The reason that some severely depressed patients do not achieve suppression of plasma 11-O.H.C.S. after dexamethasone is uncertain. It is possible that adrenocortical activity has become relatively autonomous in these patients, similar to the situation in Cushing's syndrome. However, the reversible nature of the defect in most patients, the absence of any clinical features of Cushing's syndrome, and the preservation of a normal diurnal rhythm make this unlikely. Other possibilities are that dexamethasone fails to reach the hypothalamus in the resistant patients or that the steroid-sensitive neurones are subjected to an abnormal drive from other limbic areas.

The conflicting reports that deal with levels of blood 11-O.H.C.S. and their relation to depression have been reviewed by Coppen (1967). Our own findings are similar to those reported by Brooksbank and Coppen (1967)—namely, that morning levels of 11-O.H.C.S. are within the normal range, and that the diurnal variation is not affected. We found a significant fall in the 4.30 p.m. 11-O.H.C.S. levels in the recovered depressed group, but we did not test the non-depressed patients after a similar time in hospital, because they had received a variety of drugs during treatment.

We have eliminated interfering drug effects so far as is possible. No patient received any drug except amylobarbitone for at least a week before and during testing (apart from one recovered depressive who was also having imipramine). As a group the depressives needed larger amounts of amylobarbitone during the initial testing period than they did before discharge. Barbiturates have been shown to interfere with the release of A.C.T.H. (Krieger and Krieger, 1967), but there is no evidence that they can enhance A.C.T.H. release. We have also found that neither amylobarbitone nor imipramine interferes with the estimation of plasma 11-O.H.C.S. by fluorescence (Carroll, 1968).

The results presented here show that the failure of plasma 11-O.H.C.S. to suppress with dexamethasone may not be diagnostic of adrenocortical hyperplasia if the patient is severely depressed. They also indicate the need for further definition of hypothalamic-pituitary function in depressive illness.

It is a pleasure to thank Dr. Cameron Baird and Dr. Margot Baillie for generously providing facilities for one of us (B. J. C.) to perform the 11-O.H.C.S. determinations. We also thank Messrs. Geigy Pharmaceuticals for financial support.

Addendum

Since preparing this paper we have considered another possible explanation for our results. Doig *et al.* (1966) reported that 7 out of 12 depressives showed a plasma 11-O.H.C.S. peak at 3 a.m. instead of at 6 a.m. as in normal people. They suggested that the diurnal curve of plasma 11-O.H.C.S. was shifted "to the left" in these patients. On recovery normal 6 a.m. peaking was found.

On consideration of these data we felt that the midnight dose of dexamethasone may have been given too late in the cycle to affect the diurnal rise in our depressives. We have now compared the effects of dexamethasone given at 9 p.m. and at midnight on four further patients. The results are shown in Table III. Normal suppression clearly failed to occur on both occasions. These figures indicate that the failure of the depressed patients to suppress their plasma 11-O.H.C.S. levels is not simply a function of the timing of dexamethasone administration in relation to an altered diurnal rhythm.

TABLE III

Case No.	Basal Plasma 11-O.H.C.S. (μg./100 ml.)		Plasma 11-O.H.C.S. at 8.30 a.m. after Dexamethasone 2 mg. at:	
	8.30 a.m.	4.30 p.m.	9 p.m.	12 Midnight
1	35	23·7	34·6	35·4
2	20	10·4	14·6	19·2
3	28·2	20·1	21	27·2
4	28·6	19·5	22·4	17·2

REFERENCES

Brooksbank, B. W. L., and Coppen, A. (1967). *Brit. J. Psychiat.*, **113**, 395.
Carroll, B. J. (1968). Unpublished observations.
Coppen, A. (1967). *Brit. J. Psychiat.*, **113**, 1237.
Doig, R. J., Mummery, R. V., Wills, M. R., and Elkes, A. (1966). *Brit. J. Psychiat.*, **112**, 1263.
Fawcett, J. A., and Bunney, W. E. (1967). *Arch. gen. Psychiat.*, **16**, 517.
Gibbons, J. L. (1966). *Excerpta Medica International Congress Series*, No. 111, Abstract No. 370. Amsterdam.
Krieger, D. T., and Krieger, H. P. (1967). *Science*, **155**, 1421.
McHardy-Young, S., Harris, P. W. R., Lessoff, M. H., and Lyne, C. (1967). *Brit. med. J.*, **2**, 740.
Mattingly, D. (1962). *J. clin. Path.*, **15**, 374.
Nugent, C. A., Nichols, T., and Tyler, F. H. (1965). *Arch. intern. Med.*, **116**, 172.

Viral Antibody Levels and Clinical Status in Acute Exacerbations of Chronic Bronchitis: a Controlled Prospective Study*

A. C. STENHOUSE,† B.MED.SC., M.B., M.R.C.P., M.R.A.C.P.

Brit. med. J., 1968, **3**, 287–290

Summary: A controlled prospective study was made of a group of patients with chronic bronchitis, in which serum antibodies against a group of viruses and Mycoplasma pneumoniae were estimated at regular intervals. Sixteen significant rises in antibody titre were shown, of which eight were associated with clinical acute exacerbations of bronchitis. In individual patients no correlation was found between the number of acute exacerbations or the aetiological agent and persistent change in ventilatory function as expressed by the F.E.V.$_{0.75}$.

This study was compared with the results of a previous parallel investigation of the same patients done to study the significance of rhinovirus infections. In the present investigation 12% of the acute exacerbations were associated with the 11 agents tested compared with 14% associated with rhinoviruses in the earlier work.

* This study is included in material to be presented in a thesis as partial fulfilment of the M.D. degree of the University of Otago, New Zealand.

† Wellcome Research Fellow, University Department of Medicine, the Royal Hospital, Sheffield, and the University of Sheffield Virus Research Laboratory, Lodge Moor Hospital, Sheffield. Address from 21 September: London School of Hygiene and Tropical Medicine, Keppel Street, London W.C.1.

Introduction

In healthy subjects the viruses isolated from common acute respiratory infections differ according to the age of the person

under study and to the degree of involvement of the upper and lower respiratory tract. In adults rhinoviruses are the most important single cause of upper respiratory tract infection, and these, together with parainfluenza types 1, 2, and 3 viruses, respiratory syncytial virus, and adenoviruses, make up the two-fifths of acute upper respiratory tract infections that can be defined at present. In lower respiratory tract infections influenza type A and B viruses, parainfluenza type 3 virus, adenoviruses, and the pleuropneumonia-like organism *Mycoplasma pneumoniae* make up the one-third that can be recognized (Chanock *et al.*, 1965).

The majority of reports pertaining to the viral aetiology of acute exacerbations in chronic bronchitis are based on serological studies, and a wide range of agents are implicated. Influenza A virus, particularly during epidemics and pandemics, has been shown to cause exacerbations often severe enough to require admission of the patient to hospital (Tyrrell, 1952; Stuart-Harris *et al.*, 1953; Walker *et al.*, 1958; Murdoch *et al.*, 1959; Ross *et al.*, 1966). Similarly influenza B virus and parainfluenza types 1 and 3 viruses may at times be associated with up to 7% of exacerbations (Stark *et al.*, 1965). In addition to these viruses, which in healthy subjects occasionally cause lower respiratory tract infection, two recent studies have shown that rhinoviruses can be identified in 23 and 14% of acute exacerbations (Eadie *et al.*, 1966; Stenhouse, 1967). Respiratory syncytial virus, which in normal adults is responsible for a mild upper respiratory illness, has also been claimed by some observers to possess an association with acute exacerbations (Sommerville, 1963; Carilli *et al.*, 1964). Thus the distinction present in healthy adults between viruses responsible for upper and for lower respiratory tract infections is not applicable in the case of patients with chronic bronchitis.

The purpose of this paper is, firstly, to consider the presence or absence and the type of respiratory illness associated with significant serological rises in a prospective study of a group of chronic bronchitic patients and controls. Secondly, an attempt is made to assess the relative importance of different agents in causing acute exacerbations by comparing the present results with a parallel and previously described study, in the same subjects, on the significance of rhinovirus infection in acute exacerbations of chronic bronchitis (Stenhouse, 1967).

Materials and Methods

Clinical.—The study groups comprised 34 chronic bronchitis patients having a mean age of 61.3 years, compared with a mean age of 51 years for the 19 control subjects. The chronic bronchitis patients fulfilled the criteria for chronic bronchitis as defined by the Medical Research Council (1965). The term "acute exacerbation of bronchitis" embraces a spectrum of clinical disorders of varying severity and was defined in this study as "an alteration in the quiescent state of the patient with an increase in either the quantity or purulence of the sputum." On a number of occasions the bronchitic patients presented with an increase in wheeze and shortness of breath which was not accompanied by a change in the quantity or purulence of the sputum produced; these were classified as "non-specific bronchitis" and were not included in this study as acute exacerbations. The criteria used for the diagnosis of "common cold" and "influenza" were those outlined by Tyrrell (1965).

Virological.—Venepuncture blood samples were taken routinely at two-monthly intervals. In addition, acute and convalescent specimens were obtained at the times of acute respiratory illness. After separation the serum was stored at −20° C. All serum specimens from a particular subject were examined for antibodies to a particular agent under the same experimental conditions by the complement fixation method as described by Grist *et al.* (1966). A fourfold or greater rise in antibody levels between consecutive specimens was required as evidence of recent infection. The consecutive specimens consisted of acute and convalescent sera, together with the comparison of one serum sample with its immediate predecessor, taken routinely at two-monthly intervals. Each serum specimen was examined for antibodies to influenza A, B, and C viruses, parainfluenza 1, 2, and 3 viruses, the adenovirus group, respiratory syncytial virus, the psittacosis group, Q fever, and *M. pneumoniae.* All the antigens used were supplied by Dr. C. P. Bradstreet, Central Laboratory of the Public Health Laboratory Service, Colindale, and were stored at 4° C.

Results

Table I presents the total number of acute respiratory and acute upper respiratory infections that occurred in the chronic bronchitic and control groups. The finding of 2.0 and 2.6 acute upper respiratory infections per subject per year for the control and chronic bronchitis groups represents a comparable incidence for the numbers involved. However, the occurrence of acute exacerbations in the bronchitis subjects is reflected in the increased total acute respiratory infections of 3.6 per subject per year for the bronchitis patients compared with 2.0 in the control group.

TABLE I.—*Total Number of Acute Respiratory Tract Infections and Acute Upper Respiratory Tract Infections in the Chronic Bronchitic and Control Groups*

Group	No. of Subjects	Acute Upper Respiratory Infections		Total Acute Respiratory Infections	
		No.	No. per Subject per Year	No.	No. per Subject per Year
Chronic bronchitis ..	34	56	2·6	79	3·6
Control	19	21	2·0	21	2·0

From the 267 sera examined from both the bronchitis patients and the control group 17 fourfold or greater antibody rises were demonstrated, of which 16 occurred in the chronic bronchitic patients. These rises occurred between two consecutive samples from the same person and/or from paired serum samples obtained during an acute exacerbation. No fourfold or greater falls in antibody levels occurred over a period short enough to be of significance. The single positive result in the control series was to influenza A virus in a subject in whom clinical influenza was diagnosed. The number and titre of the significant antibody rises for each of the agents tested is listed in Table II. No significant serological rises occurred with influenza C virus, parainfluenza type 2 and 3 viruses, adenovirus group, and the Q fever agent.

TABLE II.—*Fourfold or Greater Complement-fixing (C.F.) Antibody Rises to Antigen Tested*

Antigen	No. of Subjects with Fourfold or Greater C.F. Antibody Rises	C.F. Antibody Levels in Consecutive Sera*
Influenza A	6	16/64, 8/64, 16/64, 32/128, 8/32, 4/16†
Influenza B	2	4/16, 4/64
Influenza C	0	—
Parainfluenza 1	1	<4/16
Parainfluenza 2	0	—
Parainfluenza 3	0	—
Respiratory syncytial ..	3	16/64, 8/32, 4/16
Adenovirus	0	—
Psittacosis	3	16/64, 4/16, <4/32
Q fever	0	—
M. pneumoniae	2	32/128, 16/128

* Numerator = acute serum. Denominator = convalescent serum. Antibody titre expressed as a reciprocal.
† The single rise that occurred in a control subject.

Table III presents the number of serological rises to particular agents in the chronic bronchitis patients in relation to their clinical status at the time that the antibody response occurred. Thus from a total of 16 significant antibody rises only eight could be correlated with an acute exacerbation of bronchitis—four antibody responses occurred to influenza A virus, two to

influenza B virus, and one each to the psittacosis agent and respiratory syncytial virus. Of the remaining eight serological rises three were recorded in the absence of clinical illness, including both of the rises to *M. pneumoniae* and a single rise to influenza A virus. A significant antibody rise to respiratory syncytial virus was recorded in a patient presenting clinically a common cold. The four significant antibody rises unrelated to a specific clinical illness were associated with a variety of causes. With the rise to parainfluenza type 1 virus the subject presented three clinical illnesses in the interval between the sera tested, the two intervening serum specimens being lost. With respiratory syncytial virus and one of the rises to the psittacosis agent the patients failed to report for a convalescent sample and it was considered that the period between the specimens was too long to be conclusive. The other antibody rise to the psittacosis agent was in association with a rhinovirus isolation made at the time of the clinical illness.

TABLE III.—*Number of Significant Serological Rises in Chronic Bronchitis Patients in Relation to Clinical Status*

Agent Tested	Acute Exacerbations	Acute Upper Respiratory Infection	No Clinical Illness	Not Relatable
Influenza A	4	—	1	—
Influenza B	2	—	—	—
Parainfluenza 1 ..	—	—	—	1
Respiratory syncytial ..	1	1	—	1
Psittacosis	1	—	—	2
M. pneumoniae	—	—	2	—

From the total of 79 acute respiratory infections in the chronic bronchitis patients only 64 may be included in this study as acute exacerbations unassociated with rhinovirus infection. Thus 8 out of 64 (12%) acute exacerbations were related to the 11 specific agents tested by the complement fixation method. Considering the relatively small number of acute exacerbations and serological conversions recorded, it is not possible to associate, on a statistical basis, the agents as the cause of the exacerbations. However, a much higher incidence of virus infections occurred during acute exacerbations than in the quiescent periods. No significant antibody rises or virus isolations were recorded in the chronic bronchitis patients presenting with 22 episodes of " non-specific bronchitis."

A description of the clinical illness typically associated with influenza type A and B virus infections in chronic bronchitis patients is presented.

Case 1.—On 11 March 1965 a 73-year-old man experienced a sudden onset of malaise. The following day he became more short of breath and the quantity of mucoid sputum increased. There were no symptoms referable to the upper respiratory tract. This illness was diagnosed as an acute exacerbation of bronchitis. The level of complement-fixing antibodies to influenza A virus was 1 in 16 on 12 March and had risen to 1 in 64 on 6 April. He had remained well in the intervening period.

Case 2.—A 60-year-old man developed headache, rhinorrhoea, increased wheeze, and cough on 26 February 1965. His sputum became purulent and he was more short of breath. A diagnosis of a common cold with an acute exacerbation of bronchitis was made. His symptoms subsided over the ensuing 10 days. The complement-fixing antibody level to influenza B virus was 1 in 4 on 27 February and 1 in 64 on 27 March.

Discussion

Carilli *et al.* (1964) reported that their patients with chronic bronchitis presented eight times the number of acute respiratory illnesses compared with a control series. This finding is different from the present study, in which the Sheffield bronchitis patients presented an incidence of 3.6 acute respiratory illnesses as compared with 2.0 per subject per year for the healthy controls. This discrepancy may be due to the small number of respiratory illnesses in the 10 subjects in the control group of Carilli *et al.* (1964), since the incidence of acute exacerbations in their bronchitis patients was 2.6 per subject per year, a finding consistent with other published work.

The incidence of acute exacerbations, as reported by Moffat and Sutherland (1967), Eadie *et al.* (1966), and the present study, is 0.8, 1.6, and 3.2 per subject per year respectively. This increasing incidence is inversely related to the findings in the ventilatory function of these subjects. Moffat and Sutherland (1967) required an indirect maximum breathing capacity greater than 50 litres per minute as a prerequisite for inclusion of a patient in their study, whereas the mean maximum breathing capacity in this study was 30.2 l./min. (unpublished observation). Eadie *et al.* (1966) used the peak flow-meter reading as an index of airways obstruction, and their subjects appeared to fall in an intermediate position between the present study and that of Moffat and Sutherland (1967). Thus it appears that as the functional disability increases there is an increased incidence of acute exacerbations of chronic bronchitis. This may be due to an increased involvement of the lower respiratory tract in a greater percentage of acute upper respiratory infections. Thus in the study by Eadie *et al.* (1966) 21 out of 75 acute respiratory infections involved only the upper respiratory tract compared with 6 out of 79 in the present study. Alternatively, increased primary involvement of the lower respiratory tract may occur, as evidenced by the published reports of the clinical illnesses associated with rhinovirus infections in the chronic bronchitis patients (Eadie *et al.*, 1966; Stenhouse, 1967).

The part played by exacerbations in the progression of the disease is more difficult to discern. Though ventilatory function studies were performed in the present investigation, it was not possible to correlate either the number of acute exacerbations or infections due to a particular virus with a persistent change in the maximum breathing capacity (unpublished observation). Howard (1967), studying this problem in a larger group and for a longer period, noted that the majority of acute exacerbations treated with antibiotics had very little effect on individual forced expiratory volume ($F.E.V._{0.75}$) curves.

It is of interest to record that the 22 episodes of " non-specific bronchitis " were confined to a few of the chronic bronchitis patients in this study (unpublished observations). In addition no virus infections, using isolation and serological studies, were found to be associated with this change. Though the numbers involved are small, this observation may merit further study, since this group can be recognized clinically as a separate entity and thus the aetiological basis for and the ultimate management of this reaction may be different from that defined as " an acute exacerbation of chronic bronchitis."

Out of the total of 17 significant antibody rises 16 occurred in the chronic bronchitis patients, and this would suggest that, although not all the rises were relatable to clinical illness, the bronchitic group received more frequent antigenic stimulation. This might be expected to lead to higher antibody levels in a greater percentage of bronchitis patients as compared with controls. This is not borne out, however, by the study of Ross *et al.* (1966), who reported no difference in the percentage or level of antibodies to a wide range of viruses in a chronic bronchitis and a control group when matched for age.

A parallel study of the same subjects indicated that 14% of the acute exacerbations were associated with a rhinovirus infection (Stenhouse, 1967). This should be compared with the 12% due to the 11 agents studied serologically. Eadie *et al.* (1966) found 23% of acute exacerbations associated with a rhinovirus, and the associated serological studies showed only two significant antibody rises, one each to influenza A and C viruses. These reports place the various agents responsible for the initiation of acute exacerbations in some perspective and emphasize the important role of the rhinovirus. They do not indicate, however, the probable variation from year to year which may occur as a reflection of the varied incidence of the different respiratory viruses present in the community.

It is a pleasure to record my appreciation of the help received from Professor C. H. Stuart-Harris in all phases of this study, for his constructive criticism of this paper, and for the permission given to investigate patients under his care. I should like to thank the chief technician, Mr. J. Skelton, and the technical staffs of the Virus Research Laboratory, Lodge Moor Hospital, and of the department of medicine, the Royal Hospital, Sheffield, for their considerable part in this study; in particular, Miss Lynda Bebbington for her excellent assistance at all times.

References

Carilli, A. D., Gohd, R. S., and Gordon, W. (1964). *New Engl. J. Med.*, 270, 123.
Chanock, R. M., Mufson, M. A., and Johnson, K. M. (1965). *Progr. med. Virol.*, 7, 208.
Eadie, M. B., Stott, E. J., and Grist, N. R. (1966). *Brit. med. J.*, 2, 671.
Grist, N. R., Ross, C. A. C., Bell, E. J., and Stott, E. J. (1966). *Diagnostic Methods in Clinical Virology*. Oxford.
Howard, P. (1967). *Brit. med. J.*, 3, 392.
Medical Research Council (1965). *Brit. med. J.*, 1, 775.
Moffat, M. A. J., and Sutherland, J. A. W. (1967). *Brit. med. J.*, 1, 601.
Murdoch, J. McC., Leckie, W. J. H., Downie, J., Swain, R. H. A., and Gould, J. C. (1959). *Brit. med. J.*, 2, 1277.
Ross, C. A. C., McMichael, S., Eadie, M. B., Lees, A. W., Murray, E. A., and Pinkerton, I. (1966). *Thorax*, 21, 461.
Sommerville, R. G. (1963). *Lancet*, 2, 1247.
Stark, J. E., Heath, R. B., and Curwen, M. P. (1965). *Thorax*, 20, 124.
Stenhouse, A. C. (1967). *Brit. med. J.*, 3, 461.
Stuart-Harris, C. H., Pownall, M., Scothorne, C. M., and Franks, Z. (1953). *Quart. J. Med.*, 22, 121.
Tyrrell, D. A. J. (1952). *Quart. J. Med.*, 21, 291.
Tyrrell, D. A. J. (1965). *Common Colds and Related Diseases*. London.
Walker, W. C., Douglas, A. C., Leckie, W. J. H., Pines, A., and Grant, I. W. B. (1958). *Lancet*, 1, 449.

Medical Memoranda

Paraquat Poisoning Treated by Forced Diuresis

Brit. med. J., 1968, 3, 290–291

Eight cases of poisoning (six fatal) by the weed-killer paraquat (1,1'dimethyl-4,4'-dipyridilium) have now been reported (Bullivant, 1966; Clark, McElligott, and Hurst, 1966; Almog and Tal, 1967; *Brit. med. J.*, 1967; Mourin, 1967; Campbell, 1968; Oreopoulos, Soyannwo, Sinniah, Fenton, McGeown, and Bruce, 1968). The following is a report on one such case treated by forced diuresis in which the patient recovered.

Case Report

The patient, a man aged 32, was admitted to hospital on 25 November 1967 after ingestion of about 45 g. of Weedol (which contains 5% paraquat) in an attempt to poison himself. The Weedol had been partially dissolved in water. He was first seen one and a half hours after ingestion with a complaint of fiery periumbilical pain which had begun about 15 minutes after swallowing the weed-killer. He had not vomited.

He had previously been physically healthy. Since March 1967 he had noticed a change in mood with subsequent intermittent episodes of depression, loss of interest, insomnia, and paranoid feelings. He began to contemplate suicide in the latter three months when the depression deepened.

Examination on admission showed epigastric tenderness and guarding. The liver edge was just palpable below the costal margin. There was no evidence of pre-existing cardiac, pulmonary, or renal disease. Shortly after admission gastric lavage was performed and no solid Weedol particles were visible in the aspirate. Forced diuresis was begun.

Next day he complained of anterior chest tightness, frontal headache, and photophobia. Chest auscultation revealed generalized bilateral sibilant rhonchi. The abdominal pain disappeared by the eighth day and tenderness by the tenth day. The liver edge remained palpable throughout. He remained free of oral and pharyngeal ulceration. Clinical examination of the chest showed disappearance of rhonchi by the tenth day and there was no evidence of pulmonary hypertension at any time. The temperature rose on only one occasion to 99° F. (37.2° C.). A five-day course of intramuscular penicillin was started on admission and was followed by a five-day course of prednisolone, begun on the fifth day.

Investigations.—Daily chest *x*-ray films and electrocardiograms performed from time of admission showed no abnormality. The haemoglobin and absolute values were found to be normal. The white cell count was 6,650/cu. mm. on admission and 13,500/cu. mm. on the twelfth day. The E.S.R., blood urea, urine urea, serum electrolytes, serum bicarbonate, standard liver function tests, and arterial blood gas values were all persistently normal. Occult blood was not found in the stools. Creatinine clearance just before discharge was 58 ml./min.

Forced diuresis was performed by the method of Linton, Luke, Speirs, and Kennedy (1964) and continued for 24 hours. Throughout the forced diuresis two-hourly aliquots of urine were collected and blood samples were obtained at intervals. The concentrations of paraquat in each of these specimens and in one specimen of gastric aspirate were determined by the method of Daniel and Gage (1966). The lower limit of sensitivity by this method is 10 μg. of paraquat. This quantitative method is efficient but time-consuming. The urine cell counts were performed by the method of McGeachie and Kennedy (1963).

TABLE I.—*Paraquat Excretion and Serum Concentrations*

Time in Hours	Volume of Urine (ml.)	Paraquat Conc. in Urine (μg./100 ml.)	Total Paraquat in Urine (μg.)	Paraquat Conc. in Serum (μg./100 ml.)
Pre-forced diuresis ..	75	14,800	11,100	
0–2	1,775	840	14,910	85 (0 hours)
2–4	575	510	2,932	40 (4 hours)
4–6	750	490	3,675	—
6–8	950	330	3,135	—
8–10	900	230	2,070	40 (10 hours)
10–12	950	230	2,185	—
12–14	500	230	1,150	—
14–16	1,100	105	1,155	Nil (15 hours)
16–18	700	95	665	Nil (17 hours)
18–20	850	95	808	Nil (19 hours)
20–22	1,400	95	1,330	Nil (21 hours)
22–24	650	95	618	—
Total ..			46·0 mg.	

Table I shows the values of paraquat excretion and serum concentrations. Table II compares paraquat clearance with urine flow rate. It is apparent from this that the trend is for higher clearance of paraquat with the higher urine flow rate. The urine cell count figures are shown in Table III. Cell excretion reached a peak about the fourth day and thereafter fell to normal levels.