TABLE I

*Percentage loss of various amino acids in Buffalo milk casein*

(Average values of three samples)

| Amino acids | Average % loss of heated B.M.C.* for 1 h | Average % loss of heated B.M.C.* for 24 h | Average % loss of $H_2O_2$ treated and heated B.M.C.* for 1 h | | | |
|---|---|---|---|---|---|---|
| | | | concentration of $H_2O_2$ | | | |
| | | | 0·02% | 0·04% | 0·08% | 0·10% |
| 1. Lysine | 2·8 | 67·2 | 13·2 | 27·0 | 54·1 | 67·6 |
| 2. Histidine | 2·7 | 65·3 | 13·4 | 26·8 | 53·4 | 66·8 |
| 3. Arginine | 2·3 | 56·1 | 12·9 | 22·5 | 42·4 | 56·1 |
| 4. Serine | 2·1 | 50·4 | 13·6 | 23·4 | 38·4 | 49·2 |
| 5. Glycine | 1·8 | 42·7 | 8·6 | 17·4 | 34·1 | 42·8 |
| 6. Aspartic acid | 2·4 | 57·6 | 12·6 | 24·4 | 50·8 | 62·0 |
| 7. Threonine | 2·6 | 62·6 | 17·1 | 26·2 | 48·2 | 59·5 |
| 8. Alanine | 2·1 | 50·6 | 12·7 | 17·5 | 35·1 | 50·4 |
| 9. Tyrosine | 2·4 | 58·8 | 12·1 | 24·2 | 49·2 | 60·4 |
| 10. Valine | 2·9 | 69·6 | 13·6 | 26·2 | 54·4 | 69·4 |
| 11. Methionine | 2·6 | 62·4 | 13·7 | 26·8 | 52·4 | 68·2 |
| 12. Phenylalanine | 2·3 | 56·6 | 13·8 | 22·9 | 44·8 | 53·6 |
| 13. Leucines | 1·7 | 40·8 | 8·2 | 16·4 | 33·1 | 40·6 |

B.M.C.*—Buffalo milk casein.

samples of the hydrolysates prepared under identical conditions. The developed chromatogram was used for the estimation of amino acids densitometrically. The results are given in Table 1.

## RESULTS AND DISCUSSION

From the above results it is clearly indicated that thermally treated casein for a period of 1 h at 110°C has undergone a small decrease (2·5%) in most of the amino acids. When casein is heated for 24 h under identical conditions the loss is over 50%.

In the case of casein isolated from milk treated with $H_2O_2$, it is observed that even at a very low concentration of 0·02%, there is over 10% loss of amino acids on heating for an hour at 110°C. At higher concentrations of $H_2O_2$ (0·1% by wt/v) there is a marked decrease of amino acids over 60%.

$H_2O_2$ treated casein on pyrolysis at 110°C readily turns brown releasing gaseous products. So the $H_2O_2$ treated samples (0·02 wt/v) should not be heated for over 30 min. at 110°C.

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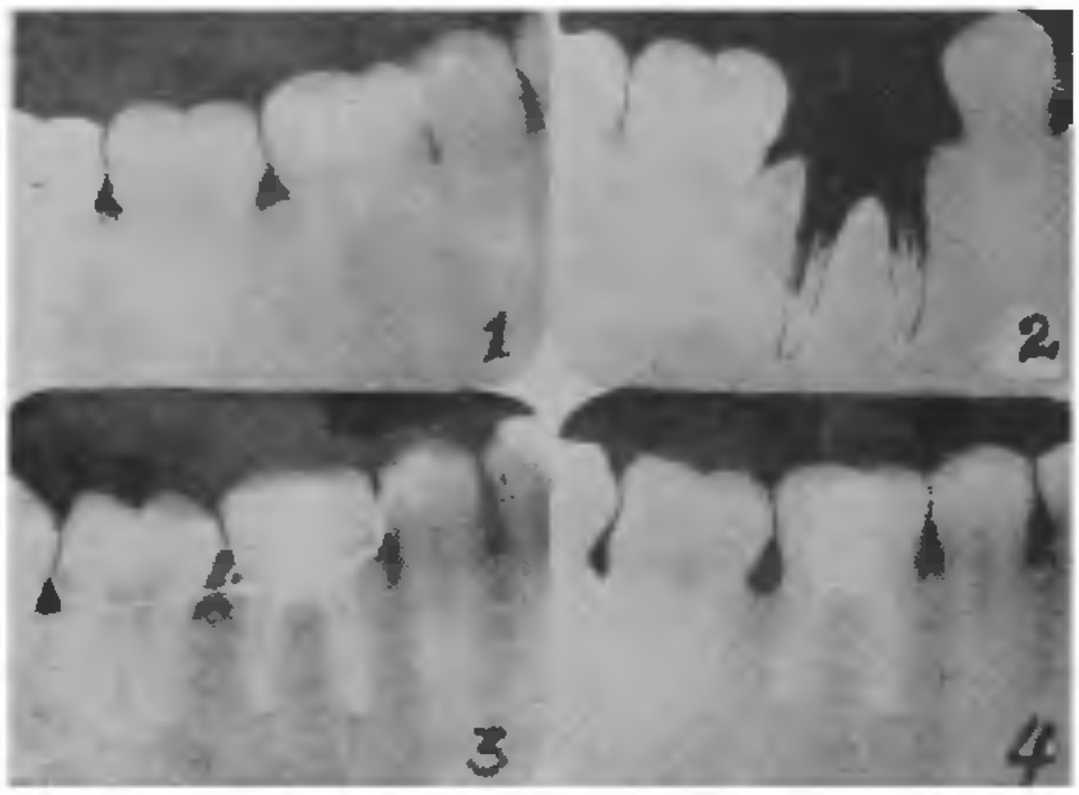
## ON THE REPLANTATION OF HUMAN MOLARS

MODERN operative dental practice involves *replantation* of the traumatically displaced teeth as well as *intentional replantation*[1,2], attempting to treat and retain the permanent teeth affected with caries—in such cases where *in vivo* endodontic surgery is not feasible. *In vitro* endodontic treatments are carried out in the shortest possible time thus maintaining the viability of the supporting structures of the extracted teeth[3]. Further, the limitation of endodontic therapy of the carious teeth, extent of coronal destruction and periodontal involvement and, oral hygiene are some of the important criteria to be considered while attempting an *intentional replantation* towards a conservative management. Present study was undertaken with

reference to the adult molars and to check the viability of the periodontal ligaments when excised, though a few attempts have been made in relation to the replantation of permanent anterior teeth[1–5].

Root canals of carefully extracted molars of ten adult humans were treated *in vitro* (Figs. 1 and 2) with ready-made gutta percha points and zinc oxide-eugenol cement. Zinc-free silver amalgam fillings were provided at the apical region, following the removal of 2–3 mm of the apical portion of the roots. Crowns of the teeth were restored by permanent fillings and the occlusal surfaces of the teeth were ground to prevent contact with the opposing teeth. Effective reinsertion in the socket was made with wiring with neighbouring teeth (Figs. 3 and 4), for a period of 6–8 weeks.




FIGS. 1–4. Successive stages of replantation procedure of human molar.

Though the exact biological processes involved in the successful replantation of intentionally or accidentally displaced teeth are not known, the procedure is gaining importance owing to its aesthetic value especially in teeth with calcified root canals where radiographic evidence of apical pathology is not exhibited. Extensive root resorption has been reported[6] in the replantation studies of the anterior teeth of children where the ligaments and the supporting tissue were suggested to be in embryonic stage. Present study confirms the periodontal membrane and the exposed adult teeth portions being viable at least for a period of 20–30 min., as in, 90% of the analysed samples no resorption occurred for a period of 4–5 years. In spite of the indication of the post-operative regeneration of the periodontal ligament being similar to the periosteum of the bone grafts[6], the successful replantation of the adult molars strongly favours experimental furtherance in terms of physiological stability or loss, and histology of regenerating supportive tissue, over extended periods.

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## IDENTITY OF THE PATHOGEN CAUSING ERGOT OF PEARL MILLET IN INDIA

ERGOT contamination of pearl millet (*Pennisetum typhoideum*) is a serious problem in India, since consumption of ergoty pearl millet leads to disease in humans characterized by nausea, vomiting, giddiness and prolonged sleepiness[1].

The pathogen causing ergot of pearl millet in India was identified by Shinde and Bhide[2] as *Claviceps microcephala*. Loveless[3] designated the pathogen causing ergot of pearl millet in Rhodesia as *Claviceps fusiformis*. An attempt was, therefore, made to establish the correct identity of the pathogen of pearl millet in India based on morphological and chemotaxonomic criteria.

A number of samples of ergot of pearl millet was collected from various parts of India and examined. The sclerotia were obpyriform or irregularly shaped up to 0·7 mm long and up to 0·4 mm wide, generally slightly curved with maximum width at the base and usually narrowing towards the tip. The conidia were hyaline, fusiform to broadly falcate, and 28·0–40·0 $\mu$ wide. Comparison of the Indian material with the type material of *Claviceps fusiformis* established that the two were identical.

A study of the alkaloids in the honeydew stage, after extraction and quantitation by the method described earlier[4] for sclerotial stage, showed that 100 gm of the material contained about 5 mg of the alkaloid. The alkaloid consisted of agroclavine, elymoclavine, chanoclavine, setoclavine and penniclavine. The alkaloid profile was found to be similar to that seen in the sclerotial stage. However, quantitatively in the sclerotial stage the alkaloid content was found to be ten fold higher than that in the honeydew stage. The pathogen which causes ergot of pearl millet produces