where impotence or a disorder of ejaculation had persisted from the first attempt at heterosexual coitus. If a constitutional factor is of importance in the aetiology of these disorders, then it may be more relevant in cases of early onset than in cases of late onset. In the latter group, where the disorder of sexual potency developed after a period of normal heterosexual coitus, the aetiology was more often part of a psychogenic reaction to environmental factors. In Table III the androgyny scores of early-onset impotence and late-onset impotence are compared with the control group. A significant difference is seen to exist between the androgyny measurements of the early-onset and control groups.

TABLE II.—*Comparison by "t" Test of Neurotic and Non-neurotic Patients with the Control Group*

Group	Mean	S.D.	Compared with Control	
			t	P
Control (N = 70)				
Androgyny score	90·8	4·9		
Disorder of potency (N = 55)				
Neurotic constitution (N = 31)				
Androgyny score	87·0	6·0	3·1	0·001
Normal constitution (N = 24)				
Androgyny score	88·8	5·6	1·6	0·10

TABLE III.—*Comparison by "t" Test of Early-onset and Late-onset Impotence with Control Group*

Group	Mean	S.D.	Compared with Control	
			t	P
Control (N = 70)	90·8	4·9		
Early-onset impotence (N = 13) ..	87·5	5·1	2·2	0·02
Late-onset impotence (N = 22) ..	88·9	5·4	1·5	0·1 N.S.

There is some evidence from this study to suggest that abnormalities of physical constitution relating to sexual dimorphism are present in patients with disordered sexual potency. This constitutional factor, however, appears to be closely related to the determinants of neurotic constitution in general: a similar conclusion was arrived at by Coppen in his study of homosexuals. Since neurotic constitution occurred significantly more often in the early-onset group ($\chi^2 = 4.24$, $P < 0.05$), the same general factor for neurotic behaviour might account for the difference in the early-onset and late-onset groups.

Constitutional factors similar to those which determine neurotic behaviour may be relevant to the aetiology of disorders of sexual potency, particularly those of early onset. Such factors might account for the poor prognosis for recovery of potency which was found at follow-up in this group (Johnson, 1965). Although constitutional factors are usually thought to have a complex genetic basis, it seems not unreasonable to assume, as Tanner (1964) suggests, that they may also be due in part to the "ineradicable effects of early experience."

Summary

The androgyny scores of 55 males referred to a psychiatric hospital because of a disorder of sexual potency were compared with a control group of normal males. A significant difference in androgyny was demonstrated between the groups, with a tendency to gynandromorphy in those with impaired potency. This difference was more significant in the "early-onset" disorders and in those with neurotic constitution. These findings lend support to the suggestion that constitutional factors may be important in the aetiology of some of these disorders.

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Preliminary Communications

Brain Development and the E.E.G. of Normal Children of Various Ethnical Groups

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In any group of young children some appear to be more advanced than others, at least in certain aspects of their development. In a mixed population individual variations may be very wide, and ethnical factors may complicate evaluation of the results of investigation. From the work of Faladé (1955), in Senegal, and of Geber (1956) and Geber and Dean (1957, 1964), in Uganda, the psychomotor and somatic development of both newborn and young African children seems to be much more advanced than that of European babies of similar age-groups. These authors' observations were based on the technique of neurological examination utilized by the pupils of André Thomas. Their extensive work has recently been confirmed by the studies of E. M. Brett (personal communication, 1965) in Europe. Both genetic and environmental factors seem to influence subsequent development, and psychometric tests based on European or American culture in later childhood may not be applicable to African children (Geber and Dean, 1964).

Competent clinical observation is usually the best method of rapidly detecting similarities and differences in individual performance. It is, however, difficult to standardize clinical observations of which no permanent record has been made, which depend on the skill of the examiner, and which are not amenable to further processing. At present the terms "cerebral function" and "higher nervous activity" have to be considered as concepts rather than as known quantities. Some physiological aspects of cerebral function, however, may be measured, and among these the electrical activity of the brain has been extensively employed in developmental studies, both in animals and in man. Unfortunately there has been little uniformity in the techniques used by different workers in various countries, and the statistical methods of analysis applied to the evaluation of the recorded data tend to blurr individual variations.

Brain development, and that of its functions, are often regarded as linear processes which, like height or weight, follow, during body-growth, either a smooth curve or a straight line,

according to the mathematics applied. In the dog, however, maturation of the electrical activity of the brain appears to take place in a series of steps, particularly in the first six months of extrauterine life (Pampiglione, 1963). Moreover, in some regions of the canine brain the patterns of electrical activity evolve in a way that differs from that of other cerebral regions in the same animal.

It has been suggested (Grafstein, 1964 ; Dreyfus-Brisac, 1964 ; Ellingson, 1964) that the maturation of some electroencephalographic (E.E.G.) patterns in kittens, in premature children, and in normal newborn babies might be a somewhat uneven process. In recent combined neurophysiological and histological observations it has been shown that dendritic growth in the cerebral cortex and elsewhere runs parallel to some of the steps in the maturation of electrical cerebral events in young kittens, rats, and rabbits (Purpura *et al.*, 1964 ; Schadé and Pascoe, 1964).

Very little information is available regarding the ranges of individual variation in the patterns of electrical activity of the brain in young children and babies at different ages. In the present study an attempt has been made to assess whether the evolutionary steps in the maturation of the electrical activity might be strictly related to age, or whether some genetic factor might to a certain extent determine individual variations in the electrical milestones. A total of 40 children are under consideration in this paper, while further long-term studies are being envisaged.

Material and Method

The investigations were carried out on a mixed population of babies and children living in the Greater London area: 19 were of European (9 Anglo-Saxon, 10 Mediterranean), 4 of Indian, and 17 of African negro origin.

Serial E.E.G. studies were made up to six times in each child over a period of 6 to 10 months from the first test ; the ages of the children ranged from 5 months to 3 years.

These 40 children had been selected from a larger group, the criterion being that, from the clinical information available, it was unlikely that gross brain disorders were present. No attempt was made to standardize the many environmental factors that might have influenced each child's development. The E.E.G.s, however, were taken in comparable circumstances in all the children, who were left as free as possible. Sedation was never given. The electrodes (silver–silver chloride disks fixed with collodion) were placed on the scalp according to measurements from bony landmarks (Pampiglione, 1956). Each test lasted between 20 and 40 minutes. All the E.E.G.s were taken with a portable eight-channel Offner type T apparatus, and included babies and children resident in the Greater London area, as well as my own children and those of friends. The consent of the parents or guardians had been obtained in all cases.

The E.E.G. data were classified according to extrauterine age at the time of each test and to the apparent ethnical group to which the child seemed to belong (inspection, supplemented by certain details in the family history).

Results and Discussion

All the E.E.G.s were classified in terms both of frequency of the rhythmic components and of their distribution over the cerebral hemispheres, during the waking state, after passive closure of the eyelids for a few seconds, and during the remaining part of the test when the child was looking around or playing. No systematic observations were made of the evolution of E.E.G. patterns during sleep.

Passive closure of the eyelids was a very strong stimulus in eliciting E.E.G. changes in all the babies and young children tested. The basic technique was that of the " peep-bo," familiar to all parents and nannies. It was essential that the eyelids should be completely closed and that the baby or child should not peep. An acceptable alternative was that, even if the eyes remained open, vision should be occluded behind a cloth, or a hand, covering them completely, the technique being similar to that illustrated in the study of young dogs (Pampiglione, 1963). Usually within a third of a second from the occlusion of vision a considerable amount of rhythmic activity appeared in the E.E.G., recorded from electrodes placed in the occipital region. Although in some children similar activity could be recorded also from the posterior temporal and parietal electrodes, a midline occipital electrode placed about 1 cm. above the inion was essential for detection of the characteristic E.E.G. changes elicited by the passive occlusion of vision in all the babies examined.

In the serial E.E.G. studies it was noticed that, while in both European and Indian children a definite alpha rhythm at 8–9 c/s did not appear until the age of 24–28 months, in most African negro children this maturational change was already present by the age of about 18–24 months. The amplitude of this activity, usually confined to the occipital region in this age-group, was of the order of 50–100 microvolts, there being no substantial differences between the various ethnical groups. In all children the alpha rhythm was promptly blocked on opening the eyes.

In babies who had not reached the age at which a definite 8–9 c/s alpha rhythm appeared, a well-formed sinusoidal rhythmic activity at about 5–6 c/s in the occipital region was observed after eye-closure. This activity was already present by the age of 5–7 months in the African negro children, while in most European children it was not seen in this frequency range before the age of 9 months. The amplitude of this activity, which was promptly blocked on eye-opening, was of the order of 100–200 microvolts in the occipital region.

It is suggested that the age at which changes occur in the frequency of the rhythmic activity elicited by passive eye-closure should be regarded as a critical period in the process of maturation of the electrical activity of the brain. It therefore seems probable, from the observations made on this group of children, that some individual differences in the rate of E.E.G. development might be related, among other possibilities, to an ethnical or genetic factor which affects cerebral maturation in a way yet to be understood. These preliminary studies, which were made on a small sample of babies and children because of the limited research facilities available, will have to be checked on larger numbers.

Should the above findings be confirmed they might add some measurable criteria to our existing theories of brain function and learning in young children. Through carefully combined studies on the same children it should be possible to assess whether early maturation of the electrical activity of the brain might be related to early acquisition of motor skills, or intellectual abilities, or of particular types of emotional response.

In the absence of special facilities these observations had to be made in the course of other neurophysiological studies over the past four years, some with the support of the Polio Research Fund (mobile unit for the study of encephalitis), some with the apparatus of the Medical Research Council (E.E.G. studies in measles), and some with the support of the Clarissa Norman Research Fund ; I am indebted to all the technicians who helped with these projects.

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Medical Memoranda

Acute Massive Gangrene of Tongue

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Gangrene of the tongue with sloughing of the gangrenous area due to atherosclerotic arterial occlusion appears to have been reported only once previously in the British literature—by Bergan (1959). The rareness of the condition is because of the rich arterial blood supply of the tongue. The chief blood supply is by the lingual artery; however, the ascending pharyngeal artery and the external facial artery contribute deep branches to the tongue.

Case Report

The patient, a woman aged 72 years, was seen on 6 December 1962. She complained of aching pains in the temporal areas bilaterally for four weeks. The pains appeared to radiate into the jaws and were associated with vague aches in the posterior cervical area; they were intensified by chewing. There were no significant findings on physical examination; the mouth could be opened fully. The temporal arteries were pulsating, palpable, but not tender. The blood-pressure was 140 mm. of mercury systolic and 80 mm. of mercury diastolic.

The patient was a known epileptic of long standing under control with phenacemide (Phenurone). In 1954 she developed mild exertional angina. In 1962 she was treated for sub-thyroidism with thyroxine.

The pains were thought to be due to a mild temporo-mandibular arthritis and treatment was started with local heat and oral salicylates. No improvement occurred and she was admitted on 14 December 1962 to St. Luke's Hospital for further investigations.

After admission she developed tenderness over the bifurcation of the carotid artery on the right side. Investigations showed a normal skull *x*-ray and radiographs of the temporor-mandibular region were also normal. Examination of the blood showed a haemoglobin of 12.1 g./100 ml.; white cell count 6,700 per c.mm. with a normal distribution. The erythrocyte sedimentation rate was 54 mm. in one hour (Wintrobe).

The patient had a low-grade pyrexia for the first four days after admission with a maximum reading of 99.2° F. (37.3° C.). She was treated with oxyphenbutazone 25 mg. six-hourly, with no significant improvement.

On 22 December she complained of difficulty in speaking and her speech was found to be slurred. It was felt that this was due to a minor cerebral ischaemic episode. Muscle power, sensation, and co-ordination were normal, as were the deep nervous reflexes. This episode lasted a few hours and cleared completely.

During the subsequent week the patient was reluctant to chew as the pain intensified in the jaws and in the temples. On 28 December she complained of a sudden intolerable pain in the tongue and face; her speech became unclear. On examination there was cyanosis and coldness of the skin of the centre of the mandible; the lower lip, upper lip, and tip of the tongue were deeply cyanosed. The tongue began to swell rapidly. The pulsation of the temporal arteries had disappeared and the facial arteries could not be felt. Examination of the central nervous system was normal, the blood-pressure was unchanged. The pain was eased by intramuscular pethidine and the tongue became numb.

A diagnosis of vascular occlusion involving the lingual artery was made with reluctance, in view of the known vascularity of the tongue and lips. A bilateral stellate ganglion block was performed; intramuscular papaverine hydrochloride and intravenous heparin were started.

Twelve hours after the infarction good circulation was present in the lips and mandible; however, the tongue was mottled and grossly oedematous. A pulse could be felt in the temporal arteries. It was impossible to inspect the pharynx but the airway was clear. It was decided that carotid exploration was not indicated.

The patient was kept on oral fluids, oral antiseptics, Hibitane lozenges, intramuscular papaverine and penicillin, and intravenous heparin 5,000 units every four hours.

By the fourth day the tongue was grey but less oedematous. The patient was able to speak with difficulty and to swallow some fluids. She was free of pain. There was profuse salivation which made her mop her lips frequently. At the end of 10 days heparin was discontinued and phenindione therapy started. The prothrombin index was maintained at about 30%. The haemoglobin was 11.8 g./100 ml. and white cell count 7,100 per c.mm. The erythrocyte sedimentation rate was 34 mm. in one hour (Wintrobe).

The tongue was now a light brown colour, healthy vascular tissue could be recognized at its root, and posteriorly the infarcted area extended to within 1 cm. of the circumvallate papillae. The infarcted area started to slough from the edges (Fig. 1) and a gross oral foetor developed. The slough separated on the twentieth day aided by minor surgery, after which the tongue oozed blood and a blood transfusion of one pint (0.6 l.) of blood was given and anticoagulants were discontinued.

The slough (Fig. 2) measured 4.5 cm. in maximum diameter and 4.3 cm. in maximum width. Microscopically complete necrosis was present.

Progress was subsequently uneventful. The patient could tolerate a denture two weeks after removal of the slough and she was discharged on 6 February 1963. A photograph (Fig. 3) taken three months after discharge shows the remaining stump of the tongue.