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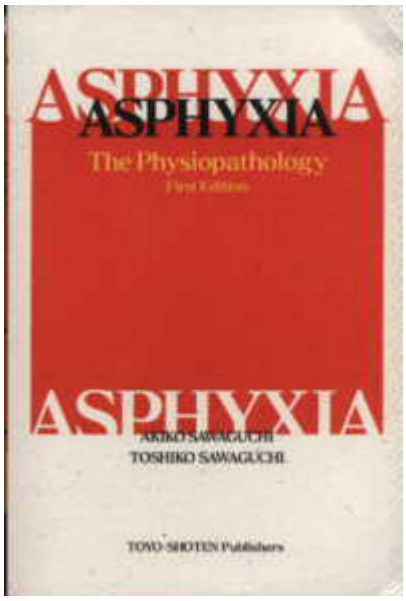
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NEW IDEAS IN PATHOPHYSIOLOGY OF ASPHYXIA

Asphyxia-The Pathophysiology, 1st Edition, *by* Akiko Sawaguchi and Toshiko Sawaguchi. paperback, 5.5" x 8".
Toyo-Shoten Publishers, 97 Yaraicho, Shinjuku, Tokyo 162-0805, Japan. Publication Date 2001. 140 pages, ISBN 4-88595-340-5. Price Yen 4000.00

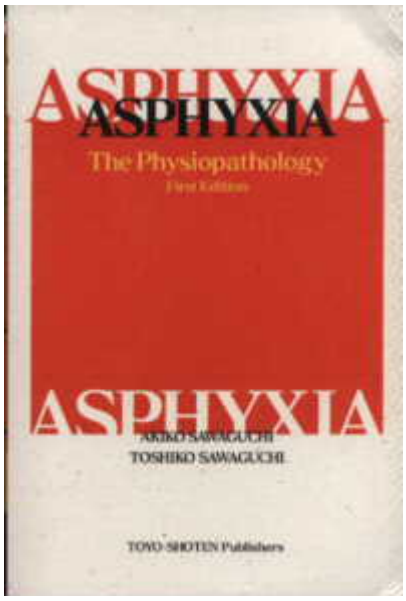


Akiko and Toshiko Sawaguchi

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Asphyxial deaths remain one of the most important areas of investigation of a forensic pathologist. A number of post-mortem findings have been attributed to asphyxial deaths, such as cyanosis, petechial haemorrhages, congestion and oedema of internal organs, fluidity of blood and engorgement of the right side of the heart[1] , but these have been vehemently challenged by a number of pathologists, foremost among them being Bernard Knight[2] . Lester Adelson called them the 'obsolete diagnostic quintet'[3] . Since, this controversy is not likely to be over soon, many pathologists started looking at other directions for diagnosis of asphyxia - one of them being the search for a perfect "chemical marker". Is there a "chemical marker" in the body, the presence of which would strongly point to death by asphyxia?

The book under review attempts to answer this question. Funded in part by a grant from the Ministry of Education in Japan, this book is based mainly on the work of the author herself. Over the years she has conducted a number of elegant experiments on animals and has amassed an amazing wealth of data on the pathophysiology of asphyxial deaths. Most of her work has been published either in the *Journal of the Tokyo Women Medical College* or in *Japanese Journal of Legal Medicine*, which is not surprising as she hails from the Tokyo Women's Medical University, Japan. This data has remained insulated from the English speaking world so far. This book attempts to provide all that valuable data to the English speaking world for the first time. Anthony Busuttil, Regius Professor of Forensic Medicine, at the University of Edinburgh thinks such works "deserve credit and recognition".[4]

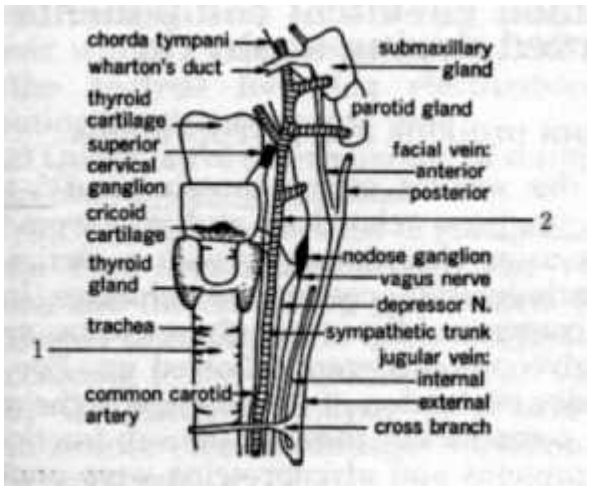


..Are we moving towards an era, where in doubtful cases, a pathologist could just order a few routine blood tests (of the corpse) and write out the cause of death? Well, we will have to wait and see, but what I definitely can say, that about a century ago, one would have refuted this assertion vehemently in relation to clinical diagnosis...

On the very first page of the book, the author defines asphyxia very aptly - it is in very broad sense a condition in which an abnormality in the arterial blood gases (hypoxemia or hypercapnia) occurs due to a disruption in breathing mechanics, so that the host can no longer maintain normal activities. Three types of asphyxia are then defined according to the clinical course. Acute asphyxia is said to occur when the time interval between the onset of asphyxia till death ranges from a few minutes to a little over ten minutes. Typical examples are hanging, complete ligature strangulation, and complete airway closure by any other means. In subacute asphyxia the duration of survival is more than ten minutes as in incomplete airway closure or airway narrowing and delayed death from drowning. Asphyxia of a still longer duration (typically between 50 and 183 minutes, as the author informs us at several places in her book) results in adaptive and compensatory responses. This is known as chronic asphyxia. All her work has to be seen in the context of these definitions set out by her in the beginning.

Let us now have a look at the kind of work she has done. She has taken rabbits and dogs and has asphyxiated them experimentally in a number of ways. Why rabbits and dogs, and not other animals? As she explains on page 42, an animal had to be chosen, that could withstand the experimental conditions without anesthesia, and rabbits seemed to suit the bill very well. Dogs were chosen for a selected number of purposes only - as for the estimation of blood sugar after asphyxia - because "antibodies may be used in these animals for insulin analysis related to carbohydrate metabolism" (page 81).

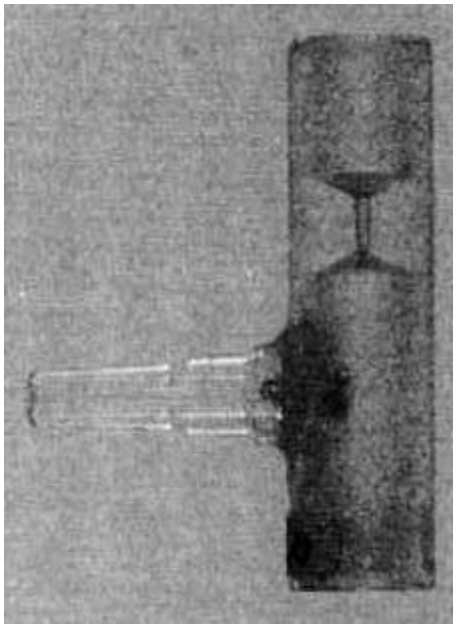
Surgical operation in the rabbit to expose neck structures. 1 represents the site where the trachea is obstructed. 2 represents the site where a canula or probe is installed for insertion into the carotid artery. This figure appears on page 43 of the book.

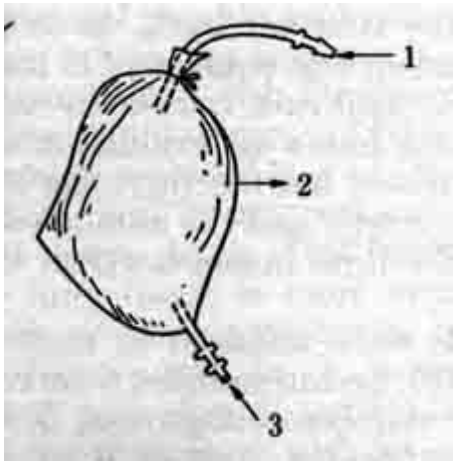


Six different methods were employed to asphyxiate these animals and various blood constituents studied after that. The methods were (i) Pressure on trachea (ii) Asphyxiator (iii) Tracheostenotic cannula (iv) Hanging (v) Strangulation and (vi) Compression of nerves and blood vessels. In addition a control group was killed by four different methods - by cyanide, strychnine, air embolism and by blows to the neck. Of these the strychnine group gave several findings similar to those of asphyxia, as strychnine in large amounts is known to paralyze the respiratory center and this effect can be construed as equivalent to asphyxiation.

In the first group the pressure on the neck was not applied directly. Instead, by an elaborate surgical procedure the trachea of the animals was exposed which was then occluded by applying pressure with a pair of haemostatic forceps. Asphyxiator is an interesting device, which mimics a closed room. For the rabbits, this was an airtight box measuring 25x40x25 cm (capacity 25 L) and made of clear resin, and for the dog it measured 30x60x30 cm (Capacity 54 L). It could be hermetically sealed by attaching highly elastic rubber pieces to the sections where the main body and its cover came in contact. The typical survival time for the animals in this box was about 2 to 2.5 hours, which mimicked chronic asphyxiating conditions in a closed room.

Tracheostenotic cannula designed at the author's laboratory (top), and Vinyl bag (bottom). Tracheal cannula is attached at point 1, and oxygen monitor at point 3. These figures appear on page 77 of the book.

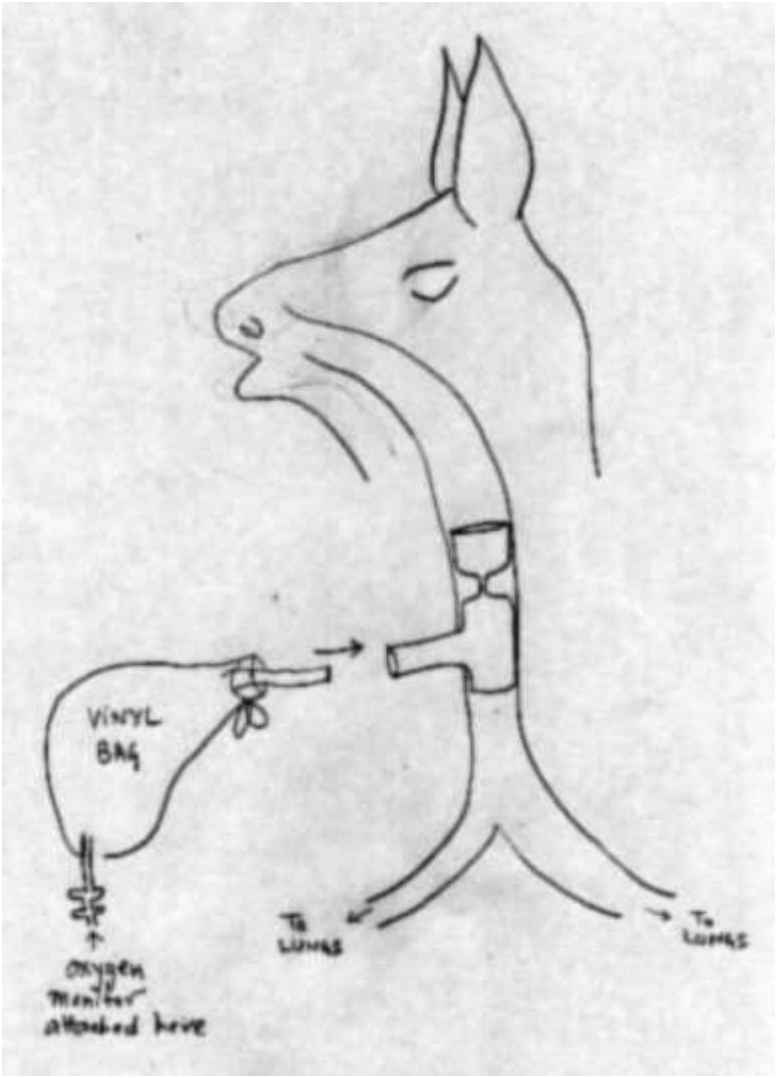




Tracheostenotic cannula is again an extremely interesting device (and something of which I for one, heard for the first time!). It is a tube which can be inserted in the trachea. The tube can be surgically inserted in the trachea in such a way that the little nozzle juts out (please refer to accompanying figure).

The tube has a small stenotic portion which is about 3.0 mm long and just 0.5 mm wide. To the nozzle can be attached a small vinyl bag (see the figure below) through which 3,400 to 5,000 ml of air could be delivered in strictly measured quantities. Things could be so arranged, that the animal could be made to breathe either through the nose - in which case, it had to overcome the resistance of the stenotic part of the cannula, although the supply of air remained unlimited - or through the vinyl bag - in which case there was no resistance, but the supply of air was limited. As can easily be seen, while the latter case simulates locking up in a room, the former corresponds more to conditions like hanging and strangulation. The typical survival period in the latter (in which the animal was made to breathe from air in the vinyl bag) was less than 60 minutes, while in the latter case it was between 50-183 minutes. In this way, the authors could produce conditions of subacute and chronic asphyxia.

This is how the cannula is inserted in the animal (This picture does not appear anywhere in the book. This is the reviewer's own interpretation of what the authors describe)



In addition, some animals were hanged and others strangulated. Details of how they were hanged are not provided (on page 92, the authors just say that "apparatus for head fixation and hanging.. .was designed at our laboratory"), but the details of strangulation are provided. Two synthetic resin ropes measuring 0.5 cm in diameter were wrapped round the neck once and pulled from both ends with a traction force amounting to 2.5 to 3.5 times the body weight (the traction force is an important parameter in strangulation. In hanging, it is necessarily equal to 1).

Then there is a last group of animals in which the death was caused by compression of major blood vessels and the nerves of the neck. This was also achieved by first exposing these structures by an elaborate surgical procedure and then directly clamping them. As Busutill quite rightly points out in his review of this book, these "carefully controlled experimental designs.. .do not hold true to any reproducible extent in actual practice.. .Yet they provide ample food for thought".[5]

What about the results? Well, they are as interesting as can be. The authors have studied an amazing array of biochemical markers including serum proteins, glycoproteins, serum enzymes and isozymes, blood sugar, hormone levels, and blood lipids. Other things studied are the dynamics of body fluid and acid-base equilibrium, serum electrolytes, blood gases, CSF gases and acid-base equilibrium and partial pressures of the gases contained in the brain, liver and kidney tissues. Regarding the results, the reader may want to explore the book himself, but I will give just a sampling here. The authors found that the serum total protein level rises immediately after lethal asphyxia which they ascribe to "condensation of the blood during the asphyxial process" (pages

49-50). Although the total protein rises, there is a reduction in the alpha-1 and gamma globulin fractions and an increase in the albumin fraction and alpha-2 and beta globulin fractions. The authors ascribe the increase in the albumin fraction to the "escape of blood components as a consequence of acidosis .. and damage to the vascular wall". Similarly they found an increase in calcium in asphyxial deaths, especially those due to strangulation, and this they ascribe to the pressure on the parathyroid gland!

Abnormal arterial blood gas contents and acid-base equilibrium in acute asphyxia caused by tracheal obstruction pressure. (experimental animals : rabbits weighing approximately 3.0 kg). This table appears on page 115.

N=7 (x ± S.D.)	before	2~3 min.	7~8 min.	after death
Po_2 (Torr)	102.8 ± 17.0	21.5 ± 7.6	3.3 ± 1.2	1.6 ± 0.9
Pco_2 (Torr)	23.2 ± 6.7	57.6 ± 8.2	76.2 ± 2.3	88.1 ± 6.3
pH	7.371 ± 0.068	7.051 ± 0.090	6.897 ± 0.011	6.840 ± 0.030
BE (mMol/l)	-8.1 ± 2.6	-15.5 ± 3.4	-19.6 ± 1.1	-21.1 ± 2.4
HCO_3^-	14.7 ± 4.5	15.8 ± 1.2	14.8 ± 0.3	15.1 ± 0.5

Regarding the depth of study the authors have done on blood gases in asphyxial deaths, I am reproducing just one table on the left. This should give the readers some idea, what to expect from this book.

So are we moving towards an era, where in doubtful cases, a pathologist could just order a few routine blood tests (of the corpse) and write out the cause of death? Well, we will have to wait and see, but what I definitely can say, that about a century ago, one would have refuted this assertion vehemently in relation to clinical diagnosis. At that time the quartet of inspection, palpation, percussion and auscultation was the holy grail of correct diagnosis, but how many clinicians routinely employ it today? They are just happy to order blood tests, X-rays and other investigations at the drop of a hat without an iota of doubt that they will miss out something. Who knows forensic pathology may reflect this state of affairs a century hence. This book definitely prepares a ground work for that.

Who will benefit from the book most? Anyone with an interest in forensic pathology - students, forensic practitioners, death investigators, biochemists. But the ones who are really going to love this book will be those, who plan to reproduce this study in their own labs, and may be take this work further.

References

(1) Camps, Francis E. (Ed.). Gradwohl's Legal Medicine, 3rd Edition, 1976, Bristol: John Wright & Sons Ltd. Pages 327-329. (Back to the review)

(2) Knight B. Forensic Pathology, 2nd Edition, Arnold, London, Pages 347-351. (Back to the review)

(3) Quoted by Knight B. Vide supra, page 347. (Back to the review)

(4) Busuttil A. Book Review of "Asphyxia - The Pathophysiology, 1st Edition by Akiko Sawaguchi and Toshiko Sawaguchi". Journal of Clinical Forensic Medicine 2001;8:185. (Back to the review)

(5) Busuttil A. Vide Supra. (Back to the review)

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