

administered by a devoted father, and stayed there (on and off) till she was 14 months old. Every three months I go back to the hospital for a check-up, during which I stick out my tongue and have to remember not to wear polo-necked jerseys so that the doctor can feel my neck. I have graduated down from consultants and registrars, and am now seen by whoever happens to be around, which I count as progress.

I cannot say that I ever forget that I have had cancer. But it is amazing what people learn to live with. It has been an eye-opener in many ways. Firstly, I am impressed by the need for research in communication between medical staff and patients with cancer. I was never offered any information about my illness, and although my questions were certainly answered, I know (by checking with my medical colleagues) that some of them were answered dishonestly. Most doctors seem to be unable to confront their own feelings about cancer.

Secondly, anyone with cancer has to come face to face with society's attitudes to malignant disease and these are extremely fatalistic. I'm sure that some of my friends expected me to drop dead almost immediately—or at least to *look* different—and when I didn't they didn't know where to look. Susan Sontag has written about all this in her excellent book *Illness as Metaphor*,² which I advise anyone concerned with the treatment of patients with cancer to read. Children, as always, go to the heart of the matter. I remember explaining to my 10-year-old son what it was that I had on my tongue, using suitably childlike expressions. "You mean you've got cancer," he said. The biggest bane of my life as a patient with cancer is the lady in the pink overall (a voluntary helper, I imagine) who weighs people in the out-

patients clinic. "Oh good," she says, "you've put on weight"—when I am still desperately trying to lose my post-baby bulge.

Learning to live in the present

While I definitely could have done without the experience of having cancer, there is no doubt that it has permanently altered my attitudes to the conduct of my own life. I have learnt to live in the present, which seems by far the best way to live. I have ceased to be impressed by the ephemera of academic life, and am interested only in doing the work I want to do as well as I can. I reckon that if people have unfulfilled ambitions they ought to fulfil them, so this year I am going to write a novel, not start another research project. All those tortuous wranglings with conscience about work versus motherhood suddenly seem very clear to me. Children are precious and lovely and, although they do not "need" their mothers as our cultural ideology of motherhood suggests, I, as a mother, certainly need them.

Oddest of all is the fact that only this confrontation with death has enabled me to realise how happy I am. That, I'm sure, must be a poignant reflection on the kind of society in which we live.

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Today's Treatment

Drug-induced diseases

Drug-induced neurological disease

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Most clinicians acquire a wide practical knowledge of drug-induced disease. Drug trials and drug legislation cannot anticipate every circumstantial interaction or protoplasmic defect, and pharmaceutical research must continue to develop drugs, which of their nature cannot be subjected to large trials, to combat rare diseases. Patients and the public should respect the inevitability of side effects from powerful modern drugs, and doctors must improve their expertise in applied therapeutics and yet be prepared, where the possibility of adverse reactions arises, to seek the help of colleagues, hospital pharmacists, the Committee on Safety of Medicines, and medical representatives of the

drug company concerned. The following discussion on drug-induced neurological disease is not intended to be comprehensive but rather to draw attention to some of the more important conditions.

Both old and young vary in their tolerance of drugs, and research is needed into the development of screening tests checking the patient's individual susceptibility to an iatrogenic challenge. For a few children the earliest challenge presented may arise from drugs that cross from the maternal to fetal circulation. Thus diazepam (Valium, Atensine) may cause respiratory depression in the newborn, lithium cause hypotonia, and antiemetics containing pyridoxine (for instance, Ancoloxin, Benadon, and Debendox) produce neonatal convulsions that require treatment with high doses of pyridoxine. Convulsions can also occur in breast-fed babies whose mothers are taking indomethacin (Indocid, Imbrilon). For most children the earliest risk of iatrogenic disease is that from vaccination, unmasking any constitutional weakness and given at a time of considerable susceptibility to intercurrent infection. The perils of pertussis vaccination are open to conflicting interpretations, however, and the relation to encephalopathy is far from proved.²

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Cerebrospinal disorders

All antibiotics are potentially neurotoxic. Given systemically the danger of convulsions and brain damage is potentiated by renal failure or blockade—as with probenecid (Colbenemid, Benemid), and this syndrome may also result from the intrathecal injection of noxious agents including undiluted penicillin or streptomycin. (No other antibiotic should be given by this route.) With high blood concentrations the aminoglycosides (streptomycin, kanamycin, and gentamicin) and the diuretics, ethacrynic acid (Edecrin) and frusemide (Lasix, Dryptal), are ototoxic. In contrast, aseptic meningitis may result from inadequate antibiotic treatment or poor penetration of the cerebrospinal fluid by wide-spectrum antibiotics. Bacterial meningitis and fungal disorders of the nervous system may arise during treatment with cytotoxic drugs or prolonged steroid administration. Another syndrome produced by drugs, including antibiotics, is iatrogenic intracranial hypertension (pseudotumour cerebri). The condition may present with bulging fontanelles, associated with the use of tetracycline in infancy, or arise in infants, children, teenagers, and young adults as a result of steroid administration (especially triamcinolone) or withdrawal, nalidixic acid (Negram), nitrofurantoin (Furadantin), or oral contraceptives.

Cerebrovascular disorders

Drugs affecting blood vessels and the constituents of the blood may all result in complications affecting the central nervous system. The incidence of complications from oral contraceptives has undoubtedly decreased since the introduction of the low oestrogen pill, but migraine may be intensified and drug-induced chorea, strokes, sinus thrombosis, subarachnoid haemorrhage, and subdural haematomas are still reported. Dexamethasone (Decadron), a powerful glucocorticoid, is invaluable in treating cerebral oedema, but its overenthusiastic use may result in failure to diagnose intracranial tumours or subdural haematomas. Other syndromes related to the administration of steroids include psychotic reactions, drug-sensitive relapsing polyneuritis, spinal compression from collapsed osteomalacic vertebrae, cataract, glaucoma, and a severe proximal myopathy that develops most readily when the fluorine-containing analogues are used but is seen with all steroid preparations.

Muscular disorders

Penicillamine, tetracycline, aminoglycosides—for instance, intraperitoneal streptomycin or neomycin—phenytoin, practolol, quinidine and procainamide may compete with neuromuscular blocking agents and induce respiratory paralysis. Just occasionally, especially in the presence of renal failure, they may produce a true myasthenic syndrome, and anti-acetylcholine receptor antibodies have been identified in D-penicillamine-induced myasthenia gravis. Suxamethonium, alone or as the result of interaction with anaesthetic agents, can induce a syndrome of malignant hyperpyrexia and extreme muscular rigidity in genetically susceptible individuals. Drug-induced myopathies are caused by a wide variety of drugs including disulfiram, emetine, polymyxin E, allopurinol, and steroids. Many are improved on withdrawing treatment thus aiding the differentiation from carcinomatous, endocrine, or metabolic myopathies.

Peripheral neuropathies

Drugs may damage Schwann cells, myelin, axons, or the distal ends of peripheral nerves. Fibres of different sizes are selectively affected with manifestations affecting predominantly the motor or sensory nerves. Coexistent damage may affect anterior horn

cells, cranial nerves, the eye, and muscles or other tissues of the body, so that, despite clinical variability, it is possible to distinguish distinctive patterns for particular drugs.

Isoniazid neuropathy is an axonal sensorimotor neuropathy with mental symptoms and skin changes similar to pellagra, and conditioned by a reversible pyridoxine depletion due to the genetically linked slow acetylation of isoniazid.

Thalidomide—Less notorious than phocomelia is the unpleasant burning dysaesthesiae of hands and feet with peripheral impairment of pain sensation often restricted to the digits after thalidomide. Unfortunately half of the patients remain without improvement after many years.

Nitrofurantoin (Furadantin) in the presence of renal impairment may, through failure of detoxication, lead to a refractory, symmetrical mixed neuropathy with reduced motor and sensory nerve conduction and segmental demyelination.

Clioquinol—A particularly virulent condition, subacute myelo-optic neuropathy, with a demyelinating syndrome of optic neuritis, myelopathy, and peripheral neuropathy has now been aetiologically linked with taking clioquinol (Enterovioform) for traveller's diarrhoea.

Chloroquine—With prolonged, high doses of chloroquine a motor polyneuropathy may develop. Recovery is rapid after drug withdrawal, but there may be an associated retinopathy and a myopathy with vacuolation and glycogen accumulation in muscle fibres.

Vinca alkaloids (vincristine and vinblastine), used to treat leukaemia, are particularly liable to neurotoxic effects, producing a painful sensorimotor polyneuropathy with axonal degeneration, secondary demyelination, and focal necrosis of muscle fibres. The autonomic nervous system may be involved, with constipation and intestinal obstruction, and, especially with intrathecal use, there may be extensive neuronal damage, convulsions, and ophthalmoplegia.

Perhexiline maleate (Pexid) may produce a reversible distal sensory neuropathy, proximal myopathy, and autonomic dysfunction with a raised protein content in cerebrospinal fluid that may lead to further complications.

Disopyramide (Norpace, Rhythmolan), another cardiac drug, may produce a mild sensorimotor neuropathy of large fibre type with dysaesthesia of the feet.

Glofibrate (Atromid S) has occasionally produced a proximal myopathy with a distal motor neuropathy, severe myalgia, and muscle stiffness.

Disulfiram (Antabuse) and *cyanamide* (Abstem), used to treat alcohol addiction, can produce sensorimotor neuropathies and disulfiram may also cause optic neuritis and myopathy.

Metronidazole—A final example of a drug-induced polyneuropathy is that of metronidazole (Flagyl), used as a bactericide, protozoacide, and potentiating agent for radiotherapy. It may produce a mild distal sensory neuropathy, but occasionally a more severe neuritis with intolerable dysaesthesiae can result.

Convulsive disorders

The list of drugs capable of producing convulsions is formidable and examples are given in order to stress how such reactions arise; from irritants injected into the subarachnoid space, such as penicillin, water-soluble contrast media and antimitotic drugs; intravenous injections, particularly at an unstable pH—for instance, the penicillins and drugs to correct cardiac arrhythmias, such as lignocaine and disopyramide (Norpace, Rhythmolan); penetrative antituberculosis drugs affecting pyridoxine metabolism, such as isoniazid and cycloserine; most antidepressives, including maprotiline (Ludiomil), amitriptyline (Tryptizol), and viloxazine (Vivalan) have mild cerebral irritant properties and this function of chlorpromazine (Largactic) has been used to display instabilities in the electroencephalogram; hypoglycaemic agents, by causing too rapid a reduction in blood sugar; electrolyte imbalance, as from dilutional hyponatraemia caused by infusion of 5% dextrose solution; and pheny-

toin in toxic doses becomes a convulsant, inducing attacks that may resemble hysteroepilepsy.

In narcotic institutions care is taken to titrate the withdrawal of barbiturates, more so than opiates, to prevent the risk to life of withdrawal fits. Similarly, if fits develop after a suicidal overdosage of barbiturates the treatment is always phenobarbitone and not other anticonvulsants. The cause of status epilepticus in established epilepsy is almost invariably the result of a reduction in anticonvulsants. Doctors should recognise that the taking of anticonvulsants is an imposition and that a psychological reaction is to be expected, particularly from teenagers. It is imperative to emphasise the advantages of regular medication in reducing the chances of kindling attacks in later life and to keep the regimen as simple and non-interfering as possible.

Anticonvulsant medication

Successful treatment with anticonvulsants depends on stable, therapeutic blood levels. Drug interactions, however, may occur in one or more phases of drug action: the drug release phase, for instance, within the intestine; the pharmacokinetic phase in which absorption, distribution, enzymic biotransformation, and excretion determine the concentration of the drug; and the pharmacodynamic phase in which drug and receptor combine to give a response.¹ Both phenobarbitone and phenytoin are potent inducers of hepatic enzymes lowering the serum concentrations of other drugs. At the same time, competition for plasma protein binding sites or inhibition of phenytoin metabolism by isoniazid or sulthiame (Ospolot) may rapidly produce toxic concentrations of phenytoin. The side effects of phenobarbitone include mental dulling and paradoxical hyperkinesia, especially when used in a brain-damaged child. Carbamazepine (Tegretol), an effective drug for focal epilepsy, may cause water intoxication and the practice of pushing fluids in a burnt epileptic child may thus be rendered hazardous.

However, the most commonly seen drug-induced neurological state without doubt is the subacute toxic-ataxic syndrome with anticonvulsants. Fine nystagmus occurs with therapeutic levels of phenytoin but coarse nystagmus, dysarthria, or ataxia is indicative of the toxic-ataxic syndrome. With variations—including hypotonia, truncal ataxia, drowsiness, and confusion—this syndrome is seen not only with phenytoin but with other anticonvulsants such as primidone (Mysoline)—as an acute reaction on starting treatment—phenobarbitone, and carbamazepine (Tegretol). A less common syndrome and less readily diagnosed is the choreic-athetotic syndrome produced by toxic levels of the same drugs. The principal causes of these syndromes in practice are attempts to maintain patients on 400 mg or more of phenytoin without regular serum estimations or the thoughtless prescribing of combinations of Garoin (combined phenytoin and phenobarbitone) and phenytoin (Epanutin). Chronic phenytoin toxicity may lead to high levels of cerebrospinal fluid protein, encephalopathy, cerebellar degeneration, and a peripheral neuropathy (this latter is essentially of theoretical interest). Phenytoin is the best studied of all drugs and for this reason, perhaps, its side effects seem particularly horrendous. A myopathy has even been described as a sequel to anticonvulsant osteomalacia.

Extrapyramidal and allied syndromes

Few non-psychiatrists have the opportunity to study the normal responses of patients to lithium carbonate (Camcolit, Phasal, Priadel). Lithium salts are prescribed to treat and prevent manic-depressive episodes and probably act by altering electrolyte and amine levels in the brain. The toxic effects may include bizarre combinations of neurological symptoms, which are inadequately described in published reports; thus, to quote a personal case, I was asked whether a patient who happened to be

on lithium had Parkinsonism, cerebrovascular disease, or an underlying tumour. He appeared depressed with a slow, thick hesitant speech, kept one eye closed because of photophobia, had bilateral hand tremor, ankle clonus, flexor plantar responses, and locomotor apraxia. He responded dramatically to a reduction in the dose of lithium carbonate.

The side effects that may occur with plasma concentrations below 1.8 mmol/l include fine tremor, weakness, polydipsia, polyuria, hypothyroidism, and hypokalaemia; and any disturbance of water or electrolyte balance—as from diuretics, strict dieting, and sauna baths or interaction with potentially nephrotoxic agents such as tetracycline—can precipitate more serious reactions. Within the lower toxic range these may include ataxia, blurred vision, confusion, drowsiness, fasciculation, and slurred speech; and, with more severe poisoning, hyperreflexia, extensor spasms, convulsions, toxic psychosis, syncope, and coma. Serious interactions can occur with haloperidol, phenytoin, and diazepam. Lithium potentiates the side effects of other psychotropic drugs but combined treatment is possible, provided such drugs are started at a lower dosage than usual.

All effective major tranquillisers are capable of inducing extrapyramidal disturbances. This propensity is seen mainly with the piperazine series of phenothiazines (Stelazine, Stemetil, Fentazin, and fluphenazine), and butyrophenones (haloperidol); but a mild Parkinsonian syndrome can also occur in older patients on tricyclic antidepressants and with reserpine or tetrabenazine (Nitoman). Extrapyramidal movement disorders may follow the withdrawal of antihistamines, glutethimide (Doriden), antidepressants, and narcotics, probably due to an imbalance between cholinergic and dopaminergic activity in the basal ganglia, or be induced by the antimalarial amodiaquine (Camoquin). In one series,³ 40% of patients treated for major psychoses with phenothiazines developed extrapyramidal reactions: about 21% developed akathisia (motor restlessness), 16% Parkinsonism, and 3% dystonias—torticollis, writhing, grimacing, and oculogyric crises. These dose-related disorders are usually reversible; but not so the persistent (tardive) dyskinesias, which are related to the total amount ingested. Tardive dyskinesias are the scourge of long-stay mental institutions, affecting 12% of male and 20% of female patients.⁴ Typically, they affect the bucco-linguo-masticatory muscles in quasi-compulsive, stereotyped movements that may simulate the potentially reversible dyskinetic movements produced by levodopa. There is no value in the prophylactic administration of anticholinergic drugs for these conditions, and drug-induced Parkinsonism does not respond to levodopa. Retinal damage may result from the piperidine derivatives such as thioridazine (Melleril); and haloperidol (Seranace) has proved unsafe in thyrotoxicosis. A particular warning¹ is given against using the antiemetic metoclopramide (Maxolon) in childhood, as a single dose may incite acute dystonia or a reaction simulating an acute encephalopathy.

The incidence of severe side effects with monoamine oxidase inhibitors is highest in patients who are slow acetylators of isoniazid. Acute reactions include insomnia, agitation, hallucinations, hyperreflexia, hyperpyrexia with neck rigidity, convulsions, and hypo- or hyper-tension. More chronic side effects include tremor, peripheral neuropathy, orthostatic hypotension, hypomania, oculomotor paralysis, and toxic amblyopia; but most hazardous are the hypertensive reactions with headache and intracranial haemorrhage when a monoamine oxidase inhibitor is given simultaneously with certain drugs or foods. The adverse neurological manifestations of the tricyclic antidepressants and allied drugs usually stem from their anticholinergic actions, precipitating glaucoma, convulsions, or various states of hallucinosis, delirium, or manic excitement. With acute overdosage, hyperpyrexia, hypertension, and seizures may result in a coma potentially reversible with physostigmine. The major hazard of the benzodiazepines comes from the variability of response: thus a paradoxical reaction can occur with excitement or depression instead of sedation. There is also the risk of dependence of the barbiturate-alcohol type with with-

drawal reactions including convulsions and potentiation of other drugs; thus hypotonia, ataxia, erratic behaviour, and drowsiness may be observed with clonazepam (Rivotril), especially when used in conjunction with other anticonvulsants.

Parkinsonism is a degenerative disease often accompanied by depression and loss of drug tolerance. Although anticholinergic drugs may add to the risk of confusional states, they act synergistically with levodopa and their subsequent withdrawal may lead to hypersalivation and intense rigidity. Currently, an immediate problem is the need to nullify the dyskinetic and on-off effects resulting from long-term treatment with levodopa. In this regard the efficacy of bromocriptine (used successfully in lower dosage for endocrine dysfunction, anorexia nervosa, and hypertension) has been questioned. Twenty-five to 40% of Parkinsonian patients are unable to tolerate therapeutic doses of this drug, and in those who have received levodopa refractory visual hallucinations and confusional states often

occur.⁵ Bromocriptine may also have the harmful effect of hypersensitising dopaminergic receptors.⁶

The most serious omissions in this review include a description of drugs that produce confusional states, depression, or damage the ocular system. Drug syndromes should always be considered, especially when a patient apparently benefiting from treatment suddenly develops untoward and unexplained symptoms.

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MATERIA NON MEDICA

Thatching

Thatching has come to mean something different. Jackie Charlton is now its most famous exponent. Watch him next time on television casually tossing the temporal hairs on the left across to join the temporal hairs on the right. Better than a topper? Better to be honestly bald. Here, where the bare granite bones break through the thin bog skin, thatching has its old meaning. Its exponents are few and aging. The knowledge is dying with the knowledgeable.

The best thatch is wheat straw. It will last six or seven years. It needs no ropes to tie it down. Sally rods act as ties. It is dry and warm but hard to get. The Lagan of east Donegal is the nearest place and not many go there.

Bent grass from the sand dunes is the usual material. It is gathered in sheaves and delivered as a beart. Each beart has twenty sheaves. It is laid on the roof and tied down with ropes that are tied to stones that stick out from the wall near the top.

Mary Ward has an old house built after the famine. Her family are grown and all away except Patrick, who works in a factory in Gweedore. She is getting a new council house built in front of the old one next year. She won't let them toss the old house: it could be a store or a byre or anything in time. The room is slated, the kitchen thatched, and the byre has a cement roof. The kitchen was thatched last summer. She needed only one beart, which cost her £4. The ropes cost £3.50. She paid two old men £2 each and gave them their dinner to do the job. The day after they finished it poured with rain and the roof leaked. She went out and bought plastic sheeting to cover the thatch. It cost her £5.50. "It can stay there now till the roof caves in." It may well outlast the slates and cement. It will certainly outlive that last poor thatching.—B DECLAN BONAR (physician, Dungloe, Co Donegal).

It's an ill wind . . .

In the previous winter there had been typhus in Kurdistan. Craigie had recently introduced his vaccine. This seemed an opportunity to test its efficacy. The plan was to inoculate all the inhabitants of one Kurdish village, to leave surrounding villages unvaccinated, and to return in spring to assess results.

Permission was obtained from the health authorities of Iraq and from the Medical Directorate of PAIFORCE. The chief medical officer, Sulaimaniya Liwa, and the medical officer of Halebja, the village chosen, gave willing help.

The major in command of the PAIFORCE laboratory, his sergeant and batman, my wife (also a bacteriologist), and I journeyed by train to Kirkuk, then by Iraq government ambulance to Halebja, where we installed ourselves in the two offices of the hospital. At first, we asked the population to come to us for vaccination and were helped by the local musician, who, with his whistle, led a procession—after the style of the Pied Piper.

All the inhabitants could not come to the hospital, so after a few days we went out to the village, each of us accompanied by a cheerfully willing small boy carrying a haversack with vaccine, syringes, lamentably few needles, and methylated spirit to "sterilise" them. (Fortunately for our peace of mind, we did not then know about

Hepatitis B virus; but perhaps the population was already saturated.)

Sometimes the local Kurds would play tricks on us, by making a V sign to indicate vaccination on their arms with a stain other than our Ziehl-Neelsen, or hiding behind grain sacks. This was all good fun and roars of laughter followed discovery. There was no difficulty in vaccinating a household once the master had been done. He would pull out his womenfolk, then the children, in descending order of age; in one instance the youngest brought the cat.

The second round of vaccinations went almost as well as the first and we felt that we had done a thorough job. In the spring we returned to assess the results. There had, indeed, been no typhus in Halebja, but neither had there been any in the surrounding villages. We should have known better; typhus is unlikely to strike twice in succeeding years.

But our efforts had not been fruitless. To our joy we found that the children had a new toy: a hollow reed into which was inserted a solid stick and with this they sucked and they squirted to their hearts' content.—C P BEATTIE (emeritus professor, Sutton-in-Ashfield).

Quacks and cluckers

1978 was a good year for birds. Those seen in the garden included the resident house martins, a family of partridges, a flock of goldfinches, treecreepers, a tawny owl, and a kestrel, with pride of place going to the woodcocks which nested in the woods behind the house. A pair of pigeons had the impudence to nest in the hen run, where they successfully reared their young on hen food and fresh garden vegetables. Once the fledglings had flown, my horticultural instincts gained the upper hand and I asked a general practitioner friend to fire a few warning shots into the air to discourage further ravaging of the greens. This he happily did. But he forgot to collect his decoy pigeon, and by the following morning my brassicas had taken a beating.

The hens remain the major ornithological interest, although "cocky" (*BMJ*, 1976, **2**, 580) is no more. My youngest daughter kept a daily chart which related egg production to weather, feeding, etc and could provide the basis of a future PhD thesis. We collected 6159 eggs, excluding those laid secretly in the garden by hens that escaped from their run. On 4 May a hen went broody, and three weeks later she started to foster 15-day-old shaver chicks obtained from the Poultry Research Institute. A second clucker fostered 15 thorbers. The overall perinatal mortality was 13.3%. The shavers are remarkable birds. They ignore their hen house and would rather roost in a yew tree, even in six inches of snow. The six cockerels vie with each other in welcoming the dawn, and, irrespective of the time, greet any car with its lights on coming down the drive after dark with a cacophony of crowing.

The pullets are elegant white birds which fly like pheasants. They first laid on 28 November and two have continued to lay in spite of the appalling weather. We tried to coax them to use the nesting boxes by putting out a wooden egg. This vanished, and if, as I suspect, it was taken by rats, then I hope they were as irritated by their find as I was by my loss. Still, one of the pullets has solved the rat problem. Has anyone else got a hen that lays eggs 10 feet up a yew tree in a disused pigeon's nest?—J F MUNRO (consultant physician, Edinburgh).