牙醫師護療癌症患者之職責

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由於許多的癌症病源是因口腔受到各種慢性刺激而引起，因此；爲了醫療及預防癌症，牙醫師應注意並協助患者維持口腔內的健康以除去或避免各種慢性刺激。

New York Dental Association 訂出了下列的大綱，每一患者每天可例行治療一部分，藉而達到預防癌症之發生：

1. 修補斷爛之牙齒，尤其是突出或鋸齒形之牙齒邊緣。
2. 口腔內膿漏應竭力治療，以除去具傳染性之慢性病灶及已經鬆動的牙齒。
3. 預防及改正早期咬合不正。
4. 如有缺牙應早日鑲補，以預防鄰近變位，否則會導致咬合不正，及食物殘渣填塞於牙間。
5. 對於不良之牙科後形物，應改正或除去，以避免或減少刺激之來源。
6. 應避免繼續使用苛性藥物去治療軟組織病灶。
7. 應避免過量使用煙草。（少抽煙）
8. 避免因職業習慣引起之慢性刺激，如修鞋匠用口咬鐵釘。
9. 避免使用所有刺激性之漱口水及潔牙劑。
10. 避免過度暴露太陽輻射線，以免產生口唇癌及皮膚癌之可能性。
11. 加強早期診治梅毒。

於癌症控制上，牙醫師應於記錄病歷、口腔檢查、診斷及將患者會診專科醫師治療後，仍應直接負起對患者之口腔醫療工作。茲分下列幾項說明之：

㈠口腔內衛生保健：

特別是口腔癌患者，其口內衛生狀況惡劣，會引起牙關緊閉、痛及傳染而影響全身性健康。

㈡修綴牙齒：

對於粗糙不平的牙齒，填補物及牙冠邊緣不良，不合適之假牙切勿保留於口腔內，對於所有牙齒均應重新修補。

㈢拔牙：

如果牙齒及牙周組織經臨床檢查及X光研究，確定有嚴重齲齒，且齒槽骨吸收已很厲害，或牙根尖周已被侵害。斯時，此類牙齒均應拔去，對於有問題之牙齒，牙根斷片及傳染均應竭力除去，不論牙齒其狀況如何，如果已被 X-ray 照射到亦應拔除，在放射線治療前7—10天應先行拔牙，因此類被腫瘤圍閉之鬆動牙齒，可於口腔外科手術治療癌症時，同時將牙齒除去，如果外科治療前過早拔牙，常會導致癌症之蔓延。

㈣放射線治療前後之醫護工作：

口腔於放射線治療之前，應盡可能保持於最佳健康狀況下，此工作亦須及早施行，因放射線治療癌症是愈早愈好的。治療之前，牙齒必須先清潔及填補，牙肉亦需健康，牙髓暴露，牙尖周傳染，有齒齦炎及牙周組織炎之牙齒均應在放射線治療前拔除，有時因需給大劑量放射線治療，或治療範圍大，可將全部牙齒拔除之。

放射線治療後，口腔易感到痛楚，牙醫師更應協助患者注意保持口內清潔，如有腫痛反應而致假牙不能配戴於牙床上，必須待各種反應完全消失後才能配放，如放射線治療部位有外形及體積之改變，則假牙必須修改始能合適應用。

㈤口腔外科治療前後之工作：

外科治療前，應將口腔保持最佳健康狀況，以減少傳染及吸入口腔物質，如下頜骨切除一部份時，牙醫師應製造一牙夾作暫時維持下頜存留斷片之位置。

口腔外科治療癌症後，引起臉部畸形之情形鮮見，但如果有畸形時需作補修以免病人心理上有不良感受。

註：

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Oral & Facial Cancer

RHEUMATIC FEVER

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August 14, 1969, it was a cold and fine Thursday morning. Upon the bare branches of the tall oak trees, a soft white coat clothing them showing that had been a snow fall last night. People from the suburb areas around Melbourne were busy on their ways to the city where the offices were concernti ıted. Thousands of cars lining up on the road and flashes of sunlight reflected from them composing a beautiful picture. A beam of sunlight breaking through the window crept quietly into this room, an operation theatre at the Royal Melbourne Hospital.

On the operation table at the middle of the room lay a young boy about 18 years of age. Pale and weak, he was under general anaesthesia. The patient had been suffering from chronic rheumatic valvular disease. In the course of ten years, his heart was failing and could no more meet the demand of the body. Actually he was dying.

Quietly, the members of a special surgical team were working busily preparing a major operation on him. The chief surgeon was named George Westlake, a famous heart surgeon in Melbourne as well as in Australia. The clock striked eight when the first incision was made on his chest. The sternum would be splitted up and the chest opened. The great vessels of the heart and lung were by-passed by the heart-lung machine, a techuique called cardio-pulmonary by-pass.

The defect of the heart was on the mitral valves caused by rheumatic fever. Rheumatic fever is an acute, but frequently recurring, infection characterized by fever and swelling of joints. Although rheumatic fever is painful and temporarily disabling, it is rarely fatal. But its sequelae in the form of damage to the heart are so common and so serious that it ranks today as one of the major infectious disease problems of childhood and early adolescent life. For many years, the aetiology and causes of rheumatic fever were not known. Recent experiments and reports of medical investigators showing that the disease might be induced by a group of bacteria, group A beta haemolytic streptococcus.

There is no evidence, as yet, that rheumatic fever is caused by any infective agents.. But it

is now generally believed that rheumatic fever is an abnormal tissue reaction to the products of haemolytic streptococcal infection in a sensitised individual.

The cellular components of haemolytic streptococcus are mainly composed of mucopolysaccharide, Protein and mucopeptide. From which. according to these cellular components, the bacteria is separated into a number of serological distinct groups, A to O; and types, 1 to 50. Amongest these components, M protein, a specific protein, is most significant in its antigenic property in producing antibodies in the circulation of the individual and is thought to have relation in the pathogenesis of rheumatic fever. All the extracellular products of haemolytic strptococcus, streptolysin O; streptokinase; hyaluronidase; diphosphopyridine Nucleolidase (DPNase); and deoxyribonuclease (DNase), with the exception of streptolysin S are also antigenic and evoke detectable antibodies in the sera of patients following a streptococcal infection Patient suffering from acute rheumatic fever with an abnormally high anti-streptolysin titre is demonstrated, thus, supporting the hypothesis of haemolytic streptococcal induced rheumatic fever.

The greatest incidence of rheumatic fever occurs between the age of 6 to 15 years with a peak at about 8 years of age. The rheumatic susceptibility is supposed to be affected by many factors. Extensive investigations by teams of medical workers show a familial tendency, thus genetic factors are first come into consideration. Other factors such as race; age; nutrition; Endocrine and environmental factors are also studied. High incidence among the poor families icdicated the socioeconomic importance in rheumatic fever.

The clinical manifestations of acute rheumatic fever are: carditis, chorea, arthritis, subcutaneous nodules and erythema marginatum. The prognosis of the disease is directly related to the severity of carditis. In the acute phase, patient may die of severe fulminating myocarditis. Fragmentation of the cardiac muscle fibers leads to early failure of the heart. The endocardium and pericardium are also involved in the process of acute inflammation. Recurrences continue for several years after the initial infection, each recurrence may add further damage to the heart. The usual site exposed to the attack are the heart valves. By far the commonest valvular defect resulting from rheumatic fever is the damage done to the mitral valves. Aortic valves are also but less common affected, while pulmonary valvular damage is rare.

In the valves, this leads to postinflammatory adhesions and contraction with accompanying stenosis and incompetence. Following the acute attack the thin glistening membranes of the valves may gradually become thickened and fibrotic, and the commissures between may become adherent. From the time of the initial attack of rheumatic fever until the development of symptoms of mitral stenosis, there is frequently a Lapse of many years. Once symptoms develop, the condition is downhill. Later, the leaflets or cusps, may become calcified. In addition, the chordae tendinae may become so thickened and shortened that the papillary muscles seem directly implanted in the cusps. Because of the slow and insidious progress of the disease, the patient is frequently not fully aware of how limited his activities have become until he is no longer able to carry on his occupation.

Normally, the leaflets open widely providing an opening for blood flow during ventricular diastole. However, with gradual thickening and fibrosis this opening gradually narrows. The sealing together of the two leaflets in a fibrous union begins by the formation of fibrous adhesion which bridge the commissures at the edge of the leaflets. When this degree of narrowing has occured, the capacity to increase the flow through the narrowed valves is limited. As a result of thes pressure elevation, Pulmonary hypertension develops involving the pulmonary arteries. Because of the pulmonary hypertension, the work of the right ventricle is increased, and in advanced cases, right heart failure with secondary tricuspid incompetence develops. When this occurs, there is oliguria, and pain in the right upper quadrant of the abdomen from liver enlargement and portal

hypertension. Atrial fibrillation eventually occurs and increases markedly the hazard of peripheral or pulmonary embolism. Repeated, massive and fatal pulmonary infraction may supervene, once right heart failure has developed.

Mitral incompetence is an almost constant accompaniment of mitral stenosis. Mitral incompetence produces a decreased cardiac output and increases the work of the left ventricle since a portion of the blood which would be normally ejected through the incompetent mitral valves. This leads to increase pressure in the left atrium similar to that which occurs in mitral stenosis. There will be ventricular enlargement and a left ventricular lift. Pulmonic incompetence may be present due to increasing pulmonary hypertension and dilation of the pulmonary arteries.

In mild and moderate cases, symptomatic treatments using digitalis and diuretics with potassium supplements can help the patient temporary. In sever stenotic valves, commissurotomy is the usual surgical treatment. But post-commissurotomized incompetence of the valves is so common that it lays another hazard on the patient. Recently, exicsion of the damaged valves and replacement by artificial valves is the common form of surgical treatment.

In order to reduce the rate of occurence of rheumatic heart disease in children, rheumatic fever must be checked. Recently, reports from workers and investigators all over the world show a declined incidence in rheumatic fever, especially after the administration of anti-biotics such as penicillin and sulfonamides, the anti-streptococcal agents. Wide spread public health knowledge among the citizens; improving the living conditions; and prophylactic regimens carry out in children become the primary prevention of rheumatic fever occurence. Forseeing the future, with advanced medical research and technique, rheumatic fever would be eliminated from being one of the major causes of a disabling disease----rheumatic heart disease.

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