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Study Of Vitreous Humour Electrolytes To Assess The Postmortem Interval And Cause Of Death

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ABSTRACT

Forensic medicine deals with application of medical knowledge to solve medico legal problems to help in the administration of justice. With the ever increasing homicidal cases the estimation of time since death became an Achilles heel in Forensic medicine. Estimation of time since death plays an important role in investigation of crime. A cross-sectional study was carried out on 100 cases subjected to autopsy at district hospital, Belgaum, Karnataka, India and also from bodies dying of chronic lingering diseases at district hospital and Jawaharlal Nehru Medical college, hospital & research centre, Belgaum, Karnataka, India. Postmortem interval (PMI)/Time since death (TSD) was estimated using vitreous potassium. A linear association was obtained upto certain hours, when the mean values of potassium were plotted against PMI on a graph. Applying the statistical data from vitreous potassium concentration, the PMI can be calculated by using the formula, PMI= (2.99 x K+ conc. -6.26) + 3.29 hrs. This formula holds good, when the time since death is within 48 hrs.

Keywords: Postmortem interval; Time since death; Vitreous potassium; Vitreous humor electrolytes

Introduction

The rate of homicide and other unnatural deaths is increasing. Whenever an expert witness appears in the court to give evidence, the legal basis in the court of law is to enquire where? When? What is the postmortem interval (PMI)/ time since death (TSD)? This shows the importance of estimation of time since death for the people of law and law enforcement officers in streamlining investigating procedure.

Estimation of time since death helps the investigating officer to:-

1) Gather important information in the investigation of unattended deaths

2) Include the suspects who happened to be in and around the scene of crime in a case of homicide and also to exclude those who might have not been present.

3) Know approximately how far the alleged vehicle might have traveled by this time in a hit and run case; in deaths on rail lines following accidents or following natural and unnatural deaths in running trains.

In the early period i.e., during second half of 18th century the forensic experts made use of postmortem changes like postmortem cooling, postmortem lividity and rigor mortis to estimate PMI.[1] Unfortunately, the accuracy was low the margin of error remained large and unpredictable. Therefore the second half of the 19^{th} century onwards, chemical tests were introduced to determine PMI by estimating the electrolyte concentration of various body fluids like blood, CSF, synovial fluid, pericardial fluid, vitreous humor.

Of all these body fluids vitreous humor is the only fluid which is unique and preferred because it is anatomically separated, resistant to putrefaction for a long time, most sterile. Even in most brutal homicide, caused by multiple stab injuries, eyes are always spared.[2] Following death, there is cessation of metabolism and energy production. Autolysis starts leading to dissolution of the chemical, physical and morphological integrity of the vitreous body, cessation of active membrane transport, loss of selective membrane permeability. Therefore, this leads to diffusion of ions from intracellular fluid to extracellular fluid according to their concentration gradients till the equilibrium is reached.

At present Dr. Sturner's equation[3] which was proposed in 1964 (PMI= 7.14 X K conc. -39.1) is been widely quoted and used by Forensic pathologists. Coe in 1973[4] discussed cases of people dying in hot environments and established that the slope for value of potassium rise in hot conditions was very steep, that is the rise of potassium in vitreous humor was much more rapid than the predicted value from any of the published graphs. In 1977, Koumura and Oshiro[5] found a linear relationship between potassium values of vitreous humor and PMI but the slope of the line for ambient temperature of 26^0 - 29^0C was considerably steeper, than the slope of specimens obtained from bodies residing in temperatures of 13^0 - 17^0 C. Similar findings were reported by a number of other authors.[6,7,8]Therefore in any study the external variables have to be taken into consideration and a universal formula cannot be applied to determine PMI.

Thus in the present study an effort is made to derive a formula to estimate near accurate PMI in this tropical region from postmortem chemistry (sodium, potassium, chloride) of vitreous humor. An effort is also made to know if the postmortem vitreous electrolyte concentration can help in determining the cause of death or their relationship to pathophysiology before death.

Material

The study was carried out in the department of forensic medicine with assistance from department of Biochemistry, Jawaharlal Nehru Medical college, hospital and research centre, Belgaum, Karnataka, India. Ethical clearance was obtained from college ethical committee.

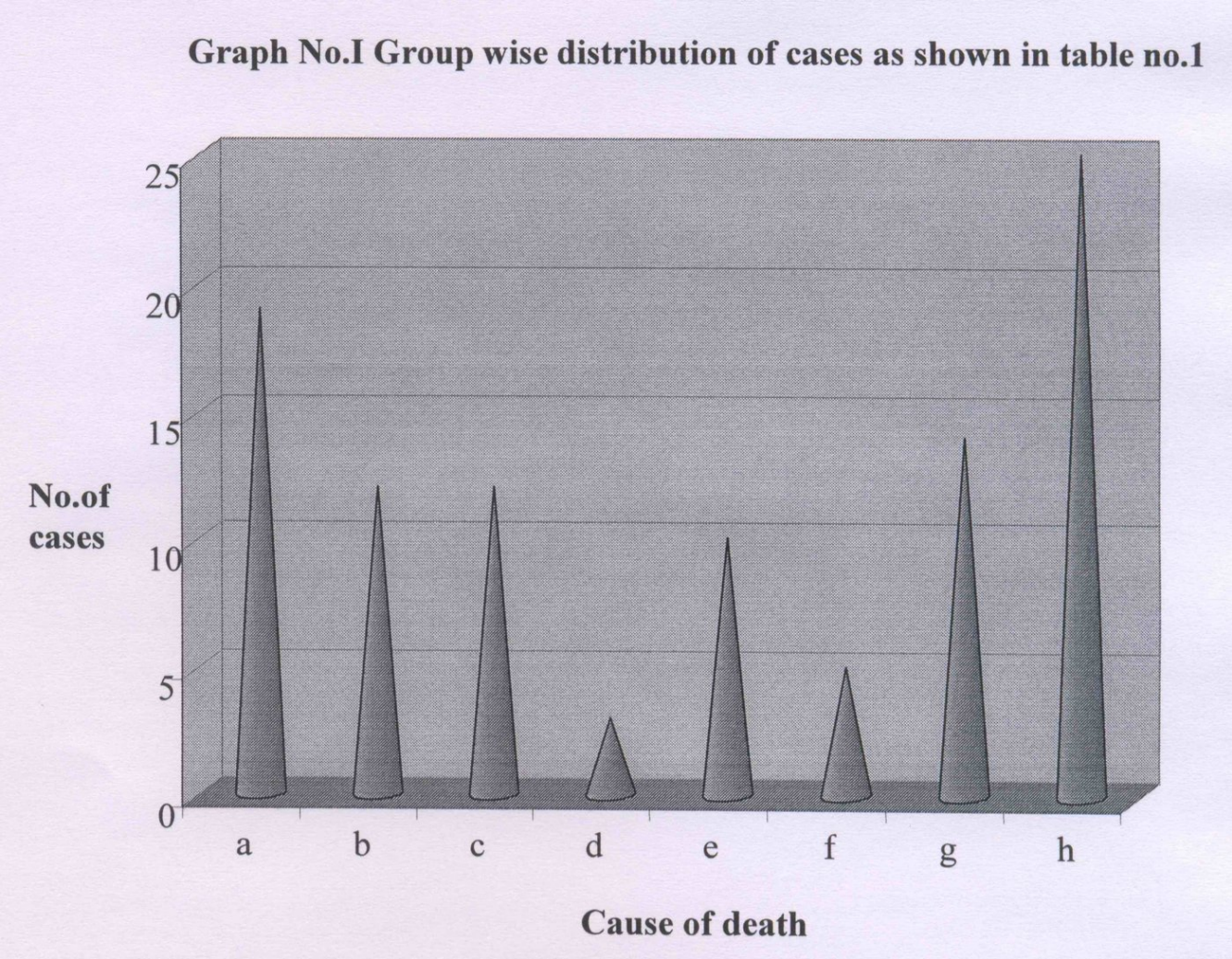
The vitreous humor was sampled from 100 cases subjected to autopsy at district hospital and also from bodies dying of chronic lingering diseases at district hospital and Jawaharlal Nehru Medical

College, hospital & research centre, Belgaum. The time of death and cause of death was established by means of separate questionnaires answered by relatives, friends, ambulance crew, investigating officer and by going through the inquest report and medical records in case of hospital deaths. Those cases, whose time of death on enquiry from different sources was found to differ by more than ±15 min, were not included in the study. The time since death thus obtained was further cross-verified by postmortem changes like hypostasis, rigor mortis and putrefaction.

In the forensic group (n=75), deaths were due to road traffic accidents, fall from height, head injury, burns, electrocution, asphyxial death and death due to poisoning. In the pathology group (n=25), deaths were due to chronic lingering diseases, like cancer, infectious disease, cardiac failure, myocardial infarction. The ambient room temperature of the mortuary was 22-24^0 C. Samples were stored at 4^0 C for upto maximum 24 hours for further analysis.

Methods

All the vitreous humor from both the eyes were collected and estimated separately for sodium, potassium and chloride. The sample was collected using 10ml syringe and number 20 needle in a clean sterile bulb. The needle was introduced in the eye through the outer canthus, 4.5 cms lateral to limbus and 5 ml of vitreous humor was aspirated as gradually as possible. The sample was immediately centrifuged for 10 minutes at 3000 revolutions per minute. The supernatant was used for estimation after diluting with required amount of ion free water.



Graph I. Group wise distribution of cases as shown in table no.2.

Sodium and potassium were estimated by **flame photometer**.[9] Flame photometer 'Mediflame' 127 (Systronics) is a dual channel instrument, capable of simultaneous estimation of sodium (Na) and potassium (K). The fluid under analysis is sprayed as a fine mist into a non - luminous flame which becomes colored according to the characteristic wavelength emissions of the elements (Na: 589nm; K: 768nm) which is detected by a detector. The outputs of the detectors are processed by the electronic metering unit and the results are appropriately displayed on the digital panel. Standard stock solutions of sodium (200mmol/L) and potassium (10mmol/L) were first prepared. **Standard stock solution of sodium** = 11.69 gms of pure dry analytical grade sodium chloride was dissolved in a liter of glass distilled water. **Standard stock solution of potassium** = 0.746 gms of pure dry analytical grade potassium chloride was dissolved in a liter of glass distilled water. **Preparation of standard mixed working solutions: -** The above standard stock solutions were mixed as per the given table below and diluted to 100ml with glass distilled water.

Na stock solution(ml)	K stock solution(ml)	Na conc. (mmol/L)	K conc. (mmol/L)
0.50	0.1	1.0	0.01
0.55	0.2	1.1	0.02
0.60	0.3	1.2	0.03
0.65	0.4	1.3	0.04
0.70	0.5	1.4	0.05
0.75	0.6	1.5	0.06
0.80	0.7	1.6	0.07
0.85	0.8	1.7	0.08

Table 1.

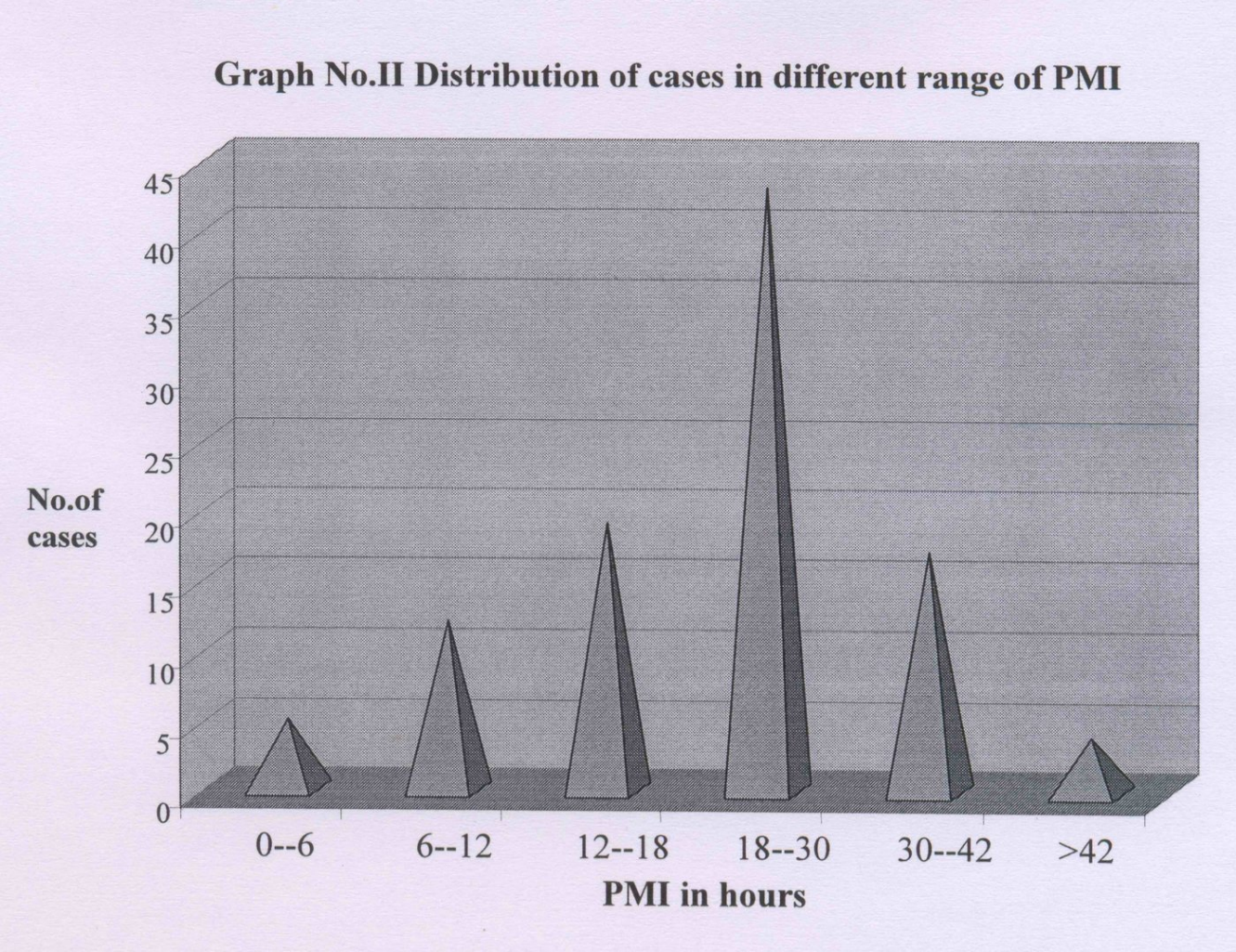
1.0/0.01 mmol/L of Na/K solution was aspirated and adjusted for a read out of 100 for Na only. Next 1.7/0.08 mmol/L of Na/K solution was aspirated and adjusted for a readout of 170 for Na and 80 for K. The standard mixed working solutions were repeatedly run until the readings were stabilized. The unit thus stands calibrated. **Preparation of sample:** 0.2 ml of vitreous humor was diluted with 19.8 ml of glass distilled water.

Chloride was estimated based on the modified Schoenfeld and Lewellen **colorimetric method**.[10] Chloride ions react with undissociated mercuric thiocyanate to form undissociated mercuric chloride and free thiocyanate ions. The free thiocyanate ions react with ferric ion to form the highly colored, reddish complex of ferric thiocyanate with an absorption peak at 505nm. The concentration of the standard used was 100mmol/L. Commercial reference control serum was used to ensure adequate quality control. All the glassware and cuvettes required for the assay were washed with nitric acid and rinsed with double distilled water. The values of both the right and left eye were added and the average was taken for statistical analysis.

Inclusion criteria: Only crystal clear, vitreous humor was used for analysis (with the onset of putrefaction the fluid becomes cloudy and brownish in color). Cases with known time since death and cause of death alone were included in the study group.

Exclusion criteria: Cases with known or suspected ocular diseases or trauma and vitreous contaminated with blood were excluded. Time of death was kept confidential to avoid any bias in results.

Results



Graph II. Distribution of cases in different range of PMI.

100 cases of known postmortem interval (PMI) were included in this study. Of the 100 cases 74 were male and 26 were female with the age ranging from 20-60 years (average age= 37.5 yrs). The reported PMI in the present study was in the range of 3-56 hours. The distribution of cases in different range of PMI is shown in graph no.2. Deaths due to both natural and unnatural causes were included in this study.

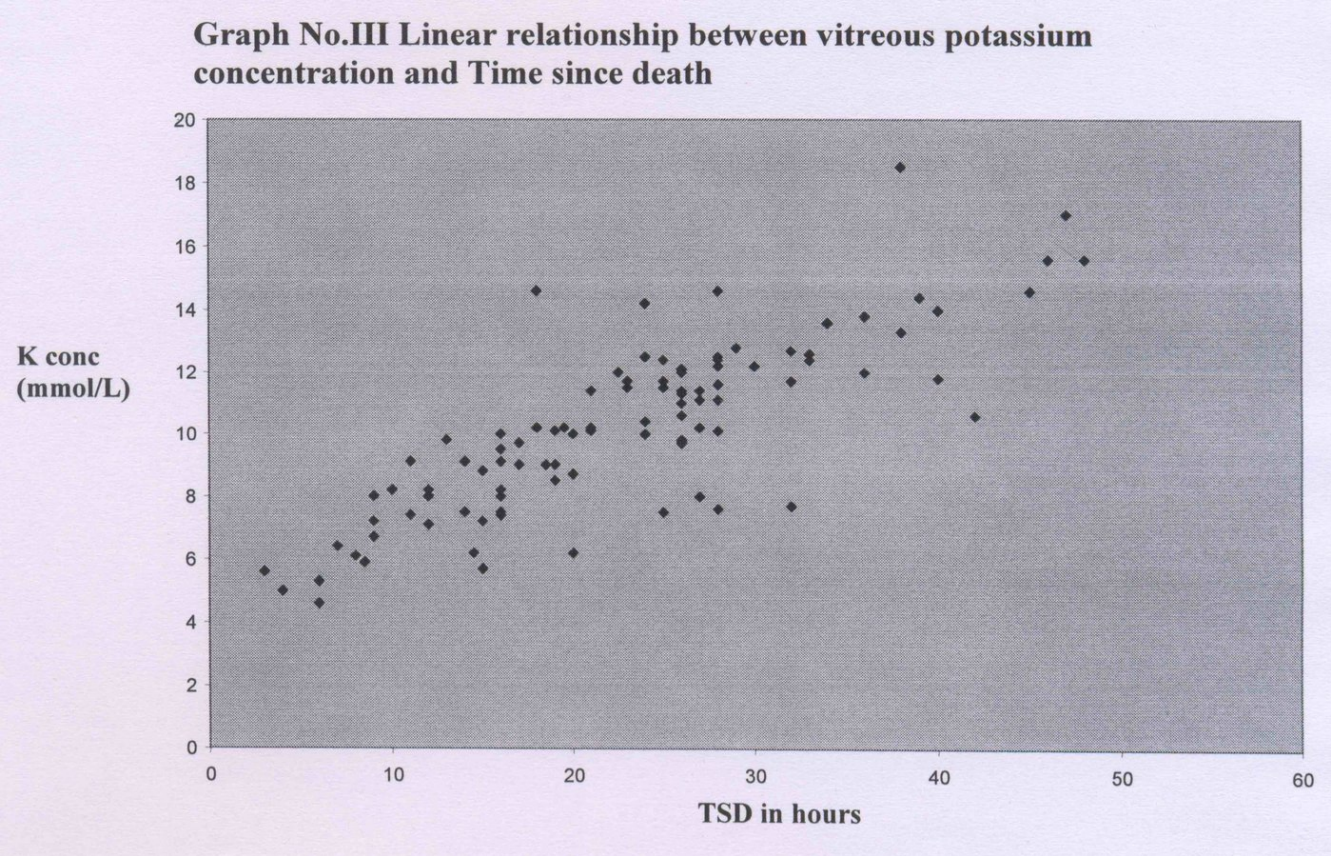
By applying simple regression analysis, the following statistical results were derived; r (*correlation coefficient*) and R (*regression coefficient*). The *mean* value of postmortem vitreous potassium concentration for all the cases was 10.21 ± 2.79 mmol/L (mean ± SD) and *coefficient of variance* was 0.27%. The *mean* time since death for all the cases was 22.59 ± 10.16 hours (mean ± SD). The mean value of potassium and mean PMI for different groups (eight) are shown in table no. 2.

Cause of death	No. of cases	Chloride (mmol/L)	Sodium (mmol/L)	Potassium (mmol/L)	PMI(hrs)
RTA (a)	19	133.09 + 7.98	150.0 + 13.21	11.21 + 2.47	24.63 + 9.02
Poisoning (b)	12	127.09 + 9.12	143.05 + 13.39	11.58 + 3.05	26.67 + 8.86
Burns (c)	12	104.0 + 4.6	116.2 + 8.58	7.30 + 1.77	20.29 + 8.89
Fall from height(d)	03	97.18 + 11.26	110.45 + 7.5	12.80 + 1.63	32.67 + 9.50
Stab/assault(e)	10	118.21 + 9.59	131.63 + 5.94	10.36 + 2.98	24.50 + 6.60
Electrocution (f)	05	113.56 + 13.55	132.35 + 7.89	10.70 + 2.33	20.60 + 6.42
Asphyxial deaths (g)	14	124.99 + 10.22	136.44 + 13.26	12.23 + 1.85	29.71 + 10.46
Natural causes (h)	25	119.61 + 25.26	138.65 + 12.18	7.90 + 2.49	15.60 + 10.42
Total	100	125.14 + 12.62	140.15 + 15.13	10.21 + 2.79	22.59 + 10.16

Table 2. Average electrolyte concentration and PMI in different groups

The *correlation coefficient* (r) between the two variables was r = 0.83, which is statistically significant (t=14.13, df = 98, p<0.0001). In order to establish linear association between potassium concentration and PMI, a scatter diagram was plotted (Graph no. III). The potassium levels increased in the vitreous humor in a regular fashion with an increasing postmortem interval. This increase was rapid initially but soon levels off to a more gradual rate of increase. After 48 hours of PMI the potassium concentration did not rise significantly. This could be due to attainment of diffusion equilibrium of potassium across the cell membrane.

Highly significant changes occur during the PMI in the levels of potassium in the vitreous humor. These changes are proportional to the time that has elapsed since death and provide a theoretical basis on which the time of death can be calculated, by extrapolation of a line joining the two results. From the data the least square linear regression equation between PMI (*the dependent variable*), potassium concentration (*the independent variable*) was found to be y=a+bx. The 'a' and 'b' represent the intercept and regression coefficient respectively. These constants were estimated from data and the *regression equation* was PMI=2.91x K + conc-6.26.



Graph III. Linear relationship between vitreous potassium concentration and Time since death

The *analysis of variance* (ANOVA) was done to test the linear relationship between potassium concentration and PMI (ANOVA no.1).

Source of variation	Degree of freedom(df)	Source of squares(SS)	Mean of squares(MSS)	F ratio
Due to regression	1	7661.78	7661.78	239.35
About regression or error	98	3136.05	32.01	
Total	**99**	10,797.83	P<0.0001	

ANOVA No.I

This confirms the linear relationship between potassium concentration and PMI (p<0.0001). For per unit change in potassium concentration, change expected in PMI is 2.91 hours. To test the hypothesis that value of potassium concentration increased with the value of PMI, that is b=0 against b ?0, students't' test was used. The't' value was found to be 15.302(df = 98), which was statistically significant (p<0.0001).

Source of variation	Degree of freedom(df)	Source of squares(SS)	Mean of squares(MSS)	F ratio
Due to regression	1	622.18	622.18	220.36
About regression or error	98	267.70	2.82	
Total	99	899.88	P<0.0001	

ANOVA No.II

When the role was reversed, that is when potassium concentration was taken as *dependent variable* and PMI as *independent variable* , the *regression equation of potassium concentration* on PMI was, K= 4.48 + 0.24 PMI. The ANOVA table of this is shown in ANOVA no.2, which confirms the linear relationship between the two (t =13.826, df = 98, p<0.0001). For per unit change in PMI, change expected in potassium concentration was 0.24mmol/L. Vitreous sodium and chloride did not show linear relationship with postmortem interval and moreover their concentration remained constant for few hours postmortem which was later followed by decline in their concentration at a rate of 1mmol/L/hour.

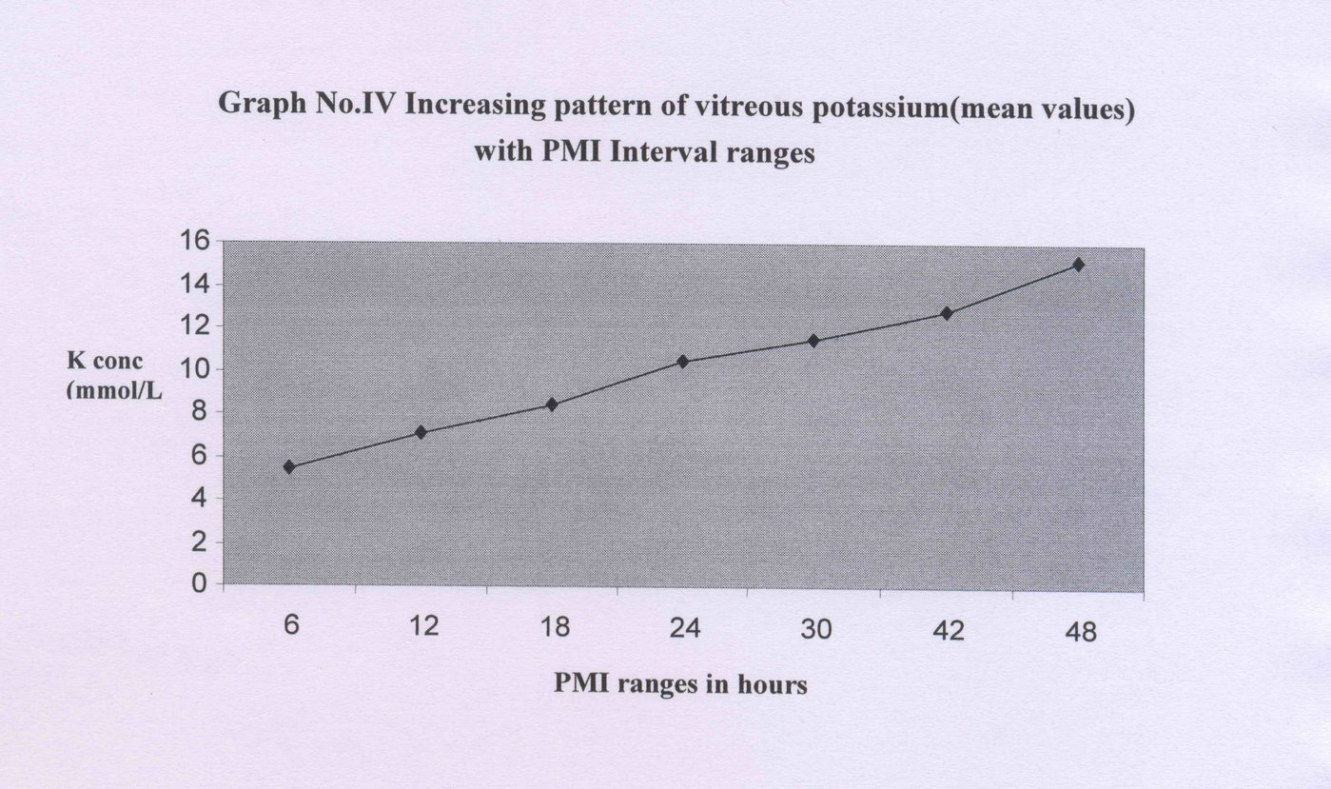
Discussion

Over more than one and half centuries, Forensic experts have attempted to estimate the accurate time since death or PMI. Almost all workers admit that the level of accuracy is not 100%, and more importantly the 95% confidence limit increases with increasing PMI and becomes unpredictable with wide limits. Thus only a range of preferred times can be offered to the investigating officer within which death was likely to have taken place.

The normal vitreous concentration of potassium is 2.6-4.2mmol/L, sodium is 118-124mmol/L and chloride is 108-142mmol/L.[11] During lifetime, potassium is almost intracellular. High intracellular concentration of potassium is maintained by Na^+-K^+ pump. After death this Na^+-K^+ pump does not operate, therefore K+ is leaked out of cell, leading to high postmortem levels. It is postulated that normal antemortem entry route of potassium into vitreous humor is through ciliary body. After death, autolysis of the vascular choroids and retinal cells are responsible for its rise).[12]

The levels of sodium and chloride in vitreous remained constant for prolonged postmortem intervals. Later, their levels decreased at a rate of 1mmol/L per hour.

The concentration of vitreous potassium increased with increase in postmortem interval upto 48 hours after which there was a slight fall. The slope of the group as a whole was 0.24meq/hour with an intercept at zero time of 3.6meq. The 95% confidence limit for all the cases was ±3.29 hours, when PMI was within 48 hours.



Graph IV. Increasing pattern of vitreous potassium (mean values) with PMI Interval ranges

It was Naumann in 1959,[12] who first reported this relationship. Later on Sturner and Ganter in 1964[3] devised linear regression model upto 104 postmortem hours with 95% confidence limit of 9.5 hours to describe this relationship which was extensively evaluated by the follow up investigators. Adelson et al[13] emphatically suggested that the rise was in arithmetic progression. Our findings were consistent with the works of several other investigators.[12,13,14,15,16,17,18,19,20]

They noted that potassium increased in a regular fashion and the average rate of rise was 0.17mmol/L.

There was a nonlinear relationship between sodium and PMI. In the present study, sodium values ranged between 99.80 to 186.21 mmol/L, the *average* sodium concentration was 140.15 mmol/L, with *standard deviation* of 15.13 and the *coefficient of variance* was 0.10%. The sodium concentration remained constant for an average of 26 hour postmortem. Our findings were consistent with the works of several investigators.[12,16,21,22] Later the concentration of sodium starts declining at a rate of 1 mmol/L.

There was a nonlinear relationship between chloride and PMI. In the present study, chloride values ranged between 95.82 to 149.29 mmol/L, with an average of 125.14 mmol/L, with standard deviation of 12.62 and coefficient of variance was 0.10%. The chloride concentration remained constant for an average of 18 hours postmortem. Our findings were consistent with the works of several investigators.[12,21,22,23]

Groups/ PMI (hrs)	RTA	Poisoning	Burns	Fall from height	Stab/Assault	Electrocution	Asphyxia	Natural death	Mean K value of all groups
0-6	--	--	5.6	--	--	--	--	5.3	5.45
6-12	7.4	--	5.9	--	--	8	--	7.04	7.09
12-18	8.75	7.1	7.46	--	8.1	9	9.75	8.48	8.38
18-24	11.32	10.13	6.2	11.5	8.85	14.6	10.9	--	10.5
24-30	11.63	11.08	8.2	14.6	11.86	11.25	12.06	10.9	11.45
30-42	13.17	14	10.6	12.4	14.4	14	11.55	--	12.87
>42	15	15.6	--	--	--	15.6	14.6	--	15.2

Table 3. Mean vitreous potassium concentration (mmol/L) in different PMI ranges in different groups

The linear rise of vitreous potassium upto 48 hours postmortem was described by the following simple regression equation.

Our equation: PMI = 2.99 x K conc - 6.26 **or** K^+ = 4.48 + 0.24 x PMI

Sturner's equation[16] : PMI = 7.14 x K^+ conc -39.1 **or** K^+ = 5.476 + 0.14 x PMI

J.G.Farmer's equation[8]: PMI = 0.61x K^+ conc - 27.31 **or** K^+ = 238 + 4.752 x PMI

Any formula reported till date are valid only for adult individuals and cannot be used to establish PMI of infant deaths. The advantage of this equation is that, in case, where the PMI is in question, it can be determined by simply estimating the vitreous K^+ concentration and inserting its value into the formula. However, many authors have stated different 95% *confidence limit* . In Sturner's and Ganter's study, their *confidence limit* is ± 9.5 hours.[3] The *confidence limit* continues to increase with increasing postmortem interval. In this study the 95% *confidence limit* was ± 3.29 hours, when the PMI is within 48 hours.

The rise of vitreous potassium concentration upto 48 hours of death in the present study was in contrast to longer time period reported by western workers. It could be because of the tropical climate in India , which causes the dead body to putrefy faster leading to faster biochemical changes. B. Madea and others state that the reasons for *variable confidence limit* are electrolyte imbalance at the time of death, duration of terminal episode, age, ambient temperature, uremia, presence of osmotically active substances like alcohol and drugs.[24] External factors which influence the validity of the test are sampling techniques, analytical instrumentation and environmental temperature during the PMI. Internal factors that are recognized at the

present time which influence vitreous potassium are the age of the individual, the duration of the terminal episode and the presence or absence of nitrogen retention).[16] C.Hanssge et al emphasized the limitations of various methods, the need for assessing multiple parameters, and the need to express the probable postmortem interval as a range. They appropriately cite the words of Davey (1839): "Much judgment, however, and nice discrimination may be requisite on the part of the medical man . . . so as to enable him when called on for his opinion, to give one which will be satisfactory to the legal officers-and to himself, on reflection."[25]

In the 75 medico legal cases, the electrolyte concentration was within the normal range except for burns cases, where the vitreous electrolyte concentration was either below the normal range or very close to lower limit of normal range. This shows the antemortem electrolyte imbalance, which could be the pointer to the primary cause of death or it can be used as corroborative evidence.

In the 25 natural death cases (MI, cirrhosis of liver, pulmonary tuberculosis, HIV with opportunistic infections, carcinoma of colon and rectum, meningitis, status asthmatics, gastroenteritis), cirrhosis of liver showed vitreous electrolyte concentration less than the normal range. This indicates that there was electrolyte imbalance just before death, which resembled the low salt pattern of Coe's study.[23] Gastroenteritis showed elevated electrolyte concentration, resembling the dehydration pattern of Coe's study.[23] In the rest of the group, the vitreous electrolyte concentration was erratic and was not specific to any cause of death. In all these groups, the terminal episode was more than 12 hours.

Conclusion

Of the three electrolytes Na^+, K^+, Cl^- estimated from postmortem vitreous, only K + serves as a useful marker for estimating time since death in the early postmortem period. The sodium and chloride values are of little significance in achieving an accurate estimate of PMI.

From the above discussion, it is also evident that vitreous electrolyte imbalance occurs in deaths due to dehydration, burns and cirrhosis. However, their usefulness in determining the cause of death or their relationship to pathophysiology before death continue to be a subject of further studies.

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