than 66,000, the mean value for all the counts presented here. It is possible that this discrepancy is to some extent due to the factor of cellular lysis in urine. This method is accurate to about 10%, and the limits of the normal range coincide very closely with those found by Rofe (1955), who investigated the urinary cellular excretion of 12 normal male subjects by a method rather more elaborate than that described here.

Since the completion of the work presented above, it came to our notice that Hamburger *et al.* (1950) had already described a similar method of estimating the urinary cell excretion rate. No details are given in their paper of the number of subjects used or the number of counts performed. They gave 30,000 white and epithelial cells an hour as the maximum normal excretion rate. This method was subsequently employed by Richet (1953) for the estimation of red-cell excretion rates in the urines of cases of nephritis.

Faced as he often is with a report of positive culture from a urinary specimen and a report on the deposit which, owing to the method by which it is obtained, is often meaningless, the clinician may well be at a loss to decide whether or not his patient has an infected urinary tract or whether the organisms grown represent no more than contaminants. It is felt that this method, by providing a reasonably accurate quantitative evaluation of the urinary deposit, may resolve this all too common difficulty.

Although the primary purpose of this paper is the presentation of a method, some of the results we have obtained are of sufficient interest to merit brief discussion.

The highest counts were obtained from ambulant males in the morning, all of whom had voided their night urine prior to the period of collection of the specimen. The difference between these counts and those from ambulant males in the afternoon and from resting males in the morning was significant. On the other hand, no such differences existed between the counts for ambulant and resting males in the afternoon, while the differences between resting males in the two periods in the day were not significant.

It appears that the resumption of activity at the beginning of the day may be responsible for a rise in the rate of cellular excretion and that this rate then falls later in the day irrespective of the posture of the subject at that time. This conclusion is supported by the results obtained from the resting male subjects in the afternoon and the female subjects at each period of the day. The males who were resting in the afternoon had been ambulant in the morning, and the mean of their cell excretion rates was higher than that of the males resting in the morning, who had been in bed all night, and had not been allowed up before the period of the count. The resting female patients, however, who were used for morning counts had been up for an hour or two before the period of urine collection began, and the counts obtained on them are slightly higher than the corresponding ones for the males. Similarly, those female subjects used for the afternoon counts were allowed up in the morning and their cell excretion rates did not differ from those of the resting males counted during the same period. Most of these differences do not attain a level of statistical significance, perhaps because the groups studied were not large enough, but they all show a consistent trend.

It should be emphasized that, as the results of the assessment of variance show, there is no characteristic pattern of cell excretion in any one subject, and therefore that the limits of the normal as defined for the groups apply equally to the individual.

All female subjects were catheterized, thereby eliminating the urethra as the source of the cells observed, and no difference between the rates of cell excretion in sexes was found. This would suggest that the cells counted originated above the urethra.

Summary

The rate of excretion of leucocytes and non-squamous epithelial cells has been estimated in normal subjects by

a simple and reliable method. The normal rate varied between 18,000 and 196,000 an hour.

The results are analysed according to the time of day at which the urine was collected and the posture, sex, and age of the subjects. In three normal males no characteristic rate of excretion over a period of several days was observed.

The rates of excretion in normal urines and in a few pathological urines are compared.

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MULTIPLE STOKES-ADAMS EPISODES DURING ANAESTHESIA

BY

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Many cases of cardiac arrest have been documented in the medical literature, and the treatment of such cases is becoming well established. An immediate diagnostic thoracotomy, should the peripheral pulses become impalpable, has been advocated by some authors (Rao *et al.*, 1954; Milstein and Brock, 1954; Mullens, 1955), but others have taken a more conservative attitude (Johnstone, 1955a). As thoracotomy may be definitely contraindicated in "arrest" associated with Stokes-Adams attacks, and because such episodes during anaesthesia have only rarely been reported, the recording of such a case appears justified. McLemore and Levine (1955) presented a series of cases in which, incidentally, Stokes-Adams episodes were associated with anaesthesia, but the details of the drugs administered and the techniques followed were not given.

Case Report

An 80-year-old white man (80 kg.) was admitted to hospital with acute intestinal obstruction. For 15 years he had had orthopnoea with arteriosclerotic heart disease, and for six years hypertension (170/100) with complete heart-block. Electrocardiograms had previously revealed myocardial infarction. He suffered from chronic pulmonary emphysema, and intermittent episodes of congestive heart failure had occurred until the time of admission, when he showed minimal oedema and crepitant rales at the lung bases. He had been fully digitalized. Abdominal distension and vomiting were present.

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Laparotomy was planned for undiagnosed intestinal obstruction. Following premedication (pethidine hydrochloride 50 mg.) the abdomen was opened under an "arrow-head" block (Evans, 1949) (lignocaine, 700 mg., and adrenaline, 0.3 mg.). A further 40 mg. of pethidine hydrochloride was given in intermittent doses through the intravenous drip during this period. On exploration of the peritoneal cavity it became apparent that the surgical manipulations would require supplementary anaesthesia. Thiopentone sodium (80 mg., followed by 160 mg.) was given intravenously to produce light anaesthesia. Oxygen (100%) was administered by mask and later by manual ventilation following suxamethonium (25 mg.) intravenously. "Cyclaine" (5%) was used to anaesthetize the larynx, the patient was again ventilated with oxygen, and a cuffed endotracheal tube was passed. Ventilation was continued with nitrous oxide (60%) and oxygen (40%) until spontaneous respirations returned.

During this time the patient developed transient but recurrent episodes in which he appeared pallid and pulseless. The electrocardiogram showed intermittent asystole, ventricular tachycardia, or ventricular fibrillation. A diagnosis of Stokes-Adams episodes was made. During the episodes, which lasted up to three or more minutes, and which followed each other at one- to three-minute intervals, the respirations remained of good volume, but they underwent transient depression, and a few seconds of apnoea occurred, following the return of the peripheral pulse on each occasion. During the latter part of each attack the patient's pupils became transiently dilated.

A large internal hernia was reduced and the surgery rapidly completed (40 minutes), during which time 100% oxygen was continually administered, and suxamethonium (105 mg.) injected intravenously as required for surgical relaxation. The patient was then extubated and oxygen continued by mask. The episodes persisted almost continually during surgery and post-operatively, and disappeared quite suddenly two hours and forty minutes after the onset. At the start of these incidents procaine amide (100 mg.) had been twice administered, as it was felt that the ventricular fibrillation justified this medication, but no other specific treatment was given. Later nikethamide (5 ml.) was administered and repeated, to lighten the thiopentone hypnosis, and thereafter the patient was conscious and rational between attacks. As the respiratory pattern suggested that a "chemoreceptor" drive was controlling his respirations, the oxygen which had been administered during all the attacks was withheld on two occasions. Only one further attack occurred.

The patient showed no signs of cerebral damage following these long periods of "arrest" and made an uninterrupted recovery, being discharged home on the 17th post-operative day.

Discussion

The mechanisms which may have contributed to the sudden occurrence of Stokes-Adams attacks in this patient include vagal or sympathetic overactivity, or factors concerning ventilation management.

Vagal Stimuli.—Even in the presence of complete heart-block, visceral, laryngeal, or pulmonary reflexes may have contributed to these episodes. In this respect, however, Johnstone (1955b) concludes (as a result of observations of laryngeal movements and electrocardiographic changes) that suxamethonium provides better and safer conditions for endotracheal intubation than other relaxants. In this patient, also, topical anaesthesia of the larynx was performed, and no irritant vapours were used. In spite of his conclusion, however, Johnstone has shown that a definite slowing of pulse occurs in some patients (a muscarinic acetylcholine-like effect) following the administration of suxamethonium, and in retrospect there seems very little doubt that atropine should have been included in the premedication of this patient. In one of the more severe cases reported by McLeomore and Levine (1955) atropine had also been omitted from the premedication.

Sympathetic Overactivity.—The effect of excessive adrenaline on the intact Purkinje cells may give rise to ventricular arrhythmias which may be depressed by procaine, but in this patient it seems unlikely that the adrenaline in the infiltration and block could have caused any deleterious effect, and the administration of procaine amide can be shown to be definitely contraindicated in Stokes-Adams syndrome.

Ventilatory Management.—Anoxia, which may always be a factor in the administration of an anaesthetic, and which could contribute to cardiovascular collapse, had been avoided in this instance. Conversely, it is difficult to dissociate the onset of the attacks from the administration of 100% oxygen by manual inflation. It is known that the administration of high oxygen concentrations may have a deleterious effect on the respiratory mechanism in chronic pulmonary disease associated with hypercarbia, and Stead *et al.* (1954) have shown, in animals, that sudden lowering of the alveolar carbon dioxide levels, after prolonged hypercarbia, may raise the plasma potassium to cardiotoxic levels, precipitating ventricular fibrillation. It is an interesting conjecture whether efficient ventilation with a high percentage of oxygen, to which he was unaccustomed, might not have been a factor on the onset of these attacks in this patient. There is little doubt that the episodes disappeared when manual oxygenation was withheld.

This experience brings to the fore several points of interest, particularly the differentiation between these attacks and "true" cardiac arrest. It is doubtful whether this man would have survived a thoracotomy, and it is worth noting that these patients seem to recover with little specific treatment.

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HEPARIN AND SYMPATHETIC BLOCK IN TREATMENT OF VENOUS THROMBOSIS

EXPERIMENTAL INVESTIGATIONS

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At present there are two main methods of treatment of venous thrombosis: (1) by anticoagulant agents, and especially by two main compounds of this group—heparin and dicoumarol; and (2) by paravertebral procaine block of the sympathetic system. Both methods are supported by evaluation of clinical results, but it is difficult to compare them with precision. Encouraged by Professor T. Butkiewicz, I decided to investigate this problem experimentally.

Experiments were designed to produce thrombosis and then to apply treatment either by heparin or by procaine block of the sympathetic system. The importance of experiments such as we carried out on 50 dogs may be gathered when it is realized that in the accessible medical world literature we could find no experimental work evaluating comparatively these methods of treatment.