

## GOUT IN A GENERAL PRACTICE

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*Drink wine and have the gout and  
drink no wine and have the gout*

(Proverb quoted by Thomas Sydenham 1624-1689)

Gout was described by Hippocrates, and is known to have been prevalent in Imperial Rome, but neither its causes, nor the mode of action of its specific remedy, colchicine, are yet understood, and even its incidence is uncertain.

The condition was commonly diagnosed in England in the eighteenth and nineteenth centuries. In more recent times, Kerr Pringle (1936) considered that in the preceding forty years its incidence had fallen, and that this fall had been accelerated during the first World War until, five or ten years prior to 1936, the numbers started to rise again. He considered that the factors concerned in the fall included greater diagnostic accuracy, and the privations and restricted war-time diet. Langdon Brown (1938) also referred to the diminished incidence of gout, and stated that the last time he saw any considerable number of cases was when food restrictions were removed in 1919.

Cohen (1955) quotes Gräfe (1931) who observed that after the 1914-18 war, gout disappeared from Central Europe. This was also remarked on by Van Breemen (1938). He considered that the more intensive bodily exercise performed during the war was of significance, in addition to the effects of a starvation diet. Kersley (1958) noted with interest how many people had their first attack of gout after the austerity of the war when they visited the United States or Switzerland.

In the United States, the disease was thought to be uncommon, until the work of Williamson (1920), Hench (1936), and others showed this view to be wrong.

What is the position in this country fifteen years after the end

of the second World War and six years after the end of all food rationing? This paper is based on observations over the past eight years in a large industrial general practice of some 10,000 patients in Coventry.

The writers assess the incidence of gout in their practice and refer in some detail to the diagnostic criteria employed. The gouty subjects are compared in some social respects with a random sample of 135 non-gouty men of 30 years and over, taken from the patients of the practice. It is proposed to call this the social control series, to distinguish it from a separate smaller series of both sexes used for biochemical control purposes. Lastly, as the paper proceeds, certain medical and social features are related with those of published series from hospital and specialist sources.

### Medical Observations

At the end of January 1960, we knew of 16 patients with gout alive in our practice. All were males. Eleven were having acute mon-articular attacks and, of these, five had different sites affected at different times (e.g., a great toe in one attack, and the opposite toe or a foot in another).

Two patients were in the poly-articular stage of the disease. In each the gout had commenced with acute mon-articular attacks.

The three remaining patients suffered from chronic gouty arthritis. In all three, acute attacks at times supervened on the chronic arthritis.

*Sites involved.* Cohen (1955) states that the big toe is involved initially in 60 to 70 per cent of patients. It will be seen from table I that the great toe was involved initially in twelve of our cases. The foot in three cases, and the ankle and wrist jointly in one case were the other sites first involved. None of our patients presented with an acute bursitis.

TABLE I  
FREQUENCY OF SITE INVOLVEMENT BY GOUT

<i>Site involved</i>	<i>Cases where site was involved in initial attack</i>	<i>Cases where site was subsequently affected</i>	<i>Totals</i>
Great toe .. ..	12	3	15
Foot .. .. .	3	1	4
Ankle .. .. .	1	3	4
Wrist .. .. .	1	2	3
Elbow .. .. .	0	4	4
Knee .. .. .	0	3	3
Hand .. .. .	0	2	2
Finger .. .. .	0	1	1
Shoulder .. ..	0	1	1

In all, 15 out of 16 cases had a great toe affected initially or subsequently. McCracken *et al.* (1946) describe a total great toe involvement of 97 per cent, and Brøchner-Mortensen (1941) of 71 per cent.

*The classical attack.* Since Sydenham's enduring, personal account (1696), this has been described by many writers, recently and notably by Wood Jones (1950), a fellow sufferer.

As an acute attack of gout has a limited duration it is more likely to be seen in general than in specialist practice. We have ourselves observed 12 of our patients with podagra.

*The presence of tophi.* Five of our patients had tophi. In three, the tophi were in the ear alone, while one had deposits in the olecranon bursa as well as the ear. In the fifth the tophi were peri-articular, affecting the hand, knee and olecranon bursa. In each case at least 5 years had elapsed between the onset of the disease and the observation of tophi.

Garrod (1876) found tophi in 17 of his 37 cases of gout and, of these, seven had them in the ears alone and nine had them in the ears and in other sites also.

Hench (1936) observed tophi in 53 of his 100 cases, 46 being aural. Kerr Pringle (1936) found tophi in 7 of his 33 patients, while Brøchner-Mortensen (1941) found them in 33 of his 100 cases, 28 being aural.

Kerr Pringle (1936) writes that when tophi are present they are diagnostic, but their absence means little and diagnosis should not be delayed till they appear.

*Biochemical findings.* Because of its non-specific character, we do not propose to do more than mention the erythrocyte sedimentation rate. The Rose-Waaler test was used and found to be negative in two patients where the possibility of rheumatoid arthritis had arisen.

All of our patients had a blood count, urine test, and blood urea estimation to exclude secondary gout due to blood or renal disease (Gutman *et al.* 1956).

Uric acid determinations in whole blood have now been generally abandoned as less reliable than serum or plasma values (Jacobson 1938). The serum uric acid estimations were carried out by the method of Bidmead (1951), slightly modified as to the time of reading. It is essential to success that the colorimetric test be carried out by a skilled technician, accustomed to performing the test frequently, and using only freshly prepared reagents (Talbot 1957*a* and *b*). These conditions obtain in Coventry.

Bauer and Calkins (1959) state that one cannot interpret serum urate values accurately unless the standard for each method has been established by a sufficient number of determinations on normal subjects. In table VI below we include particulars of tests performed on 80 normal males of 30 years and over, and on 30 normal adult females taken at random from our practice. From the results obtained it was clear that we might safely use the generally accepted lower limit for the gouty male of 6.0 mg./100 ml. (Maxwell Lockie, 1957; Talbott, 1957*a*; and Mason, 1959). Cohen (1955) states that values above 5.0 mg./100 ml. suggest, and above 6.0 mg./100 ml. establish, a hyperuricaemia.

Four patients in our series had S.U.A. values of 8.0 mg./100 ml. or more. Six patients were in the range 7.0 to 7.9 mg./100 ml. The remaining six had readings between 6.2 and 6.9 mg./100 ml. It was interesting to find that our most advanced case of chronic gouty arthritis (case 1—see table II) had the lowest average serum urate, 6.2 mg./100 ml., while two of the three patients in the highest range (cases 9 and 10) had these levels determined during their first attacks of gout. In case 9 this level fell from 8.3 mg./100 ml. to 7.2 mg./100 ml. 2 months after the attack and in case 10 the level of 8.0 mg./100 ml. fell to the borderline of 5.7 mg./100 ml. 3 months after recovery from his podagra.

Talbott (1955) points out the importance of withholding uricosuric drugs such as salicylates, aspirin, benemid for 48 hours before determining the serum uric acid level. As patients frequently medicate themselves, they should be questioned on this point. The serum uric acid level may be raised in conditions other than gout, such as chronic renal disease, blood dyscrasias, and lymphoblastomas (Talbott 1957*a*). The incidence of symptomless hyperuricaemia in the general population is unknown, a matter that is discussed further below, and it is quite possible for a chance finding of hyperuricaemia to be made in some other form of arthritis. Maxwell Lockie (1957) at the suggestion of Dr J. J. Bunim, investigated and found hyperuricaemia in at least ten per cent of his patients with rheumatoid arthritis. This runs counter to the experience of Duthie and Davidson (1955) that values out with the normal range are rare in this disease.

To sum up, it is clear that in gout the old rule applies that laboratory findings can be used to support a clinical diagnosis, but not to replace it.

*X-ray changes.* Three of our patients showed what appeared to be specific radiological changes. Case 1 who has had gout for 35 years showed changes in the feet and hands, case 3 with a 29-year history showed changes in the feet, and case 16 with a 13-year history

showed changes in the feet, hands, and elbows. Two further patients case 2 and case 6 in whom the disease had existed for 16 and 14 years respectively, had x-ray appearances of the feet suggestive of gout. There were no signs of gout in the x rays of any of the remaining eleven patients in the series, although in two the disease had lasted for 10 years.

Radiological findings in gout are late and apt to be misleading. According to Talbott (1957a) the characteristic finding in the gouty joint represents circular or oval areas of decreased density with a distinct and sharply defined periphery. Campbell Golding (1955) writes that the diagnosis of gout in the great toe is complicated by this joint being a common site of osteo-arthritis. In the hand, the typical radiological findings are described by Campbell Golding as small punched out erosions due to deposition of sodium biurate in the bony surface near the joint margins, loss of joint cartilage, the absence of osteoporosis, and a spotted appearance of the carpal bones. As the erosions enlarge, they invade more of the cortex of the bone rather than the articular surface.

Erosions of the articular surfaces are also found in rheumatoid arthritis, and when they have a smooth outline, Campbell Golding (ibid.) observes that "the radiologist . . . may have to confess that he is unable to decide whether the lesions are due to gout or rheumatoid arthritis. Fortunately there is usually sufficient clinical evidence to make the diagnosis secure".

*Response to colchicine therapy.* Fourteen of our 16 patients have shown a rapid and convincing response to colchicine in acute gout. The value of colchicine lies in its having no uricosuric effect to frustrate a diagnostic serum uric acid determination, while its therapeutic action is specific for gout alone.

Gutman and Yü (1952) point out that colchicine is not always promptly and completely effective in acute gout, particularly if treatment has been delayed for weeks or months. They observed a refractory response in a quarter of 87 attacks suffered by 46 gouty subjects. Two of our patients failed to benefit from colchicine but responded well to phenylbutazone. Mason (1959) states that phenylbutazone, subject to contra-indications, bids fair to replace colchicine as the drug of choice for the acute attack. Trommer (1957) has written of its use as a presumptive test in the diagnosis of acute gout. The excellent response of our patients to colchicine may be due to the early exhibition of the drug which is possible in general practice.

*Exciting factors.* Patients often attribute the initiation of gouty attacks to a specific article of food or drink. Our patients have variously blamed gravy, patent meat-extract, strawberries, sweet-

bread, and liver. Moreno (1952), however, found that in no case did an attack of gout follow when his patients were persuaded to partake of the article of diet they had incriminated.

Confinement to bed through illness, accident, or operation has been followed by acute gout in one of the patients in the series, and also in two patients now dead, whom we have attended in the past 8 years. Neither is included in the series. One is referred to below in connection with his mode of death.

Exposure to cold and unaccustomed exercise were blamed by one patient, while the trauma of prolonged walking and standing at the motor show was blamed by another. Both patients developed podagra.

None of our patients blamed alcoholic excess for an attack.

*Associated medical conditions.* One of our patients (case 1) has had coronary thrombosis, and during the 8 years of observation two gouty patients not included in the series have died of this condition. There are signs of heart failure in one other patient (case 3). One patient (case 5) is a chronic bronchitic, and another (case 10) has had two severe attacks of phlebitis. Three patients are hypertensive (cases 3, 6, and 9). A patient not in this series died of cerebral haemorrhage in 1953. None of our cases gave a history of allergy.

Of renal conditions, only one patient (case 1) gave a history of urinary stone. He had passed a stone at the age of fourteen, 26 years before his first attack of gout. He is now aged 75, and has had coronary thrombosis. His urea clearance test is 58 per cent of normal and his blood urea 45 mg. per cent. One patient (case 15), in the polyarticular stage of the disease, has persistent albuminuria. As in all the remaining patients, the blood urea of these two men is within normal limits.

Albuminuria was described by such older writers as Garrod (1876) and Dyce Duckworth (1889) as common in chronic gout. Brøchner-Mortensen (1941) found this sign in 21 of his hundred cases. Recently Talbott (1957a) and Maxwell Lockie (1957) have written of its occurrence as an antecedent to acute gout.

### Social Observations

*Sex.* All of our cases were male. In women the disease occurs generally, but not exclusively, after the menopause. Authorities vary in their assessment of the sex frequency. Of the 504 cases of Kuzell *et al.* (1955) in the United States, 26 per cent were post-menopausal women. They believe that acute, painful shoulders are frequently seen in the disease in women, in contrast to the frequency of podagra in men. McCracken *et al.* (1946) also in the

United States found that 9 per cent of patients were women, while Moreno (1952) gives an incidence of 11 per cent in Buenos Aires. Other workers such as Hench (1936), Kinell and Haden (1940), Brøchner-Mortensen (1941) and Bauer and Calkins (1957) state that less than 5 per cent of their cases are women. Talbott (1957a) endorses this low figure and states that tophaceous gout in a female is rare in his experience.

In this country Kellgren *et al.* (1953) reporting a field survey of rheumatic complaints at Leigh encountered only one patient among 1,896 women interviewed who complained of gout in the 5 years prior to the survey. Lawrence (1960) states that no clinical gout in females was encountered in two other surveys at Wensleydale and Rhondda Fach.

*Age of onset and duration of disease before diagnosis.* Gout is, in the main, a disease of middle life. In our series it did not become manifest before the age of thirty. Four patients had their first attack in the decade 30—39 years, eight in the decade 40—49 years, and three in the decade 50—59 years. One patient first had gout at the age of 74. The average age at onset was 44.8 years which is in close agreement with the figure of 47.7 years quoted by Brøchner-Mortensen (1941) and of 46.8 years by McCracken *et al.* (1946).

TABLE II  
AGE AT ONSET AND DURATION OF DISEASE BEFORE DIAGNOSIS

Case	Present age	Age at first attack	Duration of disease before diagnosis
1	75	40	8 years
2	46	30	9 years
3	59	30	20 years
4	57	48	Diagnosed in first attack
5	69	59	6 years
6	56	42	9 years
7	46	41	Diagnosed in first attack
8	41	31	5 years
9	56	55	Diagnosed in first attack
10	75	74	Diagnosed in first attack
11	41	35	6 years
12	46	44	Diagnosed in first attack
13	43	41	Diagnosed in first attack
14	58	56	1 year
15	45	42	3 years
16	62	49	6 years

Table II also records the lapse of time in our series between the first attack of gout and its diagnosis. An immediate clinical diagnosis, subsequently confirmed, was made in six patients. Case 3 on the other hand had 20 years elapse before his condition was

diagnosed. The average length of time between onset and diagnosis was 4.6 years.

In the American literature, the corresponding period given by Hench (1936) for his series was 15 years, while Kinell and Haden (1940) give an average of 7 years and McCracken *et al.* (1946) of 10 years.

No merit can be claimed for early diagnosis in this series. The general practitioner is the first doctor to see the patient, and, if he keeps gout in mind, he is in the position to make an early presumptive diagnosis. Conversely, long delays do not necessarily mean that the doctor has been at fault. A stoical patient may tide himself over a gouty crisis using household analgesics, and not consulting his doctor. Several of our patients did so, early in the course of their disease. Since years may elapse between attacks, there may be considerable delay before medical aid is sought. Case 2 had 9 years pass between his first and second attacks, while case 8 had 5 years between his first and second, and 3 between his second and third attacks. Between these crises the affected parts had appeared normal, and the patients had no complaints.

*Social status and occupation.* Coventry is a prosperous industrial city where employment is full, if sometimes precarious, and wages on the whole are high. With the exception of old-age pensioners, most of the patients in our practice have enjoyed a good standard of living in the past 8 years.

Of the 16 patients with gout, one had a junior executive post in a factory. There were two skilled, ten semi-skilled (one a part-time, licensed-club secretary), and no unskilled factory workers among them. There were, besides, one butcher, one publican, and one retired painter and decorator to represent the traditional callings of the gouty.

Our patients are classified in social groups after the method of the Registrar General and compared in table III with the 135 non-gouty males of 30 years and over in the social control series.

TABLE III  
SOCIAL CLASSIFICATION OF 16 GOUTY AND 135 NON-GOUTY MALES

<i>Social group</i>	<i>I</i>	<i>II</i>	<i>III</i>	<i>IV</i>	<i>V</i>
Gouty (16)	0	3	3	10	0
Non-gouty (135)	0	5 (4 per cent)	34 (25 per cent)	78 (58 per cent)	18 (13 per cent)

It will be seen that although 13 per cent of the control series are unskilled workers (group V) compared with none in the gouty



series. As against this, the highest social group represented (group II) make up only 4 per cent in the control series. Sydenham (1696) wrote that "this disease . . . kills more Rich than Poor, more Wise than Fools". Although our figures appear to be consistent with this view, it must be realized that the distribution would be radically altered, if only two gouty patients belonged to a different social group.

*Habitus, diet, and alcohol.* Seven gouty patients were obese, two grossly so, and five were heavily built. Thus three-quarters were above average weight. In the social control series only 4 per cent were obese, and 17 per cent of heavy build, a total of only 21 per cent above average weight. The tendency to obesity and sthenic constitution in gout has been well remarked in the literature. In recent years Brøchner-Mortensen (1941) found 71 per cent of his patients overweight, while Kuzell *et al.* (1955) state that this applied to more than half of their cases.

Since people vary greatly in their views of what is an average helping of food, it was not possible to assess accurately by questioning, the dietary intake of our patients. There are, however, obvious and accurate measures of alcoholic consumption. The staple drink of our patients is beer. Spirits and wines are drunk rarely, and on special occasions.

Garrod (1876) writes: "There is no truth in medicine better established than the fact that the use of fermented liquors is the most powerful of all the pre-disposing causes of gout; nay so powerful that it may be a question whether gout would have been known to man-kind had such beverages not been indulged in". Such strong views are not now generally held. Yet Williamson (1920) found that among 116 patients admitted with gout to a Chicago charity hospital, more than half were heavy drinkers, four were abstainers, nine occasional and 41 moderate drinkers. Of Brøchner-Mortensen's 100 patients (1941) 13 were abstainers and 17 drank more than ten bottles of beer daily. Cohen (1955) writes that the belief that alcoholic drinks induce gout is founded on ill-recorded data. In his series of 100 patients the average and the distribution of intake did not differ materially from a similar sample in the non-gouty, and 30 per cent said they took only an occasional drink or were total abstainers. Cohen adds that patients' statements in this connexion are not however always reliable.

In table IV we compare the beer consumption of our gouty patients with that of the social control series. These figures suggest that the gouty are the heavier drinkers, although individual patients are abstemious or partake only moderately of alcohol. To eliminate possible errors due to the different distribution of the social groups

in the series, a separate analysis of the beer consumption of the largest common social group, group IV, has been included in the table. The results of this comparison do not differ greatly from those of the whole series. Again, it is necessary to remember the small number of gouty subjects.

TABLE IV  
DISTRIBUTION OF BEER CONSUMPTION IN GOUTY PATIENTS AND CONTROL SERIES

<i>Weekly beer consumption</i>	<i>All gouty patients</i> (16)	<i>Whole control series</i> (135)	<i>Gouty patients in social group IV</i> (10)	<i>Controls in social group IV</i> (88)
		per cent		per cent
Abstainers or near abstainers ..	1	28 (21)	nil	17 (19)
½—5 pints ..	3	40 (30)	3	30 (34)
6—15 pints ..	3	46 (34)	2	26 (30)
16—25 pints ..	6	10 (7)	3	8 (9)
26 pints and over ..	3	11 (8)	2	7 (8)
Average age ..	55 years	52 years	52 years	52 years

### Family History

Of our 16 patients, two are brothers, and a history of gout was found in the family of five others. Thus, there is a positive family history in seven out of 16 cases. Of the 12 affected relatives, six were dead, two fathers and a mother, a paternal and a maternal grandfather, and a maternal uncle. Of the relatives alive there were five gouty brothers, but no gouty sister. There was one gouty son, but no gouty daughter or aunt. None of the relatives of the 135 men in the social control series was known to be gouty.

Among the older writers, Scudamore (1823) found a family history among the parents, grandparents, uncles and aunts of 60 per cent of his 522 patients, while Garrod (1876) found such a history in the antecedents of half of his hospital and three-quarters of his private cases.

More recently, Kerr Pringle (1936) elicited a gouty family history in nine of his 33 cases, while Hill (1938) gives a figure of 45 per cent of his male patients. Abroad, Brøchner-Mortensen (1941) reports a family history of gout in only 11 per cent of his patients, while, on the other hand, Talbott (1957*a*) found a family incidence in more than half his cases at the Buffalo General Hospital. Talbott stresses that the greater the interest of the physician the higher the percentage of (positive) family histories found. The occasional tendency to suppress a family history of gout as akin to alcoholism and therefore shameful, should be kept in mind.

We were able to interview and obtain serum uric acid estimations in a total of 53 adult relatives of 13 of our gouty patients, comprising 28 men and 25 women. Besides the six living relatives with gout, single instances of symptomless hyperuricaemia (S.U.A.>6.0 mg./100 ml.) were found in the families of three patients, two of whom had no relative with overt gout.

That hyperuricaemia is to be found in some symptomless relatives of gouty patients was noted by Folin and Denis (1915), Jacobson (1938), and Talbott (1940). Its connexion with the inheritance of gout has been variously interpreted (Smyth *et al.* 1948, Stecher *et al.* 1949, Hauge and Harvold 1955).

Table V shows the composition and relationship of the relatives and of the incidence of gout and hyperuricaemia among them.

TABLE V  
RESULTS OF INVESTIGATION OF THE LIVING RELATIVES OF 13 GOUTY PATIENTS

<i>Relationship</i>	<i>Total</i>	<i>Gouty</i>	<i>Hyperuricaemic</i>
Father .. ..	3	0	1
Brother .. ..	17	5	1
Son .. .. .	8	1	0
Mother .. ..	3	0	0
Sister .. .. .	15	0	1
Daughter .. ..	7	0	0

As indicated earlier in this paper, we have carried out serum uric acid estimations on 80 normal men aged 30 years and over, and on 30 normal adult women, both groups taken at random from our practice. One man and one woman were found to have symptomless hyperuricaemia.

Reference to the occurrence of symptomless hyperuricaemia in the absence of a family history of gout has been made by Hauge and Harvold (1955) and Talbott (1957a). The incidence is unknown but Talbott reports a series of 1,500 samples of blood obtained at routine examinations of college students aged 18 to 21 years. Of these 68 were found to have levels higher than 6.0 mg./100 ml., most of which were persistent.

In table VI we compare the serum uric acid levels of our own biochemical control group with the levels found in the relatives of our gouty patients.

It will be seen that the serum uric acid level of the male relatives is significantly higher than that of the control group, whereas between the female groups there is no significant difference.

Hauge and Harvold (1955) in a large scale investigation found

that the serum uric acid level of both male and female relatives were significantly higher in all age groups than the levels of their control series. Like Stecher *et al.* (1949) they demonstrated a significant increase with age in the serum urate levels of the female relatives. No such increase could be shown in the male relatives or male and female controls.

TABLE VI  
SERUM URIC ACID ESTIMATIONS—SUMMARY AND COMPARISONS

Sex		No. of subjects	Mean mg./100 ml.	S.D.	Average age
Male	{ controls ..	80	3.84±0.12	1.05	51
	{ relatives ..	28	4.74±0.25	1.33	50
Female	{ controls ..	30	3.19±0.25	1.39	51
	{ relatives ..	25	3.19±0.21	1.06	49

### Incidence

There are 71 male and 36 female West Indians on our National Health list of patients. No gout was observed among them.

Negroes were thought to be immune from gout, but Garrod (1876) quotes a Dr Quarrier who stated that when serving as sailors in the British Navy they were apt to become gouty. The disease was not demonstrated beyond doubt until Cohen (1948) observed its occurrence in three negro brothers, albeit of mixed descent. Further cases have been reported since then, both male and female.

We have 468 male and 59 female Indian and Pakistani patients. No proved gout was found among them, but in 1954 a provisional diagnosis was made in the case of a middle-aged, male Indian. Language difficulties prevented a satisfactory history being elicited, and the patient left the district before the diagnosis could be confirmed. The only reference to gout in India, that we could trace, was the much quoted paper by Das Gupta (1935), concerning a community numbering forty to fifty thousand in Nepal. We understand that the Nepalese belong to a different ethnic group from our patients. Das Gupta reported a remarkably high incidence of gout—between 6 and 7 per cent. He described it as common in women and children. In one of his cases the diagnosis was regarded as being confirmed by the presence of uric acid crystals in the urine, and he was led to conclude that gout was an infectious disease antagonistic to tuberculosis.

If we exclude our immigrant patients we find that the 16 cases of gout have arisen in a total of 9,655 patients, of whom 4,743 are male and 4,912 female. The overall incidence is therefore almost

0.2 per cent. Narrowing the field to 3,703 males of 15 years and over the incidence is 0.4 per cent. In the age group of 30 years and over, in which we actually found gout, there are 2,720 males and the incidence rises to 0.6 per cent.

### Discussion

Our patients number approximately 3.5 per cent of the population of Coventry. According to our calculations, we have in the practice proportionately 2.4 per cent more men of 15 years and over, and 1.4 per cent more men of 30 years and over than the population of England and Wales as estimated by the Registrar General at 30 June 1959. Possibly, in the local context these small differences might be a little greater since our practice does not extend in strength into the housing estates built since the recent war. These estates house a high proportion of children. The difference between the age distribution of patients in our practice and the general population from which they are drawn will inflate slightly our figure for the overall incidence of gout, but its affect upon the incidence among men of 30 and over will be for all practical purposes negligible.

The question arises whether genetic factors favouring gout are more or are less common locally than elsewhere. In fact, nine of our 16 patients were born in or within 12 miles of Coventry and seven were born in other parts of the country. Of the social control series 54 had been born in or near Coventry and 81 elsewhere. (The more than four-fold increase in Coventry's population this century owes much to a large influx of workers from other parts of the British Isles.)

Wages tend to be high locally; it is reasonable to believe that as in other prosperous parts of the country, more will be spent on food and drink than in less favoured areas.

The literature on the incidence of gout is scanty. A few references concern the percentage of hospital admissions due to the disease, but, since admission is rarely required, this gives no indication of its prevalence in the general population. Several authorities have estimated its frequency among patients attending rheumatism clinics. Hench (1936) reported this as five per cent, and Kinell and Haden (1940) as 3.7 per cent. Smyth *et al.* (1948) found that about four per cent of patients visiting the Rackham Arthritis Unit at Ann Arbor were victims of gout. Assuming that gout occurred with the same frequency among that fraction of civilians (*viz.*: 2.2 per cent) who were found in a nation-wide canvass of 9,000 families in the United States to have histories of "chronic arthritis and rheumatism" (Collins, 1933), they arrive at an estimate of the incidence of gout in the United States of 0.088 per cent. Since early attacks of gout might not be classified as "chronic arthritis

and rheumatism" it seems possible that this figure is an underestimate. Talbott (1957*a*) describes a figure of 300,000 cases of gout in the United States (approximately 0.2 per cent of the population in 1950) as probably an underestimate. We are not aware how this figure was arrived at, but note with interest that we have found the same overall incidence.

Apart from visits connected with the compilation of this paper only four of our patients had to attend hospital or rheumatism clinic for their gout.

In this country Kellgren *et al.* (1953) carried out a survey of rheumatic complaints among the 48,714 inhabitants of Leigh. A random sample was taken of every tenth house and a specially trained, medico-social worker interviewed all individuals normally resident there who were 15 years and over, with regard to rheumatic complaints during the previous 5 years. All individuals with such complaints were subsequently visited by a physician, who elicited the history and carried out a simple clinical examination to exclude non-rheumatic complaints and to classify the rheumatic conditions. They found five men in 1,619 and one woman in 1,896 who complained of gout in the previous 5 years. The incidence in males of 15 and over in this series was therefore 0.31 per cent. This compares with the incidence determined by us in a different way, of 0.4 per cent in 3,703 males of the same age group in our practice.

Lawrence (1960) states that he confirmed the Leigh incidence by a smaller survey in greater detail. He adds that similar surveys in other parts of the country showed the following prevalences:

Wensleydale	Males	0.5 per cent
Rhondda Fach	Males	0.0 per cent

Thus, while the Yorkshire survey showed a higher incidence than in our practice, no gout at all was encountered in the Welsh survey. Information from other areas would be of great interest.

### Summary and Conclusions

Sixteen male but no female patients in a large industrial general practice are known to have gout.

The cases are described, and the paramount importance of the clinical findings in establishing the diagnosis is emphasized. The general practitioner who sees the acute attack at its height is privileged in this respect.

Fourteen patients responded promptly to colchicine. In general practice this, in the writers' view, remains the drug of choice for the acute gouty crisis.

There is a striking preponderance of overweight individuals

among the gouty subjects.

Seven patients give a positive family history for gout, and two others have each one close relative with symptomless hyperuricaemia. Fifty-three relatives of 13 of the patients were interviewed and investigated for gout and hyperuricaemia.

The social status and habits of the patients, including their consumption of alcohol, are compared with those of a control series.

Four of the 16 patients required reference to hospital.

The incidence of gout in the practice is 0.2 per cent overall, 0.4 per cent in males of 15 years and over, and 0.6 per cent in males of 30 years and over.

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“ . . . Gouty persons should therefore make it a rule in this particular, to drink such liquors as will not inebriate, if drunk in large quantity, or injure the stomach by their chillness. Of this kind as I before hinted is our small beer, and in other countries a similar liquor may be made by diluting wine well with water. As to water alone, I esteem it crude and pernicious, and have found it so to my cost; but young persons may drink it with safety, and it is at this day the common drink of the greatest part of mankind, who are happier in their poverty than we are with all our luxury and abundance. This is confirmed by the great multitude of diseases with which we are afflicted upon this account, as the stone, gout, apoplexy, palsy, etc., besides the injury done to the mind in being driven from its natural rectitude, by the disturbance which the fiery spirits of such liquors, together with the animal spirits which assist the thinking powers occasion, by volatilizing the mind too much, and suggesting vain and idle notions, instead of solid and weighty reasonings, and thus at length rendering us drolls and buffoons instead of wise men; between which the difference is almost as great as between a substance and a shadow. But enough of this . . . ”

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