extended phase II study in previously untreated patients is indicated.

1. Legha SS. Interferons in the treatment of malignant melanoma. A review of recent trials. *Cancer* 1986, **57**, 1675–1677.
2. Welander CE, *et al.* In: Kisner D, Smith J, eds. *Interferon Alfa-2: Pre-clinical and Clinical Evaluation*. Leiden, Martinus Nijhoff, 1985, 29–33.
3. Comus RL. DTIC (NSC-45388) in malignant melanoma: a perspective. *Cancer Treat Rep* 1976, **60**, 165–176.

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Intracranial Tumours and Blood Groups

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THE RELATION between blood groups and diseases such as carcinoma of the stomach, peptic ulcer [1] and diabetes [2] is known. However, reports regarding blood groups and intracranial tumours [3–9] conflict. We have retrospectively studied the distribution of different blood groups in patients with intracranial tumours compared with that in the general population.

All consecutive case records of patients with histologically verified intracranial glioma, medulloblastoma, schwannoma and meningioma from 1981–1986 treated at the National Institute of Mental Health and Neurosciences, Bangalore, were reviewed and the blood groups of the patients noted. The distribution of blood groups in the general population in and around Bangalore City was obtained from the Indian Red Cross Society [10].

The total number of patients treated for intracranial tumours during the 5 years was 1287. Of these, 668 (52%) had glioma, 310 (24%) meningioma, 215 (17%) schwannoma and 94 (7%) medulloblastoma. The distribution of the four major blood groups in this population was compared with that in the general population by the χ^2 test. A significantly greater proportion of patients with tumours had blood group A ($P < 0.001$), whilst more individuals in the general population had blood group O.

Of our patients with glioma, significantly more were in group A and significantly fewer in group O (both $P < 0.001$) compared with individuals in the general population. Compared with the general population, more of our patients with medulloblastoma were in groups B and AB, and the proportion was significantly higher in group B ($P < 0.001$). Contrary to this observation, Atwell [11] found no significant difference in blood group distribution in a study of children with embryonic tumours. Significantly fewer of our patients with schwannoma were in group O than the proportion in the general population, whilst most of our patients with meningioma were in blood group O or A. Yates and Pearce [9] found a higher association of meningiomas with blood group A. However, Mayer *et al.* [7] found meningiomas more frequent in group B types. Although the proportion of our group A patients with meningioma was higher than the proportion of individuals with group A in the general population, this difference was not significant.

Silverstone and Cooper [8] compared blood groups in astrocytoma patients with those in the general population. They found a significantly higher frequency of astrocytoma in group A individuals and a lesser frequency in those with group O and suggested that anti-A may provide some protection. Yates and Pearce [9] found astrocytoma less frequent in group O types and Campbell *et al.* [3] reported that glioma was more frequent in group A individuals. In contrast, Carter *et al.* [4] in a study of patients with glioblastoma and Garcia *et al.* [5] in a study of patients with astrocytoma did not find any positive correlation.

It is not known how blood group can affect the frequency of tumour in the central nervous system. Further studies of tumour immunology and blood groups are indicated.

Table 1. Blood groups in patients with intracranial tumours and in a general population

	A	B	AB	O	Total
General population	1390 (26%)	1118 (21%)	239 (4%)	2682 (50%)	5429
Patients with tumours	412 (32%)	309 (24%)	64 (5%)	502 (39%)	1287
Glioma	223 (33%)	146 (22%)	39 (6%)	260 (39%)	668
Medulloblastoma	14 (15%)	41 (44%)	9 (10%)	30 (32%)	94
Schwannoma	72 (33%)	57 (27%)	11 (5%)	75 (35%)	215
Meningioma	97 (31%)	62 (20%)	6 (2%)	145 (47%)	310

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1. Aird I, Bentall HH, Roberts JAF. A relationship between cancer of stomach and the ABO blood groups. *Br Med J* 1953, **1**, 799–801.
2. McConnell RB, Pyke DA, Roberts JAF. Blood groups in diabetes mellitus. *Br Med J* 1956, **1**, 772–776.
3. Campbell ACP, Gaisford W, Patterson E, Steward JK. Tumours in children: A survey carried out in the Manchester region. *Br Med J* 1961, **1**, 448–452.
4. Carter RL, Hitchcock ER, Sato F. Malignant gliomas and ABO blood groups. *Br Med J* 1964, **1**, 122.
5. Garcia JH, Okazaki H, Aronson SM. Blood group frequencies and astrocytoma. *J Neurosurg* 1963, **20**, 397–399.
6. Manuila A. Blood groups and disease—hard facts and delusions. *JAMA* 1958, **167**, 2047–2053.

7. Mayer E, Diamond LK, Levine RP, Mayer M. Suspected correlation between blood group frequency and pituitary adenomas. *Science* 1956, **124**, 932–934.
8. Silverstone B, Cooper DR. Astrocytomas and blood groups. *J Neurosurg* 1961, **18**, 602–604.
9. Yates PO, Pearce KM. Recent change in blood group distribution of astrocytomas. *Lancet* 1960, **i**, 194–195.
10. Prasanna HA, Srinivasamurthy. The distribution of blood groups in and around Bangalore. Proceedings of the 12th national conference of the Indian Society of Blood Transfusion and Immunohaematology. Bangalore, India, 1986, 14–15.
11. Atwell JD. Distribution of ABO blood groups in children with embryonic tumours. *Br Med J* 1962, **1**, 89–90.

Table 1. Root surface tumour extension in relation to RBD

Root surface	No.	RBD+	RBD−
Gross tumour	26	19	7
Microscopic tumour only	4	3	1
No tumour	13	4	9
Total	43	26	17

RBD = Radiologically demonstrable bone destruction: + = present, − = absent.

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Periodontal Space: Major Route to Bone in Oral Cancer

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ORAL CANCERS involve the bone by invading and destroying the periosteum, and points such as the occlusal ridge and foraminae are natural routes of entry [1–3]. We report the preliminary observations of our prospective study, which suggest that one natural point of entry, the periodontal space, is the major route to both the maxilla and mandible.

We have so far studied 43 untreated patients with oral squamous cell cancers. All were dentulous, with primary tumours of the gingiva or an adjacent site growing onto the gingiva, and all had a "tumour touched tooth". 26 had lesions related to the lower and 17 to the upper gingiva, respectively. Bone involvement was assessed by radiography, both conventional views of the jawbones and intra-oral dental radiograph of the tumour-touched tooth. This tooth was then extracted, and its root surface inspected for the presence of tumour. Scrapings were taken from the root surface, and submitted for histopathology.

Table 1 summarises the observations. 30 (70%) patients had tumour extension on the root surface. The tumour was grossly visible in 87% of these cases, and extended from the level of gingival attachment to varying lengths along the root surface, to the tip. Radiography, despite a high chance of false negativity, is considered the primary investigation to diagnose bone involvement. The relation between periodontal spread and bone involvement, evidenced by radiologically demonstrable bone destruction is shown in Table 1. As many as 74% (22/34) of the patients with root surface tumour extension had radiologically demonstrable bone destruction, compared with only 31% (4/13) without root surface tumour extension. This correlation was statistically significant ($P < 0.01$, χ^2 test). Of the 4 patients with radiologically demonstrable bone destruction but no root surface tumour extension, 1 had involvement from a fixed submandibular lymph node, and in the other 3 the route was unknown. Of the 26 patients who had bone involvement, the periodontal space was the route of entry in 85% (22).

It is the cancers of the gingiva, buccoalveolar sulci and floor of mouth that are most likely to involve the bone. These cancers have the highest causal relation with tobacco chewing, a habit that also results in poor dental hygiene [4], and possibly leads to chronic periodontitis. Normally periodontal ligament is tough, and probably as resistant to tumour invasion as the periosteum, however, chronic periodontitis weakens the ligament and the surrounding bone, which becomes more susceptible to tumour invasion than the periosteum. Given the high frequency of periodontal spread and bone involvement in the present study, we suggest that in countries with a high incidence of oral cancers and associated poor dental hygiene, periodontal space is the major route to bone, as we previously hypothesised [5].

Our study identifies examination of the root surface of extracted teeth as a new investigation, helpful in diagnosing bone involvement. Whilst radiography cannot give histological confirmation, even when positive, root surface examination can. Therefore, root surface examination supplements, if not supplants, radiography in establishing bone involvement.

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1. Byars LT. Extent of mandibular resection required for treatment of oral cancer. *AMA Arch Surg* 1955, **70**, 914–920.
2. McGregor AD, McDonald DG. Routes of entry of squamous cell carcinoma to the mandible. *Head Neck Surg* 1988, **10**, 294–301.
3. O'Brien CJ, Carter RL, Soo KC, Barr LC, Hamlyn PJ, Shaw HJ. Invasion of the mandible by squamous carcinomas of the oral cavity and oropharynx. *Head Neck Surg* 1986, **8**, 247–256.
4. Sankaranarayanan R, Duffy SW, Day NE, Nair MK, Padmakumari G. A case control investigation of cancer of the oral tongue and floor of the mouth in South India. *Int J Cancer* 1989, **44**, 617–622.
5. Bhattathiri VN, Nair MK. *Medical Hypotheses* (in press).

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