concealed haemorrhage, I at once applied my hand over the uterus and found it tightly contracted, while the pulse was of excellent quality. Without any apparent reason, and in spite of energetic stimulation, the patient grew slowly worse, the pulse becoming rapid and weak, the eyes sinking back in their sockets, and the face assuming a drawn and Hippocratic expression.

"Thorough examination failed to reveal the slightest cause for the condition. The hand introduced into the uterus could find no trace of rupture. Eight hours after delivery I requested a colleague to see her in consultation, but he also was unable to offer any explanation. It then occurred to me that the condition might possibly be the result of her morbid forebodings, and acting upon that supposition I administered a large dose of morphine hypodermically, which was promptly followed by sound sleep, a marked improvement in the character of the pulse, and some rapid change for the better in the general appearance. Upon awakening a few hours later the patient felt very comfortable and made an uninterrupted recovery."

From my own experience I can record a case which had a tragic ending. These are my notes.

M. W., aged 29, primipara. On April 25th, 1925, attended by her family doctor, a careful and experienced obstetrician. I was called in after the second stage of labour as the patient had collapsed. Labour, expected on the 27th, started on the 22nd. Poor pains, on the 23rd and 24th, were treated with morphine, hyoscine, and chloral. The membranes ruptured on the 25th at 9 a.m. The cervix was then fully dilated and quite effaced. At 11 a.m. 1/2 c.cm. of pituitrin was given. The feeble contractions increased slightly, but were not efficient. About 12 o'clock low forceps were applied under light chloroform anaesthesia induced by the doctor himself. The pelvic floor was very rigid, and twenty minutes were occupied in extracting the child. The patient collapsed very shortly afterwards. There was no haemorrhage, and only a slight tear of the perineal body. As there was no obvious cause for the collapse, and as the placenta had not come away, the doctor explored the vagina and cervix with his hand. He could feel that the placenta was still in the uterus, but, the patient's condition being very serious, he considered it unwise and probably unnecessary to remove it. He sent for assistance and started a submammary infusion of saline.

I arrived at 1 p.m., less than an hour after the delivery. The patient was then very pale, pulseless at the wrist, but not at all restless. The uterus was as high as the umbilicus, not relaxed, but contracting only slightly and at long intervals. There was no bleeding. She was at once given 1/2 c.cm. of omnopon, and glucose was added to the saline. She rallied slowly. By 4 p.m. her colour had returned, and the pulse was countable at the wrist—120, very small. At 7 p.m. the pulse was still 120, still very small, and the patient was sleeping. The condition of the uterus was unchanged. At 10 p.m. the doctor injected ergotamine hypodermically, as a "safeguard" for the night.

I saw her again the next morning at 11 o'clock; she had had a restless night and had passed two very small clots, without pain. The temperature was 98.4° F.; it had been up to 99° F. Her condition was much better. Her colour had returned and she talked freely. There was no abdominal pain nor rigidity. The uterus was unchanged. Before considering the question of removal of the placenta, we decided to move the patient into hospital (her mother had been taken to hospital for removal of adherent placenta after the patient's own birth). The patient readily agreed, but while talking quite cheerfully about the necessary arrangements her face suddenly set, her eyes stared (we thought a fit was impending), and she died in two minutes or less. Traction on the tongue, artificial respiration, strychnine injected hypodermically and into the heart, all failed to revive her.

Afterwards I inspected the vagina and cervix, but found only a moderate tear of the perineal body. I passed my hand into the uterus, but found no rupture. The placenta peeled off with no unusual difficulty. The uterine wall was thin and relaxed. There was no sensation of free fluid in the pouch of Douglas. The ultimate diagnosis was death from emotional shock.

The patient was of gipsy type, very deeply pigmented, and she was said by her father to be of a very emotional temperament. I have seen the statement made that darkly pigmented people are specially vulnerable to shock, whether physical or

mental. Unfortunately no record of the blood pressure was taken, and no post-mortem examination of the pelvic floor muscles was made. It is noteworthy that the pelvic floor was described as being very rigid.

The exact cause of death in these cases is not known. Possibly direct cardiac inhibition occurs, but it has been shown experimentally by Cannon¹ and others that mental emotion will stimulate the suprarenal gland to excessive secretion. Now although adrenaline causes a prolonged vaso-constriction in skin vessels, it also produces "an increase in the volume of blood present in voluntary muscle and in the intestine." This has been demonstrated by Dr. G. A. Clark¹⁰ in the physiological department of Sheffield University. We have seen that in traumatic shock the capillaries of muscles are relaxed. The effect of adrenaline either injected into or secreted by a patient already or recently the subject of traumatic shock, will therefore be harmful and possibly even fatal.

In the fatal case just described the placenta was retained. The relationship of shock to retention of the placenta is not quite clear. Personally I prefer the view that it is the shock which paralyses the uterus and causes retention of the placenta to that which holds that the adherent placenta promotes shock. Hence I agree that no attempt should be made to remove the placenta while the shock persists, unless, of course, partial separation leads to haemorrhage.

Finally, we are left with the prevention of emotional shock. This is too wide a subject to deal with here. It is perhaps best summed up in careful and considerate ante-natal care whereby the woman is helped to approach her confinement with a mind freed from anxiety and apprehension.

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CEREBRAL CORTICAL STRUCTURE AND ITS RELATIONS TO MENTAL DISEASE

BY

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It seems reasonably safe to assume that the average medical man has little or no knowledge of the intricacies of the histological construction of the brain, now technically known as cyto-architectonics, or of the significance and practical utility of these developments in general and special practice. Yet English law may compel every medical man to certify, when necessary, cases of mental deficiency and mental disease; and the most recent Act of all implies a hope that more cures may be effected than heretofore. The profession is thus at a double disadvantage; and so it may be helpful if some brief account be given, as simply as possible, of recent advances in the study of brain structure and its relationship to mental disorder.

HISTOLOGICAL STRUCTURE

The microscopical study of the cells of the human brain is obviously attended by many and great technical difficulties, and is thus beyond the efforts of any but a limited and fortunate few. The work of these few has, however, had far-reaching results, culminating, at the moment, in the work of von Economo of Vienna, whose estimate of the number of brain cells in the normal healthy cerebral cortex is 14,000 millions, or some 5,000 millions more than the previous estimate of Donaldson of the Wistar Institute. These millions of brain cells are not arranged haphazard, as so many modern histological textbooks might lead one to suppose, but are definitely located in layers or laminae, of which English observers, like Bolton, Watson, Mott, and others, have recognized five.

It is perhaps not so well known as it might be that mammals are the only animal forms with a definite neo-pallial cerebral cortex or true end-brain. As the mammalian scale is ascended from the marsupials, through the anthropoids, up to man, this neo-pallial cerebral cortex becomes increasingly complex, culminating in the human brain. Originally a three-layered histological cortex, it becomes, in the higher anthropoids and man, a five-layered one. The successive results of these increments are to be seen in the enormous number of cells in the human brain, far exceeding that of any other mammal. In all mammals, however, these brain cells are arranged in a laminated manner, and the five of man were described and named by Shaw Bolton in 1912 as follows:

1. The outer fibre lamina.
2. The outer cell lamina, chiefly composed of pyramidal cells.
3. The middle cell lamina of granular or minute Golgi Type 2 cells.
4. The inner fibre lamina of axons.
5. The inner cell lamina of polymorphic cells.

From his investigations of the mammalian cerebral cortex Watson came to almost similar conclusions; but he pointed out that the mammalian neo-pallial cerebral cortex is primarily built up on an infragranular basis—that is, of layers lying within or internal to Bolton's third layer. He therefore simplified the nomenclature of the cerebral laminae by suggesting that the cortex should be considered as consisting of three main functional divisions: an internal infragranular cortex composed of polymorphic cells, an external supragranular cortex composed of pyramidal cells, with a third or granular layer between the other two, sharply delimiting the one from the other. There is no part of the body where structure so strictly accords with function as the nervous system, and that some significant functional importance attaches to these three contrasted forms of cells is certain. The views of Mott, Watson, and Bolton thereon are well known, and have been substantiated by others. I do not propose to give a detailed account of the work of these and other investigators, but to give a general introduction to von Economo's more recent researches.

Von Economo's smaller work, in 1929, on the cyto-architectonics of the human cerebral cortex is, in the main, confirmatory of the previous work of the English observers, though naturally differing in details. It extends the application of cortical cytology to other forms of mental disorder and disease, and indicates the different areas and laminae affected in some of the psychoses. Such a work is therefore of importance to the profession generally. Von Economo differs from English investigators inasmuch as he describes the cerebral cortex as a six-layered structure, not a five; and derives this extra layer by regarding the small pyramidal cells of the outermost part of Bolton's second layer as a separate and additional granular layer. These differences in arrange-

ment and nomenclature will be rendered clear by Fig. 1, and by the comparative table below.

FIG. 1.—Layers of the cerebral cortex, as described and named by von Economo (on the left), and by Shaw Bolton (on the right).

BOLTON	WATSON	V. ECONOMO
1. Outer fibre lamina	Supragranular	I. Molecular layer
2. Outer cell lamina, chiefly pyramidal cells		II. External granular layer of fine pyramidal cells
3. Middle cell lamina, chiefly minute granule cells of Golgi Type 2 variety	Granular	III. Pyramidal cell lamina of numerous large pyramidal cells
4. Inner fibre lamina	Infragranular	IV. Internal granular layer
5. Inner cell lamina of polymorphic cells		V. Ganglionic layer, chiefly fine pyramidal cells
		VI. Layer of fusiform cells

It is to be specially noted that von Economo bases his classification of cortical laminae on cells only, whereas previous English investigators have included the well-defined strata of incoming or outgoing axons. Again, it will be observed that he separates off the small pyramidal cells forming the outermost part of Bolton's outer cell lamina as a separate layer of granules; a new point of some significance, as it means that the supragranular cortex is provided with a receptor layer as well as the older infragranular cortex. Against these minor differences is the major fact that all observers agree in regarding the human cerebral cortex as being a laminated structure with three main types of functional cells (pyramidal, granular, and polymorphic or fusiform), and that some functional significance attaches to each of these three principal strata. In von Economo's own and most recent words:

"In applying our present knowledge of cyto-architectonics to cerebral pathology, we must remember the following principles. The three chief forms of cells of the cortex (pyramidal, granule, and fusiform cells) are arranged in horizontal layers. The cortex consists of six such laminae, each of which has probably a certain function in the physiological activity of the grey matter."

LOCALIZATION OF FUNCTION

While it is undoubtedly true that certain portions of the cerebral cortex seem to specialize in particular functions, the conception of brain centres obtained from most textbooks of medicine is extremely misleading; and Broca's area, still unhappily described as the speech centre, forms an example of the persistence of an antiquated ignorance of cerebral function. Speech probably involves many portions of the cortex and myriads of cells in all layers. Notwithstanding this, certain portions of brain cortex do have specialized functions, as is proved by the findings of cyto-architectonics. Brodman, Campbell, Marinesco, Betz, and many others, divide the cortex, from the known arrangement of its cells, into as many as upwards of a hundred areas. It suffices to say, with von Economo, that there are at least five main structural types of cortical cell construction and lamination. Thus the area known generally as the Rolandic motor area is largely devoid of granule cells, which it does not need, because it is concerned with outgoing cerebral impulses. This comparative absence of granule cells enables us to recognize such portions of the cerebral cortex under the microscope with considerable facility, and von Economo calls these areas the agranular cortex. Areas, on the other hand, which are primarily concerned with the receipt of impulses from the chief exteroceptive senses, such as sight, hearing, taste, and touch in all its forms, will be particularly rich in granule cells, thus forming easily recognizable cortical areas, termed granular cortex. With these exceptions, the rest of the cortex is mainly made up of slight, but recognizable, variants of the five or six cellular layers. Correlating these facts with clinical conditions the Viennese and other Continental schools find that mental disorder is rarely associated with area lesions, but shows a predilection for certain laminae in one or other of the five types of cortex.

PATHOLOGICAL CHANGES

With the recognition of these five or six cortical layers of the brain there has subsequently accrued, especially as the result of English work, a considerable knowledge of their pathological alterations in association with certain mental disorders, especially in mental deficiency. It is now well established that mental deficiency results from a lack of development of the cells of either or both of the pyramidal and fusiform cortical layers, with a consequent diminution in their numbers, a disordered structural formation, a consequent microcephaly, and disordered psychological reactions to the environment best summed up, perhaps, by a single word, unsanity. Bolton, in his classic work on *The Brain in Health and Disease*, extended and applied our knowledge of cortical lamination to both the amentias and the dementias; and von Economo has been able to go even further in applying it to both psychology and psychiatry.

The agranular cortex, for example, shows no change in general paralysis, which chiefly affects some of the cell laminae in the anterior frontal lobe of the brain, parts of the parietal and temporal lobes, and certain other sites. According to Jakob, the majority of organic mental diseases do not attack areas of the brain, but show a widespread affection of the cortex with a predilection for certain regions. In demential psychotic disturbances of the personality, von Economo finds that the chief lesions are in the third cortical layer. On the other hand, those diseases which primarily affect the capacity for expression and motility show a particular affinity for the cells of the fifth and sixth layers. In those cases of senile dementia which show a striking extrapyramidal muscular rigidity, in addition to their psychic symptoms, von Economo finds a marked affection of the cells of the fifth and sixth

laminae. In many other cases of dementia there is not only a destruction of cortical cells, but also a proliferation of the glial cells, as instanced by von Economo, and also found in a case now under examination from a recent post-mortem at Stoke Park Colony. Mental deficiency, on the other hand, does not result so much from a dissolution of brain cells, as from their inherent inability to develop, many of them remaining in the neuroblastic condition; and feeble-mindedness is, according to Hammarberg, a closely allied condition of inhibition of proper cell development.

FIG. 2.—Cortical areas of von Economo. 1. Agranular cortex, chiefly confined to motor areas. 5. Granular cortex, chiefly found in sensory areas. 2. Frontal type. 3. Parietal type. 4. Polar type. Nos. 2 to 4 variants of the type illustrated in Fig. 1. No. 3 in the diagram corresponds to focal points of intelligence.

But cyto-architectonics is not of importance only to the psychiatrist. It promises results for the psychologist also, inasmuch as cellular studies of the cortex suggest that there are present richly receptive focal points of intelligence (von Economo) in both the prefrontal and parietal areas; but again the warning must be sounded that "for every kind of psychic expression the functioning of the entire cerebrum is necessary."

Thus does the apparently dulllest and most forbidding of subjects, cyto-architectonics, hold out the greatest hope for the future of mental science in all its branches. And indeed it cannot now be long before the brain gives up the last of its secrets—the fashioning of mind out of the functions of neurones and their synapses.

ACIDOSIS AND SEBORRHOEA

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The problem of seborrhoea is one of great importance and complexity. The minor manifestations of this disorder are so common as to be almost universal, while the major dermatoses of this group form a very large part of all skin cases seen in private and hospital practice. The subject is therefore one of interest to the general practitioner as well as to the dermatologist, and merits investigation from more than one point of view. MacLeod¹ and Dowling² have added much to our knowledge of the organisms concerned in the production of some of these disorders, but the seborrhoeic diathesis has received little attention, except from the clinician.

Three factors play a part in the production of most seborrhoeic affections—namely, a constitutional, an infective, and a precipitating factor. A brief explanation of each of these features is necessary.