The fate of the 200-odd cases examined may be of some interest. The following figures are approximate, but fairly accurate:

40% of them were dismissed by me. Half an hour or more elapses between the time of arrest and the time of examination. Arrest has a sobering effect. All these dismissed cases showed evidence of having had a considerable amount of alcohol, with the exception of three, all of whom were diabetics suffering from overdosage of insulin. This is the only condition I have come across which simulates drunkenness closely. Of the 60% certified by me about one-third plead guilty, of the remainder about one half elect to be tried by the magistrate; the others ask for trial by jury at the County of London sessions.

The magistrates find about 75% guilty. The court of sessions finds about 75% not guilty.

Even when the accused wins his case at sessions it is expensive; when he loses, it is very expensive, and the penalty is severe. Even so the gamble is well worth while. This 75% acquittal is a curious phenomenon. Cases which appear to be guilty beyond any possible doubt are frequently thrown out by the jury. It may be that the reason for this is to be found in the wording of the charge: "Being so much under the influence of alcohol as to be incapable of having proper control of a motor vehicle." Many cases are arrested as they are about to start their cars; others are able to show that they have in fact driven their cars some distance without mishap. In the first case there is no absolute proof that they were incapable; in the second it would seem that they were capable. Magistrates recognize the flaw in this sort of reasoning: juries apparently do not.

I have no legal training, and my opinion is of little value; but I believe that there would be more convictions and justice would be better served if the charge were amended to something like this: "Being in charge of a motor vehicle while so much under the influence of alcohol as to have one or more faculties noticeably deranged thereby."—I am, etc.,

London, W.9. JAMES GOSSIP.

Anticoagulants in Leukaemia

SIR,—Sir Lionel Whitby in Part II of his Refresher Course on leukaemia (December 29, 1951, p. 1573) has painted a picture of this disease which is easy to see and understand, and for which I am grateful.

In the treatment of chronic leukaemia there is, I suggest, one omission. He does not mention the use of anticoagulants in those cases where the thrombotic manifestations are prominent. In two of the three cases of chronic leukaemia which have come under my care mesenteric thromboses occurred, in one case agonizing in the intensity of pain. These were completely relieved of thrombotic signs and symptoms by keeping the prothrombin level around 65% of normal by the use of dicoumarol.

Sir Lionel mentions the distressing symptom of priapism and the possibility of surgical treatment. One of my patients developed this symptom and was relieved by anticoagulant treatment. I thought these observations might be of use to others.—I am, etc.,

Leeds, 8. R. A. MURRAY SCOTT.

Naevi in Pregnancy

SIR,—The letter of Mr. John Stallworthy (December 1, p. 1341) referring to the occasional appearance and growth of naevi associated with pregnancy prompts me to record a case I saw a few years ago.

The patient, a married woman with one child, noticed shortly after parturition the appearance of a small red papule in front of and just above the tragus of the right ear. There had been neither naevus nor telangiectasis on this site before or during pregnancy. The papule, which gradually increased in size, was itchy and bled when scratched. A few months later similar but smaller papules appeared on the tip of the helix of the same ear. Over the next two and a half years further lesions made their appearance while those that had appeared originally continued to increase in size. The new lesions were situated behind the right ear and on the skin of the face in front of the tragus.

There were no other naevi on her body, but she stated that at the age of 9 a red mark, unrelated to trauma, had appeared on the skin of her face in front of the right ear. This area, which was approximately 1½ in. (3.7 cm.) long by ¼ in. (0.6 cm.) broad, was telangiectatic and was noticed at the time of the examination.

On examination it was seen that the tragus and upper part of the helix of the right ear had been replaced by tumour tissue, and there were two lesions on the skin of the face near the ear. A similar lesion was present over the mastoid area behind the ear. The growths were not large, had a port-wine colour, were firm but rather spongy in texture, and blanched on pressure. General examination revealed no abnormality and a blood picture was normal apart from a slight degree of hypochromic anaemia. The W.R. was negative. A biopsy report stated that the lesion was a simple tumour of either lymph or haemangiomatous type, or both, with a heavy chronic inflammatory type of perivascular exudate in which eosinophils were outstanding.

The patient was treated with *x*-ray therapy with considerable regression of the tumour, but because of my National Service duties I was unable to follow the case further.

Although there are few references in the literature to the spontaneous appearance or growth of naevi in association with pregnancy the condition has been described by Whitfield (*British Encyclopaedia of Medical Practice*, 1937, **5**, 149) under the title "Progressive Angiomas of Pregnancy." He records a case of his own and quotes Gans as having figured a section of one from the cheek of a girl two months pregnant. Along the same lines Enticknap (*British Medical Journal*, 1946, **2**, 51) recorded a fatal case of angioblastoma occurring in the breast of a woman three months pregnant, which he considered to have arisen from a pre-existing simple haemangioma.

I would like to thank my former chief, Dr. A. D. McLachlan, physician, Diseases of the Skin, Western Infirmary, Glasgow, for permission to record the above case.

—I am, etc.,

Glasgow. W. O. THOMSON.

Cough Fracture in Late Pregnancy

SIR,—Dr. N. Wynn-Williams in his article on cough fracture (December 22, 1951, p. 1494) refers to a paper by myself and others on this condition in late pregnancy (*British Medical Journal*, 1949, **1**, 135). We described four cases and were able to find six others in the literature. The notable feature was that, in all, the left lower ribs were involved. It was our opinion that this fracture was relatively common in late pregnancy, and probably largely unrecognized. Dr. Wynn-Williams suggests that this fracture and pregnancy are a "fortuitous association" and not a clinical entity.

In the last two years I have seen four more examples in pregnant women, all left sided and affecting the lower ribs. No special effort was made to find them, other than a casual request to one or two of the assistants in the antenatal clinic to keep a look-out. Two were discovered in this way. The third came to light after I had overheard a conversation between a doctor and his wife about a patient requesting a visit. The fourth came to me because of chest pain of two years' duration.

I agree with Dr. Wynn-Williams that cough fracture unassociated with tuberculosis is common enough, and have seen six other such patients in the last four years; however, in these there is no such clear-cut tendency for the left side to be affected as there seems to be in our series. We discussed some of the possible causes of this left-sided predominance in our paper. The fact that all cases so far reported have occurred in the last three months of pregnancy cannot easily be dismissed as due to chance.—I am, etc.,

Ipswich. J. W. PAULLEY.

Skin Resistance to Ultra-violet Light

SIR,—Dr. Klaus A. J. Järvinen, of Helsinki (December 8, 1951, p. 1377), has reported some interesting observations on the effect of systemic cortisone therapy on the reaction

of the skin to ultra-violet light. He has found that it doubles the minimal erythematous dose, and he also states that when the same patients were submitted to six minutes' exposure before, during, and after cortisone therapy the effect of cortisone was to promote pigmentation and, as one might therefore expect, to diminish the harmful erythematous and vesicular reaction.

From these simple and precise observations Dr. Järvinen draws the somewhat surprising conclusion that in this respect cortisone is not increasing the resistance of the skin to the injurious effect of ultra-violet light, but rather suppressing the normal protective mechanism by which this resistance is augmented and maintained. This can be true only if we regard erythema and vesiculation as a protective mechanism and pigmentation as an injurious effect. Since the opposite is a more prevalent teleological interpretation of the phenomena of photosensitivity, I would like to submit that his experiments indicate a true increase in the resistance of the skin to ultra-violet light as exemplified by the doubling of the minimal erythematous dose and that they also suggest a stimulation of the protective mechanism if we are entitled to regard pigmentation in this light.—I am, etc.,

Merck and Co., Ltd., Montreal. J. H. LAURIE, Medical Director.

Night Pain with Fractured Clavicle

SIR,—Within the last 10 weeks I have had two cases in children under 5 of fracture of the outer third of the clavicle. Clinically they have been misleading in that there is no deformity; there is practically no loss of function—the child has been able to dress and undress itself apparently without pain, including pushing the affected arm into a jersey, etc.; there is no apparent general disturbance. There has, however, been one feature common to both cases, and that is pain at night in bed. I suppose the anatomical structure maintains position while erect, but on lying down and turning over in bed pressure is brought on the fracture, or is it relaxation of muscles while asleep? Both cases were found on *x*-ray examination.

I wonder if any other people have found this pain-in-bed story connected with fracture of the outer third of the clavicle in young children.—I am, etc.,

Hadlow, Kent. J. B. MARSHALL.

Treatment of Anthisan Poisoning

SIR,—Your report of a further two cases of "anthisan" poisoning in children (December 22, 1951, p. 1530) prompts me to take out of their context some of the results of an investigation into the cerebral effects of histamine. This investigation, which will be published eventually, necessitated the administration of anthisan in toxic doses to a large number of animals. The findings were so uniform in all the species examined (mouse, rat, guinea-pig, and rabbit) as to favour the view that they might also apply to human beings, especially as the clinical features of anthisan poisoning are very similar in man and animals.

Anthisan is a central convulsant—i.e., the convulsions seen in poisoning are not a secondary effect of interference with respiration, as is sometimes suggested, but depend on a direct action of the drug upon nervous centres. The average convulsive dose is very near the average lethal dose, and the gravest view must therefore be taken of any case presenting convulsions. Death is the result of respiratory depression, which occurs after the animal has passed through the convulsive stage. With slow absorption of the drug the central nervous system shows successively depression, excitement and increased reflex activity, tonic-clonic convulsions, coma, and respiratory depression. These stages correspond to the severity of the intoxication, and all four stages are therefore only seen with fatal or near fatal doses. Anthisan may be classified with atropine as a "deliriant narcotic," and their similarity of action is not fortuitous.

Orthodox anticonvulsant drug therapy is likely to prove disastrous in anthisan poisoning. The administration of ether, chloroform, soluble barbitone, or "nembutal" to experimental animals, while leading to a prompt cessation of the convulsions, greatly diminishes the survival rate by hastening and deepening the stage of respiratory depression. It should be remembered that convulsions, unless of great severity and long duration, are not a life-threatening symptom in themselves. Their abolition is therefore not usually a legitimate therapeutic aim unless it is achieved by means which relieve rather than deepen the underlying intoxication of the central nervous system.

The effect of histamine on anthisan poisoning is markedly diphasic. In relatively small doses it has an important anticonvulsant and life-saving action, while in larger doses it increases mortality. With simultaneous administration the greatest life-saving effect is produced by an anthisan:histamine acid phosphate ratio (weight for weight) of 4:1 in the guinea-pig and 1:1 in the mouse, using average lethal doses of anthisan. To translate this finding into terms of human therapy is extremely difficult, not only because of species difference but also because poisoning in human cases is usually by mouth and the amount swallowed is rarely known. Moreover, animal experiments suggest that it is very difficult to dislodge anthisan from its central receptors, and histamine therapy becomes successively less effective as the interval between anthisan and histamine administration grows. In spite of this, a cautious trial of histamine in these desperate cases can be recommended, especially as absorption of anthisan is often still proceeding when they are first seen by a physician. If histamine is tried as an antidote, steps must be taken to protect the patient from the acute and severe hyperchlorhydria which will ensue.

Morphine occupies theoretically an ambivalent position in the treatment of anthisan poisoning. As a respiratory depressant it would seem to be contraindicated, as a histamine-releasing drug it might be an ideal therapeutic agent. Preliminary trials in mice have not yet given an unequivocal answer, but I think that a clinical trial of its efficacy as an antidote would be justifiable. Judging from animal experiments, it is certainly preferable as a sedative to barbiturates in these cases.

Adrenaline and atropine hasten death from anthisan poisoning in experimental animals.

On these imperfect data we may base the following scheme of treatment:

1. Wash out the stomach.
2. Treat the patient in a darkened room and avoid all unnecessary stimulation.
3. *Do not administer inhalation anaesthetics or barbiturates.*
4. If drug therapy seems desirable, try morphine or histamine, the latter in doses no greater than the amount (weight for weight of histamine acid phosphate) thought to have been absorbed by the patient.
5. Watch for respiratory depression, and give respiratory stimulants if it supervenes.
6. Take any supportive and eliminative measures which are not in conflict with the above.

An improved outlook in anthisan poisoning will result from the conscientious reporting of all cases with full notes on the treatment, even if unsuccessful. Meanwhile a certain number of lives may be saved by attention to these (largely negative) injunctions.

These observations probably apply to a greater or lesser extent to all antihistamine drugs. They are all central convulsants in toxic doses, and it is to be assumed that the mechanism of production of toxic manifestations is the same. I cannot conclude these remarks without making reference to the great help which I have received from Dr. Roy Maxwell, of May and Baker, Ltd., both in the supply of drugs and in the giving of expert advice.—I am, etc.,

Edinburgh. ERICH GEIRINGER.

Discovery of the Formol Gel Reaction

SIR,—I am very much indebted indeed to Colonel W. C. Spackman for his letter (October 13, p. 911) about the discovery of the formol gel reaction, as it provides a very good opportunity for me to give a short account of the history of this discovery in the light of real facts, and not as presented in some publications.

In September, 1920, while I was a medical student and assistant at the Diagnostic Laboratory of Dr. Gaté, of the Bacteriological Institute of Lyons (France), there was a violent explosion that destroyed part of the Institute, including the refrigerator in which I used to keep the mixture of positive and the mixture of negative