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Effect of forceps compression and traction on the newborn heart as monitored by ECG

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INTRODUCTION.

Obstetric forceps have been in use since the early 1600's having been first described by the Chamberlen family.[1] However, no extensive data was published on the effect of forceps until 1861 when Kristellar attached a spring gauge to the handle of the forceps to measure force used during traction.[2] In 1935 Wylie inserted a spring scale in a Bill axis traction handle to measure traction force exerted during delivery. His measurements were of peak force only. His data indicated that primigravidas and babies of increasing weight required more force.[3] Strain gages were used on the Bill axis traction handle and the De Lee-Simpson forceps by Fleming and associates in 1959. They recorded the entire course of traction and compression during delivery.[4]

One basic reason for the evaluation of the effect of forceps on the fetal and newborn heart stands forth. Theory has long stated that compression and traction by forceps causes a bradycardia in the fetus to a point of almost half the normal fetal heart rate. Since over one million obstetric forceps deliveries occur per year in this country alone,[5] we need to know if this bradycardia is actually caused by the forceps, and if so, by what exact mechanism.

Larks and Longo noted several causes for fetal bradycardia during delivery: pressure on the fetal head, cardiovascular reflexes from the fetal neck or elsewhere, marked fall in maternal blood pressure, twisting or knotting of the umbilical cord, increased intrauterine pressure, and inhalation anesthesia. Their study revealed slowing of the fetal heart rate which they associated with traction on the fetal head. The rate dropped to 72 beats per minute with continued traction, but they noted no morphologic changes in the QRS complex.[6] Kelly, in his studies in 1963, noted that bradycardia occured in 73% of the pulls; this occured as commonly with the vacuum extractor as with the forceps. Tachycardia occured after forceps traction ceased in 25% of deliveries. Tachycardia after vacuum extraction occured slightly more frequently.[7] Prystowsky produced bradycardia by compression manually of the skull of the newborn infant and attributed this to medullary ischemia.[8] Ullery disagrees with this concept as a result of his research. He states, "In all cases definite bradycardia occured with traction. The etiology of this bradycardia is obscure." He noted that the slowing of the fetal heart closely attended the application of the forceps thus making medullary ischemia an unlikely explanation. He suggests rather a reflex neural mechanism in response to forceps traction. The difference during spontaneous and forceps deliveries in average fetal heart rate depression varied so little that no statistical significance could be concluded.[9] This study was conducted in an attempt to resolve the existing confusion by precise measurements of fetal heart variations in newborn infants subjected to quantitated forceps compression and traction.

MATERIALS AND METHODS

The Simpson forceps and Bill axis traction handle were used. The strain gage instrumentation of this equipment has previously been described.[3] The recording of the compression and traction was by a Sanborn twin channel strain gage amplifier and recorder. Traction was recorded upward from the baseline of the upper channel. Calibration was 10 kg. = 1 cm. Compression was recorded downward from the baseline of the lower channel with a calibration of 1 kg. = 1 cm. The paper speed was set so that 1 cm. = 10 secs. (Fig. 1 shows a typical recording of a forceps and compression recording.) The electrocardiograms were recorded on a

Sanborn Cardio-Visette. All instruments were easy to calibrate and were relatively free of drift over long periods of time. Calibration of the forceps has previously been described by Pearse.[5] The instruments were calibrated prior to each series of studies.

In 1964, thirty-nine newborn infants were studied and in 1965, thirty-seven were studied. Age ranged from 30 minutes to 48 hours. All babies were picked at random without regard as to sex, weight, mode of delivery, type of anesthesia, or health status of the mother. All studies were conducted by the same two individuals working together, one of whom is this author. (See Table 1.)

A baseline ECG was run on each infant at rest using Lead I. The series done in 1964 measured only the effect of forceps compression. Eight of the thirty-nine patients were done with the Shute forceps without strain gage instrumentation. The other thirty-one were compressed using the Simpson forceps with measured compression. Increments of 500 g., 1000 g., and 1500 g. were used in occiput anterior and occiput posterior forceps applications. In some instances 2000 g. of compression could be applied. The infants were placed on a flat wooden table on a blanket. They were placed upon their backs for the occiput posterior application and prone for the occiput anterior applications.

The 1965 series measured the normal ECG, ECG with compression alone, and then ECG during compression combined with traction. For twenty infants a false pelvic outlet was constructed by attaching two metal shoulder braces to a wooden table. The shoulder braces had fitted foam rubber pads and were placed nine centimeters apart. The infants were then pulled through this in both the occiput anterior and occiput posterior positions while compression and traction as well as the ECG were monitered. Thus, compression and traction were measured with the outlet offering resistance to the head and then to the shoulders after the head had been pulled through the outlet.

Respiratory rate was measured in ten newborns. A chest spirometer was used to measure the respiratory rate and depth versus increasing increments of compression of 0, 500, 1000, 1500, and 2000 grams.

The average ECG measurement for each segment, interval, or height was determined by measuring ten complexes per maneuver and determining the average per ten complexes. (ie. - 10 QRS complexes per maneuver were measured to determine the average QRS complex duration per maneuver.) Lead I was used for all recordings.

RESULTS.

No significant change was found in the average fetal heart rate with the application of compression in the 1964 series. The rate did not vary directly or indirectly with increasing increments of compression. (See Table II.) The range of variation was from -3 to to +1 beats/min. change. The largest fall in FHR was from 147 to 100 beats/min., but with increasing compressive pressure the rate came back to 125. The highest rate obtained was 185 and the lowest was 97.

Although the rate did not change, there were minor changes within the ECG intervals. (Table III.)

The height of the various waves which represent the electrical activity of the heart was found to increase slightly with compression in some instances. Wave durations are not significantly changed, but longer durations of the cardiac cycle are accounted for by the P and T wave. Usually a shorter T-P interval is seen in a tachycardia but was particularly significant in this series. Duration of the cardiac cycle was increased, also, by a longer S-T segment.

There is, despite no apparent change in rate, some change in cardiac activity. Occasionally there were fairly profound changes in the ECG pattern with no change in rate. (Table IV.)

No difference was noted when the Shute forceps were used as compared to the Simpson forceps. The age range of 30 minutes to 48 hours made no difference in the findings.

A comparison of the studies of 1964 and 1965 for average values is noted in Table V. The average heart rate was similar and no significant deviations were noted. The heart rate in this series also did not decrease with compression. The addition of traction had no effect on the rate nor did the position of the head inside or outside of the constructed "pelvic outlet." (See Fig. 2.) No pattern trends could be noted within the ECG such as variations in intervals, waves, or activity as measured by wave height. (Fig. 3 & 4.) The T-P interval was noted to be non-existent (0) when the rate was increased in this series. No reason is apparent for this.

Twenty-nine babies in the 1965 series had compressive forces of greater than 2000 gms. exerted on the skull. The range was 500 to 7460 grams. This group, when divided into less than and greater than 2000 grams, revealed a decrease in duration of the T and P wave, and QRS complex. But a compensating increase in the P-R interval duration and an increased fetal heart rate was also noted. (Table VI.)

The force exerted by traction ranged from 2.5 kg. to 11 kg. This is much less than that reported initially by Pearse. His recordings averaged about 20 kg.[5] The difference might be accounted for on the basis of ease of passage of the newborn versus the fetus under experimental conditions.

A small series (ten newborns) measuring respiratory rate and depth with skull compression indicated no change. Thus one can initially assume that no compensation or reaction is initiated by the respiratory system. The results of this series verify again the original work by Pearse that compression and traction do not increase together on a proportional basis.[5]

DISCUSSION

An attempt was made to objectively compare two series of newborns. One series used only measured compression with the Simpson forceps and one measured compression with the Shute forceps. The other series measured compression with the Simpson forceps and

and traction with the Bill axis traction handle. The effect on the heart was monitored by the electrocardiogram in both series.

Measured compression caused no significant change in rate, though changes within the ECG occurred, such as an increase in cardiac electrical activity. Shute forceps had no different effects when compared with Simpson forceps in this series though the Shute forceps is theoretically constructed to exert less compression.[10]

Compression and traction combined had no effect on the newborn heart rate, the reverse of what has previously been reported to occur during delivery when these forces are applied.[6,7,8,9] The reason for this difference is not immediately apparent. Respiration did not vary in the newborn when compression and traction was applied, so no compensation occurred as a result of this system. Therefore, hypoxia secondary to compression seems unlikely.[8]

Reynolds reports that the chemoreceptors are inactive until several days after birth. The carotid sinus reflex does not become active until the infant is forty-five days of age. Adrenal sympathetic activity is used early in life only as a last resort mechanism.[11] This leaves the factors of total pressure on the undelivered infant and the fetal-maternal relationship via the umbilical cord as possible contributory sources for the bradycardia reported. Supporting the importance of these other factors, Brady reports that the fetal heart rate often increases or decreases 10 - 25 beats/minute with uterine contraction in the later part of the first stage of labor. This rate change was usually less than forty-eight seconds in duration. Bradycardia in the fetus also occurred with a hypotensive episode in the mother as seen secondary to spinal anesthesia.[12] In conclusion, one must look elsewhere for the cause of bradycardia during delivery rather than to the direct effect of forceps compression and traction.

SUMMARY

1. Forceps compression and traction on the newborn does not cause a bradycardia, whether inside or outside the mechanical "pelvic outlet."
2. Respiratory rate and depth does not vary with forceps compression, and therefore does not serve as a compensatory factor to these forces in the new-born.
3. There were variations within the ECG, without variation of rate, during the application of forceps compression and traction. No consistent

pattern change was noted.

4. Factors not evaluated which may prove to be important in evaluating bradycardia during delivery are total maternal compression on the fetus and the relationship between the mother and fetus via the umbilical cord.

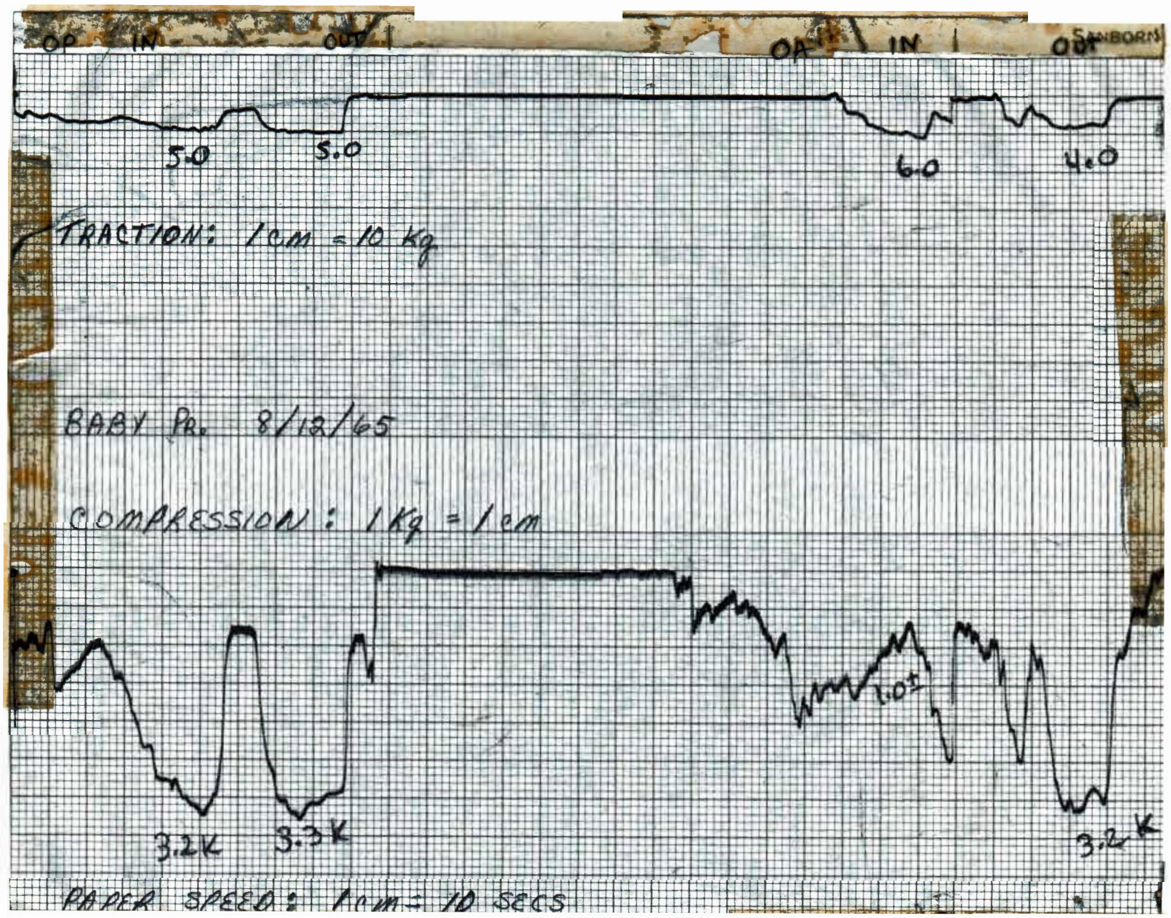

Figure 1. This is a typical recording of forceps compression and traction during an application. The application was done on a newborn who was pulled through the constructed pelvic outlet. "In" and "Out" indicate the position of the head before and after being pulled through the outlet respectively.

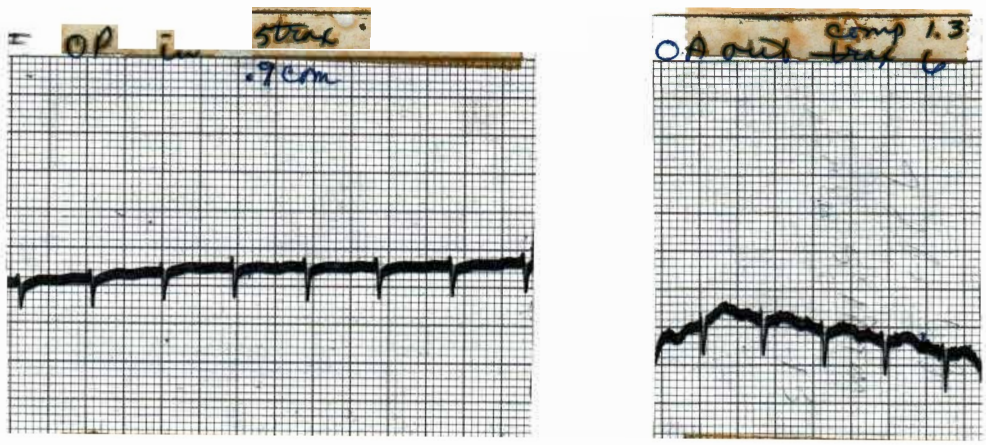

Figure 2. Typical ECG recording with the infants head in and out of the constructed pelvic outlet.

KEY: x = traction force applied outside the constructed pelvic outlet.
o = traction force applied inside the constructed pelvic outlet.

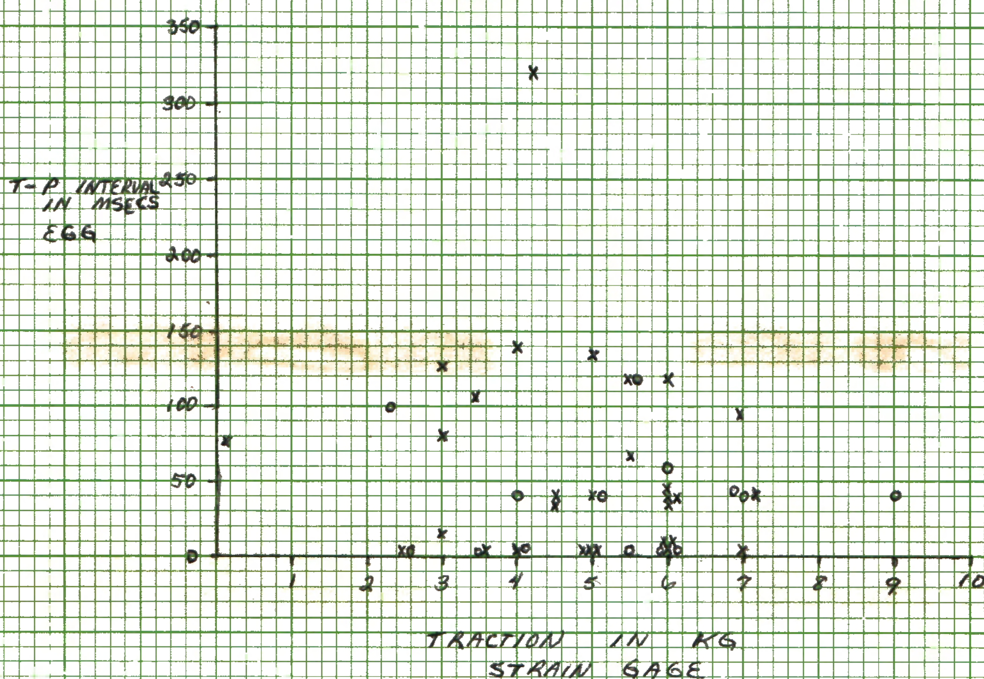

Figure 3. By comparing the T-P interval in msecs against the traction in kg. inside and outside the constructed pelvic outlet, no correlation between increments of applied traction and changes in duration of the T-P interval can be found. The forceps application for this series was occiput anterior.

KEY: x = compression force applied outside the constructed pelvic outlet.
o = compression foree applied inside the constructed pelvic outlet.

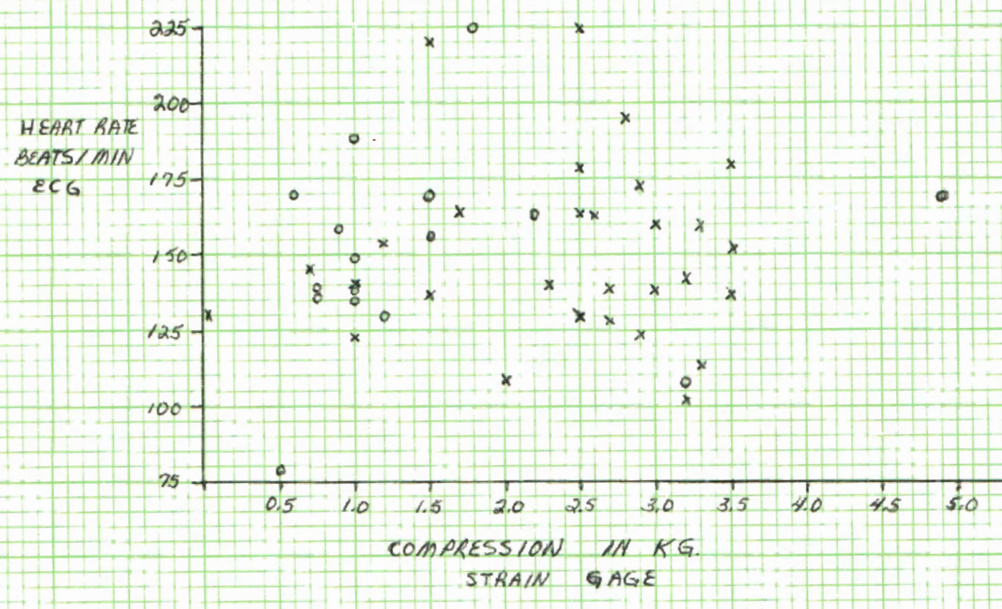

Figure 4. By comparing the heart rate in beats/ min. against the compression in kg. inside and outside the constructed pelvic outlet, no correlation between increments of rate versus compression by forceps on the newborn can be seen. The forceps application for this series was occiput posterior.

SEX	NBR. #	MIN. WT.	MAX. WT.	MIN. AGE	MAX. AGE
♂	18	6 lbs. 8 oz.	9 lbs. 4 oz.	½ hr.	48 hr.
♀	20*	5 lbs. 8½ oz.	9 lbs.	½ hr.	"
♂	18	6 lbs. 14½ oz.	10 lbs. 8½ oz.	½ hr.	"
♀	18*	5 lbs. 6½ oz.	9 lbs. 11 oz.	½ hr.	"

Table 1. *One child in 1964 and one in 1965 are not listed as their sex was not recorded.

Forceps Application	Rate Resting	Compression 500 gms.	Compression 1000 gms.	Compression 1500 gms.
OA	132 beats/ min.	+1	-1	-1
OP		-2	-2	0

Table II. Indicates average deviation in beats/ min. from average resting rate (132 beats/ min.) with forceps application.

	Normal average height	Average change with compression
P wave	0.078 mV	+ 0.011 mV
Q wave	0.233 mV	+ 0.067 mV
R wave	0.873 mV	+ 0.100 mV
T wave	0.910 mV	+ 0.024 mV
	Normal average duration	
P wave	80.4 msec	+ 3.2 msec
QRS complex	37.2 msec	- 2.6 msec
T wave	174 msec	+ 3.0 msec
P-R interval	149 msec	--
S-T segment	241 msec	+ 8.6 msec
T-P interval	80.8 msec	- 8.2 msec

Table III. Average normal height and average change caused by compression for the various ECG entities.

Baby F. # 9	Born: 9:05 a.m. on 7/13/64 ECG: 2:30 p.m. on 7/13/64							
Application of Forceps		OP	OP	OP	OA	OA	OA	OA
Am't of Pressure in Grams	0	500	1000	1500	500	1000	1500	2000
HR in beats per minute	136	145	132	132	140	144	142	141
T msecs	160	160	200	180	160	192	180	180
P "	100	80	120	120	80	100	128	120
QRS "	40	32	40	24	32	40	20	20
T mm	.5	1.2	1.2	1.1	1.0	1.1	1.1	1.2
P "	.6	1.0	.9	1.2	1.0	1.1	1.1	1.0
Q "	.4	1.0	1.0	1.0	1.2	1.2	1.0	1.0
R "	5.1	8.5	5.5	6.5	6.0	7.0	6.4	7.0
T-P msecs	60	60	64	80	40	48	60	92
P-R "	144	160	192	160	160	160	160	184
S-T "	60	64	60	60	60	60	60	72

Table IV. Typical recording of the average of 10 samples per maneuver showing variation within the ECG with only a slight change in rate.

Duration	1964	1965
T wave	174 msec	164 msec
P wave	80.4 "	84 "
QRS complex	37.2 "	41 "
P-R interval	149 "	151 "
S-T segment	67 "	80 "
T-P interval	80.8 "	74 "
<u>Height</u>		
P wave	.78 mm	.49 mm
Q wave	2.3 "	1.5 "
R wave	8.7 "	5.8 "
T wave	.91 "	.53 "
Heart Rate	132 beats/min.	130 beats/min.

Table V. Comparison of values for 1964 and 1965. Standard Lead I was used for all values.

ECG		<2000 gm. average	>2000 gm. average	<2000 gm. average change from control	>2000 gm. average change from control
T	msecs	163	166.5	+3.0	-9.8
P	"	85	83	-2.0	-7.0
QRS	"	41	41	-1.6	-2.4
T-P	"	74	73	-12.0	-16.0
P-R	"	152	151	-2.0	+1.0
S-T	"	80	81	-4.0	+5.0
T	mm	.5	.56	-.32	-.13
P	"	.5	.45	-.01	0
Q	"	1.5	1.6	0	+.01
R	"	5.8	5.6	-.10	-.10
FHR	beats/ min.	131	129	+1.0	+7.0

Table VI. Comparison of compression forces on the newborn heart at <2000 gm. and >2000 gm. Range: 500 gm. to 7460 gm. Some infants are listed in both the <2000 and >2000 gm. columns.

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