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THE AUDITORY BRAINSTEM RESPONSE (ABR) EVALUATES RISK FACTORS FOR HEARING LOSS IN THE NEWBORN

Robert Galambos and Paul-Andre Despland

Speech, Hearing and Neurosensory Center
Childrens Hospital Complex
San Diego, California 92123 and
Department of Neurosciences
University of California, San Diego
La Jolla, California 92093

SUMMARY

Fourteen of 100 unselected patients in an intensive care nursery were found by the auditory brainstem evoked response (ABR) method to suffer significant hearing loss; of these 8 were ultimately discharged home. Analysis of the 100 clinical records identified 9 risk factors of which most, like low Apgar scores, are already known (Table I). However, neonatal asphyxia appeared to be associated with hearing loss only when repeated episodes of acidosis accompanied it (Table III). We conclude that the ABR readily identifies the hard-of-hearing premature and estimates the type and amount of his peripheral hearing loss, and that physiological events associated with prolonged perfusion of the cochlea with blood low in pH may be the most common cause of hearing disorder in this group.

SPECULATION

Since this study points to the consequences of postnatal acidosis as an important factor in producing damage to the cochlea, it reraises the old question of the relative roles played by hypoxia and low pH in producing the irreversible brain damage that can occur at and near birth. The statistic suggesting that nearly 10 per cent of the babies discharged from an intensive care nursery suffer an irreversible peripheral hearing loss may surprise and dismay neonatologists, and needs in any event to be validated by studies on similar populations. Whether these losses are permanent, furthermore, can only be settled by appropriate follow-up studies.

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About 10 percent of the graduates of an intensive care nursery show peripheral hearing disorder. The clinical histories of those affected show frequent bouts of acidosis at and after birth.

INTRODUCTION

The factors that place neonates at-risk for hearing loss have been listed (e.g., 13) and can be classified under three major headings: genetic disorders, of which there are at least 60 different types (16); maternal infection (e.g., rubella, toxoplasmosis, syphilis: 14); and a long list of peri-and postnatal states and events such as prematurity, infection, hypoxia, drugs and hyperbilirubinemia (e.g., 3,8,9). At the present time, most of the newborns falling into these categories - and hence the ones most likely to suffer hearing loss - are isolated in the infant intensive care nursery (ICN) at large hospital centers because of their obvious congenital anomalies, very small size, unusually low Apgar scores, history of infection, etc. The actual incidence of significant hearing loss in such babies has been estimated at around 1:20 (22).

In a companion paper (7) we have reported our tests on 108 ICN patients using the auditory brainstem response (ABR), a new physiological method for evaluating the audiological and neurological status of infants, children and adults (see 6,20 for reviews). In this group, 18 were found to have audiological disorder, neurological disorder, or both. In the present paper we deal with a subpopulation of that group consisting of 100 babies representing 97% of the ICN population during a 5-month period. Our primary aim was to identify every baby with a hearing loss and to specify its magnitude and type (conductive, sensorineural, central); thereafter we examined the clinical histories of the entire group in an attempt to uncover the otonoxious risk-factors and arrange them in order of importance.

METHODS

As described previously in detail (7) the ABR was computer-extracted from the EEG recorded through conventional electrodes attached to the vertex and mastoid. Stimuli were clicks delivered monaurally through an earphone taped over the ear. The infants were all tested by the same person, about 30 mins. post-feeding and while they were in natural sleep.

RESULTS

A. ABR Audiometry

1. Infants with normal hearing.

Figure 1 shows ABRs derived from left and right ear stimulation of a premature infant. This baby is considered to be audiologically and neurologically normal because, as discussed in our previous paper (7), the 60 dB stimulus to each ear yielded an ABR whose wave V latency was normal for age, and the 30 dB stimuli successfully evoked recognizable responses bilaterally. In the 100 infants of this series 86 (125 ears) fulfilled these criteria for normal hearing.

The latency of wave V decreases, as Fig. 1 shows, when stimulus intensity increases. The plot of this relationship, the so-called intensity series, is shown in Fig. 2 along with similar plots for normal adults and infants at different ages. These curves provide the norms against which the intensity series from a given patient is to be compared. As we shall shortly see the infant with significant hearing loss, like the adult, produces a wave V latency curve that deviates in characteristic ways from that expected at his age.

2. Infants with hearing loss.

The 14 patients (28 ears) who failed to meet the ABR criteria for normal hearing consisted of 11 prematures, 2 newborns and one aged 18 months. These patients fall into the following categories defined in our previous report (7).

Group I: 3 infants who produced no ABR to clicks at a level of 90 dB, the limit of our apparatus. Two died within a few days of birth while the third was ultimately discharged in good health. We classify these infants as having a hearing loss of at least 90 dB at the time of test.

Group III: 2 infants with ABR responses permitting a diagnosis of both audiological and neurological disorder.

Group IV: 9 infants neurologically normal by ABR but with elevated auditory threshold.

The intensity series' of the 11 babies of Groups III and IV are plotted in Fig. 3. Two abnormal features in these curves provide the diagnostic audiological information. First, in all cases the threshold ABR appears at an intensity of 40 dB or higher instead of at the 15-25 dB level estimated as normal for infants of 35 wks g.a. and older (7,21,24); on this basis a hearing loss of the order of at least 20 dB existed in the "best" ears in the group. Second, the patient curves deviate from those of infants of comparable age, being either somewhat displaced to the right (2 cases), or showing a very steep slope at intensities near an elevated threshold (9 cases). These deviations are known to characterize conductive and sensorineural hearing loss, respectively, in adults whose losses have been established by conventional audiometric procedures (10,26). On this basis we consider 9 of the 11 infants to suffer sensorineural losses varying between about 45 and 75 dB in magnitude (re the threshold for adults), and 2 to show a small conductive loss approaching 40 dB.

B. The High-Risk Factors for Hearing Loss in Newborns

1. Identifying the factors.

The effort to identify the clinical factors predisposing for these hearing losses began with a search through the charts of the affected babies for probable or likely causes. This search yielded the nine categories of apparently relevant information shown at the left of Table I; it includes weight and gestational age at birth, Apgar scores at 1 and 5 mins., acidosis, ototoxic damage, various signs of central nervous system disorder, the number of days on assisted ventilation, and visible evidence of congenital malformation. Appropriate subdivisions of these nine categories were then established as also shown in Table I, and the number of normal and abnormal infants falling into each category was tabulated. To decide whether a particular subdivision represented a significant high-risk factor in the clinical history the following arbitrary rule was applied: a risk factor is identified when the number of babies with abnormal ABRs exceeds or approximates the number of normals. In this way, 9 risk factors and 3 possible ones were extracted as indicated in the right hand column of Table I.

All the items included in this list, except for the factor acidosis, are either known or suspected risk-factors for hearing loss. By "acidosis" we mean a blood pH measurement of 7.25 or below within an hour or two of birth, and/or entries in the baby's chart showing that on 2 or more days during treatment blood pH sank below 7.25.

Table II lists the risk factors found in the clinical records of the babies with abnormal ABRs. Infants 1 and 2 in the Table showed only congenital cleft palate abnormalities; one was deaf to the 90 dB signal while the other did not respond below about 55 dB. A third baby with cleft palate also had low Apgar scores; his hearing loss was of the conductive type amounting to about 40 dB. Three risk factors (asphyxia, acidosis, coma) were involved in 2 cases of which one gave no response from either ear at 90 dB and died within 48 hours while the other had a sensorineural loss of about 40-45 dB. The baby for whom 4 factors are listed suffered an intracranial hemorrhage; the ABR identified impaired conduction through the brainstem and a 60 dB sensorineural type of loss. Five risk factors were involved for one patient who gave no responses to clicks below 80 dB. Six risk factors can be listed for 2 babies, one with a mild sensorineural loss by ABR, the other with a mild conductive loss and a prolonged I-V interval (indicating neurological disorder) in addition. Seven risk factors characterized the histories of 5 babies, one of whom showed only wave I and hence was also a neurological case; the sensorineural hearing loss present was moderate in one and severe in four. As already stated, the ABR threshold and intensity series for each of these babies is plotted in Fig. 3.

Further examination of Table II offers suggestions about the relative importance of the risk factors themselves. Infants with cleft palate are obviously at high risk, a fact already clearly stated in the literature (1,11,16). The factors most frequently represented in the table are low Apgar scores (11 cases) and acidosis (10 cases), there being 2 infants for whom these alone (along with a comatose state undoubtedly secondary to them) are identified. Eight in this asphyxiated group were born very prematurely; six of them presented with cardiovascular anomalies, six developed the RDS syndrome and 2 suffered intracranial hemorrhages. One asphyxiated baby showed cleft palate anomalies, a risk factor which alone can account for the hearing loss.

Since low Apgar score appears most frequently in Table II, hypoxia may be the most important of the perinatal factors predisposing for hearing loss. Table III, however, suggests this may not be so. Table III contains all of the 22 babies in our sample with unusually low Apgar scores measured at birth. Of these, 11, or only half of them, showed hearing loss by ABR. Those with hearing loss differed from those without mainly in the pH history during treatment. If blood pH below 7.25 was recorded on several occasions, hearing loss was likely. If pH had even transiently dropped below 7.21 on 2 or more days, hearing loss was highly likely. This suggests that hypoxia at birth was not the main cause of the hearing losses found; instead, infants showed impaired auditory function when the hypoxia-related acidosis persisted for a prolonged period at birth, and/or when several periods of severe acidosis appeared postnatally during treatment.

DISCUSSION

According to this study the ABR method can reliably identify hearing-impaired infants in the ICN. The method uncovered a total of 14 in the population of 100 tested; of these 8 were subsequently discharged home. Using this latter group only, an 8 percent estimate for the incidence of peripheral hearing loss in such a population seems reasonable; this resembles the 5 per cent estimate arrived at in a similar study (22). Evidently, somewhere between 1:10 and 1:20 of the babies discharged from a typical intensive care unit may leave for home with a significant peripheral hearing loss. These losses appear to be permanent, as judged from the retests done on about half of the babies in our series, but this conclusion needs confirmation.

The losses found included 12 of the sensorineural type, i.e., disorders within the cochlea, and 2 involving the conductive apparatus. In magnitude the threshold elevations varied from about 40 dB (re adult) to beyond 90 dB. The prevalence of sensorineural loss in babies asphyxiated at birth has already been noted (8,9) but its presence in 2 of the 3 cleft-palate patients is not in accord with Bess *et al.* (1) who reported mostly conductive losses in such children. Impedance audiometry on our 2 cases, however, cleared them both for middle ear disorder.

Most of the 9 risk factors identified in this study have already been recognized. Certain other well-known factors, however, do not appear in our list and their absence is easily explained: our sample did not contain, for example, a baby whose parents were deaf, whose mother had had a rubella infection or who himself was hyperbilirubinemic. (Parenthetically, the ABR has identified hearing loss due to each of these factors in our ICN during the past several years). Thus our list is not exhaustive with respect to factors that place an infant at-risk for hearing loss; it gives only those factors extractable from the histories of this particular sample of 100 babies.

The rejection of ototoxic drugs as a risk factor (Table I) requires an explanation. We do not of course question that such drugs at high blood levels for prolonged periods will produce cochlear damage. In this study, however, 65 patients received substantial quantities of one or more of them (gentamycin, ampicillin, penicillin, kanamycin) but only 11 displayed hearing loss. Furthermore, every baby who required assisted ventilation and/or perfusions routinely received one or more of the potentially ototoxic drugs, often for protracted periods; out of the 23 or 24 who received comparably large amounts for comparable times, however, only about half showed hearing loss by ABR, and in every instance at least one other risk factor (e.g., acidosis) was also available as an explanation. To exclude ototoxic drugs with certainty would require data about renal clearances, blood levels of drug, etc., which unfortunately are unavailable for these babies. Nevertheless, our somewhat subjective evidence is not compatible with the idea that the ototoxic drugs administered to this group was an important factor in producing their hearing losses.

The ABR has identified two types of lesions in this series: damage to the cochlea and damage to the brainstem (7, Figs. 7 and 9). In asphyxiated neonates the peculiar susceptability of the brainstem auditory pathways has been well documented by anatomical studies in animals (18,27) and man (12), but changes at the cochlear level have not been found (12,23). Most of our abnormal babies, however, showed only cochlear damage (i.e., elevated auditory threshold and an ABR latency-intensity curve characteristic of sensorineural hearing loss); the ABR additionally revealed brainstem damage in only a small number of the most severely affected infants. Thus, the physiological (ABR) evidence suggests the pathological sequence to be cochlear damage first, central damage next, whereas the morphological evidence suggests the opposite should be true. This discrepancy would appear to be resolvable only through appropriate animal studies where ABR physiological results before death are correlated with the post-mortem morphological findings in both the cochlea and brainstem.

Evaluating the etiological factor(s) responsible for the irreversible damage to cochlea and brainstem also will require further study. Our hypothesis that prolonged acidosis at and after birth is closely related to irreversible hearing loss is being advanced for the first time, so far as we are aware, and more data will of course be required to confirm or disconfirm it. Furthermore, even if the hypothesis were correct, the acidotic state in a newborn can have many causes and consequences, and the present study does not specify which of many possible events might be otonoxious ones. Finally, it is possible, though we think improbable, that we have omitted some crucial item in the analysis of risk factors summarized in Table I.

Our analysis indicates that acidosis is a more important predictor of hearing loss than any other physiological variable, including even the hypoxia which it so often accompanies (Table III). It has already been pointed out that in the presence of severe hypoxia the prevention or correction of an acidosis can have important beneficial consequences. An early and dramatic example was described by Nahas (19): dogs, prevented from breathing by succinyl choline injection, received infusions of tris-buffer which maintained blood pH at near-normal values; none of the dogs shows significant change in CSF or arterial pressure during the hour-long experimental period and all recovered completely, whereas about 40 percent of the controls subjected to this drastic procedure died. Another example is provided by Dawes et al. (5), who asphyxiated newborn monkeys for times up to 15 minutes, infusing base into some to prevent the accompanying acidosis: the amount of brain damage seen histologically in equally severely hypoxic infants correlated well with the drop in blood pH permitted, and some of the animals with near-normal pH values during the procedure showed no brain damage at all. Dawes summarizes these and related studies in his 1968 monograph (4). Still another example comes from Hrbek et al. (15), who separately varied the O_2 and pH levels of blood perfusing the fetal sheep: the deterioration in brain function at a given level of hypoxia depended upon how much the pH of the circulating blood was dropped.

In their recent discussions of the damaging effects of neonatal asphyxia neither Strang (25) nor Myers (18) isolates and directly addresses the acidosis question we raise here, although the former does describe the changes attending base infusions to correct acidosis in human neonates, and the latter points to lactic acid accumulation in the brain as a major biochemical correlate of the anatomical damage measured in the monkey. In the anatomical reports on the brains of asphyxiated babies, where gliosis (2) and a drop in cell count at the cochlear nucleus (12) are described, this same failure to consider acidosis specifically as an etiological factor exists: the authors apparently attribute the pathological changes solely to the oxygen lack, although the clinical histories of their babies who survived for days or weeks on assisted ventilation show, as do ours (Table III), frequent and protracted periods of severe acidosis (2,17).

In any event, and despite the uncertainties about the acidotic state which future observations must clarify, we have shown here that nearly all of the nongenetic hearing losses uncovered in our series were associated with episodes at birth and thereafter during which the brain and cochlea were perfused by blood low in pH. If, as the analysis suggests, the events associated with the acidosis, not the hypoxia, were mainly responsible for the irreversible damages measured by ABR, then any neonate in whom acidosis is a problem during the postnatal period should be considered at-risk for hearing loss. This group obviously includes, among others, babies with cardiac anomalies and those requiring long periods of assisted ventilation.

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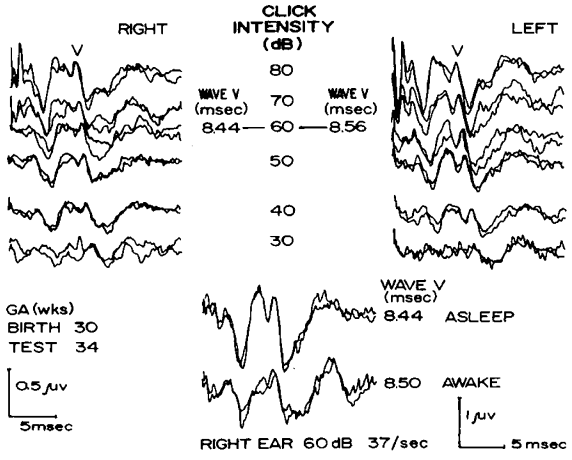

Fig. 1. ABRs recorded from a normal infant of 34 wks gestational age. Each tracing represented the average of 2000 responses to click stimuli at the indicated intensity; two tracings were obtained at each intensity and superimposed. Wave V is located (V) in the 80 dB records and its latency is given for the 60 dB responses. Both left and right ear monaural stimulation yield similar responses at all stimulus levels including 30 dB (re adult threshold for the click). The two ABRs at the bottom of the figure (calibration: lower right) compare the sleep and waking records.

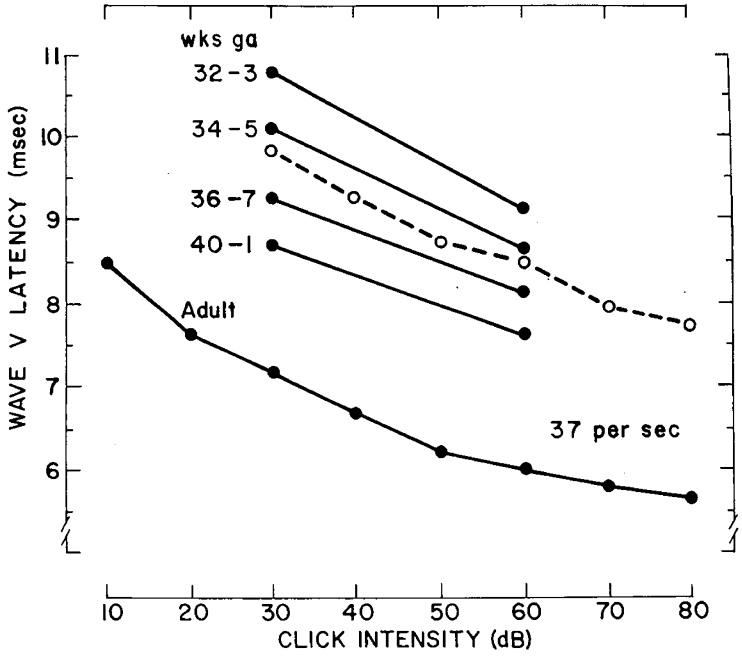

Fig. 2. Normal latency-intensity curves for adults and for premature infants of various ages (solid circles). The wave V latencies measured on the recordings of Fig. 1 are given by open circles.

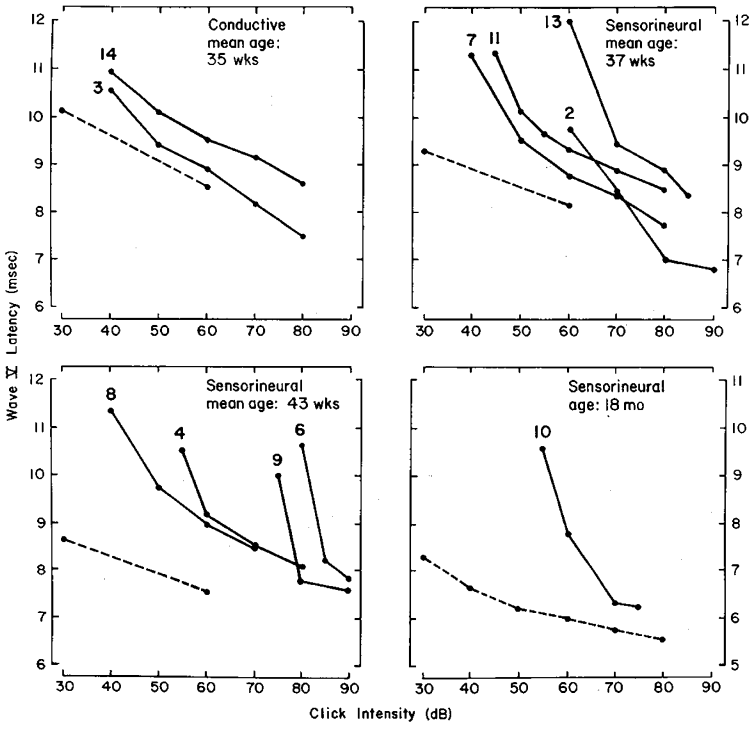

Fig. 3. Latency-intensity curves for 11 of the infants with hearing loss. Normal curve for the age indicated is shown by dashed line. Number on tracing identifies baby in Tables II and III. Left and right ear losses were approximately equal in all cases; curves give mean values for the 2 ears.

TABLE I

CLINICAL DATA		TESTED BY ABR				RISK FACTOR?
		TOTAL	NORMAL	ABNORMAL AUDIO-LOGICAL[1]	ABNORMAL NEURO-LOGICAL	
GESTATIONAL AGE AT BIRTH (WKS)	26-30	23	15	6	2	YES
	31-34	27	25	1(2)		
	35-38	30	26	0(2)		
	39-42	20	18	2		
WEIGHT AT BIRTH (GMS)	< 1000	9	2	6	1	YES
	1000-1500	16	13	1(2)	1	
	1600-2500	44	43	0(1)		
	2600-4000	31	28	1(3)	1	
APGAR SCORES AT 1/5 MINS	<5/6	22	11	9(10)	1	YES
	>6/7	78	75	0(2)	1	
POST-NATAL BLOOD pH	>7.26	83	79	0(3)	1	
	<7.25	17	7	9	1	YES
OTOTOXIC DRUGS	NO	35	31	0(2)	2	
	YES	65	54	11		
NEUROLOGICAL STATUS	BRAIN HAEM	8	3	3	2	YES
	DEEP COMA	10	4	6		YES
	NORMAL	82	79	2(3)		
SEIZURES	1-5 DAYS	9	6	1	2	PERHAPS
	STATUS EPIL	2	0	2		PERHAPS
	NONE	89	80	6(9)		
RESPIRATORY DISTRESS SYNDROME-DAYS ON VENTILATION	<1	27	25	0(2)		
	1-10	63	59	2(3)	1	
	>11	10	2	7	1	YES
CONGENITAL MALFORMATION	CARDIAC	9	3	5	1	YES
	FACE	7	4	3		YES
	TRIS-18	1	0	1		PERHAPS
	NONE	83	79	3	1	

[1] Number in parenthesis adds infants with craniofacial malformation.

TABLE II

Risk factors (left column) associated with abnormal ABRs for the 14 infants identified in this study

RISK FACTOR	ABNORMAL ABR													
	AUDIOLOGICAL												NEURO	
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
VERY YOUNG						X	X	X	X	X	X	X	X	X
LOW WEIGHT						X	X	X	X	X	X	X	X	X
ASPHYXIA			X	X	X	X	X	X	X	X	X	X		X
ACIDOSIS				X	X	X	X	X	X	X	X	X		X
HEMORRHAGE							X			X	X		X	X
COMA				X	X	X		X	X			X	X	
GENES: HEART								X	X	X	X	X		X
GENES: FACE	X	X	X											
RDS							X	X	X	X	X	X		
TOTALS	1	1	2	3	3	5	6	7	7	7	7	7	4	6

TABLE III

Blood pH measurements on the low-Apgar babies in the present study.

HEARING NORMAL BY ABR				HEARING LOSS BY ABR				
	pH Measured					pH Measured		
Apgar (1/5mins)	First[a]	7.21-[b] 7.25	below[b] 7.21	Baby Number	Apgar	First[a]	7.21[b] 7.25	below[b] 7.21
1/3	+	+	0	3	3/5	+	+	+
1/3	0	0	0	4	1/3	+	0	0
1/5	+	0	0	5[c]	0/2	+		
2/6	+	0	0	6	2/5	+	+	+
2/6	0	0	0	7	0/0	+	+	+
2/9	+	+	0	8	5/6	0	+	+
3/4	+	0	0	9	2/5	0	+	+
3/5	0	+	0	10	2/3		+	+
3/6	0	0	0	11	1/3	0	+	+
2/6	+	0	0	12[c]	2/5			
2/7	+	0	0	14	2/5	0	+	+

a. If blood pH was recorded within an hour or two of birth, and if below 7.25, + is entered; if above 7.25, 0.

b. If chart records 2 or more instances of the pH indicated on 2 or more treatment days, + is entered.

c. Baby died within a few days.

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