EMERGENCIES IN GENERAL PRACTICE

AGRICULTURAL PESTICIDES

BY

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This article sets out to help general practitioners, especially those in areas of intensive agriculture or horticulture, to deal with emergencies arising from the use of chemical pesticides. A short article can only emphasize the more important facts, in the hope that interested practitioners will then seek further information.

Chemicals Used

There is now a large and annually increasing range of pesticidal chemicals, with many proprietary brands and special-purpose variants. These chemicals differ so much in toxicity to animals and potential hazard in use that any attempt to divide them into such simple groups as "comparatively safe" and "hazardous," as in the Table, may need revision later. The safest chemical can be dangerous if sufficiently mishandled, while the most hazardous can be safely used so long as adequate precautions are maintained.

Pesticides in Common Use

Purpose	"Comparatively Safe"	"Hazardous"
General herbicides	Borates Sodium chlorate *C.M.U.	Sulphuric acid (corrosive) Arsenites
Selective weed-killers	Sodium trichloracetate 2,4D } Hormone- 2,4,5-T } type M.C.P.A. } growth *M.C.P.B. } regulators *Pentachlorphenol	D.N.C. (dinitro-*o*-cresol) D.N.B.P. (dinoseb, dinitrobutyl phenol)
Fungicides ..	Copper compounds Sulphur Zineb. Thiram Lime sulphur Dilute D.N.C. or D.N.B.P. Captan	Lead arsenate Organic mercurials, e.g., ethyl, methyl, phenyl, or other mercurial salt. (Also used in seed-dressings)
Crop defoliants ..	Monochloracetate	D.N.C. Arsenites
Insecticides and acaricides	Tar oils Derris Pyrethrum. "PCPPCBS" D.D.T. B.H.C. and lindane *Aldrin, Dieldrin *Chlordane, Heptachlor Organic phosphorus compounds: *Malathion *Methyl-demeton	Lead arsenate Nicotine Organic phosphorus compounds: Parathion T.E.P.P. (H.E.T.P.) Schradan Dimefox Demeton

* These chemicals are of recent introduction and their comparative safety is provisional.

Over 90% of British pesticide usage involves the comparatively safe chemicals listed above. Many growers still prefer to pay professional spraying contractors to carry out hazardous operations, but the more experienced are now tending to use the hazardous chemicals themselves.

Occupational Hazards

The main occupational risks are unremoved splashes and smears of chemical on the skin, chiefly on the hands, from handling concentrated preparations or contaminated equipment, and the prolonged inhalation of airborne mist or dust whilst spraying. Ingestion may sometimes occur—for example, from blowing or sucking at blocked jets or contamination of food or water. Eye contamination may produce local effects but hardly ever any risk of poisoning. Vapour concentrations out of doors are rarely high enough to be of toxicological importance; the important inhalational hazard is that of particles, not vapour. Frequently repeated minor over-exposures are markedly cumulative in their effects, but early effects are usually slight and tend to be ignored or misconstrued until acute illness begins to show itself. Risks are dictated not simply by the nature of the chemical used but also by the suitability and serviceability of equipment, the worker's own care and cleanliness, and the duration of his exposure.

The chief users of the hazardous chemicals are spraying contractors and their employees, farm workers, workers in greenhouses, orchards, hopfields, and market gardens, private farmers, workers in seed-dressing establishments, and scientific workers. Deliberate or accidental ingestion may occur in adults or children, as dangerous chemicals are often insecurely stored by purchasers. The hazardous chemicals are not easily available to or much used by amateur gardeners, whose occupational exposure and risk is therefore small.

The period of most intensive use and consequent risk is from early April to August or September, the peak months being May, June, and July. Periods of fine hot weather intensify field spraying and sharply increase risks. Pesticide usage is most concentrated in the Eastern, South-eastern, and Midland counties, but is quickly extending.

As in most other countries, fatalities occurred in England during the early years of use. Eight agricultural deaths occurred between 1946 and 1951, all in contract spraying operators in hot weather, from the intensive use of D.N.C. as weed-killer, obviously without adequate safety precautions. The ninth death occurred in a glasshouse worker in Guernsey in 1954, due to over-absorption of parathion. There have also been a number of cases of mild poisoning, but it is encouraging that there has been no fatality or serious accident from the use of D.N.C. since 1951, despite its wider use.

Emergencies from "Comparatively Safe" Chemicals

With chemicals of comparatively low toxicity, emergencies are only likely from accidental or deliberate ingestion of large single doses. The effects would then probably include violent nausea, prompt severe vomiting, purging, and abdominal pain, leading on to mental confusion, collapse, tremors, stupor, circulatory failure, respiratory distress, cyanosis, convulsions, and death in coma. With such compounds the full severity of the case may not be apparent for some hours or even days. If there is prompt vomiting or a gastric wash-out is performed quickly, a patient may survive the immediate effects, later to show prolonged weakness, or delayed further illness, due to secondary effects on, for example, the liver, kidney, or nervous system. Such effects may at times be due to the solvent, not to the active chemical. The prognosis should be guarded for at least one or two weeks.

General procedures in such medical emergencies are suggested below. The modes of toxic action of the less hazardous chemicals are rarely known, and, with two exceptions, there are no specific antidotes or pharmacological antagonists to be recommended. The most urgent needs are the rapid removal of residual chemical from the stomach, followed by good nursing, symptomatic and supportive treatment, with careful observation during the recovery period for late effects on the liver, kidney, blood, nervous system, or gastro-intestinal tract.

Poisoning by the comparatively safe organic phosphorus insecticides malathion and methyl-demeton should be treated as later advised for the more toxic organic phosphorus compounds. Pentachlorphenol closely resembles D.N.C. and D.N.B.P. in its manner of disrupting carbohydrate metabolism, and acute poisoning should be treated as suggested for the more toxic compounds.

Emergency Procedure in Pesticide Poisoning

Most of the following suggestions are elementary, but they are often forgotten, despite their proved importance in any case of acute poisoning:

1. Make urgent inquiries about what chemicals had been used in the past few days, and examine any available instructional leaflets or container-labels from the immediate place of work. Note that workers often mix a hazardous with a safe chemical—for example, parathion with D.D.T., or T.E.P.P. with a copper fungicide. Treat as for the most dangerous.

2. Start any recommended treatment immediately. Cases with rapid onset tend to deteriorate further, and speed is vital.

3. Unless already done, carefully remove all outer working-clothes and thoroughly wash the exposed skin with soap and water to remove unabsorbed chemical.

4. If the chemical is known or believed to have been swallowed, induce vomiting unless the patient has already vomited repeatedly. In all cases it is preferable to pass a well-lubricated gastric tube, evacuate the stomach contents, and wash out repeatedly with warm water. About one pint (half a litre) of a dilute solution of sodium bicarbonate (1%) may then be left in the stomach. If available, activated charcoal (1 or 2 tablespoonfuls) could be given to conscious patients.

5. If the patient is obviously ill, disturb him as little as is compatible with treatment. Exertion will dramatically increase the severity of effects, especially with the hazardous compounds, and may lead to death.

6. Rapid effective sedation is therefore necessary for restlessness, excitement, or apparent imminence of convulsions—for example, injection of sodium amylobarbitone, 0.4–0.8 g. (7–12 gr.), or sodium phenobarbitone, 100–200 mg. (1½–3 gr.), intravenously in urgent cases, or otherwise intramuscularly. Morphine should not be used.

7. Anticipate coming therapeutic needs—for example, for oxygen supplies, saline, or glucose-saline infusion.

8. Do not send a seriously ill patient off to hospital unless it is quite certain that this will be the best for him, and that he can receive all necessary treatment *en route*.

9. Save vomitus, chemical container, and the worker's clothing, in case of later inquiries.

10. If in doubt about diagnosis, treatment, or procedure seek outside medical help.*

11. The patient's workmates may also be approaching a dangerous degree of over-exposure. They should be examined and not allowed to return to work or home until it is quite certain that they are fit to do so.

12. Ensure that the occurrence is notified to the local Ministry of Agriculture safety inspector.

Their extent of usage and worker-hazards suggests that attention should now be focused on D.N.C. and organic phosphorus compounds, and then on the less widely used arsenical, mercurial, and nicotine compounds.

Dinitrocresol Poisoning

Absorbed D.N.C. or D.N.B.P. rapidly upsets carbohydrate metabolism and increases the basal metabolic rate, an effect usually tolerable in cool or cold weather conditions, but liable to cause dangerous effects when heat loss is limited by, for example, hot sultry weather. The general clinical picture is that of a worker with yellow-stained hands, face, or hair, with excessive sweating, thirst, and air-hunger.

Mild cases may show nothing but copious sweating and thirst; the patients are sometimes feeling particularly well, presumably owing to the moderately increased metabolic rate. *Severe cases* are likely to show profuse and continuous sweating, fast pulse, nausea, vomiting, weakness, malaise, restlessness, dehydration, yellow sclera, scanty urine of fluorescent yellow hue, albuminuria, and deep rapid breathing. *Fulminating cases* may be drenched in sweat, dyspnoeic, cyanosed, comatose, or having convulsions even when first seen. There is usually a history of a few days' progressively increasing, but ignored, sweating and tiredness. Body temperature may be raised.

Laboratory tests in severe cases would show a tremendous rise in basal metabolic rate and the presence of D.N.C. in the blood and the urine. Photoabsorptiometric methods of determining D.N.C. levels in the blood have been described by V. H. Parker* and D. G. Harvey,† and I have recorded a simple comparator method, eminently suitable for both emergency and routine purposes.‡ All hospitals in the major cereal-growing areas should be equipped to determine blood D.N.C. levels very quickly (see Appendix for details of collection, etc.). Symptoms are likely to be absent below 25–30 μg. of D.N.C. per ml. of whole blood, mild at 30–40 μg. per ml., severe at 45–55 μg. per ml., very severe at 55–65 μg. per ml., and critical at over 65–70 μg. per ml. The analytical methods apply equally well to D.N.C. and D.N.B.P., which are equivalent in hazard and effect. Since serum protein binds most of the circulating D.N.C., serum D.N.C. levels are twice as high as those given above for whole blood; some hospital laboratories quote serum levels. Urinary D.N.C. levels are not a good index of absorption or of the severity of the case. Whole-blood D.N.C. levels normally decrease at about 1 μg. per ml. of blood a day on removal from further exposure. The yellow staining of the skin is a specific persistent dyeing action, extremely difficult to avoid even in careful workers, and needing days or weeks to wear off. It does not therefore reflect the severity of the case, nor even the certainty that the case is one of D.N.C. poisoning.

Treatment

There is as yet no known specific antagonist to the effects of D.N.C. in the body, and treatment is aimed at the rapid promotion of maximum heat loss and the complete relief of anoxia, restlessness, and dehydration. *Mild cases* require little but a few days' enforced rest, freedom from further exposure, frequent cold sponging, minimum clothing and bedding, and abundant soft drinks. They should not resume work with D.N.C. for a month, or until the blood D.N.C. level is down to 5 μg. per ml. (All users are strongly urged by manufacturers to seek medical advice as soon as they notice unusual sweating.)

The most effective emergency method of cooling the severely affected patient is to remove his clothing and cover him with a wet sheet subjected to vigorous air movement to cause evaporative cooling—for example, by relays of helpers with large fans, electric fans, or a reversed vacuum cleaner acting as a blower, preferably carried out in the shade with free ventilation. Water should periodically be sprinkled on the drying sheet, or a fresh wet sheet used. Cold wet cloths to the head, ice-packs, ice to suck, and abundant cold drinks for the conscious patient, and, if possible, a cold-water rubber-bed or wetted stretcher, all promote further heat loss.

Restlessness demands the use of quick-acting barbiturates. *Dehydration* is almost certain to exist; conscious patients should drink copiously and often of cold drinks with added glucose (1 oz. per pint—40 g. per litre) and salt (1 teaspoonful per pint). An unconscious patient should ideally have a fast intravenous glucose-saline drip. Failing this, large volumes of rectal saline should be administered. *Dyspnoea* demands oxygen therapy by nasal tube, oral tube, face-mask, improvised "cellophane" or paper head-tent and open tube, or resuscitator.

*Specialist advice in such cases may be sought from the Medical Research Council's Department for Research in Industrial Medicine, the London Hospital, London, E.1. (Tel.: Bishopsgate 3255); or, when suggested on container labels, from the medical departments of the larger manufacturing concerns.

Analyst, 1949, **74**, 646.
†*Lancet*, 1952, **1**, 796.
‡Ibid., 1954, **1**, 981.

The acute illness may last for 12–24 hours, and the above treatment may be required throughout that time. The *prognosis* in the fulminating cases is bad despite energetic treatment. Severe cases need a guarded prognosis for the first 24–36 hours, but thereafter the outlook improves. No delayed after-effects have as yet been reported in recovered acute cases. There should be no exposure to D.N.C., D.N.B.P., or other hazardous chemicals for at least two months; this usually means until the next season.

Organic Phosphorus Insecticides

All present-day organic phosphorus (O.P.) insecticides cause similar toxic effects, by inactivating acetylcholinesterase enzyme (ChE) of the tissues and thus permitting acetylcholine to accumulate dangerously. Since ChE regenerates only slowly, frequent small over-exposures are cumulative in their effects but often symptomless until ChE reserves fall to perhaps 5–20% normal. Illness then occurs, first affecting involuntary muscles and secretory glands, then the voluntary muscles, and finally vital brain centres.

Early on mild cases with gradual onset show malaise, fatigue, weakness, cold sweats, pallor, nausea, vomiting, abdominal discomfort, irritability, apprehension, or restlessness. *Severe cases* develop headaches visual disturbances, confusion, vomiting, colic, diarrhoea, dyspnoea, and fine fibrillary tremors of hands, lids, face, or tongue. *Very serious cases* develop salivation, lacrimation, bronchorrhoea, severe diarrhoea, incontinence, basal rales, circulatory collapse, cyanosis, severe dyspnoea with a sense of suffocation, mental confusion passing to stupor, muscular weakness with tremors and convulsions, and coma. *Fatal cases* end with gross pulmonary oedema, cyanosis, bradycardia, and fall in body temperature.

The pupils may be pinpoint and fixed, and there may also be a sense of asthma-like tightness of the chest. These effects can, however, be misleading, being producible by the local eye or chest effects of exposure to spray mist of T.E.P.P., parathion, or demeton. Where present, they are presumptive of exposure, but not necessarily of poisoning. On the other hand, severe poisoning can occur with some organic phosphorus compounds—for example, schradan or dimefox—without miosis or bronchial constriction. The clinical diagnosis of organic phosphorus poisoning relies on concurrent effects on secretions, muscles, and brain. There are no external indications of contamination, but there will be a history of exposure within the past few days. The confirmation of diagnosis relies on demonstrating reduced ChE activity in the circulating blood. The technique is specialized, but is now available at certain regional laboratories (see Appendix). A simple colorimetric method has recently been introduced, suitable for emergency and routine testing. Activity is usually expressed as percentage of normality of healthy adults. Values above 80% normality imply that no significant absorption has taken place; there is no important upper limit. Cases with values between 50% and 80% normality are usually symptomless, but indicate over-absorption. Slightly early effects, mainly abdominal discomfort and cold sweats, may occur at 20–30% normality. Severe cases would be expected to have very low ChE activity in the blood—for example, 5–10%—but some apparently severe cases of parathion poisoning have had up to 50% normal values, while some cases of over-exposure with ChE levels reduced to 5% normality have proved symptomless. Thus, ChE levels are vitally important for confirmation of the diagnosis of an illness or over-exposure in workers, but low activity results may bear no obvious relationship to the clinical severity of effects.

Treatment

The recommended general emergency procedures should be carried out as they apply. The specific antagonist to organic-phosphorus-induced hypersecretion, involuntary muscle contraction, and some central effects is *atropine sulphate*. Unfortunately, it does not prevent muscular tremors or weakness. All but very minor cases should be given atropine sulphate *immediately* (unless the worker has already had an initial oral dose elsewhere) in *high dosage* (initially 1–2 mg. (1/50–1/30 gr.)) intramuscularly, or intravenously in very ill patients; then ½–1 mg. (1/100–1/50 gr.) subcutaneously, *repeatedly* (at intervals of ¼–3 hours, depending on the severity of the case) and *for a prolonged period* (12–48 hours, if the patient's condition remains poor). The atropine dosage needed in organic phosphorus poisoning vastly exceeds orthodox dosage schedules for this drug. The risk is of giving too little in severe cases, rather than too much. In a recent severe case, for example, 45 mg. of atropine (¾ gr.) was given intravenously in three hours, with recovery.

Here a special word of warning must be given:

1. If atropine is given erroneously in D.N.C. poisoning, the patient will stop sweating and may then die. The two types of poisoning must never be confused.

2. Practitioners should send the receiving hospital details of atropine and other drug dosage already given.

Restlessness must be combated immediately by quick-acting barbiturates. *Dyspnoea* demands continuous oxygen. *Pulmonary oedema* requires quick recognition and postural drainage. A recent suicidal case in Germany showed dramatic improvement in lung oedema after the injection of a mercurial diuretic; in a severe case injectio mersalyli (2 ml.) should be given intravenously.

Sudden failure of respiration justifies prolonged artificial respiration, preferably with positive pressure. Breathing may be resumed and survival follow. Care should be taken not to overload an already struggling circulatory system by saline infusion or transfusion of plasma or blood. On theoretical grounds, harm would result. Similarly, morphine should not be used. Good nursing is required, and the patient must not be left unattended while acutely ill.

The *prognosis* in very severe cases is poor, despite treatment. In such cases it is apparently not enough to have dried up the secretions and eased the involuntary muscles and anoxia; central effects dictate survival. Severe cases may be pulled through by intensive treatment. Mild cases usually respond extremely well to atropine. Full recovery can usually be expected, even in severe cases, if the first few days are survived, but careful observation should be kept for delayed effects on the nervous system for some weeks after organic phosphorus poisoning.

Cholinesterase activity returns to normal at the rate of about 1% a day, so a severely affected person should have no further exposure for at least three months. It should be noted that persons with a temporarily low cholinesterase activity may be hypersensitive not only to all organic phosphorus insecticides, but also to anticholinesterase muscle relaxants, procaine, neostigmine, eserine, or D.F.P.

Arsenical Preparations

There is increasing use of arsenites as weed-killers and defoliants. The toxic effects of over-exposures may include reddened eyes, skin rashes, sore throat, hoarseness, coryza, vomiting, diarrhoea, headaches, hallucinations, haematuria, aphasia, and peripheral nerve lesions. Eventual confirmation of the diagnosis is by finding arsenic in the urine in excess of the normal 0.008–0.15 mg. of As per litre of urine. The specifix antidote to be used along with the general emergency procedures is dimercaprol in doses of 200 mg. intramuscularly every four hours for two days, and reduced doses for ten days.

Organic Mercurials

Organic mercurials occur in many fungicidal seed-dressings and spraying mixtures. Fatalities have occurred in Europe owing to the volatility of ethyl and methyl compounds, but inhaled dusts are also hazardous. The possible effects include gastric upsets, headaches, trembling, clumsiness, slurred speech, confusion, ataxia, nervous irritability, and

change in temperament. Rashes or blistering of exposed skin may occur as local effects of primary irritancy. The diagnosis may be confirmed by finding over 20–30 μg. of Hg per litre of urine. Dimercaprol therapy should be carried out in severe cases, although the results are not likely to be as favourable as in arsenical intoxication. The neurotoxic effects of mercurial poisoning tend to persist or slowly increase, and prognosis must be guarded. The skin lesions respond well to orthodox treatment as for thermal or minor chemical burns.

Nicotine Preparations

Nicotine still has considerable use in horticulture. Its toxic effects, most serious in non-smokers, include nausea, vomiting, pallor, vertigo, palpitations, tachycardia, chest pain, excitability, diplopia, incoordination, and collapse. Nicotine is less cumulative as a result of minor over-exposure than are most pesticidal materials. There is no specific antagonist to the effects of absorbed nicotine, treatment being that suggested under general emergency procedures, and symptomatic.

Prevention of Over-exposure to Pesticides

During the coming four to five months over 2,000,000 acres in this country will be sprayed with crop-protection chemicals. About one-tenth of this acreage will involve the use of hazardous pesticides, often under conditions of some risk. The safeguarding of potential worker-casualties from these modern agricultural operations remains a matter of great concern to users, employers, distributors, manufacturers, and Government departments. Many real steps have been taken to ensure safety, so far as this is currently possible, by mutual collaboration between these groups and by legislative measures. The practitioner is himself able to make some important contributions. He should be able to recognize and treat casualties as effectively as present knowledge permits. Medical treatment often stands a poor chance of success in serious cases, however, so whenever an opportunity presents itself he should enjoin on his user-patients the need for care, especially in the prevention and avoidance of contamination. He could also detect unsafe working methods by arranging for periodic analyses of blood D.N.C. levels in major users of dinitro-weed-killers, or for ChE levels in organic phosphorus insecticide users, and could with advantage carry out periodic scrutiny of workers, especially of contract spraying operators, during their season of major risks.

APPENDIX

The following details about laboratories to which specimens may be sent for blood D.N.C. and blood cholinesterase estimations are based on information provided by the Ministry of Health.

Blood D.N.C. Estimation.—Five millilitres of oxalated blood is required for this estimation. Blood must be taken in a dry syringe not previously sterilized with spirit. The specimen, or the patient, should be sent to the hospital to which the practitioner normally refers patients for pathological tests of any kind.

Blood Cholinesterase Estimation.—Five millilitres of oxalated blood, taken under the same conditions as for D.N.C. estimation, is required. Specimens may be sent either direct to Dr. E. N. Allott, Group Laboratory, High Street, Lewisham, London, S.E.13 ; Dr. E. M. Darmady, Pathological Laboratory, Central Laboratory, Milton Road, Portsmouth ; Dr. D. H. Fulton, Memorial Hospital, Peterborough ; or Dr. P. Kidd, Pathological Department, Royal Infirmary, Worcester ; or, alternatively, the specimen should be sent to the hospital laboratory which the practitioner normally uses. Specimens sent through the post should go by letter post and be marked "Pathological Specimen (Agricultural Poison)."

Next article on Emergencies in General Practice.—"Diabetic and Insulin Coma," by Dr. J. D. N. Nabarro.

SELECTION OF PATIENTS FOR PSYCHOTHERAPY

Which patients with neurosis should be accepted for systematic therapy? This is a question that confronts every psychiatrist, and it is made no easier by the very large number of such patients now coming forward for treatment. In a lecture at the St. Marylebone Hospital for Psychiatry and Child Guidance on March 23, Dr. E. A. BENNET addressed himself to this problem.

Psychotherapy, said Dr. Bennet, took many forms: the kindly interest shown by the family doctor towards his patient; the psychological element accompanying physical treatments in psychiatry; art therapy; and, in particular, the methods ranging from explanation and suggestion to the often rigidly ordered types of analysis employed in the neuroses. Before accepting any patient with neurosis for systematic treatment—their present concern—at least two preliminary interviews were desirable, and these should be therapeutic as well as diagnostic.

Therapist and Patient

Psychotherapy, unlike most methods of treatment in general medicine and surgery, was always conducted upon a personal basis. The therapist was in the treatment, and for better or for worse his personality counted. Patients might select their therapist when treatment was arranged privately, but in hospital or clinic this was rarely possible. Nevertheless, in a department with a big staff it was essential, in the selection of patients, to take note of the personalities, predilections, and preferred methods of the therapists. One therapist might choose patients in the younger age groups, another do his best work with those in the second half of life, a third use supportive methods, and a fourth formal analysis.

Further, in selecting, the psychiatrist must do his best to decide whether a particular patient was likely to respond to the treatment available and so justify the heavy expenditure of medical time involved, before adding his name to the waiting-list. The dull individual with an intelligence below the average should be rejected; likewise those with established psychotic symptoms, and reluctant patients sent (or brought) for treatment they did not want. Although such patients might be treatable, the demand for treatment was so high that the needs of others ruled them out.

The patient's expression of a need for psychological treatment should be taken seriously in the selection. An intelligent person, aware that there was something within his personality which needed attention, was often quite right. He might be successful in life and the antithesis of the "chronics" who frequented every medical out-patient department and became, to the student, the prototype of the "psychological case." Sometimes these patients reported that they had asked their doctor to send them to a psychiatrist and had been told that they did not need to see one—that they were not "the neurotic type." But, eventually, the doctor was persuaded to ask for an appointment. An importunate attitude on the part of a patient might even indicate suitability for psychotherapy.

The value of an affirmative diagnosis was often overlooked by doctors who diagnosed neurosis by the exclusion of organic disease, went on Dr. Bennet. Their medical training was responsible for this, and it was an attitude that frequently bred invalidism. Fruitless and often expensive investigations were disheartening, especially to an anxious patient, and the possibility of psychotherapy might be welcomed. Nor were psychotherapy and physical investigation mutually exclusive, but could well proceed *pari passu*. Likewise, when necessary, sedatives and other medicines should be prescribed by the psychiatrist, whatever type of psychotherapy he might be using. Some analysts would disagree with this and insist that medicine must be prescribed by another doctor, claiming that otherwise the transference