should be to the much-visited U.S.A. and Canada. No doubt the adverse reports in the medical press about the irritations encountered during service abroad are a reason for avoiding the former colonies now emerging to self-government. These reports are, however, hardly ever refuted or discussed, and, while not disputing the integrity of such letters, it is only fair to point out that it is unlikely that anyone can expect to get more out of this type of service than they put into it.

In my opinion the permanent medical directorate of Professor Hill's proposed Commonwealth Medical Council should be a Government department staffed by experienced medical advisers from the Ministry of Health, the Colonial Office, the Commonwealth Relations Office, and the Foreign Office. The membership of the council itself might include all Government departments and organizations who send medical personnel overseas, such as H.M. Forces, the British Council, missionary societies, and industrial firms, etc. The B.M.A. might wish to be represented, but a semi-voluntary organization under the auspices of the B.M.A. in my view would not carry the central governmental authority and political awareness to make the work effective at home and abroad. If the plans are made and implemented within the framework of such a council, British doctors would have a tremendous opportunity to show their mettle and help to avoid the experiences which have dismayed us all in the Belgian Congo. The history of British medicine overseas is, to say the least of it, a fine one, and it would be a sad affair if the doctors of to-day, cosy in their successful N.H.S., were to allow foreign doctors and scientists to usurp their record in this particular field.

Personally neither I nor my family have the slightest regret for my tour of duty in the Colonial Service. We enjoyed it all and I gained considerable valuable experience. I did not find it difficult to re-enter the N.H.S., but must admit that I think I would have been more useful abroad with experience of the N.H.S. behind rather than in front of me. Given the proper organization and encouragement, the young doctors of to-day could be one of our most valuable exports, especially to Africa, if terms like good will, service, human relationships, help to the sick and needy, and the example of the British way of life are to mean anything at all in the vast reorganization that is happening now.

The best age for the doctor to do this service is when he and his family are fairly young. Children thrive in hot climates until they are 6, education is not a serious problem, and the wife gets plenty of willing help. At the end of one or two tours a decision has to be made whether to return home or stay, but appointment to a post at home need cause no anxiety if conscientious and efficient work has been done. The return to the young doctor in terms of experience, broadening of outlook, and self-confidence is far greater than anything which can be obtained in a comparable time in England to-day. —I am, etc.,

Syston, Leicester. J. A. WARD.

SIR,—In my letter in your issue of December 17 (p. 1801) I made a statement which was in error. The Nigerian Widows' and Orphans' Fund entitled pensions have not in fact been halved or reduced in any way for paid-up members. The information received by us was given in all good faith and was in fact checked by cable. I am very pleased to be able to say that this information was in fact false and regret any alarm which may have been caused by it.—I am, etc.,

Nairobi. P. E. C. MANSON-BAHR,
Chairman, Services Subcommittee of the B.M.A. Kenya Branch.

Splenic Rupture in Leukaemia

SIR,—We are interested in the description by Mr. M. J. Flood and Mr. R. A. Carpenter (January 7, p. 35) of a patient with acute myeloid leukaemia who had ruptured his spleen.

In July, 1959, we treated a man of 34 who had a spontaneous rupture of his spleen, and on further investigation was found to be suffering from chronic myeloid leukaemia. On direct questioning he denied any injury, but admitted to "a touch of malaria" in 1948 when in Malaya. On admission to the Mansfield Nursing Home he gave a history of recent tiredness and of 48 hours' abdominal pain which started in the left side of the abdomen and later became generalized. On examination, he looked ill and lay still. His temperature was 99.2° F. (37.3° C.), pulse 112, blood-pressure 140/80. His tongue was furred. The abdomen was slightly distended, with generalized release tenderness and rigidity of the left upper quadrant. The bowel sounds were diminished and there was rectal tenderness. His haemoglobin was 87% and there was slight anisocytosis and poikilocytosis seen on the film. Under a general anaesthesia given by Dr. A. G. Hegarty, the enlarged spleen could be felt. At laparotomy the spleen was found to be ruptured along its free edge and there was over one pint of blood free in the peritoneal cavity. Dr. A. B. Hill reported that the changes in the excised spleen and the liver biopsy were suggestive of granulocytic leukaemia. The total peripheral nucleated white-cell count was 300,000 per c.mm., of which about 14,000 were blast cells. This information was not available at the time of operation. The patient has remained well and has been on intermittent busulphan therapy since his splenectomy.

Two other uncommon modes of presentation of leukaemia have been seen by us. The first was an old lady who presented with an anal ulcer and was found to have an enlarged spleen and myeloid leukaemia. The second was an apparently healthy man of 36 who presented with painless haematuria and was found on examination to have petechiae on the right shoulder. Cysto-urethroscopy while bleeding showed a bloody efflux from both sides. He was found to have an acute leukaemia, blast cells forming 73% of the white-cell series in the sternal-marrow examination. He died one week later from a cerebral haemorrhage. Gastro-intestinal bleeding associated with leukaemia has occurred in 3 of our last 700 admissions for haematemesis and melaena.—We are, etc.,

J. C. PEASE.
J. N. WARD-MCQUAID.

Mansfield and District General Hospital, Notts.

SIR,—I was very interested to read the case of spontaneous rupture of the spleen in acute myeloid leukaemia described by Mr. M. J. Flood and Mr. R. A. Carpenter (January 7, p. 35), having encountered a similar case.

A 40-year-old man presented himself in the casualty department, complaining of abdominal pain. He collapsed while waiting and died almost immediately. On post-mortem examination, the only abnormal features were pallor of the body and a ruptured spleen, with a large amount of blood in the abdominal cavity. The spleen was enlarged about three-fingerbreadths below the costal margin. On histological examination, sections of spleen revealed a picture of acute myeloid leukaemia, which was also confirmed by peripheral blood and bone-marrow examination. So far as I could ascertain, there was no history of injury or trauma before his visit to the casualty depart-

ment, and his only complaints were some shortness of breath and feeling off-colour for a few weeks.

I have also been told of a similar case where only a post-mortem examination revealed the real cause of death. As neither case was published, I would infer that spontaneous rupture of the spleen in acute myeloid leukaemia occurs more commonly than suggested by the authors.—I am, etc.,

R.A.F. Hospital,
Wroughton, Wilts. A. TALERMAN.

Phenylbutazone and Leukaemia

SIR,—We were interested to read the article by Dr. R. H. D. Bean, "Phenylbutazone and Leukaemia" (November 26, p. 1552), and also the letter by Dr. A. Lawrence on this subject (December 10, p. 1736). Although blood changes complicating phenylbutazone therapy are well recognized, there has been only one report of transient leucopenia complicating therapy with hydroxyphenylbutazone ("tanderil," G27.202).[1] This drug has previously been shown to have a similar anti-rheumatic and analgesic effect to phenylbutazone[2] but apparently less toxic side-effects.

We have recently seen a man aged 62 years who developed transient leucopenia and thrombocytopenia while on hydroxyphenylbutazone therapy. He was first seen in June, 1959, complaining of episodes of pain, swelling, and redness of both big toes, which settled spontaneously but were soon followed by an acute arthritis of the left knee, which was relieved within twelve hours of starting colchicine therapy. The serum uric acid level was 17 mg./100 ml., and a diagnosis of gout was made.

A routine blood-count showed: haemoglobin 19.3 g./100 ml. (132% Haldane), haematocrit 58%, with normal white cell and platelet counts. Polycythaemia was suspected and confirmed by the demonstration of an increased red-cell mass, using a ^{51}Cr technique. It was felt that the hyperuricaemia was attributable to the polycythaemia. The patient was venesected twice and received two one-month courses of busulphan between September and November, 1959, with temporary suppression of the white-cell count but no effect on the platelet count or haemoglobin level. Maintenance therapy with soluble aspirin relieved his joint pains but caused dyspepsia. In December treatment with phenylbutazone, 400 mg. daily, was commenced, but this also caused epigastric pain and diarrhoea. As a supply of hydroxyphenylbutazone was available and preliminary reports had suggested that it was efficacious in gout, this was substituted in a dose of 300 mg. daily, later reduced to 200 mg. because of further diarrhoea. As this produced satisfactory relief of his joint pains it was continued for six weeks until thrombocytopenia and leucopenia were noted (see Table). The drug was immediately stopped, and within two weeks the white-cell and platelet counts returned to normal. Repeated monthly counts since this episode have been normal. Subsequently long-term uricosuric therapy with probenecid was begun.

Date	Haemoglobin (%) Haldane)	W.B.C. (per c.mm.)	Platelets (per c.mm.)	Treatment
14/9/59	128	5,600	157,000	Calcium aspirin 3·9 g./day Busulphan, 4 mg./day
12/10/59	121	2,700	142,000	Calcium aspirin 3·9 g./day
17/11/59	121	4,900	121,000	3·9 "
				Busulphan 2 mg./day"
15/12/59	119	4,100	119,000	Phenylbutazone 400 mg./day
5/1/60	121	4,900	117 000	G27.202 300 mg./day
15/2/60	125	4,800	78,000	Nil
19/2/60	126	4,600	63,000	Tabs. paracetamol 4–8/day
23/2/60	121	2,800	98,000	" " "
26/2/60	120	4,000	90,000	" " "
1/3/60	121	5 300	106,000	" " "
7/3/60	121	5 000	149,000	" " "
4/7/60	120	4,800	137,000	" " "

We feel that the episode of leucopenia and thrombocytopenia observed was due to the hydroxyphenylbutazone therapy. Since the course of phenylbutazone was terminated six weeks, and the course of busulphan three months, before the episode, it is felt that neither drug can be held directly responsible. It is conceivable that either, or both, of these drugs could have produced a minor degree of sustained marrow suppression undetectable in the peripheral blood, and that the additional suppressant effect of hydroxyphenylbutazone unmasked this. It is most improbable that the thrombocytopenia and leucopenia were due to an aplastic phase of polycythaemia vera[3] because it was of such short duration, recovery being rapid and maintained for six months.

The authors wish to thank Dr. P. Bauwens, Dr. J. L. Pinniger, and Dr. G. Wetherley-Mein for permission to publish, and Dr. W. S. Stoddart, of Geigy Pharmaceuticals Limited, for supplies of hydroxyphenylbutazone.

—We are, etc.,

St. Thomas' Hospital,
London S.E.1. D. A. H. YATES.
R. W. PAYNE.

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Natural Gas

SIR,—Mr. James Lawrie in his letter (December 10, p. 1735) criticizing my own (November 26, p. 1597) on the dangers of "re-formed" natural gas containing carbon monoxide, stresses the interchangeability of methods of suicide, which I had myself admitted. My main concern was with the high and growing incidence of accidental deaths due to coal-gas, an incidence which would be perpetuated if the latter were replaced by re-formed natural gas.

The possibility of replacing coal-gas by natural gas which has not been re-formed, and the advantages of such a replacement, are demonstrated by Gonzales *et al.*[1] who write:

"In New York City in 1950 and 1952 respectively, throughout the Boroughs of Richmond and Brooklyn, natural gas was substituted for the old illuminating gas which was manufactured from coal and contained CO as its chief ingredient. Natural gas contains methane (CH_4), 94.1%; ethane (C_2H_6), 3.6%; propane (C_3H_8), 1.0%; higher paraffin hydrocarbons, 0.7%; and carbon dioxide (CO_2), 0.6%. There is no carbon monoxide in natural gas, so that if it is inhaled directly with plenty of oxygen, the victim is not seriously affected but only rendered uncomfortable. Of course if the gas were inhaled in a closed chamber without oxygen, it would cause death from asphyxia like carbon dioxide. As it is rich in hydrocarbons containing much carbon, natural gas on combustion might form considerable carbon monoxide and be a source of poisoning, especially if burned under conditions in which complete combustion did not occur.

"The statistics of the Chief Medical Examiner for Richmond from 1947 to 1951 show a striking decrease in fatal cases of gas poisoning after the substitution was made:

1947—fatal cases of gas poisoning, 13 (suicide, 8; accident, 5).
1948—fatal cases of gas poisoning, 16 (suicide, 9; accident, 6; homicide, 1).
1949—fatal cases of gas poisoning, 9 (suicide, 3; accident, 6).
1950—fatal cases of gas poisoning, 0.
1951—fatal cases of gas poisoning, 0."

I believe that this has been a common experience in many parts of the United States, but have so far been