

itself abnormal; that old age is a form of disease or is due to disease, and, theoretically at least, is capable of being eliminated. We have already seen that individual cell-life, such as that of the white blood corpuscles and of the cells of many tissues, can under suitable conditions be prolonged for days or weeks or months after general death. Unicellular organisms kept under suitable conditions of nutrition have been observed to carry on their functions normally for prolonged periods and to show no degeneration such as would accompany senescence. They give rise by division to others of the same kind, which also, under favourable conditions, continue to live, to all appearance indefinitely. But these instances, although they indicate that in the simplest forms of organisation existence may be greatly extended without signs of decay, do not furnish conclusive evidence of indefinite prolongation of life. Most of the cells which constitute the body, after a period of growth and activity, sometimes more, sometimes less prolonged, eventually undergo atrophy and cease to perform satisfactorily the functions which are allotted to them. And when we consider the body as a whole, we find that in every case the life of the aggregate consists of a definite cycle of changes which, after passing through the stages of growth and maturity, always leads to senescence, and finally terminates in death. The only exception is in the reproductive cells, in which the processes of maturation and fertilisation result in rejuvenescence, so that instead of the usual downward change towards senescence, the fertilised ovum obtains a new lease of life, which is carried on into the new-formed organism. The latter again itself ultimately forms reproductive cells, and thus the life of the species is continued. It is only in the sense of its propagation in this way from one generation to another that we can speak of the indefinite continuance of life: we can only be immortal through our descendants!

AVERAGE DURATION OF LIFE AND POSSIBILITY OF ITS PROLONGATION.

The individuals of every species of animal appear to have an average duration of existence.²⁸ Some species are known the individuals of which live only for a few hours, whilst others survive for 100 years.²⁹ In man himself the average length of life would probably be greater than the three-score and ten years allotted to him by the Psalmist if we could eliminate the results of disease and accident; when these results are included it falls far short of that period. If the terms of life given in the purely mythological part of the Old Testament were credible, man would in the early stages of his history have possessed a remarkable power of resisting age and disease. But, although many here present were brought up to believe in their literal veracity, such records are no longer accepted even by the most orthodox of theologians, and the 900 odd years with which Adam and his immediate descendants are credited, culminating in the 969 of Methuselah, have been relegated, with the account of Creation and the Deluge, to their proper position in literature. When we come to the Hebrew Patriarchs, we notice a considerable diminution to have taken place in what the insurance offices term the "expectation of life." Abraham is described as having lived only to 175 years, Joseph and Joshua to 110, Moses to 120; even at that age "his eye was not dim nor his natural force abated." We cannot say that under ideal conditions all these terms are impossible; indeed, Metchnikoff is disposed to regard them as probable, for great ages are still occasionally recorded, although it is doubtful if any as considerable as these are ever substantiated. That the expectation of life was better then than now would be inferred from the apologetic tone adopted by Jacob when questioned by Pharaoh as to his age: "The days of the years of my pilgrimage are a hundred and thirty years; few and evil have the days of the years of my life been, and have not attained unto the days of the years of the life of my fathers in the days of their pilgrimage." David, to whom, before the advent of the modern statistician, we owe the idea that 70 years is to be

regarded as the normal period of life,³⁰ is himself merely stated to have "died in a good old age." The periods recorded for the Kings show a considerable falling-off as compared with the Patriarchs; but not a few were cut off by violent deaths, and many lived lives which were not ideal. Amongst eminent Greeks and Romans few very long lives are recorded, and the same is true of historical persons in mediæval and modern history. It is a long life that lasts much beyond 80; three such linked together carry us far back into history. Mankind is in this respect more favoured than most mammals, although a few of these surpass the period of man's existence.³¹ Strange that the brevity of human life should be a favourite theme of preacher and poet when the actual term of his "erring pilgrimage" is greater than that of most of his fellow creatures!

THE END OF LIFE.

The modern applications of the principles of preventive medicine and hygiene are, no doubt, operating to lengthen the average life. But even if the ravages of disease could be altogether eliminated, it is certain that, at any rate, the fixed cells of our body must eventually grow old and ultimately cease to function; when this happens to cells which are essential to the life of the organism general death must result. This will always remain the universal law, from which there is no escape. "All that lives must die, passing through nature to eternity."

Such natural death unaccelerated by disease—is not death by disease as unnatural as death by accident?—should be a quiet, painless phenomenon, unattended by violent change. As Dastre expresses it, "The need of death should appear at the end of life, just as the need of sleep appears at the end of the day." The change has been led gradually up to by an orderly succession of phases, and is itself the last manifestation of life. Were we all certain of a quiet passing—were we sure that there would be "no moaning of the bar when we go out to sea"—we could anticipate the coming of death after a ripe old age without apprehension. And if ever the time shall arrive when man will have learned to regard this change as a simple physiological process, as natural as the oncoming of sleep, the approach of the fatal shears will be as generally welcomed as it is now abhorred. Such a day is still distant; we can hardly say that its dawning is visible. Let us at least hope that, in the manner depicted by Dürer in his well-known etching, the sunshine which science irradiates may eventually put to flight the melancholy which hovers, bat-like, over the termination of our lives, and which even the anticipation of a future happier existence has not hitherto succeeded in dispersing.

ON THE VALUE OF A QUANTITATIVE ALBUMIN ESTIMATION OF THE CEREBRO-SPINAL FLUID (WITH SPECIAL REFERENCE TO THE SYNDROME OF MASSIVE COAGULATION AND XANTHOCHROMIA).

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It seems of some interest to publish the results obtained by a routine examination of the cerebro-spinal fluids of various neurological cases, especially as some of the cases of paraplegia examined present a condition of the fluid which does not seem to have found its way into English neurological text-books or published reports. Of the condition of the fluid presented by three of my cases I have only been able to find six published reports in French, German, and American literature, but I believe that it is by no means so uncommon as this would lead one to suppose, and that it forms a diagnostic sign in paraplegia of considerable importance.

The method of albumin estimation I have used, whatever its technical errors, has yielded very reliable results in my examinations, and is applicable to very small amounts of fluid. In one of my cases, although only 2 c.c. of fluid could be obtained for examination, it was possible to completely revise the diagnosis by the examination of this.

* This was regarded by Buffon as related to the period of growth, but the ratio is certainly not constant. The subject is discussed by Ray Lankester in an early work: "On Comparative Longevity in Man and Animals," 1870.

²⁹ The approximately regular periods of longevity of different species of animals furnishes a strong argument against the theory that the decay of old age is an accidental phenomenon, comparable with disease.

³⁰ The expectation of life of a healthy man of 50 is still reckoned at about 20 years.

³¹ "Hominis ævum ceterorum animalium omnium superat præter admodum paucorum."—Francis Bacon, "Historia Vitæ et Mortis," 1637.

Many writers have published the results in a series of cases where the albumin has been estimated by the Esbach method, but this is only applicable to large amounts of fluid (over 10 c.c.), and the precipitate is very flocculent, and does not lend itself to accurate readings. Auflecht's tube is better, as it requires only 3 to 4 c.c. of fluid, but for it a centrifuge of known velocity and special size is necessary for accurate readings, and graduations below 0.025 per cent. are not given.

The method I have used is a modification of Noguchi's butyric acid reaction¹; 2 c.c. of fluid are used, and the result of the test is poured into a graduated centrifuge tube, which gives readings to 0.1 c.c. with fair accuracy; each 0.1 c.c. by this method is equivalent to 0.025 per cent. Small quantities of fluid can thus be examined, and fairly reliable readings obtained from them.

The results of my examination of 38 cases of nervous diseases of various kinds by this method are tabulated below. In each case 2 c.c. of the cerebro-spinal fluid was used. For comparison the results obtained by Esbach's method in several cases are also given, and where possible both methods have been used, and the Noguchi method thus standardised. I found that 0.4 c.c. precipitate corresponds fairly closely to 1 part per 1000.

Normal fluids gave readings of 0.05 to 0.2 c.c. (corresponding to 0.1 to 0.5 part per 1000). Syphilitic meningitis and parasypilitic disease gave readings up to 0.6 c.c., or 1.5 per 1000, but in no uncomplicated case did I get readings above this level. In many cases of syphilitic nervous disease, even where some lymphocytosis was present, no increase of the albumin content was found, and this was probably owing to the disease being mainly vascular, or to the meningeal inflammation having subsided. Contrary to the statements of some text-books, I have found the albumin content raised in tabes dorsalis to as high a degree as in any case of general paralysis that I examined. In other cases it was not above the normal level, and most often this was so in the chronic cases.

The cytological examination of the fluid was made in every case, but, owing to imperfections of the centrifuge used, the results were not always comparable. Where the fluid could be thoroughly centrifuged I found that 10 to 12 lymphocytes to the high-power field (magnification $\times 350$) was found in normal fluids. In no case did I find complete absence of cells, except where the fluid had been kept for some time in a tube other than that used for centrifuging. The propensity of leucocytes to adhere to glass is well known, and probably this is what occurred in such cases. Quincke's technique for cytological examination was followed, the fluid being centrifuged at high speed for 20 minutes, and the deposit on the bottom of the tube (after being inverted till it was merely wet), was collected in a newly made capillary pipette and blown out on to a glass slide. In this way most of the cells in the fluid can be collected in a circle of 2 to 3 mm. diameter. By this method lymphocyte counts of 500 to 1000 to the high-power field are often obtained, and Boyd has reported a case in which the count was as high as 3450. Any count above 15 must be reckoned as pathological, and apart from meningitis seems to be pathognomonic of syphilitic or parasypilitic disease.

In a separate table (I.) I give certain of Quincke's figures, with which mine agree fairly closely. Many writers report increase in the cellular elements of the fluid in cases of brain tumour, disseminated sclerosis, and herpes zoster. In my cases I did not get counts which I considered pathological in any of these diseases. Unfortunately I had not an opportunity of examining any case of cerebral endothelioma before operation, but three cases of glioma, in all of which the tumour was apparent on the surface of the brain, gave normal counts; and in an acute case of disseminated sclerosis, and in a case of subacute combined degeneration, during an attack of herpes zoster, the cell counts reached the upper normal limit.

Quincke gives some very interesting results in cases of cerebral tumour. Out of 45 cases, in 41 he found the albumin content less than 0.5 part per 1000—i.e., about normal—but in four cases the content was very high:

(1) 2.7 to 4.5 parts per 1000; (2) 3.5 parts per 1000; (3) 8 per 1000; (4) 15 per 1000. He says: "In the latter cases the fluid was golden, and formed on standing a fine coagulum, without blood or cell elements, but the cause and significance of this unfortunately I cannot state." Cl. Vincent has obtained similar fluids from cases of cerebral tumour, and reports the presence of complement in some of the fluids.

TABLE I.—Results by Quincke.

Nature of case.	Albumin by Esbach method.	Characters of fluid.
Tubercular meningitis— Several cases.	Less than 0.05 %	—
12 cases.	Over 0.2 %	—
5 „	„ 0.3 %	—
3 „	„ 0.4 %	—
1 case.	0.5%—0.6 %	—
Cerebral tumour—* 41 cases.	Under 0.05 % 0.27% to 0.4 %	—
4 other cases.	0.35% 0.8% 1.5%	Golden fluid forming a fine reticulum on standing.
Landry's paralysis.	0.15%	No cells seen.
Chronic myelitis.	0.2%	—
Headache.	0.025%	—
Headache of fever.	0.05%	—

* Indicates that cases are mentioned in text.

This condition of the fluid is similar to what has been described by French writers as "Syndrome de coagulation massive et de xanthochromie sans éléments cellulaires." This was described first in 1909 by Blanchetière et Lejonne, who claim that their case is the first of the kind on record. This was a man of 66, in whom paraplegia had developed somewhat gradually four months before he was examined. The cerebro-spinal fluid, examined repeatedly during six months from the time of the first examination until the death of the patient, was constantly yellow and highly albuminous. On standing it spontaneously formed a coagulum, and sometimes coagulated in the needle. It contained no cellular elements. At the necropsy a tumour of the dura, the size of a large olive, was found. This was situated on the dorsal surface of the cord at the level of the seventh, eighth, and ninth dorsal segments, and had led to some adhesion at this level between the dura and the cord.

A similar finding is recorded by Cooper in 1910, in a case of "localised segmental lesion of the cord."

Macroscopically the fluid was clear, and of a yellowish-brown colour, looking like the clear serous fluid that is aspirated from pleural or peritoneal effusions. On standing a well-marked cobweb coagulum formed. The Noguchi and Nonne globulin tests were strongly positive, thick precipitates occurring. The Wassermann complement reaction was negative in fluid and blood. Microscopically, practically no cells were to be found either in the centrifuged deposit or in the coagulum. No bacteria could be detected. Dr. Hyman, who operated on the case, and I came to the conclusion that we were dealing with a case of chronic compression of the cord, probably due to an intraspinal tumour, but an explanation of the findings in the cerebro-spinal fluid did not suggest itself. At operation a strongly marked oedema of the membranes was found below the site of the tumour (an angiosarcoma). The transudate character of the fluid was thus explained.

Nonne, Apelt, Kleineberger, and Heilig have also collected cases of tumour of the spinal cord, with a similar condition of the fluid below the level of the tumour. In one of my cases (No. 56), where general carcinomatosis of the meninges was found at the necropsy, the fluid was yellow and highly albuminous, and contained very few cells, but it did not coagulate spontaneously. In two other cases (Nos. 57 and 58) the diagnosis of intraspinal tumour was made, or assisted, by the finding of a highly albuminous fluid, which was, however, free from any yellow colouration. In both these cases the high albumin content might not have been noticed in an ordinary examination, especially by an inexperienced observer, for lack of a quantitative standard.

¹ The Noguchi test is as follows: 2 c.c. cerebro-spinal fluid and 5 c.c. of 10 per cent. butyric acid are boiled together. 1 c.c. of normal sodium hydrate is then added, and the tube again boiled, when a flocculent precipitate is thrown down.

TABLE II.—Results Obtained by Noguchi and Esbach Methods.

Case.	Nature of disease.	Albumin (N. = Noguchi, E. = Esbach).	Other characters.
<i>General Paralysis of the Insane.</i>			
1	—	0.2 c.c. N.	Cells 170 to h.p. field.
2	—	0.3 c.c. N.	" 10 + " "
3	—	—	" 50-60 to h.p. field.
4	—	0.2 c.c. N.	" 12-15 " "
5	Congenital.	0.4 c.c. N.	Cells more than 100 to h.p. field.
6	"	0.25 c.c. N.	Cells 100-200 to h.p. field.
7	—	0.1% E.	" 100-200 " "
<i>Tabes Dorsalis.</i>			
8	—	0.2 c.c. N.	Cells 100 to h.p. field.
9	—	0.5 c.c. N.	" 10 + " "
10	—	—	" 30 " "
11	Gastric crises with no other signs.	0.6 c.c. N.	" 100-150 " "
12	—	0.5 c.c. N.	" 100 + " "
13	—	Slight.	" 100 + " "
14	— (1)	0.45 c.c. N.	" 50-60 " "
	(2)	0.4 c.c. N. (0.1% E.)	
<i>Syphilitic Paraplegia.</i>			
15*	—	4.5 c.c. N., E. 0.6%	Yellow fluid coagulating spontaneously into fine reticulum. Cells 50 to h.p. field.
16*	—	1.0 c.c. N.	" 30-50 " " A viscid, slightly yellow fluid.
17	A very chronic case.	0.15 c.c. N.	Cells 9 to h.p. field.
18	—	0.4 c.c. N.	" 100 " "
19	A case after much treatment.	0.2 c.c. N.	" 2-3 " "
<i>Gummata (cerebral).</i>			
20	—	0.5 c.c. N.	Cells 200-300 to h.p. field. A yellow fluid with decayed red cells.
21	—	0.2 c.c. N.	Cells 70-100 to h.p. field.
22	—	0.25 c.c. N.	" 80-100 " "
<i>Congenital Syphilis.</i>			
23	Functional symptoms.	0.1 c.c. N.	Cells 15-20 to h.p. field.
24	Diplegia.	No increase.	" 4-5 " "
<i>Paraplegia.</i>			
25	Syringomyelia.	0.1 c.c. N.	Cells 7 to h.p. field.
26	Gonococcal myelitis (?)	0.05 c.c. N.	" 2 " "
27	Primary lateral sclerosis.	—	" 6 " "
28*	Subacute combined degeneration during an attack of herpes zoster.	0.2 c.c. N.	" 10-15 " "
29	Syringomyelia.	No increase.	" 10 " "
30	Cerebral arterio-sclerosis, ? due to lead.	—	" 4 " "
31	Pseudo-bulbar paralysis.	0.1 c.c. N.	" 3-4 " "
32	Functional ?	0.2 c.c. N.	" 12 " "
<i>Disseminated Sclerosis.</i>			
33	—	0.1 c.c. N.	Cells very few.
34	? syphilitic.	0.3 c.c. N.	Cells 6 to h.p. field.
35	—	†	" 8 " "
36*	In acute stage.	0.2 c.c. N.	" 15 " "
37	—	—	" 4-6 " "
38	—	0.2 c.c. N.	" 5-6 " "
<i>Cerebral Tumour.</i>			
39	Glioma.	0.05 c.c. N.	Cells 2 to h.p. field.
40	+ ^s syphilitic endarteritis.	0.02 c.c. N.	" 5-6 " "
41	Glioma.	0.2 c.c. N. 0.05% E.	" 4-8 " "

† No ppt. with ammonium sulphate.

TABLE II.—Results Obtained by Noguchi and Esbach Methods (continued).

Case.	Nature of disease	Albumin (N. = Noguchi, E. = Esbach).	Other characters.
<i>Tubercular Meningitis.</i>			
42	— (1)	0.2% E.	Cells, lymphocytes and polymorph.
	(2)	0.2% E.	
	(3)	0.05% E.	
43	—	0.2% E.	Cells entirely lymphocytes.
44	—	0.2% E.	Sugar absent.
45	—	0.4% E.	Lymphocytes and polymorph. cells and tubercle bacilli.
46	—	0.1% E.	Polymorphs. and lymphocytes.
47	—	0.2% E.	All lymphocytes.
48	—	0.1% E. 0.5 c.c. N.	Lymphocytes only.
<i>Cerebro-spinal (meningococcal) Meningitis.</i>			
49	—	0.1%	Polymorph. cells. Organisms mainly extra-cellular.
50	—	0.6%	Organisms extra- and intra-cellular.
<i>Hydrocephalus.</i>			
51	Post-bas. (1)	0.15% E.	A few polymorph. Mainly lymphocytes.
	(2)	0.1% E.	No organisms seen.
52	Cong. syphilis.	Considerable increase.	Lymphocytes 75-100 to h.p. field.
	After treatment.	0.05%	" 15 to h.p. field.
53	Syphilitic. (Inactive pupils became active under treatment.)	0.1% E.	Fluid spurted in a continuous stream.
		0.4 c.c. N.	Cells 500-600 in h.p. field, completely filling the field.
<i>Cortical Hæmorrhage.</i>			
54	Purpura neonatorum.	—	(1) Uniformly admixed blood
		—	(2) Blood in a yellowish fluid.
		—	(3) Yellowish fluid, no blood.
55	? Sunstroke, recovery in 5-6 days.	0.4 c.c. N.	A yellowish fluid with thick turbidity, containing red cells in proportion of 5 to 1 of leucocytes which were mainly polymorphonuclear. No organisms.
<i>Cases showing Abnormally High Albumin Content.</i>			
<i>Spinal Tumours.</i>			
56*	Carcinomatosis of meninges.	1.4 c.c. N.	A clear yellow fluid. Sugar diminished. Cells 3 to h.p. field.
57*	Invasion of meninges by alveolar sarcoma.	1.1 c.c. N.	Clear white fluid. Cells 10-15 to h.p. field.
58*	Paraplegia, ? spinal tumour.	2.2 c.c. N.	A clear white fluid came out extremely slowly. Cells 4-5 to h.p. field.

* Indicates that case is mentioned in text.

CASE 57.—Aged 42. He had had an alveolar sarcoma of the left testis and abdominal glands removed three years before his neurological condition developed. A mass of glands close to the spleen could not be removed at the operation, but this mass diminished in size in the succeeding months. However, some two years after the operation these masses became noticeably larger, and very soon after he had symptoms of numbness and weakness of the extremities.

The patient was admitted to the National Hospital for the Paralyzed and Epileptic, under Dr. Grainger Stewart's care, about three months after these symptoms began to show themselves. He then presented a flaccid paralysis of arms and legs, with slight sensory loss. His tendon jerks were diminished or lost, and except for some difficulty of sphincter control he resembled a case of multiple neuritis. Lumbar puncture gave 8 c.c. of clear watery fluid at normal pressure. The cell content was normal (10 to 15 lymphocytes) to the high-power field ($\times 350$), but the albumin content was greatly raised; by the Noguchi test 1.1 c.c. precipitate was deposited from 2 c.c. fluid, and by the picric acid test 1.3 c.c. from the same quantity of fluid.

The paralysis after increasing for some weeks became stationary, and he was discharged three months later in much the same condition. A diagnosis of meningeal infiltration by the alveolar sarcoma was made.

CASE 58.—A girl, aged 15 years, was admitted to the National Hospital, under the care of Dr. J. A. Ormerod, in January, 1912. Fifteen months before this date she had had some pain in the back, and within a few weeks lost the use of her legs and the control of bowels and bladder. She attributed this to sleeping in a damp bed. She was treated at the Charing Cross Hospital by Dr. F. W. Mott soon after her illness started, but without much benefit.

On examination the patient showed a spastic paraplegia with complete anaesthesia up to the ninth dorsal segment. There was no tenderness or irregularity of the spine, and the Wassermann reaction of the

blood was negative. The cause of the paraplegia was very doubtful, as she showed no stigmata of congenital lues. Lumbar puncture was performed three weeks after admission, and a clear, colourless fluid was obtained, which came from the needle extremely slowly, only two or three drops per minute. It was impossible, even by raising the patient's head, to collect more than 4 c.c. of the fluid, and of this 2.0 c.c. were lost owing to the centrifuge jamming. The remaining 2 c.c. gave a precipitate of 2.2 c.c. by the Noguchi method. The cell content amounted to 4 to 5 cells to the high-power field (collected from 4 c.c. of fluid).

The diagnosis of compression of the cord, probably by a meningeal tumour, was then made, and an exploratory laminectomy advised, but the patient's parents would not give their consent to the operation.

This condition of the fluid (massive coagulation and xanthochromia) had been noted by Sicard and Descomps in 1908 as occurring in syphilitic meningitis, associated with a great increase in the lymphocyte content. Other French writers have reported cases, Mestrezat and Roger in 1909, and later Cestan and Ravant and others. The condition is fairly common and is probably well known to English neurologists. Of this I have seen two cases, one (No. 15) in whom the condition was very marked, another (No. 16) in whom it was very slight. It is considered to be due to loculation of the fluid by adhesions and thickening of the pia-arachnoid in the dorsal region of the cord, but the pathology of the condition is still obscure.

It may be noted that in both the French and American cases mentioned above the cord was compressed by a limited tumour, which in one case, at any rate, had produced meningeal adhesion, and thus cut off the lower portion of the subarachnoid space from free communication with the ventricular fluid. Apelt has recorded two cases of extradural tumour producing a similar effect on the fluid below their level, and Nonne one of intramedullary glioma. Heilig explains this effect on the spinal fluid by the vascular congestion which takes place below a lesion of the cord, the object of which may be the removal of the products of degeneration.

In one of my cases (No. 58) the tumour or compression was of a limited nature, but in the other two cases (Nos. 56 and 57) the condition of the fluid might have been explained by transudation from a vascular tumour infiltrating the meninges widely. The occurrence of a similar fluid in cases of cerebral tumour, as reported by Quincke and Vincent, indicates that the damming up of the free circulation of the fluid has much to do with the production of an albuminous yellow fluid in the portions of the subarachnoid space shut off from the ventricles, as it is very possible that in these cases the foramen magnum was plugged by the downward pressure on the brain stem forming the so-called "pressure cone." Whether this condition is compatible with life for a long enough period to allow the spinal fluid to undergo such changes is, of course, doubtful, but this hypothesis harmonises with the other causes—spinal tumour, syphilitic leptomeningitis, &c. At the same time, if this were the only explanation, one would expect a similar result in hydrocephalus, but although in such cases the spinal fluid must have been examined very many times this condition of the fluid does not seem to have been found.

It is possible that the presence of the tumour may in some cases of itself raise the albumin content, especially in the fluid shut up in a limited area of the subarachnoid space.

The yellow colouration seemed to me to be similar to what was found in cases of cortical hæmorrhage (see Table II.), but a full chemical examination was not carried out in any of the cases. French writers have examined the fluid in many cases of fracture of the skull, cortical hæmorrhage, &c., where this yellow colouration was present. Bard and Sicard consider it to be due to a special decomposition of the hæmoglobin of the blood. Gilbert and Hersher, Fuffier and Milian, on the other hand, consider it to be due to a pigment, serochrome, normally present to greater or less extent in the blood. That the pigment arises from the blood seems undoubted. In my cases the presence or absence of this pigment had little relation to the quantity of albumin. It was present with readings of 4.3, 1.4, and 1.0 c.c., and absent with readings of 1.1 and 2.2 c.c. The yellow colouration, therefore, does not seem to be an essential part of the syndrome.

It is interesting to observe from a practical point of view that the finding of a high albumin content in the cerebro-spinal fluid (above 1.0 c.c. from 2 c.c. by the Noguchi test) seems to be in almost every case an indication for operative treatment. Where the cause is syphilitic meningitis much

good may be done by promoting free circulation of the cerebro-spinal fluid to the lumbar segments of the cord, and where a level of anæsthesia or paraplegia is found the operation is frequently followed by very good results. In such cases a cyst of fluid is often found at the operation, pressing on the cord and practically acting as a tumour.

Apart from syphilitic cases, the diagnosis is limited to spinal tumour, or other compression paraplegia—e.g., Pott's disease, fracture, dislocation, &c.—where the clinical history would make the diagnosis simple. Pachymeningitis cervicalis hypertrophica might possibly cause a similar condition of the fluid, and in this condition, as shown by Horsley's results, operative treatment is often extremely beneficial.

As a diagnostic sign of spinal tumour, examination of the cerebro-spinal fluid seems to have been too much neglected, and in the absence of other evidence may be of the greatest value. As compared with other methods of differential diagnosis in cases of paraplegia, lumbar puncture and examination of the fluid requires very little neurological experience, and is not beyond the scope of a general practitioner's laboratory.

In conclusion, my thanks are due to the medical staffs of the East London Hospital for Children, Shadwell, and of the National Hospital for the Paralysed and Epileptic, Queen-square, and to Dr. A. G. Barrs for permission to make use of their cases.

Bibliography.—1. Apelt: Berliner Klinische Wochenschrift, No. 33, 1910, S. 1540. 2. Bard and Sicard: Gilbert and Hersher: Cestan and Ravant quoted in "La Pratique Neurologique" (P. Marie). 3. Blanchetière and Lejonne: Gazette des Hôpitaux, 1909, vol. lxxxii., p. 1303. 4. Wm. Boyd: Brit. Med. Jour., 1909, i., 1352. 5. Cooper: Journal of the American Medical Association, 1910, p. 2298. 6. Heilig: Monatschrift für Neurologie und Psychiatrie, Heft 2, 1911, p. 95. 7. Kleiberger, *Ibid.*, Okt., 1910, S. 346. 8. Mestrezat and Roger: Comptes Rendus de la Société de Biologie, 1909, vol. lxvi., p. 1000. 9. Nonne: Deutsche Zeitschrift für Nervenheilkunde, Band xl., Hefte 1-2, 1910, p. 161. 10. Quincke: *Ibid.*, p. 78. 11. Sicard and Descomps: Gazette des Hôpitaux, 1908, vol. lxxxi., p. 1431. 12. Cl. Vincent: Informateur des Aliénistes et des Neurologistes, No. 5, p. 147, May 25th, 1912.

THE QUANTITATIVE CUTANEOUS TUBERCULIN TEST.

(QUANTI-PIRQUET (Q P) FOR SHORT.)

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THE diagnosis of the existence in the body of tubercular disease in need of treatment is one of the most difficult of clinical problems, and the solution thereof would tend in no small measure to the simplification of the treatment of the disease. There is the need, on the one hand, of avoiding the fatal delay in waiting for striking symptoms to appear, when the time for efficient treatment may be past; and, on the other hand, of critically examining any criterion, such as the subcutaneous tuberculin test as commonly applied, to see whether the mesh is not too narrow, and latent and arrested cases as well as active ones included.

The tuberculin test should, however, if rightly applied, give the desired indication, for there seems no doubt that the grade of the reaction of the body towards tuberculin—its tuberculin-sensitiveness—is in general an index of the amount—i.e., the activity—of the disease present. The amount of this sensitiveness can in theory be measured by any of the tuberculin tests—conjunctival (Calmette), subcutaneous (Koch), cutaneous (von Pirquet), percutaneous (Moro), &c.—but the great superiority of the cutaneous test lies in the fact that it can be simultaneously applied in various strengths and a quantitative result obtained: (a) without waste of time; (b) with perfect safety; (c) without in itself altering what it is desired to measure; for any subcutaneous dose itself gives rise to an alteration—it may be temporary—in the sensitiveness.

Von Pirquet himself pointed out the possible quantitative application of his test, but it is comparatively recently that two Danish observers, Ellermann and Erlandsen^{1, 2}, have worked out an ingenious method in which the sensitiveness can be determined by a series of simple measurements and the result expressed numerically, thus affording an entirely objective standard of comparison from case to case. The original papers have not attracted the attention which they