planation of this variation in excretion rate is far from clear, although several possibilities, such as variations in kidney D-amino acid oxidase activity and renal readsorption mechanisms, have been discussed (*5, 8*). Because of the low levels of D-phenylalanine in this work, it was not possible to examine any of these physiologic hypotheses.

What is of most interest to us is the genetic information that can be obtained from a comparison of the intrapair differences and variances in excretion rates between the pairs of monozygotic and dizygotic twins. As can be seen in Table 1, the average intrapair difference for the dizygotic twin pairs is more than twice as large as that for the monozygotic pairs. The intrapair variance ratio for the dizygotics/monozygotics is 11.7, which, with 5 and 3 degrees of freedom, is significant at higher than the 5 percent level. This would mean that at least part of the observed variation in excretion rate of D-phenylalanine is the result of genetic differences. The work of Goodman (*8*) on mice is particularly relevant here, since she also found evidence for genetic control of variation in excretion rates of the D-isomers of several amino acids, including phenylalanine. It would appear that further work on the physiology and genetics of differential excretion of the D-isomers would be very rewarding.

STANLEY M. GARTLER
RICHARD E. TASHIAN
Institute for the Study of Human Variation, Columbia University, New York

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Glia/Nerve Cell Index for Cortex of the Whale

Since man occupies the top position in the phylogenetic scale and has attained the highest intellectual development, there has been a well-understood tendency to relate certain characteristics of the morphology of his brain to this development. Early attempts to parallel intellectual performance with brain weight, relationship of brain weight to body weight, and number of convolutions now have only historical interest. Certain aspects of the histology of the cerebral cortex were also thought to reflect phylogenetic development. The idea that the more highly developed cortex contains more "space" between nerve cells probably was expressed first by Nissl (*1*) and was later supported by such an authority on cerebral cytology as Economo (*2*). More recently, Friede (*3*) studied the glia index (number of glia cells per nerve cell) in the cortex of various animals and found that it increases from the frog to man. He concluded that the ascending phylogenetic development of the cortex is characterized by a relative increase in glia cells.

However, the human brain is not only the most highly developed but it is also the largest brain that is usually studied. It is possible, therefore, that certain histological characteristics of the cerebral cortex may reflect an increase in size and not in the phylogenetic development. Thus, Tower (*4*), studying the cell density of the cerebral cortex, included in his series the brain of the whale and elephant, which are the only two animals with a brain weight higher than that of man. He found that the cortical cell density was inversely correlated with brain weight, and not with the position of the animal on the phylogenetic scale.

It is the purpose of this report to show that the glia/nerve cell index reflects brain weight rather than phylogenetic development. Our histological material was a portion of that used by Tower (*4, 5*) and consisted of 20-μ paraffin sections of two whale (*Balaenoptera physalus* L.) brains which weighed 6500 g and 7150 g, respectively. Cortex from three sections, two from the frontal region and one from the occipital region, was studied. A 20-μ paraffin section of the temporal cortex of a 36-year-old woman was also examined for the purpose of the comparison of our results with those of Friede (*3*).

Photomicrographs at a magnification of 80 were made, and the glia cells and nerve cells were marked on them with the aid of direct microscopic observation of the slides. No attempt was made to differentiate between the types of glia cells, but care was taken to avoid marking endothelial nuclei. The marked cells were counted according to the following rules. In the whale cortex, the counts were made separately only for layer II; the deeper layers were counted together. Separate counts for nerve cells containing or not containing nucleoli were made. In the human cortex, layers II to IV were counted separately, layers V and VI together. All nerve cells were counted, irrespective of whether or not they contained nucleoli. Data are recorded in Table 1.

Table 1. Number of glia and nerve cells counted and the glia/nerve cell index for whale and man.

Layer	Cells counted		Glia/nerve cell index
	Glia	Nerve	
Whale, specimen No. Cst-1			
II	185	167	1.11
III to VI	2116	473	4.47
All	2301	640	3.59
Whale, specimen No. C-293			
II	277	150	1.84
III to VI	1717	264	6.50
All	1994	414	4.81
Whale, specimen No. C-293			
II	290	186	1.56
III to VI	2454	311	7.89
All	2744	497	5.52
Whale (total)			
	7039	1551	4.54
Man			
II	714	468	1.53
III	627	343	1.83
IV	870	424	2.06
V and VI	657	371	1.27
Man (total)			
	2868	1606	1.78

As can be seen from Table 1, the glia/nerve cell index of the cerebral cortex is much higher in the whale than in man. The value for the human cortex is 1.78, which is very close to the ratio of 1.68 established by Friede. Our index was obtained by investigation of only one region in the first temporal convolution. Because of the agreement of our value with that of Friede, we did not investigate other regions. In the whale, we studied three regions, two from one animal and one from the other. Though the ratio varied from 3.59 to 5.52, it was significantly higher than in man, the average for all three regions being 4.54. Statistical analysis of these results was made, applying the formula

$$t = \frac{x - y}{\sqrt{\frac{x(1-x)}{N_1} + \frac{y(1-y)}{N_2}}}$$

and the results were found to be highly significant.

These values for the whale were obtained when all nerve cells, whether they contained nucleoli or not, were counted. By applying such a method, we incurred an error resulting from the fact that when particles are counted in histological sections, the true number per volume is smaller than that counted (*6*). This error increases with the increase of the ratio between the size of the particles and the thickness of sections. Since the nerve cells of the whale are larger than those of man, and since all our sections were 20 μ thick, we overestimated the number of nerve cells for the whale cor-

tex to a slightly greater extent than we did for the human cortex. Therefore, the actual difference between the indices should be even higher than that given. In order to find out the size of the error with which we were dealing, we also counted in the whale only such nerve cells as contained nucleoli. The glia/nerve cell index obtained in this manner was 5.86.

It is interesting to note that the index for the whale was consistently lower in the second cortical layer. This layer is also more cellular than the rest of the cortex, and it is possible that these two characteristics are correlated. The differences between the indices of the three regions of the whale cortex may represent consistent regional variations, or be only an accidental finding. This problem requires further investigation. We did not intend to establish absolute values, but only to compare the index for man with that for the whale.

Thus our results indicate that the increase in the number of glia cells per nerve cell is not correlated with the phylogenetic development, but with brain size. The significance of this increase is not known, but it may be suggested that it is related to the increase in the size of the nerve cells, which have longer processes and require more assistance from the supportive tissue to meet their metabolic needs. It may be of great interest for the understanding of the physiology of glia cells to determine whether one particular type of glia cell is involved in this increase.

A. Hawkins
J. Olszewski
Department of Pathology, College of Medicine, University of Saskatchewan, Saskatoon

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Lack of Congenital Malformations in Normal Human Pregnancies after Transabdominal Amniocentesis

Recently considerable interest has been shown in studying human amniotic fluid. There are many references scattered throughout the literature which suggest that analysis of amniotic fluid may be of diagnostic value. Amniotic fluid may be considered as an additional body fluid compartment in the pregnant mother. Theoretically, amniotic fluid should reflect physiologic and pathologic conditions of the fetus or maternal host, or both, just as whole blood, plasma, urine, and cerebrospinal fluid indicate pathologic conditions in the nonpregnant host.

Table 1. Experience with transabdominal amniocentesis in 50 normal patients.

Gestation (wk)	Patients (No.)	Taps	
		Successful	Unsuccessful
20–24	5	5	0
24–28	8	8	0
28–32	13	12	1
32–36	13	11	2
36–40	11	10	1
Total	50	46	4

One reason amniotic fluid has not been studied extensively throughout various stages of gestation in human beings is that most physicians and investigators do not realize how easily and safely it can be obtained. Rivett (*1*) discussed the theoretical complications of transabdominal amniocentesis in human beings. More recently, Trasler and her associates (*2*) reported experimental evidence of congenital malformations in mice following puncture of the amniotic sac. They suggested that the procedure may produce similar congenital malformations in human beings.

The purpose of this report is to describe our results with 50 transabdominal amniocenteses in normal human beings during the last two trimesters of pregnancy. An 18-gage spinal needle with a trochar was used for these tests. From 15 to 25 ml of fluid was withdrawn when possible. Table 1 lists the patients by weeks of gestation and indicates the results obtained. There were 46 successful and four unsuccessful taps. The only maternal complications immediately following amniocentesis were two patients who developed infections of the urinary tract. We attribute these to faulty sterile technique in preoperative catheterization. None of the patients had premature labor precipitated by the procedure. All of the abdominal wounds healed without infection.

Each mother was followed during her prenatal course, delivery, and postnatal course. The placenta and fetus were carefully examined for evidence of trauma or other abnormalities which might have resulted from puncture of the amniotic sac. All the placentas appeared normal. The infants were all perfectly formed and were without external signs of congenital malformations. No evidence of fetal trauma was found.

The only complication was one primagravida who developed acute preeclampsia 5 weeks after amniocentesis. She experienced a complete placental separation during the thirty-fourth week of pregnancy, and the infant was stillborn. The stillborn infant had no anatomic abnormalities or evidences of trauma. Because this complication occurred such a long time after transabdominal amniocentesis, we do not feel that the procedure was a causative factor.

In our experience, transabdominal amniocentesis is a safe and easy way to obtain amniotic fluid in normal human beings during the last two trimesters of pregnancy. There was no evidence of maternal or fetal trauma. In contrast to the high incidence of congenital malformations produced by amniotomy in mice, we found no congenital malformations in human beings following transabdominal amniocentesis. Perhaps these differences are related to the stage of pregnancy when amniocentesis is performed and the ratio of fetal volume to amniotic fluid volume in various species.

Henry M. Parrish*
Frank R. Lock
Mary E. Rountree
Department of Obstetrics and Gynecology, Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, North Carolina

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* American Cancer Society fellow in epidemiology and biometry, Department of Public Health, Yale University School of Medicine, New Haven, Conn.

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Monitoring of Low-Frequency Phenomena

Many physical phenomena occur at frequencies which are generally so low that they require visual attention during monitoring or recording. Whether such phenomena are detected through their concomitant electric activity [for example, electric activity of the heart (ECG) or brain (EEG)] or through the use of electric transducers (for example, for measuring blood pressure or other fluctuating pressures), audio monitoring frees the visual attention of the experimenter for the observation of other phenomena or for the performance of other tasks. The experimenter is given a constant indication of the experiment and may be confident that he will hear any changes as soon as they happen.

A transistor regenerative oscillator was adapted from an experimental model (*1*) to convert subaudible frequencies into audio frequencies. Other transistor oscillators have been described (*2*) which could be similarly adapted. The frequency of oscillation of the oscillator varies inversely with the supply voltage.