In those cases in which microscopy of the lesion was carried out the results have been reported. That it was not done in every case is no great defect: microscopical evidence of chronicity is probably unreliable (Tanner, 1953). Clinical differentiation of acute from chronic ulceration is also arbitrary. At one end of the scale is the patient with severe symptoms for many years, who at laparotomy is found to have a minimal lesion; at the other is the patient—often elderly—who has no symptoms until haemorrhage or stricture ensues, and who is found to have a gross chronic ulcer. However, from a consideration of these cases it would appear that the earlier a patient dies the more acute the appearances (Case 1); and the longer he survives the more chronic these are (Case 4).

More important from a clinical aspect would seem to be the anatomical site of the ulcer (see Fig.) and whether its

Diagram showing the anatomical site of the ulcers.

base is formed by peritoneum (Case 1), pancreas (Case 6), or blood vessel (Case 4). Perforation or haemorrhage brings a grave prognosis to elderly patients who are convalescing from accident or surgery, and in six of these patients the main symptom was haemorrhage.

It is apparent from a study of these cases that there may be some warning that ulceration is taking place. There was none in Cases 1, 4, and 7 (all of whom died); but there was warning with Cases 2, 3, 5, and 6 (of whom one died), and it is possible that early treatment might have saved life.

Conclusions

Peptic ulceration after trauma may be regarded as the abnormal reaction of an ulcer-prone patient to a physiological stimulus. Until such a time as the physiological reaction to stress and trauma can be measured and safely regulated, the best way to deal with this complication would be by prophylaxis and by early recognition and treatment. Prophylaxis is difficult, as the ulcer-prone patient does not declare himself. It is not the practice of surgeons to ask patients awaiting consultation to fill in "pro formas" on personal habits and previous and family history; were it so, however, it would be worth asking, "Have you suffered much from indigestion?" or, "Do you take stomach powders, tablets, or mixtures?" and if the patient be over the age of 50 and a man particular heed should be paid to such an admission. A careful general history is as important in surgery as it is in medicine.

If such a history is elicited the regime after operation should not be too active, and inquiry should be made into upper abdominal symptoms. In addition to the daily examination of the calves, a hand might well be applied to the epigastrium. If relevant symptoms appear, antacids and atropine should be given, and if tarry stools are passed the matter should be reported immediately, a serious view taken, and arrangements made at once to collect serum for cross-matching in preparation for a transfusion of blood that may be needed with great urgency.

Summary

Eight cases are presented of ulceration arising as a complication of operation or injury.

This ulceration may arise *de novo* or may be an exacerbation of previous ulceration.

A watchful outlook should be maintained for epigastric postprandial pain; particularly in the early convalescent phase. Melaena and coffee-ground vomit must be recognized as alarm signals, and immediate steps should be taken to arrange for rapid blood transfusion if necessary.

Energetic medical treatment may well avert death from a complication which has a high mortality rate.

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RECENT EXPERIENCE OF ENCEPHALITIS IN CHILDHOOD

BY

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This communication is based on a clinical study of 93 children with encephalitis seen since 1946 in the children's wards of three hospitals associated with the Department of Child Health in the University of Durham. This experience has been so varied that it invites an attempt to analyse it.

The term "encephalitis" is used without attempting to indicate any particular aetiological factor, whether of virus, allergic, or toxic origin. What is meant, therefore, is the occurrence, in a previously healthy child, of the classical symptoms or signs of disturbed cerebral function, which, after the exclusion of bacterial and vascular conditions and of chemical poisonings, are presumed to be the result of non-suppurative disorder of the brain.

The patients fall into two groups: (1) those associated or following on a recognized infective condition (measles, 12; pertussis, 8; varicella, 3; rubella, 1; mumps, 1; herpes simplex, 1; acute rheumatic fever, 1; infectious mononucleosis, 1), of which there are 28; and (2) those of unknown aetiology with no obvious causative factor, of which there are 65.

In attempting to define the clinical pictures of these diseases, this material could have been dealt with in two

ways. First, the cases associated with known infections might have been analysed to see if each had a distinctive clinical picture, and those of unknown aetiology could then be related to or compared with them. Bearing in mind that encephalitis may be the only manifestation of infections such as mumps, measles, and rubella, studies such as those of Greenfield (1950) have been of immense value in indicating the variety of conditions which can give rise to encephalitis, but they have been concerned solely with fatal cases and, even so, have shown quite clearly that there has been no uniformity of the clinical pattern in the various pathological groups. Second, all the cases might have been analysed according to the pattern of the clinical illness, regardless of aetiology or pathology, to ascertain whether any types were discernible. It is realized that in the absence of virus and histological studies, cases of encephalitis lethargica, subacute inclusion encephalitis (Dawson, 1933, 1934), haemorrhagic encephalitis (Baker, 1935), acute haemorrhagic leuco-encephalitis (Weston Hurst, 1941), and polioencephalitis (McAlpine *et al.*, 1947; Brewis and Neubauer, 1948; Jennings *et al.*, 1949; Kelleher *et al.*, 1949; Barrett *et al.*, 1952) may be grouped together in this way. This second method of analysis was chosen because it reveals clinical patterns of illness, irrespective of aetiology, which offer guidance to the practitioner in giving a prognosis.

Four main patterns of illness have been distinguished, although in this attempt at classification it has not been possible to insist on rigid criteria, and many of the signs and symptoms of impaired function of the nervous system have been common to all. Only the more obvious differences in onset, course, and duration have been used, and in some minor respects overlap between the types has occurred; in addition to which many cases could not be classified and have been described as a heterogeneous group.

Type 1.—Cases presenting as a sudden cerebral calamity in a previously healthy child or following an indefinite prodromal phase. Convulsions were prolonged or repeated, and unconsciousness lasted several hours or even two to three days. The duration of the acute phase was short, death or returning consciousness occurring in two to three days.

Type 2.—Cases beginning usually with a non-specific and meningitic illness with pleocytosis in the C.S.F. and followed by the acute onset of unconsciousness, stupor, or somnolence of a few days' duration, from which a rapid or sudden recovery frequently occurred. Cranial nerve paralysis and lower motor weakness were often encountered and recovery was the rule.

Type 3.—Cases often with a prolonged and insidious prodromal phase of indefinite symptoms or occasional convulsions, with periods of well-being followed by gradual unsteadiness developing into gross ataxia, tremors, and continuous choreiform movements of the whole body, particularly of the eyes. The illness was long, and was associated with emotional instability. Usually these cases had been diagnosed as chorea or cerebral tumour.

Type 4.—Cases which resembled encephalitis lethargica, with a short prodromal phase, and a slowly developing somnolence or coma. This lasted several days and was followed by cranial nerve involvement and a slow return to consciousness, interrupted by mental and emotional instability.

Type 1. Sudden Onset with Convulsions and Unconsciousness

Illustrative Case.—A. G., aged 10. On previous day, shivering, restlessness, and vomiting; no headache. Later

unable to speak; convulsion with bilateral clonic movements lasting 1½ hours. Temperature 107.8° F. (42.1° C.) reduced to 101° F. (38.3° C.) by sponging. On admission gravely ill, unconscious and vomiting; limbs immobile and flaccid. Face constantly twitching; eyes deviated, coarse nystagmus. No meningeal signs; C.S.F., 22 lymphocytes. Reflexes absent; W.B.C., 32,300. Remained unresponsive, with sporadic twitching of right hand, fingers, and toes; later, tonic neck reflexes present; head and eyes deviated to right, with facial weakness and hemipasticity. Constant variation in physical signs. Rapid deterioration, death 33 hours after first convulsion. Necropsy revealed small perivascular haemorrhages and cuffing in right basal ganglia, frontal cortex, brain stem, and spinal cord, and also perivascular collections of round cells in areas of the occipital and parietal cortex. No neuronal changes.

There were 19 cases of unknown aetiology in this category, 13 of them being below the age of 2 years. In nine there had been prodromal symptoms of malaise, listlessness, slight cold, and restlessness for one to five days. Eleven patients had vomited, six had loose offensive motions, and two had abdominal pain, although the bowel disturbance did not usually continue into the illness. In addition there were three cases of measles, four of pertussis, one of mononucleosis, and one of mumps which were similar in type.

Convulsions, generalized in 21 cases and limited to twitching or spasms of the face or limbs in 7, varied in number from 2 to 50 before admission. They had been continuous in 17 patients, and after admission were still continuous in 13, of whom 7 died. All patients were unconscious or stuporous from the onset of the fits. The temperature was raised in all but five, a high rising temperature being associated with a bad prognosis. Meningeal signs were present in only four cases due to measles or where the aetiology was uncertain, but were present in five of the other six cases of known infection. The C.S.F., examined in 26 cases, was normal in 18, while there was a mild pleocytosis of 12 to 56 cells in the remaining eight, one of which was blood-stained. Of these latter cases five were fatal.

The subsequent course of the illness in the survivors was very variable. Convulsions recurred in six cases and lasted for a few hours; drowsiness or coma for a day or two, accompanied by retardation, aphasia, slurring of speech, spasticity, choreo-athetosis, and ataxia were seen. Often recovery was quick, the illness lasting a week or less in nine cases, one to two weeks in four, two to four weeks in five, and two months in one case. The longer the illness the more likely were sequelae to develop; two cases were left hemiplegic with fits, three mentally retarded, and one with fits. Hemiplegia occurred in three other cases, one a fatal case and the others recovered completely.

Nine patients died, usually after short illnesses—six of 48 hours, two of three days, and one of 16 days. The last-mentioned case was that of a baby who continued to have convulsions for 10 days after admission and died a few hours after ventriculography. At necropsy, infiltration and old haemorrhages into the arachnoid were the only findings. Perivascular demyelination and haemorrhage were found in the mid-brain of one patient and the cortical areas of another; while perivascular cuffing and neuronophagia of polioclastic type, petechial haematomata, and capillary thrombi with infiltration were the appearances respectively in the other three patients on whom necropsies were done.

Type 2. Acute Onset with Short Somnolent Phase and Paralysis

Illustrative Case.—D. D., aged 10. Headache, sore throat, and fever lasting one day; then well for five days. Relapsed with pain in neck, headache, and sleepiness. Vomited continuously next day, but rational and co-operative. Marked meningeal signs, nystagmus, and right facial weakness, diminished tendon reflexes. Temperature 101° F. (38.3° C.); C.S.F., 45 cells, mainly polymorphs. Subsequent deterioration with deepening coma lasting five days, bilateral facial palsy, weakness of left arm and right shoulder, and diffi-

culty in swallowing. Slow improvement; becoming rational, though incontinent, excitable, and difficult to control. Went home 18 days after admission with paralysis of right face, deltoid, and left thigh, and emotionally labile. Went through a difficult phase, being bad-tempered, excitable, and nervous; often rude and vulgar. Speech staccato; appetite voracious; thin and restless. Eighteen months later had steadied down, was doing well at school, and had recovered neurologically.

There were 15 cases in this category, all of unknown aetiology, all children in their early years, and most were encountered during outbreaks of poliomyelitis. Thirteen had some form of prodromal illness lasting two to eight days and consisting of headache, vomiting, fever, transient diplopia, disturbance of micturition, drowsiness, aphasia, and dysphagia. Fits were present in three cases, and in two cases siblings had a febrile meningitic illness about the same time. All but three were febrile. Meningeal signs were present in 12 cases, but in all there was a pleocytosis in the C.S.F.

The mental state on admission was variably described as: unconscious (2), stuporous (3), persistently asleep (3), retarded but rousable with difficulty (4), uncontrollable following three days' somnolence (1), conscious but aphasic (1), rational, becoming semicomatose for a week later (1).

Recovery of mental clarity occurred early: nine patients became conscious on or before the fourth day, four during the next three days, and only one took longer than a week. Sometimes this return of consciousness was very dramatic and sudden; a child having been deeply asleep or semicomatose for some days would waken in a few hours and become quite alert, would be found wandering round the cubicle, or, in one case, drinking out of the cubicle tap. Similarly, the return of speech followed the same pattern, and so striking was this apparent transformation from lethargy and sleep that the condition became known as the "Snow White syndrome."

The cranial nerves were not infrequently involved; strabismus (1), ptosis (1), facial palsy (3), aphasia (4), dysphagia (3). Paralysis of an arm or a leg occurred once each, and only five cases failed to show some form of lower motor weakness at some stage of the illness. Pyramidal signs were seen in six patients.

Only one death occurred: that of a 14-year-old child who was stuporous but responding on admission, and who became progressively comatose and died on the fourth day. At necropsy the lumbar cord was congested and showed punctate haemorrhages anteriorly, and perivascular cuffing was seen in the cervical cord and the thalamic region.

In only three cases did permanent sequelae persist: two cases of unilateral facial palsy and one of foot-drop; although temporary emotional instability was occasionally encountered.

Type 3. Insidious Onset with Continuous Choreiform Movements and Ataxia

Illustrative Case.—E. R., a male child aged 18 months. Previously well; then off his feet slowly; began to shake, and eyes moved from side to side. Very miserable; no temperature; C.S.F. normal. Seen again in one month; no progress and still very ataxic. A month after this, although bright and friendly, he had difficulty in balancing even when sitting still. His head was moving up and down regularly, and he was making mouthing movements. Unable to crawl, but could roll over. Reflexes, fundi, and C.S.F. normal. W.R. negative. E.E.G. indefinite. Developed basal ganglion tremor, particularly in arms. Excessive salivation. Improved for a time and could crawl, and the involuntary movements almost disappeared. Three weeks later he had again deteriorated, and the tremors were as bad as before, affecting head, eyelids, mouth, and to a less extent the limbs, and being continuous in sleep. These movements gradually became worse; arms, legs, and head jerked about uncontrollably; the eyes were never still, and

rolled upwards. He became uncooperative and resented interference. His condition was unchanged a year later.

There were nine cases similar in type to this, all of unknown aetiology, and all except one were under 5 years. There was an insidious onset from two weeks to three months, and occasionally isolated fits occurred two to three weeks beforehand, followed by a period of well-being before the characteristic signs developed. Sometimes a gradual unsteadiness of the limbs was the first manifestation, followed by stumbling and ataxia with tremors, squints and continuous eye movements or oculogyric crises, or a typical chorea-like illness. In four cases irritability, hysterical behaviour, or personality changes were also present, and frequently these patients were thought to be suffering from an intracranial tumour. The temperature was normal in all except one case.

The patients might be co-operative, lying quietly and slightly drowsy, or irritable, wildly excited and restless, violently choreiform and ataxic, with gross nystagmoid movements of the eyes. Coarse tremor on voluntary movement, nodding of the head, jerky speech, and salivation were present in some cases. Meningeal signs were seen in only one case, the C.S.F. contained a few cells in four cases, and the Lange curve carried out in five was normal. The subsequent course was usually slow improvement, often punctuated by relapses, emotional lability, staccato speech, outbursts of temper or excessive cheerfulness, and with slowly improving ataxia. The illness was usually long—between two and three months from the onset—although in two cases it was only two to three weeks.

Two patients died: one with continuous writhing and twitching, upward deviation of the eyes, slow deterioration, and finally opisthotonos with death in the eighth week; the brain showed occasional microscopic haemorrhages and signs of neuronal degeneration. The other case showed a progressive increase of the choreiform movements, alternating with phases of quiescence, when the temperature subsided for a time, but, following mental degeneration and blindness, the illness terminated in a convulsion in the seventeenth week. Minor changes with neuronal degeneration were seen in the mid-brain and cervical cord.

Sequelae were not infrequent: only three cases left hospital apparently normal; one was unchanged and had gross tremor and ataxia, myoclonus, and eye movements more than two years later; one was abnormally restless; one was irritable and domineering; and another was attending a psychiatric clinic.

The cases of this type were very similar in description to those of subacute inclusion encephalitis, although the normal Lange curves in four cases and the subsequent course did not conform to the usual fatal result or complete disablement reported by most observers (Campbell *et al.*, 1952; Foley and Williams, 1953), and at necropsy in two cases no inclusions were found.

Contrasted with the prolonged illness and very marked disturbance of cerebral function, the pathological changes were surprisingly scanty.

Type 4. Somnolent and Lethargic Illness with Spasticity and Retardation

Illustrative Case.—B. H., a boy aged 4½. Illness began two days before admission, with abdominal pain, repeated vomiting, and large offensive stools; drowsy; temperature 101° F. (38.3° C.), but no abnormal signs. On morning of admission he was found in a state of coma, with slight muscular rigidity and bilateral extensor responses; mild meningeal signs; C.S.F. normal. If undisturbed he lay as though peacefully asleep, but he resented interference, grimacing and crying, but otherwise aphasic. The face had an "ironed-out" appearance, and, although occasionally his attention was attracted, lethargy and sleep rapidly returned. Breathing was shallow and periodic, with deep sighing; inability to swallow necessitated tube-feeding almost continuously for six weeks. State of consciousness varied;

flexibilitas cerea developed and increasing spasticity—in the arms, of “cogwheel” type. Illness complicated by hypostatic pneumonia, generalized rigidity, double incontinence, and thrombosis of the inferior vena cava, the last-named being compensated by large venous anastomoses over the abdominal wall. Slow improvement occurred, and emotional responses returned, together with ability to swallow and a decrease in spasticity. Further progress was relatively rapid, rigidity of the left side of the body being slower to recover than the right. Euphoria and emotional lability were seen for a long time and walking was ataxic. He left hospital in three months, and when seen one year later was happy but somewhat backward, and still had slight spasticity of the left arm.

Five cases of unknown aetiology showed this type of illness—an infant and four young children. Prodromal symptoms were present in all, lasting from two to three days to one week. They consisted of headache, vomiting, abdominal pain, screaming and staring, occasionally twitching or a definite fit. In addition in this group there were seven cases of measles, three of pertussis, one of acute rheumatic fever, and one of rubella.

Meningeal signs were present in nine cases and a pleocytosis up to 150 cells in eight—not always the same cases. When first seen these patients were comatose or apparently deeply asleep. The cranial nerves were often affected, squints (9), ptosis (3), facial paralysis (9), pharyngeal paralysis (7), and a mask-like face or “dead-pan” appearance (5) being seen.

The course of the disease varied in duration up to two and a half months, five cases due to the acute infections had illnesses lasting one to two weeks only. Usually the drowsiness or coma increased for a few days, then improved gradually, being followed by variable periods of stupor, aphasia, dysphagia (two cases required tube feeding for three to six weeks), wild tremor and ataxia, twitches and occasional unilateral or bilateral fits, spasticity with extensor responses, flexibilitas cerea, irritability, screaming, salivation, and yawning. There was no sudden awakening from the lethargy such as occurred in the Type 2 cases.

There were two deaths, both in measles cases; 10 cases recovered completely; two were much improved but were mentally reduced; and one was apparently of normal intelligence but excessively noisy. One of the measles cases was backward, with infantile speech; another, who left hospital in a hopeless state and was little better a year later, when seen five years after the illness was second in his class at school, though his temper was uncertain. One pertussis patient was also left mentally retarded.

Cases with a Varied Clinical Picture not Resembling Other Types

There were 24 cases, 17 of unknown aetiology, which could not easily be placed in any of the other categories, although they showed certain similarities to them, often to more than one type. In many there were bizarre features; one child attacked another in the next bed and had alternating phases of aggression and docility; eventually he was transferred to a psychiatric ward, where he had several fits followed by stupor for three weeks. He made a complete recovery.

Three cases were thought to be associated with sensitivity reactions, sulphadimidine and “sulphatriad” probably being the responsible agents in two instances. The rashes were all urticarial and of mixed type, showing morbilliform, scarlatiniform, and petechial elements. There was sudden onset of drowsiness and slurred speech or aphasia. All had extensor responses, two were pyrexial, and initially the C.S.F. was normal in all, though later one showed a pleocytosis of 24 cells. Recovery was rapid, within two to three days, and with fading of the rash. One has a persistently abnormal E.E.G. and violent bursts of temper.

Four cases had illnesses resembling cerebellar tumour in which a variable period of dysphagia, dysarthria, disorientation, and fits preceded nystagmus, cerebellar ataxia, and

sometimes indefinite pyramidal signs. So marked were these signs that air encephalography was carried out in one case, with negative results. A diagnosis of tumour was originally suggested in many cases in the whole series, but exploration was carried out in only three other cases.

There were three cases following varicella which had a not dissimilar appearance to those just mentioned, but the diagnosis was never in doubt, and they all recovered quickly.

In two cases there was an illness progressing over five and sixteen days respectively, consisting of headache, vomiting, drowsiness, and apathy, and in one a fit, followed by a slowly developing hemiplegia. The temperature was normal and meningeal signs were absent; the C.S.F. in one case contained 22 cells, the other fluid being normal. On admission the patients were apathetic or fretful, becoming increasingly drowsy for a few days. One recovered from the hemiplegia in three weeks; the other was left with a spastic hemiplegia and an impulsive temperament. These cases were not unlike those described by Weston Hurst (1941), except that neither was fatal.

The remainder of this group were made up of two cases of measles, one of pertussis, and one of herpes simplex which could not be classified elsewhere.

The fatal case was that of a child of 3 months who was taken ill suddenly with continuous loud screaming and extreme restlessness. The temperature was only 99.3° F. (37.4° C.), and vomiting was absent. Apart from a depressed fontanelle, there were initially no abnormal signs. The screaming continued for five days, and was followed by unconsciousness and generalized spasticity. The C.S.F. was repeatedly blood-stained, was under normal pressure, and later became xanthochromic. The child lay completely unresponsive, developing nystagmus and intermittent squints and bilateral optic atrophy, apparently being blind and deaf. The limbs remained spastic, with occasional tremulous movements. The fontanelle later became tense, but otherwise the condition remained unchanged for three months, when, following a short period of continual spasms affecting the limbs, and hyperthermia, the child died. Different diagnoses had been entertained at different times, among them Schilder's encephalopathy, cerebral venous thrombosis, and cerebral sclerosis of Krabbe; but at necropsy the changes, though not gross, were compatible with haemorrhagic leuco-encephalitis.

Four other cases had sequelae, one was mentally defective and a severe epileptic, another was difficult to manage and had fits, a third had a slight hemiparesis, and a fourth was affected by sudden attacks of falling or stumbling.

The following Table shows the results according to type of case :

Type	Cases	Deaths	Complete Recovery	Survivors with Severe Disability
1	28	9 (32%)	13 (46.4%)	6 (31.6%)
2	15	1 (6.6%)	11 (73.4%)	
3	9	2 (22.2%)	3 (33.3%)	1 (14%)
4	17	2 (11.6%)	10 (58.8%)	3 (20%)
Others	24	1 (4.1%)	18 (75%)	5 (21.7%)
Total	93	15 (16%)	55 (59%)	15 (19%)

Summary

A study of 93 cases of encephalitis is presented; 65 (12 deaths) were of unknown aetiology and 28 (3 deaths) followed definite infections.

Sequelae were encountered in 23 of the 78 survivors, an incidence of almost 29%. They were severe or disabling (fits, mental reduction, or hemiplegia) in 15 cases, or 19% of the survivors.

An attempt has been made to group many of the cases according to the type of the clinical illness, and it has been found that there appears to be no constant relationship between the clinical picture and the aetiology or pathology. The histological changes in the brain have

often been surprisingly mild and have been disproportionate to the degree of cerebral disturbance seen during life.

It is suggested that, to some extent, the severity of the illness may be assessed by observing the mode of onset, the depth and duration of unconsciousness, the frequency of convulsions, and the persistence of signs indicating focal damage of the brain.

I am indebted to Professor Sir James Spence for much help and criticism in preparing this paper, and for allowing me to use many of the cases under his care. My thanks are due also to Dr. George Davison, Dr. F. J. W. Miller, Dr. S. D. M. Court, and Dr. C. E. Cooper for inviting me to see and to use the records of their cases.

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EXTRAMEDULLARY PLASMOCYTOMA

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Plasma-cell tumours may occur in bone and in soft tissues. Multiple myeloma is the most common manifestation, and its clinical and pathological features are well known. The solitary plasmacytoma of bone and extramedullary plasma-cell tumours are less common, while the interrelationship of the three conditions is still not finally proved. This case of extramedullary plasmacytoma is recorded because of its rarity, its unusual clinical presentation, and its interest as a case for differential diagnosis.

Case History

A man aged 55, who had lived in Jamaica all his life, was admitted to the Royal Infirmary, Sheffield, in August, 1952. In March, 1950, he had noticed a swelling on the left side of his neck. The following day he had a pyrexia of 102° F. (38.9° C.), and was treated by his doctor with penicillin injections. After a week the temperature subsided, but the swelling in his neck remained. The temperature rose again for several days in May, 1950. A radiograph of his chest and a full blood count at this time revealed only a "slight anaemia." His doctor diagnosed tuberculous glands of the neck. Thereafter, at intervals of six weeks, he had pyrexia, occasionally as high as 104° F. (40° C.), which persisted for several days before subsiding. In April, 1952, a lymph-node biopsy was reported on as Hodgkin's disease.

On examination three enlarged lymph nodes were present in the left anterior triangle of the neck. The largest of the three was approximately 2 cm. in diameter. They were freely mobile and painless. Examination of the oral cavity was normal and there was no evidence of dental caries.

The nose, nasopharynx, pharynx, and larynx were examined by an otolaryngologist and pronounced normal. A complete clinical examination revealed no other abnormality.

Investigations.—X-ray examination of the chest, skull, lumbar spine, long bones, and accessory air sinuses revealed nothing abnormal. The Wassermann and Kahn reactions were negative. Agglutination reactions: *Brucella abortus* and *Brucella melitensis* less than 1/10. The urine was examined on many occasions: albumin was constantly present, but tests for Bence Jones proteose were repeatedly negative. Plasma protein: serum protein, 8.3 g. per 100 ml. (albumin 4 g., globulin 4.3 g.). Haematology: haemoglobin, 82%; red cells, 4,000,000; leucocytes, 7,000 (band cells 3%, lymphocytes 24%, polymorphs 64%, monocytes 6%, eosinophils 3%). The white-cell morphology appeared to be within normal limits. Platelets 620,000 per c.mm. (Lempert). Bone marrow (left ilium): The abnormal features included the presence of some macronormoblasts and an occasional megaloid cell. Abnormal nests of plasmocytes and their precursors were seen—in all, 5.6% of 500 cells. There was megakaryocytic hyperplasia.

Biopsy of the cervical lymph node on August 29 was reported on by Professor Willis as follows: "The extremely heavy infiltration of this lymph node by plasma cells is, I think, rather more than might be accounted for by any inflammatory disease, and my diagnosis is plasmacytoma. Whether this may be due to a primary growth in the upper respiratory passages or to skeletal myelomatosis can only be decided by other investigations, especially x-ray studies of the bone. The high plasma protein level favours myelomatosis. Bence Jones proteosuria should be carefully watched for."

The patient's general condition remained good and no further pyrexia occurred. As the radiological examination of the bones, the clinical state of the patient, and the other investigations were against a diagnosis of multiple myeloma, the lesion in the neck was treated with x-irradiation as a case of primary extramedullary plasmacytoma. Satisfactory regression of the remaining enlarged glands took place.

Since his return to Jamaica he has remained in good health. No Bence Jones proteosuria has been demonstrated. Radiological findings (skull, thorax, vertebral column, pelvis, long bones of upper and lower extremities) were normal in May, 1953.

Discussion

Solitary plasmacytoma is an established clinical condition. Although any part of the skeleton may be the site of such a lesion there appears to be a special predilection for the innominate bone and the long bones of the lower limbs. Amongst recorded cases are those of Rogers (1930), Chesterman (1936, 1948), Leedham-Green *et al.* (1938), Willis (1941), and Lumb (1948).

Willis (1941) considers that in order to establish a diagnosis of solitary plasmacytoma the following criteria must be fulfilled: microscopical proof that the tumour is of plasma-cell nature; exclusion of the possibility that the lesion is the first evidence of generalized myelomatosis; there must be radiological proof that no skeletal lesions are present, both at the time of examination and at subsequent examinations at repeated intervals; and Bence Jones proteosuria shall be absent on repeated examinations.

The validity of this last criterion is questioned by Lumb (1948), who found Bence Jones proteose in the urine of a case proved at necropsy to be one of solitary plasmacytoma of the sacrum. He concludes that the proteosuria is consequent on actual tumour volume—namely, the amount of bone-marrow involvement.

Plasmacytomas of soft tissue are much less often encountered. Hellwig (1943) found 127 published cases in the literature reviewed between 1905 and 1942, to which he added a case of his own. The lesions in most cases are situated in the upper air passages, especially the nasopharynx, maxillary antrum, and oral cavity. Cases have