At the conclusion of the arteriography, while the common carotid arteries were still exposed, the left common carotid artery was ligated completely. I realized it was only a temporizing measure, but I reasoned that if the direct attack could be deferred for a few days some healing might occur in the wall of the aneurysm and so lessen the risk of a torrential haemorrhage occurring from the aneurysm when it was exposed.

Two weeks later, by means of a coronal scalp flap and a left frontal bone flap performed under general anaesthesia, the left-sided aneurysm was attacked. The left frontal lobe was elevated off the floor of the anterior cranial fossa, as in a pituitary operation, and the left internal carotid artery was exposed as it lay immediately behind and lateral to the optic nerve. The artery was then followed to its bifurcation, and in the process of doing this the neck of the aneurysm was exposed in the angle between the diverging anterior and middle cerebral arteries. The neck was about 7 or 8 mm. in diameter. The fundus of the aneurysm was not seen, for it projected upwards into the frontal lobe. Two silver clips, each slightly longer than standard size, were then applied to the neck of the sac in such a way that they occluded it without narrowing the origins of the anterior and middle cerebral arteries. The clips were then buttressed by being wrapped round with muscle, and the craniotomy was closed.

Ten days later the coronal scalp flap was reopened and a right frontal craniotomy performed. The smaller aneurysm on the right side was thus located, being treated in the same way by the application of silver clips to its neck (Fig. 3).

FIG. 3.—Post-operative radiograph of skull showing position of silver clip placed on the necks of the two aneurysms, and also the position of the two bone flaps.

Convalescence from both these operations was straightforward, and the patient made a complete recovery except for bilateral loss of smell, indicating damage to his olfactory nerves during elevation of the frontal lobes. Within two months of his second craniotomy he had resumed his work as a company accountant, and one and a half years later he reported that he was symptom-free except for loss of smell, and that he had been most successful in his profession. His wife in an independent statement reported that he was mentally normal in every way.

I wish to thank those physicians who entrusted my unit with their patients. Fifteen patients were referred by colleagues in the Dunedin Hospital, 14 by colleagues in the Christchurch Hospital, and the remainder from other hospitals. Four of the cases were treated by my associate, Mr. Anthony James, who has kindly allowed me to include them in this report. I particularly wish to acknowledge

the encouragement which I received from Professor F. G. Bell, Dr. J. A. D. Iverach, and Dr. R. H. Q. Baxter during my first endeavours. Dr. A. C. Begg and Mr. M. Allen collaborated with the radiographic studies.

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PRESENT STATUS OF THE Rh-Hr BLOOD TYPES

BY

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There have been considerable advances in our understanding of the Rh-Hr blood types during the past few years. The purpose of this article is to summarize the present status of the subject in order to take into account especially the newer knowledge concerning genes r^y , R^z , R^{1w} , and the R^0 variants.

At present the existence of three "standard" Rh antisera is recognized—namely, anti-Rh₀, anti-rh', and anti-rh". Sera giving reactions reciprocally related to those of the Rh antisera are designated as Hr antisera, and two such sera have been shown to exist—namely, anti-hr' and anti-hr". Despite claims to the contrary published in the literature, the existence of an antiserum and corresponding factor reciprocally related to Rh₀ has not been convincingly demonstrated to date (Wiener, 1950). With the aid of the three standard Rh antisera the existence of eight sharply defined allelic genes has been established. These genes can be identified by the reactions with the three Rh antisera alone (Wiener and Hyman, 1948), and the reactions with the Hr antisera merely define additional characteristics not essential for their differentiation (see accompanying Table).

Table Showing Present Status of Rh Series of Allelic Genes

Gene	Corresponding Agglutinin	Reactions with Rh Sera				Reactions with Hr Sera	
		Anti-Rh ₀	Anti-rh'	Anti-rh"	Anti-rh ^w	Anti-hr'	Anti-hr"
r^y	rh	Neg.	Neg.	Neg.	Neg.	Pos.	Pos.
r^y	rh'	"	Pos.	"	"	Neg.	"
r^y	rh"	"	Neg.	Pos.	"	Pos.	Neg.
r^y	rh ^w	"	Pos.	"	"	Neg.	Pos.
R^0	Rh ₀	Pos.	Neg.	Neg.	"	Pos.	Pos.
R^1	Rh ₁	"	Pos.	"	"	Neg.	"
R^2	Rh ₂	"	Neg.	Pos.	"	Pos.	Neg.
R^3	Rh ₃	"	Pos.	"	"	Neg.	Pos.
R^{1w}	Rh _{1w}	"	Neg.	Pos.	"	Pos.	Pos.
R^0	Rh ₀	Weak	Neg.	"	Pos.	Pos.	"
R^1	Rh ₁	"	Pos.	"	"	Neg.	"
R^2	Rh ₂	"	Neg.	Pos.	"	Pos.	Neg.

The eight allelic Rh genes are designated (Wiener, 1949) by the symbols r , r' , r'' , r^v , R^0 , R^1 , R^2 , and R^z , respectively. As shown in the table, the capital "R" is used to represent these blood properties which contain the factor Rh₀; the small "r," on the other hand, implies the absence of factor Rh₀. For verbal use, however, it is not necessary to say "large R" or "small r" as the case may be, because the qualifying subscripts and superscripts fully identify the symbols.

To avoid ambiguity, distinctive symbols are used for genes and agglutinogens; the gene symbols are printed in italics, have superscripts only, and the "h" is omitted. For example, "R-one," which is easy to say, clearly refers to a gene, and "R-H-one," which is also easy to say, clearly refers to an agglutinin or a blood type. The equivalent designation for this type in the Fisher-Race nomenclature would be "large C, large D, small e," which is neither easy to say nor easy to absorb, and in this nomenclature there are no distinctive symbols for genotypes and phenotypes. Phenotype Rh₂rh includes genotypes R^2r , R^2R^0 , and R^0r'' . In the Fisher-Race nomenclature, in this case for both phenotype and genotype the same symbol cDE/cde is used, and this is misleading if not incorrect, especially since no tests have been made for "little d" factor.

In 1943 I observed that there were bloods, especially among members of the Negroid race, which reacted weakly with one or more of the Rh antisera (Wiener, 1944). These bloods were said to contain "intermediate" Rh factors or Rh variants, and it was recognized that these weak reactions were due to some qualitative genetically determined difference in the agglutinin. It was pointed out that when these Rh variants are taken into account the postulated series of Rh allelic genes had to be extended considerably. If, as I postulated, these weak reactions are due to qualitative difference in the agglutinogens it should be possible to find new Rh antisera specific for these Rh variants. This expectation has been fulfilled in part through the work (Race *et al.*, 1948) on variants of the rh' (C) factor.

Some individuals of type Rh₁ have blood containing a variant of the rh' factor as shown by the weak reactions with anti-rh' serum. Among these the existence of an agglutinin Rh₁^w (CC^wDe) has been completely established, and potent and specific anti-rh^w (anti-C^w) sera are available to test for this variant (Callender and Race, 1946-7). In the papers of British workers the Rh₁^w agglutinin and its corresponding gene have been designated as C^wDe. If the policy that the designations for the genes and agglutinogens should fully define the serological reactions is to be continued then the correct symbol should be CC^wDe instead of C^wDe (Wiener and Gordon, 1949). With regard to other rh' variants—namely, bloods that react weakly with anti-rh' sera but are rh^w negative—specific antisera are not available and these are pooled together under the original symbol.

Of considerable practical importance are the variants of the Rh₀ factor (Wiener, 1944; Race *et al.*, 1948-9), because the Rh₀ factor is the one usually responsible for clinical complications. When mothers are sensitized to the Rh₀ factor, babies whose blood contains an Rh₀ variant have proved to be erythroblastotic; similarly, transfusion of blood containing an Rh₀ variant to a patient sensitized to the Rh₀ factor could give rise to a haemolytic reaction. Unfortunately, no sera have been obtained to date specific for the Rh₀ variants, which are still recognized primarily by their weaker reactions with Rh₀ antisera. Nevertheless, some distinctive designation for bloods containing Rh₀

variants seems desirable, and it is therefore proposed to use a capital Germanic **R** to indicate the presence of an Rh₀ variant, as shown in the Table. For oral use, however, the term "Rh₀ variant" is convenient, and is therefore retained.

It hardly seems necessary to point out once more the need for a single uniform Rh-Hr nomenclature, such as exists for the ABO and MN systems. To argue that duplicate nomenclatures are simpler and clearer than a single one is sheer nonsense. It is hoped that Sir Lionel Whitby, President of the International Society of Haematology, will appoint a fact-finding committee to investigate this matter. The appointment of such a committee now would give interested workers ample time to submit to its members evidence relating to this question. After the evidence had been digested a report could be submitted to the meeting, and published after it had been endorsed by the society.

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FOETAL LOSS IN BREECH PRESENTATION: PRIMIGRAVIDAE AND MULTIGRAVIDAE COMPARED

BY

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There is a general impression that breech presentation in a woman who has already borne babies is a less serious matter than in a woman pregnant for the first time. Some doubt has been thrown on the accuracy of this impression by the published figures of foetal loss in breech delivery—for example, Peel and Clayton (1948), who presented a large collected series (Table I).

TABLE I.—The "Corrected" Foetal Mortality in Breech Presentation (the Combined Results from the Series of Six Separate Authors—Collected by Peel and Clayton (1948))

	Primigravidae	Multigravidae	Total Breech Deliveries
Combined figures	11.3% (738 cases)	9.8% (919 cases)	1,657 cases
Range in the individual series	5.3%–29%	2.2%–23.2%	

S.E. of the difference = 1.5; observed % difference = 1.5. Thus the difference in this large composite series is not significant.

It was decided to compare the results of primigravid and multigravid breech delivery obtained in St. Mary's Hospital, Manchester, and to study the factors leading to the foetal loss.

The material comprised 243 breech deliveries and extractions performed in 1947 and 1948. These two years were chosen because I was resident in the hospital, and delivered some of the cases, supervised others, and knew the conditions under which the remainder were delivered. It was a policy to admit to hospital for delivery all breech presentations seen at the clinic, both in primigravidae and multigravidae, and, as a rule, twin pregnancies were delivered in hospital rather than by the district service. Thus,