(Oxford Standards Laboratory)—that is, they were type V, W, X, or Z—but pressure of work prevented more detailed identification.

The MacConkey agar in use at the laboratory gave a high proportion of positive plates in early cases, when a fragment of blood-stained mucus was plated after repeated washing; but results in convalescents, though occasionally positive, were evidently unreliable. The first of the media described by Wilson and Blair (the tellurite-iron citrate-neutral red medium) was brought into use within a few days of the publication of their paper, one plate of Wilson and Blair's medium and one of MacConkey's agar being used for each specimen. The results are shown in the table.

Table of Results with Blair and Wilson's Medium and those with MacConkey's Agar

Case No.	Acute or Convalescent	No. of Flexner Colonies		Remarks
		MacConkey Plate	Wilson-Blair Plate	
240	A	1	1	
246	A	+	+	
259	C	Neg.	+	
262	C	20	Uncountable	Pure culture of Flexner on Wilson-Blair plate
267	A	20	Uncountable	
271	C	10	10	
272	C	2	10	
275	A	Neg.	Uncountable	Pure culture of Flexner on Wilson-Blair plate
284	A	Uncountable	"	
285	A	Uncountable	"	Little to choose between the two media
298	C	Neg.	"	
300	C	"	"	
341	C	9	Neg.	
342	C	1	100	
343	C	1	100	
353	C	Neg.	30	
372	C	Neg.	Uncountable	Pure culture of Flexner on Wilson-Blair plate
383	C	"	30	" " "
384	C	"	100	" " "
392	C	"	10	" " "

Positive on both media : 8
 " " Wilson and Blair's medium only : 10
 " " MacConkey agar only : 2
 Total positive on Wilson and Blair's medium : 18 (90%)
 " " " MacConkey's agar : 10 (50%)

It will be noted that of the 20 consecutive stools from which Flexner dysentery bacilli were isolated 8 were positive on both media, 2 on MacConkey agar only, and 10 on the Wilson-Blair medium only—i.e., 50% of the positive stools would have been missed had only MacConkey's agar been used. Of 46 consecutive negative stools, the Wilson-Blair plate was in one case spoilt; 22 of the remaining 45 were either sterile or showed only minute colonies of *Streptococcus faecalis*. The corresponding MacConkey plates often showed non-lactose-fermenting colonies which required subculture and inoculation of sugars for identification. Thus the Wilson-Blair medium will sometimes have an advantage of two or three days in speed of reporting a negative result, a useful point when many convalescents are being dealt with. On the other hand, this high selectivity must be kept in mind, lest other pathogens be missed, as the following shows:

After some 20 consecutive cases of Flexner dysentery had occurred the stools of certain ward contacts (clinically uninfected) were examined. From one of these Sonne dysentery bacilli were isolated; this patient was presumably a carrier. Later a single case of Sonne dysentery occurred in the same ward. The Wilson-Blair plates from both these patients were of course negative.

From one batch of medium both bile salt and lactose were omitted in error. This batch appeared to give equally good results, but would probably prove unreliable on more extended trial. As Wilson and Blair point out, the medium, after the addition of tellurite, is very sensitive to heat. Neglect of this point leads to loss of selectivity.

The weight of inoculum which the medium will stand is surprising. Apart from *Str. faecalis*, which causes no trouble, and an occasional strain of *B. coli*, little except Flexner's dysentery bacilli will grow upon the medium. Using one or two large loopfuls of heavy faecal suspension or preferably of washed mucus, 27 out of 65 consecutive plates showed no growth of unwanted organisms, the plates being either pure cultures of Flexner's dysentery bacilli or sterile (neglecting the minute colonies of *Str. faecalis*). The growth of Flexner dysentery bacilli is neither inhibited nor enhanced, since subcultures on Wilson and Blair's medium and on MacConkey's agar give equal growth. The heavier growth of Flexner dysentery bacilli on the primary plates is due entirely to the heavy inoculum possible on this medium.

Summary

One of the new media described by Wilson and Blair for the Flexner group has been used in an outbreak of this type of dysentery. The results confirm the value of this medium.

My thanks are due to Dr. J. M. Gibson for permission to publish these notes, and to Dr. Honoria Twomey for much practical help.

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CAROTID CAVERNOUS ANEURYSM WITH FISTULA

BY

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The following case of carotid cavernous aneurysm may be of interest. I saw the patient at the ophthalmic department of a county hospital and later with my colleague Mr. Rupert Parry at Cardiff Royal Infirmary; she was subsequently transferred from the ophthalmic department to the surgical unit under Prof. Lambert Rogers.

Report of Case

History.—Ten days before I saw her in November, 1937, she was awakened by a violent fit of sneezing. Immediately afterwards there was a great strain over her right eye and a hissing noise in the right side of her head which synchronized with the pulse. Her vision gradually depreciated. Two days after sneezing she developed diplopia; her vision with a +1 D sphere was: right 6/24, left 6/18. The pupil on the right side reacted sluggishly and there was paresis of the right external rectus; the fundi were normal. There was a history of antispecific treatment; her Wassermann and Khan reactions were negative, and the systolic blood pressure 190 mm. Radiographs of the skull were negative with the exception of deep meningeal grooves. At Christmas-time she developed total ophthalmoplegia in the right eye, with marked exophthalmos, ptosis, and chemosis; her vision was reduced to 6/60 in this eye. She was given potassium iodide internally, and towards the middle of January, 1938, the exophthalmos diminished somewhat; there were slight movements in the upper lid, and she could look up and in to some extent; the chemosis had lessened, but the large conjunctival veins were very varicose and engorged. Towards the end of January there was ptosis of the left eye, slight chemosis, and diminished eye movements; vision was 6/36 with correction, the fundus normal, and the field of vision full. Total ophthalmoplegia soon followed, with marked exophthalmos and chemosis. There was a recurrence of similar signs in the right eye, but the congestion in the conjunctival veins was more pronounced. There was some surgical emphysema on the right side of the forehead, and the noise in the head seemed to be over the right eye. When a stethoscope was applied to the skull a loud whirring bruit could be heard beneath the right side of the

frontal bone. Compression of the right common carotid artery caused the bruit to disappear, and with it the sensation of noise in the head. There was no sensory change in the trigeminal field. The patient's appearance was as in Figs. 1 and 2.

FIG. 1.

FIG. 2.

At no time was pulsating exophthalmos present; the only fundus abnormality was dilated retinal veins. Fortunately in this case compression of the internal carotid artery did not provoke faintness or giddiness, so, according to Matas's test, operation for ligation of the internal carotid could be undertaken without undue concern.

Operation.—This was performed by Prof. Lambert Rogers on March 29, 1938. At this stage the patient was practically blind in both eyes. Under local anaesthesia the confluence of the three carotid arteries was exposed on the right side of the neck. The internal carotid was noticed to be larger than normal, approximating in size to the common carotid. The internal and external carotid arteries were ligated with silk; the buzzing ceased, and no untoward symptoms were noted. The wound was closed.

Progress.—Six hours later the patient was conscious and able to use both upper limbs. The buzzing in the head had ceased. On the following day she became drowsy, slow in speech, and incontinent of urine; the pupils reacted sluggishly, but the chemosis was less and the exophthalmos had diminished. Left facial weakness was now noted, and left monoplegia. The right plantar reflex was flexor and the left extensor in type. Later in the day the left leg was paralysed. She was now hemiplegic and stuporous. Recovery then gradually took place, and despite some residual hemiplegia she expressed herself well pleased with the result of the operation, since she could now see and the buzzing in her head had ceased. It had previously been most distressing, and had kept her awake at night. Five weeks after the ligation her appearance was as shown in Fig. 3.

FIG. 3.

FIG. 4.

In May, 1938, her vision was: right 6/6, left 6/6; the only abnormalities were a very slight exophthalmos in the right eye and dilated conjunctival veins. I still see her at intervals. There is no change in her eye condition and little change in her hemiplegic condition; she does her housework and goes for long walks. Fig. 4 is a recent photograph.

Probable Pathological Condition

The pathological condition present in this case can only be surmised from the signs and symptoms which were present in the 31 cases mentioned in the literature on this subject in which necropsies were performed. Charcot established the fact that in no part of its course is the internal carotid so thin-walled as in its passage through the cavernous sinus. This portion of it has little support; it is surrounded by the cavernous venous channel, and is consequently liable to aneurysmal development. In this case I will presume that the aneurysm was on the point of bursting; the patient sneezed violently, and the aneurysm burst into the cavernous sinus, the resulting carotid fistula accounting for the immediate pulsating noise in the head. The cavernous sinus dilated because of the consequent arterial pressure within the venous sinus. There was damming up of the venous blood return from the right eye, with consequent exophthalmos. The enlarged cavernous sinus pushed forward the internal carotid artery and bent the optic nerve, with consequent loss of vision. The total ophthalmoplegia and ptosis were due to the kinking of the third, fourth, and sixth nerves in their passage through the dura, and to the direct pressure of the dilated sinus. The vicious back-pressure in the dilated sinus caused dilatation in the intercavernous sinus; then the left cavernous sinus dilated, and as a consequence there followed exophthalmos, ophthalmoplegia, ptosis, chemosis, and loss of vision in the left eye.

Immediately after the external and internal carotid arteries on the right side were ligated the head noise ceased, vision improved, and the other signs gradually disappeared, leaving no doubt that this was a case of right carotid cavernous arteriovenous fistula.

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Medical Memoranda

Thyroxine as a Practical Treatment of Myxoedema

It is well known that thyroxine is as effective as thyroid extract in the treatment of myxoedema. Nevertheless it is seldom used in practice, for it is more expensive than thyroid extract and is poorly absorbed when given by mouth. As thyroid extract could not be tolerated in the following case, injections of thyroxine were given. Our purpose is to describe a possible method of using thyroxine in such cases. Although we do not advocate its employment as a routine treatment of myxoedema, we think it worth recording that it is a practicable alternative.

CASE REPORT

The patient, a married woman aged 56, was first admitted to University College Hospital in August, 1938, having had diarrhoea and vomiting for four months. On admission she was found to be an obvious case of myxoedema, with a dry skin, thin hair, husky voice, and slow cerebration. Her basal metabolic rate was -53%, and while in hospital her pulse rate varied between 58 and 82. On three occasions thyroid extract was given by mouth, in doses varying from 0.5 grain twice daily to 1 grain three times daily, but each time the treatment had to be stopped owing to persistent vomiting.

She was readmitted to hospital in March, 1941, having had an attack of oedema affecting the upper part of her face, including the eyelids, after taking three tablets of thyroid extract. She also complained of pains in the arms, thighs, and calves. Her condition was about the same as it had been when she was first seen, but she had put on a stone in weight. The basal metabolic rate was -49%.