PAPERS AND ORIGINALS

Virus Cross-infection in Paediatric Wards

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Summary

In a survey of virus cross-infection in paediatric wards there were 15 cross-infections due to respiratory syncytial (R.S.) virus and 16 due to influenza A, both during a four-month period, and 19 due to parainfluenza viruses over two years. The illnesses produced by these infections acquired in hospital ranged from a slight cold to severe pneumonia: in 17 of the 50 cases the illness involved the lower respiratory tract. A measure of cross-infection frequency in the form of a "cross-infection rate" has been devised, and it is suggested that this can be used to assess the influence of factors such as ward design and admission policy on the frequency of cross-infection.

Introduction

In the past cross-infection was a serious hazard for children admitted to hospital. Today it is assumed that with preventive immunization, the increasing use of single rooms in children's wards, and the availability of effective antibiotics against most pathogenic bacteria the problem has been solved. Those who work in wards, nurseries, and other institutions admitting children know that this is not true, but they are often uncertain of the nature and extent of the problem.

We have been studying this for some years (Ditchburn *et al.*, 1971) and, though our evidence is incomplete, feel we should share our experiences so far. In this paper we examine cross-infection due to respiratory syncytial (R.S.) virus, influenza A virus, and the parainfluenza viruses. We have measured the extent and clinical importance of the problem and attempted to examine statistically the influence of ward design on the frequency of cross-infection with R.S. virus and influenza A.

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Methods

PERIOD OF STUDY

For R.S. virus and influenza A the survey extended from 14 December 1971 to 31 April 1972, during which period both viruses were epidemic in the Newcastle area. Parainfluenza virus infections as judged by hospital admissions are spread more evenly throughout the year, with smaller and less sharply defined epidemics, so that a longer study period for parainfluenza viruses was necessary, extending from November 1970 to October 1972.

DEFINITION OF CROSS-INFECTION

Virus cross-infection was considered to have taken place when a child acquired an infection after being in the ward longer than the accepted incubation period for the virus. For R.S. virus this period is from five to eight days; for influenza A, one to two days.

Less is known about the incubation period for parainfluenza viruses, and from the information available in this study it was not possible to define this period accurately. However, it appeared that in some cases the incubation period may be as short as two days, and therefore children with parainfluenza virus infections who developed symptoms two or more days after admission were included.

THE WARDS

The wards studied statistically for evidence of cross-infection with influenza A or R.S. virus were divided according to their design: group A consisted of four wards in which open ward structure is combined with some cubicles; group B consisted of four wards composed almost entirely of individual cubicles (table I).

For this study group A wards were designated by the numbers 1 to 4. Wards 1 to 3 admitted children of all ages, and ward 4 only children of under 5 years. Wards 1 and 2 are divided into two sections separated by a corridor and an administrative area 20 yds (18 m) wide, one half mainly for children over 5 and the other mainly for those under 5. Ward 3 has one section only. Ward 4 consists of four units each containing four cots, and four single-cot cubicles. Whenever possible, children of less than 1 year were admitted into single-cot cubicles.

Group B wards were designated 5 to 8. Wards 5 and 6 admitted children of under 5 years. Ward 7, which admitted children of all ages, consists of two sections, one with six single cubicles for children under a year and the other a separate open section containing cots and beds for older children; it is included in group B because all but one of the children with either influenza A or R.S. virus infection were admitted to cubicles. Ward 8 admitted only children under 1 year. The proportion of single-bedded cubicles in the eight wards is shown in table I.

In eight other wards virus surveillance was less intense, and these were therefore not included in our statistical analysis of the frequency of cross-infection and the influence of ward design. The cross-infections detected on these wards are included in the total figures shown in table II.

TABLE I—*Percentage of Total Number of Cots or Beds in Each Ward Which are in Single Cubicles*

Group A:	Percentage Cubicles	Group B:	Percentage Cubicles
1	25	5	100
2	26	6	100
3	37	7	100*
4	20	8	87†

*Includes a separate open section (see text).
†Includes one unit with three cots.

TABLE II—*Number of Cross-infections Related to Admissions of Virus Infection*

Virus Type	No. of Admissions	No. of Cross-infections
R.S. virus	219	15
Influenza A	61	16
Parainfluenza virus type 1	55	5
Parainfluenza virus type 2	9	0
Parainfluenza virus type 3	56	13
Parainfluenza virus type 4A	7	1
Parainfluenza virus type 4B	7	0
Total	414	50

CROSS-INFECTION RATE

The frequency of virus cross-infection in the past has not been accurately assessed, mainly because of varying degrees of clinical and laboratory surveillance of possible cases. Variations in the numbers of children admitted with a particular virus infection and in the length of hospital stay have made it difficult to compare the frequency of cross-infection in different units.

Knowing the statistical difficulties, we still felt it necessary to devise some comparative measure in the form of a "cross-infection rate." The basis for calculation in this study was the number of cross-infections which occurred when the number of susceptible child days was related to the number of child days of primary infection in the ward. The possibility of tertiary cases (a child becoming infected by a secondary case who had himself acquired the infection in the ward) was not taken into account.

The cross-infection rate was calculated as follows:

$$\text{Cross-infection per million susceptible days per infective day} = \frac{\text{Number of cross-infections} \times 10^6}{(\text{Number at risk} \times \text{mean stay}) \times (\text{Number of infected} \times \text{their mean stay})}.$$

It was not possible to allow for the length of time of virus excretion of each infected child nor for adult carriage of viruses. The assumption has been made that both these factors were similar in wards of groups A and B.

VIROLOGY

Cough/nose swabs and nasopharyngeal secretions were obtained from each child; the methods of collection and laboratory investigations have been described elsewhere (McQuillin and Gardner, 1968; Sturdy *et al.*, 1969; Ditchburn *et al.*, 1971).

CLINICAL CATEGORIES

Each respiratory infection was assigned to a long-accepted clinical category (pneumonia, bronchiolitis, bronchitis, croup, tonsillitis, pharyngitis, cold) previously described (Gardner *et al.*, 1960). One child with a febrile illness without respiratory signs could not be classified in this way.

Results

EXTENT AND CLINICAL IMPORTANCE OF CROSS-INFECTION

The study of cross-infection with R.S. virus and influenza A extended over four months. During this period 219 children were admitted to hospital with illnesses due to R.S. virus infection and 15 children acquired the infection in hospital; 61 children were admitted with illnesses due to influenza A virus, and 16 acquired the infection in hospital. These 31 infections were experienced by 30 children, one child being simultaneously infected with both R.S. and influenza A viruses.

Over the two-year study period for parainfluenza viruses 134 children were admitted to hospital with illnesses due to parainfluenza viruses, and 19 probably acquired the infection in hospital (table II). Five of these cross-infections were readmissions shortly after discharge from hospital. All had been in contact with known cases of parainfluenza infection while in the wards, and the interval was probably too short for them to have acquired the infection at home.

For several reasons these figures are likely to underestimate the frequency of cross-infection. In the first place, some cases will occur after discharge from hospital, and these will escape notice unless the illness produced is severe enough to require readmission. This is particularly applicable to R.S. virus, with a relatively long incubation period. Secondly, virus surveillance was less regular for some wards than for others. In the case of parainfluenza viruses there could well be a higher threshold for the recognition of cross-infection as these illnesses were less epidemic in distribution. Nevertheless, daily virus surveillance was felt to be sufficiently close to detect most of the episodes of R.S. virus and influenza A cross-infections occurring on those wards subjected to statistical analysis.

CLINICAL PICTURE AND AGE INCIDENCE OF ILLNESSES PRODUCED BY CROSS-INFECTION

The details of respiratory illnesses and the ages of the cross-infected children for R.S. virus, influenza A virus, and the parainfluenza viruses respectively are given in tables III, IV, and V. The primary reasons for their admission to hospital are also shown.

In addition, the history of one child not shown in the tables merits separate description. This 3-month-old infant died at home as a cot death. He had been admitted to hospital with gastroenteritis and discharged 10 days previously. During his stay in hospital there was a child with a parainfluenza type 3 infection in the adjacent cubicle. No immediate history of respiratory infection was obtained from other members of the family but parainfluenza type 3 was found in respiratory tract secretions and in the lungs after death; the lungs also showed

TABLE III—*Ages, Reasons for Admission to Hospital, and Illnesses Developed by 15 Children who acquired R.S. Virus Infection in Hospital*

Case No.	Age	Reason for Admission	R.S. Virus Associated Illness
1	4 weeks	Severe cold	Cold
2	11 weeks	Social problem	Cold
3	12 weeks	Social problem	Bronchiolitis
4	13 weeks	Gastroenteritis	Cold
5	15 weeks	Severe cold	Bronchiolitis
6	16 weeks	Fractured femur	Bronchiolitis
7	17 weeks	Meningomyelocele	Bronchiolitis
8	21 weeks	Social problem	Cold
9	25 weeks	Burns	Bronchiolitis
10	28 weeks	Social problem	Bronchiolitis
11	30 weeks	Cervical abscess	Bronchiolitis
12	52 weeks	Congenital heart disease	Cold
13	88 weeks	Hydrocephalus	Cold
14	110 weeks	Failure to thrive	Cold
15	6 years	Social problem	Cold

TABLE IV—*Ages, Reasons for Admission to Hospital, and Illnesses Developed by 16 Children who acquired Influenza A Virus in Hospital*

Case No.	Age	Reason for Admission	Influenza A Associated Illness
16	3 months	Congenital emphysema	Pneumonia
17	11 months	Mental retardation	Pneumonia
18	13 months	Mental retardation	Pharyngitis
19	16 months	Scalds	Fever
20	18 months	Failure to thrive	Cold
21	18 months	Pneumonia	Pneumonia
22	18 months	Social problem	Cold
23	22 months	Social problem	Pharyngitis
24	22 months	Meningococcal septicaemia	Cold
25	22 months	Hydrocephalus	Cold
26	24 months	Mental retardation	Cold
27	26 months	Congenital heart disease	Cold
28	30 months	Cystic fibrosis	Bronchitis
29	38 months	Meningomyelocele	Cold
30	5 years	Asthma	Pharyngitis
31	9 years	Portal hypertension	Otitis media

TABLE V—*Ages, Reason for Admission to Hospital, and Illnesses Developed by 19 Children who probably acquired Parainfluenza Virus Infection in Hospital*

Case No.	Age	Reason for Admission	Parainfluenza Associated Illness	Para-Influenza Type
32	2½ months	Sonnei dysentery	Cold	3
33	5 months	Sonnei dysentery	Cold	3
34	6 months	Immune deficiency syndrome	Pneumonia	3
35	6 months	Acute leukaemia	Cold	4A
36	7 months	Croup due to para. 1*	Bronchitis	3
37	7 months	Failure to thrive	Pharyngitis	3
38	8 months	Bronchitis due to para. 1	Bronchitis	3
39	10 months	Failure to thrive	Pharyngitis	3
40	15 months	Axillary abscess	Cold	3
41	15 months	Febrile convulsion with upper respiratory tract infection*	Pneumonia	1
42	16 months	Diarrhoea and vomiting	Cold	3
43	21 months	Febrile convulsion with otitis media	Croup	1
44	30 months	Developmental retardation	Cold	3
45	31 months	Cystic fibrosis	Bronchitis	1
46	31 months	Cystic fibrosis	Bronchitis	3
47	31 months	Social problem	Cold	3
48	30 months	Accidental poisoning*	Croup	1
49	4 years	Sonnei dysentery	Cold	3
50	11 years	Appendicitis*	Otitis media	1

*Previous recent admission.

histological changes of early bronchiolitis. We cannot state with certainty that this infant acquired the parainfluenza virus in hospital, nor that the virus was the cause of death. We can only note the association and chain of events and hope that observations in other cases will clarify the role of viruses in these "unexpected deaths." A larger body of evidence which we are accumulating makes it almost certain that viruses are involved in a proportion of sudden unexpected deaths in young infants (Ferris *et al.*, 1973).

SOURCE OF INFECTION

In 14 out of the 15 cases of R.S. virus cross-infection, and in 15 out of the 16 cases of influenza A cross-infection, at least one child on the same ward was known to have been excreting the same virus at the appropriate time. In the other two cases no source was identified.

In 14 of the 19 cases of parainfluenza virus cross-infection it was again possible to trace one or more contacts among children on the same ward. In one case, however, the probable source of infection was the house physician, from whom the virus was isolated at the time of a heavy cold, and who had himself probably acquired his infection from a child in an adjacent ward. In a further two cases carriage by staff from infected children on adjacent wards was probable, though specimens from possible carriers were unfortunately not obtained. In the remaining two cases no source for the cross-infection was identified.

EXAMPLES OF CROSS-INFECTION IN DETAIL

The following examples illustrate the ways cross-infection occurred.

Ward 1.—This ward consists of two main sections separated by a corridor and adjoining "administrative" rooms. The nursing, domestic, and medical staff for each part is the same, but the children are admitted to and remain within one or other section of the ward. There were two outbreaks of influenza. The first followed the admission on the same day and to the open ward of two children with febrile convulsions due to influenza A. The first, aged 11 months, was adjacent to a child of 9 years with portal hypertension and who, three days later, developed otitis media due to influenza A. The second infector, aged 18 months, was admitted to the other section of the ward next to a child of 2½ years with cystic fibrosis; nine days later this child developed a sharp attack of bronchitis due to influenza A.

The second outbreak followed the admission of a child of 12 months with a febrile convulsion due to influenza A. For a variety of reasons this active, mobile infant remained in the ward for 11 days. Three infections due to influenza A subsequently followed. The first, a child aged 18 months, was admitted to a cubicle with severely infected scabies three days after the index case, and developed a cold nine days later. The second, a child aged two admitted to the open ward for the investigation of severe mental retardation five days after the index case, developed a cold seven days later. The third infection was in a child aged 18 months with R.S. virus pneumonia admitted to a cubicle eight days after the index case; five days later, when he had almost recovered from his first pneumonia, a second pneumonia developed associated with influenza A. The long excretion of influenza A in childhood infections (Brocklebank *et al.*, 1972) makes patients infections for long periods, and, despite the short incubation period, relatively long intervals may occur before the susceptible contact develops symptoms.

Ward 4.—The ward which experienced this outbreak consists of four units each with four cots and four single-cot cubicles. Four children while in the ward were infected with R.S. virus. The first, aged seven months, was in a four-cot unit for investigation of failure to thrive and developed bronchiolitis after 40 days due to R.S. virus. The probable sources of infection were three children admitted six, seven, and eight days before the secondary bronchiolitis. The first was in the ward for four days with otitis media, the second for two days with bronchitis, and the third for eight days with bronchiolitis, all due to R.S. virus. The second acquired R.S. virus infection occurred in a child of 18 months with hydrocephalus in an adjacent four-cot unit. This child developed a cold due to R.S. virus 12 days after the first of the three potential infectors described above was admitted. The third episode of R.S. virus cross-infection involved a 13-month-old child admitted to a four-cot unit for the investigation of failure to thrive; nine days later this child developed an R.S. virus cold. The probable infector was a child of four years admitted to the same unit with bronchitis due to R.S. virus infection. The fourth child, aged 3 months, was admitted to a four-cot unit with a fractured femur; after 14 days he developed R.S. virus bronchiolitis. The probable infector was a child of 13 months who had been admitted to an adjacent single-cot cubicle with bronchiolitis six days previously.

Ward 2.—On 21 June 1972 a 6-year-old boy was admitted with a sore throat, high fever, and headache, and parainfluenza type 1 was identified in his nasopharyngeal secretions. A few days later a 2-year-old child who had already been in the ward for eight months with cystic fibrosis developed an exacerbation of his bronchitis, and on 30 June parainfluenza type 1 was identified in his nasopharyngeal secretions. On 27 June the house physician on the ward developed a

heavy cold and on 1 July was found to be excreting parainfluenza type 1, by which time his cold was improving. The same virus was recovered a week later from an 11-year-old boy who developed a heavy cold while convalescing from an appendicectomy on an adjacent ward. This ward was covered by the same houseman, and it therefore seemed likely that at the time of his cold he carried the virus from the first ward to the second. Two further children were probably involved in this outbreak. The first, aged 21 months, had been in the ward with otitis media and a febrile convulsion from 21 to 26 June, and was then readmitted on 30 June with croup due to parainfluenza type 1. The second, aged 2½ years, was in the ward on 3 and 4 July, having accidentally taken nitrazepam, and was readmitted on 9 July, again with croup due to parainfluenza type 1.

CROSS-INFECTION RATE AND WARD DESIGN

As stated above, we attempted both to measure the frequency of cross-infection and to relate this to ward design. This analysis was restricted to the eight wards in groups A and B, and to R.S. virus and influenza A infections only. Virus surveillance was carried out daily on these wards, and was felt to be sufficiently reliable at these epidemic times to make it probable that most episodes of cross-infection were identified. A further reason for excluding parainfluenza infections from this analysis was our uncertainty about the length of the incubation period for these infections.

The cross-infection rate in wards of groups A and B for R.S. virus and influenza A respectively are shown in tables VI and VII. The overall rate for group A wards was greater than that for group B wards for both viruses. The numbers of cross-infections were too small to make statistical comparisons between the rates, but there is a clear trend for the rates to be lower in wards consisting entirely of single-cot cubicles than in those combining some cubicles with an open area.

TABLE VI—*Ward Cross-infection Rate for R.S. Virus*

	Total Admissions	R.S. Virus Admissions	No. of Susceptible Child Days*	No. of Infective Child Days†	Cross-infection	
					No.	Rate
Group A:						
Ward 1	370	19	2,036	198	3	7·4
„ 2	323	17	2,142	189	2	4·9
„ 3	95	14	855	110	0	0
„ 4	309	34	1,854	196	4	11·0
Total	1,097	84	—	—	9	7·1‡
Group B:						
Ward 5	124	31	1,395	209	2	6·9
„ 6	125	9	1,044	61	1	15·7
„ 7	288	14	1,638	168	0	0
„ 8	224	16	1,456	219	1	3·1
Total	761	70	—	—	4	4·2‡

*No. of susceptible child days = No. at risk × their mean stay in hospital.
†No. of infected children × their mean stay in hospital.
‡The overall cross-infection rate is weighted for each ward.

TABLE VII—*Ward Cross-infection Rate for Influenza A*

	Total Admissions	Influenza A Admissions	No. of* Susceptible Child Days	No. of† Infective Child Days	Cross-infection	
					No.	Rate
Group A:						
Ward 1	370	14	2,065	78	3	18·5
„ 2	323	15	2,156	60	5	38·6
„ 3	95	3	828	11	2	219·6
„ 4	309	14	1,770	85	4	26·6
Total	1,097	46	—	—	14	31·0
Group B:						
Ward 5	124	3	1,815	15	0	—
„ 6	125	2	984	16	0	—
„ 7	288	5	1,698	22	1	26·8
„ 8	224	0	0	0	0	—
Total	761	10	—	—	1	12·4

*No. of susceptible child days = No. at risk × their mean stay in hospital.
‡No. of infected children × their mean stay in hospital.

Conclusions

Though the total number of cross-infections was small, the fact that 19 children acquired lower respiratory tract infections or croup emphasizes the potentially serious nature of cross-infection with R.S. virus, influenza A virus, and the parainfluenza viruses. Age is the first factor in determining severity, with young infants more vulnerable to R.S. virus and parainfluenza type 3 virus, and children between one and five years to influenza A and the other parainfluenza viruses. The second factor which may increase the risk of acquiring infection and its severity is the nature of the primary illness which resulted in the child's admission. We have shown elsewhere (Gardner *et al.*, 1967) in a series of deaths associated with respiratory infection, that one in three children had predisposing disease or malformation—mainly congenital heart disease, malformations of the nervous system, severe mental handicap, and cystic fibrosis. These conditions were present in a similar proportion (30%) of the children with cross-infection in this study.

Another important factor increasing the risk of cross-infection is the duration of virus excretion, which may be as long as 14 days for R.S. virus (Gardner *et al.*, 1970), 10 days for influenza A (Brocklebank *et al.*, 1972), but an undetermined period for the parainfluenza viruses.

We believe there is a need for a comparative index of cross-infection, and have suggested a method by which a cross-infection rate can be calculated. This, we hope, will be of value as a standard both for comparisons of cross-infection frequency in different centres and for monitoring the effect of new measures for control introduced in any one centre over the years.

As with all indices used to monitor situations subject only to gradual change and involving relatively small numbers—for example, the infant mortality rate—it will be the interpretation of trends in cross-infection rate which will be of most practical value; statistically significant comparisons will be less helpful, except over long periods or where major changes have taken place. We have given an example of how such a trend can be measured, by examining the influence of two types of ward design on cross-infection. The similarity of the two groups of wards in respect of other factors which tend to limit the frequency of cross-infection, such as a high standard of nursing care and the availability of rapid virus diagnosis, has made the differences in cross-infection rates too small to be statistically significant. But such is our concern about the severe nature of some of the illnesses acquired, that we feel compelled to emphasize the clear trend for rates to be lower in wards consisting entirely of cubicles. It would seem wise that, whenever possible, children with acute respiratory illnesses should be admitted to individual cubicles, and that these should not be reserved solely for infants under 1 year of age as is sometimes the practice. This is supported by the observations of Sterner (1972).

In the absence of antiviral agents or effective immunization against respiratory virus infection in children, what means of limiting cross-infection are available?

Firstly, it is unwise to admit children to hospital with conditions which predispose to respiratory infection if this can be avoided, especially during the winter or in epidemic times. If admission is necessary then the single-cot cubicle, though not an absolute barrier, is superior to the open ward.

Secondly, if all infants and young children with respiratory symptoms or febrile or unexpected convulsions could be isolated for the first 24-48 hours rapid virus diagnosis would enable the uninfected to remain separate, while those infected by the same virus could be nursed together.

Finally, though individual cubicles play an important part in the prevention of cross-infection, the fact that some virus cross-infections occurred even in wards consisting entirely of cubicles suggests that modes of transmission other than direct spread from patient to patient must sometimes play a part. Despite the difficulties, we are currently attempting to study the role of carriage by ward staff, parents, and visitors in the cross-infection process.

We are well aware of the many problems faced by those in charge of an acute children's ward today, and that wards whose design is satisfactory on other grounds will be in use for a long time. Nevertheless, without asking too much of busy resident doctors and ward sisters, we believe that the knowledge of the facts and the applications of the conclusions of this study could do much to improve the situation.

We are indebted to the M.R.C. for continuing support. We are also grateful to the medical and nursing staff of all the hospitals who showed the patient and good-tempered co-operation without which this type of study would be impossible.

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Excretion Urography in Acute Renal Failure

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Summary

High-dose excretion urography has been carried out in 32 patients presenting with non-obstructive acute oliguric or non-oliguric renal failure. An early, dense, persisting nephrogram has been observed in all patients with acute uncomplicated tubular necrosis and in patients with acute oliguric pyelonephritis. This appearance is modified by the presence of pre-existing renal disease. Different patterns have been observed in patients with acute glomerular disease, severe renal ischaemia, and chronic glomerular disease. The study demonstrates that careful analysis of the evolution of the nephrogram in patients with acute renal failure provides valuable information as to the nature of the parenchymal disease.

Introduction

High-dose excretion urography is of established value in the investigation of patients with non-oliguric renal failure, both to find or exclude treatable postrenal obstruction and, by defining the renal outlines, to help diagnose the nature and severity of chronic parenchymal disease (Fry and Cattell, 1971a, 1971b). It has been less extensively used in patients with oliguric renal failure, partly because of fear that the investigation might be hazardous (Schencker, 1964; Fry and Cattell, 1970) and partly because of doubt whether any useful information could be obtained. Recent studies of our own and others (Mahaffy *et al.*, 1969; Brown *et al.*, 1970; Meadows *et al.*, 1971) have, however, shown that high-dose urography is safe in oliguric subjects and is capable of defining both renal size and the presence or absence of obstruction.

In the course of these studies it became apparent that critical examination of changes in the density of the kidney during excretion urography—the nephrographic pattern (Fry and Cattell, 1972)—could also yield information about the nature of the parenchymal disease in patients with acute non-obstructive renal failure (Moseley *et al.*, 1971). To extend these observations we now report the urographic findings in 32 patients presenting with acute non-obstructive oliguric and non-oliguric renal failure. This study clearly indicates that careful analysis of the nephrographic pattern taken in conjunction with renal size and the presence or absence of pelvicalyceal filling will provide valuable information about the nature of the renal lesion in these patients.

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Patients and Methods

Altogether, 32 patients, aged 22-69 years, have been studied (see table*). All had presented with renal failure of recent acute onset without any previous history of renal disease. In many cases a presumptive clinical diagnosis had been made but in all some doubt existed as to the true diagnosis. Most (22) were oliguric (less than 500 ml of urine per day) and all were uraemic. Prerenal circulatory failure had been excluded by the time of study as judged by persisting oliguria and/or increasing uraemia despite adequate volume replacement and intensive diuretic treatment with mannitol and frusemide. For the purpose of the present investigation patients with postrenal obstruction were also excluded.

High-dose excretion urography was performed as soon as possible after admission to hospital and exclusion of prerenal circulatory failure. Severely uraemic and fluid-overloaded patients were dialysed to improve their clinical condition before the investigation.

Urographic Technique.—Fluid restriction was avoided. 1 ml/lb body weight (2·2 ml/kg) Hypaque 45% (sodium diatrizoate) or an equivalent dose of Conray 420 (sodium iothalamate) was injected intravenously over a period of 3–5 minutes. Preliminary tomograms of the renal areas were obtained at the same time as the control plain films. Whenever possible films were taken immediately at the end of injection, at 5, 10, 30, and 60 minutes, and at intervals up to 24 hours. Tomograms were always obtained immediately after injection and with most of the subsequent films. In a few patients the full range of films was not taken owing to their clinical condition. Special care was taken to ensure that tomographic cuts throughout the examination were comparable with regard both to radiographic exposure and to level of cut.

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