Current Practice

CHILD CARE IN GENERAL PRACTICE

Nephritis and Nephrosis

GAVIN C. ARNEIL,* M.D., PH.D., M.R.C.P., F.R.C.P.ED., F.R.C.P.GLASG.

Bright's disease is so bedevilled by differing classifications that the medical practitioner cannot but be confused. For clarity I have adopted the following simple scheme:

(1) Pyelonephritis	
(2) Haemorrhagic nephritis	Chronic renal failure
(3) The nephrotic syndrome	("chronic nephritis")
(4) Hypertensive states	

In childhood the most important groups are pyelonephritis, haemorrhagic nephritis, and the nephrotic syndrome, in that order. Pyelonephritis is discussed elsewhere in this series.

Haemorrhagic Nephritis

Acute Nephritis

This is a bilateral disease of the kidneys occurring most commonly in the child of school age. Approximately 85% of cases have associated infection with Lancefield group A, β -haemolytic streptococci. The patient usually presents with a complaint of passing brown urine. The urine may be tested for haemoglobin by tablet tests such as Occultest. The smoky colour of nephritic urine is related to the presence of lysed cells. Microscopic examination is essential to observe the red cells, cellular casts, and white blood cells which will be present in excess.

The second commonest cause of haematuria in children is acute pyelonephritis, and one should be on guard against this possibility if frequency or dysuria is complained of. Periorbital

oedema or enlarged cervical glands may have been noted. A complaint of headache or blurring of vision is a likely indication of severe and potentially dangerous hypertension. A story of a preceding respiratory infection may be obtained.

The general examination of the patient includes inspection of the skin for concomitant rash (purpuric or scarlatiniform). Periorbital oedema is often present and generalized oedema occasionally present. Examination of the cardiovascular system is particularly important. By 1958 it had become clear to us that a characteristic feature of acute nephritis was expansion of the blood volume accounting for temporary cardiomegaly and believed to be due to sodium retention and poor kidney function in water excretion. That increase in extra-cellular fluid is not usually accompanied by an increase in body weight even when oedema is present presumably is in part due to a shift of fluid within the body compartments. This "hypertonic" hypervolaemia is visible on radiographs of the chest as engorgement of the lung fields, enlargement of the heart, and sometimes as congestive cardiac failure (Fig. 1). To avoid aggravating the "hypertonic" hypervolaemia it is important to reduce the amount of sodium (salt) and water in the patient's diet.

Another possible cardiovascular complication is severe hypertension. The blood-pressure should be recorded at least once daily for the first week of illness until it settles as diuresis occurs. If a general practitioner is not in a position to carry out daily recording of blood-pressure, then the child is better in hospital.

Attention is principally focused on the diastolic blood-pressure. If this rises above 100 mm./Hg in a child with acute nephritis, phenobarbitone should be given (gr. $\frac{1}{2}$ -1 (30-60

FIG. 1.—Alteration in cardiac and pulmonary vascular pattern in acute nephritis. These manifestations of hypervolaemia are frequently unrecognized. Note the cardiac enlargement and pulmonary plethra in (a). The second radiograph (b) was taken one week later. During this week diuresis occurred and hypertension ceased.

* Leonard Gow Lecturer in Child Health, University of Glasgow.

mg.) b.i.d.) as a prophylaxis against convulsions. If the diastolic pressure exceeds 115 mm./Hg then hypertensive encephalopathy is imminent and immediate transfer to hospital is indicated. A complaint of headache or blurring of vision suggests such acute hypertension in a child.

The great majority of streptococcal infections will be of tonsils, pharynx, nose, or ears. It is common practice to take a swab of one tonsil, have it cultured, and defer treatment with antibiotics until a report is to hand. This is not logical. A negative throat swab does not mean that streptococcal infection is absent. We know that 85% of these patients have streptococcal infection (as judged by a high antistreptolysin titre—that is, above 200 units/ml.), that virtually all such streptococci are sensitive to penicillin, and that the duration of haematuria is related to that of streptococcal infection. In my opinion one should assume that a streptococcal infection is present in all cases of acute nephritis, and it should be treated with penicillin. Such treatment should not await a report on a throat swab.

The child should urinate into a chamber pot and not a water closet. The urine should be collected each day in a suitable bottle (a quart bottle is ample) during the oliguric phase. In this way the practitioner can assess the severity of the oliguria. The mother should be instructed to inform the practitioner at once if the child complains of headache, dizziness, or blurring of vision.

Treatment

Disposal.—A child with mild acute nephritis may well be treated at home if it is a good home with reliable parents and if the general practitioner is able to see the patient at least once daily during the initial period of hypertension and oliguria. A child from a poor home, or far from daily medical help, or with severe hypertension (diastolic blood-pressure >100 mm./Hg) or marked oliguria should be sent to hospital.

Antibiotic Therapy.—Treatment is specifically aimed at the elimination of streptococcal infection. The drug of choice is penicillin, which may be given either orally or intramuscularly. For the patient treated at home I would suggest oral penicillin-G (or penicillin-V) in dosage of 250 mg. four times daily. Since other members of the family may harbour the streptococcus that is responsible and could re-infect the patient, I would suggest full therapeutic dosage for a week, followed by prophylactic levels (penicillin-G, 250 mg. b.i.d.) for some weeks.

Diet.—The diet at first should be moderately low in water, salt, and protein. A simple diet would be one pint (568 ml.) of milk and one pint of water per day, with tablespoonfuls of lactose or sucrose added *ad lib*. In addition glucose sweets may be given freely. This strictly limited diet is continued while oliguria, hypertension, and sodium retention persist. Usually within a week diuresis occurs, blood-pressure falls, and dietary restriction can be relaxed. Cereals should then be prescribed to supplement the diet, and other "essentials" such as ice-cream, jellies, and so on can be added. After a few days on this regime the intake of milk is doubled to two pints daily and the patient is by then well on the road to recovery.

Rest in Bed.—This is of debatable value. While the hypertension and oliguria persist one would advise rest in bed. Entertainment such as jigsaws and comics should be provided and television or radio may be arranged in the room. The patient probably harbours β -haemolytic streptococci which could infect other members of the family, so he should be kept as separate from them as is practicable until 48 hours after he has started penicillin treatment.

When diuresis has occurred and hypertension lessens, the amount of activity may be increased, but it will be four to six weeks before the E.S.R. is normal. The speed of return

to activity should be judged by the rate at which haematuria (microscopic) disappears and the E.S.R. returns to normal.

Schoolwork.—It is highly desirable that the child should carry on with his school-work while he recuperates. In hospitals individual tuition should begin two days after admission. Not only does this keep up the progress of the patient's work but it helps to prevent boredom.

Treatment of Complications

The common complications are severe oliguria, progressing to anuria and acute uraemia; hypertensive encephalopathy; left heart failure.

Acute Uraemia.—In most instances the blood-urea level in a child with acute nephritis is raised to 50–100 mg./100 ml. blood (normal 15–30 mg.). When impairment of renal function is severe and oliguria prolonged, the blood urea rises progressively, an index of acute uraemia. It follows that when marked oliguria is detected (<10 fl. oz. (284 ml.) daily) the blood urea should be monitored every few days. If complete anuria occurs it is important further to reduce the fluid intake to one pint (568 ml.) only per day, and this should consist of equal parts of milk and water with three tablespoonfuls (50 g.) of lactose added. The child should go to hospital. He may well be vomiting, and if so treatment will probably consist of an intravenous drip of 500 ml. of 20% fructose daily. Treatment is continued on basically conservative lines because the condition is self-limited and recovery occurs spontaneously. The blood-urea level rises by about 50 mg. daily, and when it exceeds 400 mg./100 ml., or the serum-phosphorus level rises above 13 mg./100 ml., then dialysis may be required. This is very rarely necessary. Dialysis may be carried out by an extracorporeal artificial kidney or preferably by peritoneal dialysis, which is probably safer in small children. Dialysis removes the excess urea, phosphorus, potassium, and acid metabolites from the body fluids, and returns the serum phosphorus, calcium, sodium chloride, and potassium to normal. If the tonicity of the dialysing fluid is increased one can decrease the fluid content of the patient by removing water. Haemodialysis is more rapid (six hours), but it is difficult, more dangerous, and requires experts and instruments. Peritoneal dialysis is slower (36 hours) but relatively easy and can be carried out in any hospital with a biochemistry laboratory.

The other hazard of acute uraemia is a rise of plasma potassium to dangerous levels (>7.5 mEq/l.) at which the heart is affected. This danger is detected easily by repeated E.C.G. studies, and the plasma-potassium level may be lowered by the introduction of an ion-exchange resin (Resonium-A) rectally or orally. This removes potassium in exchange for sodium and may obviate or delay the need for dialysis.

Hypertensive Encephalopathy.—This is preceded by a rapid rise in diastolic blood-pressure, headaches, and blurring of vision. Since a fit may be the presenting symptom, subcutaneous phenobarbitone (Gardenal Sodium gr. 1½) should be given as soon as this threatens. It is for this reason that the blood-pressure should be checked in any child who has a fit for the first time. The convulsion may be stopped by an intramuscular injection of paraldehyde in dosage of 1 ml. per stone (6.4 kg.) of body weight. The child should immediately be sent to hospital, care being taken to ensure an adequate airway. A venesection of about 10 fl. oz. (250–300 ml.) will help to lower the blood-pressure for a short period.

In hospital a variety of procedures may be used—the administration of hypotensive drugs, lumbar puncture, hypertonic fluids rectally or intravenously, and further sedation.

Acute Heart Failure.—The signs of this are dyspnoea, tachypnoea, liver enlargement, cervical venous engorgement, and fine rales audible at the bases. Digoxin in initial dosage of 0.5 mg.

orally for a 5-year-old child may be given. The child should be propped up and sent to hospital. Venesection is rarely required.

Prognosis and Later Treatment

The prognosis for haemorrhagic nephritis is now very good indeed. Less than 1% of cases die in the acute stages, and probably 95% recover rapidly and completely. The small residue continue to have haematuria and proteinuria which persists for months or years. There is no specific treatment for this, except perhaps to treat intercurrent infections more promptly than usual. A considerable proportion of such cases eventually become asymptomatic but a few progress to chronic renal failure, usually after many years. Recovery is often more rapid in children in whom hypertension is initially marked. The 15% of children in whom the A.S.O. is low, that is to say in whom the acute nephritis is apparently not due to streptococcal infection, do not have so favourable a prognosis.

Anaphylactoid Nephritis

Anaphylactoid purpura (formerly called Henoch-Schönlein purpura) may be complicated by polyarthritides, colic, melaena, and acute nephritis. The nephritis usually begins some days or weeks after the onset of purpura and is first detected as proteinuria, followed by haematuria with casts in the urine.

This form of acute nephritis has a less good prognosis and is more likely to go on to subacute nephritis with a nephrotic phase and finally chronic renal failure, often with hypertension. The presence of a purpuric rash, fresh or fading, on the extensor surfaces of the limb joints makes diagnosis easy.

The Nephrotic Syndrome

The nephrotic syndrome is a clinical entity in which gross proteinuria is always present, oedema is usually present, and hypertension and/or haematuria is occasionally present. The serum levels of total protein, albumin, and gamma-globulin are low and the cholesterol level very high. Many different conditions may produce this syndrome but fortunately most of these are very rare. One such is a congenital familial type of nephrosis, obvious from the early weeks of life, unresponsive to steroid therapy, rare, and lethal. Secondary nephrosis may complicate heavy-metal poisoning, collagen diseases, malaria, or renal vein thrombosis. For all practical purposes the practitioner will need to deal with children suffering from the common so-called idiopathic or primary nephrosis.

Idiopathic Nephrosis

This illness occurs most frequently in the pre-school child; the incidence then rapidly diminishes until by puberty it is a rare condition. The total incidence is less than one-fifth of acute nephritis. Nephrosis presents as insidious periorbital oedema, followed by ankle and lumbo-sacral oedema and by ascites. The affected child becomes wasted and thin, though this wasting is masked by the rounded contours of oedema, which moves in response to gravitational changes of posture, affecting dependent parts. Such children are very vulnerable to bacterial infection, especially those due to the pneumococcus. Before effective steroid and antibiotic therapy became available about 20% of affected children died of infection within a year of the onset, and 20% of renal failure in the next three years.

The diagnosis is usually easily made from the clinical signs and the urinary findings of oliguria and gross proteinuria. Difficulty occasionally arises in differentiating the rare cases

with haematuria from acute nephritis. Renal biopsy is usually unnecessary as a diagnostic procedure in a child.

The child with nephrosis of recent onset should be treated in hospital, because basic treatment usually includes giving high doses of steroids, a therapy which has many possible hazards. Before admission he should be given a diet containing very little salt.

Treatment

Diet.—The child is given a low sodium diet (1–2 g. daily), high in protein content, provided that the level of blood urea is satisfactory (<80 mg./100 ml.). This is continued until the patient is free from symptoms, or until uraemia ensues. During steroid therapy every effort should be made to keep the calorie intake of the child down to what he was receiving beforehand, so as to prevent the gross increase in weight which commonly complicates this form of treatment. Nor must he be allowed to over-eat on leaving hospital. If he does he may develop gross obesity, leaving permanent “striae gravidarum.”

Steroid Therapy.—Many differing methods of treatment are available, but most consist of a period of two to four weeks on high dosage—for example 40–80 mg. of prednisolone—followed by a gradual reduction over a period of weeks. A further period of weeks or months of low dosage or intermittent steroid therapy follows, and during this period the child is generally allowed home.

The initial results of steroid therapy are excellent in the uncomplicated case in childhood—that is, if the patient has no haematuria. Diuresis begins one week after the start of treatment and is usually complete by the twentieth day. Protein-

FIG. 2.—Diuresis provoked by prednisolone. Note the dramatic diuresis with loss of gravitational oedema and ascites which occurred in a period of six days on steroid therapy.

uria decreases synchronously and when the clinical response is good may vanish altogether. Oedema and ascites disappear, and the serum proteins, cholesterol, and the E.S.R. return to normal levels. Vulnerability to infection ceases. The underlying wasting becomes obvious as the soft contours of oedema shrink away (see Fig. 2).

The side-effects of steroid therapy are Cushingoid obesity (centripetal fat), cutaneous striae, hirsutism, and “tomato” or “moon” face. More serious toxic effects are hypertensive crises, permanent diabetes mellitus, osteoporosis, vertebral collapse, convulsions, and psychological upsets. Such complications usually occur only on intensive treatment, and the child should not receive more than 20 mg. prednisolone daily at home, and 10 mg. daily is more usual.

In about one-third of the children no relapse occurs and they remain well thereafter. The remainder relapse once, twice, or oftener. Such relapses of proteinuria and oedema often follow an upper respiratory infection and may be tran-

sient and self-limited or may persist. If relapse persists for more than three weeks further intensive treatment in hospital is required. It is probably not wise for the family doctor to tinker about with small doses or increments of steroid therapy.

Diuretic Therapy.—If haematuria, hypertension, and a high level of blood urea are present steroid therapy is unlikely to be effective and diuretics are employed. A very high dosage has to be given at first. The patient will be treated in hospital on a low sodium diet (1–2 g. daily) and started on 1–4 g. chlorothiazide (or 100–400 mg. of hydrochlorothiazide) daily, together with 1–4 g. of potassium chloride orally to lessen the risk of hypokalaemia. Such therapy often produces incomplete diuresis, and 25–100 mg. of spironolactone-A are then added. Complete diuresis usually results. The dose of the diuretics is progressively reduced to the lowest level compatible with virtual absence of oedema (continuing supplementary potassium chloride), and the patient may then be sent home. A suitable dosage might be 1 g. of KCl, or 1 g. of chlorothiazide, and 25 mg. of spironolactone-A daily. Many other diuretics are available but those described are reliable and effective.

Antibiotic Therapy.—The vulnerability of the child with oedematous nephritis to infection, and in particular to pneumococcal infection, demands prompt antibiotic therapy for any intercurrent infection which arises. Penicillin, tetracycline, and cloxacillin may be used in full dosage (250 mg. four times daily). Continuous prophylactic therapy is not necessary but should be employed when repeated relapses occur as a result of intercurrent infections.

Prognosis

It is now possible to relieve the oedema completely in almost all children with the nephrotic syndrome. In those without haematuria the proteinuria—and consequently all other signs and symptoms—can be eliminated in the great majority, but unfortunately in two-thirds of these children relapses occur. One of the problems facing us to-day is how to stop this happening. Death from infection is now a rarity. About one-third of children have persisting proteinuria with relapses over a period of many years, and we do not yet know how long it will be before chronic renal failure ensues. The minority who have haematuria from the outset fare badly, and many die of renal failure within three years of onset.

Chronic Renal Failure

Chronic renal failure, the so-called “chronic nephritis,” characterized by polyuria, slight proteinuria, and the presence of casts in the urine has many causes in children. It is not usually a sequel to acute nephritis in children and for this reason the term chronic renal failure should be discarded. A summary of causes might be as follows:

Common Causes :	1. Developmental anomaly of renal tract.
	2. Recurrent pyelonephritis.
Uncommon Causes :	1. Sequel to the nephrotic syndrome, anaphylactoid nephritis, or acute nephritis.
	2. Miscellaneous rare causes—for example, lead poisoning, cystinosis, primary oxaluria.

Signs and Symptoms

The syndrome may be summarized as follows:

General Symptoms : These are anaemia, pallor, lassitude, and liability to infection.

Renal Dwarfism : Failure to thrive results in loss of weight gain and progressive dwarfism, hence the term “renal dwarf” (Fig. 3).

Renal Rickets : The failure of the kidney to clear phosphate normally may result in a severe degree of osteoporosis and rickets (renal rickets or renal osteodystrophy).

Thirst and Polyuria : This complaint is often accompanied by one of recurring enuresis, which may be the presenting feature. The kidney is unable to concentrate urea in the urine and consequently a larger volume of urine must be passed to wash out the daily quota of urea. What goes out must come in

FIG. 3.—(Right.) A child with renal dwarfism and rickets due to chronic renal failure. She is the same age as the child on the left who is of normal average height and weight for her age.

—hence the pathological thirst. These children are in fact drinking to live. The specific gravity of the urine is fixed.

Hypertension and Hypertensive Retinopathy : Hypertension is not always present but sometimes appears in the late stages and may cause typical retinal changes.

Treatment

In dealing with this problem one must temper enthusiasm with humanity. All these children will die, and they should be allowed to live comfortably and happily at home as much as possible, rather than under ultra-scientific (but eventually ineffectual) care in a hospital.

Fluids : The intake should be as large as possible. The fluids should be varied, milk diluted, and excess of fruit juices avoided. Lemonade is popular in a variety of hues.

Diet : The protein intake should be reduced to about 20 g. daily. Milk should be sparingly given and carbohydrate intake encouraged. Sweets, jam, and biscuits are usually popular. This reduced protein intake helps to slow the rise in the level of blood urea.

Alkalis : The use of alkalis helps to ameliorate the progressive metabolic acidosis associated with uraemia. Sodium bicarbonate and/or sodium citrate in dosage of 120–240 gr.

daily is useful. Eventually tetany occurs, owing to the low plasma-calcium level present as a result of phosphate retention, and then alkali treatment must be stopped.

Calcium Gluconate: The administration of 10 ml. of 10% solution intravenously will effectively, but temporarily, stop painful carpopedal spasm.

Blood Transfusion: Progressive intractable anaemia is the rule and repeated transfusions make the child feel better. If transfused in the morning the child usually need not be admitted to hospital overnight—a privilege usually welcomed.

Vitamin D: Sometimes a dosage of 20,000 i.u. vitamin D,

daily may heal renal rickets but the plasma-calcium level must be monitored lest hypercalcaemia arise.

Surgery: This seldom helps except in rare cases of obstruction of the urinary tract. In such cases if chronic renal failure is present surgery has been delayed too long.

Dialysis: In terminal chronic renal failure neither peritoneal dialysis nor haemodialysis is indicated; they simply prolong dying.

Sedation: In the terminal phase the child, bleeding, drowsy, and uraemic, should be deeply sedated and the use of morphia need not be avoided.

ANY QUESTIONS?

We publish below a selection of questions and answers of general interest.

Diabetes from Emotional Shock

Q.—Is there any evidence that an acute emotional shock can result in diabetes mellitus? There is no previous history of this condition in the patient or any members of the family.

A.—This is a controversial question. What might be termed the "party line" states that emotional stress plays little if any part in the aetiology of diabetes. This view is held by Lawrence in this country and by the inheritors of Joslin's teaching in the United States, and they point for support to the rarity of diabetes among the fighting services in the two world wars. On the other hand there have been attempts,^{1,2} largely by psychiatrists, to invoke emotional factors in the onset of the disease, and there are some remarkable individual case records.³

The aggravation of existing diabetes by emotional stress is well recognized,⁴ and when the disease presents for the first time after such a stress it is fashionable to invoke an inherent weakness of the pancreas. Nevertheless every now and then examples occur, like the one quoted in the question, and it is difficult to deny completely a causative role to the emotions. Perhaps there is a parallel to the experimental production of hyperglycaemia and glycosuria by hypothalamic stimulation?

REFERENCES

- 1 Menninger, W. C., *J. nerv. ment. Dis.*, 1935, 81, 1.
- 2 Slawson, P. F., Flynn, W. R., and Kollar, E. J., *J. Amer. med. Ass.*, 1963, 185, 166.
- 3 Phillips, W., *Lancet*, 1957, 1, 281.
- 4 Walker, J. B., *Practitioner*, 1957, 178, 590.

Paroxysmal Sneezing

Q.—What could be the cause of paroxysms of sneezing unconnected with coryza and not apparently of allergic origin? Is there any treatment?

A.—Paroxysms of sneezing are nearly always due to sensitivity of the nasal mucous membrane, and this sensitivity is usually due to something inhaled. However, some sufferers experience paroxysms of sneezing with change of temperature or with exposure to bright lights, like looking into the sunlight, so clearly things other than irritant inhalants (allergic) can provoke a paroxysm of sneezing.

The best thing in such cases is to use a simple sedative nasal spray containing a weak solution of ephedrine.

Corticosteroids and Oral Contraceptives

Q.—Are oral contraceptives contraindicated in patients who are being given A.C.T.H. or corticosteroids daily for asthma?

A.—The use of corticosteroids or of A.C.T.H. for the treatment of severe asthma is not a contraindication to the simultaneous use of oral contraceptives. In some cases it is found that oral contraceptives (or oestrogens) decrease the dose of corticosteroid or A.C.T.H. required to produce the pharmacological effect. This is obviously advantageous, but it does mean that if the dose of corticosteroid is not reduced Cushingoid side-effects might appear.

Allergy to Rum

Q.—A man develops a generalized rash three to four days after drinking a tot of rum, but not after other alcoholic drinks. Can rum cause an allergic response?

A.—Allergic reactions can be caused by wines, spirits, beer, etc. Sensitivity probably develops most commonly to yeast or fungi used in their preparation, but allergy may also develop to sugar-cane, grain, grapes, etc., used in the preparation of individual drinks.

Ventilation of Fall-out Shelter

Q.—How long could 1,300 people remain in a basement fall-out shelter of 85,000 cubic feet (2,406 cu.m.) capacity? How many people could remain in for 48 hours? How much oxygen per hour would be required to replace that used (CO₂ would be extracted) for an indefinite period?

A.—Answers to this question must depend on the activity of the persons during their stay in the shelter. Oxygen requirements have been assessed for persons at complete rest (sitting or lying down) as half a litre of oxygen per minute and for those doing moderate activity (walking about, standing, gossiping, and doing minor chores) as 1 litre a minute. At rest 1,300 people could remain

in the shelter for three hours; if moderately active for 1½ hours. At rest 80 people could remain for 48 hours; if moderately active 40 people could remain for 48 hours.

For an indefinite period, assuming some moderate activity is taking place, a replacement rate of oxygen would be 2 cubic feet (0.056 cu.m.) per person per hour, carbon dioxide being removed. Periodic sampling should be carried out if practicable. It should be remembered, however, that, especially for an indefinite period, O₂ supply and CO₂ removal are not the only requirements. Attention should be paid to control of temperature air movement and humidity, and arrangements made for the removal of any other possible toxic hazards from smoking, cooking, or volatile substances which may accumulate in the shelter.

Examination for Myopia

Q.—At what age should a child who has one parent with fairly severe myopia be first examined for the condition?

A.—Though in rare cases myopia is congenital, more commonly it is first apparent after the age of 4. Unless the child shows evidence of poor vision examination can be delayed until this age.

Flashlight in Babies' Eyes

Q.—Is the intense light of flashlight photography injurious to the eyes of a young infant? If so, from what age is it safe to take such photographs?

A.—I know of no evidence that taking flashlight photographs of a young infant causes damage to the eyes. The amount of light energy reaching the retina is much less than that required to cause a retinal burn.

Tetanus Toxoid for the Injured

Q.—Why should a patient who has had a full immunizing course of tetanus toxoid have to receive a further dose of toxoid when he is injured within the effective period of immunity? Is there not a limit to the number of booster doses of toxoid which are necessary?

A.—Administration of a dose of adsorbed tetanus vaccine is an essential step in the prevention of tetanus in the injured. This has the function of either (a) reinforcing previous immunization or (b) starting a primary course.