remain away from employment for a specified period. Most of the plants set 6 weeks or 2 months as the required post-natal leave; one set it at 4 weeks, one at 4 months, and one at 6 months. Eleven required the amount of post-natal leave to be decided by a physician (the plant physician or the woman's own physician).

Extent of Return to Work After Delivery.—It was very difficult to find out how many women returned to work after childbirth. Most firms did not keep records of this type. Companies that had been employing women for many years, although they did not keep records of the number of returns, frequently were able to offer estimates. The textile mills had a very high percentage of returns. Some electrical plants also had a high percentage.

Reinstatement and Seniority.—Among the establishments that had high percentages of returns to work the question of reinstatement and seniority was probably a factor in motivating an early return. In several of these plants reinstatement and seniority privileges were effective for only 2 to 3 months after childbirth. If the period in which these privileges were effective were lengthened, women might be inclined to take longer post-natal leave."

Some British Statistics

An investigation on employment of women in industry before and after childbirth in this country carried out during 1940-2 by the Industrial Welfare Society gives the following general information. Twenty-six firms were approached; they all permit a return to work, but the period after pregnancy varies as follows:

At a minimum of 4 weeks	..	..	..	..	2 firms
" " 5 "	..	..	..	..	3 "
" " 6 "	..	..	..	..	6 "
" " 8 "	..	..	..	..	7 "
" " 12 "	..	..	..	..	4 "
On application at any time	..	..	..	..	1 firm
At any time a vacancy occurs	..	..	..	..	1 "
When fit to return	..	..	..	..	1 "
Left to opinion of welfare officer	..	..	..	..	1 "

The statistics of 29 firms who keep records of their pregnant workers gave the following figures:

Firm	Pregnancies	Returned to Work	Firm	Pregnancies	Returned to Work
A	12	1	P	6	4
B	20	2	Q	122	8
C	37	1	R	13	4
D	11	2	S	25	4
E	50	1	T	57	2
F	13	10	U	17	3
G	21	2	V	17	3
H	21	6	W	57	7
I	24	1	X	17	3
J	9	1	Y	24	3
K	3	1	Z	1	1
L	48	3	AA	1	1
M	12	3	BA	4	1
N	11	2	CA	9	2
O	4	4			
				666	85

One firm commented: "The great difficulty of mothers returning to work is the lack of day nurseries. The unmarried mothers, unless they have sympathetic parents, are forced to have their babies adopted, often against their will. Many married women would like to return to work if there were facilities for these very young babies." Another stated: "Two girls returned to work after one month—both through financial difficulties." None of the firms quoted had an antenatal clinic of their own.

Summary of Conclusions

Surprisingly few mothers return to work after confinement. In our own series only 18 out of 83 had returned to any work, excluding housewife duties, up to 17 months after confinement.

Death of the baby or financial matters were the main reasons given for the return to paid employment.

From our experience of the normal working mother we conclude that six months is a reasonable time after pregnancy for return to work to be considered.

We are grateful to Mr. R. R. Hyde of the Industrial Welfare Society, Dr. Katherine Bain, and Dr. Barbara Ann Hewell of the Children's Bureau, U.S. Department of Labor, for their co-operation, and to Sister Harwood of our own works antenatal clinic.

S. Solomon, M. Kaslik, and N. Kiven (*Ann. intern. Med.*, 1944, 21, 639) record three cases of periarteritis nodosa in which the condition was diagnosed during life and confirmed at necropsy. The diagnosis should be considered in patients with prolonged fever of unknown origin who have irregular or baffling symptoms referable to many parts of the body. A biopsy will usually reveal characteristic changes in the smaller arteries. The disease is probably an allergic reaction to many different antigens. There is no specific treatment.

Medical Memoranda

A Diagnostic Problem: An Unusual Case of Hepatitis

The following case is interesting in view of the prevalence of hepatitis. A girl aged 13 months was admitted to the hospital on Sept. 6 as a case of notified cerebrospinal fever (post-basis). Except for occasional "snuffles" the history disclosed nothing abnormal until two weeks before admission. She was then said to have had an attack of bronchopneumonia, treated for four days with 8 g. of sulphathiazole. Subsequently the child had been fretful, and for two days before admission there had been neck rigidity, head retraction, and persistent vomiting.

CLINICAL HISTORY

On examination the child's condition seemed satisfactory—T. 97°, P. 120, R. 26—but she was irritable and had a stiff neck with head retraction; Kernig's sign and Brudzinski's reflexes were negative. The gums were inflamed owing to teething, and the liver was palpable two fingerbreadths below the right costal margin, the upper limit of superficial dullness being percussed at the fifth interspace. Lumbar puncture was performed. The fluid was clear and under normal pressure, and contained "2 leucocytes per c.mm., protein 10 mg., globulin 0, chlorides 770 mg., sugar rather in excess. W.R. negative."

During the next three days the (presumable) meningism disappeared and vomiting ceased. The stools remained normal throughout the illness, while the urine contained a trace of albumin. On Sept. 10 a swelling appeared in the right renal angle. It was hard, deep-seated, immobile, but not attached to adjacent bony structures, and was associated with slight oedema of the overlying skin. On Sept. 11 the lump was larger and softer, but its nature remained obscure. At 4.30 p.m. precisely that afternoon the child screamed violently, became cyanosed, and appeared to be in pain. On examination her colour was ghastly, her extremities cold, pulse imperceptible, temperature subnormal, and respirations about 80 per minute. She was gravely ill, and the dramatic suddenness of the change suggested a catastrophe such as an internal haemorrhage or spontaneous pneumothorax. No thoracic signs were elicited, but the liver was over five fingerbreadths below the right costal margin, while the upper limit of superficial dullness was level with the sixth rib on percussion.

Surgical advice was sought, and Mr. Kennedy of Plymouth kindly saw the child. Nevertheless, no diagnosis was made, and the only course available was treatment for shock on the usual lines. By 8.30 p.m. her condition had improved slightly—T. 98.4°, P. 160, R. 88—and the liver was only three fingerbreadths below the costal margin, but at 10.35 p.m. she collapsed and died, her malady undiagnosed.

POST-MORTEM EXAMINATION

The necropsy was performed by Dr. M. R. Thomas, with the kind permission of Dr. Wordley. Externally there were no distinctive marks and the lumbar swelling noted during life had disappeared. About half a pint of haemorrhagic fluid was found in the right pleural cavity. Otherwise, apart from petechial haemorrhages on the serous surfaces and a small pericardial effusion, the heart and lungs seemed normal. In the abdomen half a pint of bile-stained ascites was found. The liver was tremendously enlarged and engorged, and the cut surface was yellow. The hepatic artery and biliary system appeared normal, as also the duodenum and other viscera, though the right kidney was displaced backward into the loin by the enlarged liver, thus accounting for the mysterious lumbar swelling.

Histologically, the liver showed generalized parenchymatous necrosis, particularly marked in the portal zones. There was neither hyperplasia of liver cells nor bile-duct proliferation. The organ was stained and examined for spirochaetes, but none was seen, nor was evidence of syphilis found. Other organs showed no special changes except scattered petechial haemorrhages.

DISCUSSION

It is unfortunate that investigations, including blood counts and urine analysis, were not done, but precise indications were wanting until too late. First, the cause of the meningism remains obscure. It might have been due to the hepatic lesion or even to "teething." It should be remembered that occasionally meningism is a marked feature of intolerance to sulphamide therapy, particularly in cerebrospinal fever—a highly improbable cause in this instance, however, eight days later. Alternatively Weil's disease with a meningeal onset merits consideration, but it is unlikely that such a case would have been apyrexial throughout, nor were any spirochaetes demonstrated.

The puzzling lumbar swelling received satisfactory explanation at necropsy. More difficult of explanation was the sudden and catastrophic deterioration in the child's condition, with apparent hepatic displacement and enlargement. The rapid accumulation of a haemorrhagic pleural effusion—singular indeed in respect of the complete absence of physical signs in the chest—was probably largely responsible. Sudden cardiac

failure may also have aggravated the general condition and increased the size of the liver from acute passive congestion. No doubt the various effusions and petechial haemorrhages were direct consequences of hepatic failure.

In view of the extensive necrosis it is perhaps surprising that jaundice was completely absent. Either the liver destruction was too rapid or this was really an example of extreme fatty degeneration of the organ following an acute infection—bronchopneumonia in this instance, as was stated in the history. The latter explanation, however, was not supported by the histological findings, but certainly the possibility of a fulminating virus hepatitis cannot be completely excluded.

Lastly, of drugs sulphathiazole alone was given, to a total of 8 g. two weeks before death. Fatal cases of hepatitis are known to have followed sulphonamide therapy, and symptoms have not always declared themselves until after the final withdrawal of the drug. Of all possible causes, this does seem the most feasible; but, unfortunately, the available evidence is inconclusive.

I am indebted to Dr. T. Peirson, medical officer of health for Plymouth, for permission to publish this case; to Dr. D. F. Johnstone, medical superintendent of this hospital, for his interest in the preparation of the paper; and to Dr. M. R. Thomas for the post-mortem examination and preparation of histological material.

J. L. FLUKER, M.B., M.R.C.P.,
Resident Medical Officer,
City Isolation Hospital, Plymouth.

Splenomegaly in Bacillary Dysentery

As we have not found a reference to splenomegaly in dysentery in the limited amount of medical literature which we have been able to search we think the following account of a series of cases may be of interest.

A SERIES OF CASES IN THE MIDDLE EAST

Enlargement of the spleen was found in 50 (5.77%) out of a total of 866 cases of bacillary dysentery admitted to a general hospital in the Middle East between Aug., 1943, and Jan., 1944. The cases had occurred regularly throughout this period. There was no evidence of the presence of any other disease which might have caused splenic enlargement. In none of the cases was there any lymphadenopathy or clinical evidence of anaemia. At least one blood slide was examined in each case, but no malaria parasites were discovered. Only four cases gave a history of previous single attacks of malaria—10/12, 2, 3, and 3 years previously. Three cases were not included in the series, as it was considered malaria might be the cause of the splenomegaly, two of the patients being Cingalese and the other a British "other rank" who gave a history of three attacks of malaria two years previously. Three other cases were excluded, in two of which the splenomegaly persisted for 21 and 28 days respectively, and in the third there was a history of two attacks of short-term fever in the previous two months. It seems unlikely that enteric fever could produce this almost constant picture of sudden onset of diarrhoea, associated with blood and mucus in the stools, fever for 2 to 3 days, absence of rose spots, and fitness for duty in 10 to 12 days.

Twenty of the patients had been in the Middle East for less than four months, and of these eight had been abroad for only four weeks or less. The cases were not confined to one particular camp, but were admitted from various surrounding sites over a wide area.

CLINICAL DETAILS

Except for the splenomegaly all the patients showed the usual clinical features of bacillary dysentery. They were of the mild or moderately severe type. The average duration of diarrhoea was 2.88 days (maximum 10 days; minimum 1 day—7 cases). The temperature on admission ranged from 99° to 102° in 38 cases, but in 12 there was no fever. Two cases were febrile for 3 and 5 days respectively, but the remainder had a normal temperature after 48 hours.

In 46 cases blood and mucus appeared in the stools; 23 cases on microscopy showed bacillary exudate and 27 cases indefinite exudate; 4 cases had liquid stools with mucus and showed indefinite exudate. Of 31 cases in which a culture was performed 13 showed the Flexner bacillus (Flexner I, 3 cases; Flexner II, 6 cases; undetermined, 4 cases) and 18 showed no pathogens.

In 49 cases the spleen was palpable on the day of admission and in one on the day after admission (3rd day of disease). In 37 cases the spleen on inspiration reached a point 1 fingerbreadth below the costal margin, in 10 cases 2 fingerbreadths, and in 3 cases 3 fingerbreadths. In 43 cases the spleen remained palpable for 2 to 6 days only, but in 7 cases the edge could just be felt on discharge from hospital on the 15th to 18th day of disease. In one of these cases, which it was possible to

review, the spleen was no longer palpable 3 months later. Total and differential white blood cell counts were carried out in 22 cases. No constant abnormality was present. The average total was 6,959 (maximum 15,300, minimum 3,200). The average proportion of polymorphonuclear leucocytes was 62%.

DISCUSSION

The cause of the splenic enlargement in these cases is not understood. As already stated, malaria, enteric group fevers, and other obvious causes of the splenomegaly were excluded. It is suggested that it is a non-specific phenomenon, and this is borne out by the fact that similar splenic enlargement was found to occur equally commonly in certain other infectious diseases in the Middle East (Illingworth, 1944, personal communication). Illingworth reported that splenic enlargement occurred in 6.3% of 221 cases of diphtheria seen by him, 5% of 81 cases of tonsillitis, 7.8% of 42 cases of scarlet fever, and 5.3% of 38 cases of measles. In only 4 of these cases was there a previous history of malaria and in no case was there any other discoverable cause for the splenomegaly. In all except one case the splenic enlargement had subsided within a fortnight of the onset of the infection. It is interesting that the frequency of splenomegaly in these infectious diseases was approximately the same as that of the cases of bacillary dysentery reported above.

We are indebted to Major R. S. Illingworth for his invaluable help and also to Lieut.-Col. Bodley Scott for his criticism and advice. We should also like to thank Col. W. B. Stevenson, officer commanding a Middle East general hospital and Brig. W. K. Morrison, officiating D.M.S., M.E.F., for permission to publish this article.

R. P. WARIN, M.D.,
Capt., R.A.M.C.

R. S. ALEXANDER, M.B., Ch.B.,
Capt., R.A.M.C.

Jaundice during Arsenotherapy

In view of the general interest taken in the causation of jaundice during arsenotherapy, it may be of value to record some observations made at the neurological clinic, Metropolitan Hospital, on a consecutive unselected series of 12 cases. These patients were all suffering from neurosyphilis; their ages ranged from 13 to 64 years. Treatment has consisted of intramuscular or subcutaneous injections of 3 c.cm. of acetylarsan (May and Baker), together with 0.5 c.cm. of intramuscular bismotab in courses of 10 to 12 injections, with intervals of one month. Potassium iodide orally was also given. By the use of this preparation of arsenic given by these routes over a period of 2 to 4 years, possible transference of minute quantities of serum from patient to patient, as suggested by Major Salaman (1944) to be a potential cause of jaundice, is avoided.

These patients were carefully observed while undergoing treatment, and it is significant that not a single case of jaundice occurred. This suggests that where the intramuscular or subcutaneous rather than the intravenous route is used there is little likelihood of jaundice occurring. This seems to be supporting evidence for the belief that the hepatitis which is sometimes produced during arsenotherapy is not a direct toxic effect due to the arsenic but is rather of an infective origin, as is now thought to be the case in homologous serum jaundice or in the jaundice occurring after yellow-fever vaccination. Particulars of the cases are as follows:

Case	Sex and Age	Disease	W.R.	Commencement of Arsenotherapy	No. of Courses	No. of Inj.
1	M 55	Meningo-vasc. syph.	+	Mar., 1941	4	10
2	M 13	Cong. G.P.I.	+	" "	5	12
3	F 49	A.R. pupils	-	" "	2	12
		" Syph. died out				
4	F 36	Meningo-vasc. syph.	+	Nov., 1941	5	12
5	F 36	Cong. tabes dorsalis	+	Nov., 1942	5	12
6	M 57	Meningo-vasc. syph.	+	Feb., 1942	5	12
7	F 33	G.P.I.	+	Apr., 1942	4	12
8	F 49	Tabes dorsalis	+	June, 1942	4	12
9	M 56	Meningo-vasc. syph.	+	Aug., 1942	4	12
10	F 36	Cong. tabes dorsalis	+	Nov., 1942	3	12
11	M 55	Taboparesis	+	June, 1943	3	12
12	M 41	"	+	Aug., 1943	3	10

Cases 2 and 7 received malaria therapy previously. All the above cases now have a negative Wassermann reaction.

I wish to thank Dr. Worster Drought and Dr. N. G. Hulbert for access to the case records, and the latter also for his kind help and advice.

Metropolitan Hospital, E.8.

B. GREEN, M.R.C.S., L.R.C.P.

REFERENCES

- Findlay, G. M., *et al.* (1944). *Lancet*, **2**, 340.
Marshall, J. (1943). *Brit. J. vener. Dis.*, **19**, 52.
Salaman, M. H. (1944). *Proc. roy. Soc. Med.*, **37**, 458.