate the ends of the veins and stitch them together.

The wound is closed longitudinally with interrupted silkworm sutures, the ends of which are left long and tied together, as short ends will irritate the sensitive scrotum. Drainage usually is not necessary. Rubber tissue or oiled silk is placed over the dressing to prevent the possibility of contamination by urine. The dressing is held in place by a figure-of-eight roller bandage which supports the testicles. After a few days it is convenient to use a smaller dressing and a large suspensory bandage. The patient is kept in bed for ten days, and a suspensory worn only until the wound is thoroughly healed, then discarded. Otherwise there is danger of forming the "suspensory habit."

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MEANINGLESS DIAGNOSES.

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Medical science has shown an astounding progress in our generation, not only in knowledge but also in wisdom, and with this has come a refreshing education of the public in matters regarding disease and its treatment. This education, rather than interfering with the efficiency of the medical man by the obtrusiveness of the public with whom a little learning might have been a dangerous thing, has brought about a most invigorating cooperation and has increased the trust and confidence of the doctor's position.

This confidence physicians as a body should strive to merit, and it is, therefore, not in bad taste now to point out at least one matter in which, even in this enlightened day, many fall by the wayside. Advance in medicine has emphasized the fact that the *sine qua non* predicated the rational treatment of disease is the establishing of a diagnosis. Increased facilities for clinical examination have given us, in many cases, material evidence on which to base our diagnoses, and in others some tangible reasons for induction and inference.

The result of this is that we are now in a position to

be thoroughly honest. Heretofore, perhaps quite subconsciously, we have not always been utterly frank with ourselves, and the time has now come when the profession should discard several ill-used and time-worn diagnoses that have expressed clinical entities, which if not fantastic, have at least never been proved to exist. These diagnoses have been used much too frequently to cover our ignorance and to pander to the desire of the patient to know what is the matter. This is a perfectly natural desire, too, and is so thoroughly relieved by an answer that it has tempted too many to resort to the diagnoses about to be mentioned.

Perhaps the most commonly used of these meaningless diagnoses is that of "biliousness," and there is no other outranks it in giving satisfaction to the patient. Just what is "biliousness"? Does it mean blocking of the biliary exit? If it does we should have jaundice and all the symptoms of biliary obstruction, and if that is so, then we are surely dealing with cholelithiasis, cholangitis, cholecystitis or new growth; in other words, a definite clinical entity which "biliousness" does not express. Does it mean insufficient secretion or excretion of bile? If so there should be those chemical changes in the stools and in general metabolism which are never looked for by the physician who diagnoses "biliousness." No, "biliousness" does not mean any of the above things, but is applied to those cases of general malaise whose true cause, if not to be found in constipation, can not be or is not ascertained. When "biliousness" has been diagnosed, nothing has been diagnosed. The diagnostician has satisfied both himself and patient and has cloaked his ignorance at the sacrifice of his frankness.

How many of us have encountered "typhoid pneumonia"? To-day there is firmly fixed in the minds of many practitioners the idea that such a combination of diseases actually exists, and when they announce the diagnosis they mean not the pneumonic type of typhoid (which, of course, does exist as well as the intestinal, meningitic and nephritic forms, and is not uncommon), but a true coincidence of the diseases. Such cases usually are typhoid pure and simple, and should there be any doubt a blood examination and Widal reaction will dispel it. Most of us, either directly or indirectly, have been influenced by the teachings of Osler, and it used to be a salient point of his that the "typhoid pneumonias" were usually typhoid, perhaps pneumonia, but never both. There is one possibility of such a diagnosis being true, and that is in a case of pneumonia caused by the typhoid bacillus, which would be striking for its rarity, that is, when not a part of typhoid fever.

"Typhoid malaria" is to be deprecated as severely as "typhoid pneumonia," for such cases are invariably one or the other. Naturally it is easily possible for a malarial patient to contract typhoid, and then he has typhoid following malaria. Such cases, however, are not the ones usually included in that class diagnosed as "typhoid malaria."

What a vogue "ptomain poisoning" has attained! It must be conceded that ptomain poisoning does exist, and may be very common for all we know, but that is no justification for calling every case of acute gastroenteritis by that name. We are entirely ignorant as to whether such cases are actually caused by ptomains. Most of us appreciate the training in chemistry, the equipment and the time necessary to isolate ptomains, and yet many physicians blithely diagnose ptomain poisoning. It has been my experience to hear a practitioner testify at an inquest that ptomains are to be rec-

ognized by a microscopic examination of the gastric contents!

What is "gastric fever" of which we hear so commonly? It never has become quite clear to me, and probably is not quite clear to those who diagnose it, just what type of case it represents. Who first described the disease, in what works in medicine do we find accounts of it and ought we not record its death?

There are many more meaningless diagnoses than these, and it may be that each section of the country has its own particular foibles of this nature, but should not all conscientious physicians discard the use of these terms, and if there are none better to be found in the present terminology, why use terminology at all? It is self-evident that the expression "I don't know" is better than "biliousness" and the rest of the category, and brings the comfort that there is after all much doubt left, in the clearing of which we can find use for our years.

Our distance from Utopia in medicine, as in all things, is vast, and though we shall never attain the ideal, we make one step towards it when we face our ignorance when we find it, and we make still another when we try to overcome it. Rather than call things by false names or meaningless names, let us call them by no names at all, and so, for the love of Æsculapius and Hippocrates, let us hear little more of "biliousness," "ptomain poisoning" and the like.

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COMATOSE PERNICIOUS MALARIA.

WITH REPORT OF THREE CASES.

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The comatose form of malaria is met more frequently than any other of the pernicious varieties. Even in temperate latitudes it is not extremely rare. Its diagnosis compels an answer to the very old but frequently occurring and often difficult question, that of the cause of coma in a given case. Its prompt recognition is very important because of the effect on the mortality of the early institution of correct treatment. These considerations should make it a subject of more than usual interest.

ETIOLOGY.

Although any variety of the malarial plasmodia may give rise to pernicious symptoms, in the vast majority of cases such symptoms are due to the estivo-autumnal parasite. Nothnagel states that so far only two cases of comatose malaria have been observed in which the blood showed the ordinary tertian parasite. Aside from favoring conditions pre-existing in the individual, the development of coma depends on three factors: the number of parasites present; the amount of toxin secreted by them; and, most important of all, the localization of the parasites in the brain. In temperate latitudes malarial coma occurs almost always in the summer and fall, as do all other pernicious manifestations of the disease. In the tropics it occurs throughout the year and is quite common. The attacks usually occur in persons who have suffered from malaria for some time and have not been properly treated. Cardamitis believes that predisposing causes to comatose attacks are previous defective conditions of the organism, due to

anemia, cachexia, physical or mental fatigue, the puerperium, insolation, alcoholism, etc.

PATHOLOGY.

The pathology of comatose malaria is the same in most respects as that of any severe malarial infection. There are the breaking down of the red cells, the oligocythemia and hemoglobinemia, the melanemia and pigmentation of the whites, and in most cases the relative increase in the mononuclears. There is also in the majority of cases, as in the less severe forms of malaria, a leucopenia, but in some of the fatal comatose cases a leucocytosis is observed (Craig). The changes in the spleen and liver are the same as those produced in any severe case of malaria.

The kidneys usually show the changes of acute nephritis, combined with the peculiar lesions due to the malarial infection. The urine contains albumin and hyalin and granular casts. "It can be stated, as a rule, that all fatal cases of malaria show albuminous urine containing casts prior to death" (Craig).

The most striking pathologic lesions, and those which may be said to be peculiar to the comatose form, are those found in the brain. Actual thrombi of malarial parasites occur in this organ blocking the capillaries. These can be readily demonstrated after death by simply placing a fragment of the brain tissue under a cover-glass, and the appearance presented is one likely to make a deep impression on the mind of the observer. The secondary changes are pronounced and consist of congestion of the capillaries, minute hemorrhages into the brain substances, and accumulation of pigment in varying amount. As has been shown by the researches of Marchiafava and Monti, changes occur in the protoplasm and nucleus of the nerve cells, leading to complete degeneration of the diseased tissue.

Guarnieri has described changes which take place in the retina in pernicious malaria. These consist of hemorrhages and congestion of the capillaries with impairment of function. Lellos has related a number of cases of meningitis which were apparently due to malarial infection and were cured by quinin.

The peripheral blood usually contains large numbers of malarial organisms of the estivo-autumnal variety.

SYMPTOMS.

The coma may come on suddenly like an apoplectic stroke, whence the old phrase "*intermittens apoplectica*," or, as is more common, it may develop gradually during a paroxysm of fever, having been preceded by slight disturbances of the sensorium such as apathy, somnolence, restlessness and melancholia. The coma may disappear with the fall of the temperature to reappear again within twelve or twenty-four hours, or it may continue over a period of one to four days until death or recovery ensues.

The face is somewhat flushed, the pulse at first full and bounding, later weak, frequent and thready; the respirations are hurried and often stertorous. Attempts to rouse the patient call forth no response or only an unintelligible muttering. The patient usually lies perfectly quiet, but there may be restless movements of the limbs. The skin is warm and toward the latter part of the attack is bathed in sweat. It often has an icteric tint. The tongue is thickly coated. Feces and urine pass involuntarily. Retention of urine is not infrequent.

The temperature is not usually high, sometimes sub-