TABLE II.—Cases at Lichfield (all males)

Case No.	Symptoms						
	Age	Shivering	Head-ache	Sore Throat	Cough	Muscle Pains	Temperature on Day of Disease
1	18	—	—	Dry	+	—	101.6° F.; 2nd (38.7° C.)
2	18	—	+	Dry	+	+	101.0° F.; 2nd (38.3° C.)
3	19	+	—	Dry	+	—	98.4° F.; 2nd (36.9° C.)
4	22	+	+	+	—	+	102.8° F.; 1st (39.3° C.)

Saline Washings.—Virus was not isolated from these washings.
C.F.T.—Second sera showed 4- or 8-fold rise in titre of complement-fixing antibody to Type A strain antigen.
Result.—Influenza type A.

TABLE III.—Cases at Donnington (all males)

Case No.	Symptoms						
	Age	Shivering	Head-ache	Sore Throat	Cough	Muscle Pains	Temperature on Day of Disease
1	19	+	+	—	—	+	99.6° F.; 3rd (37.5° C.)
2	18	+	+	—	—	+	99.8° F.; 3rd (37.7° C.)
3	18	+	+	—	—	+	98.0° F.; 3rd (36.7° C.)
4	19	+	+	—	—	+	100.0° F.; 2nd (37.8° C.)
5	20	—	+	—	+	+	100.2° F.; 3rd (37.9° C.)
6	19	+	+	—	+	—	100.8° F.; 3rd (38.2° C.)
7	20	+	—	+	+	—	100.2° F.; 5th (37.9° C.)
8	18	+	+	—	+	—	99.4° F.; 3rd (37.4° C.)

Saline Washings.—Type A strain of virus isolated in one case from washings taken on Feb. 9, 1949.
C.F.T.—Second sera showed 8- to 32-fold rise in titre of complement-fixing antibody to Type A strain antigen.
Result.—Influenza type A.

Complement fixation tests on sera from four cases at Malvern showed results with Type A strain antigens similar to those in the Tables.

Preventive Measures

Early diagnosis and rapid isolation of the patient were aimed at in order to check the spread throughout each unit. Strict attention was paid to the adequate ventilation of barrack rooms and to the spacing of beds to the optimum. General measures on the maintenance of the health of the troops, the shaking and airing of blankets in the open, and the sweeping of floors, previously sprinkled with water, were adopted. Routine gargling and the spraying of rooms with disinfectant were not carried out, nor was prophylactic inoculation attempted. Unit cinemas were closed for a short time in two areas at the height of the outbreak, but the numbers of cases occurring did not warrant restricting the movement of troops or cancelling the intake of National Service men to training establishments.

The movements of the first three cases occurring in each unit prior to the onset of symptoms were investigated, with, however, inconclusive results. No direct connexion with the European epidemic could be traced.

Summary

An outbreak of influenza of the Virus A type occurring in Army units in Wales and the West Midlands is reported. Symptoms, treatment, laboratory investigations, and preventive measures adopted are described.

Our thanks are due to Lieutenant-General Neil Cantlie, C.B., M.C., K.H.P., Director-General Army Medical Services, and Major-General R. W. Galloway, C.B., C.B.E., D.S.O., K.H.S., D.D.M.S. Western Command, for permission to publish this report.

SPONTANEOUS INTRA-ABDOMINAL HAEMORRHAGE

BY

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Spontaneous intraperitoneal haemorrhage—i.e., that occurring without any obvious cause such as cirrhosis of the liver (James, 1930) and rupture of aneurysm—has been recorded in 26 cases*—19 collected by Berk, Rothschild, and Doane† (1941), three reported by Marks and Freedlander (1945), and one each by Keusenhoff (1934), Matheson (1934), Crile and Newell (1940), and Haugen (1944).

In only eight of these cases were necropsies performed. In Keusenhoff's case and in two cases recorded by Moorehead and McLester (1936) a definite arterial rupture was found; but in the remaining five no single vessel could be held responsible, though in Matheson's case the haematoma was related to the branches of the ileocolic artery. Four of the cases had clinical or morbid anatomical evidence of hypertension (Moorehead and McLester two, and the fatal case of Marks and Freedlander) or arteriosclerosis (Keusenhoff). Haugen's case, which followed appendicectomy, like those of Matheson (1934), Churchman (1911), and Hartley and MacKechnie (1934), showed no evidence of vascular disease, but in these cases blood-pressure readings before the catastrophe or reports on the histology of the kidneys are lacking.

Three cases of spontaneous abdominal haemorrhage, two of which included a haemoperitoneum, are described below.

Case 1

A woman aged 58 was admitted with a four-months history of discharging ear (? cerebral abscess). She had had pain in the left hypochondrium for one month accompanied by vomiting when severe. Investigations before admission showed: haemoglobin, 66%; leucocytes, 17,300 per c.mm. (89% neutrophils); serum Wassermann reaction, negative.

On examination the temperature was 99° F. (37.2° C.), pulse 110. She had a profuse discharge from the right ear, and diplopia on looking above the horizontal. The abdomen appeared normal. The B.P. was 140/80. Leucocytes numbered 43,000 per c.mm. Two days after admission she had severe upper abdominal pain and urge to defaecate, but the bowels were not opened. On examination she was pale, the pulse was 160, and there was extreme tenderness in the upper half of the abdomen. She died in less than an hour.

Necropsy.—Intracranial disease was not discovered. Very marked purulent bronchitis and emphysema were found. On opening the abdomen large blood clots were seen in the peritoneal cavity. The greater omentum and lesser sac also contained blood clot. Careful dissection of the branches of the coeliac axis, the superior mesenteric artery, and the tributaries of the portal vein failed to reveal any macroscopic evidence of structural change in their walls or of thrombosis. Smaller vessels within the area of haemorrhage in the pancreas were also minutely examined without any abnormality being detected. Both renal arteries contained thrombi, the kidneys appearing very pale but otherwise normal. The bowel contents were black, but there was no macroscopic lesion in the intestinal mucosa.

Histology.—*Omentum:*—A huge area of haemorrhage occupied the omental fat tissue, which included a central area of

*Since submission of this paper a case of malignant hypertension with haemoperitoneum has been published by De Navasquez and French.

†The case of Florence and Ducuing (1913) was traumatic.

haemorrhagic necrosis. This area was surrounded by macrophages containing haemosiderin. Outside the haemorrhagic areas the arterioles showed fibrinoid thickening of the intima, and collars of lymphocytes, eosinophils, and macrophages. In addition, several islets of haemopoiesis were found. *Myocardium*:—Only the vascular changes were striking. The arterioles showed subintimal swelling, sometimes in the form of appositional loosely woven pink cushions and sometimes in the form of intensely eosinophil hillocks over which endothelial cells appeared unusually prominent and sometimes detached. *Spleen*:—There was very extensive and marked hyaline fibrinoid infiltration of walls of arterioles with reduplication of elastic laminae of some vessels. *Pancreas*:—Marked hyalinosis of arterioles was present. Stroma was rich in fat grapes and contained extensive areas of haemorrhage. *Stomach*:—Arterioles of outer layers of wall, outside and in the periphery of blood clot, showed marked hyaline fibrinoid infiltration. Innumerable collagen shreds could be seen in the clot. *Kidney*:—A fair number of glomeruli showed hyalinosis of the afferent arterioles. Many glomeruli appeared enlarged, with fusion and hyalinization of loops and increase in nuclei. There were tubular casts but no crescent formation. An occasional glomerulus showed fibrinoid change in some of its loops. Another section showed several collections of lymphocytes, the most conspicuous being around a hyalinized vessel the lumen of which was completely occluded by hyaline material, part of which showed fibrinoid change.

Comment.—Necropsy revealed no intracranial lesion and no source for the haemorrhage. Histologically there were widespread arteriolar thickening and kidney changes, as normally seen in hypertension, to which the massive intraperitoneal haemorrhage may therefore be attributed, in spite of the readings, which were, however, taken only after the onset of the disease. That the arteriolar changes may have given rise to the haemorrhage is possible, especially as similar lesions occurring in dermatomyositis have been shown (Pagel, Woolf, and Asher, 1948) to lead to extravasation of blood, though this was admittedly microscopic. Goldblatt's (1938) finding of petechial and larger haemorrhages in dogs with experimental malignant hypertension provides further support. The petechial haemorrhages in such cases were associated with dissection of necrotic or hyalinized arteriolar walls by red blood corpuscles. On the other hand, the possibility of a haemorrhagic diathesis may be considered, although Bruce's (quoted by Berk, Rothschild, and Doane, 1941) similar case with recovery was subsequently investigated for bleeding tendency without success. Multiple small discrete haematomata in the greater omentum and mesentery in Hartley and MacKechnie's case, however, suggest that their patient may have had a haemorrhagic diathesis.

The following case of abdominal haemorrhage affecting chiefly the ilio-psoas muscle, though lacking a haemoperitoneum, showed in other respects a striking resemblance to Case 1, and is therefore included.

Case 2

A woman aged 65 had had rheumatoid arthritis for eight years, and recently the onset of dyspnoea, nycturia, and swelling of the feet. On examination myxoedema, congestive cardiac failure, and advanced rheumatoid arthritis were found. The B.P. was 180/100. She collapsed and died two days after admission.

Necropsy.—Very pale and thin body. Basilar artery and circle of Willis showed severe atheroma. Right pleura adherent and oedematous, with numerous small haemorrhages (up to 0.4 cm. diameter), marked left ventricular hypertrophy, and atheroma of endocardium below aortic valves. Severe atheroma of coronary arteries. Femoral arteries showed medial sclerosis. Kidneys granular, with variegated cortex. There were extensive areas of intramuscular haemorrhage up to 5 by 4 cm. diameter in the superficial portions of the rectus abdominis and in the ilio-psoas muscles.

Histology.—*Psoas muscle*:—There was a massive haemorrhage beneath the fascia but no appreciable haemorrhage in the muscle itself. In addition to the extensions of the extravasation along the interfascicular stroma there were striking cellular and muscular changes. (a) The cellular changes consisted of dense perivascular and interstitial infiltration with plasma cells and lymphocytes, the former being predominant around the vessels, which, though for the most part appearing normal or showing slight fibrous thickening, occasionally showed intensely eosinophil eccentric intimal cushions as described in the myocardium in Case 1. (b) The muscular changes occurred only between the strata of extravasated blood, and consisted of vacuolation and shrinking of sarcoplasm with evidence of cellular regeneration. *Heart muscle*:—Conspicuous eccentric subintimal cushion formation in small arterioles. *Kidney*:—Extreme glomerular hyalinization of the wire-loop type, many glomeruli being completely atresic. There was also atrophy of the renal tubules.

Comment.—The presence of severe arteriolar disease and the clinically observed hypertension suggest a primarily vascular cause for the haemorrhages in this case. This is corroborated by the confinement of the pictures of muscular degeneration to the haemorrhagic areas. The perivascular, lymphocytic, and plasma-cell cuffing in the area of haemorrhage in these two cases is probably a local reaction to the latter, and is described in cases of perirenal haematoma by Heilmann (1930).

Case 3

A man aged 63 was admitted two hours after an acute onset of pain in the right hypochondrium radiating to the right scapular angle. The pain was dull and continuous, and he felt as though blown up with wind. There was slight nausea but no vomiting. A similar attack a year previously, diagnosed as a coronary thrombosis, cleared up after a week in bed.

On examination he was seen to be obese and pale, and he was sweating. The pulse was 72, temperature 97° F. (36.1° C.), and respirations 20. The blood pressure was 220/125. The abdomen was distended and tympanitic, without shifting dullness. There was tenderness but no rigidity in all areas, most pronounced in the right upper quadrant and extending round the flank to the right loin. Peristalsis was diminished. Rectal examination showed no abnormality and the urine was normal. On two occasions after admission he suddenly became shocked and his blood pressure fell to 60/40 but with a pulse rate of only 80. On each occasion he recovered in a few hours. On the afternoon following admission the tenderness and the gnawing pain were localized to the right iliac fossa. Laparotomy was performed for "acute appendicitis." There was much free blood in the peritoneal cavity and adhesions were present, mainly on the right side. In the right iliac fossa a fresh retroperitoneal haematoma was spreading towards the right loin, and a large boggy swelling was palpated in the position of the right kidney. Death occurred 30 hours after operation.

Necropsy.—Short, fat man. Right pararectal operation wound. *Heart*:—Weight, 800 g.; left ventricular hypertrophy. *Aorta*:—Gross atheroma. Peritoneum contained 60 oz. (1.7 litres) of fresh blood; omentum adherent to anterior abdominal wall. *Intestines*:—Gross distension; coil of terminal ileum adherent to pelvic peritoneum and obstructed by an omental band. Adhesions between gall-bladder and transverse colon. There was a large blood clot in the right perirenal fat which extended to the splenic flexure. The right suprarenal gland was expanded by a dense blood clot; external measurements 7 by 3 by 3 cm. The right kidney was surrounded by blood clot but was normal, apart from autolysis, and its capsule was intact. The renal artery was free from thrombosis, but the suprarenal artery could not be identified owing to the advanced autolysis.

Histology.—Suprarenal gland:—wholesale necrosis of the cortex, also of the capsular fat tissue. Some islets of parenchyma were still preserved in the central areas, where there was fibrinoid infiltration of the septa. With staining for elastic tissue the outlines of vein walls were visible in the centre of the blood clot.

Comment.—Perirenal haematoma was first described by Wunderlich (1856) under the title of "Apoplexie des

Nierenlagers," and was later classified into four categories by Ponfick (quoted by Coenen, 1910). Case 3 is an example of Ponfick's haematoma renis extracapsulare. Coenen, who collected 12 cases of perirenal haematoma and reported one of his own, mentions nephritis, renal sarcoma, arteriosclerosis, renal tuberculosis, chronic splenomegaly with gastritis, and haemophilia as the associated conditions. Heilmann (1930), who reported four cases of perirenal haematoma, suggests spasm of the capsular vessels as an aetiological factor. Cases of gross suprarenal haemorrhage have been reported in adults (Keele and Keele, 1942; Burnett, 1948). Eleven cases had previously been described. Only three of these cases, including those of Keele and Keele, were unilateral, as was the case under discussion, and in no instance was there a haemoperitoneum. In addition to atheroma and a possible spastic mechanism owing to hypertension, thrombosis of the suprarenal vein as in Keele and Keele's case should be considered as the proximate cause.

Summary

Three cases of intra-abdominal haemorrhage are reported. All three had evidence of hypertension.

The first case was one of spontaneous intraperitoneal haemorrhage with no morbid anatomical source for the bleeding apart from hypertensive changes in the smaller vessels.

The second case showed intramuscular haematoma; hypertensive arteriolar disease was sufficient to account for the haemorrhage.

The third case showed a haemoperitoneum from rupture of a perirenal haematoma. The origin of this is discussed. There was clinical evidence of severe hypertension.

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The British Council for the Welfare of Spastics has produced an admirable pamphlet entitled "Notes for Parents on the Home Care of Children Handicapped by Cerebral Palsy." It has been prepared by Dr. J. H. Crosland, of the Physical Medicine Department, Charing Cross Hospital, and Mr. H. P. Weston, executive secretary of the council. After a short account of the causes and nature of cerebral palsy the authors describe in detail how to care for spastic and athetoid children, while emphasizing that they must not be coddled. While these children must receive what help they need, the parents' natural anxiety should not prompt them to make their children over-dependent. Self-confidence and a sense of security must be inculcated into the children, and there are illustrations of special furniture, toys, and feeding utensils that will help the children to live as independently as possible. Special sections are included on the training of speech and excretion. Particulars may be obtained from the council's honorary executive secretary at 107, Norfolk Avenue, Sanderstead, Surrey. The pamphlet is obtainable from the same address for 1s. 3d. post free.

EFFECT OF PRESSURE COOKING ON VITAMIN C CONTENT OF VEGETABLES

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We have recently been investigating the effect of pressure cooking upon the loss of vitamin C in vegetables. As it may be some time before we can continue this work, and as there is considerable topical interest in the question, the results so far obtained are here briefly reported.

Method

In order that the results may be interpreted readily, we have compared the effects of pressure cooking with those of cooking in an ordinary saucepan. Since it is known that factors such as the volume of water and the period of cooking greatly affect the loss of vitamin C in the saucepan, the method adopted was that known to conserve the vitamin as much as possible. This method, advocated by the Ministry of Food, involves the use of a small volume of water, bringing this to the boil before putting in the vegetable, and cooking for the minimal time. With one exception, 200 g. of vegetable was cooked in 200 ml. of water in the saucepan, as this was found to be the minimal amount of liquid necessary to prevent the saucepan boiling dry. The exception was spinach, to which, as in common practice, water was not added.

The pressure cooker used was one of the now popular variety with a capacity of 7 pints (3.96 litres). The vegetables were cooked in this according to the directions supplied by the manufacturer. Again 200 g. of vegetables was used, but only 100 ml. of water. A larger volume of water is unnecessary, and would undoubtedly result in a greater loss of vitamin C by leaching. Care was taken to avoid overcooking with either method.

In every experiment six samples of each raw vegetable, so far as possible equivalent and representative, were taken. Two were for the determination of the initial content of vitamin C, two for the pressure cooking, and two for saucepan cooking. Two different portions of each raw sample of vegetable and three of each cooked sample were taken for analysis. The vitamin C was also assayed in the cooking-water. Thus for every experiment 20 samples were assayed, each assay being made in duplicate. In all, 14 experiments were performed on 10 different vegetables. The method of estimation used was that of Harris and Olliver (1942).

Results

In view of the varying nature of different vegetables and of different specimens of the same type, it was not to be expected that the variation in results between the two methods of cooking would be uniform. In all of the 10 vegetables examined there was no evidence that saucepan cooking was better than pressure cooking in retaining vitamin C. In six vegetables—cauliflower, broccoli, winter cabbage, new carrots, turnips, and swedes—the retention was consistently better in pressure cooking by between 3 and 28% of the original amount of vitamin C. In the other four vegetables—spring cabbage, broad beans, old carrots, and spinach—pressure cooking gave a slightly higher retention in some experiments and a slightly lower retention in others. The average difference in