nasal polypi. The thin bony walls of the ethmoidal labyrinth are destroyed by chronic sinusitis. This allows infection through the cribriform plate.

I would, however, disagree with the opinion that meningitis rarely occurs in patients with spontaneous C.S.F. rhinorrhoea. I have diagnosed this condition in three of my patients. Two of them developed meningitis. The diagnosis is not difficult provided one bears it in mind. A watery nasal discharge is a very common symptom in an otolaryngological clinic. It is usually due to allergic rhinitis or chronic rhinitis due to nose drops. In C.S.F. rhinorrhoea a sample of the fluid will contain glucose. This never happens in allergic rhinitis. A rapid test is to insert a Clinitest stick into each nostril. It will confirm the presence of glucose and show the side of the C.S.F. leak.

Tomography does not help to confirm a bony defect in the cribriform plate. The area is difficult to see clearly by *x*-rays. Looking for radioactive isotopes in the nose after they have been put into the lumbar theca is an important development. It may help us to diagnose this condition more often.—I am, etc.,

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Miliary Tuberculosis in a Nonagenarian

SIR,—In old age tuberculosis usually occurs as a chronic pulmonary infection, often indolent enough for grandchildren to catch the disease unaware. Even in "open" cases, symptoms and fever can be absent because age slows metabolism and defences remain firm. Miliary tuberculosis is now uncommon. Fifteen deaths from the disease were notified in 1962–66, with 26 cases in the preceding five years (General Register Office, 1967). But it should stay high in the list of differential diagnoses for pyrexia of unknown origin. We record this case as a reminder that miliary tuberculosis may occur at any age.

A young-looking 93-year-old woman had night sweats and felt tired for a month. She had no cough, breathlessness, headache, anorexia, dysuria, or ankle swelling. She had fever, without rigors, nine days before admission, and routine blood agglutination tests were negative. Her past health was excellent but her brother died of Addison's disease. She looked well, her temperature was 38.4°C., the pulse was regular at 76, and blood pressure 190/70 mm. Hg. Her skin, retinae, and lungs were clinically normal and the spleen and lymph glands were not enlarged. A soft apical systolic murmur was heard in her heart, and a provisional diagnosis of bacterial endocarditis was made. The haemoglobin was 11.83 g./100 ml., W.B.C. 8,000 per cm., E.S.R. 32 mm. Three blood cultures were sterile, repeated blood agglutination and sedimentation tests were normal, and the urine showed no abnormality. Stool cultures were negative and the chest *x*-ray was normal. A barium meal and liver function tests were normal and L.E. tests negative. Another mid-stream urine showed 60 mg. of protein, many leucocytes, and a growth of paracolon bacteria. Neither a course of ampicillin nor nitrofurantoin altered the fever. A chest *x*-ray two weeks later was again normal, but an abdominal *x*-ray showed a calcified gland. The hectic fever slowly subsided, the patient became doubly incontinent and died. At post-mortem a pulmonary embolus accounted for her death, and tuberculous ulceration of the lower ileum, with generalized miliary tuberculosis, was found. Calcified mesenteric glands were present and the organism was bovine in type. The infection was considered to be a recrudescence of an old intestinal infection.

This woman was not "geriatric" until her fatal febrile illness. A tuberculous focus presumably broke into the circulation after being quiescent for perhaps 80 years. A similar case was seen 18 years ago where radiotherapy appeared to have caused the dissemination. The patient, aged 76, had destruction of one lumbar vertebra and no primary neoplasm was discovered. After radiotherapy, given for pain, she developed a hectic fever and later miliary lesions in the optic fundi.[1] In the first month of this disease chest *x*-ray changes are often absent, as in these two cases. One of us (R.W.A.) had diagnosed, at post mortem, three other cases, aged 60, 68, and 83, in the last four years. The Registrar General's figures are bound to be an underestimate of the true incidence.

The diagnosis is made from the sputum, stool, or urine; occasionally biopsy of the marrow, liver, or pleura is necessary. Acid-fast staining and cultures for tuberculosis are essential in the old as in the prime of life.

We thank Dr. G. A. MacGregor for permission to publish this case.

—We are, etc.,

R. MILTON.
R. P. YADAVA.
R. W. AINSWORTH.

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REFERENCE

1 Macgregor, G. A., 1968, Personal communication.

Gastrointestinal Haemorrhage and Aspirin

SIR,—Dr. H. B. Valman and his associates in their paper on gastrointestinal haemorrhage and aspirin (14 December, p. 661) make the provocative statement, "Though the mechanism of bleeding after aspirin ingestion is still obscure some facts are known," but unfortunately they fail to disclose what some of the facts are. Since I have been interested in the problem of haemorrhage in surgical procedures since 1937, when I proposed the use of vitamin K for the treatment of cholaemic bleeding,[1] and have given considerable thought and effort to the bleeding problem, I should like to make a few comments on aspirin.

This drug has a systemic vascular effect which can be measured by the prolongation of the Duke bleeding time, which I have utilized for developing the aspirin tolerance test.[2] The action of aspirin is attributable to its acetyl linkage, since sodium salicylate has no such effect. Aspirin appears to act by preventing the vascular contraction after mechanical injury of the micro-circulation. Many normal subjects have a slight but distinct increase of the bleeding time two hours following the ingestion of 0·65 g. of aspirin. It is likely that it is this group which shows the occult gastrointestinal bleeding from aspirin. In the Minot–von Willebrand syndrome the bleeding time is significantly prolonged by aspirin, and these subjects have the more severe gastrointestinal bleeding from the drug.

The two conditions that should always be considered in idiopathic gastrointestinal haemorrhage are the Minot–von Willebrand syndrome and telangiectasia. The first disease is readily detectable even in the mild form by the aspirin tolerance test. Telangiectasia, which is equally as common, is likely to be overlooked because no specific laboratory tests are available and the diagnosis must depend on a careful physical examination, skill in recognizing the skin lesions, and on the hereditary history. When the diagnosis of these two diseases is established, aspirin as a factor in bleeding becomes more understandable.—I am, etc.,

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REFERENCES

1 Quick, A. J., *Amer. J. Physiol.*, 1937, **118**, 260.
2 Quick, A. J., *Amer. J. clin. Path.*, 1967, **47**, 459.

Motorway Madness

SIR,—In reading the newspaper reports of the dreadful pile-ups on the M1 and M4 in fog recently, two neglected explanations occurred to me:

(1) *Relative sensory deprivation.*—Safe motoring depends on the driver perceiving and acting upon a stream of visual and auditory clues. In thick fog a motorist is almost in the situation of a solitary astronaut in a blacked-out space capsule. In such conditions he may make disastrous errors of judgement. He may even begin to hallucinate or have delusions if his sensory input falls below a certain critical level.

(2) *Toxic effects of petrochemical fumes trapped at low level in fog.*—The central nervous system is very sensitive to petrochemical intoxication. Under certain atmospheric conditions the petrochemical content of fog at ground level in traffic may be high enough to affect perception and judgement adversely.

Taken together, these two factors could account for normally rational, careful drivers blinding along at 70 m.p.h. into almost certain destruction. Two remedies might help: infra-red lamps and sensing devices on all cars, and respirators for motorists to wear in fog.—I am, etc.,

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Management of Acute Salicylate Poisoning

SIR,—I read with interest the articles on the treatment of acute salicylate poisoning by Drs. A. G. Morgan and A. Polak (4 January, p. 16) and by Dr. T. M. Savege and others (4 January, p. 35). In reviving the combined use of bicarbonate and acetazolamide in adult salicylate poisoning Drs. Morgan and Polak have achieved an effective form of treatment in terms of alkalinization of the urine and reduction of plasma salicylate, but their regimen is no more effective than more generally accepted regimens of forced alkaline diuresis. They express concern at the high infusion rates of the latter forms of treatment, but in my experience of 307 adults admitted to the Regional Poisoning Treatment Centre, Royal Infirmary, Edinburgh, with acute salicylate overdosage, clinical complica-