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A quantitative study of Australian Aboriginal and Caucasian brains*

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INTRODUCTION

At the beginning of this century the morphology of Aboriginal brains was studied extensively (Ariens-Kappers, 1929; Duckworth, 1907/08; Flashman, 1908, 1916; Karplus, 1902; Klaatsch, 1911; Kohlbrugge, 1909; Miklouho-Maclay, 1884; Packer, 1949; Rolleston, 1887/88; Shellshear, 1934, 1937, 1939; Woollard, 1929, 1931). Several authors measured brain weights, brain volumes, skull capacities, and other parameters (Brackebusch, 1905; Davis, 1868; Duckworth, 1904*a*, *b*, 1907/08; Hauger, 1921; Karplus, 1902; Krause, 1897; Packer, 1949; Rolleston, 1887/88; Shellshear, 1937; Woollard, 1929, 1931). Woollard (1929) determined the weight of the cerebral cortex of Aboriginal brains by cutting away the cortex from 2 mm thick brain slices with a scalpel. Most of the studies published to date have been rather small. A brain weight study of 63 cases was published by Harper & Mina (1981). A detailed literature review of studies concerning Aborigines was given by Moodie & Pedersen (1971).

This is a quantitative study of 8 male Australian Aboriginals and 11 male Caucasians measuring volumes of the whole brain and a variety of brain regions.

MATERIALS AND METHODS

Eight male Aboriginal brains from the Department of Neuropathology, Royal Perth Hospital, Perth, Western Australia, and eleven male Caucasian brains from the Institute of Forensic Medicine, University of Hamburg, Germany, formed the basis of this study. The Aboriginal brains were derived from the northwestern part of Australia near Carnarvon and from Kalgoorlie. Most of the Aborigines in that area live in missions and Aboriginal settlements. Table 1 summarises the age, body height, body weight, cause of death, delay between death and autopsy, and pathological findings of the 19 cases.

Fresh brain weights, including the leptomeninges, were determined at autopsy. After fixation in 10 % formalin solution brain weights were measured again using a weighing machine with an accuracy of 0·1 g. Brain volumes were determined by buoyancy measurements in water according to:

$$BV = (BW - BWW)/SGW \quad (1)$$

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Table 1. *Case characteristics of eleven male Caucasian and eight male Aboriginal brains*

Case no.	Age years	BH cm	BW kg	Time until autopsy	Cause of death	Pathological findings
				Part a: Caucasians		
H 28/81	34	165	78	1 d	Carbon monoxide poisoning	Arteriosclerosis, brain oedema
H 27/81	42	169	74	2 d	Right sided heart failure, bronchitis	Arteriosclerosis
H 26/81	51	168	69	2 d	Hanging	Arteriosclerosis
H 25/81	44	176	73	1 d	Hanging	Adrenal adenoma, arteriosclerosis
H 24/81	39	181	87	2 d	Blood loss, alcohol intoxication	Fatty liver, arteriosclerosis
H 21/81	59	175	85	1 d	Myocardial infarct	Emphysema, brain oedema, arteriosclerosis
H 20/81	53	177	83	2 d	Myocardial infarct	Arteriosclerosis
H 19/81	45	175	58	2 d	Blood loss	Thyroid cyst
H 18/81	58	172	98	2 d	Myocardial infarct	Anthracosis of the lung, arteriosclerosis
H 16/81	51	174	92	1 d	Cor pulmonale	Emphysema, arteriosclerosis
H 15/81	23	178	64	1 d	Hanging	Drug intoxication, brain oedema
Mean	45	174	78	1·5 d	—	—
				Part b: Aborigines		
X80–405	55	170	59	5 d	Pneumonia	—
X80–628	35	178	94	1 d	Rupture of liver and spleen	Fatty liver
X81–121	33	168	42	3 d	Undetermined	Pancreatitis, liver cirrhosis
X81–203	30	167	51	2 d	Bullet wound	—
X81–214	70	167	78	2 d	Myocardial infarct	Arteriosclerosis
X81–395	25	192	89	1 d	Undetermined	Lung oedema
X81–554	51	185	74	4 d	Pneumonia	—
X82–274	33	150	85	1 d	Pancreatitis	Diabetes, fatty liver, deaf, dumb, blind in left eye
Mean	42	172	72	2·5 d	—	—

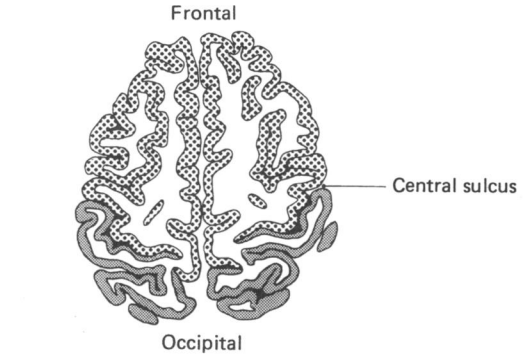
BH, body height; BW, body weight; d, day(s).

(BV = brain volume, BW = brain weight, BWW = brain weight in water, SGW = specific gravity of water).

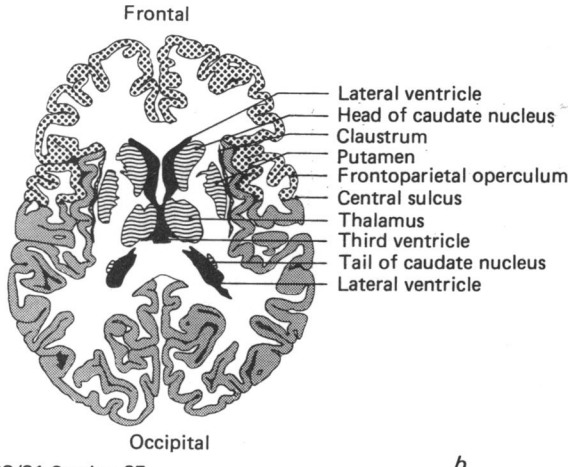
The leptomeninges were removed and the cerebellum and brainstem were cut from the cerebral hemispheres at the pons-mesencephalic junction. The cerebellum was separated from the brainstem at the cerebellar peduncles, and the medulla was sectioned at the level of the obex. Thus, the brain was divided into three parts: prosencephalon-mesencephalon unit (in the following text also referred to as cerebral hemispheres), cerebellum, and pons-medulla oblongata unit (also referred to as the brainstem).

Precentral and postcentral gyri were stained with ink for easy recognition after sectioning. The cerebral hemispheres were embedded in 3 % agarose and cut into 2 mm slices in the horizontal bicommissural plane with a macrovibratome which has been described elsewhere (Klekamp, Riedel, Herrmann & Kretschmann, 1985).

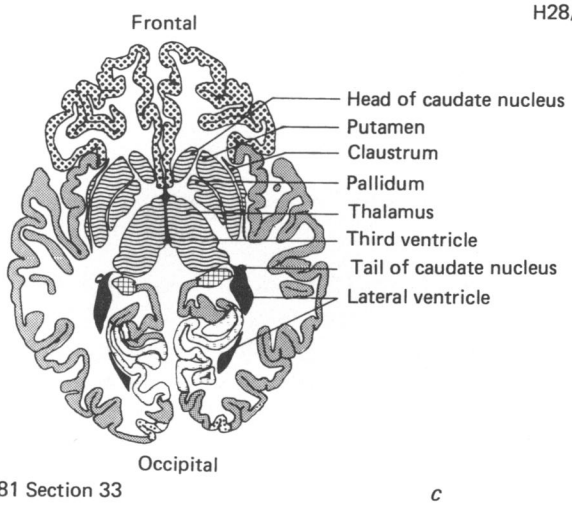
After each 2 mm cut the brain-agarose block was photographed alongside a scale from a distance of 60 cm (Nikon F2, Nikkor 1:3·5/55 mm on Agfaortho 25, 12



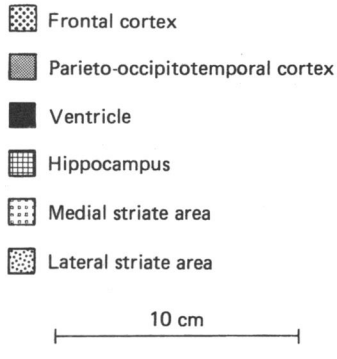
H28/81 Section 9 *a*

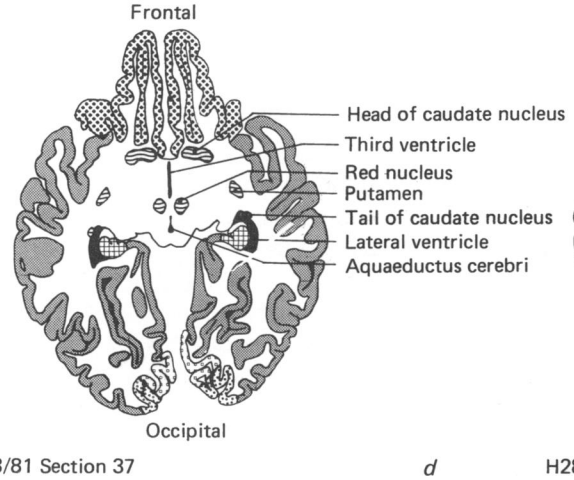


H28/81 Section 27 *b*

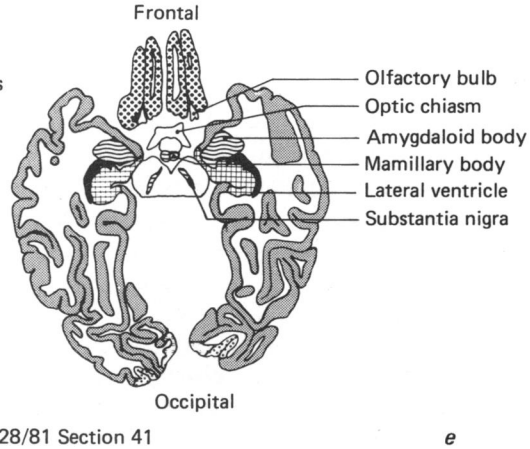


H28/81 Section 33 *c*





H28/81 Section 37 *d*



H28/81 Section 41 *e*

Fig. 1 (*a–e*). Definition of cortical areas exemplified for a 34 years old Caucasian (H 28/81).

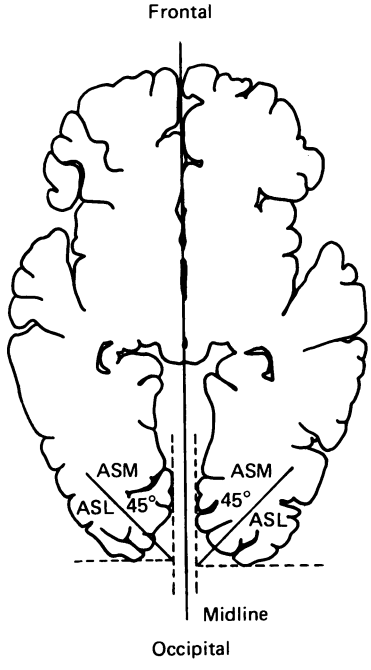


Fig. 2. Definition of medial (*ASM*) and lateral (*ASL*) striate cortex.

ASA). Brain slices were put into plastic bags with 15 ml of 10 % formalin solution and affixed to stiff paper sheets. Black and white prints were made of each slice using a special photographic paper (Agfa Copyline P 90, Agfa Gevaert) at a 1 × 1 magnification. Because of fixation artifacts volume measurements were not done for X 81-203 and H 19/81 except for the cerebral hemispheres, cerebellum, and brainstem.

To exclude significant neurological disorders all brains were examined histologically. The following structures were examined: pre- and postcentral gyrus, frontal cortex, hippocampus, visual cortex, thalamus, basal ganglia, red nucleus, substantia nigra, mamillary bodies, vermis, and medulla oblongata. [Minor changes were observed for X 81-214 (atrophy of superior vermis) and H 18/81 (atrophy of superior vermis and slight cortical atrophy). X 81-121 had chronic Wernicke's encephalopathy. Despite these histological findings all the measured fresh volumes of these brains were above (X 81-214 and H 18/81) or close to (X 81-121) the mean value for each area. Therefore, we did not exclude them from this study.

All measured structures were identified macroscopically. Borders were drawn onto the photos and the areas measured by electronic planimetry (MOP-AM-02, Kontron) using a computer (Wang 2200). The following criteria were used. Right and left hemispheres were measured separately. The cerebral cortex included all cortical structures of the prosencephalon without the amygdaloid bodies. The frontal cortex was limited by the central sulcus. The median border of the frontal cortex was marked by the sulcus limiting the lobulus paracentralis (pars marginalis sulci cinguli). In deeper sections only cortex in front of the corpus callosum was considered as frontal cortex. Insular cortex was delimited by the sulcus circularis insulae (Fig. 1).

The parieto-occipitotemporal cortex consisted of all cortical areas excluding

frontal cortex but including visual cortex and hippocampus. The visual cortex was defined as the cortical area where the stria of Gennari could be identified macroscopically with a bright halogen lamp. The medial and lateral parts of the visual cortex were measured separately. The border was constructed geometrically as indicated in Figure 2.

Only the retrocommissural part of the hippocampal formation was measured. The alveus and fimbria hippocampi were excluded. The median border was defined as between praesubiculum and subiculum using the halogen lamp. This definition is in accordance with Stephan (1975) and Braak (1980). For the cerebral hemispheres and the areas of the cerebral cortex, except the hippocampus and visual cortex, every second slice was measured (section distance 4 mm). For the hippocampus and striate cortex all slices were measured (section distance 2 mm). The planimetric volume (PV) was calculated using:

$$PV = \sum_{i=1}^{n} A_i \times d \tag{2}$$

(A_i = planimetric area of slice i; d = section distance). The fresh brain volume (FBV) was calculated using the fresh brain weight (FBW) and the specific gravity of the fixed brain (SGB):

$$FBV = FBW/SGB. \tag{3}$$

The fresh volume of the prosencephalon-mesencephalon unit (PMFV) was calculated according to:

$$PMFV = FBV \times (PMW_{fix}/BW_{fix}) \tag{4}$$

with PMW_{fix} = weight of the fixed prosencephalon-mesencephalon unit and BW_{fix} = weight of the fixed brain. The volumes calculated in this way gave fresh volumes using:

$$AFV = PMFV \times (APV/PMPV) \tag{5}$$

(AFV = fresh volume of an area; APV = planimetric volume of an area; and PMPV = planimetric volume of the prosencephalon-mesencephalon unit). All values presented in this study are given as means plus or minus the standard deviation. They were defined as significantly different if the p value was less than 0·05, determined using Student's *t*-test. Regression lines in Figures 3–10 are shown if they reached a level of significance of at least 95 %. All subsequent data refer to 'fresh' brain weights and volumes unless otherwise specified.

RESULTS

In the following text the values for Aborigines are always listed first. The mean age for Aborigines and Caucasians did not differ significantly (42 ± 16 years, and 45 ± 11 years, respectively), nor did body heights (172 ± 13 cm, and 174 ± 5 cm, respectively). The fresh brain weight for Aboriginals was significantly less (1241 ± 88 g) than for Caucasians (1421 ± 101 g). The fresh brain volumes of both populations showed a similar significant relationship (1199 ± 84 ml to 1386 ± 98 ml). The specific gravity of the fixed brains was significantly different being $1{\cdot}035 \pm 0{\cdot}004$ g/ml for Aborigines and $1{\cdot}025 \pm 0{\cdot}004$ g/ml for Caucasians. The volumes of the cerebral hemispheres and cerebellum were significantly less for Aborigines (1039 ± 79 ml to 1204 ± 84 ml, and 134 ± 13 ml to 155 ± 17 ml, respectively). The volume of the

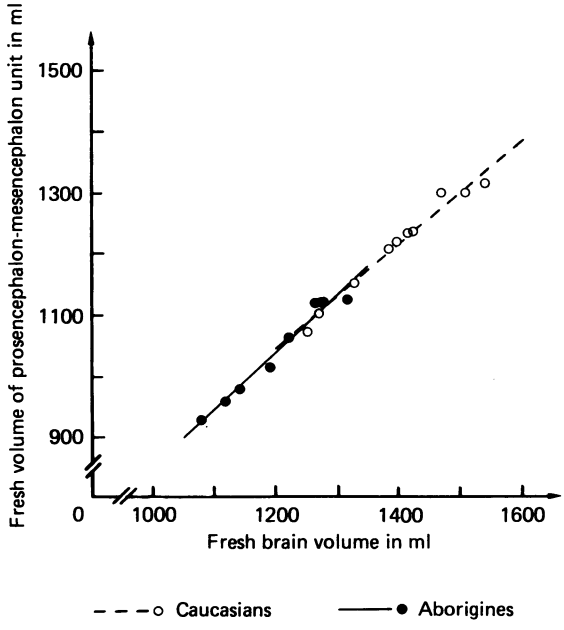


Fig. 3. Relationship between fresh volume of the prosencephalon-mesencephalon unit and fresh brain volume for eight male Aboriginal and eleven male Caucasian brains, $r(C) = 0{\cdot}99$; $y = 0{\cdot}85x + 24{\cdot}14$ (Caucasians), $r(A) = 0{\cdot}99$; $y = 0{\cdot}93x - 71{\cdot}06$ (Aborigines).

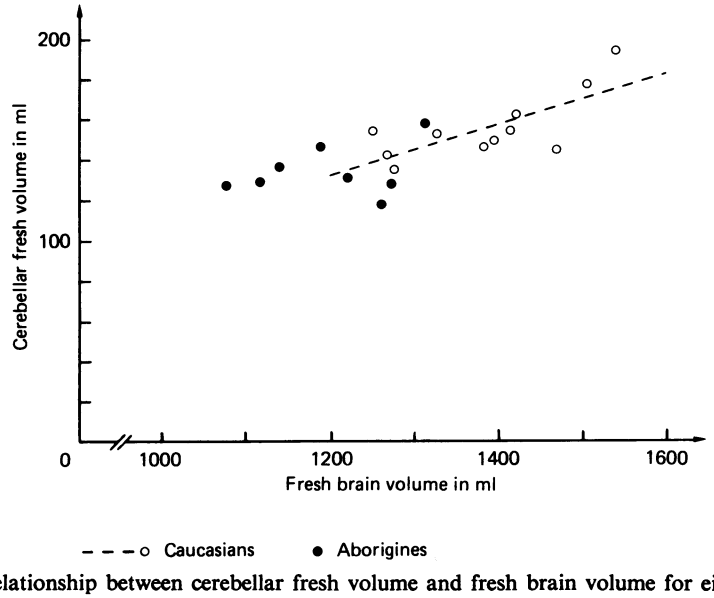


Fig. 4. Relationship between cerebellar fresh volume and fresh brain volume for eight male Aboriginal and eleven male Caucasian brains, $r(C) = 0{\cdot}75$; $y = 0{\cdot}13x - 19{\cdot}08$ (Caucasians), $r(A) = 0{\cdot}33$ (Aborigines).

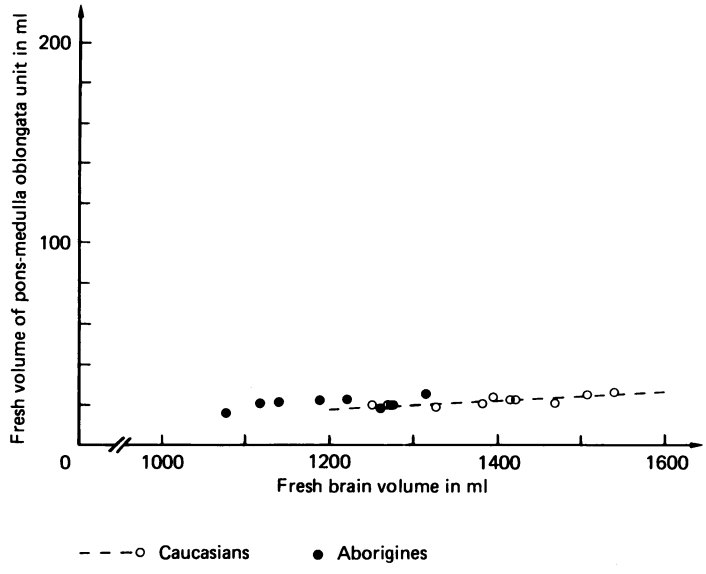


Fig. 5. Relationship between fresh volume of the pons-medulla oblongata unit and fresh brain volume for eight male Aboriginal and eleven male Caucasian brains, $r(C) = 0{\cdot}85$; $y = 0{\cdot}02x - 7{\cdot}56$ (Caucasians), $r(A) = 0{\cdot}60$ (Aborigines).

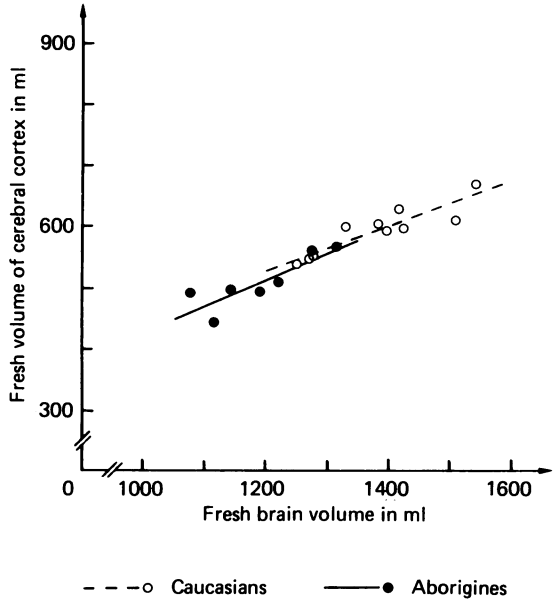


Fig. 6. Relationship between fresh volume of the cerebral cortex and fresh brain volume for seven male Aboriginal and ten male Caucasian brains, $r(C) = 0{\cdot}91$; $y = 0{\cdot}37x + 82{\cdot}52$ (Caucasians), $r(A) = 0{\cdot}89$; $y = 0{\cdot}43x - 1{\cdot}84$ (Aborigines).

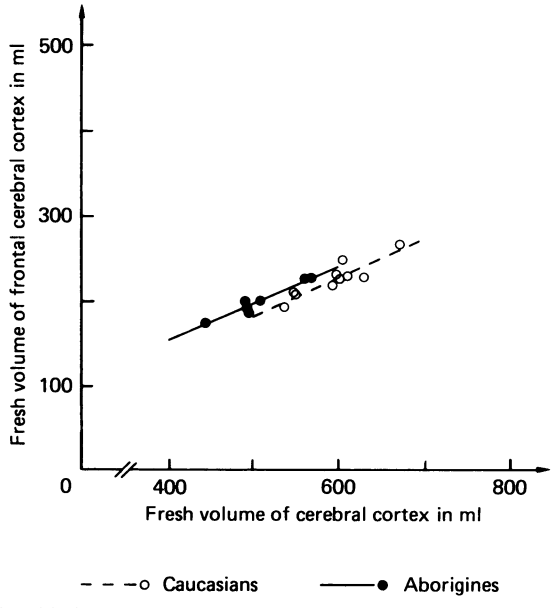


Fig. 7. Relationship between fresh volume of the frontal cortex and fresh volume of the cerebral cortex for seven male Aboriginal and ten male Caucasian brains, $r(C) = 0{\cdot}92$; $y = 0{\cdot}46x - 46{\cdot}76$ (Caucasians), $r(A) = 0{\cdot}98$; $y = 0{\cdot}44x - 21{\cdot}96$ (Aborigines).

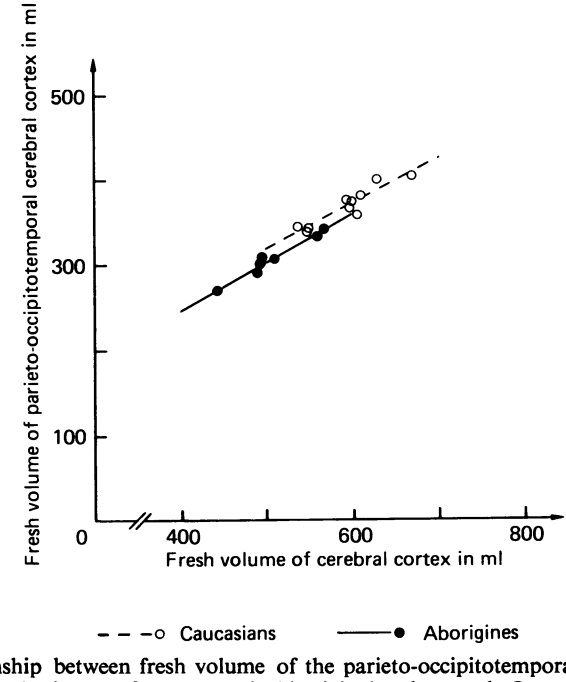


Fig. 8. Relationship between fresh volume of the parieto-occipitotemporal cortex and fresh volume of the cerebral cortex for seven male Aboriginal and ten male Caucasian brains, $r(C) = 0{\cdot}94$; $y = 0{\cdot}54x + 50{\cdot}67$ (Caucasians), $r(A) = 0{\cdot}99$; $y = 0{\cdot}57x + 16{\cdot}25$ (Aborigines).

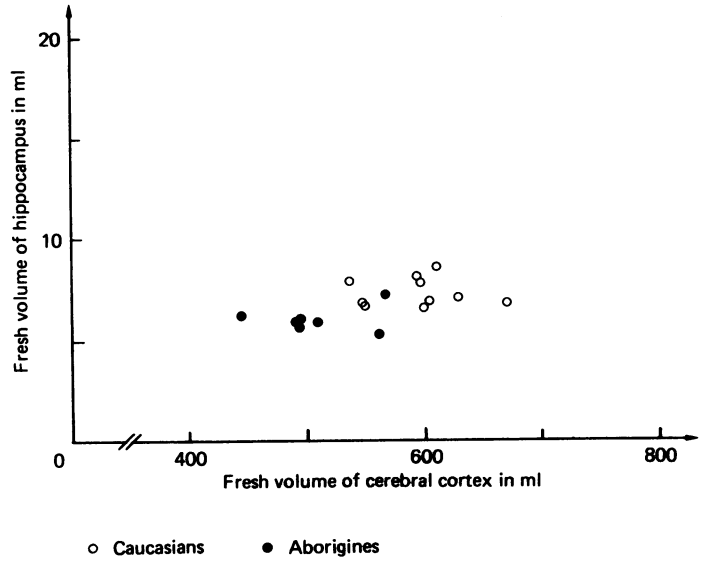


Fig. 9. Relationship between fresh volume of the hippocampus and fresh volume of the cerebral cortex for seven male Aboriginal and ten male Caucasian brains, $r(C) = -0{\cdot}07$ (Caucasians) and $r(A) = 0{\cdot}22$ (Aborigines).

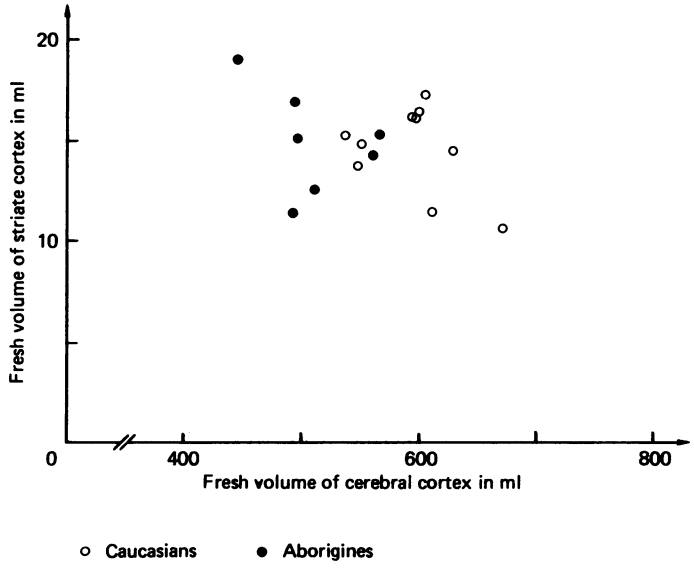


Fig. 10. Relationship between fresh volume of the striate cortex and fresh volume of the cerebral cortex for seven male Aboriginal and ten male Caucasian brains, $r(C) = -0{\cdot}43$ (Caucasians) and $r(A) = -0{\cdot}41$ (Aborigines).

Table 2. *Fresh brain weight in g, specific gravity of the brain in g/ml, and fresh volumes of several brain parts in ml*

Case no.	FBW	FBV	SGB	CFV	PMOFV	PMl	PMt	CCl	CCt
H 28/81	1305	1268	1·029	142	20	553	1101	274	548
H 27/81	1450	1414	1·026	154	23	610	1232	310	629
H 26/81	1360	1327	1·025	152	19	570	1152	296	600
H 25/81	1415	1382	1·024	146	21	599	1207	302	605
H 24/81	1420	1395	1·018	149	24	601	1217	294	594
H 21/81	1303	1276	1·021	135	20	557	1119	274	550
H 20/81	1550	1507	1·029	177	25	651	1300	308	611
H 19/81	1505	1469	1·025	144	21	—	1299	—	—
H 18/81	1450	1422	1·020	162	23	616	1234	300	597
H 16/81	1285	1250	1·028	154	20	526	1072	261	537
H 15/81	1585	1540	1·029	194	27	654	1316	335	671
Mean	1421	1386	1·025	155	22	594	1204	295	594
s	101	98	0·004	17	3	42	84	21	41
X80–405	1158	1116	1·03	129	21	481	960	222	445
X80–628	1230	1188	1·035	146	23	510	1017	249	494
X81–121	1170	1140	1·026	136	22	488	980	248	496
X81–203	1304	1261	1·034	118	19	—	1120	—	—
X81–214	1364	1315	1·037	158	26	559	1125	283	568
X81–395	1320	1273	1·037	128	20	539	1119	271	561
X81–554	1268	1220	1·039	131	23	536	1063	258	510
X82–274	1116	1076	1·037	127	16	462	929	245	429
Mean	1241	1199	1·035	134	21	511	1039	254	509
s	88	84	0·004	13	3	36	79	20	43

FBW, fresh brain weight; FBV, fresh brain volume; SGB, specific gravity of the brain; PMOFV, fresh volume of pons-medulla oblongata unit; PMl, PMt, fresh volume of prosencephalon-mesencephalon unit left and total; CCl, CCt, fresh volume of cerebral cortex left and total; s, standard deviation.

pons-medulla oblongata unit showed no significant difference (21 ± 3 ml and 22 ± 3 ml).

The percentages of total brain volume which the three units represented were as follows. The cerebral hemispheres were 86·7 ± 1·2 % for Aboriginals and 86·9 ± 0·9 % for Caucasians, and the cerebellum 11·2 ± 1·0 % and 11·2 ± 0·8 %, respectively. Neither of these was significantly different. However, the pons-medulla oblongata unit was significantly bigger in Aborigines (1·8 ± 0·2 %) than in Caucasians (1·6 ± 0·1 %).

No significant differences were identified between left and right hemispheres for any area examined. Therefore, only combined right and left hemisphere volumes for each area are mentioned in this text. However, more detailed information is given in Tables 2–4.

The cerebral cortical volume was significantly less in Aboriginal brains compared to Caucasian brains (509 ± 43 ml and 594 ± 41 ml, respectively). Similar relationships were found for frontal cortex (202 ± 19 ml and 226 ± 21 ml), parieto-occipitotemporal cortex (307 ± 25 ml and 369 ± 23 ml), and hippocampus (6·0 ± 0·6 ml and 7·3 ± 0·7 ml), respectively. However, there was no significant difference when cerebral cortex was expressed as a percentage of total brain volume (42·8 ± 1·9 % for Aborigines and 43·2 ± 1·3 % for Caucasians). Significant differences in percentage of cerebral cortex were found for frontal cortex (39·6 ± 1·0 % and 38·0 ± 1·5 % and parieto-occipitotemporal cortex (60·3 ± 1·0 % and 62·1 ± 1·5 %), but none for hippocampus (1·2 ± 0·2 % for both populations).

Table 3. *Fresh volumes of cerebral cortical regions in ml*

Case no.	FCCl	FCCt	POTCCl	POTCCt	HPl	HPt	ASMl	ASMt	ASLl	ASLt	ASFVl	AFVt
H 28/81	104	210	170	338	3·4	6·8	4·9	10·0	1·9	3·7	6·8	13·7
H 27/81	113	228	198	401	3·4	7·1	6·0	11·8	1·0	2·7	7·0	14·5
H 26/81	110	226	187	374	3·3	6·6	6·1	12·4	2·1	4·1	8·2	16·4
H 25/81	124	248	178	358	3·5	6·9	6·6	13·4	1·8	3·9	8·5	17·3
H 24/81	108	218	186	376	4·0	8·1	4·7	10·7	3·0	5·5	7·7	16·2
H 21/81	102	208	173	343	3·3	6·7	4·1	8·9	3·0	5·9	7·1	14·8
H 20/81	114	231	194	380	4·2	8·6	4·4	9·5	0·9	2·1	5·3	11·5
H 18/81	116	231	185	366	3·9	7·8	6·1	11·3	2·3	4·8	8·4	16·1
H 16/81	93	194	168	344	3·8	7·9	6·3	11·8	1·2	3·4	7·5	15·2
H 15/81	134	266	201	405	3·3	6·8	4·7	9·3	0·8	1·4	5·5	10·6
Mean	112	226	184	369	3·6	7·3	5·4	10·9	1·8	3·8	7·2	14·6
s	12	21	12	23	0·3	0·7	0·9	1·5	0·8	1·4	1·1	2·2
X80–405	87	175	135	269	3·1	6·2	6·3	12·3	3·1	6·8	9·4	19·0
X80–628	97	193	152	301	2·7	5·6	6·4	12·8	1·8	4·1	8·2	16·9
X81–121	95	188	152	308	3·1	6·0	4·9	8·9	2·9	6·2	7·8	15·1
X81–214	112	227	172	342	3·5	7·2	5·5	10·2	2·0	5·1	7·5	15·3
X81–395	111	227	160	334	2·7	5·3	3·6	8·5	3·0	5·8	6·6	14·3
X81–554	101	202	157	308	3·0	5·9	4·5	8·4	1·8	4·1	6·3	12·5
X82–274	100	202	145	290	2·9	5·9	3·5	7·4	2·1	4·0	5·6	11·4
Mean	100	202	153	307	3·0	6·0	5·0	9·8	2·4	5·2	7·3	14·9
s	9	19	12	25	0·3	0·6	1·2	2·1	0·6	1·1	1·3	2·6

FCCl, FCCt, fresh volume of frontal cerebral cortex left and total; POTCCl, POTCCt, fresh volume of parieto-occipitotemporal cerebral cortex left and total; HPl, HPt, fresh volume of hippocampus left and total; ASMl, ASMt, fresh volume of medial striate cortex left and total; ASLl, ASLt, fresh volume of lateral striate cortex left and total; ASFVl, ASFVt, fresh volume of striate cortex left and total; s, standard deviation.

Table 4. *Summary of relations between several fresh brain volumes*

Relations	Percentages for Caucasians mean	s	Percentages for Aborigines mean	s
CFV/FBV	11·2	0·8	11·2	1·0
PMOFV/FBV	1·6	0·1	1·8	0·2
PMFV/FBV	86·9	0·9	86·7	1·2
CCFV/FBV	43·2	1·3	42·8	1·9
FCCFV/CCFV	38·0	1·5	39·6	1·0
POTCCFV/CCFV	62·1	1·5	60·3	1·0
HPFV/CCFV	1·2	0·2	1·2	0·2
ASFV/CCFV	2·5	0·4	3·0	0·7
ASMFV/ASFV	75·2	8·2	65·4	5·6
ASLFV/ASFV	25·1	7·9	34·7	5·6

FBV, fresh brain volume; CCFV, fresh volume of cerebral cortex; ASFV, fresh volume of striate cortex; CFV, fresh volume of cerebellum; PMOFV, fresh volume of pons-medulla oblongata unit; PMFV, fresh volume of prosencephalon-mesencephalon unit; FCCFV, fresh volume of frontal cerebral cortex; POTCCFV, fresh volume of parieto-occipitotemporal cortex; HPFV, fresh volume of hippocampus; ASMFV, fresh volume of medial striate cortex; ASLFV, fresh volume of lateral striate cortex.

The volume of the visual cortex was greater for Aborigines (14·9 ± 2·6 ml) than for Caucasians (14·6 ± 2·2 ml). The difference was not statistically significant. No statistical difference was found for the volume of the medial area striata (9·8 ± 2·1 ml to 10·9 ± 1·5 ml). The lateral part of the visual cortex was significantly larger in Aboriginal brains (5·2 ± 1·1 ml to 3·8 ± 1·4 ml).

The striate area in Aboriginal brains represented a higher percentage of cerebral cortical volume than in Caucasian brains (3·0 ± 0·7 % and 2·5 ± 0·4 %, respectively), but this was not statistically significant. The distribution of the visual cortex in the two populations showed significant differences. The medial part represented a lower percentage of visual cortex in Aborigines (65·4 ± 5·6 %) than in Caucasians (75·2 ± 8·2 %), and *vice versa* for the lateral visual cortex (34·7 ± 5·6 % and 25·1 ± 7·9 %, respectively).

DISCUSSION

In comparing these populations, some comments must be made regarding differences in fixation time, cause of death, delay between death and autopsy, age distribution, and body height (Table 1).

The brains of the Caucasian population were collected between May and July 1981, the Aboriginal brains were sampled from 1980 to 1982. Thus, the duration of fixation was different for the two populations. According to Kato (1938) and Paul (1971) variations in volume occur during formalin fixation. Cammermeyer (1956) observed different degrees of shrinkage in formalin for different parts of cat spinal cord. These influences have, to a large degree, been corrected by calculating the fresh volumes of each area in relation to the volume change of the whole brain. However, differences in shrinkage values between grey and white matter could not be corrected. The delay between death and autopsy was not significantly different, although delays of up to 5 days occurred for Aboriginal brains (Table 1). Correlated with time after death, Brandes (1927) and Tobias (1970) observed a postmortem enlargement of the brain by up to 9 %. Age distribution was not significantly different in the two populations. According to Haug (1975) and Spann (1956) a decrease in brain weight can be observed commencing in about the sixtieth year of life. Miller, Alston & Corsellis (1980) found a decrease in brain weight of 3·5 % for each decade of life. However, because of the low mean ages of both populations in this study age-related shrinkage of the brains is unlikely to play a significant role.

A variety of diseases may influence brain weight. Low brain weights have been observed after several chronic illnesses (Spann, 1956; Tobias, 1970). Correspondingly mean fresh brain weights were found to be higher in populations from Forensic Departments compared to those from Departments of Pathology (Matiegka, 1902). However, both populations for this study came from Forensic Departments. With regard to the body weight to body height ratio in case X 81-121, undernutrition must be suspected. Wernicke's encephalopathy was documented in this case. Because all measured volumes were in the mean range for each area, this brain was not excluded from the study.

The mean values for brain weights determined in this study are in accordance with measurements by other authors for Caucasians (Dekaban & Sadowsky, 1978; Holloway, 1980; Kretschmann *et al.* 1979; Paul, 1971; Spann & Dustmann, 1965; Zilles, 1972) and for Aborigines (Davis, 1868; Harper & Mina, 1981; Karplus, 1902; Packer, 1949; Rolleston, 1887/88). Before genetic variations can be implicated for

these differences in brain weights and most volumes, some of the environmental factors known to influence the development of the human brain must be considered

There are numerous studies concerning nutritional influences on the growth of the rat and human brain (Cragg, 1972; Dobbing, 1968, 1970, 1974; Dobbing & Sands, 1970, 1971; Stoch & Smythe, 1963, 1967; Winick, 1969; Winick, Fish & Rosso, 1968; Winick, Rosso & Waterlow, 1970; Winick, Brasel, Velasco & Rosso, 1974). According to Dobbing & Sands (1973) and to Winick, Rosso & Waterlow (1970) each area of a brain has its own growth period. During this period each area is susceptible (vulnerable period) to undernutrition. In man two growth phases have been distinguished. The first involves neuronal development and migration during early pregnancy, the other relates to neuronal maturation, glial proliferation, and myelination.

The second growth period causes the largest volume increase in brain development, starts in the second half of pregnancy, and lasts until the second postnatal year. Stresses in early pregnancy may lead to neuronal deficiencies, whereas stresses during late pregnancy or early life may lead to glial and myelination deficits (Dobbing & Sands, 1970). Studying the effects of undernutrition on the development of rat brains, several authors (Cragg, 1972; Dobbing & Sands, 1971; Winick, Fish & Rosso, 1968; Zamenhof, Marthens & Margolis, 1968) have found that volume deficits due to undernutrition during vulnerable periods cannot be compensated completely by optimal nutrition in later life, unless optimal nutrition starts during the vulnerable period (Winick, Fish & Rosso, 1968). Winick, Brasel, Velasco & Rosso, (1974) showed that undernutrition of pregnant rats led to lower brain weights in their offspring. According to Winick, Rosso & Waterlow (1970) different areas of a brain show volume depletions in correlation with their growth velocity. Fast growing areas can be damaged to a greater extent than slowly growing parts. Kretschmann *et al.* (1986*a*) determined the maximal volume increase of the human brain to occur about 400 days post-conception. Studying undernourished children several authors have found decreased brain weights compared to normal mean values (Beckett, 1970; Brown, 1966; Davies & Davis, 1970; Naeye & Burlington, 1965; Parekh, Pherwani & Udani, 1970). Stoch & Smythe (1963) noted a deficit of 2·28 cm in the head circumference of undernourished South African children 6 to 7 years of age. Despite improved nutritional status the deficit compared to age-matched controls was 2·46 cm four years later (Stoch & Smythe, 1967). Calculating the skull capacity, this corresponded to a decrease of 163 ml or 13·7 % compared to their control group. Brandt (1981) showed that a reduction in head circumference of children with a birth weight under 2500 g can be corrected in the first 6 to 9 months of life by optimal nutrition. Thereafter no improvements can be expected.

According to 'Aboriginal illth' (1981) life expectancy for Aborigines is about 20 years less than for the white Australian population. Infant mortality is about twice as high ('Aboriginal illth', 1981; Edmonds, Roberts & Schlafrig, 1970; Hausfeld, 1977). Aboriginal neonates have a lower mean birth weight compared to white Australian neonates (Brown & Barrett, 1972; Seward & Stanley, 1981). However, using the postnatal increases in body height, body weight and head circumference as indicators for nutritional status, no gross differences could be observed between European and Aboriginal children until 6 months of age. At the age of 1 year body weight for Aboriginal children lagged behind by 2 kg, body height and head circumference showing smaller deficits (Kettle, 1966). Comparable results were obtained by

Jose & Welch (1970), Kirke (1969) and Maxwell & Elliott (1969). Aboriginal children undernourished in early infancy could only partly compensate for these deficits in later life (Edwards, 1970; Maxwell & Elliott, 1969).

Aboriginal children living in missions and rural areas showed greater deficits than those living in larger cities (Cockington, 1980). The main reasons quoted for the observed deficiencies are undernutrition due to cessation of breast feeding and the high rate of infectious diseases (Cox, 1979; Edwards, 1970; Forbes, Williams & Macdonald, 1973; Maxwell & Elliott, 1969; Walker & Harry, 1972).

Genetic differences in head circumference between different populations have not been identified so far (Nellhaus, 1968). Comparative studies of brain weights of black and white Americans by Ho, Roessmann, Straumfjord & Monroe (1980) and Ho, Roessmann, Hause & Monroe (1981) showed no differences up to the age of 6 years. Thereafter, brain weights of black Americans were significantly lower. The authors concluded that environmental factors such as undernutrition, alcoholism, and social circumstances were responsible rather than genetic differences.

Associated with improved health, social and nutritional status of Aboriginals in the Northern Territory from 1930 to 1971, body height of young male Aboriginals has increased by 5·5 cm, and body weight by 6·4 kg (Barrett & Brown, 1971; Brown & Barrett, 1972, 1973). Similar processes might account for comparable observations by Kretschmann *et al.* (1979) for head circumference and brain weight of the European population from 1880 to 1976. They found an increase in the average brain weight of 69 g.

On the other hand the whole life style of Aborigines has changed drastically. Formerly living as nomads in the Australian deserts and bushlands they now live in missions, Aboriginal settlements, and cities. This might be one reason for the high rate of infectious diseases in Aboriginal children (Campbell & Barrett, 1953; Moodie, 1973; Sinclair, 1977). Diseases related to modern civilisation such as hypertension and diabetes are now observed more often in Aborigines (Wise *et al.* 1970). In contrast to nutritional status, health status of Aboriginals living as nomads is better compared to Aboriginals living in cities (Abbie, 1974; Hitchcock & Gracey, 1975; Wise *et al.* 1970).

Another major health problem related to their drastic change in life style is alcoholism. Hunt (1981) observed in 80 % of all Aboriginal families at least one alcoholic. Kamien (1975) found an alcohol intake of more than 80 g per day in 53 % of male Aborigines in New South Wales. Thirty six per cent of male Aborigines 14–19 years old drank alcohol regularly and 46 % of male Aborigines 20–29 years old had an alcohol intake of 120 to 180 g per day. The incidence of pneumonia, gastroenteritis, liver cirrhosis, pancreatitis, and accidents due to alcoholism is very high in Aboriginals (Hunt, 1981). Harper, Kril & Holloway (1985) observed a decrease of 71 g of the mean brain weight in a population of alcoholics compared to non-alcoholic controls. Correspondingly the brain volume was also reduced in alcoholics. The reduction of brain volume was found to be due to a loss of white matter rather than grey matter. In another study, Harper & Kril (1985) found decreases in the mean brain weight of 41 g in alcoholics without encephalopathy or liver disease, of 89 g in alcoholics with Wernicke's encephalopathy and of 109 g in alcoholics with liver disease.

The differences in nutritional and health status of Aboriginals compared to Caucasians could explain a fresh brain weight difference of 180 g or 12·6 % quite easily. On the other hand, different genetic influences on brain development cannot be

excluded. Further evidence for both arguments can be found in the data presented in this study.

The mean values of the volumes for the cerebral hemispheres, brainstem, and cerebellum of Caucasians are comparable to those obtained by Breuer (1973), Haug (1970), Jenkins & Truex (1963), Lange & Albring (1979), Paul (1971), Schlenska (1969), Wessely (1970) and Zilles (1972). The volumes of prosencephalon-mesencephalon unit and cerebellum were significantly less in Aborigines. For pons-medulla oblongata unit no significant difference could be found. The time of maximal growth of the cerebellum was determined by Kretschmann *et al.* (1986*b*) to be around 464 days post-conception. At birth only 13 % of the final cerebellar volume has been attained. The vulnerable period is probably around the sixth postnatal month, which corresponds to the delays in physical development in Aboriginal children. So the lower results for Aboriginal cerebellar weights could be due to disturbances in brain development by undernutrition, infectious diseases etc. as described above.

Winick, Rosso & Waterlow (1970) found rapid increases in DNA-content in the prosencephalon and cerebellum during their vulnerable period, but a slow rate of increase for the brainstem. According to studies on rat brains by Fish & Winick (1969) fast growing areas are more susceptible to undernutrition than slowly growing areas. Therefore, the brainstem should be less sensitive to undernutrition than other parts of the brain. This was shown by Winick, Rosso & Waterlow (1970) studying 16 brains of marasmic children. DNA-content in prosencephalon and cerebellum was reduced compared to a control group, whereas the difference in DNA-content of the brainstem was less striking. This finding could explain why no significant difference in the absolute volume of the pons-medulla oblongata unit was noted between the two populations in this study.

The volume of the cerebral cortex was significantly less in Aboriginal brains. Our results for Caucasians are comparable with other studies (Fischer, 1981; Haug, 1970; Henneberg, 1910/11; Jäger, 1914; Kretschmann & Wingert, 1970; Lange & Albring, 1979; Paul, 1971; Zilles, 1972). The higher percentage of frontal cortex in Aboriginal brains cannot be explained so far.

Studies by Haug (1970) and Fischer (1981) revealed similar values for the volume of the hippocampus of Caucasians. The volume of the hippocampus in Aborigines was significantly less. Due to the large individual variation in size of the hippocampus as already stated by Gertz, Lindenberg & Piavis (1972) and Kretschmann *et al.* (1986*b*), this area does not correlate with brain size (see Fig. 9). The sample size in this study is too small to draw conclusions with regard to this. Kretschmann *et al.* (1986*b*) determined that the vulnerable period for the hippocampus is around 313 days post-conception. Considering that delays in Aboriginal physical development were observed from the sixth postnatal month onwards, a less striking difference in the fresh volume of the hippocampus would have been expected.

No significant difference was found for the fresh volume of the visual cortex ($14{\cdot}6 \pm 2{\cdot}2$ ml for Caucasians and $14{\cdot}9 \pm 2{\cdot}6$ ml for Aborigines). Similar values for Caucasians were determined by Gerhardt & Kreht (1933), Lange & Albring (1979), and Sauer *et al.* (1983). The half value time of the visual cortex is around 265 days post-conception (Kretschmann *et al.* 1986*b*), which predates delays in physical development in Aboriginal children. Rabinowicz (1981) found that the final thickness of visual cortex is attained by the sixth prenatal month. However, Sauer *et al.* (1983)

state that there is a growth spurt of the visual cortex at the time of birth and that the maximum volume is only attained by 400 days post-conception.

The development of a normal optic system is dependent upon normally functioning eyes. Studying Rhesus monkeys (*Macaca mulatta*), Wiesel (1982) showed that the growth of the visual cortex could be inhibited by closing one eye for some time about the sixth postnatal week. Noorden (1981) stated that congenital diseases of the eye have to be corrected as quickly as possible to ensure a normal development of the optic system. For diseases starting after 6 years of life the time of correction made no difference to the final outcome. Both these studies prove that the perinatal period is critical for the development of the optic system which corresponds to the growth spurt of the visual cortex. In this regard it should be noted that case X 82-274 was blind in his left eye. It is not known when his blindness started, but he had the smallest visual cortex of all Aboriginals studied.

Taylor (1981) noted that Aboriginals had a higher visual acuity compared to white Australians. Kearins (1976, 1981), studying the visual memory of Aboriginal and white Australian children of different age groups, found that Aboriginal children had a better visual memory and concluded that they rely to a greater extent on visual strategies to solve problems compared to white Australians who rely more on verbal strategies. These differences in visual acuity and visual memory could be interpreted as an adaptation to living conditions in the bush and desert regions of Australia.

As in this study, several other authors have found that the visual cortex is distributed to a greater extent on the lateral surface of the brain in Aborigines (Flashman, 1916; Shellshear, 1937; Woollard, 1929). This might be explained by the hypothesis that the visual cortex is pushed medially by growth of the parietal cortex. This process might have occurred to a lesser degree in Aborigines, as might be concluded from their smaller percentage of parieto-occipitotemporal cortex. However, this type of distribution of the visual cortex is not unique to Aboriginal brains (Antoni, 1914) and in this study one Caucasian brain (H 28/81) had a large lateral visual cortex, too.

All the findings and hypotheses presented in this study must be interpreted with caution. No studies exist about the growth and development of Aboriginal brains. Although this is a small study, highly significant differences between both populations were identified for most of the areas studied. Further studies must be undertaken in order to determine the importance of genetic and environmental influences on the development of the brains of the Aborigines. In particular, studies of the visual cortex hold much promise because of the associated clinical differences identified by Taylor (1981) and Kearins (1976, 1981).

SUMMARY

The brain volumes of 8 male Australian Aborigines and 11 male Caucasians were determined. Total brain volume was significantly smaller for Aborigines (1199 ± 84 ml) compared to Caucasians (1386 ± 98 ml). Significantly smaller volumes were also found for cerebellum, prosencephalon-mesencephalon unit, cerebral cortex, frontal cortex, parieto-occipitotemporal cortex, and hippocampus. Volumes of pons-medulla oblongata unit (21 ± 3 ml for Aborigines and 22 ± 3 ml for Caucasians) and visual cortex (14·9 ml ± 2·6 ml and 14·6 ± 2·2 ml, respectively) did not differ significantly. The striate cortex extended further onto the lateral surface of the occipital lobe in Aboriginal brains. The frontal portion of cerebral cortex was larger in Aboriginal than in Caucasian brains.

According to the specific growth periods for the areas studied, these differences could be explained by the higher incidence of malnutrition and infectious diseases for Aboriginals during the development of the brain in early childhood, especially after the 6th postnatal month. However, genetic influences cannot be excluded. The results for the visual cortex of Aborigines might represent an adaptation to living conditions in the bush and desert regions of Australia.

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