DIFFERENTIAL DIAGNOSIS OF MENTAL DISORDER IN LATER LIFE*

By BRIAN A. O'CONNELL, M.B., D.P.M.

Department of Psychiatry, St. George's Hospital, S.W.1

The problems raised by mental disorders of the elderly, already a serious social and medical issue, are likely to be aggravated in the future by the progressive increase, relative and absolute, in the numbers of the old in the population. The assumption in the past that most of the mental disorder of later life was due to organic brain damage led to an undue pessimism and for long obscured the fact that reversible mental illness is common in this age group as in others. That such illness should be recognized and treated is a matter of some urgency.

Mental disorders of later life are here considered as those which present over the age of 60, which age commonly determines retirement and pension benefit.

Normal Ageing

The majority of old people live out their lives without having any mental disorder. They show, however, *pari passu* with the normal decline in physical prowess certain psychological and intellectual changes. For some, retirement spells the end of a productive life and gives rise to vague fears of insecurity and loneliness. Narrowing of mental perspective and restriction of interests may follow the lowering of mental and physical energy. The failing physical resources lead to a loss of personal security and foster dependence. Krapf (1953) regards as the fundamental anomaly in old age the loss of ability to project oneself into the future. Having no great interest in the future, these people tend to dwell in the past, and because it fosters security, they often show a rigid adherence to long-established routines with a diminished capacity for embarking on new lines of thought.

Neurosis

Having regard to these changes (and particularly when they are complicated by social stress), one is tempted to interpret the appearance of neurotic symptoms as a comprehensible reaction to circumstances. It must be emphasized, how-

ever, that neurosis rarely arises *de novo* in senescence. The normal adult adjusts to the restrictions imposed by age. The elderly patient presenting with neurotic behaviour—aggressiveness, jealousy, hypochondriasis, dramatic appeals for help and the like, will most often be found to have shown evidence of immaturity and maladjustment in the previous history. Thus, for example, Busse (1954) found that where a poor relationship with the surviving children existed (and this is often put forward by the patient as a cause of his trouble), it could not be attributed solely to the events of old age, but was associated with a life-long history of a poor marital and sexual adjustment and an inability to establish mature relationships.

But where neurotic patterns appear for the first time in senescence, the case calls for careful consideration as this development often conceals something more serious, namely psychotic illness whether organic or functional. Thus, severe hypochondriasis may herald the onset of cerebral arteriosclerosis, or anxiety symptoms may cloak a psychotic depression. The same caution must be exercised where in an established neurotic the reaction pattern alters markedly; for example, the onset of gross conversion symptoms in, say, a life-long neurasthenic not previously subject to them, may be the indication—at first sight unexpectedly—of organic cerebral disease. A diagnosis of neuroticism in the elderly who have not been previously neurotic or who show new neurotic patterns, is made only after the exclusion of (1) functional psychosis (mainly affective, more rarely paraphrenic), and (2) organic psychosis (primary, as in senile dementia and cerebral arteriosclerosis; secondary as, for example, in brain tumour). Apart from these, the only other organic mental state seen often at this age is delirium.

*Lecture given April 1955, at St. George's Hospital, in course of Seminar organized by British Post Graduate Medical Federation.

Reversible and Irreversible Psychosis

In the past, undue emphasis on pathology fostered the idea that most mental disorder in the elderly was of irreversible kind. Kraepelin and Bleuler appear to have regarded the occurrence of reversible affective psychosis in the senium as a rarity. Certainly, before the days of effective treatment, cases of affective psychosis in the aged admitted to mental hospitals generally ended their days there, thus leaving little to choose prognostically between them and the organic psychoses. But experience with electrical treatment suggested its extension to include elderly cases with affective symptoms. The results show that in the aged a considerable proportion of mental illness is of the affective type and is reversible and does not then proceed to emotional and intellectual disorganization (Evans, 1943; Mayer-Gross, 1945; Feldman *et al.*, 1946; Wilbur and Fortes, 1947; Gallinek, 1948; Roth and Morrissey, 1952; Levy, 1952; Robinson and de Mott, 1953; Norris and Post, 1954; O'Connell, 1954; Ehrenberg and Gullingsrud, 1955). It may be remarked that many of the patients reported on, in whom reversal of illness was secured, were in the ninth decade and almost all showed some of the physical complications of old age—hernia, osteoarthritis, hypertension, cardio-vascular disease, etc. This emphasizes the safety of the treatment, despite superstitions to the contrary which are sometimes encountered.

Follow-up studies of treated cases of affective psychosis show a sustained recovery rate of between 65 per cent. and 75 per cent. after a period of two years. Senile dementia, on the other hand, is intractable and has a hopeless prognosis. The death rate two years after admission to hospital is variously reported as 78 per cent. (Roth and Morrissey, 1952), 75 per cent. (O'Connell, 1954) and 70.7 per cent. (Camargo and Preston, 1945). Contrasting death rates after the same time in affective psychosis are 10 per cent. (Roth and Morrissey, 1952) and 27 per cent. (O'Connell, 1954). Thus it is seen that affective psychosis is a benign condition with a good prognosis when treated. If not recognized, it leads, at the best, to prolonged suffering and at the worst to death from inanition, inter-current infection or suicide. Senile dementia, on the other hand, is a malignant disease leading to disintegration and death. The other primary organic brain disease, cerebral arteriosclerosis, runs a somewhat similar course, although there are at least better prospects of temporary remission in the earlier stages.

Paraphrenia (paranoid form of schizophrenia of late onset and characterized by good social preservation) may present at this time of life but, though chronic, is not fatal although liable to periodic but irregular exacerbation.

Delirious states of the elderly tend to end in early death or recovery.

Affective Psychosis

Affective psychosis accounts for about half the cases of mental illness referred over the age of 60 for psychiatric opinion. Reported figures are 54 per cent. (Roth and Morrissey, 1952) for hospital admissions, 58 per cent. (Norris and Post, 1953) for out-patient referrals and 43 per cent. (Curran, 1955) for private practice. Affective psychosis occurs in two forms, depression and mania.

1. *Depression.* Depression is the form of affective disorder in 95 per cent. of cases, the remainder being of mania type. Depression in this age group is practically always of the involutional kind, with some or all of the following features—anxiety and agitation with much lamentation, somatic concerns, ideas of guilt, depressive delusions of hypochondriacal or nihilistic kind (their bowels are blocked or they are financially ruined, etc.), intractable insomnia with, classically, early morning wakening; loss of weight, poor appetite, constipation, disturbance of concentration and fears of insanity and vague complaints of weight and tension in the head. Rarely, instead of agitation there is retardation. The mood characteristically changes for the better towards evening (diurnal variation). It must not be supposed that all cases will have such florid features, for many do not complain directly of depression but of vague physical symptoms, anxiety and fears of insanity. As mentioned, such mild cases might easily be dismissed as neurotic unless a careful history be taken. In this connection the onset of the illness is of the greatest importance. This is usually abrupt, often precipitated by psychological or physical trauma, but without evidence of previous failure in adaptation. Elsewhere I have reported the average duration of symptoms before referral as about 18 weeks in these cases, compared with three to six years in cases of senile dementia, this impression being confirmed by a recent examination of material in a private practice. Some cases of affective psychosis will come to light through a suicidal attempt. Suicidal attempts in the elderly almost invariably indicate a psychotic depressive background. They rarely arise in the case of a dementing illness since, as Batchelor and Napier (1953) point out, the retention of affect and volition is a prerequisite for the act. In the past history of depressives there may be evidence of previous mood swings, sometimes necessitating hospital admission.

In contrast to degenerative brain disease there is no real intellectual deterioration though there may be a spurious imitation of it. The severe depressive pays little attention to his surroundings and cannot recall what he has not registered. This

may give rise to a misleading appearance of deterioration. Further, mild confusion may actually arise on the basis of exhaustion, inadequate fluid or vitamin intake or excessive sedation. These possibilities show the need for careful evaluation of (i) the presence of actual depressive symptoms, (ii) how real is the apparent deterioration, (iii) what other factors might account for it. It is common experience that with the application of electrical treatment and reversal of the affective disorder the 'organic signs' may completely disappear (Maddon *et al.*, 1952; Prout and Hamilton, 1952; Tewfik, 1953). The similar disappearance of 'organic signs' in depressed patients recovering spontaneously has been reported by Robertson and Browne (1953). Batchelor and Napier (1953) report the appearance of confusion in severely depressed people prior to suicidal attempts. It was perhaps the occurrence of such quasi-organic features that gave currency in the past to the use of the term senile depression.

2. *Mania*. About 5 per cent. of affective disorder in the aged presents in this way. There is a more or less sudden onset with hyperactivity, elation, flight of ideas and push of talk, grandiosity, poor sleep and fatigue and occasionally erotic behaviour. Such cases are more resistant to treatment than cases of depression.

Degenerative Brain Disease

Senile dementia and cerebral arteriosclerosis between them account for much less than half of the mental illness presenting in those over 60. Thus reported figures for hospital admissions are 34 per cent. (Robertson and Browne, 1953), and 29 per cent. (Roth and Morrissey, 1952); for out-patient referrals, 24 per cent. (Norris and Post, 1954); and for private practice 23 per cent. (Curran, 1955). As previously indicated, these diseases carry an ominous prognosis. While it is true that the clinical conditions of senile dementia and cerebral arteriosclerosis arise on the basis of characteristic pathological brain changes, it is well to remember that there is no consistent correlation between the histological change and the degree of dementia and that equally severe changes occur in the brains of apparently normal old people (Gellerstedt, 1933; Rothschild, 1937). This would seem to indicate that factors other than actual brain damage play a part in the clinical picture. It is inferred that more robust personalities can accommodate better to brain damage and have, so to speak, a greater mental reserve, although presumably if the changes are severe enough even the most robust will exhibit psychotic features. Studies such as those of Williams (1942) and Post (1944) direct attention to the part played by personality factors and social stresses. Sands and Rothschild (1952) believe the

clinical picture to be the product of special vulnerabilities in the personality coupled with 'sociopsychiatric' stress in the presence of organic cerebral disease. This formulation covers a fact of clinical experience—that psychological trauma seems often to initiate a rapid decline in the course of organic brain disease. Such trauma acts, so to speak, as the last straw to break down an already precarious adjustment. Since in our present state of knowledge nothing can be done to reverse the organic brain damage, the main attack on this problem is directed to reducing social stress in the aged (Sheldon, 1954).

Senile dementia. This is a condition associated with structural brain changes and showing insidiously progressive disorganization of the personality in the intellectual and emotional spheres. The long and rather uneventful history (usually several years) of gradual intellectual failure is most characteristic—there is difficulty in dating the onset of the illness, unlike the case of affective psychosis. A general reduction in initiative, with easily induced fatigue, is soon followed by slow failure of memory, particularly for recent events, with a tendency to fill in the gaps with fabrication and pseudo-reminiscence; concentration and attention flag, the patient becomes disorientated, especially in time, and grasp and judgment begin to fail. The whole syndrome tends to be worse at night, during which, especially as insomnia may be prominent, the patient may show confused activity. Delusions of a paranoid or grandiose type, frequently bizarre or fantastic, may be present. They are typically changing and are not woven into a system—this is to be expected in view of the very poor memory and disturbance of comprehension. At one time or another all cases of senile dementia show confusional episodes. Emotional disintegration proceeds with the intellectual decline. For a time the patients may be irritable and obstinate, but the characteristic affect is one of emotional lability on a background of shallow euphoria. If faced with tasks they cannot overcome, they often show what Goldstein (1952) calls the catastrophic condition—in a moment they change from good humour and co-operation to opposition and tears, with temporary disintegration of such faculties as they retain. Sustained depression, however, of the kind described in affective psychosis, and presenting with ideas of guilt, etc., is not seen and should not be confused with the emotional lability of the organic.

Some cases of senile dementia may be referred through the Courts for indecent exposure. Such behaviour, occurring in an old person for the first time, should always suggest the possibility of brain damage, although this is by no means invariably the case. In the later stages of the illness there may

be incontinence of urine and faeces and the final condition is one of profound and helpless dementia.

Cerebral arteriosclerosis. The better, although temporary, prospects of remission in this illness make its differentiation from senile dementia worth while. The distinction is often possible, at least in the early stages, on the criteria laid down by Rothschild (1947)—a fluctuating course which may have commenced abruptly (with a cerebrovascular accident), more patchy impairment of intellect and relatively well retained personality, with fair degree of insight, emotional incontinence (sudden spasmodic weeping and laughing) and focal cerebral signs, with a tendency in longstanding cases to develop pseudobulbar palsy. There is usually a history at some time of apoplectiform or syncopal attacks, less often of convulsive seizures. There may have been transient paresis, aphasia or hemianopia. It may be remarked that radial and retinal arteriosclerosis are no guide to the diagnosis. The illness is frequently heralded by a hypochondriacal-like picture, with headache, vertigo, tinnitus, malaise, fatigue, insomnia and loss of appetite.

These prodromal symptoms are often seen in the course of hypertension and may indeed bring it to notice. Repeated hypertensive crises may leave in their train a gradually developing dementia and intellectual impairment has even been demonstrated in cases of essential hypertension before the appearance of any neurological signs (Apter, 1951; Reitan, 1954). It seems reasonable to suppose that at least in some patients early and active treatment of the hypertensive condition might avert later cerebrovascular damage. While it is true that a degree of systolic hypertension is normal in old age and that many hypertensives suffer more from anxiety and morbid preoccupation about their condition than from any functional effects of the condition itself, the stage at which psychological management is of most value is surely that when the hypertension is still labile.

Secondary Organic Brain Disorder

The clinical picture of degenerative brain disease may be simulated by a variety of somatic and toxic factors, some of them reversible.

1. **General paralysis.** Expansive delusions are not now so common and the case may present as simple dementia. The C.S.F., W.R. should be done in suspected cases, since the blood W.R. may be negative.

2. **Tumour.** In the elderly, headache, vomiting and papilloedema may be absent. There may be no focal signs, or those in the psychic sphere may be missed and, for example, aphasia be regarded as loss of memory, spatial disorientation as senile wandering. Mental symptoms may be the first and for a long time the only symptoms (Sachs,

1950; Kahn and Schlesinger, 1951; Waggoner and Bagghi, 1954). If the illness begins abruptly with a 'stroke' and without development of papilloedema, it may be confused with a cerebrovascular accident. As Allison (1952) points out, subsequent development of progressive mental deterioration, with or without hemiplegia, favours a diagnosis of tumour since the picture in cerebrovascular accidents is one of maximum disturbance at the outset and gradual recession thereafter. E.E.G. examination may be helpful. Skull and chest X-rays should be carried out and a search made for primary growths. If increased intracranial pressure is suspected, lumbar puncture should only be carried out in a neurosurgical unit. Air studies and angiography may also be required.

3. **Subdural haematoma.** In cases of recent deterioration, enquiry should be made for head injury which it must be remembered may commonly occur in a syncopal attack. Evidence of increased pressure and localizing neurological signs may be absent in the early stages and the condition may result in progressive impairment of intellect and memory. Clarke and Cooper (1954) describe such a case and, reviewing the literature on the associated mental changes, conclude 'that in any patient who complains of headache and manifests non-specific mental changes accompanied by drowsiness, which may or may not fluctuate, a subdural haematoma should be considered, even in the absence of known trauma, increased intracranial tension and abnormal neurological signs.' The recency of onset and fluctuating level of consciousness are important points.

Head injury, even in the absence of haematoma, may give rise to dementia, especially in those predisposed by cerebral arteriosclerosis or chronic alcoholism.

4. **Alcoholism and epilepsy.** In the chronic stages patients may show evidence of dementia. It should be remembered that such cases are apt to fall and any sudden deterioration in the mental condition may suggest haematoma.

5. **Neurological conditions.** Dementia may occur in longstanding cases of encephalitis or disseminated sclerosis.

6. **Abscess.** A mild confusional state, with drowsiness and memory impairment may result from a cerebral abscess. In any patient with a history of head injury, middle ear infection or chronic chest illness, changes in the behaviour or in the level of consciousness should suggest this possibility.

7. **Chronic heart failure and uraemia.** Again evidence of dementia may be present, although since heart and kidney malfunction is frequently

associated with cerebral arteriosclerosis it may be difficult to say which is the aetiological factor.

Paranoid States.

1. Paranoid delusions may be prominent in affective psychoses. They are then sustained and in tune with the mood. For example, a man who believes himself to be in disgrace may think that others shun him, or one who believes he has committed irreparable crimes may fear that he will be put to death. This is essentially secondary to the depression rather than a paranoid state in itself.

2. In organic brain disease, paranoid delusions may arise out of the disturbance of grasp with misinterpretation. The delusions are not woven into a system and because of the poor memory and labile mood are constantly changing—they are frequently fantastic. Again the paranoid features are secondary.

3. A small number of patients, in the absence of affective features or intellectual deterioration, exhibit paranoid delusions of a sustained and systematized kind, often associated with hallucinations in clear consciousness and sometimes with ideas of being influenced by others in supernatural ways. In these the paranoid features are primary. These are cases of paraphrenia which on examination will commonly be found to have been present for many years.

Delirium

Old people are peculiarly susceptible to a variety of factors which induce delirium. Disordered orientation, fear and hallucinations, especially in the visual field, are the most characteristic features of the condition. Classically, there is a fluctuation in the level of awareness and the patient is frequently worse at night, becoming noisy and overactive. Memory is disturbed and there may be confabulation and paraphasia, while delusions of a changing kind are common. Physically there may be tremor, inco-ordination, slurred speech, incontinence, pupillary changes, elevated temperature, leucocytosis, albuminuria and glycosuria. The onset of delirium in any illness in the elderly is a serious event and unless treatment is promptly initiated the mortality is high. It is therefore well to recognize prodromal symptoms—restlessness and apprehension, irritability with increased sensitivity to noise and light, emotional instability, insomnia and terrifying dreams. Delirium may arise in the following conditions:

1. In the course of senile dementia or cerebral arteriosclerosis. It may be the first manifestation of the latter condition, in which case a fair degree of recovery may be expected, although repeated attacks will leave behind an increasing dementia.

2. In the acute initial stages of a severe affective

disorder (Batchelor and Napier, 1953; Robertson and Brown, 1953).

3. In acute infections, especially pneumonia and urinary infections, and in such it may not arise until the convalescent stage. Urinary infections are specially liable to be missed in the elderly and are a frequent cause of senile confusion.

4. After operations in the elderly, especially for cataract and prostate, and also following fractures.

5. With excessive sedation, especially by bromides and barbiturates. Bromide should not be given to elderly people as a routine, especially if there is any suggestion of arteriosclerosis. Paraldehyde is probably the drug of choice, although recent reports (Howell, 1954; Winkelmann, 1954) speak of the effectiveness and safety of chlorpromazine in the elderly, although the resulting hypotension calls for caution when there is vascular damage. In the case of bromism, a blood examination will settle the issue. Because of his reduced tolerance, alcohol may easily precipitate delirium in the elderly arteriosclerotic.

6. In acute heart failure—here anoxaemia is presumably the important factor and may also be responsible for the delirium occasionally seen in cases of severe haemorrhage and pernicious anaemia.

7. Uraemia and cholaemia.

8. Dehydration, alkalosis (as, for example, treating urinary infections or chronic dyspepsia) and hypoglycaemia are suggested by Allison (1952) as further contributory causes of delirium. In the latter connection he suggests that in the elderly diabetic it is better to restrict the total calories and reduce the insulin, since the effects of repeated hypoglycaemia disturb cerebral function.

9. Non-specific cases of delirium, without evident aetiology, are sometimes assumed to be due to deficiency factors, such as avitaminosis.

Summary

1. Neurosis arising *de novo* in senescence may conceal organic or functional psychosis.

2. The primary degenerative brain diseases—cerebral arteriosclerosis and senile dementia, account for a minority of cases (roughly 40 per cent.) They have a history of insidious onset (occasionally abrupt in cerebral arteriosclerosis) and a hopeless prognosis.

3. Reversible functional psychosis of the affective type is common in the elderly and accounts for at least half the cases. It is of abrupt onset and of good prognosis.

4. Paraphrenic psychosis occasionally presents for the first time after 60. It runs a chronic course but does not have a high mortality.

5. Mental illness of acute or subacute kind, much of it reversible, may arise on the basis of a

variety of other physical factors. Delirium in the elderly tends to end in recovery or death.

6. Every case of mental disorder in the aged demands a thorough history, searching physical and especially neurological examination and the use of special diagnostic aids where indicated.

Acknowledgments

I am indebted to Dr. Desmond Curran for providing me with figures relating to his private practice.

BIBLIOGRAPHY

- ALLISON, R. S. (1952), *Brit. med. J.*, **ii**, 1286.
 APTER, N. S., *et al.* (1951), *Amer. J. Psychiat.*, **107**, 808.
 BATCHELOR, I. R. C., and NAPIER, M. B. (1953), *Brit. med. J.*, **ii**, 1186.
 BUSSE, E. W., *et al.* (1954), *Amer. J. Psychiat.*, **110**, 897.
 CAMARGO, O., and PRESTON, G. H. (1945), *Ibid.*, **102**, 168.
 CLARKE, E., and COOPER, R. (1954), *Lancet*, **i**, 1260.
 CURRAN, D. (1955), personal communication.
 EHRENBERG, R., and GULLINGSRUD, M. J. O. (1955), *Amer. J. Psychiat.*, **111**, 743.
 EVANS, V. L. (1943), *Ibid.*, **99**, 531.
 FELDMAN, F., *et al.* (1946), *Arch. Neurol. Psychiat.*, **56**, 158.
 GALLINEK, A. (1948), *J. Nerv. Ment. Dis.*, **108**, 293.
 GELLERSTEDT, N. (1937), quoted, *Amer. J. Psychiat.*, **93**, 763.
 GOLDSTEIN, K. (1952), *Psychiatry*, **15**, 245.
 HOWELL, T. H., *et al.* (1954), *Practitioner*, **173**, 172.
 KAHN, R. L., and SCHLESINGER, B. (1951), *J. Nerv. Ment. Dis.*, **114**, 492.
 KRAPP, E. E. (1953), *Proc. Roy. Soc. Med.*, **46**, 951.
 LEVY, S. (1952), *Confinia Neurol.*, **12**, 381.
 MADDEN, J. J., *et al.* (1952), *J. Amer. med. Ass.*, **150**, 1567.
 MAYER-GROSS, W. (1945), *J. Ment. Sci.*, **91**, 101.
 NORRIS, V., and POST, F. (1954), *Brit. med. J.*, **i**, 675.
 O'CONNELL, B. A. (1954), *Ibid.*, **ii**, 1206.
 POST, F. (1944), *J. Ment. Sci.*, **90**, 554.
 PROUT, C. T., and HAMILTON, D. M. (1952), *Bull. N.Y. Acad. Med.*, **28**, 454.
 REITAN, R. M. (1954), *Amer. J. Psychiat.*, **110**, 817.
 ROBERTSON, E. E., and BROWNE, N. L. MASON (1953), *Brit. med. J.*, **ii**, 1076.
 ROBINSON, G. W., JUN., and DE MOTT, J. D. (1953), *Geriatrics*, **8**, 259.
 ROTH, M., and MORRISSEY, J. D. (1952), *J. Ment. Sci.*, **98**, 66.
 ROTHSCILD, D. (1937), *Amer. J. Psychiat.*, **93**, 757.
 ROTHSCILD, D. (1947), *Geriatrics*, **2**, 155.
 SACHS, E., JUN. (1950), *J. Ment. Sci.*, **96**, 998.
 SANDS, S. L., and ROTHSCILD, D. (1952), *J. Nerv. Ment. Dis.*, **116**, 233.
 SHELDON, J. H. (1954), *Lancet*, **ii**, 151.
 TEWFIK, G. I. (1953), *Ibid.*, **ii**, 110.
 WAGGONER, R. W., and BAGGHI, B. K. (1954), *Amer. J. Psychiat.*, **110**, 904.
 WILBUR, C. B., and FORTES, A. (1947), *Ibid.*, **104**, 48.
 WILLIAMS, H. W., *et al.* (1942), *Ibid.*, **98**, 712.
 WINKELMANN, N. W. (1954), *J. Amer. med. Ass.*, **155**, 18.

Important Medical Books

FRACTURES AND JOINT INJURIES

Fourth Edition Reprint. By Sir REGINALD WATSON-JONES, M.Ch.Orth., F.R.C.S. In two volumes, not sold separately.
 1,130 pages. 1,613 illustrations. £6 10s.

BODY FLUIDS IN SURGERY

By A. W. WILKINSON, Ch.M., F.R.C.S.E.
 222 pages. 7 illustrations. 16s.

MULTIPLE SCLEROSIS

By DOUGLAS McALPINE, M.D., F.R.C.P., NIGEL COMPSTON, M.D., M.R.C.P., and CHARLES LUMSDEN, M.D.
 310 pages. 115 illustrations. 35s.

ANATOMY OF THE AUTONOMIC NERVOUS SYSTEM

By G. A. G. MITCHELL, O.B.E., T.D., M.B., Ch.M., D.Sc., M.Sc.
 372 pages. 131 illustrations. 55s.

FRACTURES OF THE FACIAL SKELETON

By N. L. ROWE, F.D.S.R.C.S.(Eng.), M.R.C.S., L.R.C.P., H.D.D.R.C.S.(Edin.), and H. C. KILLEY, F.D.S.R.C.S.(Eng.), M.R.C.S., L.R.C.P., H.D.D.R.C.S.(Edin.).
 960 pages. 1,236 illustrations. £6

AN ATLAS OF REGIONAL DERMATOLOGY

By G. H. PERCIVAL, M.D., Ph.D., F.R.C.P., D.P.H., and T. C. DODDS, F.I.M.L.T., F.I.B.P., F.R.P.S.
 272 pages. 479 illustrations. £5

NURSING CARE OF THE NEWLY BORN INFANT

By W. S. CRAIG, B.Sc., M.D., F.R.C.P.E., F.R.S.E.
 480 pages. 272 illustrations. 35s.

THE BOKE OF CHYLDREN

By Dr. THOMAS PHAIRE. 76 pages. 7s. 6d.

E. & S. LIVINGSTONE, LTD., TEVIOT PLACE, EDINBURGH, 1