TABLE VI.—Time of Burning in 1,803 Cases

Time	Cases	Time	Cases
12 mn.—	8	12 noon—	126
1 a.m.—	6	1 p.m.—	97
2 "—	8	2 "—	120
3 "—	4	3 "—	154
4 "—	6	4 "—	148
5 "—	7	5 "—	154
6 "—	17	6 "—	111
7 "—	19	7 "—	149
8 "—	50	8 "—	121
9 "—	101	9 "—	109
10 "—	74	10 "—	100
11 "—	58	11 "—	56

ages. The last column of the table shows that the proportion of burns occurring after 8 p.m. was highest between ages 1 and 2 (38% of all burns at this age happened after 8 p.m.). The proportion varies between 23 and 30% at other children's ages, but for all ages over 16 combined it is as low as 11.7%.

TABLE VII.—Age and Time Distribution (1,803 Cases)

Age Group (Years)	Total No. Burned	Up to 8 p.m.		After 8 p.m.		Burns after 8 p.m. as % of Total at Each Age
		No.	%	No.	%	
0—	154	107	7.6	47	12.2	30.5
1—	202	126	8.9	76	19.7	37.6
2—	92	71	5.0	21	5.5	22.8
4—	147	104	7.3	43	11.1	29.2
7—	124	86	6.1	38	9.8	30.6
10—	104	74	5.2	30	7.8	28.8
13—	133	101	7.1	32	8.3	24.3
16+—	847	748	52.8	99	25.6	11.7
All ages	1,803	1,417	100.0	386	100.0	21.4

Extent of the Burn.—The average extent of the burn differed greatly in the sexes. This is seen to be due to the large proportion of males with burns of minor degree, and the greater proportion of severe burns in females (Table VIII). Only 13% of the males had

TABLE VIII.—Extent of Burn in 297 Admissions for which Data were available

Area Burned (%)	Males		Females	
	Cases	% of Total	Cases	% of Total
0—	90	55	54	41
5—	52	32	43	32
15—	12	7	21	16
30+—	10	6	15	11

burns involving over 15% of the body surface, against 27% of the females. In addition, the average area of the burn was 8% in the males and 14% in the females. The tendency for the more severe burns to occur in the female may be related to the difference in clothing.

Deaths due to Burns

In the series of 303 admissions there were 40 deaths. Fatality rates by age and sex are given in Table IX. At every age group the rate is higher for females than for males, and this in itself indicates that the excess fatality rate for females at all ages combined—17.6%, as compared with 9.6% for males—is significant. Using broader groupings, the figures

TABLE IX.—Fatality Rates by Age and Sex in 303 Admissions

Age Group (Years)	Males			Females		
	Cases	Deaths	Fatality Rate (%)	Cases	Deaths	Fatality Rate (%)
0—	77	8	10.4	65	9	13.8
11—	23	0	0	19	2	10.5
21—	13	0	0	13	0	0
31—	22	2	9.1	12	2	16.3
41—	13	1	7.7	9	3	33.3
51—	11	1	9.1	10	1	10.0
61+—	8	4	50.0	8	7	87.5
All ages	167	16	9.6	136	24	17.6
0—	100	8	8.0	84	11	13.1
21—	48	3	6.2	34	5	14.7
51+—	19	5	26.3	18	8	44.4

suggest that the fatality rate is much higher at the older ages; but the total number of deaths is too small for valid statistical tests, so that this conclusion must be accepted with reserve.

When the age distribution of the deaths in this series is compared with that of deaths from burns in Scotland as a whole for 1941* no real difference can be demonstrated (Table X).

TABLE X

Age Group	This Series	Scotland, 1941
0—	17	105
11—	2	21
31—	8	21
51+—	13	89
All ages	40	236

$$\chi^2 = 4.9. \quad P = 0.17.$$

It is possible, therefore, to arrive at a very crude estimate of the number of admissions to hospital from burns in the whole of Scotland. For if 40 deaths arose from 303 admissions, then 236 deaths would have arisen from 1,788 cases of burns serious enough to be admitted to hospital.

The mortality from scalds was lower than that of other types of domestic burn. In 132 cases of scald 9 (6.8%) deaths occurred, whereas 29 deaths occurred in the other 119 domestic cases (24.4% mortality; difference, $17.6 \pm 4.5\%$). Only 2 deaths occurred as a result of industrial burns (3.8% mortality, as compared with domestic 15.1%; difference = $11.3 \pm 5.2\%$).

Summary and Conclusions

This report gives a statistical analysis of 1,803 cases of burns treated in the burns unit of the Glasgow Royal Infirmary between June, 1942, and Aug., 1943, particularly in regard to 303 patients who were admitted to hospital. It is supplementary to the series of papers published in "Studies of Burns and Scalds: Reports of the Burns Unit, Royal Infirmary, Glasgow, 1942-3" (Med. Res. Cncl. Sp. Rep. Ser. No. 249, 1944).

The incidence of burns was high at the younger ages, but the results were more serious in older patients. The incidence was higher among males, but fatality was greater among females.

The injuries were sustained most frequently between 2 p.m. and 8 p.m., but the incidence of burns at a late hour was greatest in the early age groups. Between midnight and 8 p.m. less than half the burns sustained occurred in children under 16, but between 8 p.m. and midnight three-quarters of the accidents occurred at these young ages. The grave social implication of this finding is stressed.

We are indebted to Mr. A. M. Clark, surgeon in charge of the burns unit, for permission to publish these findings; and to Dr. L. Colebrook, Prof. J. W. S. Blacklock, and Prof. M. Greenwood for much helpful criticism.

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CHRONIC INTRACEREBRAL HAEMATOMATA THEIR PATHOLOGY, DIAGNOSIS, AND TREATMENT

BY

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Intracranial haemorrhages may result from rupture either of normal or of diseased blood vessels. Apart from tearing by accidental forces, healthy cerebral blood vessels rarely burst; on the other hand, when weakened by disease or by congenital malformation the walls of a cerebral blood vessel may give way when subjected to normal stresses and strains.

Spontaneous subarachnoid haemorrhage is a well-known cerebral catastrophe, diagnosis readily being made by lumbar puncture. The clinical picture is usually characteristic. A previously healthy young or middle-aged person is struck with a sudden and severe pain in the head which rapidly radiates into the neck and may reach as far as the fingers and toes. Consciousness may or may not be impaired, and when impairment does occur this may be immediate or delayed. Cranial nerve and limb palsies may or may not develop.

* Latest figures available.

Blood is always present in the cerebrospinal fluid. Such haemorrhages result from the rupture of aneurysms of congenital origin, which most commonly arise from the vessels forming the circle of Willis. They do not result from the weakening of the vessel walls by syphilis or by any other degenerative process. Embryologically, the endothelial element of the cerebral vascular system is laid down first as solid columns of cells, and these columns later canalize to form the blood channels. The mesodermal coats of the vessels are laid down in sheets. Occasionally these mesodermal sheets fail to meet perfectly at the apex of the junction of two vessels, thus leaving a weakness or congenital defect. It is at such a site that the endothelial coat is likely to balloon and rupture even under the forces of a normal blood pressure. An alternative hypothesis on the formation of aneurysms is that they arise from the stumps of primitive vessels which normally completely disappear. Since the aneurysms arise from vessels that lie in the subarachnoid space, it is thought that when they rupture bleeding must always take place into the subarachnoid space. Occasionally, however, a congenital aneurysm may become attached to the brain tissue and rupture intracerebrally, to cause a chronic haematoma with resulting hemiplegia, as the following two cases will show.

Case I

On the evening of Jan. 22, 1943, a woman aged 28 was admitted to the Royal Victoria Infirmary, Newcastle upon Tyne, under the care of one of us (A. G. O.). It was ascertained from a relative that two hours previously, while sitting at a table writing a letter, she had suddenly slumped forwards. At this stage of the episode she was still conscious, and complained of severe pains over the right eye; the left side of the mouth was twisted and the left arm and leg were stiff. Vomiting occurred on two occasions. Previous to the attack she had been in excellent health and had not been subject to headaches.

When first examined in hospital, the woman was found to be in a state of semicomatose; the left side of the body was paralysed; both pupils were of mid-dilatation and both reacted briskly to light; the optic nerve heads were not oedematous; the pulse rate, blood pressure, and temperature were normal; and respiration was deep and rapid. A diagnosis of a spontaneous subarachnoid haemorrhage was made, and this was later confirmed by lumbar puncture, the cerebrospinal fluid being heavily stained with blood. Later, special investigations proved that there were no metabolic disturbances resulting from dysfunction of the kidneys, pancreas, or liver. The blood Wassermann reaction was negative. We came to the conclusion that the subarachnoid haemorrhage was due to rupture of a congenital aneurysm situated somewhere on the circle of Willis, and probably on the right middle cerebral artery because of the left-sided hemiplegia.

On the third day after admission to hospital the woman was fully conscious and fully orientated, but the left-sided hemiplegia remained complete; she felt ill and suffered from considerable pain in the head, particularly over the right eye. From this time onwards very little change was noticed, save that occasionally she would become drowsy, when co-operation was imperfect. She complained continuously of severe headaches.

On Feb. 14 papilloedema was noticed for the first time; the hemiplegia was still complete, there being no evidence of any kind of improvement. On Feb. 16 she was transferred to the neurosurgical unit at the General Hospital, Newcastle upon Tyne, under the care of one of us (G. F. R.). At this date the neurological picture was as follows:

The woman was fully conscious and perfectly co-operative. Sight, speech, smell, and taste were normal. The whole of the left side of the body was densely paralysed. There was no sensory loss or any abnormal cerebellar signs. Both pupils were dilated and both reacted to light and to accommodation; there was a bilateral papilloedema of one diopetre. The blood pressure was 130/80.

Case II

During the evening of July 2, 1943, a young man aged 23 collapsed in his seat at a cinema and his friends carried him into the vestibule. Later he was taken home, and arrived there in a state of confusion, complaining of very severe headache. The next day he was admitted to the Royal Victoria Infirmary under the care of one of us (A. G. O.). At this time the man was in coma and the whole of the left side of his body was paralysed. The pupils were contracted and fixed and both eyes were pulled to the right; the optic disks were flat; both plantar reflexes were extensor; the knee-jerks were increased and the abdominal reflexes were absent. The man's head was extended and there was marked neck rigidity.

A diagnosis of subarachnoid haemorrhage was established by lumbar puncture. In the left lateral position the pressure of the

cerebrospinal fluid, as measured by manometry, was 240 mm. of water, the normal figure lying between 150 and 50 mm. Further investigations proved that there was no generalized disease of the cardiovascular system, the blood pressure being normal. No metabolic disturbances were found to point to dysfunction of the kidneys, pancreas, or liver. The blood Wassermann was negative.

The next day the man was still unconscious, but less deeply so than on the day of admission; the hemiplegia on the left side, however, was still complete. On July 6, although his mental condition had improved, he was still confused. The pressure of the cerebrospinal fluid was 80 mm. The red blood cells had disappeared from the fluid, but there was xanthochromia. On July 18 the man was still complaining of severe headaches, and the pressure of the cerebrospinal fluid had risen to 180 mm. During the next two weeks his mental condition remained unchanged and there was no improvement in the hemiplegia. On Aug. 2 he was transferred to the neurosurgical unit at the General Hospital, Newcastle upon Tyne, under the care of one of us (G. F. R.). At this time the clinical picture was as follows:

The man looked ill and obviously had lost a good deal of weight. He was confused, but could co-operate in a simple neurological examination. The left side of his body was completely paralysed and the reflexes were typical of those of an upper motor neurone lesion. There was no sensory loss, nor were there any signs to point to dysfunction of the cerebellum. A bilateral papilloedema of one diopetre was present. The blood pressure was 140/95.

Discussion on Diagnosis and Treatment

In the above two cases the diagnosis of an intracranial congenital supraclinoid aneurysm was reasonably certain. Also, because of the hemiplegia, it was obvious that the aneurysms arose either from the middle cerebral artery or from some vessel close to it. From experience it is known that, with complete rest in bed, bleeding in this type of case will usually cease spontaneously and the hole in the aneurysm close by natural processes. Ligation of the internal carotid artery in the neck, although of great value in the treatment of a cavernous or subclinoid aneurysm, is of little use in the treatment of supraclinoid aneurysms. Repeated bleedings, in spite of rest, in ruptures of supraclinoid aneurysms are best treated by exposure of the aneurysm and local ligation or repair by muscle grafts.

It was, however, in the problem of the hemiplegia that we were particularly interested, and we came to the conclusion that the possible causes of the paralysis were as follows: (i) Obliteration of the channel of the middle cerebral artery either by rupture or by thrombosis. (ii) Pressure on the middle cerebral artery by the aneurysm itself. (iii) Pressure on the middle cerebral artery by a localized blood clot in the Sylvian fissure. (iv) Pressure due to an intracerebral blood clot. (v) Hydrocephalus due to blocking of the cerebrospinal fluid pathways.

Some years ago one of us (G. F. R.) saw a young woman who had had a spontaneous subarachnoid haemorrhage, due, no doubt, to rupture of a congenital aneurysm. From the start she was densely hemiplegic; papilloedema developed at a later date. In the belief that there might be a large surface clot compressing the convexity of the brain, the motor cortex was exposed by raising a bone flap. A large subarachnoid blood clot was in fact found and easily removed. Closure of the wound was so made that a subtemporal decompression was provided. After the operation the hemiplegia did not recede and, in spite of repeated lumbar punctures which proved that there was no further bleeding, the decompression continued to bulge. The girl was densely hemiplegic a year later.

In view of this experience, it was thought that some factor other than surface compression was the cause of the hemiplegia in the two cases under consideration. The secondary rise in intracranial pressure, as shown by the delayed papilloedema, was very suggestive of recurrent bleeding, although lumbar puncture proved on several occasions that bleeding into the general subarachnoid spaces had not occurred. In view of our uncertainty of the cause of the secondary rise in intracranial pressure and of the hemiplegia, it was decided to explore the region of the anterior end of the Sylvian fissure wherein the middle cerebral artery and its many branches lie.

Operation Notes and Results

Case I

On Feb. 18, 1943, under local analgesia, a temporal exposure was made through a muscle-split approach (Rowbotham, 1942). A

piece of bone about the size of a five-shilling piece was removed. No blood was found in the extradural or subdural spaces. There was no blood clot beneath the arachnoid mater. The brain was bulging, and was obviously under pressure. All the exposed brain tissue was stained a yellowish colour. The upper temporal gyrus was widened and flattened, and beneath it a softening could be palpated with the finger-tip. The frontal gyri were normal.

By means of a brain cannula a large cyst was found at a depth of 2 cm. in the temporal lobe of the brain immediately below the Sylvian fissure. An incision was made into the brain tissue and the cyst opened; this was about 4 cm. in diameter. Its contents were fluid, consisting of altered black-coloured blood, and were easily removed by gentle suction. The wall of the cyst was smooth and, so far as could be judged by the naked eye, was formed by condensed brain tissue. There was certainly no adventitious membrane such as invariably surrounds a chronic subdural haematoma. In the roof of the cyst a small mound of hard blood was firmly attached to the cyst wall. This no doubt marked the site where rupture of the aneurysm had occurred. Also, it was obvious that the parent vessel was the middle cerebral artery or one of its main branches as it lies deep and high in the Sylvian fissure. In order to minimize the danger of further bleeding, the blood clot was covered with a muscle graft. The dura was left open to provide a decompression; the muscle and scalp layers were re-formed in the usual way, drainage of the cyst being maintained through a fine rubber tube for 12 hours.

Immediate Result.—At the end of the operation the woman's condition was no worse. The next morning there was definite improvement; she was much more alert and her attention was better sustained. Already she could make weak movements with her paralysed arm and leg. From this time onwards improvement was rapid, full power in the limbs being regained before the reflexes returned to normal. The papilloedema had receded by the third day. On the tenth day after operation the woman got out of bed, and finally walked out of hospital on March 6—that is, 16 days after operation.

Late Result.—Her convalescence was steadily progressive, and six weeks after leaving hospital she had regained her normal health. Later she became pregnant, and a child was born on Feb. 2, 1944. After the confinement her general health deteriorated considerably; she became extremely nervous, and for a period lost the use of her left arm. In September she went away for a holiday, and on her return had made a complete recovery. She was examined on Dec. 18; she then had no complaints and stated that she was feeling very well. She was a plump, healthy-looking woman, and neurologically there was not a single abnormal sign.

Case II

On Aug. 4, 1943, under local analgesia, a right temporal exploration was made by means of a periosteal slide (Rowbotham, 1942). A square of bone was removed from the anterior half of the exposure. On opening the dura mater no blood or any other abnormal collection of fluid was found in the subdural space. There was no clot in the subarachnoid space. The brain was bulging, and was obviously under pressure. All the exposed brain tissue was stained a yellowish colour. The gyri of the frontal lobe were widened and flattened, and beneath them a softening could be palpated with the finger-tip. The exposed gyri of the temporal lobe were normal.

By means of a brain cannula a large cyst was found at a depth of 2 cm. in the frontal lobe of the brain immediately above the Sylvian fissure. An incision was made into the brain tissue and the cyst opened. The cyst was about 4 cm. in diameter. Its contents were fluid, consisting of altered black-coloured blood, and were easily removed by gentle suction. The wall of the cyst was smooth and, so far as could be judged macroscopically, was formed by condensed brain tissue. There was certainly no adventitious membrane such as invariably surrounds a chronic subdural haematoma. In the floor of the cyst a small mound of hard blood was firmly attached to the cyst wall. This no doubt marked the site of rupture of the aneurysm. Also, it was obvious that the parent vessel was the middle cerebral artery or one of its main branches as it lies deep and high in the Sylvian fissure. In order to minimize the danger of further bleeding, the blood clot was covered with a muscle graft. The dura was left open to provide a decompression; the muscle and scalp layers were re-formed in the usual way, drainage of the cyst being maintained through a fine rubber tube for 12 hours.

Immediate Result.—At the end of the operation the man's condition was no worse. The next morning there was definite improvement. He looked better and said that he felt better. He was much more alert, and his attention was better sustained. Already the hemiplegia was beginning to recede; weak but definite movements were possible in both the leg and the arm. From this time onwards improvement was rapid, full power returning before the reflexes became normal. On the fifth post-operative day there was no sign

of papilloedema and the decompression was flat. On Sept. 16 the man had completely recovered from his illness and walked briskly out of hospital.

Late Result.—Some weeks after his discharge from hospital he had a peculiar feeling as if things were receding from him; also the left side of the face began to twitch. He did not lose consciousness, and felt well again in a few hours. A similar episode occurred some weeks later. In Feb., 1944, he returned to his employment as a melter in a steel-smelting works and has worked regularly ever since, no time having been lost because of ill-health. On examination on Nov. 20 he was a healthy-looking man of excellent physique. He had no complaints, and, apart from the operative defect in his skull, which is of little importance, not a single abnormal sign was to be found.

Commentary on the Underlying Neuro-physiological Mechanism

Probably a combination of cerebral shock and widespread arterial spasm accounted for the sudden impairment of consciousness that occurred at the moment of rupture of the aneurysm. The sudden loss of power on one side of the body probably was due partly to the effect of the above two factors and partly to the fact that the blood which should have been passing along the channel of the middle cerebral artery was passing out of the hole in the aneurysm. The deepening unconsciousness and deepening paralysis which further questioning confirmed or established were due to increasing haemorrhage. As the bleeding ceased and as cerebral shock and arterial spasm receded, consciousness to a large extent returned. The mental dulling which remained was probably due to the pressure of the blood that had been extravasated.

On the other hand, even though consciousness largely returned there was no improvement in the hemiplegia in either of the two cases under consideration. Subsequent events, however, proved that the channel of the middle cerebral artery had not been irretrievably destroyed, because full recovery of function finally did take place. Therefore, although we did not know it at the time, the paralysis must have been due either to vascular spasm or to pressure interfering with the arterialization of the motor cortex. Moreover, the ischaemia could not have been complete, because absolute ischaemia even for a few moments leads to irreversible injury and to permanent paralysis of function.

We felt that if the paralysis were due to the pressure of the extravasated blood it would be reasonable to assume that with time the blood would absorb and the paralysis resolve. The opposite, in fact, happened. Instead of the patients slowly recovering, intracranial pressure began to rise after an interval, and it is more than possible that they would have died of rising intracranial pressure. Even if death had not resulted it is very unlikely that recovery from the paralysis would have occurred from natural processes. The secondary rise in pressure was due to the breakdown of the intracerebral blood clot. As the clot broke down its molecular content increased, leading to a rise in osmotic pressure, so that fluid was drawn into the blood clot from surrounding tissues to form a blood cyst of increasing size. This increase in size accounts for the secondary rise in intracranial pressure, which was evidenced in both cases by the appearance of papilloedema and measured in one case by lumbar manometry. After drainage of the blood cyst recovery from the hemiplegia rapidly took place, thus proving the paralysis was due to the pressure of the cyst. The pressure could either have acted directly on the motor cortex by pushing up from below or have acted on the middle cerebral artery, causing partial obstruction of its lumen.

We came to the conclusion that pressure on the middle cerebral artery was probably the mechanism causing the hemiplegia, since recovery from the paralysis was so rapid. In our experience, in cases of hemiplegia due to long-standing compression, relief of the pressure does not lead to such quick improvement as occurred in the above two cases. It might be objected that recovery of function takes place rapidly after the removal of a large extradural blood clot caused by bleeding in the middle meningeal vessels. In such cases, however, the compression has lasted at most for one or two days, whereas in the two cases under discussion the compression and the hemiplegia had lasted 27 days in one case and 32 days in the other.

It might be suggested that at the time of operation it would also have been wise to expose the aneurysm, removing it or ligating its base, as well as draining the haematoma, so as to prevent the recurrence of further haemorrhages. This was not done because opening of the anterior end of the Sylvian fissure introduces the danger of starting up bleeding which is difficult to control, leading to the possible necessity for ligating the middle cerebral artery, with resulting hemiplegia of a permanent kind.

We should like to point out that the nature of the illness was explained to each patient before leaving hospital. The decision not to lead a completely sedentary life—one to have a child and the other to work before an open furnace—was made by the patients themselves. We believe they made the right decision. It is wrong to allow the sword of Damocles to hang too closely above one's head.

The above, so far as we are concerned, is a new venture into the therapeutics of intracerebral haemorrhages, and it has been a very successful one. It must be realized, however, that we chose to operate at the stage of the chronic subdural haematoma and not at the stage of an acute intracerebral haemorrhage. Possibly in those cases of hemiplegia following a subarachnoid haemorrhage it is also wise to operate even when there is no secondary rise in intracranial pressure. We doubt, on the other hand, whether surgery has much to offer in the treatment of ordinary apoplexy or, in fact, in the early stages of a cerebral haemorrhage due to any cause.

1. Ophthalmic artery. 2. Anterior cerebral artery. 3. Middle cerebral leath of arteries. 4. Posterior communicating artery. 5. Internal carotid artery. 6. The point at which the internal carotid artery leaves the cavernous sinus to enter the subarachnoid space. 7. The region in which the chronic cerebral haematomata were found in the two cases described in this paper.

Arteries which arise above the level of the cavernous sinus or clinoid process are classified as supraclinoid aneurysms; they burst usually into the subarachnoid space, rarely into the brain tissue, and sometimes into both. Intraclinoid aneurysms rarely burst either into the subarachnoid space or into the cavernous sinus itself. They do, however, commonly stretch the structures enclosed within the walls of the cavernous sinus, causing in particular a third-nerve palsy.

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With its issue for January, 1945, *The World's Children* starts its twenty-fifth annual volume and reverts to monthly publication. According to the leading article, the journal began as a record of the suffering caused to children as a result of the first world war, and in those days it earned the reputation of being, in the words of a Scottish newspaper, "the most melancholy magazine in existence." But there have also been bright spots in the twenty-four years' record of the welfare of children, and they too have had full place in the pages of *The World's Children*. It is published by the Save the Children Fund, 20, Gordon Square, W.C.1.

SULPHANILYLBENZAMIDE, SULPHANILYL-AMIDOBENZAMIDE, AND SUCCINYL SULPHATHIAZOLE IN THE CHEMOTHERAPY OF SONNE DYSENTERY

BY

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The treatment of bacterial infection of the intestinal tract by chemical agents was greatly encouraged by the introduction of sulphanilylguanidine (sulphaguanidine) (S.A.G.) by Marshall and his colleagues. The use of the drug is based upon the poor absorption from the gut of a compound soluble enough in the gut contents to be antibacterial to the pathogens concerned. S.A.G. is a rather poor antibacterial substance, and, while effective against dysentery due to Flexner strains, it has proved much less satisfactory in Sonne infections, particularly in effecting bacteriological cure of the Sonne carrier. To meet the difficulty, Brownlee and Tonkin (1941) have suggested the use of sulphanilylbenzamide (S.A.B.); and more recently sulphanilylamidobenzamide (S.A.A.B.) has been introduced. The constitution of these two drugs is shown below:

A point bearing on the toxicity of these two drugs is that, while their absorption is greater than that of S.A.G., they are acetylated to a less degree. The solubility of S.A.B. varies from 91 mg. per 100 c.cm. in an acid urine (pH 5.8) to 484 mg. in an alkaline urine (pH 7.8). The corresponding solubilities of S.A.A.B. are 112 and 95 mg. per 100 c.cm. (Brownlee, unpublished communication).

We are reporting the results of a clinical trial of S.A.B. on 41 patients of all ages suffering from Sonne dysentery, of S.A.A.B. on 112 patients, and of succinyl sulphathiazole (sulphasuxidine) (S.S.) on a small series of 26 for comparison. The majority were barrier-nursed, while the remainder were nursed in separate glass-walled rooms. In Sonne dysentery the symptoms are usually mild and the disease is more of a nuisance than a danger to the patient. Chemotherapy is therefore directed to the clearance of *Bact. sonnei* from the bowel rather than cure of the "illness," but it does in fact have a beneficial effect in that, in acute cases, within 24 to 48 hours the stools are usually formed, are normal in colour, and contain no blood or mucus when S.A.B. is used. This is true, though to a slightly less degree, for S.A.A.B.

The use of the rectal swab (Cruickshank and Swyer, 1940) and the introduction of desoxycholate citrate agar (Hynes, 1942) have shown that the convalescent carrier is much more common than was at one time believed. In fact, it has been stated that more than half of the clinically infected cases may still be excreting the organism 3 to 4 weeks after onset. Therefore elimination of this carrier state is of the greatest importance. The existence of the carrier being now generally accepted, control untreated cases were dispensed with in the present series and all bacteriologically proved cases were drug-treated. The patients' stay in hospital was divided into a preliminary observation period which averaged 9 days for the S.A.B., 4.5 for the S.A.A.B., and 6 for the S.S. series respectively, a treatment period of 5 days, followed by a second observation period averaging 14.8, 10.9, and 13.2 days for the same three series. As will be seen from an examination of Table II, the length of both preliminary and second observation periods varied considerably. This was caused by such events as concurrent diseases which delayed discharge of