epidemic form. It seemed possible at the school where the dropping cases of scarlet fever occurred that we were seeing a phenomenon ("the warning rise") rather similar to that seen in cerebro-spinal fever¹⁷ (and possibly in diphtheria)—that is to say that when the carrier rate (that is, the carrier epidemic) reaches a certain height (namely, 20 to 30 per cent.) clinical cases may occur. The observations were, however, too fragmentary for this to be anything but a surmise.

In this school thorough "spacing out" of beds was carried out, together with improvement of the ventilation of the dormitories, and since then no more cases have occurred, six months having now elapsed.

This infection with haemolytic streptococci was in one or two instances, I think, definitely due to unsatisfactory conditions (overcrowding and poor ventilation) in sleeping quarters.

In connexion with tonsillitis the question of milk must be raised. I have only dealt with three samples of public school milk, and all were definitely dirty and bacteriologically bad. Other evidence seemed to show that the milk was not responsible for the tonsillitis, but, apart from this, I should not feel happy as a school medical officer unless either pasteurized or Grade A milk were in use. Pasteurization is best done on delivery at the school premises, as at Dartmouth.

I trust that medical officers of schools will forgive me if I suggest that there are great opportunities for interesting epidemiological study still unexplored in public schools. I suggest as examples:

(a) The epidemiology and bacteriology of otitis media in relation to scarlet fever, measles, influenza, and febricula.

(b) Relative sickness rates of boys' and girls' schools, and of day and boarding schools.

(c) Relative incidence of droplet infections upon children whose tonsils have been enucleated and whose adenoids have been removed, compared with children who have not been operated upon.

The adoption or recommendation of some uniform system of medical records by the Medical Officers of Schools Association would be of great service. I hope that association will renew its activities, which have been so helpful to me in preparing this paper, and keep before its members their paramount duty of devoting as much time and thought to the preservation of health in schools as they do to the care and treatment of the sick pupils. I urge, too, that the modern boy or girl is an intelligent being, and should be treated as such, and encouraged to take an interest and pride in keeping fit. This can be attained without fussing or coddling; public opinion nowhere has greater influence than in schools, and it should be formed to regard the training of the body against infection as being as manly an aim as its training to achieve athletic success or to endure fatigue.

SUMMARY.

In the following summary of my points I advance them not as "conclusions," or as proved by my observations, but merely for convenience.

1. "Droplet" infections are responsible for at least 80 per cent. of all sickness in our public schools. There is a well-marked "danger period"—that is, the first two months of the Lent term.

2. It is probable that no immunity to influenza or febricula is conferred by previous attack, and evidence of the protective value of vaccines is inconclusive. There is, however, slight evidence that some protection may be afforded against complications, particularly pneumonia; the subject deserves more systematic study. Vaccines if given should be administered well before the beginning of the danger period, probably best in November.

3. Intensive prophylaxis during the first half of the Lent term would probably amply repay the trouble. It should include:

(a) Special efforts to prevent boys returning to school after the Christmas holidays infected with influenza or febricula.

(b) Temperature-taking for three weeks.

(c) Immediate isolation of all pyrexias and catarrhs.

(d) No work before breakfast for at least the first six weeks of the term.

(e) All hot baths and showers taken during the day or after games to be followed by cold showers.

(f) Prevention of chill in watching matches, sports, etc.

(g) Increased provision for drying clothes, uniforms, and boots.

4. Infection mainly takes place in sleeping quarters, and proper spacing-out of beds and thorough "cross" ventilation in dormitories is of paramount importance. No school authority has done its duty to its pupils unless it has provided dormitory accommodation sufficient to allow of at least 3 ft. clear space between the edges of beds and thorough and through ventilation. Until these essential wants are met the provision of properly sited, amply spaced, and "cross"-ventilated dormitories should take precedence of all building requirements.

5. Milk should be pasteurized.

[Dr. Glover exhibited a large number of diagrams; of these it has been possible to reproduce one only.]

REFERENCES.

- ¹ Special Report Series, 75 and 111. ² Medical Research Council, Special Report Series, No. 111, p. 9. ³ *British Medical Journal*, 1922, i, 134; and *Journ. Roy. San. Inst.*, January, 1927, 480. ⁴ *Journ. Roy. San. Inst.*, January 1927, 485. ⁵ Public Health Reports, Washington, 43, 11, March 16th, 1928. ⁶ Ministry of Health Report No. 4, Influenza, 411. ⁷ Parsons, H. Franklin: Second Report on Influenza, L.G.B., 1893, 75. ⁸ Ministry of Health Report No. 4, Influenza, 400. ⁹ *British Medical Journal*, 1922, i, 134. ¹⁰ Op. cit., 135. ¹¹ *British Medical Journal*, April 2nd and October 29th, 1927. ¹² Ibid., October 15th, 1927, 700. ¹³ Public Health Reports, Washington, March, 1928. ¹⁴ *Journ. Roy. San. Inst.*, January, 1927, 485. ¹⁵ "Cd. 7535," Stationery Office. ¹⁶ *Health at School*, Rivingtons, 1905, p. 140. ¹⁷ Glover: Medical Research Council, Special Report No. 50, p. 140.

TREATMENT OF BURNS AND SCALDS BY TANNIC ACID.*

BY

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BURNS and scalds are accidents of great importance in domestic and industrial life. They are often fatal, or inflict serious incapacity and deformity on those who survive. These serious consequences, together with the comparative frequency of the accidents, constitute a grave problem for the medical profession. The object of this paper is to give a very brief account of the more important studies which have been made in the subject, to review our present knowledge of it, and to inquire into the results of treatment. More especially the recently introduced tannic acid method of treatment is discussed and described, and the results in a series of personally observed cases are presented. For ease of description thermal injuries, either by scalding or by actual burning, will be included under the term "burns."

CLINICAL COURSE AND PATHOLOGY.

A consideration of the clinical course and pathology of the condition is essential to a discussion of its treatment. It is customary to divide the clinical course into three stages, thus: (1) shock, (2) inflammation, (3) healing.

This classification is inadequate and misleading, and we prefer a division into four stages in the following way:

I. Shock ("initial or primary shock").

II. Acute toxæmia ("secondary or toxic shock").

III. Septic toxæmia.

IV. Healing.

Before proceeding to a consideration of these stages we shall refer briefly to the nature and causation of "shock." For a more complete account of the condition reference should be made to the numerous and exhaustive papers published by the Medical Research Committee.¹² The question has been reviewed by Cannon⁴ in relation to experimental traumatic shock, and the conclusion seems warranted that shock resulting from severe trauma, such as war wounds, is of two kinds—namely, "initial or primary shock" and "late or secondary shock," and these

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two conditions differ in causation, severity, and consequences. "Initial shock" is of nervous origin, is similar in nature to fainting or syncope, but longer in duration, and is due to a primary disturbance of the nervous vasomotor mechanism. "Late shock," on the other hand, is due to the action of a toxic agent absorbed into the circulation from the site of injury. This toxin apparently is a product of protein disintegration at the site of injury. In a later section we shall show that the acute toxæmia ("secondary or toxic shock") which follows injury by burning is of similar origin. The term "shock" will be reserved for the condition of "initial shock." It appears more appropriate to designate the "late or toxic shock" as acute toxæmia, for it is essentially comparable in origin, pathology, and consequences with other acute toxic conditions.

STAGE I.—*Stage of Shock ("Initial or Primary Shock").*

The main clinical features are prostration, subnormal temperature, pallor, low blood pressure with a small rapid pulse, and cold skin. It arises immediately after the injury, and in the majority of cases is transient and slight in degree, and is rarely of serious significance. During the period 1913 to 1925 at the Royal Hospital for Sick Children, Edinburgh, 80 cases of burns terminated fatally. Of these, only 2 (2½ per cent.) died during the stage of shock. In the case of burns the source of shock is probably widespread damage to the numerous nerve-endings in the skin.

STAGE II.—*Stage of Acute Toxæmia ("Secondary or Toxic Shock").*

There is no doubt that this toxæmic phase is the real danger period in the clinical course, and is largely responsible for the high mortality of burns. It commences between six and twenty-four hours after injury, and in a large majority of fatal cases terminates in death within forty-eight hours from the time of injury. Of the 80 fatal cases previously mentioned 64 (80 per cent.) died in the stage of acute toxæmia, and of these 50 (78 per cent.) died within forty-eight hours from the time of infliction of the burn. Between the stages of shock and acute toxæmia there is usually an interval during which the patient looks well, even in cases of extensive lesions.

With the onset of toxæmia the temperature rises, often abruptly, and, more especially in children, it may attain remarkable heights—106° F. is by no means an uncommon level, and it may reach 110° F. shortly before death. The patient becomes restless, the expression is anxious, the pupils dilated, and the colour dusky or livid. Vomiting is a common feature of this as of other toxic states, and the vomitus is frequently "coffee-ground" in character as a result of hæmorrhagic gastritis. The blood pressure is low, the pulse rapid and small, and the respiration rate increased. Death is usually preceded for a few hours by coma; convulsions, in our experience, are rare.

It is obvious that since acute toxæmia is the most serious danger to life in injuries by burning a knowledge of its origin is of great significance.

Since Cumin⁵ of Glasgow, in 1823, published reports of post-mortem examinations on a series of five fatal cases many theories have been propounded to account for the fatal issue. Such theories are now of historical interest only; the original references can be obtained from Bardeen's³ admirable article.

Avdakoff,² in 1876, was the first to suggest that a specific toxin entered the circulation after the injury. Since then evidence of a most conclusive nature has been forthcoming in support of his contention. We may cite as especially significant the work of Ajello and Parascandolo,¹ who first practised experimentally a modification of "exsanguination-transfusion"; that of Kijanitzin;⁶ the parabiosis and other experiments of Vogt;¹⁶ the histo-pathological studies of Bardeen,³ McCrae,¹¹ and Weiskotten;¹⁷ and the recent researches of Robertson and Boyd.¹⁵ These researches proved conclusively that a toxin appears in the circulating blood within a few hours of the infliction of the burn. The toxin is formed at the burned area, is absorbed into the

blood stream, and gives rise to the symptoms of acute toxæmia. The exact nature of the toxin has not yet been determined, but it is probably a product of protein disintegration occurring under the action of excessive heat,¹³ and it is said to consist chemically of primary and secondary proteoses.¹⁵

Stages III and IV need not be discussed in this paper. In the collection of 80 fatal cases 12 (15 per cent.) died during the third stage.

TREATMENT.

The principles of treatment as indicated by the pathological condition in each stage will now be considered.

Stage I.—In shock the main lines of treatment are to avoid undue exposure to cold and restore normal body temperature, to administer fluids, and to relieve pain. Pain is frequently severe, and for this drugs of the morphine group should be given to adults. The depressant effects of morphine or heroin in young children, however, must be borne in mind. Treatment of the injury by local measures is begun at the earliest possible opportunity, which is frequently during the first stage, and involves cleansing and dressing of the affected area. Experience has convinced us of the value of a light general anaesthetic when the areas are being cleansed.

Stage II.—In this stage the primary indication of treatment is to prevent the absorption into the circulation of the toxin which is produced in the burned area. No antitoxin is available. To Davidson⁴ belongs the credit of showing that absorption of the toxin may best be prevented by inducing coagulation of the damaged tissue, thus rendering the toxin non-soluble. For this purpose he employed a local application of tannic acid and found that it minimized acute toxæmia or prevented it entirely.

Stage III.—The treatment is that of a septic wound and need not be elaborated here. After primary tannic acid treatment, however, the management of this stage is considerably modified in these circumstances, since the application of wet dressings may be a source of danger.

Stage IV.—The treatment in this stage is that of a healing sore.

PERSONAL OBSERVATIONS.

Our series consists of 42 cases in children and 8 in adults, in which the principles of treatment outlined above were adhered to.

Details of Management.

On admission the patient is put to bed at once. From the commencement of treatment the burned area is exposed to the air. No dressings are applied and the bedclothes are supported on a suitable "cage," inside which electric lamp bulbs are fitted. If shock is present, the foot of the bed is raised and artificial heat administered by means of the electric bulbs. Subcutaneous saline infusion is given if shock is severe. Morphine or heroin is prescribed for the relief of intense pain.

Cleansing.—Having secured these arrangements, cleansing of the burned area is carried out immediately. Under a light gas and oxygen or ether anaesthesia any blisters are opened and evacuated and all epithelium which is loose or raised by blistering must be completely removed, in order that the tannic acid may come into direct contact with the damaged tissue beneath. It is in this tissue that toxin formation and absorption occurs, and of it coagulation must be secured. The area is rapidly cleansed with ether, which exerts a drying and purifying effect and is specially useful if oily or greasy substances have been previously applied, for grease prevents effective contact of the watery solution of tannic acid.

Application of Tannic Acid.—An aqueous solution of tannic acid (2.5 per cent. in warm sterile water) is now applied. It must be freshly prepared for each application, since tannic acid in solution is rapidly changed to gallic acid, which has no power to precipitate or coagulate protein. The warm solution is sprayed on the affected areas from an ordinary nasal spray atomizer, and the parts are then dried by means of the electric lamp bulbs in the bed-cage. Spraying and drying are carried out at frequent intervals until all the burned area is covered by a thin

layer of brown coagulated tissue. In hospital practice we have found it most convenient to carry out the spraying at intervals of one hour, but, if practicable, as, for example, where a special nurse or attendant is available, the length of the interval may be considerably reduced. Coagulation may occur more rapidly in some areas than in others. So soon, therefore, as the desired result is observed in any part, that part should be protected by gauze during subsequent spraying. Application of tannic acid is stopped when a thin coagulum has formed, in order to avoid a too deep corrosive action of the acid. The risk of such an action is extremely slight when a weak solution is used, and can be entirely obviated by frequent observations of the area. The periphery of the burned area affected by a first degree lesion frequently shows a light chocolate brown after about eight applications. In the case of second and third degree areas a satisfactory coagulum is usually obtained after eight to twelve applications. Areas more deeply involved may require several further treatments.

Special Circumstances.—Special care is necessary in treating burns of the face. The eyes, the nostrils, and the external auditory meatus must be protected during spraying by means of pledgets of moist wool, and in this region it is convenient to dispense with artificial drying. In any case if the temperature be above 101°F ., drying the parts by artificial heat is both unnecessary and inadvisable. In some cases it is not practicable to use the spray method of application to the whole of the burned area, as, for example, when both front and back of the trunk are involved. We employ the method of the spray and free exposure for the more extensively affected area and the method of tannic compresses to the less extensively injured surface which cannot be simultaneously exposed. The details of the latter method are as follows.

Sterile gauze swabs very lightly wrung out of the warm solution of tannic acid are applied to the burned area and covered with a light layer of sterile wool and a bandage. The part is examined at the end of eight, twelve, sixteen, and twenty hours from the time of the first application, and when a light brown coagulum has formed the dressings are removed. To avoid the risk of pulling off the coagulum when removing the dressings, they should first be moistened with fresh solution. Coagulation may not be uniform, and it will be necessary in such a case to apply fresh compresses to areas which are not sufficiently tanned, such areas being examined subsequently at intervals of four hours. When compresses are used the formation of coagulum is usually satisfactory in from twelve to twenty hours. The parts are then protected by a thin layer of dry gauze covered by cotton-wool. We have also used this method of compresses in cases when the lesions were not extensive and the patients were not admitted to hospital.

Local Effects.—The immediate local effects of tannic acid treatment may now be enumerated. At an early stage of the process of coagulation, frequently after the first application, pain is relieved and rapidly disappears. Application by the spray is painless and, indeed, can sometimes be carried out without rousing a sleeping patient. The entire affected area becomes covered by a thin brown, or brownish-black, leathery coagulum, which when dry is perfectly insensitive and can be handled freely without fear of damage to it or of conveying infection to the underlying tissue. By virtue of its tough rigid character the coagulum acts as a splint and ensures the best conditions for the healing processes which are in progress beneath it.

Care of Burned Area.—From the time when the coagulum has completely formed until it is removed no special local treatment is necessary. The bedclothes are supported and the areas exposed directly to the air. Several important points in the care of the area, however, require mention. As is well known, burns are, as a rule, more extensive than appears at first sight to be the case, and blisters frequently develop in areas which initially presented the characters of a first degree lesion. These are dealt with in the usual way and the raw areas sprayed with fresh solution. Occasionally fluid accumulates under the coagulum, and such a collection should be at once evacuated through an aperture cut for the purpose with scissors. Very little care is necessary in adults to protect the coagulum from trauma. As has been mentioned, parts subjected to pressure are covered with dry gauze, and in children recumbency is secured by tying down the limbs. If the perineum is

involved the bowels are regulated by rectal lavage, and nursing is facilitated if the patient lies in the prone position with a pillow supporting the pelvis. Soiling of the areas by urine may give rise to release of toxin and sepsis.

Treatment of sepsis in the third stage is the most difficult problem in the tannic acid method. Signs of a mild degree of infection, as evidenced by a discharge of a little sero-purulent fluid from the edges and some reddening of the surrounding skin, are occasionally seen at different parts of the area. In this case no active treatment is necessary, and the parts may with safety be left alone. Gross infection is fortunately a very rare occurrence, and is usually associated with the formation of sloughs in more deeply burned areas. In such a case a collection of pus may float up the coagulum, which can then be easily stripped off, or, should the coagulum continue to adhere firmly, it should be softened by vaseline and is then readily removed.

In no circumstances whatsoever should wet dressings be applied over the coagulum. We have observed on several occasions that, while the coagulum is present, and particularly during the first week, the presence of moisture, whether in an unopened blister or in dressings applied over the area, is associated with some signs and symptoms of acute toxæmia. Apparently in these circumstances the toxin, previously imprisoned in the coagulum, becomes released and gains access to the blood stream. Only when all coagulum has been removed may wet dressings be employed in the treatment of sepsis. On this point we have confirmed the observations of Davidson.

In the uncomplicated case the process of healing advances under cover of the protective coagulum layer. In superficial lesions, such as those of second and third degree, epithelium covers the raw surface completely in from eight to fourteen days. In deeper lesions the coagulated layer remains firmly attached to the subcutaneous tissue until loosened by the growth of granulations, usually between the eighth and sixteenth days. The coagulum should be left until it can be easily peeled off. Then an ointment dressing should be applied to any areas which are still uncovered by epithelium. The most satisfactory is sterile vaseline prepared in the following way. Squares of gauze or lint are laid out flat in a tin box along with a quantity of vaseline sufficient to cause a generous impregnation of the material, and the whole sterilized in the autoclave. The squares can be removed one by one as required and applied directly to the area.

Lastly, the tannic acid method can be commenced as late as seventy-two hours from the time of injury, whether or not other methods of local treatment have been previously used. After this period the toxæmic danger has largely passed, and in the untreated case frank sepsis will be manifest. The lesion should then be treated as an infected wound.

GENERAL TREATMENT.

Abundant fluids are given. Oral administration of fluids is, as a rule, sufficient, since toxæmia is mild or absent. In the severest cases, where a considerable quantity of toxin enters the blood stream before coagulation of the injured tissues has been effected, the procedure of "exsanguination-transfusion," as recommended by Robertson,¹⁴ is a valuable accessory method of dealing with the toxic state. A quantity of toxic blood is removed from the patient by bleeding, and is replaced by an equal, or slightly greater, quantity of fresh blood from a suitable donor. We found that removal of 120 to 150 c.cm. in an infant, and of 250 to 300 c.cm. in a child of 3 years, was sufficient exsanguination. In an infant exsanguination and transfusion can easily be carried out by puncture of the superior longitudinal sinus through the fontanelle, preferably with a special needle, described by Dott.⁷

In young children, and especially in infants under the age of 1 year, it is of the utmost importance to estimate the temperature frequently. If toxæmia is severe the temperature may rise rapidly to 106°F . or higher, and, as a consequence of this high temperature, death will shortly ensue unless active measures are taken to reduce it. Personal clothing and, if necessary, the bedclothes, should be lightened or removed, and tepid sponging may be required. Artificial cooling is a life-saving measure in

these cases. In our experience of the tannic acid treatment of burns hyperpyrexia has been almost unknown, largely because acute toxæmia is usually mild or absent, but also in part because the procedure of supporting the bedclothes on a cage and exposing the parts to the air assists natural regulation of the body temperature.

RESULTS OF THE INVESTIGATIONS.

Our personal experience of the influence of the tannic acid treatment on the clinical course, the mortality, and prognosis in these cases will now be summarized.

Clinical Course.

Stage I.—It was almost invariably noted that application of tannic acid was followed rapidly by the relief of pain. From the first, therefore, this form of treatment promotes analgesia, and is associated with no discomfort whatsoever.

Stage II.—The action of tannic acid in preventing or minimizing the absorption of toxic products from the burned area has been amply proved in this series. Of 50 cases only one died in the stage of acute toxæmia, and this unfortunate result was probably due as much to severe prolonged shock as to toxæmia *per se*. The incidence of acute toxæmia in cases treated by tannic acid before the onset of this stage may be shown in tabular form.

Treated before Onset of Stage II—40 Cases.

	No. of Cases.	Average Surface Involvement.
No toxæmia	20	11 per cent.
Mild toxæmia	16	12 " "
Moderately severe toxæmia... ..	1	27 " "
Dangerous toxæmia	2	40 " "
Fatal toxæmia	1	27 " "

In the patient suffering from moderately severe toxæmia the burns were very extensive and coagulation proved slow. The general condition, however, gave rise to no anxiety. Dangerous toxæmia was the result of special circumstances. In one case the lesions involved 60 per cent. of the total surface of the body. The other was a case, probably unsuited to the tannic acid method of treatment, in which the tissues of one limb had been broiled by slow heat while the patient was unconscious from carbon dioxide poisoning. The case of fatal toxæmia has already been mentioned. In 8 cases treatment was commenced during the toxic phase, and in all the symptoms and signs disappeared rapidly when coagulation of the injured tissues was effected. When it is recalled that 80 per cent. of deaths from burns occurred in this stage with previous methods of treatment the significance and importance of these results will be appreciated.

Stage III.—The incidence of sepsis in cases treated by tannic acid is shown in tabulated form.

Patients treated by Tannic Acid before onset of Sepsis—48 Cases.

	No. of Cases.
No sepsis	32
Mild sepsis	12
Moderate degree of sepsis	2
Severe and fatal sepsis	2

The incidence of sepsis increases with the depth of the lesion, the presence of fluid, especially in blisters, which appear at the edges in the first few days, and the number of hours between the time of injury and commencement of treatment. In one or more of these factors was found the explanation of sepsis of mild or moderate degree. Severe sepsis was due to special circumstances, and occurred in the same cases in which severe acute toxæmia was observed. In both death resulted from sepsis.

Stage IV.—The protection afforded to the growing epithelium and the splint-like action of the coagulum, as well as the low incidence of sepsis, have been important factors in minimizing scar tissue formation and preventing contractures. In only one case has skin grafting been necessary.

Mortality and Prognosis.

The total number of deaths in the series was 7, of which only 4 were directly attributable to the injury. The remaining 3 deaths were in children, of whom 2 succumbed to virulent acute enteritis during epidemic periods and 1 to bronchopneumonia following whooping-cough. The death rate was, therefore, 8 per cent. Three children and one adult were

victims. Of the children, one died from severe shock, one from acute toxæmia after prolonged shock, and one from sepsis and exhaustion. Death in the adult occurred in the fifth week from sepsis.

It is obvious that a prognosis based on the extent of the area affected, relative to the total surface of the body, is very considerably influenced by the use of the tannic acid method of treatment. Observations by Fraser⁸ and Klauder¹⁰ indicated that in children the prognosis was bad when 11 or 12 per cent. of the entire surface of the body was involved. In this series of 42 cases in children, 20 suffered from lesions involving 12 per cent. or more of the total body surface, and of these 17 recovered. Moreover, in the great majority the clinical course was marked, not as in the past by severe and prolonged illness, suffering, and distress, but by an appearance of comfort, health, and well-being from the commencement of the treatment.

CONCLUSIONS.

1. Fifty cases of burns have been treated by tannic acid.
 2. It has been shown that this method of treatment promotes rapid analgesia; there is complete absence of pain and discomfort while the coagulum is present, and during this time there is no necessity for dressing.
 3. In many cases tannic acid prevents acute toxæmia and invariably lessens the severity of this phase when it develops. The prognosis as to life, especially in children injured by extensive burns, is thus greatly improved.
 4. Sepsis is usually avoided in superficial lesions treated by the tannic acid method, and when sepsis occurs in deeper lesions it does so late in the course of the case and is of a mild degree.
 5. Scarring is probably less marked than in other forms of local treatment on account of the protection afforded to the growing epithelium, the diminution of sepsis, and, hence, the minimization of granulation tissue which is the precursor of scar.
 6. The death rate from burns in this series was 8 per cent.; the figure compares very favourably with any which have been quoted from the results of treatment by other methods.
 7. A prognosis based on the extent of the injury is considerably influenced by the tannic acid treatment. The outlook in children is not necessarily grave even if 40 per cent. of the entire surface of the body is affected.
 8. In view of the success with which this treatment has been attended we would recommend that its use be extended.
- Tannic acid should form part of the armamentarium of the country practitioner.
- In mines, factories, etc., it should find a place in the first-aid equipment, and its application by the compress method could be included in first-aid training.
- Thus valuable time could be saved during transport of patients to hospital and the maximum benefits be derived from its life-saving properties.
- Tannic acid can be conveniently kept in powder form, made up in packets containing 110 grains. One such packet dissolved in half a pint of water gives a 2.5 per cent. solution.

I wish to thank Mr. N. M. Dott and Miss Gertrude Herzfeld, surgeons to the Royal Hospital for Sick Children, Edinburgh, and Professor D. P. D. Wilkie, University of Edinburgh, for the opportunity afforded for this investigation. To the resident staff of both hospitals during the period of investigation I am deeply indebted for assistance and co-operation.

REFERENCES.

- 1 Ajello and Parascandolo: Abstract in *Centralbl. f. innere Med.*, 1896, 1203.
- 2 Avdakoff: Dissertation, St. Petersburg, 1876 (quoted by Kijanitzin and Bardeen).
- 3 Bardeen: *Johns Hopkins Hosp. Rep.*, 1898, viii, 137; *Journ. Exper. Med.*, 1897, 2, 501.
- 4 Cannon: *Arch. Surg.*, 1922, 4, 1.
- 5 Cumins: *Edin. Med. and Surg. Journ.*, 1823, 19, 337.
- 6 Davidson: *Surg., Gynecol. and Obstet.*, 1925, 41, 202.
- 7 Dott: *Fraser's Surgery of Childhood*, vol. i, ch. 5, p. 66, 1926.
- 8 Fraser: *Surgery of Childhood*, vol. i, ch. 3, London, 1926.
- 9 Kijanitzin: *Arch. f. Path. Anat.*, etc., 1893, 131, 436.
- 10 Klauder: *New York Med. Journ.*, 1913, 98, 234.
- 11 McCrae: *Trans. Assoc. Amer. Phys.*, 1901, 16, 153.
- 12 Medical Research Committee, Special Report Series, 1918-19 (Reports of the Special Investigation Committee on Surgical Shock and Allied Conditions).
- 13 Pfeiffer: *Arch. f. Path. Anat.*, etc., 1905, 180, 367.
- 14 Robertson: *Canad. Med. Ass. Journ.*, 1921, 11, 744; *Arch. Surg.*, 1924, 9, 1.
- 15 Robertson and Boyd: *Amer. Journ. Dis. Child.*, 1923, 25, 163; *Journ. Lab. and Clin. Med.*, 1923, 9, 1.
- 16 Vogt: *Zeit. f. Exper. Path. u. Therap.*, 1912, ii, 191.
- 17 Weiskotten: *Journ. Amer. Med. Assoc.*, 1919, 72, 259.