Papers and Originals

Infants, Toddlers, and Aspirin*

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Aspirin causes more accidental deaths in young children than any other drug, and indeed more than the next four drugs combined. Moreover, in the pre-school child more aspirin deaths are caused by faulty therapeutics than by accident.

It can be admitted that aspirin-poisoning in young children is already well documented. Clinical studies include those of Williams and Panting (1937), Dodd *et al.* (1937), Barnett *et al.* (1942), Troll and Menten (1945), Erganian *et al.* (1947), Lipman *et al.* (1949), Riley and Worley (1956), and Segar and Holliday (1958). Winters (1959) has made a particular study of the metabolic changes, and Tschetter (1963) has written a valuable review of the whole subject. Aspirin deaths continue. What is more, they are increasing.

The unheeded warnings of the past have appeared mainly in specialist journals, and the present mortality justifies a paper available to the profession as a whole and particularly to general practitioners and, through them, to parents and pharmacists.

The present paper attempts to discover why aspirin deaths occur. The clinical manifestations of aspirin-poisoning are considered in detail, but the treatment in hospital is given only in outline.

Material and Method

In studying the national mortality the Returns of the Registrars-General for England and Wales, and for Scotland, were used up to the year 1962, the latest year available.

Continuity was maintained and the clinical element introduced by a study of cases admitted with aspirin-poisoning to the Royal Hospital for Sick Children, Glasgow, from January 1963 to April 1965, and to the children's unit at Stobhill Hospital, Glasgow, in 1963 and 1964. The total was 105 cases. Twenty-six cases were excluded because the salicylate level had not been estimated (21 cases) or was less than 5 mg./100 ml. (5 cases). This left 79 cases, of whom 67 took aspirin accidentally and 12 were given it therapeutically.

The matter seemed urgent, indicated by the number of deaths at the above hospitals, so it was decided to make this a retrospective study.

Blood salicylate was measured by the method of Smith and Talbot (1950), which seems to give readings rather lower than that of Trinder (1954), used by Done (1960) and others.

Latterly it became customary to measure the plasma standard bicarbonate on the Astrup apparatus, but the method

of Van Slyke and Neill (1924) for measuring plasma CO₂ content was used throughout the series, and it is these figures which are quoted. Though both the hospitals involved used the same method, it was noted that the Stobhill figure was never below 10 and not infrequently above 20, differing from the figures from the R.H.S.C. in both respects. The findings of the two hospitals are therefore shown separately in Table III.

Incidence of Mortality

Table I, derived from the returns of the Registrar-General, is self-explanatory. In recent years aspirin has caused more deaths than the next four poisons combined.

TABLE I.—Average Number of Accidental Deaths a Year at Different Periods from Certain Poisons, Great Britain, Children 0-4 Years

	1950-4	1955-9	1960-2
Salicylates	5.0	4.2	11.4
Barbiturates	1.4	1.2	1.7
Iron	7.6	1.2	2.3
Antihistamines	4.2	1.2	0.3
Strychnine	4.6	3.4	1.7
Tranquillizers	0.0	0.2	3.0

Those who are accustomed to deal mainly with adults, or who have read reviews with an adult slant (Greenberg, 1950), may find these figures surprising, for the pattern of poisoning in adults differs from that in children. This is well seen in some recent American figures (U.S. Department of Health, Education, and Welfare, 1961), summarized in Table II.

TABLE II.—Accidental Deaths from Salicylates and Barbiturates, U.S.A., 1955-9

Age	Poison	Number	Ratio
0-4 years	Barbiturates	45	1
	Salicylates	428	9.5
Over 4 years, including adults	Barbiturates	1,521	8.6
	Salicylates	177	1

Campbell (1963) comments that there is a peak mortality from aspirin-poisoning in the pre-school years, and from a diagram reproduced on a small scale in his paper this appears to fall at about the age of 3 years. The average age of the 67 patients in the accidental group in the present series was 31 months, and of 11 in the therapeutic group (excluding a child of 12 years) 15 months. The different ages of the two groups can be seen in the Chart, and this is not confined to the present series. Riley and Worley (1956) found a similar distribution, and the age difference intrudes throughout the literature. It is not surprising that children under 1 year of age seldom poison themselves accidentally, but it is surprising that children over 2 years seldom show therapeutic poisoning.

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Campbell's peak age for mortality corresponds with the peak age for accidental poisoning, and is quite different from the peak age for therapeutic poisoning, which strongly suggests that the therapeutic deaths have not been registered as accidental deaths. This suggestion would not stand if there were far fewer therapeutic than accidental deaths, but it will be shown that the reverse is the case.

In the present series 2 of the 67 patients in the accidental group died, but 6 of the 12 in the therapeutic group died. Again this is similar to the findings of Riley and Worley, who had no deaths in 13 accidental cases and 5 deaths in 23 therapeutic cases. The conclusion is that many more children die of therapeutic than of accidental poisoning, and this is almost entirely due to the greater susceptibility to salicylate poisoning of the child under 2 years of age.

Even at this point the danger of aspirin may be underestimated. Children may die undiagnosed, and Arena (1963) believes that some cases of aspirin-poisoning are labelled "virus encephalitis." One need not have missed the diagnosis completely to use such a label, and indeed the cause of death in one case in the present series was certified as "toxic encephalopathy." A mother is not told that she killed her child, albeit unwittingly, if there is any doubt at all.

Accidental Poisoning

Accidental poisoning by salicylate is easier to study than therapeutic poisoning, as it involves only one episode of ingestion and is uncomplicated by disease. Winters (1959) describes three stages of poisoning, and it will be convenient to consider the present series in relation to these stages.

1. There is first an increase in pulmonary ventilation, due to the stimulant effect of salicylate on the respiratory centre. This causes alkalosis, with a rise in the blood pH. In the present series there were eight children, who were admitted within four hours of ingestion and who were already overbreathing. They showed no biochemical evidence of acidosis sufficient to cause this overbreathing. Wallgren, commenting on a communication by Odin (1932), quotes a striking demonstration of the initial alkalosis. He gave salicylate to two infants with spasmophilia in order to cure them by inducing acidosis. "When I tried aspirin treatment for two infants with spasmophilia," he said, "the effect was the direct opposite of what had been expected. In spite of the fact that deep respiration distinctly arose, there occurred an increase in the electrical super-excitation and the tetania became aggravated. The aspirin was at once discontinued. . . ." The initial "deep respiration" is not in fact a sign of acidosis but a toxic effect resulting in transient alkalosis. This effect may persist in the adult or schoolchild, but is present for only an hour or two in the very young child, when the acidosis of the third phase takes over. Apart from the eight mentioned above, all the children showing symptoms on admission were in the stage of acidosis (Winters's third phase). The stimulant effect of the salicylate on the respiratory centre does of course persist while the blood salicylate is raised, and is added to rather than superseded by the hyperventilation of acidosis, so that the

hyperventilation of the severely poisoned child becomes very great. This masked tendency to alkalosis should curb heroic treatment with alkali.

2. There is an increased metabolic rate related to the action of the drug in uncoupling oxidative phosphorylation, with increased CO₂ production and fever. The fever is potentiated by dehydration. It may be difficult to believe that an established "antipyretic" drug shows fever as a toxic effect, but it has been recognized for a long time and was discussed at length by Dodd et al. (1937). In Segar and Holliday's (1958) 43 cases, of which 32 were therapeutic, 27 had temperatures of 103° F. (39.4° C.) or over, and 12 had temperatures of 105° F. (40.6° C.) or over. The incidence of fever was not so high in the present series, but one child had a temperature of 107° F. (41.7° C.) as the result of accidental ingestion. There are many cases in the literature where continuing fever has led to the treatment of aspirin-poisoning with heroic doses of aspirin.

3. Disturbed carbohydrate and lipid metabolism also occurs. Glycogenolysis is speeded and glycogen synthesis reduced. The blood-sugar may be normal or raised. Levels of over 200 mg./100 ml. are quite common. Blood-sugar levels are available in only four accidental poisonings in this series, being normal in three and raised (287 mg.) in one. Low blood-sugars may result from starvation in the therapeutic group, and did so in two of the present series, but are not a common feature of salicylate-poisoning. The idea that salicylate produces a low blood-sugar probably stems from the work of Reid et al. (1957) and Read and Lightbody (1960), who treated diabetes with aspirin, though they point out that whereas aspirin reduces the hyperglycaemia of diabetes it does not produce hypoglycaemia either in the diabetic or in the normal. Hypoglycaemia in reputed salicylate-poisoning has been described by Mortimer and Lepow (1962) and by Cotton and Fahlberg (1964), but their experience seems unusual.

Fatty-acid catabolism is accelerated and ketones are produced in excess. The precise reason for the latter is unknown, but it is very marked in the first year or two of life (Done, 1963), when acidosis is most severe.

The 67 cases of accidental poisoning in the present series can now be considered as a whole, and are summarized in Table III.

TABLE III.—Analysis of Cases of Accidental Poisoning								
Group	Time between Ingestion and Admission	Hospital	Age in Months	Hours after Ingestion	Blood Levels on Admission			No.
					Salicylate (mg./100 ml.)	HCO ₃ ' (mEq/L.)	pH	
I	0-12 hours	54 cases. Average time after ingestion, 3.3 hours						
II	12-24 hours	A	36	15	36	—	—	1
			24	12	48	13	—	2
			27	16	44	6	7.14	3
		B	19	20	67	6	7.27	4
			28	16	43	10	—	5
			26	12	58	11	—	6
III	24 hours or more	A	27	28	52	7	7.29	7
			23	24	52	7	7.23	8
			27	44	32	5	—	9
			15	24	50	14	7.20	10
		B	36	36	44	12	—	11
			24	48	57	10	—	12
			22	36	54	10	—	13

Few of the children in group I were seriously ill. Evacuation of the stomach and attention to fluid balance sufficed in most. On the other hand, group I contained the three highest salicylate levels in the series—78 mg./100 ml. three hours after ingestion, 82 mg. at nine hours, and 95 mg. at five hours. The first was treated by exchange transfusion, the others by peritoneal dialysis, and all recovered.

Four of the six in group II were very ill, and two of the seven in group III died. It will be seen that there is no essential difference between groups II and III as regards age, salicylate level, and biochemistry. The difference lies in the time since ingestion. Done (1960) pointed out that there is no close correlation between observed salicylate level and the symptoms, but he went on to produce a nomogram involving

salicylate level and time from which he deduced an imaginary peak salicylate level (S_0) which did correlate with the severity of the symptoms. It is therefore possible that the salicylate levels in group III had all been higher before admission; but it is by no means certain, as no attention had been paid to salicylate elimination before they came into hospital. It is also doubtful if the levels of those in group III had ever been as high as the three highest in group I, who recovered. The important factor is time since ingestion. This is the first hint of why children die of aspirin-poisoning. They die because admission to hospital is delayed, and it is delayed because of a failure in diagnosis. Neither of the two children who died was sent to hospital with a diagnosis of aspirin-poisoning.

Therapeutic Poisoning

Therapeutic poisoning differs from accidental in two main respects.

First, aspirin is being given to a child who is unwell, perhaps with a simple febrile illness, perhaps because his parents think he is "teething." His fluid intake and output are probably low, and he may be constipated, all of which encourage the accumulation of salicylate in the body (Langmead, 1906). The child most likely to be given aspirin is the child most likely to be poisoned by relatively small doses.

Secondly, aspirin is being administered over a long period. A good description of the fate of aspirin in the body is given by Martin (1964). Unbound salicylate in the blood is in equilibrium with that bound to albumen, and blood salicylate is in turn in equilibrium with tissue salicylate. Inflammatory fluids with a high protein content may attain a salicylate level approaching that in the plasma. Conversely, salicylate levels have to be high for salicylates to enter tissues where metabolism is slow, and it may take 32 hours for free salicylate in the C.S.F. to approach that in the plasma (Mayer *et al.*, 1959). With prolonged high salicylate levels a wider range of tissues may become involved, and one cannot expect that a rapid correction of the plasma biochemistry will be mirrored in an equally rapid correction in the tissues. The toxic action of salicylates on some tissues is not understood, but it is at least possible that sustained high salicylate levels may result in irreversible cell damage. Martin insists that the distribution of salicylate should always be studied with respect to time.

The cases of accidental poisoning were divided into three groups on a basis of time since ingestion. The cases of

therapeutic poisoning, set out in Table IV, can be regarded as a fourth group in this time-series, remembering that a high salicylate level has been present for days rather than hours.

Three of the cases in Table IV are included on debatable grounds. One (Case 5), much older than the others, was poisoned during treatment of rheumatic fever. She is included mainly because she had an alkaline blood pH (7.52) before treatment was begun, stressing the difference in response to salicylate of the toddler and the school-child. One (Case 7) was sent to Belvidere Fever Hospital as a case of pneumonia, and died despite a prompt diagnosis of aspirin-poisoning, which led to the admission of her twin (Case 6), believed by the mother to be identical, to R.H.S.C. In Case 9 the debate is only about the cause of death. There is no doubt he had salicylate-poisoning, but necropsy showed he also had acute laryngotracheobronchitis.

Apart from Cases 5 and 6, all the children were referred to hospital with an incorrect diagnosis. Diagnosis in each case was made in hospital because the hyperpnoea suggested salicylate-poisoning, subsequently confirmed from the history and the blood-salicylate level. The children who died were very ill on admission, with greyish cyanosis, peripheral collapse, restlessness, and varying signs of involvement of the central nervous system—twitching, coma, or rigidity. Less severely poisoned children showed peripheral vasodilatation, and this clinical contrast is of prognostic importance.

If we exclude the three debatable cases, then four children died out of nine. If we include them, six children died out of twelve. Riley and Worley (1956) decided it was best to attribute the deaths in their series to a combination of salicylate-poisoning and infection. It seems to us to be important to be more specific about the role of salicylate.

A case can be made out against salicylates as the cause of death. The salicylate level in those who die may be only moderately raised, the same age and biochemical picture may be found in the dead and the survivors (compare Cases 4 and 8, and Cases 6 and 7), and there is no lesion demonstrable at necropsy which is both lethal and diagnostic of salicylate-poisoning. Apart from the salicylate level the biochemical picture can be imitated by a small child with a severe infection. It is fairly common for small children to die with no clear-cut cause of death evident at necropsy, and the fact that they were given salicylates during life could be purely coincidental.

Such arguments are valid in the individual case. Nobody could be absolutely certain that one particular child died of salicylate-poisoning. When the cases are considered together the impression is different. There is no doubt that these children suffered from severe salicylate-poisoning. That one or two might have died from some unidentified or apparently minor cause is possible; that half of them should have done so is just not credible. To blame an unidentified infection in an individual case may be wise and kind, to exculpate salicylates in general is dangerous folly.

Individuals respond differently to salicylates. A few show frank idiosyncrasy. Similar doses of aspirin will produce different salicylate levels in different children of like size and age, and excretion rates vary. The tissue response to the blood level may vary also. It is not really surprising that similar cases—for example, Case 4 and Case 8—should have different outcomes.

Case 5 is very instructive. The patient was in coma for 24 hours. The only cause for this was salicylate-poisoning. Her salicylate level was only 45 mg./100 ml., and it is most unlikely that it was ever any higher. If this can happen to a child of 12 it should come as no surprise that an infant much more susceptible to salicylates should die with salicylate levels that have never been above the thirties, if they have been there long enough. It is the time factor, the length of time for

TABLE IV.—Analysis of Cases of Therapeutic Poisoning

No.	Age (Months)	Aspirin			Blood Levels		Result	Comments
		Time since Begun (Days)	Hours since Stopped	Total (gr.)	Salic. (mg./100 ml.)	HCO ₃ ' (mEq/l.)		
1	5	14	4	175	30	—	Lived	
2	35	2½	36	30	22	12	"	Hyperpnoeic. Note time since salicylate stopped
3	19	1	24	10	36	10	"	Reputedly, one dose of 10 gr. (0.6 g.)
4	11	1½	4	55	68	6	"	pH 7.28
5	144	30	12	++	45	8	"	pH 7.52. Acute rheumatism
6	15	5	3	40	35	16	"	Twin of Case 7
7	15	4½	12	35	37	15	Died	pH 7.0 before death
8	16	1½	218	45	70	10	"	Cf. Case 4
9	18	1½	6	30	53	6	"	After 24 hours, pH 7.4, HCO ₃ ' 22, Sal. 14. P.M., acute laryngo-tracheobronchitis
10	12	3	12	250	64	10	"	Admitted "decerebrate." Sal. 25 after dialysis
11	6	5	12	82	47	9	"	T. 105° F. throughout "Decerebrate." Blood sugar 29 mg. Died 4th day, after biochemistry corrected
12	16	30	48	450	60	7	"	

which they have been exposed to such levels, that really matters. There are two ways to minimize the time factor. One is to ensure that a toxic level of dosage is never reached. The other is to recognize the signs of salicylate-poisoning as soon as they appear.

Recognition and Early Treatment of Salicylate-poisoning

Meyer (1961) has described the environment in which aspirin-poisoning occurs. About 20% of children in a working-class district of Glasgow have been given aspirin before they are 1 year old (G. C. Arneil, personal communication, 1965). Not only is aspirin-poisoning dangerous, it is likely to occur; and this thought should be in the minds of doctors, pharmacists, and parents.

When dealing with a case of reputed aspirin-poisoning the first step is to empty the stomach. Efficient emesis is probably better than efficient gastric lavage (Arnold *et al.*, 1959). Emesis (Adams, 1961) with salt and water, one tablespoonful to a glass of water, followed by gastric aspiration as a prelude to lavage, can be attempted. Syrup of ipecacuanha has been criticized as slow-acting, but Robertson (1962) found that it acted in less time than it took a child to reach an emergency room. Karlsson and Norén (1965) prefer copper sulphate (0.15 g. in 20 ml. of water under 1½ years, 0.25 g. for others). Equivalent doses of syrup of ipecacuanha would be 10 to 15 ml., given after a drink of water. The syrup of ipecacuanha must not be confused with the fluid extract, which is 14 times stronger, and potentially lethal (Bates and Grunwaldt, 1962).

The physician must then decide on the circumstantial evidence available whether to refer the child to hospital or keep him under observation at home. Observation essentially means waiting to see if he develops hyperpnoea. Vomiting is of little prognostic significance, haemorrhagic manifestations are rare, and tinnitus and nausea, which are all very well in adult textbooks, are difficult to recognize in infants and toddlers. Hyperpnoea may appear within an hour or may be delayed for 18 hours, and is a sign that hospital treatment is necessary.

If the diagnosis is not made outside hospital, and if the child is sent in with a diagnosis of respiratory infection, which is often the fate of the therapeutic case, the burden of initial diagnosis may fall upon the shoulders of a newly qualified house-physician. The following points may help him.

1. It is very seldom that the breathing of pneumonia remotely resembles the hyperpnoea of salicylate-poisoning, which is deep and pauseless, of the air-hunger type. Fever may be present in aspirin-poisoning, and an x-ray examination is not always reliable. One of the present series was reported as showing bronchopneumonia, the lungs being normal at necropsy.

2. A negative ferric chloride test, done on the urine after boiling, will exclude aspirin-poisoning. Ingested salicylate appears in the urine within an hour. Erganian *et al.* (1947) found that traces of salicylate could be detected in the urine with blood salicylate levels as low as 0.4 mg./100 ml., and in the present series the test was positive at a blood level of 2 mg./100 ml. On the other hand, the test is strongly positive with normal therapeutic levels of salicylate, so its main value is as a screening test to exclude salicylate-poisoning.

3. If a child appears to be in or near "diabetic" coma, because of hyperpnoea and glycosuria, but with a blood-sugar level of only 200–300 mg./100 ml., the diagnosis is more likely to be salicylate-poisoning, which may even show polyuria in the early stages. In true diabetic pre-coma in the child the blood-sugar level is rarely below 300 mg. The length of history may be helpful.

In short, if the doctor thinks of salicylate-poisoning he is not likely to miss it, and early diagnosis leads to recovery.

Dosage of Salicylate

Repeated doses of salicylate cause a steady rise in blood salicylate level (Erganian *et al.*, 1947; Hoffman, 1953; Martin, 1964). Most of the salicylate is excreted through the kidneys in conjugated forms. Excretion at a normal pH is slow, but increases if the urine is alkaline, the increase being in the form of free salicylate. In the young infant conjugation is less and renal function immature, so that salicylate tends to be retained. In addition, acidosis develops more readily at a given salicylate level in the infant than in the older child.

Even in a larger child in good health the salicylate levels tend to rise on a constant dosage. Erganian *et al.* found that the level after four days of treatment was about 50% higher than at the end of the first day, and in some cases twice as high. Martin found that a plateau was reached after seven doses. It is likely that infants would show greater and more prolonged rises.

Doctors may remember the dosage of salicylate as 1 to 1½ grains of salicylate per pound of body weight (0.12–0.18 g./kg.) per day. This dosage was recommended by Maggioni (1944) in treating rheumatic fever, and it is in fairly general hospital use for that disease. It does not apply to infants and toddlers, in whom rheumatic fever is very uncommon and in whom such dosage would produce potentially lethal levels within 24 hours. As early as 1930 Marriott suggested an aspirin dose of ½ gr. (30 mg.) four-hourly at the age of 6 months, 1 gr. (60 mg.) four-hourly at one year. Gellis and Kagan (1964) advised a dose of 1 gr. (60 mg.) per year of age every four hours—essentially the dose recommended by Marriott if one remembers to score six months as half a year. The *Extra Pharmacopoeia* (1952) suggests a dose for "children" of "½ to 5 grains (0.03 to 0.3 g.)"—well-intentioned but vague. According to the *British National Formulary* (1963) the total daily dose for a child of 1 year would be 6 to 8 gr. (0.36–0.5 g.) as mist. acid. acetylsal. pro inf., which is on the high side.

It should be noted that though a junior aspirin tablet contains 1½ gr. (75 mg.), tab. acid. acetylsal. sol. pro inf. B.P.C. contains 2½ gr. (150 mg.). A tablet of Paynocol contains 10 gr. (0.6 g.) of aspirin, junior Paynocol 2½ gr. (150 mg.). A Salimed tablet contains 0.5 g. of salicylamide, a substance which is entering the U.S.A. poisoning picture.

Salicylates are of real value only in rheumatic fever and rheumatoid arthritis. Tepid sponging is more effective in reducing fever, and chloral is a safe sedative. The surest way to eliminate therapeutic aspirin-poisoning of infants and toddlers is to withhold the drug entirely from such children.

If it is nevertheless decided that aspirin must be given the dose should not exceed 1 gr. (60 mg.) per year of life five times a day, the dose being calculated in fractions of a year in the young child. It should not be continued for more than two days.

We have insufficient knowledge of paracetamol to recommend it as a substitute for aspirin in this age group.

Treatment in Hospital

The customary treatment of severe salicylate-poisoning is by intravenous fluids with added sodium bicarbonate. Winters (1959) does not follow this method as a routine, and does not use alkalinizing salts unless the pH of the blood falls below 7.15. He feels that alkali may potentiate the masked alkalosis of the first stage of poisoning, causing tetany and convulsions. Kaplan and del Carmen (1958), Oliver and Dyer (1960), and Whitten *et al.* (1961) all favour the use of

bicarbonate, the last-named claiming to have produced a blood pH of 7.7 with no adverse alkalotic effect. Bicarbonate certainly seems to be indicated in the child of under 4 years, but it should be given carefully, with monitoring of the blood and urine. Hyponatraemia is a potential danger, as is hypokalaemia. Oliver and Dyer gave 3.5 to 5 mEq of sodium bicarbonate per kg. of body weight, repeating in four hours if the urine was not alkaline by that time. An indwelling catheter allows checking of the urinary pH and observation of urinary function. Salicylate may commonly have a mild toxic effect on the kidney (Morris and Graham, 1931), and occasionally a severe effect (Campbell and MacLaurin, 1958), apart from the dangers of dehydration.

Sugar, potassium (Robin *et al.*, 1959), and vitamin K may also be given. Trihydroxymethylaminomethane may be of some help as an adjuvant to bicarbonate (Strauss *et al.*, 1961; Israels and Davies, 1961), but Diamox is disappointing and probably dangerous (Kaplan and del Carmen, 1958; Schwartz *et al.*, 1959).

If there is oliguria or anuria further methods of treatment should be employed. The artificial kidney was used by Doolan *et al.* (1951) and on a 2-year-old child by Spritz *et al.* (1959), peritoneal dialysis by Elliott and Crichton (1960), and exchange transfusion by Done and Otterness (1956), Leikin and Emmanouilides (1960), and others. The relative merits of these methods were reviewed by James *et al.* (1961), and in the discussion on that paper McKay warns of the dangers of transfusion with citrated blood. The artificial kidney is the most effective method of clearing salicylate from the blood, but it may not be available or practicable, in which case peritoneal dialysis has a place if there is anuria.

Summary

The national figures for accidental poisoning in childhood show that aspirin is by far the most dangerous poison to-day.

In Glasgow, during 1963–5, there were 79 cases of aspirin-poisoning, of which 67 were accidental with two deaths, and 12 therapeutic with six deaths. Accidental and therapeutic deaths occur in different age groups, and there is reason to believe that very few therapeutic deaths are included in the accidental-poisoning returns, so the problem is even greater than the national mortality figures suggest.

No child died in the accidental group who was admitted within 24 hours of ingestion, and the therapeutic group had all been given aspirin for at least 24 hours before admission. The time factor is therefore very important, and it is linked to accurate diagnosis. Not one of the eight children who died was diagnosed correctly before admission.

The cardinal sign of aspirin-poisoning is hyperventilation. Unrecognized cases of aspirin-poisoning are often diagnosed as pneumonia.

Doubt is cast on the need for giving aspirin to young children at any time. A scale of dosage is suggested for children in whom the use of aspirin is considered imperative.

Advice is given on management outside hospital, and references to the type of treatment which may be used in hospital are quoted.

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