

in the pulmonary area was split and there was a systolic murmur (grade 3) at the apex. The Kahn test was negative. X-ray examination showed general cardiac enlargement with no particular enlargement of the left auricle. The liver section showed the changes of passive congestion only. E.C.G. showed auricular fibrillation and right bundle-branch block.

Once again he responded to digitalis and mersalyl, and was discharged free from heart failure.

#### Case 7

A 42-year-old cook from El Obeid drank a huge quantity of water one evening during Ramadan, when the Muslim takes no fluid from sunrise to sunset. Following this he felt exhausted and generally unwell. Next morning he woke with rapid palpitation of the heart, and this continued on and off for four days. He was admitted to hospital, and after an E.C.G. had been done a diagnosis of paroxysmal ventricular tachycardia was made and the rhythm was restored to normal by quinidine. He was discharged and remained well for five months, when, a month before the present admission, he had a return of the palpitation. There was no breathlessness, but he complained of abdominal distension during the attacks of palpitation.

*Examination.*—His pulse was 110 a minute, irregular from extrasystoles, and of small volume; the thyroid was normal and there were no signs of thyrotoxicosis; B.P. 130/100; the jugular venous pressure was very high. The apex beat was displaced outwards and the heart sounds were normal apart from a third heart sound heard at the apex and a systolic murmur (grade 3) heard in the same place. E.C.G. showed inversion of the T waves in leads I, V<sub>2</sub>, and V<sub>4</sub>, with a biphasic T wave in V<sub>6</sub>. X-ray examination of the chest confirmed general cardiac enlargement, and liver section showed the changes of passive congestion. He remained in hospital for two weeks without any prolonged attack of palpitation; the pulse rate then rose to 180 a minute and he became very breathless. His hands were cold and he was disorientated. Two days later the pulse rate fell to 48, and then after a few hours he collapsed and died.

#### Discussion

The aetiology of the condition remains unknown. This series of cases does suggest, however, that certain of the tentative theories put forward previously will not do. Coronary artery disease, at least of the main arteries, is not a factor: the site of the lesion is against it. Anginal pain was strongly denied and atheroma is rare in the Sudanese working-class. During 18 months I have heard only one patient complain of anginal pain, and he had syphilitic aortic incompetence. This is in strong contrast to the middle and wealthier classes of patients, in whom cardiac infarction is common, often at an early age.

Undernutrition and malnutrition were not present in these patients. The Sudan has been prosperous for some time now, and the protein content, including meat protein, is at least adequate. Young children sometimes show nutritional deficiencies and kwashiorkor occurs, but the absence of chronic liver damage in these cases is against any long period of malnutrition. The disease was found, in three cooks. Moreover, Gray (1951) reported the disease in a British Colonial Service doctor and a District Magistrate from Nigeria. Edge (1946) also described what must have been a similar case in a European from West Africa. Alcohol does not appear to have been a factor in these cases.

The disease is not limited to any race and is common in those of Arab stock. Syphilis was not a factor. The uneducated Sudanese is never afraid to admit a history of this condition, and, moreover, the Kahn test was negative in all the cases in which it was done. Eosinophilia was found in only two patients, suggesting that the high incidence in other series is due to parasitic infestation rather than being a mark of the allergic nature of the condition. Again advanced liver disease was found in only one case.

Heart failure due to pulmonary bilharziasis occurs in the Sudan, but is of a different nature from that in these cases. It presents as pure right heart failure, usually with a high cardiac output, and is easily recognized. Again, trypanosomiasis occurs in the Sudan, but it has a limited distribution and few of these patients came from infected areas.

Endocardial fibrosis thus appears as a well-defined clinical and pathological condition occurring commonly in Africa in negroes, Arabs, and Europeans alike. It is probably very widespread, and in the past has been labelled chronic ischaemic heart disease. In the Sudan it appears as heart failure for no apparent cause, occurring in adults associated with embolic incidents and usually responding to digitalis and mercurial diuretics. The cases described above have been in middle-aged and elderly patients, but I have seen unexplained cardiac enlargement associated with electrocardiographic abnormalities in younger patients, including children, which may well be due to the same cause.

**ADDENDUM.**—Since this article was submitted, the paper by Ball *et al.* (1954) on endomyocardial fibrosis has been published. The cases seem to be similar with the exception that those authors found mitral incompetence a common lesion. All cases of mitral incompetence were excluded from the present series, as, in the absence of necropsy, it was impossible to disprove a rheumatic aetiology. On the other hand there were several such cases, and in view of the Makerere findings they may well have been examples of endocardial fibrosis.

#### Summary

The clinical features of 25 cases of endocardial fibrosis with congestive heart failure occurring in Arabs in the Sudan are described. No clue to the aetiology of the condition was found.

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## MAXIMUM URINE CONCENTRATION

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One of the most easily studied functions of the kidney is its capacity to conserve water under conditions of dehydration. In these circumstances water is reabsorbed from the tubular fluid against an osmotic gradient through the influence of endogenous antidiuretic hormones, and the maximum concentration of the solutes is used as an indication of the kidney's capacity to perform osmotic work. Although osmolarity is the appropriate property which determines the extent to which the solutes can be concentrated, specific gravity is usually measured for clinical convenience. Specific gravity, however, gives only an approximate indication of the osmolarity.

The present paper reports observations on the urines of normal subjects and patients with renal disease, either after 10 to 36 hours' water deprivation or after exogenous "pitressin." The first part of the paper is concerned with the errors which may arise when specific

gravity is measured instead of osmolarity; the remainder deals with some factors concerning the attainment of maximum osmolarity.

Specific gravity was estimated by a standard ward hydrometer, correction being made for a small calibration error and an allowance of  $\pm 0.001$  being made for each  $3^\circ$  C. variation above or below  $15.6^\circ$  C. at which the hydrometer was calibrated. Detergents were not used for cleansing flasks or urinals. Osmolarity was determined by cryoscopy (depression of freezing-point in degrees centigrade  $\div 1.86 =$  osmols per litre) with a thermometer graduated in  $0.01^\circ$  C; the probable error was less than  $\pm 10$  milliosmols per litre.

Creatinine output rate was always measured in order to detect errors in urine collection. Creatinine was estimated by Bonsnes and Taussky's (1945) modification of Folin's method.

Urea concentration was determined by the method of Van Slyke and Cullen as quoted by Peters and Van Slyke (1932).

#### Validity of Specific Gravity as an Indication of Urine Osmolarity

It is generally agreed that the concentration of urine is limited mainly by its osmotic activity or the *number* of particles in solution and that the kidney is indifferent to the *weight* of the solutes. Thus the reabsorption of water by the tubules is opposed to the same extent by a light ion as by a molecule many times heavier. This accounts for the widely appreciated fact that specific gravity (which is a measure of the weight of solutions) is unreliable as an indication of osmolarity when the urine contains substantial quantities of protein or sugar, for these heavy molecules give disproportionately high specific gravity readings. It is not always realized, however, that urines of like osmolarity containing neither of these substances may still show considerable differences in specific gravity.

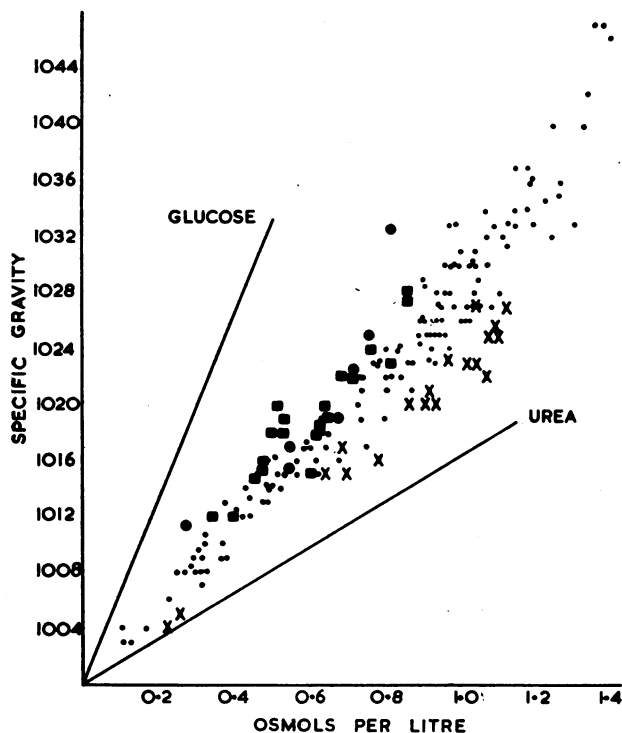


FIG. 1.—Relationship between specific gravity and osmolarity of urine. Different urines are shown as follows: With no sugar or protein (•). With + + + sugar (●). With + + + protein (■). After 25 g. urea by mouth (x). Lines are also given showing the relationship between the specific gravity and osmolarity of glucose and urea solutions.

Fig. 1 plots the osmolarity of more than 200 urines against their specific gravity. It shows that specific gravity is only a rough indication of osmolarity even in normal urines, so that a urine of specific gravity 1017 may be as concentrated, in terms of osmolarity, as a urine of specific gravity 1022, and that the error is greater at higher concentrations. The figure also shows the relatively high specific gravities of urines containing large quantities of sugar or protein, and the relatively low specific gravities of urines with high urea concentrations, collected from subjects after the ingestion of 25 g. of urea.

In addition to this unavoidable error, which is inherent in the use of specific gravity as a measure of osmolarity, there are also avoidable errors due to faulty technique in specific gravity measurement. Calibration errors of 0.002 are common, and urines, when allowed to reach room temperature on a warm day, may give specific gravity readings 0.002 or more lower than would be recorded at the correct temperature. If these errors summate, giving a 0.004 difference from the true figure in a urine which also happens to contain a high urea content (and therefore a low specific gravity in relation to its osmolarity), it will be obvious that the "specific gravity" may give a grossly misleading idea of the urine osmolarity.

#### Duration of Water Deprivation and the Attainment of Maximum Urine Osmolarity

There seems to be little information on the time taken for the water-deprived subject to achieve maximum concentration of the urine. This will depend, presumably, on many factors, the most important of which are probably the temperature and humidity of the air and the nature and quantity of the food consumed. For a concentration test, different authors recommend periods of dehydration ranging from as little as 12 (Chamberlain, 1951) to as many as 34 hours (Lashmet and Newburgh, 1932) before the onset of the urine collection period.

The following investigation was carried out on 13 convalescent patients under ordinary ward care during the

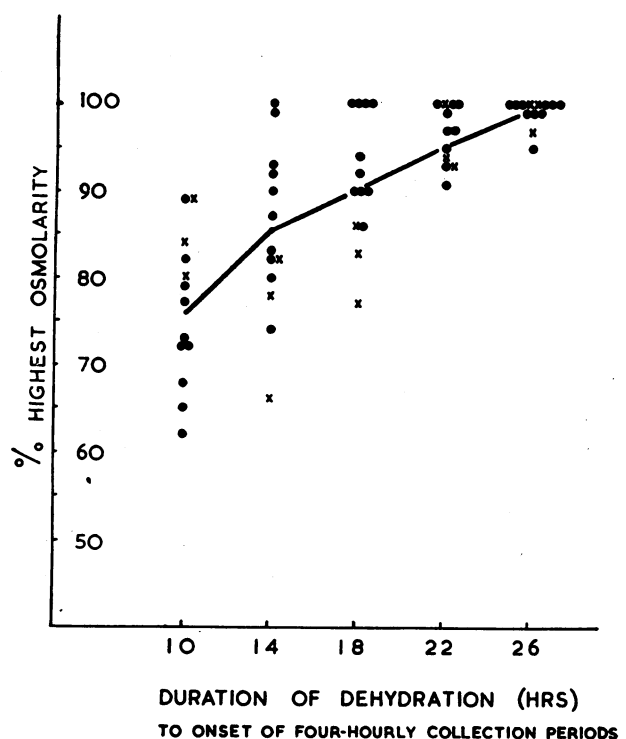


FIG. 2.—Effect of progressive water deprivation on the urine osmolarity in 13 subjects. The osmolarity is expressed as a percentage of the highest osmolarity reached in each subject during 30 hours' dehydration. Urines were collected between 8 a.m. and 4 p.m. in cases marked by a dot, and between 8 p.m. and 4 p.m. in those marked by a cross.

winter; they were considered to have normal kidney function. All were deprived of fluids for 30 hours, but allowed normal food. Four-hourly urine samples were saved during the last 20 hours of this period.

Though it is not claimed that the maximum osmolarity is always reached by 30 hours' dehydration, it is at least clear that it is often not even approached by 14 to 18 hours' (Fig. 2). The results suggest that at least 22 hours' dehydration is desirable to achieve a reasonably accurate idea of maximum osmolarity. The wide scatter of observations after shorter dehydration is equally important; it means that the maximum concentration cannot be predicted from the concentration reached after a short period of dehydration.

**Comparison of Urine Osmolarity after 24 to 36 Hours' Dehydration and after Pitressin Tannate**

Since a long period of dehydration is necessary to obtain urine of near maximum osmolarity, it would clearly be advantageous if the same effect could be achieved with less hardship to the patient. It was therefore decided to see whether equally good concentration could be obtained by using pitressin. Pitressin tannate in oil was chosen because it acts for many hours and there are no undesirable side-effects. The ampoules were warmed and well shaken before its administration.

Nineteen patients, described in Table I, were studied on two separate occasions. They were either convalescent or in a steady clinical state. Three specimens of urine were collected at four-hourly intervals between 8 a.m. and 8 p.m., on the first occasion after 24 to 36 hours' dehydration and on the second after a subcutaneous injection of 5 units of pitressin tannate in oil at 7 a.m. A moderate amount of fluid was allowed during the second test.

The subjects may be divided arbitrarily into those who after dehydration could concentrate above an approximate level of 850 m.osmols/l. and those who could not. Fig. 3 shows that among the former the effect of pitressin was conspicuously less than the effect of dehydration. In those who could not concentrate so well, however, the effect of pitressin was as good as, or even superior to, the effect of dehydration.

Table I shows a tendency for the osmolarity to increase during the day under both conditions. It is not known

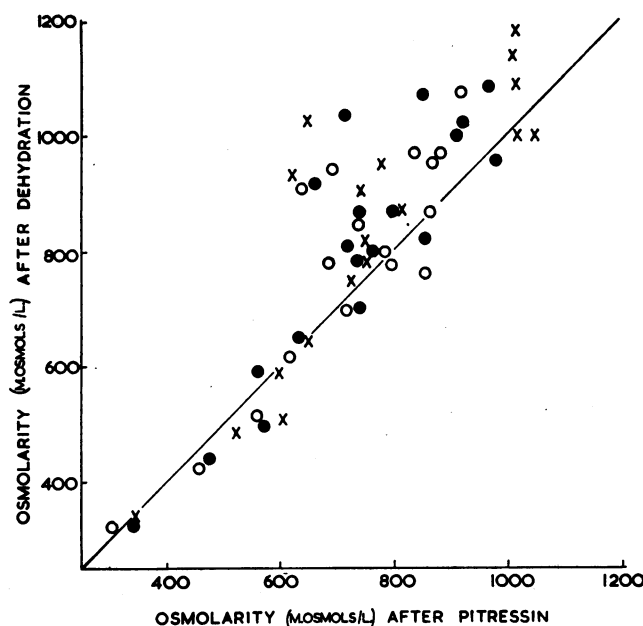


FIG. 3.—Urine osmolarity of morning (O), afternoon (●), and evening (X) specimens after 24 to 36 hours' dehydration and after 5 units of subcutaneous pitressin tannate is compared in 19 subjects (see Table I).

TABLE I.—Urine Osmolarities of Morning, Afternoon, and Evening Urines in 19 Subjects after 24 to 36 hours' Dehydration and after 5 units of Subcutaneous Pitressin Tannate

Age	Sex	Diagnosis	Urine Osmolarity (m.osmols/litre)					
			After 24-36 Hours' Dehydration			After 5 Units Pitressin		
			8 a.m.	Noon	4 p.m.	8 a.m.	Noon	4 p.m.
24	M	Peptic ulcer ..	958	1,025	1,086	876	923	1,021
62	F	Bronchiectasis ..	852	855	902	742	745	746
33	M	Eczema ..	947	1,035	1,024	692	719	651
29	F	Gall-stone ..	912	915	935	648	663	627
41	M	Peptic ulcer ..	970	1,000	1,000	885	910	1,020
55	F	Rheumatoid ..	795	810	950	783	720	780
40	F	Peptic ulcer ..	970	1,070	1,185	840	855	1,018
28	M	Pneumonia ..	1,092	1,075	1,130	925	970	1,018
32	M	Peripheral artery disease ..	870	—	—	803	—	—
43	F	Hepatitis ..	870	955	1,000	870	980	1,050
61	M	Cirrhosis ..	755	780	780	678	737	750
48	M	Chronic nephritis ..	760	818	870	855	852	812
19	F	" " " " ..	513	496	510	557	572	662
42	M	Polyarteritis ..	322	339	328	305	345	340
64	F	Lupus erythematosus ..	618	645	650	619	640	645
22	M	Chronic nephritis ..	420	435	480	456	472	528
69	F	Myelomatosis ..	775	800	820	797	760	760
50	M	Polyarteritis ..	571	592	588	—	555	599
41	F	Chronic nephritis ..	702	706	745	715	738	725

whether this was due to increasing dehydration and pitressin absorption respectively, or whether it could be accounted for by a diurnal variation in renal function common to both experiments.

**Variations in Osmolarity During Progressive Dehydration**

The osmolarity of urine during progressive water deprivation does not always steadily increase until a maximum is reached; it may fluctuate considerably (Table II). Further,

TABLE II.—Osmolarity and Solute Output Rate of Successive Urines during Progressive Dehydration in Two Subjects

	Hours of Dehydration					
	10-14	14-18	18-22	22-26	26-30	30-34
1. Osmolarity (m.osmols/l.) ..	777	1,004	945	936	1,010	1,002
Output (μ.osmols/min.) ..	576	207	360	243	306	411
2. Osmolarity (m.osmols/l.) ..	843	935	801	767	890	—
Output (μ.osmols/min.) ..	501	375	492	630	369	—

if the test is repeated under the same conditions of dehydration and times of urine collection considerable differences may occur in the two tests (Table III).

These fluctuations in osmolarity doubtless arise from a variety of causes. They are certainly not always caused by fluctuations in posterior pituitary activity or changes in solute (osmolar) output rate. Hart and Verney (1934) have

TABLE III.—Osmolarity and Solute Output Rate of Successive Urines during Progressive Dehydration. The Experiment has been Performed Twice in the Same Subject

	Hours of Dehydration				
	10-14	14-18	18-22	22-26	26-30
1. Osmolarity (m.osmols/l.) ..	952	982	1,330	1,175	1,302
Output (μ.osmols/min.) ..	546	312	324	315	303
2. Osmolarity (m.osmols/l.) ..	730	973	1,065	1,140	1,180
Output (μ.osmols/min.) ..	819	267	399	303	390

reported the occurrence of spontaneous diuresis during dehydration which can be abolished by pitressin; several of our examples, however, occurred in dehydrated subjects when under the influence of pitressin tannate. Similarly, though it is well known (McCance, 1945; Rapoport *et al.*, 1949) that a large increase in solute output is invariably followed by a reduction in osmolarity in the dehydrated subject, such a fall by no means always followed the

twofold to threefold increases in solute output which occurred in our cases. Moreover, a fall in osmolarity not infrequently accompanied a falling solute output rate (Tables II and III).

#### Influence of Urea Concentration on the Urine Osmolarity

Preliminary experiments in dehydrated subjects, in whom the osmolar output had been moderately increased by urea or sodium chloride ingestion, gave the impression that a higher osmolarity was maintained after the urea. To test the validity of this observation the following two experiments were carried out on four normal subjects. On both occasions 5 units of pitressin tannate in oil were injected subcutaneously at 11 p.m. and the bladder was emptied an hour later. A further 2.5 units of pitressin tannate were given at 8.45 a.m. and two morning specimens of urine were saved at 9 a.m. and noon. In the first experiment 25 g. of urea in 250 ml. of water and in the second 15–20 g. of salt in 500 ml. of water were taken at 11.30 a.m. In both experiments two or three further specimens were saved during the next four to five hours. Limited quantities of water were permitted to allay thirst. In this way comparable increases in osmolar output were produced by urea and salt, but the urines contained greatly different concentrations of urea.

Of the 36 observations in Fig. 4, 26 urines with higher than average urea content had a higher than average osmolarity, or vice versa, at any given solute output rate. This confirms our previous impression and suggests that maximum

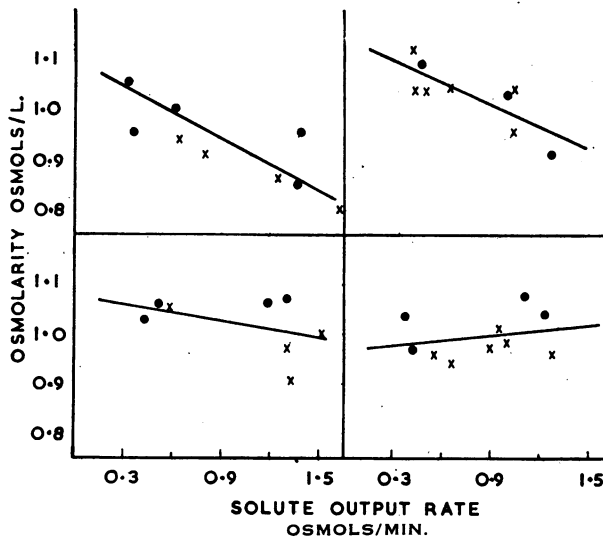


FIG. 4.—Urine osmolarity is plotted against osmolar output in four subjects. Details of the experiments are given in the text. Urines containing a greater than average proportion of urea (urea osmols ÷ total osmols) are marked by a dot, the remainder by a cross. Calculated regression lines are also shown.

osmolarity may partly depend upon the urea concentration, and that a relatively low urea content may therefore be responsible for some of the unexpected falls in osmolarity which occur during progressive dehydration.

(Statistical analysis of these results must take into account the distance of the points from the regression lines and the extent by which the urea ratios (urea osmols ÷ total osmols/l.) varied from the mean. Calculation shows that the average within-person partial correlation of urea-ratio on osmolarity, with solute output held constant, is 0.48, which differs from zero at a significance level of  $P=0.02$ .)

#### Discussion

The measurement of maximum specific gravity as an estimate of renal function is convenient and requires no expensive apparatus. Its value, however, depends on whether specific gravity is a reliable indication of osmolarity, and

whether the concentration, after the period of dehydration chosen, is sufficiently near the true maximum.

The first question is the more easily answered. We have shown that even after careful measurement (and errors of 0.004 or more are not uncommon if corrections are not made for calibration and temperature) urines of like osmolarity may vary in specific gravity by as much as 1017–1022, and 1026–1036 (Fig. 1). The greater the concentration of sodium chloride, and particularly of urea, the lower will be the specific gravity at any given osmolarity. This consideration is sufficient indication that more than one specimen should be tested when the specific gravity is borderline.

The second question, concerning the attainment of maximum osmolarity, is more complicated. We have demonstrated that under English winter conditions 14 to 18 hours' dehydration may produce inadequate concentration in a substantial proportion of cases (Fig. 2). This factor alone may mean in practice that a patient who concentrates to only 1016 after 14 to 18 hours may be capable of reaching a specific gravity greater than 1020 after a longer period of dehydration. For this reason we suggest that when precision is required the period of dehydration should exceed 22 hours; Fishberg's (1939) method of carrying out the test seems one of the most suitable and clinically convenient. The patient has no fluids after midday; the bladder is emptied at night and specimens are saved on waking and on two subsequent occasions during the morning. One of these three samples should have a specific gravity of 1022 or more if renal function is normal.

Unfortunately, it does not appear that pitressin can be used routinely in place of dehydration because, in some patients with good renal function the urine concentration after 5 units of pitressin tannate in oil was definitely inferior to the concentration after 24 to 36 hours' dehydration (Fig. 3). This confirms Taylor *et al.* (1945), though in the present experiments twice the amount of pitressin tannate was used, and osmolarity was measured instead of specific gravity. The failure of this theoretically adequate amount to produce maximum concentration is of considerable interest, but we have not enough information to state whether it is due to insufficient pitressin or to differences in hydration. The close correlation of urine concentrations after pitressin and dehydration in subjects with impaired renal function is also similar to the findings of Taylor *et al.* (1945), and suggests that pitressin may be of value in testing the function of patients with known renal disease when dehydration is considered undesirable.

Clinically, of course, it is often not necessary to know the maximum concentration. If after a short period of dehydration or an injection of pitressin the specific gravity reaches 1022 or more, it is clear that renal function is within normal limits. A long period of dehydration is indicated only when such a concentration is not reached by these simpler measures, or when a precise estimate of renal function is required. Further, if in any circumstances a carefully recorded specific gravity of 1018 is obtained, it is unlikely that the patient's symptoms are due to renal failure, though renal disease is not excluded.

The attainment of maximum osmolarity clearly depends on factors other than the length of dehydration, and poor reproducibility of maximum concentration has been commented on by others (Taylor *et al.*, 1945; McCance, 1945). Such factors include differences in solute output rate and posterior pituitary activity, but unexpected falls in osmolarity which may exceed 100 m.osmols/l. (0.003 specific gravity) sometimes occur during progressive dehydration when the solute output rate is falling and when pitressin has been administered. We have recorded some evidence that maximum osmolarity depends, among other things, upon the urea concentration. In experiments designed to vary the urea content of the urine we found that the osmolarity tended to be higher at any given solute output rate when the proportion of urea in the urine was above average. The following hypothesis is put forward as an explanation.

If the cells at the most distal site of tubular reabsorption of water were partially permeable to urea, then the "effective" osmolarity of the urea in the urine would be less than that measured by freezing-point depression, the difference varying with the urea concentration. Thus, urines with different urea concentrations might have the same "effective" osmolarity (as regards the tubule cells) but different total osmolarity as measured by freezing-point depression. Selective distal permeability to urea might also permit increased water economy without an increase in osmotic work.

If this hypothesis is correct it may mean that specific gravity is fortuitously a better indication of "effective" osmolarity than we have shown it to be of total osmolarity, for when the urea concentration is high the specific gravity is relatively low.

### Summary

The specific gravities of normal urines have been compared with their osmolarities. Considerable variation of specific gravity may occur in urines of like osmolarity.

Dehydration for less than 22 hours gives an unreliable indication of maximum osmolarity.

In some subjects with good renal function 5 units of pitressin tannate in oil produce a urine concentration significantly less than that obtained after dehydration for more than 24 hours. In patients with impaired renal function no difference in concentration is apparent.

Unexplained fluctuations in urine osmolarity occur during progressive dehydration. Possible causes are discussed.

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## SOME CURRENT PROBLEMS IN INDUSTRIAL DERMATITIS

BY

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It seems probable that industrial dermatitis is not decreasing; and the incorporation of industrial insurance in the national insurance scheme has by no means eliminated litigation, although it has altered its mechanism and character. It is often impossible for the physician, whether general practitioner or specialist, to remain entirely aloof from the medico-legal problems which arise. There are three main groups in which the difficulties encountered are mainly or entirely medical.

### 1. Doubtful Diagnosis

Doubt about the original diagnosis arises principally in distinguishing between eczematous industrial dermatitis and eczematous eruptions of "natural" or "constitutional" origin such as the common nummular or discoid eczema. Many authors—for example, Bettley (1948)—have summarized the salient diagnostic features in these cases, particular attention being given to the site of origin of the eruption and its fluctuation in severity according to changes

in or cessation of work. In such a case the family doctor, who has to deal with the case at the outset, is at some disadvantage. He has the advantage, very often, of seeing the onset and studying the earliest appearance of the eruption; but he is usually required to issue a certificate at once and to decide definitely whether the eczema is industrial or not. In some cases it is impossible to do this without a prolonged period of observation, considering the behaviour of the disorder over a period of many weeks or months. To give a firm diagnosis and certificate which has to be altered later gives rise to great dissatisfaction to all parties, not least to the patient, who is likely to feel that his doctor is letting him down. Whenever necessary, therefore, the diagnostic doubt should be very clearly explained to the patient so that he will not feel disgruntled if it is later decided that his disorder is not going to prove a source of income to him. In some such cases, when an early and hasty diagnosis has been amply disproved by subsequent events, the denial of compensation leads to dissatisfaction and a sense of grievance which largely dominates the rest of the patient's life. This unfortunate situation can be avoided by refusal to yield to a demand for exact and firm diagnosis in cases which are still doubtful. In justice to the employer it should be remembered that compensation and damages once paid can seldom be recovered, even though it be proved later that the diagnosis of industrial dermatitis was, from the outset, incorrect.

It is, of course, important to obtain a detailed description of the nature of the patient's work, and of the substances with which he comes into contact; patients are often misinformed about the chemical nature of their contacts, and information should be obtained from the factory manager or chemist if possible. A personal visit to the factory may yield vital information which can be obtained in no other way. No less important is an assessment of possible causes of dermatitis in the patient's home or hobbies. If a housewife develops an eczematous dermatitis it may be, very reasonably, attributed to household detergents; but if, in addition to her own housework, she works as an office cleaner for two hours a day, handling the same detergents, her dermatitis is apt to be labelled industrial, and to be compensated as if her paid employment were the sole cause. The amateur gardener may get dermatitis from handling certain plants; if he is an office worker the correct diagnosis may be reached quickly. But if he is a chemical process worker it is easy to jump to the wrong conclusion and to certify industrial dermatitis; in such a case the diagnosis can often be settled only by a period of observation combined, perhaps, with the results of patch tests.

The prognosis is often of greater concern to the patient than anything else. In general the dermatitis due to a primary irritant clears up in a few weeks, and after some months the skin loses any increased susceptibility to recurrence. Most of these patients can return to their former work provided they take more care than formerly to limit contact with irritants in general, and particularly with the class of irritant which caused their dermatitis.

On the other hand, when there is an allergic hypersensitivity, as can be evidenced by patch tests, the complete avoidance of contact in the future is essential, even if this means permanently abandoning a highly skilled occupation. This type of sensitivity is usually highly specific and relates only to the one substance or perhaps to a group of substances which are closely allied in chemical structure; resistance of the skin to primary irritants in general is not impaired. Unfortunately, many cases, with psychiatric and other factors intervening, do not fall into these clear groups, and in these a period of observation may be required to give the physician a shrewd idea of what the future is likely to hold.

### 2. The Friction Element

In most cases industrial dermatitis gives rise to comparatively little itching, and visible excoriations are unusual. A certain proportion of patients, however, complain of intolerable itching and even provide a demonstration in the