acknowledge some mystery in the changes characteristic of life. It is to be hoped that the acknowledgment of mystery will not lead to increasing obscurity.

I am particularly desirous of directing attention to the question of vitality just now because during the past year there have been indications of a change of opinion, and the prospect of obtaining a patient hearing is better than it has been for thirty or forty years. The spread of bacteriological investigation is familiarising an increasing number of observers with the use of high magnifying powers and methods of preparation of great importance in the higher branches of histological research; and there are many members of the profession and advanced students who are eminently qualified to judge of the facts and arguments advanced concerning the important question of the nature of vitality and to form a correct estimate of the bearing of observed facts of nature in health and disease upon the question.

I shall have to consider the probable nature of those wonderful changes which take place when lifeless nutrient matter or some of its constituent particles come into contact with matter that already lives, the nature of the new powers communicated to it, and the manner in which these new powers are transferred. Although our knowledge concerning these matters is at present imperfect I hope to show that we are able to penetrate more deeply into the mystery than anyone studying our most advanced works on biology would be led to suppose was at this time possible. Unfortunately many authorities whose doctrines have long influenced general opinion continue to repeat in most confident language statements made long ago in favour of mechanical hypotheses without adducing fresh facts or advancing new arguments.

The question of the nature of the power or endowment which establishes a sharp line of distinction between every form of living matter and every kind of matter in any other state or condition known to us, as well as all energies, forces, powers, or agencies whatever, is of absorbing interest. No physiological action, and no pathological change in nature, occurs which is not closely connected with, or directly or indirectly consequent upon, vital action of living matter. From the first movement of the matter of the living germ to the last moment of living existence—from the first slight departure of living matter from its normal condition to the establishment of morbid changes which must be fatal, the influence of vital change is paramount and is distinct from every accompanying physical and chemical action yet discovered. So fundamental are the differences between vital and physical processes that it may be safely said there is no analogy between them. The difference is not one of degree or relation, but it is essential and absolute.

To attempt to determine the nature of mind, thought, or will without in the first place determining the nature of vitality seems useless. For in all cases vitality exists long before there is evidence of any process that can be properly called mental, and certainly vitality exists in a multitude of cases in which mind is absent at every period of life, and all living beings may live for a time and die without the manifestation of mental action. Mental operations may cease although vital actions continued to be performed, but of mental action without living matter we have no knowledge. In the absence of vitality mind cannot be.

The rate at which vital actions are performed is not uniform, but varies greatly in different cases and under different conditions. The influence of all our remedial measures adopted in departures from the healthy state is due to changes brought about in processes dependent upon vital actions. We are able to facilitate or retard vital actions in some cases and to modify, check, or stop them—to reduce them when too active and to increase them when proceeding too slowly. Some of our most certain and valuable remedies act upon certain forms of living matter in the organism, while on other forms they exert no influence, and it is remarkable that of certain substances which have a very decided action upon certain forms of living matter in the human subject an overdose becomes a poison, stops vital action altogether; and yet may have no action upon the corresponding living matter of some of the lower animals, and may even be taken with impunity or perhaps constitute a harmless food. In some cases by encouraging increased action in one or more kinds of living matter in certain tissues or organs we may reduce to its normal degree exaggerated action tending to derange, damage, or destroy some more important tissue or organ near to or distant from the seat of our treatment.

The recognition of this principle enables us to be of service in the treatment of many forms of inflammation and fever, and even in certain forms of morbid growths.

(To be continued.)

FATAL PULMONARY APOPLEXY (PROBABLY EMBOLIC) IN AN APPARENTLY HEALTHY GIRL AGED FOURTEEN YEARS.

By W. PASTEUR, M.D., F.R.C.P. LOND.,
ASSISTANT PHYSICIAN TO THE MIDDLESEX HOSPITAL.

THE following case, which came under my care in June, 1895, in consultation with Dr. Marriott of Sevenoaks, presents several features of interest.

I happened to be well acquainted with the previous history of the patient, having known her from early childhood. With the exception of the usual childish ailments and of one severe illness at eleven years of age she had always enjoyed good health. Her powers of physical endurance, however, were perhaps somewhat less than her sturdy build would have led one to expect. She was healthy looking and had unusually bright colouring, a feature common to several members of her father's family. When eleven years old she had whooping-cough in the wake of influenza, which was complicated by extensive broncho-pneumonia of both lungs; the attack gradually subsided after a very critical week, during which the degree of cyanosis of the face was very unusual. She made an apparently complete recovery and resumed all her usual occupations, including violin playing, to which she devoted two or more hours a day. At this date her heart showed no indications of disease. Her last illness began quite suddenly on June 16th, as she was returning home from a two-mile walk, just after the termination of a perfectly normal "period." She was seized with breathlessness and had to be helped home. She was at once put to bed, but had to be propped with pillows into the sitting posture to enable her to breathe. In this position she was fairly comfortable and felt better next day, when first seen by Dr. Marriott, who promptly recognised the gravity of the case. When I saw her on the evening of the 18th her condition was as follows. She had orthopnoea, an anxious face, and rather quick, panting respiration, chiefly upper costal. A deep-red flush was on each cheek and a dusky pallor elsewhere. The eyes looked very large and bright. She made short, whispered replies to questions. There was an increased area of heart impulse, which was diffuse and undulating. The sounds were confused, but a sharp accentuated second sound was well heard at the base. No murmurs were made out. The pulse was running and uncountable. She had very troublesome sickness after all food or drink. She was ordered one-eighth of a grain of morphine under the skin, also hypodermic injections of strychnine and ether. On the 19th her condition remained unchanged. Morphine was ordered to be repeated if the restlessness increased and nutrient enemata. On the 20th her condition remained about the same. On the day preceding and on this day numerous moist crepitations were heard in the right axilla without obvious dullness. The breath sounds were loud at the apices. The back was not examined owing to the patient's condition. The cardiac dullness had decidedly increased and reached fully one inch beyond the right edge of the sternum. The pulse was a shade better in quality than the day previous. The heart was still churning. On one or two occasions, including this day, I could distinguish temporary murmurs, both systolic and presystolic in time, but they were never present during many successive beats. The general condition varied. The patient had bouts of agonising dyspnoea followed by periods of exhaustion and comparative quiet with disturbed sleep. On the 21st she had a very bad night, with extreme restlessness and cyanosis; the cough was very frequent and distressing, and she expectorated about three ounces of dark blood and blood-stained mucus. She rallied towards morning and was then in much the same state as at my previous visit. The physical signs were less in front of the right axilla and the breath sounds were clearer, but signs of engorgement and oedema were now present all over the posterior portions of the lungs.

She had been taking two-minim doses of tincture of digitalis in iced water with a few drops of brandy every half hour since the previous day. On the 22nd she had another bad night with decided increase of delirium and awful dyspnoea and some wandering during the day. The face, if possible, was more dusky. When seen at 9 P.M. she appeared to be moribund. The breathing consisted of a succession of short jerky sighs, the hands were icy cold, and the legs cold up to the knees. The radial pulse was fluttering and barely perceptible and there was whispering delirium. She was ordered one-tenth of a grain of morphine and stimulants with local application of warmth. She slept well after this and on the 23rd had rallied surprisingly in all directions. The improvement was maintained throughout the day, but towards evening the unfavourable symptoms returned. At midnight the patient awoke from a restless sleep and asked the nurse for some water, but before this could be given her she had fallen back dead.

It is matter for regret that an account of the post-mortem appearances is not available. The grouping of symptoms, however, and the character and development of the physical signs leave little doubt as to the nature of the case—blocking in the first instance of one of the branches of the pulmonary artery in the right lung with subsequent extension of the thrombus to other branches, pulmonary apoplexy, and death from cardiac failure. The chief interest of the case lies in the occurrence of this well-known series of events in a young girl who was apparently in perfect health. A diligent inquiry into the cause of the accident has been entirely negative in its results, and we have had to fall back on the hypothesis that the seeds of the mischief had been sown during the severe illness above referred to, possibly in the form of adherent thrombi in the right cavities of the heart, one of which became detached on June 16th.

Chandos-street, W.

NOTE ON THE PICRIC ACID TEST FOR SUGAR.

By MARK McDONALD, M.B. DUB.

WHILE carrying out some work on the urine some time since I came across certain reactions with the picric acid reduction test with which it is not generally credited. The importance which this test has acquired in the estimation, both qualitatively and quantitatively, of sugar in urine is sufficient excuse for bringing forward any points bearing on the subject.

When to a drachm of urine there are added a drachm of a saturated solution of picric acid, half a drachm of liquor potassæ, and one and a half drachms of water (as recommended by Sir George Johnson in the quantitative estimation of sugar), an orange-red colour is produced in the cold, due to the kreatinin present in the urine reducing the yellow potassium picrate to red picramate. On heating, this colour becomes more intense. Should the urine contain sugar in any quantity the effect of its presence will not be observed until the temperature reaches nearly 100° C., at which point the colouration is evidently much intensified. If, however, before heating the mixture it be largely diluted—the addition of two ounces of water produces the effect most typically as a rule—the liquid is seen to gradually darken as it is heated, until it boils. Should the urine be normal it will be observed after a few seconds' boiling that the colouration has disappeared, and prolonged boiling does not bring it back; but if the liquid be cooled the colour gradually reappears, and this reaction may be repeated with the same solution any number of times. Should the urine contain sugar in any quantity the solution as before gradually darkens as heat is applied until it boils, when the colour (due to kreatinin) disappears, but if the boiling be prolonged in this case the solution again becomes coloured. This paradoxical reaction depends on the weakness of the solution in potassium picrate. Solutions of the latter when very weak are only with great difficulty partially reduced. The picramate produced by the action of kreatinin in the cold is on boiling reoxidised to picrate; sugar, on the other hand, reduces the weak picrate solutions only on prolonged boiling. A solution of pure urinary kreatinin (prepared by Mr. S. Johnson's method) acts in a manner similar to a normal urine, more liquor potassæ being,

of course, required, as Sir George Johnson pointed out. A dextrose solution on similar treatment caused reduction of the picrate only on very prolonged boiling.

It appears, then, that under certain conditions potassium picramate, when boiled in the presence of the substances by whose oxidation it has been produced from picrate, undergoes reoxidation at the expense of these oxidised bodies. The conditions necessary are weakness of the picrate solution and capability of the oxidised bodies to easily undergo reduction. The latter condition is met with in the case of kreatinin. Other substances, as for example the alkaline sulphides, though they reduce in the cold, nevertheless do not reoxidise the picramate on boiling, as the products of their oxidation are very stable bodies. Dilution of the kreatinin solution also assists the reaction. Corresponding results are obtained if, instead of diluting the solutions before boiling, they are first boiled at the usual strength and then diluted with two ounces of water. In the case of normal urines and kreatinin solutions boiling removes the colour, while with sugar and alkaline sulphides no diminution takes place however prolonged the boiling may be. It is noteworthy in all these cases that when heat is applied after dilution the liquid assumes a distinctly darker colour before the boiling point is reached, showing that in the process of dilution and cooling some reoxidation of the picramate had taken place.

If the behaviour of normal urines and kreatinin solutions, on the one hand, and saccharine urines and dextrose solutions, on the other hand, be compared, their reducing power being equal, it is found that after prolonged boiling of the diluted solutions those which contain dextrose have a distinctly darker colour. The intensity of this final colour is proportional, too, to the quantity of dextrose present. It is possible by means of the different actions of kreatinin and sugar to determine whether the reducing effect of a urine is in part due to sugar. This can usually be effected by heating the diluted solution to nearly the boiling point in a long test-tube of small diameter, and then boiling the upper half of the liquid. The colour due to kreatinin disappears, and on boiling for a minute or two the upper part of the solution again darkens if sugar is present. It is important to remember that if the diluted liquid contains less than 1 part of dextrose in 10,000 no sugar reaction takes place, since, as Sir George Johnson noticed, its effect on alkaline picrate vanishes with higher degrees of dilution. This disturbance may be obviated by adding less water, or a dilute solution of potassium picrate may be prepared by mixing a drachm of saturated picric acid solution with half a drachm of liquor potassæ and adding eight and a half drachms of water. To one drachm of this solution one drachm of a twice or four times diluted and neutralised urine is added and the mixture treated as usual. In some cases it is necessary to compare the final colouration with that given by a kreatinin solution of equal reducing power.

Though I have occasionally during the last few months found this reaction of picric acid useful in deciding a diagnosis of glycosuria, I bring it forward less as a practical test than as a warning against a possible disturbance of the usual test for sugar.

Waterloo, Liverpool.

THE DISCOVERY OF A BULLET LOST IN THE WRIST BY MEANS OF THE ROENTGEN RAYS.

By ROBERT JONES, F.R.C.S. EDIN.,

HONORARY SURGEON TO THE ROYAL SOUTHERN HOSPITAL, LIVERPOOL;

AND

OLIVER LODGE, F.R.S.,

PROFESSOR OF PHYSICS, UNIVERSITY COLLEGE, LIVERPOOL.

A BOY aged about twelve years was brought to me by Dr. Simpson of Waterloo, Liverpool, having shot himself in the left hand just above the deep palmar arch. The wound was enlarged, but the bullet could not be found, and it was thought injudicious to prolong the search in view of the important structures in the vicinity unless one possessed a clue to its position. Professor Lodge kindly consented to take a photograph and the position of the bullet was very clearly outlined. It lies against the base of the third metacarpal bone over its articulation with the os magnum. This