TREATMENT OF ACTIVE TRACHOMA BY DIATHERMY

By Paul A. Tan and S. H. Tan

SYNOPSIS

A method of treatment of trachoma entirely by diathermy is described.

In essence it consists of the production of a superficial burn to the palpebral conjunctiva, caruncle, plica (and bulbar conjunctiva when this is involved).

A current not exceeding 30 milliamperes is used. The application of ice packs to the lids post-operatively is of utmost importance during the first 48 hours continuously.

Cure is effected within 3 weeks.

There are ½ billion people today in this world suffering from trachoma of whom twenty million are left blind as a result of it.

In the 1930's, up to 30% of diseases of the Outer Eye seen at the Eye Hospital, Singapore was due to trachoma. At the present time, however, the incidence is less than 5%, this being certainly due to the better standards of hygiene.

Before the introduction of the sulphonamides in 1938 no specific treatment for trachoma was known. Today, resolution of Stage I of the disease with systemic sulphonamides and topical tetracyclines or erythromycin can be expected in a matter of weeks, while at the next stage (Stage II, follicular or papillary hypertrophy) one cannot expect any improvement for at least 3 months. However, refractory cases are extremely common in these two groups so that a further course of treatment becomes necessary and infact there are not a few patients who are on treatment for years. Also there is evidence that different strains of this virus (chlamydia trachomatis) exists as shown by their varied response to the antibiotics and their adaptation to mice and tissue culture (Bankopf, 1959; Hurst and Reeve, 1960; Fummerss *et al*, 1960; Bell and Theobald, 1962). This paper is concerned with a method of treatment for active trachoma which has not to our knowledge been described in the literature.

INDICATIONS

The indications for this form of treatment are for all cases of Stages II and III trachoma and for all refractory cases of Stage I or where social conditions demand a quick cure.

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TECHNIQUE

The instrument, a "Curettor" consists of a rod of length 2 cm. with a spoon at one end that measures 4 mm. by 5 mm. The other end of the rod is attached to the coil of the diathermy apparatus.

Local anaesthesia is effected by instillation of cocaine followed by injecting planocaine into the fornix of the lower lid, and into the conjunctiva of the upper lid, along the line of the lid eversion. Planocaine is also injected into the plica and the caruncle.

A current not exceeding 30 milliamperes is used. With this amperage, the follicles are curetted with the constantly moving instrument until the area is "clean". Areas that appear clinically normal must be "ironed" at least once by the curettor. This includes the plicae and caruncles. Indeed, no part of the lid conjunctiva is exempt. If the disease has spread to the bulbar conjunctiva, this likewise is treated with the quickly moving curette. The operation field must be dry.

MANAGEMENT

In the ward ice packs are applied over the lids for 3 or 4 days. The application is of utmost importance in the first 48 hours continuously.

Sulphonamide drops are instilled every 2 hours. Antihistaminics are given during the first 48 hours. The patient is discharged after 1 week.

RESULT

At the end of the operation, the lid conjunctiva appears red raw.

There is marked swelling of the lids and oedema of the bulbar conjunctiva for the first 5 days.

Pain is minimal or absent.

In the course of a week, layers of necrotic tissue are seen and these are peeled off from the affected areas.

By the 10th day the swelling has completely subsided and the conjunctiva assumes its moist glistening appearance during the 3rd week.

There is little or no scarring.

COMMENT

From experience with this treatment, it would appear that the trachoma virus is very vulnerable to heat at comparatively low temperatures. This is indeed fortunate in that the temperature at which virus death occurs is well below that which would cause charring of tissue.

It is assumed that after the diathermy burn to the conjunctival epithelium only, islets of epithelium remain capable of acting as centres of regeneration.

Another more probable explanation is that only the more superficial layers of the epithelium are killed by this amount of heat, leaving the deeper layers untouched.

This form of surgical treatment is in keeping with the pathology of the disease, since the problem is most likely to be an epitheliosis in which the virus is strictly localised to the epithelial cells (Axerfeld, 1914). This statement follows on the fact that (*1*) the virus had never been found in any area other than the superficial epithelial cells, a limitation verified by the electron microscope (Mitsui and Suzuki, 1956). (*2*) The sub-epithelial lesions are a result of the liberation of a soluble and diffusible toxin elaborated from the epithelial lesion, since virus containing material capable of exciting a conjunctival inflammation is ineffective both in man and monkeys when injected into the sub-epithelial tissues through the skin of the lids (Michael and Varcea, 1932; Okamma and Mitsui, 1939; Thygeson, 1951). Further proof for this toxin is suggested by the production of a sub-acute follicular conjunctivitis after innoculation of the conjunctiva of a virus-free ultrafiltrate of trachomatous cultures in human volunteers (Mitsui *et al*, 1962).

This method of treatment was evolved by one of us (S.H.) in the pre-sulphonamide era when the disease was rampant in this part of the world.

The procedure has proved entirely effective and is devoid of complications such as symblepharon, cicatrization and lid deformity, open angle glaucoma, or any symptoms suggestive of anterior segment ischaemia.

It is cheap in comparison with the antibiotics and chemotherapeutics, which moreover have to contend with the resistant strains of the virus.

REFERENCES

1. Duke Elder: "System of ophthalmology." Kimpton, London, Vol. 8, p. 258.
2. Vanghan, Asbury and Cook: "General ophthalmology." 1971.

R2 PACEMAKERS

Chairman: M. Eliakim (Israel)

Co-Chairman: Wen-Pin Lien (Taiwan)

1. Future Power Sources—J. Edward Cheatham (Medtromic)
2. Inexpensive Pacemakers—J. McArthur (India)
3. Long Term Follow-Up of Paced Patients—M. Hori (Japan)
4. Discussants—Rae MacDonald (Australia)
   K.B. Lewis (U.S.A.)

R3 TECHNICAL PROBLEMS OF CARDIAC SURGERY IN INFANCY

Chairman: D'Arcy Sutherland (Australia)

Co-Chairman: A. Hanafiah (Indonesia)

1. Palliative versus Corrective Surgery—Peter Clarke (Australia)
2. Profound Hypothermia—Technical Aspects—B. Barratt-Boyes (N.Z.)
3. Perfusion Problems in Infants—S. Sakakibara (Japan)
4. Discussants—K. Prachuabmoh (Thailand)
   Walton Lilihei (A.C.C.)
   Stanley John (India)

PLENARY SESSION (8.30-10.45 Hours)

TUESDAY, OCTOBER 10, 1972

2nd Plenary Session

RECENT ADVANCES IN CORONARY HEART DISEASE

Chairman: I. Pinto (India)

Co-Chairman: Nong Ting (Taiwan)

1. (a) Pre-Infarction Syndrome—G. Sloman (Australia)
   (b) Discussant—Willis Hurst (A.H.A.)
2. (a) Mobile Coronary Care Unit—J. Pantridge (N. Ireland)
   (b) Discussant—Isamu Miura (Japan)
3. (a) Evaluation for Surgery—H. Swan (A.C.C.)
   (b) Discussant—L. Bernstein (Australia)
4. (a) Venous Bypass Surgery—W. Dudley Johnson (A.H.A.)
   (b) Discussant—A. Pitt (Australia)

CONCURRENT SYMPOSIUM (14.15 - 16.00 Hours)

S5 GENETICS IN CARDIOLOGY

Chairman: H.B. Wong (Singapore)

Co-Chairman: F.F. Tangco (Philippines)

1. Genetics of Congenital Heart Disease—L.S. van Mierop (A.H.A.)
2. Genetics in Hypertrophic Cardiomyopathy—R. Emanuel (U.K.)
3. Study of Twin with Hypertension—G. Mimura (Japan)
4. Genetics in Coronary Heart Disease—J. Palmer (Australia)
5. Palmar Dermatogliphics in Heart Disease—T. Takashina (Japan)

S6 SUDDEN DEATH

Chairman: Oglesby Paul (U.S.A.)

Co-Chairman: A.P. Ala (Iran)

1. Definition and Problem of Intervention—M. Oliver (U.K.)
2. Pathological Basis—J. Titus (U.S.A.)
3. Pre-Hospital Death in Singapore—T.C. Chao (Singapore)
4. Observations in Japan—S. Yoshimura (Japan)
5. Sudden Death—D. Kelly (New Zealand)

S7 PULMONARY HEART DISEASE

Chairman: J. Lequime (Belgium)

Co-Chairman: H. Sasamoto (Japan)

1. Pathologic Classification of Pulmonary Vascular Disease—C. Wagenvoort (Holland)
2. Haemodynamic Disturbances—J. Mise (Japan)
3. Role of Pulmonary Hypertension, Hypoxaemia and Right Ventricular Hypertrophy—T. Satake (Japan)
4. Ammonia Metabolism in Pulmonary Heart Disease—A. Valero (Israel)
5. Treatable Causes of Pulmonary Hypertension with Special Reference to Filiariasis—I. Obesekara (Ceylon)

S8 CARDIAC ARRHYTHMIAS

Chairman: G. Sloman (Australia)

Co-Chairman: R. Stephanian (Iran)

1. Pathogenesis of Brady and Trachy Arrhythmias—S. Toyama (Japan)
2. Syncope due to Episodes of VF and Conduction Disturbance—E. Sandoe (Denmark)
3. Bifocal Demand Pacing—B.V. Berkovits (U.S.A.)
4. Atrial Electrocardiogram in Diagnosis and Treatment of Arrhythmias—S.H. Wan (Singapore)
5. Rationale of Drug Therapy—E. Chung (U.S.A.)