The Ciba Foundation

for the Promotion of International Cooperation in Medical and Chemical Research

41 PORTLAND PLACE, LONDON W1N 4BN

PROGRAMME AND SYNOPSES OF PAPERS OF SYMPOSIUM (INCORPORATING 2nd CAMBRIDGE OPHTHALMOLOGICAL SYMPOSIUM)

CORNEAL GRAFT FAILURE

Chairman: B. R. JONES

Wednesday, Thursday and Friday,
15, 16 and 17 November 1972

Programme

WEDNESDAY, 15 NOVEMBER

9.30—10.00	Assembly: coffee
10.00—10.15	Opening by Ruth Porter
10.15—10.30	B. R. JONES: Chairman's introduction: objectives

Session 1: 10.30—12.50

Physiology and pathology of donor material

10.30—10.55	A. E. MAUMENEE: Clinical patterns of corneal graft failure
10.55—11.15	Discussion
11.15—11.35	C. H. DOHLMAN: Pathophysiology of graft failure
11.35—11.55	Discussion
11.55—12.15	E. S. SHERRARD: Quality of donor corneas for keratoplasty
12.15—12.50	Discussion of previous paper and general discussion of meeting so far
12.50	Sherry
13.10	Lunch at the Ciba Foundation

Note:

Please inform a member of the staff if lunch is not required. The wives of overseas members may join the group for lunch if they wish. During this interval informal photographs will be taken.

Session 1 (continued): 14.15—15.20

14.15—14.35	M. J. ASHWOOD-SMITH: Problems of cell survival after freezing and thawing with special reference to the cornea
14.35—15.20	Discussion of previous paper and general discussion of physiology and pathology of donor material
15.20—15.40	Tea

Session 2: 15.40—17.30

Immunological aspects

15.40—16.10 R. E. BILLINGHAM: Immunologically privileged sites and tissues

16.10—16.30 Discussion

16.30—16.55 A. M. SILVERSTEIN: Transplantation biology of the cornea

16.55—17.30 Discussion

18.30—20.00 RECEPTION at the Ciba Foundation at which Sir John McMichael, FRS, with Lady McMichael, will receive the guests.

Note:

Individual invitations will not be issued but it is hoped that all members and their wives will be able to come to the reception. Other guests will be present. Dress: informal.

THURSDAY, 16 NOVEMBER

Session 3: 9.30—10.55

Immunological and clinical aspects

9.30— 9.55 F. M. POLACK: Corneal graft rejection: clinicopathological correlation

9.55—10.15 Discussion

10.15—10.35 A. A. KHODADOUST: The allograft rejection reaction.

10.35—10.55 Discussion

10.55—11.15 Coffee

Session 4: 11.15—12.40

Clinical aspects and management of corneal graft failure

11.15—11.25 P. G. WATSON: Recurrence of host disease in the graft

11.25—11.55 Clinical experiences: Statements by members of the group

11.55—12.40 General discussion of clinical experience

12.40 Sherry

13.00 Lunch at the Ciba Foundation

Session 5: 14.15—18.00

Non-immunological and para-immunological aspects

14.15—14.35 M. FINE: Role of corneal vascularization in corneal graft reactions in man

14.35—14.55 Discussion

14.55—15.15 T. E. MOORE: Influence of surgical techniques and other mechanical factors on corneal graft failure

15.15—15.35 Discussion

15.35—15.55 Tea

15.55—16.15 N. S. C. RICE: Problems of corneal grafting in herpetic keratitis

16.15—16.35 Discussion

16.35—16.45 A. E. MAUMENEE: Role of steroids in the prevention of corneal graft failure

16.45—17.00 Discussion

17.00—18.00 General discussion

19.00 for 19.30 DINNER at the Ciba Foundation at which Professor F. G. Young, FRS, Trustee of the Ciba Foundation, will preside, with Dr Ruth Young.

Note:

Individual invitations will not be sent, but it is hoped that all members and their wives/husbands will attend the Dinner. Anyone unable to do so should inform the Deputy Director in advance. Dress: informal.

Subconjunctival

FRIDAY, 17th NOVEMBER

Session 6: 9.45—12.30

Prevention and treatment of graft failure: specific approaches

(i) Immunosuppressive therapy

9.45—10.05	M. C. BERENBAUM: Biological basis of immunosuppression
10.05—10.25	Discussion
10.25—10.45	D. B. EVANS: Some clinical problems of immunosuppression
10.45—11.10	Discussion
11.10—11.30	Coffee

(ii) Tissue-typing for corneal grafting

11.30—12.00	J. R. BATCHELOR and T. A. CASEY: The influence of HL-A compatibility upon the fate of corneal grafts.
12.00—12.30	Discussion
12.30	Sherry
12.50	Lunch at the Ciba Foundation

Note:

During this interval the photographs taken on Wednesday will be on sale

Session 6 (continued): 14.00—15.40

14.00—14.20	N. EHLERS: Influence of histocompatibility on the fate of the corneal graft
14.20—14.30	P. G. WATSON: Difficulties in the use of tissue-typing
14.30—14.50	Discussion
14.50—15.30	Final group discussion on prevention and treatment of corneal graft failure, to be opened by B. R. JONES
15.30—15.40	Chairman's closing remarks
15.40	Tea

END OF SYMPOSIUM

CLINICAL PATTERNS OF CORNEAL GRAFT FAILURE

A. E. Maumenee

The Wilmer Institute, Johns Hopkins Hospital, Baltimore

Corneal transplants tend to fail or become opaque for several different reasons. The first of these is the immediate opacification of the graft that occurs from the first day following corneal transplantation to approximately the second or third week. In technically successful transplants this is usually due to faulty endothelium on the donor button. There are other obvious early failures which are due to technical difficulties in the operative procedure.

These may vary from apposition of the donor material, flat anterior chambers, to trauma of the lens due to technical errors. Infections may result from infected donor material or stitch abscesses during the immediate post-operative period. A very interesting phenomenon that needs further investigation is the higher percentage of immediate corneal oedema that occurs in technically successful corneal grafts in aphakic eyes.

The incidence of corneal opacification as a result of an immunological response varies tremendously, dependent upon the condition of the recipient cornea. Such an immune response may occur as early as two to three weeks after transplantation, or as late as fifteen years after operation. In penetrating corneal grafts the reaction begins with an accumulation of lymphocytic cells on the posterior surface of the corneal button, then increases to a circumcorneal injection and a positive aqueous ray. Destruction of the endothelial cells is manifest by diffuse stromal corneal oedema. The recognition of this type of reaction is extremely important from a clinical point of view, for if detected in its earliest phases the destruction of endothelial cells can frequently be suppressed by the use of steroid therapy. Stromal and epithelial tissues may also be injured by the immune response,