It was unnecessary for Professor Conn to select cases of primary aldosteronism from our published results,⁶⁻⁸ since we had reported these conclusions in the same group of cases—in Professor Conn's presence—more than a year before.⁹ Professor Milne's case was not one of these. Our recent paper⁹ was a progress report on a patient described in some detail earlier.⁶ We said of this case in 1963 that the association of low plasma-renin concentration and increased aldosterone secretion suggested an aldosterone-producing adrenal tumour, and that we were studying the effects of spironolactone treatment before removing the tumour.⁶ It thus seems obtuse of Professor Conn to misconstrue² the intention of our paper⁹ as "final proof" of his more recent "thesis."

We should further make it clear that neither we nor Professor Conn and his colleagues can rightly claim priority for the prediction of low plasma renin in primary aldosteronism, or for the removal of a tumour from a patient investigated in this way. Any such credit belongs to others.^{10, 11}—We are, etc.,

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Urinary Infection in Childhood

SIR,—We would like to add our support to the comments of Dr. H. Braude, Professor J. O. Forfar, and Dr. J. C. Gould (28 November, p. 1393) on the paper by Dr. Jean M. Smellie and her collaborators (14 November, p. 1222). We also have found that growths of potential pathogens exceeding 10^5 organisms per ml. (loop/blood-agar surface count) in bag-urine specimens from babies are frequent in the absence of significant pyuria or other urinary abnormalities which would support the diagnosis of urinary infection.

Our colleagues from Edinburgh do not indicate how they are dealing with this problem, and we would like to describe two urinary investigations which we have found helpful in making a positive diagnosis of urinary infection in some babies.

The first is to do a cell-count in a white cell counting chamber on the unspun specimen.^{1, 2} In contrast to an estimation of the number of cells per high-power field in the centrifuged deposit this method eliminates the

main technical variables, although not that due to different rates of urine secretion. A count of 200 leucocytes per c.mm. or more in a clean specimen is good evidence of urinary infection at any age. Also a count of five or more red cells per c.mm. is abnormal and warrants an examination of further specimens for more definite evidence of urinary infection or other urinary-tract abnormality.

The second method we have been using over a shorter period is to dilute the specimen 1 in 100 and spread two separate loopfuls (each delivering 2 c.mm. of urine) on half-plates of blood agar. In this way an estimate of bacterial counts in the range of 10^5 to 10^7 per ml. is obtained. Our present data suggest that counts of 5×10^6 per ml. or over indicate urinary-tract infection even in bag-urine specimens.

Finally, when the urinary findings are equivocal the value of examining repeated specimens before starting chemotherapy cannot be overstressed. We consider that in spite of the difficulties of diagnosis in babies catheterization should continue to be avoided because of the danger of introducing infection in this age group as well as at other ages.—We are, etc.,

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Asymptomatic and Significant Bacteriuria

SIR,—Kass¹ uses the expression "asymptomatic bacteriuria" in respect of those patients with bacteriuria who "were not regarded by those responsible for their care as having active infection of the urinary tract." Kass was working with out-patients of the Boston City Hospital. Other authors use other equally local and subjective criteria, if indeed they attempt to define the condition at all. Clarity would be gained if authors using the term asymptomatic bacteriuria were to state which of the local and general symptoms and signs of inflammation of the urinary tract were expressly inquired after and found to be absent. As a basis for discussion may I suggest that the following should be looked for: pain on micturition, frequency, urgency, double micturition, dyspareunia, hypogastric pain or tenderness, renal pain or tenderness (elicited by percussion over the renal angle), abdominal bloating, pallor (common), a red, glossy complexion (less common), frontal headache, depression, debility, insomnia, epigastric discomfort, nausea, bad-smelling urine, and an ammoniacal smell to the patient. In children other symptoms are important—for instance, anorexia, bouts of vomiting, constipation, failure to gain weight, irritability, and screaming attacks.^{2, 3}

Significant bacteriuria is usually deemed to be present when there are 100,000 or more organisms per ml. of urine. The significance of this bacteriuria is a bacteriological one, and, while it is perfectly legitimate for bacteriologists to isolate a group of patients

with this urinary feature for special investigation, clinicians should not accept significant bacteriuria as being synonymous with inflammation of the urinary tract. Many (possibly most) patients presenting with inflammation of the urinary tract do not have significant bacteriuria, though they are not otherwise clearly distinguishable from those which have. For the clinician, bacteriuria is something of a red herring, and to him inflammation of the urinary tract is a disease of urinary tract tissues, which may or may not be accompanied by loss of organisms in the urine. We do not know if the presence or absence of bacteriuria implies a difference in kind between these two classes, or whether they are to be regarded as "open" and "closed" cases of urinary tract infection similar in principle to "open" and "closed" cases of pulmonary tuberculosis.—I am, etc.,

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Alcohol in the Driver

SIR,—In an editorial (27 February, p. 540) you refer to the B.M.A. Council's approval of the B.M.A. Alcohol and Road Accidents Committee recommendation "making it an offence for a person with a blood-alcohol concentration in excess of 80 mg./100 ml. to drive a motor vehicle on the public highway."

There are two threshold levels of quite a different order as regards performance impaired by ethanol, reasonable allowance being made for dose-response variations. The first threshold, the higher one, indicates the level above which absence of impairment is unthinkable in the majority of subjects. The second threshold indicates the level above which the safety of road users would be measurably reduced. The upper threshold is often clearly associated with drunkenness, which in turn is associated with serious social and moral implications. (However uncertain pharmacologists are as regards the meaning of drunkenness, there is every certainty that, used or misused as a label, it may be most damaging.) On the other hand, the lower threshold is not, and should not be, associated with drunkenness in normal circumstances. I think it should be stressed that the recognition of two sharply differentiated offences,¹ one comparable to a serious moral offence and the other to a speed-limit infringement, is essential. Apart from the desirability of grading penalties to fit crimes, the reputations of drivers as well as the safety of road-users are involved.

A physiological threshold is something which varies. But the concept is real and invaluable. Pain, convulsion, and head-drop thresholds therefore vary. But the variation has its measurable limits to a standard range and an ED50 (median effective dose) level of drug can be established. Readers will accept without question the implication of the ED50 of drugs in general, but allow variation in the response to ethanol to fog their appreciation of ethanol thresholds.

The maximum blood-alcohol level consistent with the safety of other road-users has