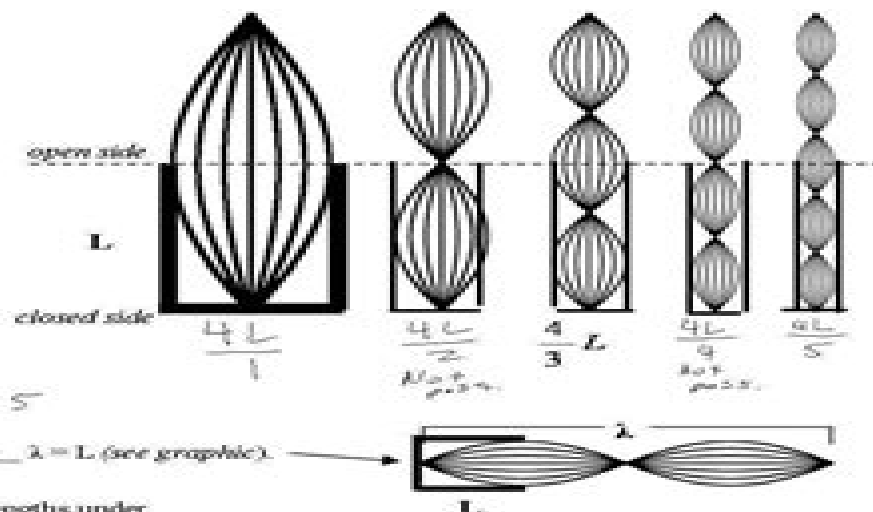


Harmonics in Closed Pipes

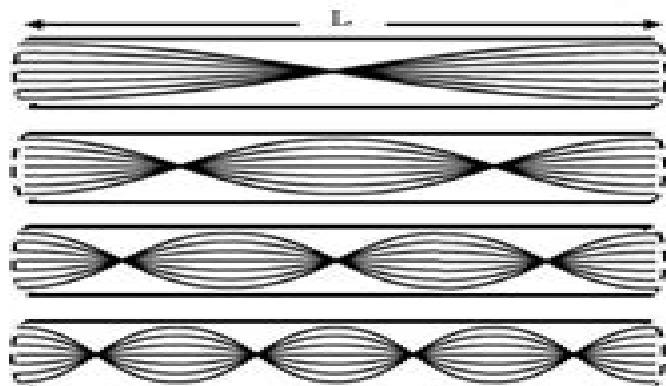
- 19) At the open end of a pipe is there a node or antinode?
- 20) At the closed end of a pipe is there a node or antinode?
So, there MUST BE a node at the closed end and the center of an antinode at the open end for the harmonic to exist.
- 21) On the graphic at the right, let's pretend we have a pipe closed at one end, as shown on the fundamental. The length of the pipe is "L".
- 22) Draw this same length pipe around each of the harmonics. The open end of each pipe will stop at the dotted line.
- 23) Which harmonics can be produced in this pipe (which have an antinode at the open end)? 1, 3, 5
- 24) So, for a pipe closed on one end (a closed pipe), give the sequence of what harmonics can be present. n can only = 1, 3, 5



- 25) Since $2AN = 1\lambda$, then the first harmonic in this diagram, $\frac{1}{4}\lambda$ $\lambda = L$ (see graphic).
- 26) So $\lambda = \underline{\frac{4}{1}L}$. Write this under H_1 .
- 27) Use the same process as for the first harmonic, write the wavelengths under the harmonics (even the ones that can't really exist). H_2 and H_3 should be obvious.
- 28) Remembering Q7-9: there must be a pattern that includes "n" sequentially. Find it.
- 29) At the right, write the equation for the wavelength of a harmonic in a closed pipe:
- 30) But which "n's" are possible? 1, 3, 5...
- 31) Calculate the wavelength of the third harmonic in a 60 cm long closed pipe (remember to convert to m).
 $\lambda_1 = 4L = 4(.6) = 2.4m$ $\lambda_3 = \frac{4L}{3} = \frac{2.4}{3} = .8m$

$$\lambda_{\text{closed pipe}} = \frac{4L}{n}$$

Harmonics in Open Pipes



- 32) Remembering the rule about nodes and antinodes, what must be present at the end of a pipe that is open?
- 33) In order for a pipe to make a sound, it must have a standing wave in it, meaning it must have at least 1 node and 1 antinode (or more).
- 34) For each successive harmonic the number of antinodes goes up by 1.
- 35) For each successive harmonic the number of nodes goes up by 1.
- 36) In the four open pipes at the left, draw the first four harmonics (number 1 is done for you). The nodes will be equally spaced and the center of an antinode must be at each end.
- 37) On a string you count the number of AN to figure out which harmonic it is. For an open pipe, you count the number of Nodes to figure out which harmonic it is.
- 38) Which harmonics can be present in an open pipe (which "n's" are present)? all of 'em

- 39) Using the same logic, figure out the sequence and give the equation for λ in an open pipe. (Hint: the drawing above will help.)

$$\lambda_{\text{open pipe}} = \frac{2L}{n}$$

$n = \text{all}$

- 40) What is the wavelength of the 3rd harmonic in a 80 cm long open pipe?

$$\lambda_1 = 1.6m$$

$$\lambda_3 = \frac{1.6}{3} = .53m$$

Optic Basics P 2 Stephen Murray Answers

Yijin Wang



Optic Basics P 2 Stephen Murray Answers:

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Successful interventional glaucoma remains a sought after goal for the glaucoma clinician for many reasons Medical costs compliance and adherence remain major barriers for the treatment of our patients A truly definitive treatment for glaucoma is most likely to be largely surgical as it is doubtful that patients will continue to use glaucoma drugs in the future for anything other than transient lowering of intraocular pressure In the coming era it seems probable that neuroprotection and neurorestoration will be combined with MIGS to lower pressure The different types of MIGS are increasing in proportion to their efficacy To date there are no fewer than 12 choices available to glaucoma specialists Choice of procedure depends on the underlying anatomy as well as the patient s individual characteristics Truthfully the number of available surgeries depends upon their taxonomy Most are based on the outflow system and the canal as illustrated in this book *Working Mother* ,2002-10 The magazine that helps career moms balance their personal and professional lives *Bookseller and the Stationery Trades' Journal* ,1980 **Glaucoma Research and Clinical Advances** John R. Samples,Paul A. Knepper,2018-04-24 Foreword Volume 2 of our serial publication continues our desire to address glaucoma with a combination of science and speculation As science expands the emphasis is on data interpretation and dogma We disagree open minds open new approaches Using methodologies that are primarily molecular and genetic we seek to refine the causes of glaucoma as well as how it is best treated especially incorporating thoughts and hypotheses about new methods of treatment Glaucoma is a complex disease and genetics proves that a variety of proteins are culpable at one level At another level however there are likely final common pathways and numerous feedback loops which have defied explanations to date The search for answers goes on in basic science researcher s laboratories and clinical ophthalmologist s offices and operating rooms We are all well served by understanding that glaucoma is a neurodegenerative disease Current attempts to solve the disease have focused on two strategic arenas the trabecular meshwork function and its impact on intraocular pressure as a major risk factor for the disease and the optic nerve dysfunction leading to visual loss Genetic mutations have yielded puzzling clues to the cause but without resolution For example mutations in myocilin and optineurin genes are closely connected to the phenotype but how do they cause the disease In the next two years priority areas of research are signaling pathway discoveries biomarker panels epigenetic factors and continued genomic studies to yield answers to the common final pathways of the disease The final pathways are complex and redundant such that the overlap of bio informatics will be challenging Current promising leads suggest the innate immune system holds important clues to both trabecular meshwork and optic nerve pathophysiology When the primary open angle glaucoma disease pathways are unraveled drug discoveries

and new treatment modalities will be available for better regulation of intraocular pressure and neuroprotection for the optic nerve This volume discusses the glaucoma pipeline from several perspectives as well as future candidate classes As always the authors herein are urged to speculate on how the cure of glaucomatous optic nerve damage will yield to new treatments John R Samples Clinical Professor Elson S Floyd College of Medicine Washington State University School of Medicine www.glaucomaconcepts.com Paul A Knepper Associate Professor of Ophthalmology Feinberg School of Medicine Northwestern University Medical School Research Scientist University of Illinois at Chicago **Forthcoming Books** Rose Arny,1999

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Table of Contents Optic Basics P 2 Stephen Murray Answers

1. Understanding the eBook Optic Basics P 2 Stephen Murray Answers
 - The Rise of Digital Reading Optic Basics P 2 Stephen Murray Answers
 - Advantages of eBooks Over Traditional Books
2. Identifying Optic Basics P 2 Stephen Murray Answers
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Optic Basics P 2 Stephen Murray Answers
 - User-Friendly Interface
4. Exploring eBook Recommendations from Optic Basics P 2 Stephen Murray Answers
 - Personalized Recommendations
 - Optic Basics P 2 Stephen Murray Answers User Reviews and Ratings
 - Optic Basics P 2 Stephen Murray Answers and Bestseller Lists
5. Accessing Optic Basics P 2 Stephen Murray Answers Free and Paid eBooks
 - Optic Basics P 2 Stephen Murray Answers Public Domain eBooks
 - Optic Basics P 2 Stephen Murray Answers eBook Subscription Services

- Optic Basics P 2 Stephen Murray Answers Budget-Friendly Options
- 6. Navigating Optic Basics P 2 Stephen Murray Answers eBook Formats
 - ePub, PDF, MOBI, and More
 - Optic Basics P 2 Stephen Murray Answers Compatibility with Devices
 - Optic Basics P 2 Stephen Murray Answers Enhanced eBook Features
- 7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Optic Basics P 2 Stephen Murray Answers
 - Highlighting and Note-Taking Optic Basics P 2 Stephen Murray Answers
 - Interactive Elements Optic Basics P 2 Stephen Murray Answers
- 8. Staying Engaged with Optic Basics P 2 Stephen Murray Answers
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers Optic Basics P 2 Stephen Murray Answers
- 9. Balancing eBooks and Physical Books Optic Basics P 2 Stephen Murray Answers
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection Optic Basics P 2 Stephen Murray Answers
- 10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
- 11. Cultivating a Reading Routine Optic Basics P 2 Stephen Murray Answers
 - Setting Reading Goals Optic Basics P 2 Stephen Murray Answers
 - Carving Out Dedicated Reading Time
- 12. Sourcing Reliable Information of Optic Basics P 2 Stephen Murray Answers
 - Fact-Checking eBook Content of Optic Basics P 2 Stephen Murray Answers
 - Distinguishing Credible Sources
- 13. Promoting Lifelong Learning
 - Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
- 14. Embracing eBook Trends

- Integration of Multimedia Elements
- Interactive and Gamified eBooks

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