



Title	ELECTROCARDIOGRAPHIC FINDINGS DURING PARTURITION AND BLOOD GAS TENSIONS IMMEDIATELY AFTER BIRTH IN THOROUGHBRED FOALS
Author(s)	YAMAMOTO, Keiji; YASUDA, Jun; TOO, Kimehiko
Citation	Japanese Journal of Veterinary Research, 39(2-4), 143-157
Issue Date	1991-12-26
DOI	10.14943/jjvr.39.2-4.143
Doc URL	http://hdl.handle.net/2115/8938
Type	bulletin (article)
File Information	KJ00002377522.pdf



[Instructions for use](#)

ELECTROCARDIOGRAPHIC FINDINGS DURING PARTURITION AND BLOOD GAS TENSIONS IMMEDIATELY AFTER BIRTH IN THOROUGHBRED FOALS.

Keiji YAMAMOTO, Jun YASUDA and Kimehiko TOO

(Accepted for publication: Nov. 12, 1991)

SUMMARY

In 101 newborn Thoroughbred foals and foaling mares, 45 fetal (FECGs) and 101 neonatal electrocardiograms (NECGs) were obtained to investigate neonatal arrhythmias and other parameters including changes in fetal (FHR) and neonatal heart rate (NHR). Moreover, umbilical arterial, venous and jugular venous blood gas tensions and pH immediately after birth were analyzed to compare with the type and the degree of neonatal arrhythmias. Before delivery, in 37 fetuses FHR gradually decreased while in 8 cases it increased after rupture of the chorio-allantois. Abnormal deliveries were related in 5 of the 8 cases. In foals born with sinus rhythm, NHR at birth was 22 beats per minute higher than the FHR immediately before birth, gradually decreased for 1-2 min and then increased. Various types of neonatal arrhythmias and their combinations were recorded in 92 of the 101 NECGs. No arrhythmias demonstrated in NECGs were detected in the FECGs.

The mean values for umbilical arterial pH, P_{CO_2} and P_{O_2} indicated that newborn foals at birth were exposed to hypoxemic, hypercapnic and acidemic conditions. The relationship between the umbilical arterial P_{O_2} value as an indicator of the degree of hypoxemia of the foal and the type of arrhythmias was not apparent. However, the P_{O_2} value in the group with severe arrhythmias was comparatively lower than that in the group with mild arrhythmias. It was suggested that hypoxemia played an important role in neonatal arrhythmias.

Key words: Arrhythmia, Blood gas, Equine, newborn, Parturition.

INTRODUCTION

In healthy foals during the adaptive period to extra-uterine life, changes in the heart rate and electrocardiographic appearance [19, 23], blood gas tensions [22, 24] and respiratory function [25] have been reported. Recently, the occurrence of many

Veterinary Hospital, Faculty of Veterinary Medicine, Hokkaido University, Sapporo 060, Japan

types of arrhythmias in Thoroughbred newborn foals has been reported [30]. In that paper, the incidence, duration and electrocardiographic features of arrhythmias, and some aspects of postnatal conditions affecting foals are described. However, the patterns of appearance of these arrhythmias, and their relationship to other findings recorded during the adaptive period has not yet been clarified.

In this study, fetal and neonatal electrocardiograms were recorded to assess the heart rate during parturition as well as the incidence, duration and frequency of neonatal arrhythmias in additional cases. Furthermore, umbilical arterial, umbilical venous and jugular venous blood gas tensions were measured immediately after birth as an indication of the degree of hypoxemia in the newborn foals to compare with the type and degree of neonatal arrhythmias.

MATERIALS AND METHODS

Electrocardiograms

One hundred and one newborn Thoroughbred foals and their mares (including 49 cases described previously [30] and 52 additional cases) were available for study at a racehorse stud in Hokkaido. Forty-five fetal electrocardiograms (FECGs, including 19 cases described in a previous report [30]) were obtained from mares during the first stage of labor as determined by the increase in skin temperature, to the time immediately before birth. Neonatal electrocardiograms (NECGs) were recorded from 101 foals, including the 45 cases from which FECG recordings had been taken. The recording techniques for both FECG and NECG are described in previous literature [30]. From these FECG and NECG tracings, the change in heart rate (HR) during the adaptive period, and the presence, duration, frequency and appearing pattern of arrhythmias in 101 foals (49 cases described and 52 additional cases) before and after birth were investigated.

Blood gas tensions

Following the setting of ECG electrodes immediately after birth, umbilical arterial blood was obtained as soon as possible, mostly within 20 sec, and then an umbilical venous sample was also obtained, mostly within 30 sec. The jugular venous samples were collected after the usual management of the foal had taken place, such as cutting of umbilical cord, so that the collection time of samples varied with the individual. For the collection of blood, a disposable syringe treated with heparin-lithium and 22 gauge disposable needle with the protector for sealing (PZ-D0322, Terumo Co. Tokyo) was used. The majority of samples were analyzed immediately, others were stored at 4 °C for 0.5–1 hr when necessary. pH, Pco₂ and Po₂ measurements were all made at 37 °C with standard pH/Blood gas analyzer equipment (IL System 1306, Allied Instr. Lab. Italy).

RESULTS

Fetal heart rate (FHR) and fetal arrhythmia

Fetal electrocardiograms could be recorded until just prior to birth, however, signal interference caused by the movement of the maternal abdominal muscles associated with strong and continuous labor prevented recording during the last few minutes of fetal life. At that time, gentle traction was applied to the fore-legs of foals to facilitate passage of the shoulders through the birth canal.

Instantaneous FHR was calculated on FECG tracings at 1 min intervals and the mean FHR change for 45 cases is shown in Fig. 1. The mean FHR gradually decreased before rupture of the chorio-allantois. After rupture of the chorio-allantois, 37 cases revealed either a further decrease or maintaining of the FHR (Fig. 2) whereas 8 cases showed an increase in FHR. One of these 8 showed sinus tachycardia (Fig. 3) for 20–30 sec continuation immediately before birth. Abnormal deliveries were observed in 5 of the 8 cases. Momentary fetal arrhythmias were detected in 6 cases (2: atrial extrasystole, 4: sinus arrhythmia). No relations were confirmed between fetal arrhythmias and NECG findings.

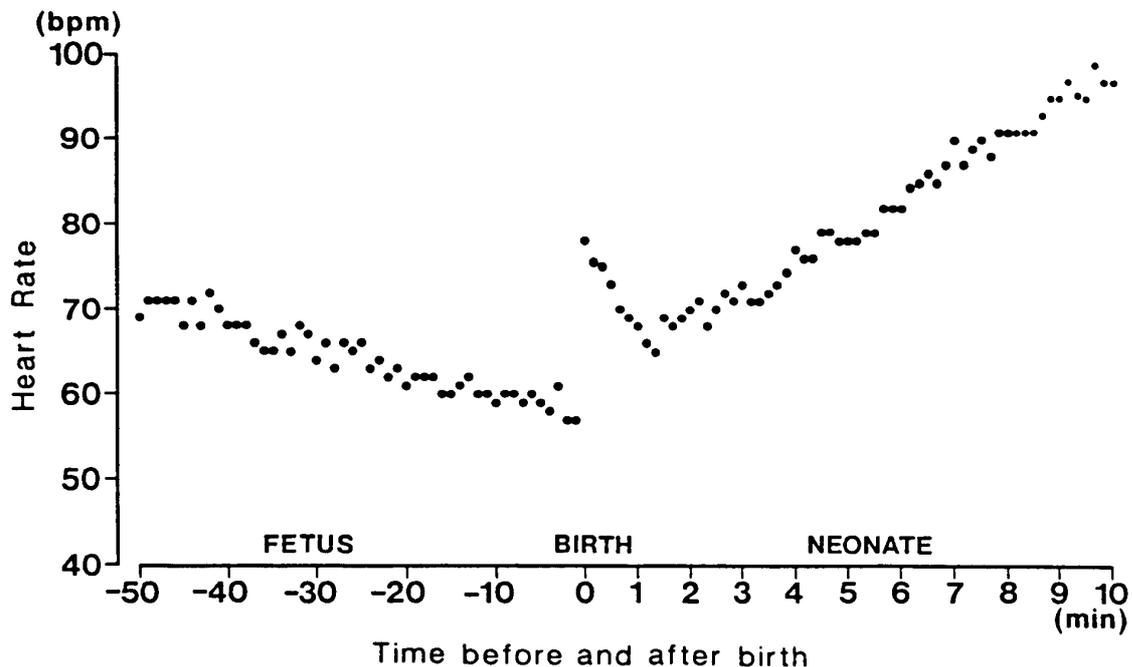


Fig. 1 Changes in mean fetal (FHR) and neonatal heart rate (NHR) during the entire course of parturition. The mean FHR of 45 cases gradually decreased before delivery. The mean NHR of foals born with sinus rhythm decreased for the first 1–2 min, and then increased.

bpm=beats per minute.

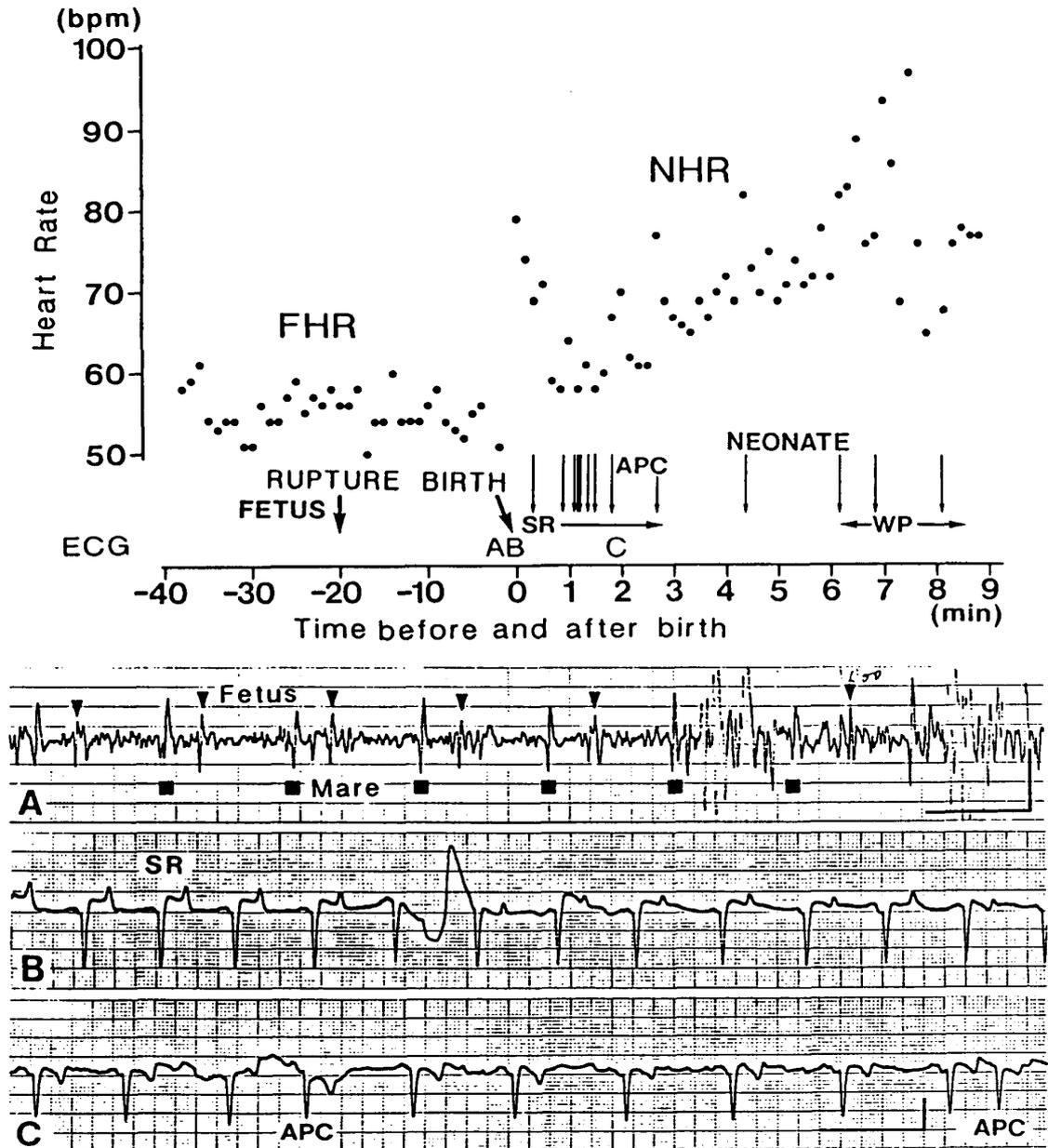


Fig. 2 Changes in FHR and NHR, pattern of arrhythmias and ECGs obtained from the case showing atrial premature contractions (APCs) after birth. Two minutes before birth (ECG A), FHR decreased to 52 beats per minute (bpm), and signal interference caused by maternal abdominal movement was recorded. At birth (ECG B), sinus rhythm (SR) was detected in ECG and the NHR was 78 bpm. After birth, APCs (vertical arrow) were recorded with high frequency (ECG C, 2 min after birth). In this case, wandering pacemaker (WP) was also observed.

Horizontal arrows indicate the maintaining the labelled rhythm.

Neonatal heart rate (NHR) and neonatal arrhythmias

Ninety two of 101 newborns demonstrated various types of arrhythmias and/or their combinations (Figs. 2-5). The patterns of arrhythmias are summarized in Tab. 1. At the beginning of recordings (mostly within 10 sec after birth), 71 showed a

Table 1 The pattern of appearance of neonatal arrhythmias and the number of foals

At the beginning of recording	Arrhythmias appearing on NECG tracings	No. of foals
SR		3
SR →	SA (WP) →	SR 6
SR →	APC →	SR 10
SR →	APC+SA (WP) →	SR 25
SR →	APC+AVB →	SR 1
SR →	APC+AVB+SA (WP) →	SR 6
SR →	CAW+APC+AVB+SA →	SR 3
CAW →	APC+AVB →	SR 1
SR →	AT+APC →	SR 1
SR →	AF →	SR 1
SR →	APC → AF →	SR 1
AT →	APC+SA (WP) →	SR 1
AF →	→	SR 12
AF →	SA (WP) →	SR 6
AF →	AVB+SA (WP) →	SR 1
AF →	APC →	SR 3
AF →	APC+SA (WP) →	SR 2
SR →	VPC+APC →	SR 1
SR →	VPC+APC+SA (WP) →	SR 5
SR →	VPC+APC+AVB →	SR 1
SR →	VPC+APC+AVB+SA (WP) →	SR 2
SR →	VPC+AT+APC →	SR 1
SR →	VPC+AT+APC+SA (WP) →	SR 1
AF+VPC →	SA (WP) →	SR 1
SR →	VT+VPC+APC+AVB+SA (WP) →	SR 1
SR →	VT+VPC+APC+SA (WP) →	SR 2
VT →	VPC+AT+SA (WP) →	SR 1
VT+AF+VPC →	→	SR 1
VT+AF+VPC →	SA (WP) →	SR 1
Total		101

SR : sinus rhythm, SA : sinus arrhythmia, WP : wandering pacemaker, APC : atrial premature contraction, AVB : atrioventricular block, CAW : continuous atrial waves, AT : atrial tachycardia, AF : atrial fibrillation, VPC : ventricular premature contraction and VT : ventricular tachycardia.

rhythm of sinus node origin. Following this, atrial premature contraction (APC), ventricular premature contraction (VPC), sinus arrhythmia (SA) including wandering pacemaker (WP) and atrioventricular block (AVB) appeared with no regularity (Fig. 2), and in a few cases, atrial tachycardia (AT), atrial fibrillation (AF) and ventricular tachycardia (VT) consequently occurred (Fig. 3). The other 30 foals demonstrated

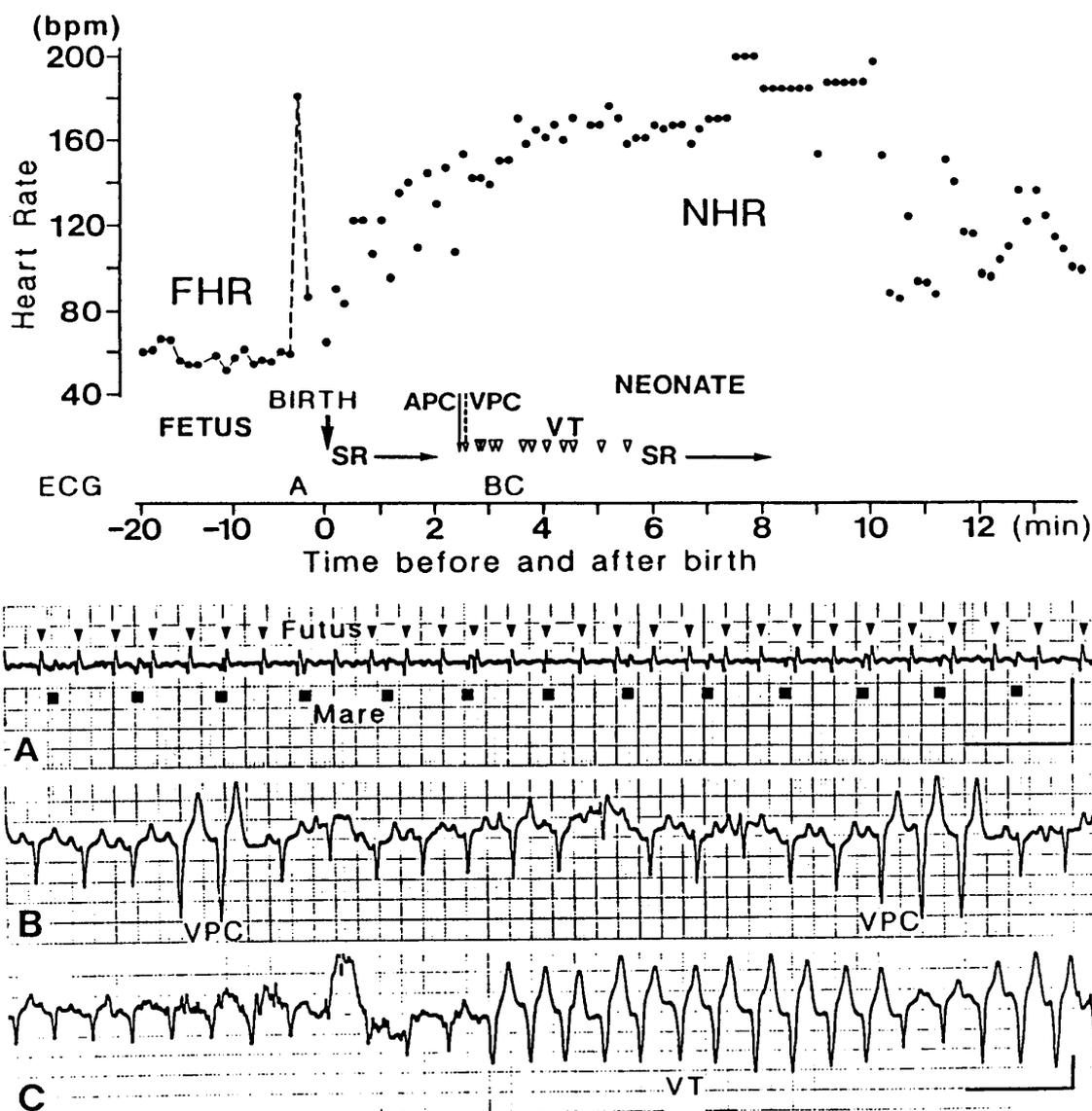


Fig. 3 Changes in FHR, NHR, pattern of arrhythmias and electrocardiograms (ECGs) from the case born in dystocia. Two minutes before birth (ECG A), paroxysmal fetal tachycardia (180 bpm, arrow heads) was recorded in FECG when the mare tried to deliver the foal while standing. Three minutes after birth (ECG B), ventricular premature contractions (VPCs, broken arrows) appeared and consequently, ventricular tachycardia (VT) occurred for 30 sec (ECG C).

ectopic rhythms at birth (25: AF, 1: AT, 2: VT+AF, 1: VT, 1: continuous atrial waves without QRS complexes). Atrial fibrillation was observed in 29 foals, 27 cases of which started before the beginning of NECG recordings, while in 2 cases the onset of AF occurred during the NECG recording (Fig. 4). Ventricular tachycardia was

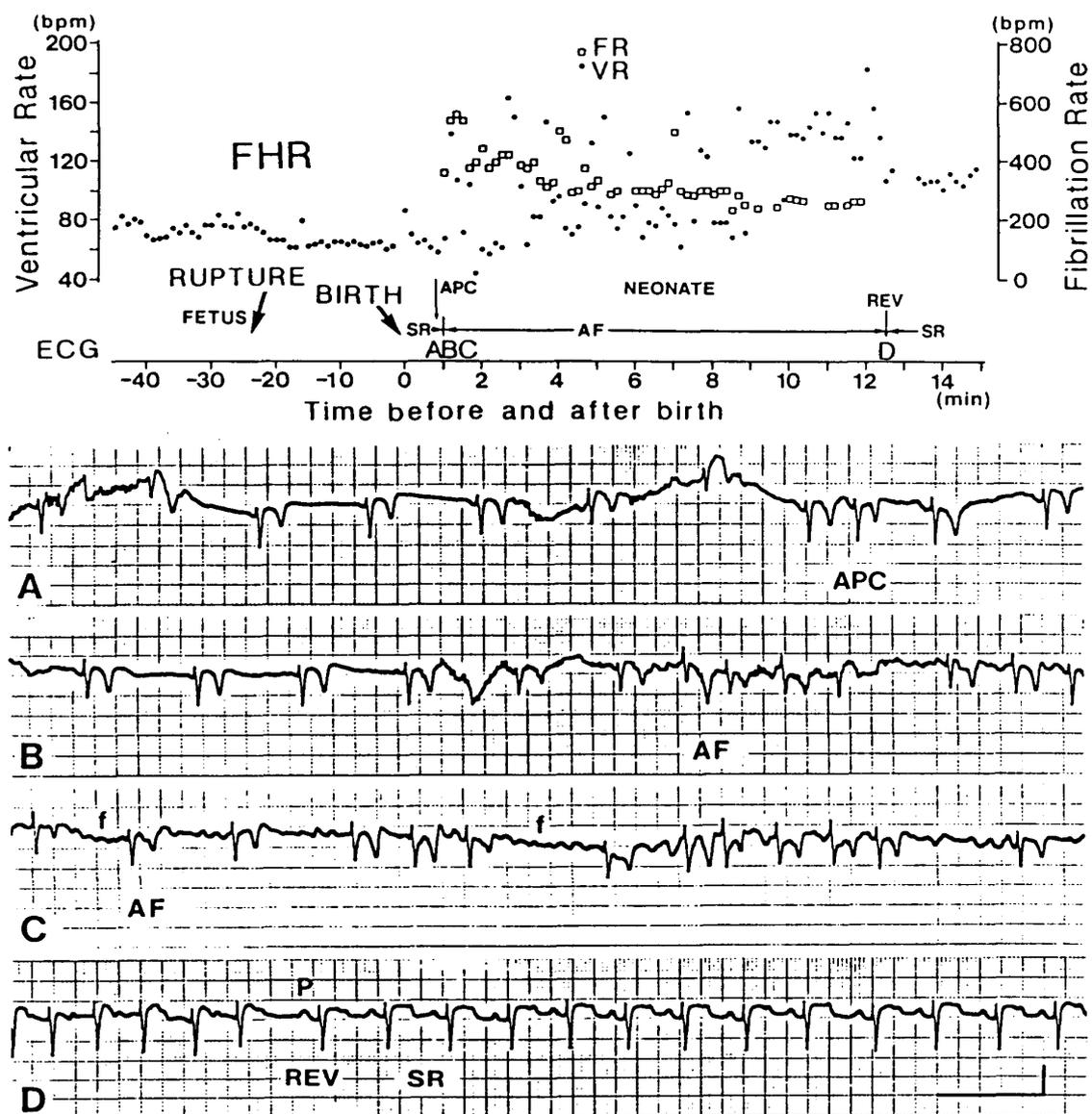


Fig. 4 Changes in FHR, neonatal ventricular rate (VR), neonatal fibrillation rate (FR), pattern of arrhythmias and ECG recordings during parturition from the case showing atrial fibrillation (AF) after birth. At birth, SR was maintained, and APC appeared 40 sec after birth (ECG A). The shift from SR to AF was recorded (ECG B) at 50 sec after birth and consequently, fibrillation waves (f) were observed (ECG C). Spontaneous reversion to SR was observed at 12.5 min after birth (ECG D).

recorded in 6 cases, 2 of which demonstrated AF simultaneously (Fig. 5). All these arrhythmias disappeared spontaneously with the passage of time and sinus rhythm (SR) was maintained.

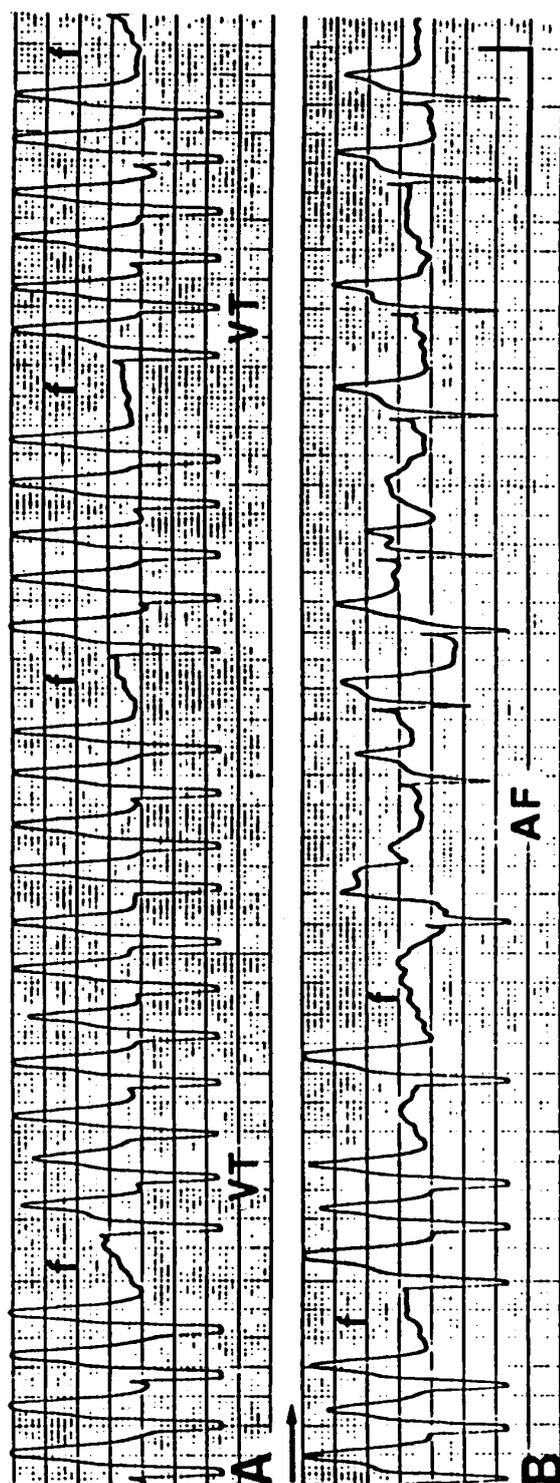


Fig. 5 ECGs obtained from the case showing AF and VT simultaneously at birth. Fibrillation waves (f) were recorded between the two waves of VT (ECG A). After the disappearance of VT, AF was detected in ECG recording (ECG B).

In the foal that revealed SR at the beginning of the NECG recording, the mean NHR gradually decreased for 1–2 min after birth then gradually increased (Figs. 1, 2). Ventricular rate of foals showing AF was unstable with the range from 50 to 200 beats per minute (bpm) during AF, although the mean of this gradually increased. Fibrillation rate showed a tendency to decrease before reversion (Fig. 4).

The incidence and duration of arrhythmias, and the frequency of extrasystoles are summarized in Tab. 2. The method for calculation of the duration of the arrhythmia has been described previously [30]. Atrial premature contraction was the most common arrhythmia in the newborns, although the range of duration and frequency varied. The duration of AF was mostly less than 10 min although in 2 cases the AF continued for over 1 hr after birth.

Table 2 The type, duration and frequency of arrhythmias

Type of arrhythmia	No. of foals*	Duration of Arrhythmia	Frequency of extrasystoles**
		Mean \pm SD, (Range)	Mean \pm SD, (Range)
SA	64		
AVB	15		
APC	68	287 \pm 209.1 (1–800)	7 \pm 12.2 (1–83)
AT	4	3 \pm 1.1 (2–5)	
AF	29	424 \pm 253.2 (47–1148) #	
VPC	18	114 \pm 132.5 (2–540)	3 \pm 2.0 (1–6)
VT	6	242 \pm 153.7 (83–540)	
CAW	4		

* Total of 101 foals were investigated.

Cases of APC and VPC include AT and VT, respectively.

** Foals with AT, AF, VT and CAW were neglected for the calculation of the frequency of extrasystoles.

This data excluded 2 cases of AF with durations of over 1 hr (5000 and 7800 sec, respectively).

Blood gas tensions immediately after birth

Sixty one umbilical arterial, 58 vmbilical venous and 75 jugular venous samples were collected from the 101 newborn foals. There were significant variations between the collection time for samples and onset of breathing of the individual foals. The values measured are shown in Tab. 3. The P_{O_2} values for umbilical arterial and venous blood were 27.0 ± 5.16 and 35.7 ± 5.87 (Mean \pm SD) mmHg, respectively. To compare the P_{O_2} , P_{CO_2} and pH values of these samples with the type of neonatal arrhythmias, foals were divided into 4 groups as follows;

Table 3 Umbilical arterial, venous and jugular venous pH, Pco₂, Po₂ values of newborn foals

Samples	pH	Pco ₂	Po ₂
	Mean±SD [Range]	Mean±SD [Range] (mmHg)	Mean±SD [Range] (mmHg)
Umbilical artery (n=61)	7.312±0.0468 [7.185–7.442]	62.0±6.99 [44.8–81.4]	27.0±5.16 [17–44]
Umbilical vein (n=58)	7.382±0.0349 [7.194–7.438]	51.2±4.29 [45.1–73.8]	35.7±5.87 [24–53]
Jugular vein (n=75)	7.238±0.0565 [6.910–7.378]	69.9±8.40 [52.7–114.2]	29.4±5.60 [15–42]

group 1: no arrhythmia without SA.

group 2: atrial ectopic beats without AF.

group 3: AF without ventricular ectopic beats.

group 4: ventricular ectopic beats.

On the basis of the duration and the frequency of extrasystoles, group 2 was further divided into 2 sub-classes.

group 2-A: APC was recorded for less than 5 beats or less than 3 min.

group 2-B: 5 or more APCs continued for over 3 min.

Umbilical arterial pH, Pco₂ and Po₂ values for each group are shown in Tab. 4. The Po₂ level of group 1 (22.5±3.20mmHg) was the lowest, followed by group 2-B (25.1±5.01mmHg), group 4 (26.8±4.65mmHg), group 2-A (27.4±3.60mmHg) and

Table 4 Umbilical arterial blood gas tensions and pH immediately after birth for divided groups.

Group	pH	Pco ₂	Po ₂
	Mean±SD	Mean±SD (mmHg)	Mean±SD (mmHg)
1 (n=4)	7.310±0.0277	64.2±7.33	22.5±3.20
2-A (n=17)	7.296±0.0424	62.6±6.43	27.4±3.60
2-B (n=13)	7.326±0.0579	61.1±7.96	25.1±5.01
3 (n=16)	7.323±0.0376	60.7±5.60	29.3±6.04
4 (n=11)	7.304±0.0473	63.1±7.74	26.8±4.65

group 3 (29.3 ± 6.04 mmHg). There are no statistically significant differences between any two types of arrhythmia. P_{O_2} levels of group 2-B which is the more severe class of group 2 had a tendency to be lower than those of group 2-A, although no statistically significant difference was evident.

General observation of mares and foals

Before delivery, abnormally thick chorio-allantois with red membranes appeared at the maternal vulval lips in 6 cases, all of which being subject to artificial rupture and management to hasten the delivery. In 4 of the 6 cases, FHR decreased to 45–60 bpm and then increased after artificial rupture. After birth, these 6 cases from abnormal deliveries showed comparatively severe arrhythmias (3 cases: AF, 2: severe APC and 1: VPC). In one case in particular, neonatal AF with a duration of over 1 hr was detected electrocardiographically, during which this foal was severely depressed and could not stand. No blood samples were obtained from this case.

Two other mares delivered foals while in the standing position, one of which showed an increase in FHR to the level of 180 bpm (fetal tachycardia, Fig. 3) while in the other, no specific pattern was detected. After birth, VT was recorded in the former (Fig. 3). Eighteen mmHg of jugular venous P_{O_2} , which is the lowest value of all cases, was measured in this case, but no data concerning blood gas tensions from the umbilical cord were obtained.

In the other mares normal deliveries were observed. All neonates except 1 foal which was destroyed for other reasons grew normally.

DISCUSSION

Cardiac arrhythmias are common in adult horses accompanying various diseases [9, 10, 12, 27, 29] or pathological heart lesions [7,8,18]. In the racehorse, exercise-induced arrhythmias [13, 26] probably related to cardiac hypoxia are also known, i. e. AF, extrasystoles. There is, however, little literature concerning arrhythmias in foals or neonates [20, 21, 23]. Our previous report suggested that in Thoroughbred newborns, many types of arrhythmias are present during the adaptive period.

In this study, including additional cases, the incidence and duration of neonatal arrhythmias were similar to the previous study [30]. The duration of AF was longer in this study and in 2 cases AF continued for over 1 hr, one of which was depressed and took a long time to stand for the first time. This case is similar to that reported by Machida et al [20], however, the other one looked normal and stood during AF.

The starting period for neonatal arrhythmias, especially AF, was 1–2 min before birth. At that time neither FECG nor NECG recordings were available. During this period, the head and cord of the fetus may be temporarily compressed resulting in the development of significant hypoxemia and acidemia [4]. Immediately after birth, umbilical arterial pH and P_{O_2} measurements were evidently lower and P_{CO_2} was higher than those of the pony fetus during late pregnancy [2, 3]. These findings suggest

that foals at birth are exposed to hypoxemic, hypercapnic and acidemic conditions.

The relationship between umbilical arterial P_{O_2} which is thought to indicate the degree of hypoxemia in newborns and the type of neonatal arrhythmia is not clear from the present study. However, there were two problems in interpretation of these results, one being the difference between the time of collecting samples and onset of breathing, and another being that in some of the cases born in dystocia which showed VPCs or VT after birth no samples could be taken from umbilical cord because it had been broken during delivery. Concerning the degree of arrhythmia, the mean P_{O_2} value in the group with severe arrhythmias was comparatively lower than that in the group with slight arrhythmias. Furthermore, one case with VT after birth demonstrated significantly low P_{O_2} and high P_{CO_2} values in the jugular venous sample. From these findings, the possibility that hypoxia played an important role in both the type and degree of neonatal arrhythmias was suggested.

In the present study of FHR, 37 of the 45 cases showed a gradual decrease in FHR during the first stage of labor and either a further decrease or a maintaining the same level of HR during the second stage. This pattern is similar to the finding reported by Too et al. [28] and is probably the common pattern in normal parturition of the horse. However, it was found in 8 cases that FHR progressively increased during the second stage of labor (including 1 case of fetal tachycardia immediately before delivery). Abnormal delivery conditions were related in 5 out of the 8 cases. In 4 cases the abnormally thick chorio-allantois with red membranes was forced through the maternal vagina, and in 1 case a mare delivered a foal while in the standing position, the foal in these cases demonstrating comparatively severe arrhythmia after birth (1: VT, 1: VPC, 2: AF, 1: severe APC). From these observations, a progressive increase of FHR including fetal tachycardia corresponding to abnormal deliveries might have been the most serious finding in the foal during the adaptive period. On the other hand, there is no evidence in this study that fetal arrhythmias occur during abnormal pregnancies.

It is generally accepted that NHR gradually increases after birth [20, 23]. Neonatal heart rate, in this study, gradually decreased for 1–2 min after birth and then increased. The mean NHR of the case born with SR at the beginning of the recording was 22 bpm higher than the mean FHR of 45 cases immediately before birth. The mechanism of the sudden rise in heart rate at birth is not clear. Before birth, compression of the fetal body and the cord due to passing through the birth canal, and continuous uterine contraction resulted in complete or partial occlusion of the cord, rise in carotid arterial pressure and gradual fall of fetal P_{aO_2} [4–6]. Many experimental works indicated that these factors could induce reflex bradycardia mediated by chemoreceptors and baroreceptors [1, 11, 15–17, 31]. Immediately after birth, foals were exposed to hypoxemic, hypercapnic and acidemic conditions (see above), although the compression of the body and the cord was released. It is known that in

the rhesus monkey, either hypoxemia or hypercapnia induces the slowing of FHR [14]. Considering these findings, the NHR decrease immediately after birth might be due to the hypoxemic, hypercapnic and acidemic condition of neonates. After the onset of breathing, the rapid improvement of conditions and the effort to stand makes NHR increase.

Neonatal arrhythmias were considered as physiological processes in newborn foals and thought to have close relations to hypoxemia, high vagal tone and the extension of atrial muscles corresponding to acute change in hemodynamics [30].

In this study, it was confirmed that foals at birth were exposed to hypoxemic, hypercapnic and acidemic conditions. However, the relationship between the degree of hypoxemia and the type of arrhythmia is not clear because of the limitations of the clinical study. To clarify the relationship between these factors and neonatal arrhythmias, further investigation including experimental studies should be performed. Moreover, the occurrence of these arrhythmias during the adaptive period in other species should be investigated.

ACKNOWLEDGMENT

The authors are grateful to Shadai Farm for their donation of the materials. We also wish to thank the veterinarians of Shadai Farm, Drs. N. Tsunoda and H. Akita for their valuable advice.

REFERENCES

- 1) ASSALI, N. S., HOLM, L. W. & SEHGAL, N. (1962): Hemodynamic changes in fetal lamb in utero in response to asphyxia, hypoxia, and hypercapnia. *Circ. Res.*, **11**, 423-430.
- 2) COMLINE, R. S. & SILVER, M. (1970): P_{O_2} , P_{CO_2} and pH levels in the umbilical and uterine blood of the mare and ewe. *J. Physiol.*, **209**, 587-608.
- 3) COMLINE, R. S. & SILVER, M. (1974): A comparative study of blood gas tensions, oxygen affinity and red cell 2, 3 DPG concentrations in fetal and maternal blood in the mare, cow and sow. *J. Physiol.*, **242**, 805-826.
- 4) COMLINE, R. S. & SILVER, M. (1975): Placental transfer of blood gases. *Br. Med. Bull.*, **31**, 25-31.
- 5) DAWES, G. S. (1961): Changes in the circulation at birth. *Br. Med. Bull.*, **17**, 148-153.
- 6) DAWES, G. S., JACOBSON, H. N., MOTT, J. C. & SHELLEY, H. J. (1960): Some observations on foetal and new-born rhesus monkeys. *J. Physiol.*, **152**, 271-298.
- 7) ELSE, R. W. & HOLMES, J. R. (1971): Pathological changes in atrial fibrillation in the horse. *Equine Vet. J.*, **3**, 56-64.
- 8) FISHER, E. W., PIRIE, H. M. & ANDREW, H. (1970): Clinical-pathological correlation of an equine cardiac arrhythmia. *Vet. Rec.*, **86**, 499-502.

- 9) GLAZIER, D. B. & DUKES, H. H. (1963): Atrial premature beats in a horse. *Irish Vet. J.*, **17**, 87-88.
- 10) GLAZIER, D. B. & NICHOLSON, J. A. (1959): Premature ventricular beats. *Irish Vet. J.*, **13**, 82-86.
- 11) HARRIS, J. L., KRUEGER, T. R. & PARER, J. T. (1982): Mechanisms of late decelerations of the fetal heart rate during hypoxia. *Am. J. Obstet. Gynecol.*, **144**, 491-496.
- 12) HILWIG, R. W. (1977): Cardiac arrhythmias in the horse. *J. Am. Vet. Med. Assoc.*, **170**, 153-163.
- 13) HOLMES, J. R. & ALPS, B. J. (1966): The effect of exercise on rhythm irregularities in the horse. *Vet. Rec.*, **78**, 672-683.
- 14) IKENOUE, T., MARTIN, Jr. C. B., MURATA, Y., ETTINGER, B. B. & LU, P. S. (1981): Effect of acute hypoxemia and respiratory acidosis on the fetal heart rate in monkeys. *Am. J. Obstet. Gynecol.*, **141**, 797-810.
- 15) ITSKOVITZ, J., GOETZMAN, B. W. & RUDOLPH, A. M. (1982): The mechanism of late deceleration of the heart rate and its relationship to oxygenation in normoxemic and chronically hypoxemic fetal lambs. *Am. J. Obstet. Gynecol.*, **142**, 66-73.
- 16) ITSKOVITZ, J., LAGAMMA, E. F. & RUDOLPH, A. M. (1983): Heart rate and blood pressure responses to umbilical cord compression in fetal lambs with special reference to the mechanism of variable deceleration. *Am. J. Obstet. Gynecol.*, **147**, 451-457.
- 17) JAMES, L. S., YEH, M., MORISHIMA, H. O., DANIEL, S. S., CARITIS, S. N., NIEMANN, W. H. & INDYK, L. (1976): Umbilical vein occlusion and transient acceleration of the fetal heart rate. Experimental observations in subhuman primates. *Am. J. Obstet. Gynecol.*, **126**, 276-283.
- 18) KIRYU, K., AMADA, A., KANEKO, M. & SATOH, H. (1974): Atrial fibrillation in the horse: Clinical and histopathological studies of two cases. II. Formal pathogenesis. *Exp. Rep. Equine Hlth. Lab.*, **11**, 70-86.
- 19) MACHIDA, N., YASUDA, J. & TOO, K. (1987): Auscultatory and phonocardiographic studies on the cardiovascular system of the newborn Thoroughbred foal. *Jpn. J. Vet. Res.*, **35**, 235-250.
- 20) MACHIDA, N., YASUDA, J. & TOO, K. (1989): Three cases of paroxysmal atrial fibrillation in the Thoroughbred newborn foal. *Equine Vet. J.*, **21**, 66-68.
- 21) MATSUI, K., AMADA, A. & SAWAZAKI, H. (1985): Second-degree atrioventricular block observed in a Thoroughbred foal at 2.5 months of age. *Jpn. J. Vet. Sci.*, **47**, 175-178.
- 22) ROSE, R. J., ROSSDALE, P. D. & LEADON, D. P. (1982): Blood gas and acid-base status in spontaneously delivered, term induced and induced premature foals. *J. Reprod. Fert., Suppl.*, **32**, 521-528.
- 23) ROSSDALE, P. D. (1967): Clinical studies on the newborn Thoroughbred foal. II. Heart rate, auscultation and electrocardiogram. *Br. Vet. J.*, **123**, 521-532.
- 24) ROSSDALE, P. D. (1968): Blood gas tensions and pH values in the normal Thoroughbred foal at birth and in the following 42h. *Biol. Neonate.*, **13**, 18-25.
- 25) ROSSDALE, P. D. (1969): Measurements of pulmonary ventilation in normal newborn Thoroughbred foals during the first three days of life. *Br. Vet. J.*, **125**, 157-162.

- 26) SENTA, T., SMETZER, D. L. & SMITH, C. R. (1970): Effects of exercise on certain electrocardiographic parameters and cardiac arrhythmias in the horse. A radiotelemetric study. *Cornell Vet.*, **60**, 552-569.
- 27) TOO, K. (1960): Abnormal electrocardiograms in the horse. *Jpn. J. Vet. Res.*, **8**, 29-34.
- 28) TOO, K., KANAGAWA, H. & KAWATA, K. (1967): Fetal and maternal electrocardiograms during parturition in a mare. *Jpn. J. Vet. Res.*, **15**, 5-14.
- 29) VIBE-PETERSEN, G. & NIELSEN, K. (1980): Electrocardiography in the horse (A report of findings in 138 horses). *Nord. Vet. -Med.*, **32**, 105-121.
- 30) YAMAMOTO, K., YASUDA, J. & TOO, K. (1991): Arrhythmias in newborn Thoroughbred foals. *Equine Vet. J.* (in press)
- 31) YEH, M., MORISHIMA, H. O., NIEMANN, W. H. & JAMES, L. S. (1975): Myocardial conduction defects in association with compression of the umbilical cord. Experimental observations on fetal baboons. *Am. J. Obstet. Gynecol.*, **121**, 951-957.