ART OF THE CLINICAL ENCOUNTER
2009 - 2010

UNIVERSITY OF CINCINNATI
DEPARTMENT OF FAMILY MEDICINE
AND
THE CINCINNATI ART MUSEUM

Instructors:

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Emily Holtrop (The Cincinnati Art Museum)

Goal:

To improve communication and observational skills utilized in the patient-doctor relationship by guided instruction in observation, description and interpretation of the visual arts.

Overview:

Students will participate in six sessions at the Cincinnati Art Museum with CAM educators and Family Medicine Faculty and two lunchtime session in the Health Professions Building (HPB.) Students will examine paintings, sculptures, and photographs in the CAM collection, followed by facilitated discussions focusing on description and interpretation gleaned from their observations.

Students will also spend ½ day per month with Dr. Hartmann, Dr. Elder, Dr. Saxena or a faculty mentor at their respective clinical sites. In the patient care setting students will record their descriptions and observations of their patient encounters as outlined below. In addition, students will sketch one patient they observe. From the descriptions collected that day, each student will choose one patient description to develop according to the guidelines listed below. Each student will complete a minimum of 3 clinical descriptions incorporating the techniques of description, interpretation, and reflection as presented in the Art Museum sessions. These will be e-mailed to the course directors and serve as the basis for the lunchtime discussions at the HPB.

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Objectives:

At the end of this elective, the student will be able to

1. Apply observational skills learned in the visual arts sessions to the clinical setting.
2. Contrast the terms **description** and **interpretation** as they apply to the visual arts and the clinical encounter.
3. Identify one's own biases and perceptions as integral elements of interpretation through examination of the cultural, ethnic, age and gender context of subjects depicted in photographs and portraiture.
4. Understand how the physician's interpretation of the patient's mood, affect, position and body language impacts the clinical encounter.
5. Using the visual arts as a model, describe how the components of observation and interpretation form the basis for response and reflection in the clinical setting.

Art Museum Session overview:

Each session at the Cincinnati Art Museum will take place on a Friday from 3:30 p.m. – 5:00p.m. Pre-selected works of art will be used during each session. Discussion will focus on descriptive and interpretive skills. As part of every session, each student will write a brief description of a presented work to be read and discussed. Clarity, accuracy and description will be emphasized. Students may also be asked to sketch a work emphasizing descriptive and observational skills.

Clinical Sessions

Students will attend one monthly ½ day session with Drs. Hartmann, Elder, Saxena or a faculty mentor. For the first 3 months of the elective, students will record a **description** of each patient encounter in the provided steno notebook. For the following 3 months students will document a **description** and key **interpretative** aspects of the encounter. During the final 3 months of the elective students will **describe, interpret** and **reflect** on aspects of each patient encounter experience. At the end of each clinical session, the student will choose one encounter to more fully document and turn in to the course directors. Students will also sketch one patient per session to practice their visual skills.

Discussion Sessions

Two discussion sessions will be held throughout the year, one evening session and one lunchtime session in room 153 of the Health Professions Bldg. The descriptive exercises turned in after each clinical session will serve as the basis for our discussion.

For our final session, we will watch a video presentation of *WIT*, perhaps the ultimate drama in clinical observation and self- reflection. A discussion will follow.

Session A: Observation and Description
Friday, October 16, 2009
3:30 – 5:00 p.m.
Cincinnati Art Museum

This session will provide a framework for the class, its objectives, and strategies. Students will also begin to learn the skill of description.

- Introduction to Cincinnati Art Museum, history and collections.
- Students will examine their own process of thinking when looking at a patient and/or an artwork.
- Students will discuss the value of description and set some guidelines for analyzing visual information in a descriptive way.
- Discussion of the use of descriptive terms vs. interpretive terms.
- Students will together examine a painting from the Cincinnati Art Museum using only descriptive terms.
- Students will break up into pairs to analyze chosen works from the Museum collection.
- Follow-up discussion.

Session B: Discussion of Clinical Observations
Friday, November 20, 2009
3:30 – 4:45 p.m.
Department of Family Medicine, Health Professions Building (HPB)

- Observational and reflection notes from the students clinical sessions will be collected and reviewed prior to these sessions.
- Verbal description exercise.
- Discussion will center on common themes and subjects from the clinical sessions centering on patient-doctor dynamics.

Session C: Bridging Description and Interpretation
Friday, December 18, 2009
3:30 – 5:00 p.m.
Cincinnati Art Museum

This session will focus on how we create interpretations based on our observations.

- Recap of previous class
- Discussion of the foundation of interpretative skills
- Partner exercises

Session D: Interpretation
Friday, January 15, 2010
3:30 – 5:00 p.m.
Cincinnati Art Museum

This session will focus on interpreting a work and how observation/description plays a part in this process.

- Recap of previous class.
- Discussion on interpretation and visual clues.
- Students will be paired up to look at the same artwork, but write their own descriptive interpretation of it.
- Students will reconvene with their partner and compare interpretations. How are they the same, how are they different? What do we learn about ourselves through these interpretations?
- Students then find a second painting, examine and interpret it. The group reconvenes and each student presents their descriptive interpretation to the group.
- Follow-up discussion.

Sessions E: Interpretation and Emotional Response
Friday, February 26, 2010
3:30 – 5:00 p.m.
Cincinnati Art Museum

- Recap of previous class.
- Continued discussion of interpretive skills and how they relate to the way we view/understand people.
- Examination of photographs of people. Students will be asked to use their descriptive/interpretive skills to understand as much of the person as they can.
- Students present their findings. The class will discuss different ways of engaging with the person in each photograph.
- Follow-up discussion

We will begin to discuss bridging interpretation with the observer's emotional response. How do emotional responses in the physician impact the clinical encounter?

Session F: Discussion of Clinical Observations
Wednesday, March 3, 2010
12:00-1:00 p.m. Lunch
Room 153 Health Professions Building

This session will focus on mood, posture, body language as these factors reflect the psychological state of the subject and the artist. Students will attempt to understand what emotions are conveyed through each artwork.

- Verbal description exercise.
- Discussion about moods and how artists use body language to communicate emotions. View works in the galleries that express different moods such as:
  - Andrea Mantegna, *A Sibyl an a Prophet*
  - Benjamin West, *Ophelia and Laertes*
- Discussion on how one might understand the psychological mood of the artist through the artwork and how those techniques can be applied in the clinical setting
- Group examination of selected photographs and continued discussion of mood.
- Follow-up discussion.

Session G: Emotional response
Friday, March 12, 2010
3:30 – 5:00 p.m.
Cincinnati Art Museum

- Examination of how we emotionally respond to works of art, what tools artists use to convey moods and feelings.
- Students write about an abstract expressionist work, paying attention to the mood and feeling evoked by the painting.

Session H: Emotional Response and Reflection
Friday, April 2, 2010
3:30 – 5:00 p.m.
Cincinnati Art Museum

This final session will focus on our own emotional response to artworks.

- Recap of last session.
- Verbal description exercise.
- Discussion recapping the main points of the last 5 sessions. What have the student's learned? How will that affect their patient relationships and doctor-doctor communication? How has clinical component aided in their learning? Discussion of Van Gogh *JAMA* articles.
- Examination of how we emotionally respond to works of art, what tools artists use to convey moods and feelings.
- Students write about an abstract expressionist work, paying attention to the mood and feeling evoked by the painting.

Session I: Viewing of WIT
TBA

We will watch a video presentation of *WIT*, perhaps the ultimate drama in observation and self reflection. A discussion will follow.

Overall, you'll notice that the curriculum as it is follows this basic progression: **description, interpretation, emotional response**. This is the basic way in which our brains process the world (in my opinion), but usually we only recognize the last element (**emotional response**). Let me give an example. Say, you come across a bloody car wreck. You immediately feel the **emotional response** of what you see (fear, shock, disgust, empathy, etc). The emotional response may be the first thing you feel, but that **emotional response** is the result of your **interpretation** of what you see. For example, you interpret this scene to be a car wreck in which the persons inside have suffered great pain. In turn, that **interpretation** is the result of your **description** of the elements that you experience through your senses (sight, sound, smell, etc). In this case, those elements might have been a mangled car, bloody bodies, etc.

(Sorry about the gruesome imagery here. I'm working from an image of an Andy Warhol screen-print of a car wreck in my mind.)

Anyway, the point here is to learn how we come to conclusions. All this happens instantaneously in most cases. We appear to go straight toward the **emotional response** because our brains process the information so quickly. By stepping back and understanding how we describe and interpret and emotionally respond, as well as developing better skills to do each of those three things, we can help the medical students pay closer attention to the way they are processing their information, coming to certain conclusions (which includes their own biases), and improve the manner in which they process this information (i.e. process the patient). Ultimately, if they are more skilled in accessing a variety of information from their patient and more skilled at making meaningful connections with their patients, not only can the doctor-patient relationship can be strengthened but also their diagnostic skills. The former is because patients generally do better if they feel that they are working in partnership with their doctor. The latter because the doctor will be taking a variety of information sources into account (rather than jumping to a diagnosis based solely on a set of lab results), including socioeconomic status, age, mood, ethnicity, lifestyle, motivation, etc.

So, here is a basic idea of how the museum sessions were run, complete with the activities that accompanied each one.

(Throughout the class, the students also observe with the doctors and record their observations and interpretations. Sometimes we ask them to draw patients too. Their notebooks of observations are discussed during three lunchtime sessions that occur at the University. Drs. Tobias and Elder usually facilitate these sessions.)

Session I: Description

In this initial session, we did the normal introductory exercises in getting to know each other. I also took the opportunity to explain a bit about the museum's history and collection of objects. It's also a good chance to cover the basic rules of the museum. For example: use only pencils, no touching the objects including museum walls and vitrines. Also, you can cover the rules of the class, which the main rule is the never read any label, not even the artist's name, artwork title, date, etc. It's also a good time to discuss the need for this class. Why are they attending? What do they want to get out of it? What's the purpose? I also liked to determine if the students have any recreational arts background or not.

The main purpose here is to help the students develop descriptive skills (observation and description are closely related here and I sometimes interchange the words). I often start with the Felix Gonzalez-Torres word portrait just to illustrate the idea that descriptions are basically the transformation of images into

words. Gonzalez-Torres's word portrait is just that. He took the image (real and figural) of the museum and broke it down into words. Conversely, when we as viewers look at the words in this artwork, we reconstruct an image of the museum. This is similar to a patient's chart. Let's say Dr. Mike Horton (from Days of Our Lives) sees a patient in the hospital and creates a patient chart for him. Before Dr. Marlena Black ("Doc") goes in to do a psych evaluation of that patient, she's going to read that patient chart, which will include age, gender, weight, vital stats, family history, and perhaps some personal comments from Dr. Horton (such as "appears older than stated age"). Sometimes race is included in the patient chart, but this is a touchy subject. Ask Dr. Tobias why. Anyway, as Doc is reading the patient chart, she is simultaneously constructing an image in her head of that person, which may or may not match what she encounters in the hospital room.

(We also do this with blind dates. Think about the setup person describing the potential date. He/she describes the potential date to his/her friend, breaking down that person's image into words. The listener then reconstructs the image in his/her head based on these words.)

An important aspect of this session is to teach the students understand the line between description and interpretation. It is similar to the line between fact and opinion. Description consists of solely understanding what we see. Interpretation is when we take these factors and combine them to produce a meaning behind their relationship. For example, a patient's symptoms and lab results are the descriptive elements (i.e. facts). The doctor's diagnosis is the interpretation (opinion). Too often, doctors jump straight to diagnoses without fully digesting all the factors involved. These first too sessions help them to step back and look more deeply at all the information they are presented with.

In the context of art, the simple objects in the image are the elements to be described. Making meaning of them is an interpretation. For example, part of a description might include something like "Presented are what appears to be a Caucasian male and what appears to be a Caucasian female. Both are without clothes although the genitals of each are covered with a leaf." Part of an interpretation might be "This is Adam and Eve."

(By the way, race is an interesting issue in this class. You'll notice that the students will only identify the race/ethnicity of a subject when it is something other than their own. Work with Dr. Tobias to determine the best time to bring up the issue of race. It can be a lengthy discussion.)

After walking through a painting together to just describe what is seen, I usually then split the group up and choose a painting for each of them to describe on their own. They worked independently on this exercise and then reconvene to present their descriptions to the group. For this initial session, I usually use works in the Cincinnati Wing. During the presentations, the students should pay attention to describing the size of the work (this is important because they are objects too, not simply images) and finding a focal point in the work, around which all the descriptions will be made. This helps to orient the listeners and helps the student to make sense of all the activity in the work. The student should try to describe as much detail as possible, including the background of the image and all the details that you and I might find tedious: description of clothes, colors of objects, jewelry, body positioning, the location of objects in relation to other objects, etc, etc. Everything. Basically, they should aim to describe these paintings as if they were describing them to someone on the phone (i.e. someone who can't see the painting). The person on the other end of the phone should be able to reconstruct the image based on these highly detailed descriptions. But all should be careful not interpret any aspect of the work. For example, Horace Pippins painting includes brightly packaged boxes. In this exercise, they are not Christmas gifts.

Session II: Description

This is a continuation of the first session, but is more focused on building the student's descriptive vocabulary. As in all the sessions, I have the students break up to analyze a painting/sculpture on their own and then present their descriptions to the class, but in this case they are asked to describe abstract, non-representational works (in the 20th century galleries). I only selected paintings/sculptures in which the subjects couldn't be identified (like the Hofman, for example). So now, the students have to figure out ways to describe things that have no names. The students again have to figure out ways to describe the abstract shapes, etc, as if they were talking to someone on the phone. One technique that can help them is the use of similes. They can determine what something looks like to help the listener make sense of what they are seeing. For example, in the de Kooning work, the students often describe the main shape as something that looks like Casper the Friendly Ghost. This is a fine tool. In a similar way, doctors often describe the appearance of a skin condition (chicken pox or mumps, I can't remember) as "dewdrops on rose petals". Again, the students should find a focal point in which to orient the listener and work their descriptions around that.

One activity that I sometimes did to help teach the art of simile was to have the group look at the Arp painting and, in rapid-fire, ask them to describe the main shape in terms of something that it looks like. The answers come quick and each time you understand the shape differently: a girl with three ponytails, a goose in mid-flight, a wheel with three spokes, three sperm trying to impregnate an egg. It's important to note that none of these are interpretations; all are simply ways to help describe something unfamiliar with familiar terms.

Session III: Interpretation

This is when things get interesting. You'll notice some frustration from the students as they limit themselves to solely presenting descriptions. This is somewhat unnatural for us to do in real life because our brains are constantly interpreting what we see. But it's good for them to understand the basis of those interpretations to begin with. But now, they get to move ahead with interpretation.

It should be noted that the students should still begin with descriptions before interpreting anything. In this exercise, I divide the group into pairs and give them each a narrative painting to describe from the Dutch, French and English galleries. Here are some of the ones that I've found best to work with:

Lancret, *The Swing*

Bol, Paloma (?) and Vertrumnus

Ter Borch, *The Music Party*

The Italian Comedians (the artist's name is escaping me here)

Each pair must interpret the painting, but do so independently and without talking to his/her partner at all. They are also not allowed, of course, to read the title of the work or any part of the label. They can be as crazy as they want in their interpretation of the painting before them, as long as they can justify the interpretations based on what they see (describe). Some students create elaborate stories for the paintings, complete with dialogues and all.

During the presentations, the students should begin with their description of the painting and then move into their interpretation. What you'll find is that the partners will sometimes have vastly different interpretations of the exact same work. And this helps to illustrate the relativity of interpretation (and of diagnoses), that we can take the exact same source of information and extract different meanings from it. During the interpretation presentations, be sure to ask the students what evidence they used to come up with particular aspects of their interpretation. By now they should be skilled at observing and describing everything and that's where their interpretations should be coming from. Sometimes you may notice that an interpretation might come from a recent event or particular interest they have (for example, someone in every class interprets the Bol painting as an attempted abortion. Only medical students do this.). Determine the sources of these interpretations so that you can illustrate how our particular lives create a unique personal lens through which we interpret the world.

Session IV: Interpretation

This is a continuation of the first class on interpretation, but one more focused on people and situations that are more true to life. Therefore, for this session, I always pull photographs from storage. Dennis always seemed to remember the photographs that I liked to use, so I never really wrote down the titles/artists of them. (sorry!) But since this is your class now anyway, maybe its best to start fresh.

My focus for artwork in this session were narrative photographs (i.e. photos that tell some sort of story), in the same manner that the paintings in the previous session were all narrative paintings. (Narrative art obviously works best for teaching interpretive skills.) Each student was given a photograph of their own to interpret, not in the somewhat fantastical manner of the previous class, but more in real-world sort of way. Asking them to answer questions such as Who is this person/these people?, What's the situation that has brought these people together?, What time period is this? What's the relationship of each person to the other? What do you know about this person/these people? What's their age, race, socioeconomic status, etc? What's going on in the photo? Again, the idea here is to develop their interpretive skills, putting ideas and relationships together to form a greater understanding of the image.

With regards to something age, ask the students to support why they think a person is of a certain age. What is their reference point? Most times, a student thinks an elderly woman in a photograph, for example, is XX age because she appears to be the same age as the student's grandmother. Therefore, that's the student's reference point. But it's important to stress that different people age different, depending on factors such as race, ethnicity, socioeconomics, health and eating habits. Reference points can be fine. The student just needs to be aware of their weaknesses.

You'll need to receive some art handling training from Cecile on how to handle works on paper before doing this class. I also go over basic rules with the students before this class: no touching the photographs (not even the mats), only use pencil, and, if you are going to sneeze, turn the hell away from the photograph!

Session V: Emotional Response

Emotional response is a little harder to define and understand, but it's something that plays a huge role in how we interpret and react to stimuli. Understanding what are emotional responses are and why they are the way they are help us to discover our own biases in interpretation. For example, Dr. Elder gives a great example of how she has a negative reaction toward her patients that smoke. She believes her reaction is this way because her own mother died of lung cancer. Therefore, she has difficulty with

patients that smoke. This emotional response has made her aware of her own bias. ("Bias" here doesn't necessarily mean a student is prejudiced or anything. It just means that certain factors make us have particular emotional responses.)

For this session, I always pulled out photographs of different types of people, mostly portraits. The photo of a girl with polio is always a good example. Others too you will find will elicit different responses. I would ask each student to think about the person in the photograph. How do you feel about this person? Who does this person remind you of? What are you concerned about in this person? What types of questions would you ask? Again all of this is designed to help the students think about their own reactions to the people they will encounter, which in their careers may range from the super rich to the homeless. Understanding how we react to people can help us understand ourselves and, as emphasized in this class, help us to remove certain barriers that might prevent us from connecting more thoroughly to them.

Session VI: Emotional Response

For this last session, the students should read a selection of medical articles about Vincent van Gogh. Dr. Tobias will give them the articles. Each is written by a different specialist and each attempts to posthumously diagnose what the artist suffered. The first part of this class was devoted to a recap of these articles. Eventually, the students learn that the theory that van Gogh suffered from glaucoma was written by an ophthalmologist, the wormwood poisoning theory written by a toxicologist, and etc, etc. The point here is that the specialists are exhibiting their own biases by thinking that van Gogh suffered from something that, coincidentally, falls within their specialties.

This is a good segway (How do you spell that word? My computer is programmed to spell-check only spanish.) into talking about our van Gogh painting. Asking the students to interpret the emotional tone of the painting, they will undoubted find morose feelings. It's helpful to encourage the students to ask themselves why they would interpret a painting of two people walking in a wooded field of flowers as something sad. Certainly there are visual aspects of the painting that lend themselves to this interpretive direction: the absence of faces, the immobility of the figures, the jail-like trees, the dark background, etc. But how much of van Gogh's biography (which the students will be well aware of after reading all those medical articles) plays a role in our emotional response? I suspect it plays a large role. It's difficult to separate what we know of van Gogh from how we see his paintings. Prior knowledge is a huge bias, which can be positive or negative, in our emotional response to paintings or people. For example, a person's family history of diagnosis can give a doctor good information for determining a diagnosis. Or it can predispose a doctor toward certain assumptions. Say a patient is in pain. But before the doctor sees him, he reads his chart and learns that the patient has a past history of drug abuse. All of the sudden, the doctor assumes that the patient is seeking painkillers not for a real condition but for the purpose of abuse.

We do this with everyday people too. Say you are going to meet your friend's new boyfriend. But before meeting him, your friend has already told you that he was once jailed for beating a former girlfriend. This prior knowledge will undoubtedly color your emotional reaction to him.

Toward the end of this discussion, I relay the information that this painting was one of the last paintings, if not the last, that van Gogh painted before committing suicide. Then I ask again if their emotional response to it has changed. Then I ask if they will ever be able to look at this painting again without

thinking about the fact that he committed suicide shortly after it was painted. Chances are, they won't be able to separate this information (this is why I wait to the end of the discussion to share this fact).

After this exercise, we move into the Ab Ex and Modern works. I think you've been with me during this class before. Before sending them off to think about their own emotional responses to these works (again, a process of looking inward), I have a little discussion with them about abstract art. This is to help eliminate the negative questions that always color their responses. Why is this art? Couldn't this person paint realistically? Why am I supposed to like this? Who decides to buy this art and put it in a museum?, etc, etc. I've found it necessary to tackle these questions upfront. Otherwise, everyone's emotional responses to the abstract works will simply be negative, not because that's how they are responding to something specific in the painting, but rather because they don't agree that this is art.

So, I encourage them to pay attention to the formal elements of the painting and to look within to discover how they emotionally respond to the colors, shapes, lines, size, movement, etc. The more introspective they can be, the more consciously aware of their emotional responses they will be with their patients. Or, at least, this is what I hope.

CHAPTER 2

Lessons from the Heart

ON A SPRING AFTERNOON several years ago, Evan McKinley was hiking in the woods near Halifax, Nova Scotia, when a pain in his chest stopped him in his tracks. McKinley was a forest ranger in his early forties, trim and extremely fit, with straw-blond hair and chiseled features. He had had a growing discomfort in his chest for the past few days, but nothing as severe as this. He wasn't sweating or lightheaded, and didn't feel feverish. But each time he took a breath, the pain got worse. McKinley slowly made his way back through the woods to the shed that housed his office. He sat and waited for the pain to pass, but it didn't. As a forest ranger, he was used to muscle aches from scaling a steep rocky trail or jogging with a loaded pack on his back. But this was different, and he decided he should see a doctor immediately.

As it happened, Dr. Pat Croskerry was working in the emergency department that day. He took McKinley's measure: a wiry, muscular man wearing the distinctive bright olive bomber jacket and pants, much like an American park ranger's uniform. McKinley's face was ruddy, as would be expected of someone who spends most of his day working outdoors, and his brow was free of per-

spiration. Croskerry listened intently as McKinley described how his chest pains had increased over the past few days and how they had worsened today. Croskerry questioned him further to get a more precise description of his symptoms. McKinley said the pains stayed in the center of his chest but did not move down his arms, into his neck, or through to his back. The pain got no worse if he changed position, and even taking a really deep breath didn't make him feel faint.

Croskerry went over a checklist of risk factors for heart and lung disease. McKinley had never smoked and had no family history of heart attack, stroke, or diabetes. He laughed, as Croskerry did, when Croskerry used the term "sedentary lifestyle." McKinley added that he felt under no particular stress, his family life was fine and he loved his job, and he had never been overweight. Croskerry then did a physical examination. First he verified that the vital signs recorded by the triage nurse were correct. McKinley's blood pressure was 110 over 60, his pulse 60 and regular, as would be expected of an athletic man. Croskerry listened with particular care to McKinley's lungs and heart, especially when he took a deep breath, but everything sounded fine. His muscles were well developed, and when Croskerry pressed on the junction between McKinley's ribs and breastbone, McKinley felt no pain. There was no swelling or tenderness in his calves or thighs. Finally, the doctor ordered an electrocardiogram, a chest x-ray, and blood work that would include tests for oxygen level and cardiac enzymes that indicate heart damage. As he expected, all of these were normal.

"I'm not at all worried about your chest pain," Croskerry told McKinley. "You probably overexerted yourself in the field and strained some muscle. My suspicion for this coming from your heart is about zero." Deeply reassured, the forest ranger went home.

The next morning, Croskerry was off duty, and read part of a

novel that he was keen to finish. He is an avid athlete and rowed on Canada's 1976 Olympic crew in Montreal. He stays in shape, and that day he had jogged four miles around the Halifax harbor. When he arrived in the emergency department in the early evening, he bumped into a colleague. "Very interesting case, that man you saw yesterday," the doctor said. "He came in this morning with an acute myocardial infarction."

Croskerry was stunned. He reviewed his notes on the emergency room chart. The colleague tried to reassure him. "If I had seen this guy, I wouldn't have gone as far as you did in ordering all those tests." But Croskerry found this cold comfort. It was not because he expected to be infallible. Rather, he recognized that he had made a common cognitive error that could have cost the forest ranger his life. "Clearly, I missed it," Croskerry told me after recounting McKinley's case. "And why did I miss it? I didn't miss it because of any egregious behavior or negligence. I missed it because my thinking was overinfluenced by how healthy this man looked." Croskerry's voice faltered for a moment. "Happily, he didn't die."

Chest pain is the second most common reason for a patient to visit an emergency room (abdominal pain is number one). Each year in the United States and Canada there are more than six million evaluations in the ER of patients like McKinley. But despite its frequency, chest pain is one of the most challenging symptoms for the clinician to unravel. In retrospect, Croskerry realized that when he saw Evan McKinley, the ranger was in the midst of unstable angina — a crescendo of chest pain, caused by coronary artery disease, that usually prefigures a heart attack. "The unstable angina didn't show on the EKG, because fifty percent of such cases don't," Croskerry said in a voice that sounded to me as if he were lecturing himself. "His unstable angina did not show up on the cardiac enzymes because there wasn't yet injury to the heart muscle, and it didn't show up on the chest x-ray because the heart

had not yet begun to fail to pump blood, so there was no fluid backup into the lungs."

The mistake Croskerry made is called a representativeness error: your thinking is guided by a prototype, so you fail to consider possibilities that contradict the prototype and thus attribute the symptoms to the wrong cause. Croskerry told me how his eyes had fixed on McKinley's trim frame and his elegant olive uniform, and how the ranger's physique and chiseled features reminded him of a young Clint Eastwood — all strong associations with health and vigor. Yes, there were unusual aspects to McKinley's angina; his pain was not typical of coronary artery disease, nor did the physical examination and tests point to the heart. But, Croskerry emphasized, that was precisely the point: "You have to be prepared in your mind for the atypical and not so quickly reassure yourself, and your patient, that everything is okay." When Croskerry now teaches students and interns about such errors, he uses Evan McKinley as an example.

More commonly, doctors make what are called attribution errors when patients fit a negative stereotype. Dr. Donald Redelmeier of the University of Toronto, who, like Croskerry, studies physician cognition, told me about a case he had recently seen on rounds. Charles Carver was in his seventies, retired from the merchant marine and living by himself in a small apartment. Over the past months, he had felt fatigued and his belly had begun to swell. When Carver came into the ER, the intern noticed alcohol on his breath, and Carver readily told him that he enjoyed a glass of rum each evening. His legs and feet, as well as his abdomen, were swollen. Carver was unshaven; his clothes were old and frayed. The intern wondered to himself how many days it had been since he bathed.

The initial presentation to Dr. Redelmeier on rounds was terse. "Charles Carver, a seventy-three-year-old retired merchant mariner, with a long history of alcohol ingestion, presents with in-

creasing fatigue and fluid retention." The intern palpated Carver's liver and told Redelmeier that it was enlarged, hard, and nodular. Redelmeier began to quiz the intern about Carver's problem. It soon became apparent that the trainee had in mind one and only one possible diagnosis: alcoholic cirrhosis. Redelmeier asked the medical team to offer other explanations for Carver's problems. He could see in their eyes that they felt burdened, that he was wasting precious time on rounds when they could be discussing much more interesting cases than that of an old, foul-smelling, rum-swilling sailor. "The intern's plan was to have this boozer sleep it off, give him some mild diuretics, and send him home as quickly as possible," Redelmeier told me.

"You are filled with a sense of disgust," Redelmeier said when we discussed the kinds of feelings that a man like Charles Carver summons in a doctor. That disgust pushes you away from him. Of course, as a doctor, it is your job to diagnose and treat him properly, but, consciously or subconsciously, you want to get the job over with and send such a man on his way. In particular, doctors consider people who seem not to be caring for themselves — alcoholics with cirrhosis, heavy smokers with end-stage emphysema, massively obese people with diabetes — as to some degree less deserving of their time and attention. Or, as in the stereotype of psychiatric patients that cloaked Anne Dodge, people who are not to be believed when they say they are following the doctor's orders. Physicians like to succeed in their treatment, and an essential ingredient for that success is a patient's cooperation. One doctor told me that patients who don't care for themselves made him feel like Sisyphus.

Redelmeier himself is prone to that visceral sense of disgust. He has taught himself to recognize the feeling and, as he put it, "plant a red flag in my mind." So, on rounds that day, Redelmeier didn't back down. He pushed the interns and residents to come up with alternative hypotheses for Carver's liver disease. He insisted on

tests for unusual conditions, like alpha-1 antitrypsin deficiency, an inherited malady that can cause lung and liver disease, and Wilson's disease, another inherited disorder, in which copper deposits damage the liver and brain.

To everyone's surprise, including Redelmeier's, Charles Carver had Wilson's disease. "They said I was a brilliant clinician," Redelmeier recalled with a chuckle. "But it wasn't really brilliance. It was just forcing myself not to make an attribution error and dismiss the case out of hand as one more scuzzy alcoholic." Redelmeier added that, in fact, Carver was not an alcoholic. He enjoyed his glass of rum a day, but it really was only one glass, as Carver's daughter confirmed. Now, along with his evening drink, Carver takes copper chelators, drugs that remove the excess metal from his tissues.

Croskerry's prototypical error illustrates the opposite pole of emotion from disgust. Croskerry embodies many of Evan McKinley's characteristics himself: both are energetic, passionate men who love their work and for whom outdoor exercise is a major part of life. Powerful positive feelings about a patient are generally held to be good, the cornerstone of humanistic medicine. We all want to feel that our physician really likes us, sees us as special, and is emotionally moved by our plight, attracted not so much by the fascinating biology of our disease but by who we are as people. Usually such positive feelings enhance our relationship with our doctor and the quality of care we receive. But not always.

Doctors must be wary of "going with your gut" when what's in your gut is a strong emotion about a patient, even a positive one. Physicians understandably care deeply about their patients and want a good outcome, which can cause them to underinvestigate problems. Doctors may make decisions that stack the deck so that they draw what seems to be a winning hand for a patient they especially like, admire, or identify with. Croskerry chose to rely

on the very first set of data — the normal EKG, chest x-ray, and blood tests — all of which indicated a favorable diagnosis for McKinley. He didn't arrange for follow-up testing.

We all tend to prefer what we hope will happen to the less appealing alternatives; this natural tendency is termed "affective error." We also lull ourselves into thinking that what we wish for will occur when we get the first inkling, however fragmentary, that our wish may come true. In short, we value too highly information that fulfills our desires. This kind of error can affect even a consummate clinician like Pat Croskerry.

The case of Evan McKinley brought me back to my conversation with Dr. Myron Falchuk. After Falchuk told me about Anne Dodge, I asked him if he had misdiagnosed a patient recently. His face fell for a moment. Then he told me about an elderly Jewish man he had seen earlier that year. "He was a wonderful, delightful character from the old country," Falchuk said. Joe Stern was in his late eighties but still spry, driving himself around Brookline and taking adult education classes. Stern complained of indigestion, specifically heartburn, for several weeks. Such symptoms are common; a general practitioner or internist usually treats them. But Falchuk knew the Stern family, and so took Joe on as his own patient. Over the course of four months, he treated him with antacids and other medications. The treatments gave him only slight relief.

Falchuk found himself enjoying Joe Stern's company so much that he ran over the allotted time at each visit. "He had a great sense of humor, and we kibitzed together in Yiddish," Falchuk recalled. "We really connected. I said to myself, Do I really have to put him through invasive tests? So I just kept adjusting his medications over four months." Falchuk paused. "Then he came in saying he felt faint and exhausted, and it was clear that something was different. He had become anemic." Falchuk performed an upper endoscopy, the same procedure he had done on Anne

Dodge, snaking a fiberoptic instrument down Stern's throat and into his esophagus and stomach. What he saw was not subtle: large growths with the characteristic pleated appearance of gastric lymphoma. A biopsy confirmed the diagnosis. The cancer clearly had been there all the time, and accounted for Stern's persistent indigestion and acid reflux.

"It's a treatable cancer," Falchuk said, "but I kicked myself over and over again. I just didn't want to subject someone of this age, whom I liked so much, to the discomfort and the strain of the procedure. And because of that, I missed the diagnosis." Fortunately, as with Evan McKinley, the ultimate outcome was good. The delay in diagnosis did not harm Joe; he went into remission. After Falchuk finished, I told him of a case of my own from many years ago: the case of Brad Miller.

Ever since he was a little boy, Brad Miller loved to run. His mother joked that it didn't matter when or where, even if he didn't have sneakers on. Growing up in Southern California, he would jog three miles to school, and on weekends he'd take the bus from Culver City west to the beach and sprint in the warm sand. Brad went east for college. He was undeterred by the sleet and broken sidewalks of New Haven, running each day in a wide arc from the university to the train station and back. Brad never joined the college track team, and doubted that his speed was sufficient to compete at the varsity level. But that didn't matter, because running just seemed to be a part of him. All through the stresses of college and graduate school, Brad used running as his tonic. He returned to Los Angeles with his doctorate in hand, his dissertation a meticulously footnoted study of ancient and contemporary female archetypes that influenced James Joyce's work. As a new English professor at a local college, he felt his life had taken a strong start out of the blocks.

"You look familiar," Brad said to me the first day I entered his

hospital room at the UCLA Medical Center. It was the early winter of 1979, and I was in my fellowship training in hematology and oncology. I studied Brad, but his face did not register.

"I see you running with two or three friends around the university," he said. "I'm a runner too — or at least was."

Nearly every evening, a pack of young doctors ran the hills of Westwood. The incline along Highland Avenue was particularly steep, from the hospital to the apex of the campus. It tested my stamina. "I must have been the one gasping for breath," I said. "Perhaps that's why I stuck in your mind."

Brad's smile was brief.

"We'll do everything possible to get you back running," I said. "The chemotherapy is difficult, I won't minimize that, but it can make all the difference."

About six weeks earlier, Brad had noted an ache in his left knee. At first he thought it was simply due to his intense training schedule for an upcoming marathon. But the ache did not go away with rest and anti-inflammatory medication. He saw a sports medicine physician, who examined the leg and recommended stretching and wearing a knee brace when he ran. Brad dutifully followed this advice, but the ache only seemed to get worse, the leg stiffer. The physician ordered an x-ray. He told Brad that it showed some kind of growth around the end of the femur, just above the knee. He said the problem was outside his area and that Brad should see a specialist. The doctor couldn't hide the gravity of what he saw with euphemisms.

The growth in Brad's leg was an osteosarcoma, a bone cancer. The surgical oncology department at UCLA, among the best in the country, had pioneered an experimental program for these types of sarcomas. In the past, people like Brad would have had the leg amputated, but a new chemotherapy drug, Adriamycin, had been developed that often shrank the tumor. Oncologists had nicknamed it "the red death" because of its cranberry color and its

terrible toxicity. Not only did it cause severe nausea, vomiting, blistering of the mouth, and reduced blood counts, but repeated doses could injure cardiac muscle, resulting in heart failure. Patients had to be monitored closely, since once the heart was damaged, there was no good way to restore its pumping capacity. The experimental strategy at UCLA involved treating patients with multiple doses of Adriamycin in the hope that the cancer would shrink enough to be surgically removed without amputation.

We began the treatment that afternoon. Despite medication to stave off vomiting, Brad spent several hours retching uncontrollably. Within a week, his white blood cell count had fallen precipitously. Because of this decline in his immune defenses, Brad was at great risk for an infection. To try to prevent this, we isolated him; he was visited only by people wearing a mask, gown, and gloves. His diet was changed to reduce exposure to bacteria in raw foods.

"Not to your taste," I observed, eyeing the untouched meal on his tray.

"My mouth hurts," Brad whispered. He had multiple oral ulcers from the chemotherapy. "And even if I could chew, it looks pretty tasteless."

We were giving Brad a special anesthetic mouthwash to try to alleviate the pain, but it clearly was not helping much. I agreed that the food looked dismal.

"What is to your taste? Fried kidney?"

Brad looked knowingly at me.

"Nothing like Joyce to lift the spirit."

I had told him when we met that I'd studied *Ulysses* in a freshman seminar. The professor had explained the relevant Irish history, especially Parnell and the Easter Rebellion; the subtle references to Catholic liturgy; and a host of other allusions that otherwise would have passed most of the class by. In the book, Leopold Bloom savors fried kidneys.

Brad was my favorite patient on the ward. Each morning when I made rounds with the residents and students, I would take an inventory of his symptoms, examining him to check on the medical team's findings and reviewing his laboratory results. Then I would linger, trying to raise his spirits and distract him from the misery of the therapy.

The protocol called for a CT scan after the third cycle of Adriamycin. If the cancer had shrunk sufficiently, the surgery would proceed. If it hadn't, or if the cancer had grown despite the chemotherapy, then there was little to be done short of amputation. And even after amputation, patients still live under a cloud, since the cancer can metastasize to the lungs or other organs.

Three cycles of chemotherapy took their toll on Brad. He became listless, difficult to engage in conversation. Then, one morning, he developed a low-grade fever of 100.2° F. The residents told me on morning rounds that they had already gotten blood and urine cultures, and that his physical examination was "nonfocal," medical jargon meaning that they had found no clear origin for an infection. People undergoing chemotherapy often get low-grade fevers after their white blood cell count falls; if the fever has no identifiable cause, a physician must use his judgment about when to begin a course of antibiotics.

"So you feel even more wiped out?" I asked Brad.

He nodded. I reviewed again a list of symptoms that might identify a source of infection: Did he have a headache, difficulty with vision, pressure in his sinuses, a sore throat, problems breathing? He answered no to each. Was he bringing up any sputum? No again. Any pain in his abdomen, diarrhea, burning on urination? None at all.

Brad said he was too exhausted to sit up on his own, so a resident took one shoulder and a student another and propped him up in bed. Brad had the body of a long-distance runner, tall and lean. Adriamycin dosages are based on body surface area rather

than weight, so with the large surface area of a person of his physique, Brad had been getting high doses. His remaining wisps of black hair were matted with sweat, and he was ashen.

I examined his eyes, ears, nose, and throat, and found nothing of note except some small ulcers on his inner cheeks and under his tongue, side effects of his treatment. Brad worked hard to take deep breaths when I examined his lungs — they were clear — and his heart sounds were strong, without a "gallop" indicating heart failure. His abdomen was soft, and there was no tenderness over his bladder.

"Enough for today," I said. Brad looked so peaked that it seemed wise to let him rest. He nodded his thanks.

Later that day, I was in the hematology lab, looking at the bone marrow biopsy of a patient with leukemia, when my beeper went off with a stat page. "Brad Miller has no blood pressure," the resident reported when I called. "His temperature is up to 104, and we're moving him to the ICU."

Septic shock. When bacteria spread through the bloodstream, they can shut down the circulation. This can be fatal even in people who are otherwise healthy, but patients with impaired immunity, like Brad, whose white blood cell count has been lowered by chemotherapy, often die.

"Do we have a source?" I asked.

"He has what looks like an abscess in his left buttock," the resident said.

Patients who lack the white cells to fight bacteria are prone to infections at sites that are routinely soiled, like the area between the buttocks.

I fell silent as I replayed in my mind the scene on rounds with Brad that morning. The abscess had certainly been there a few hours before. "Enough for today," I had said. *Not enough at all.* I had failed to ask him to roll over so I could examine his buttocks and rectum.

"We repeated his cultures and began broad-spectrum antibiotics," the resident said. "The ICU team will take over."

"Okay. Good job." As I hung up the phone, I berated myself further. *Bad job. Sloppy job.*

My heart had ached for Brad, and that deep feeling had caused me to break discipline. Normally, I had a system that I followed with every immune-deficient patient every day, beginning at the crown of their head and working down to the tip of their toes, examining every cleft and fold and orifice and organ. I had not wanted to add further to Brad's discomfort. I left the bedsheets on him. That could prove to be a fatal mistake.

I attended to the day's remaining tasks and rushed to the ICU as soon as I was free. Brad was on a respirator and opened his eyes wide to signal "hello." In addition to saline, he was receiving pressors, drugs that increase the contraction of the heart and the tone of the vessels to try to sustain the blood pressure. His heart was holding up now despite all the Adriamycin. His platelet count had fallen, as often happens in septic shock, and he was receiving platelet transfusions. The senior doctor in the ICU had already told Brad's parents how serious his situation was. I saw them sitting in a room next to the ICU, their heads bowed. At first I considered walking by, since they had not seen me, but I forced myself to go in and offer a few words of encouragement. They thanked me for my care of their son.

After a restless night, I arrived early the next morning before the residents on the ward to review all the charts of my patients. Rounds lasted an hour longer than usual, as I checked and double-checked every bit of information the team offered. I could see them growing restless, but I needed to reclaim my balance and this was the only way I knew how.

Brad Miller survived. Slowly his white blood cell count increased, and the infection was resolved. After he left the ICU, I told him that I should have examined him more thoroughly that

morning, but I did not explain why I failed to. His CT scan showed that the sarcoma had shrunk enough for him to undergo surgery without amputation. But a large portion of his thigh muscle had to be removed along with the tumor. After his surgery, running was too demanding. Occasionally I would see Brad riding his bicycle on campus, and I gave silent thanks each time I did.

One of the most celebrated statements in clinical medicine comes from a lecture delivered by Dr. Francis Weld Peabody of Harvard Medical School in 1925: "The secret of the care of the patient is in caring for the patient." This is undoubtedly true, but less obvious than it may seem. Peabody cautioned doctors about the way their training conditions them. Of necessity, we learn to suppress our emotions, to block our natural reactions to many of the awful things we see and the brutal things we must do.

Consider what happens in the ER when we try to save the life of a person smashed by a car or burned in a fire. If a doctor thought too much about the person before him, he couldn't insert his gloved hands into a hemorrhaging abdomen or maneuver a breathing tube past charred flesh. Even in less desperate circumstances — giving chemotherapy to a young woman with widespread breast cancer, say, or inserting a dialysis shunt into the arm of a blind diabetic whose kidneys have failed — we have to detach ourselves from anguish that could impede our work. But to become immune to feeling, as Peabody indicated, is to diminish the full role of the physician as a healer and relegate him to a single dimension of his job, that of a tactician. If we feel our emotions deeply, we risk recoiling or breaking down. If we erase our emotions, however, we fail to care *for* the patient. We face a paradox: feeling prevents us from being blind to our patient's soul but risks blinding us to what is wrong with him.

I asked Dr. Karen Delgado about this paradox. Delgado is an

acclaimed specialist in endocrinology and metabolism at a large urban teaching hospital who cares for patients with hormonal and metabolic disorders such as diabetes, infertility, and hypothyroidism. To my mind, she is the very model of a doctor, deeply knowledgeable about medical science and compassionate, empathetic, and generous with her patients. When I asked Delgado whether she had ever made an attribution error, she readily recalled a patient from her training in the 1970s. A young man was brought to the emergency ward of the hospital in the wee hours. The police had found him sleeping on the steps of a local art museum. He was unshaven, his clothes were dirty, and he was uncooperative, unwilling to rouse himself and respond with any clarity to the triage nurse's questions. Dr. Delgado was busy that night attending to other patients, so she "eyeballed" him and decided that he could stay on a gurney in the corridor, another homeless hippie who would be given breakfast in the morning and returned to the streets. Some hours later, she felt a nurse tugging at her sleeve. "I really want you to go back and examine that guy," the nurse said. Delgado was reluctant, but she had learned to respect an ER nurse who felt that something was really wrong with a patient.

"His blood sugar was sky-high," Delgado told me. The young man was on the brink of a diabetic coma. He had fallen asleep near the art museum because he was weak and lethargic and unable to make it back to his apartment. It turned out that he was not a vagrant but a student, and his difficulties giving the police and the triage nurse information reflected the metabolic changes that typify out-of-control diabetes.

"The hardest thing about being a doctor," Delgado said, "is that you learn best from your mistakes, mistakes made on living people." Chastened by the experience, she conjured up the picture of that young man whenever she was called to the ER to evaluate other disheveled and uncooperative people. But, Delgado continued, that was a single experience corresponding to a single

stereotype. "It is impossible to catalog all of the stereotypes that you carry in your mind," she said, "or to consistently recognize that you are fitting the individual before you into a stereotypical mold. But you don't want to have to make a mistake to learn with each stereotype." Rather, Delgado believes, patients and their families should be aware that a doctor relies on pattern recognition in his work and, understandably, draws on stereotypes to make decisions. With that knowledge, they can help him avoid attribution errors.

Is this really possible? I asked.

"Sure, it's not easy for laypeople to do," Delgado said, "because patients and their families are especially reluctant to question a doctor's thinking when their questioning suggests his thinking is colored by personal prejudice or bias." Still, Delgado thinks laypeople can diplomatically direct a doctor's attention to his reliance on stereotypes, because one of her patients had done this with her.

Ellen Barnett had recently sought out Dr. Delgado for help with a multitude of vexing symptoms. Many people who see Delgado have symptoms that are difficult to pin down — low energy, for example, or abrupt weight gain — and assume they have a hormonal or metabolic imbalance. Usually they don't. Ellen Barnett had already consulted five physicians and felt all five had shunned her. "I'm having what I call explosions, feeling hot all over, which make my skin crawl. I mean really crawl, like ants all over, and sometimes they come with terrible headaches," she told Delgado. "Really, it's like a bomb going off in my body. I know I am in menopause, and all five doctors told me that that's the cause of my problems. And two told me that I'm crazy. And, frankly, I *am* a little crazy," Barnett said with a wry smile. "Okay, I know menopausal women have hot flashes. But I think this is something else, that what I'm feeling is more than just menopause."

As Delgado listened, she recognized how easy it would be to

make an attribution error with a persistently complaining, melodramatic menopausal woman who quite accurately describes herself as kooky. So she stopped herself from casting Ellen Barnett as a stereotype and assumed for a minute that her patient was telling her something important, something meaningful, that these "explosions" were indeed different from run-of-the-mill menopausal hot flashes and hormonal migraines.

"I evaluated her very extensively," Delgado said, "and it turned out that, yes, she was menopausal, and yes, she was a strange person with lots of weird ideas, but what turned up in her urine was not from menopause or being kooky. Her catecholamine levels were through the roof. A CT scan showed a pheochromocytoma above her left kidney." A pheochromocytoma is a relatively rare endocrine tumor that produces catecholamines, chemicals like adrenaline that can cause wild swings in blood flow and blood pressure. The changes in circulation may mimic menopausal hot flashes and precipitate severe migraine-like headaches. The catecholamines can also cause psychological symptoms such as anxiety, despair, and even aggression. If untreated, the patient may have a stroke or heart or kidney failure.

"She had surgery and the tumor was removed. Now her hot flashes are much less severe, as are her headaches, at the level you would expect during menopause," Delgado said. "But Ellen is still kooky, by her own admission."

Delgado believes that patients or family members can adopt Ellen Barnett's approach. With a disarming sense of humor, she communicated that she understood she fit a certain social stereotype, and that stereotype had caused her doctors to fail to fully consider her complaints. "I didn't feel like Ellen was being obnoxious or patronizing," Delgado said, "and I didn't react and become alienated or annoyed with her. What she said enhanced her credibility and helped me avoid an attribution error."

Negative feelings that patients like Ellen Barnett trigger in a

physician are usually close to the surface. But positive feelings, like the ones Croskerry had for Evan McKinley, Falchuk had for Joe Stern, and I had for Brad Miller, are more difficult to recognize as dangerous. Since Delgado is a physician who has genuine affection for many of her patients, I asked whether she had ever fallen into that trap, the trap of affective error. She thought she had. "I had an elderly patient with thyroid cancer and considered treating him with radioactive iodine. There are difficult logistics involved with the therapy, and it really can disrupt the person's life. I was just about to refrain from treating this man when he said to me: 'Don't save me from an unpleasant test just because we're friends.'" At best, in severe circumstances, the family or friends of patients who realize that a doctor's affection may stay his hand at times can address this concern by saying: "You should know how deeply we appreciate how much care you show. Please know also that we understand you may need to do things that cause discomfort or pain."

Only a layman aware of how such feelings can color a doctor's judgment in subtle but significant ways could make such a remark. In pondering Delgado's vignette, I realized it would have been impossible for Brad Miller to muster the energy to think about our prior interactions and warn me this way when I saw him that morning on rounds. It was my job to be complete in my exam, and my charge to monitor my feelings when they might break my discipline.

Patients and their loved ones swim together with physicians in a sea of feelings. Each needs to keep an eye on a neutral shore where flags are planted to warn of perilous emotional currents.

Assuming the Worst

A YEAR LATER, JEAN STILL TALKS ABOUT THE ARGUMENT I had with her oncologist the day she was diagnosed with metastatic colon cancer. It was 11 o'clock that Friday night when the oncologist finally called me, and it didn't take long for us to come to odds. I was arguing that Jean should be offered chemotherapy choices, but the oncologist was offering palliative care only. The main reason was Jean's "functional status":

"If you knew her, you'd realize that her functional status is great!" I had known her for a long time and he had just met her that day. I hoped that launching into a description of her many activities would clear up the situation.

"I don't see that she can do much right now," he countered, cutting me off at the pass.

"She's 48 years old, she's chemo-naive . . . " Maybe describing her in "medical case" terms would help him see her in a new light.

He wasn't convinced: "She only weighed 90 pounds on admission."

"She's only 4 feet tall; she's always weighed 90 pounds!"

Convincing him felt hopeless, but I was particularly motivated to figure out why, because Jean is my aunt.

At that point, Jean was in a bad situation. Liver metastases were causing her bilirubin level to rise rapidly. If she wanted to try chemotherapy she would have to start it soon, before her bilirubin rose so high chemo would be contraindicated. Yet the oncology team had offered Jean an appointment in the palliative care clinic a week later. I agreed that going straight to palliative care was an option, but why not offer her other options? The gastroenterology oncologist could give a second opinion on Monday, but by then Jean could miss her window for starting chemo. Why was this so hard for the team to see? Was it simply that they were rounding late on a Friday night and were rushing to get home to their families? Was it because the attending physician specialized in palliative care? Or was it, as my Aunt Jean had experienced all her life, misguided assumptions about her true functional status?

Jean is an unusual-looking woman. A rare genetic disorder made her skin much darker than that of the rest of our Italian family, a striking reddish brown, with mosaic patches of white interspersed. She has almost full use of her hands, despite having contracted elbows and no thumbs. Her legs are short and thin, and I easily matched her adult height by the time I turned 7. When she took me to the zoo as a child, other kids would turn to look—tugging on their mother's hems they whispered, "Is she a midget?"

Back then I just knew that Aunt Jean was my "second mom," babysitting me, cheering at my games and performances, listening to my confidences, and encouraging me, like she would do for each of my cousins and the multiple mentees she adopted over time. As I got older she took me to her workplace, first at a disabilities advocacy organization where she worked on the passage of the Americans with Disabilities Act, then as she powered through offices in various state government departments, most recently managing labor relations for state government agencies.

Two weeks before her admission she noticed she was slightly jaundiced, but she thought she had gallstones. She kept crisscrossing the state to negotiate with union representatives, visit friends, care for her ill mother, and sing in church choir. When the jaundice got worse she walked into her primary physician's office, then drove to the hospital for a CT scan. But by the time the oncology attending saw her late at night, 48 hours of testing later, I'm sure they didn't see a "functional"-looking woman, but an unusual-looking and jaundiced woman, so exhausted and scared she could barely comprehend what they were telling her, let alone maintain her usual vigilance against erroneous assumptions.

The next morning, however, was a flurry of activity. I took the first flight that would get me to her hospital; another relative called a local oncology colleague. The oncology fellow called Jean's primary physician at home. But it was Jean who quickly figured out that she had to change the ideas formed about her the night before. She put on street clothes, got out of bed, and walked laps around the nurses' station, belting a hearty hello each time she passed by. Later that day, word came that Jean would not be discharged as planned but would remain to hear about chemotherapy choices from another oncologist. The next day Jean decided to start chemo.

Before this, I thought the challenges people with disabilities faced getting medical care were physical barriers: an examination table that doesn't lower, a mammography machine that the patient must stand up to use. But now Jean faced attitudinal barriers: clinicians' attitudes led them to assume that Jean couldn't handle chemotherapy, which they did not confirm before making treatment decisions. Jean had faced unfounded assumptions about her abilities before, but in the medical setting the assumptions seem more dangerous, in both their subtlety and their potential for harm. Recently a nurse said, "Jean's life must have always been tough; she must be tired . . . , " although Jean never implied that life was tough or that she was anything but enthusiastic about living. I wonder how often others with disabilities encounter incorrect assumptions about their physical abilities, mental abilities, social and family life, or quality of life that affect their medical care before they are explored or confirmed.

A Piece of My Mind Section Editor: Roxanne K. Young, Associate Editor.

The idea that biased attitudes might contribute to health disparities is nothing new. Unconfirmed assumptions about people with minority ethnic backgrounds, perhaps that they have few resources or a tendency toward nonadherence, could be contributing to decisions not to offer cardiac catheterizations or knee replacements. Unfortunately, the situation could be even trickier for people with disabilities. While there is no physiologic basis to assume that minorities will do worse with catheterization, knee replacement, or chemotherapy, it is reasonable to assume there *might* be medical contraindications to aggressive therapy in someone with physical disabilities. The problem comes when these assumptions are not tested with the individual patient.

When psychologists say that our biased attitudes are very hard for us to recognize, I am both comforted and frightened. Biased decisions often stem from implicit associations that the subconscious makes as the first bits of information about a situation trickle in.[1] These implicit leaps are "primed" by prior experiences and current circumstances. In medicine we are just starting to understand how these implicit associations can subconsciously lead to biased attitudes and contribute to disparities.[2,3] In this case, I'm sure the oncologists didn't explicitly think, "Jean was born with a disability and therefore we won't offer her the same choices we offer our other patients." Instead, did the attending physician's subconscious say, "Contracted arms equals other physiologic dysfunction," "90 pounds equals long-term illness and debilitation," "Genetic disorder equals mental retardation," or "In the hospital alone equals no support network" before he had a chance to consciously collect other data about the person in front of him? Since implicit associations are part of normal brain function, they are probably a common factor in medical decision making. In fact, medical training could increase our use of implicit associations as we learn to quickly recognize patterns and assess emergency situations.

Given the ubiquity of implicit associations and the conditions that encourage their use, I started to craft a scenario to explain the oncologists' limited offer. The team was working late, with a large rounding list. They were going off service that weekend. Perhaps they were "primed" by the attending's specialty of palliative care or by the physical description of Jean in her chart. They walked in to see a tired and strange-looking patient, felt pressed to make a rapid decision, and didn't ask about her physical abilities in detail.

One year after her diagnosis, Jean is working part-time, still singing in the choir, counseling her mentees, traveling with friends, and celebrating family weddings. She has had relatively few adverse effects from chemotherapy. She knows that even with the options she's had, things could have gone much worse. However, the mistaken assumptions made could have taken this functional year from my aunt's life. While Jean was able to overcome these initial barriers to her care, I wonder how often others with disabilities cannot. What if Jean had been unable to walk around the nurses' station or unable to communicate through speech? What if she was not skilled in advocacy and negotiation from her work experience? What if she had simply been too sick to realize what was happening to her?

The responsibility to overcome attitudinal barriers has to lie with those who make clinical decisions and the institutions they work in. Yet if attitudinal barriers come from something so integral and unintentional in our thinking, is there anything we can do to stop them? In the case of people with disabilities, a current congressional bill calls for medical centers to make facilities more accessible and to train medical professionals in interacting with people with disabilities.[4] While removing physical barriers to care is a laudable goal, and training might increase our awareness of barriers, I wonder if we could also find ways to systematically confront implicit assumptions we've made about vulnerable patients. Oncologists have already described such a system for geriatric patients, calling for the use of the Comprehensive Geriatric Assessment tool with every new elderly patient before treatment decisions are made. Having experienced life as a busy clinician, I also can't help but wonder if changing the circumstances in which we make medical decisions (time pressured, tired, without colleague input) would make us less likely to rely on implicit assumptions.

Perhaps the first step is realizing that implicit assumptions leading to biases can happen in any of us at any time. A few weeks ago, Jean and I were sitting at the kitchen table sipping coffee, having one of our treasured chats. When we started talking about her medical experiences, I proudly pontificated on the lessons I found in her story:

"Oncologists should know that people with disabilities could do just as well with cancer treatment as people without disabilities!"

Jean rolled her eyes at me.

"'Just as well?' When my mother took me home as a newborn, they told her I was not going to live more than a couple of months. All my life I have fought to enjoy more of what life has to offer. I think people with disabilities could have better cancer treatment outcomes because of the fighting spirit that has helped them beat the odds all along."

Better outcomes. Why had I assumed less?

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Acknowledgment: I thank my aunt, Jean, whose name has been changed, for her invaluable advice throughout life, including her contributions to this essay; and Peter A. Ubel, MD, and Ronald J. Buckanovich, MD, PhD, for editing assistance.

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The Fine Art of Optometry

Many of the world's greatest Impressionist artists suffered from severe ocular diseases that heavily influenced their works of art. **By Amy Hellem, Editor-in-Chief**

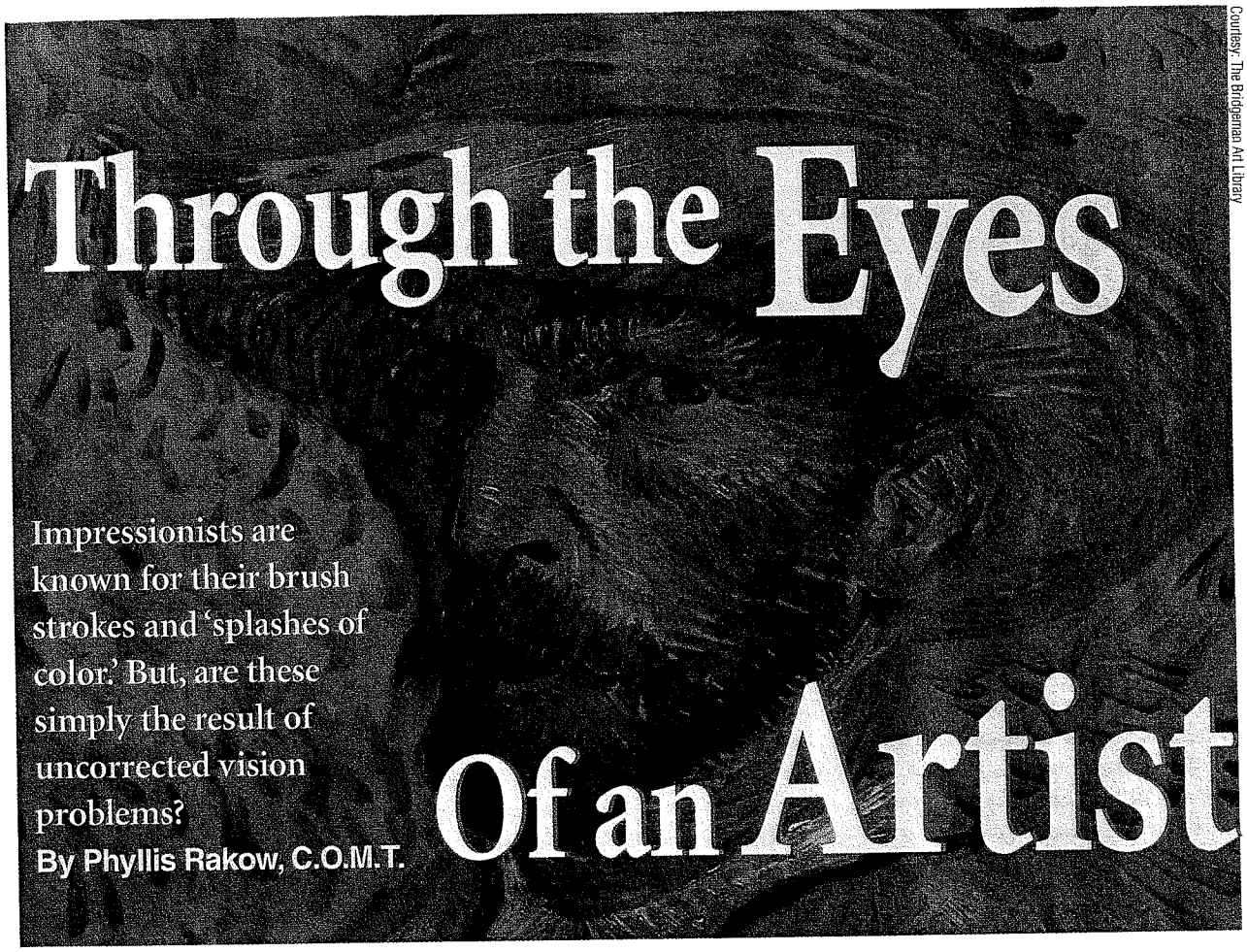
As optometrists, you endeavor to make the world more beautiful for your patients. But, in rare instances, malady can breed great beauty. Such is the case with some of the world's most renowned Impressionist artists.

In this month's special feature, *Through the Eyes of an Artist (page 31)*, Phyllis Rakow, C.O.M.T., takes readers on a journey of some of the world's great Impressionist artists to uncover the visual problems that may have had profound effects on their finest masterpieces. Here is a snapshot of the geniuses that Ms. Rakow discusses in her eye-opening report:

- Paul Cezanne was myopic, but he refused to wear glasses—a reality that clearly came out in his art. Cezanne's close-ups are quite distinct, with fine details. His landscapes, on the other hand, are consistently hazy and out of focus.
- Toward the end of Auguste Renoir's career, the artist used different colors—much less blue and more warm tones. One possible explanation: cataracts, which filter out a disproportionate amount of violet, blue and green while leaving red and brown unaffected.
- Claude Monet had bilateral cataracts. For years, his color perception was so poor that he could not distinguish between colors without reading the labels on the paint tubes. Following unilateral surgery, he said, "If I was condemned to see nature as I see it now, I'd prefer to be blind and keep my memories of the beauties I've always seen."
- Why did Vincent van Gogh use so much yellow in his later paintings? He probably had a seizure disorder and was likely mismanaged and slightly overdosed with digitalis, which caused digitalis toxicity and yellow vision. He also had pica, which caused abnormal cravings for a variety of toxic substances known to cause yellow vision—including his paints, which he often attempted to eat.
- The early works of Edgar Degas show the finest details but, as he aged, he lost central vision, painted featureless faces and even had to ask his model to identify the colors of his pastels. Degas was diagnosed with "chorioretinitis," a term that, in the 19th century, encompassed many conditions that scarred the retina—including age-related macular degeneration. Over time, he began using different colors, likely as a result of blue cone deficiency.
- Camille Pissarro suffered from chronic dacryocystitis of his lacrimal sac with fistula formation. When he was young, he painted landscapes with peasants in rural Île de France. However, following diagnosis, he was advised to avoid wind and dust and lived in constant fear that exposure would cause the dacryocystitis to recur. As a result, he moved his easel and canvasses indoors and created some of his finest paintings looking out of windows at the boulevards of Paris and the rooftops of Rouen.
- Mary Cassatt, who is considered to be the most famous American Impressionist, had diabetes. Unfortunately, since insulin was not discovered until 1920, she had to depend on dietary restrictions and bizarre methods of treatment, including radium therapy. This, coupled with worsening cataract, led her to switch from oils to pastels (which are more forgiving). What's more, her smooth brush strokes became harsh and broken.

We will never know whether these artists would have reached the same heights if they had better vision. But one thing is certain: Although they may not have been able to fully see the world around them, they certainly made it more beautiful for the rest of us. ■

Amy Hellem
Editor-in-Chief



Courtesy: The Bridgeman Art Library

Through the Eyes Of an Artist

Impressionists are known for their brush strokes and 'splashes of color.' But, are these simply the result of uncorrected vision problems?

By Phyllis Rakow, C.O.M.T.

Van Gogh's supposed xanthopsia could very well have manifested in his artwork—especially in the yellow tones of his "Self-Portrait with Straw Hat," 1887. Vincent van Gogh. Van Gogh Museum, Amsterdam.

The world of the uncorrected myope is an Impressionist painting: misty, muted and out-of-focus. In the late 19th century, traditional realism was challenged by a new breed of artists who quickly filled their canvases with splashes of color, crude brush strokes and fuzzy images. Most art historians surmised that these artists were merely trying to capture a fleeting moment, an impression; others believed that eyes dimmed by defects or disease explained the broken brush strokes, the lack of detail, and the brief glimpses of nature and of life that crossed their canvases.

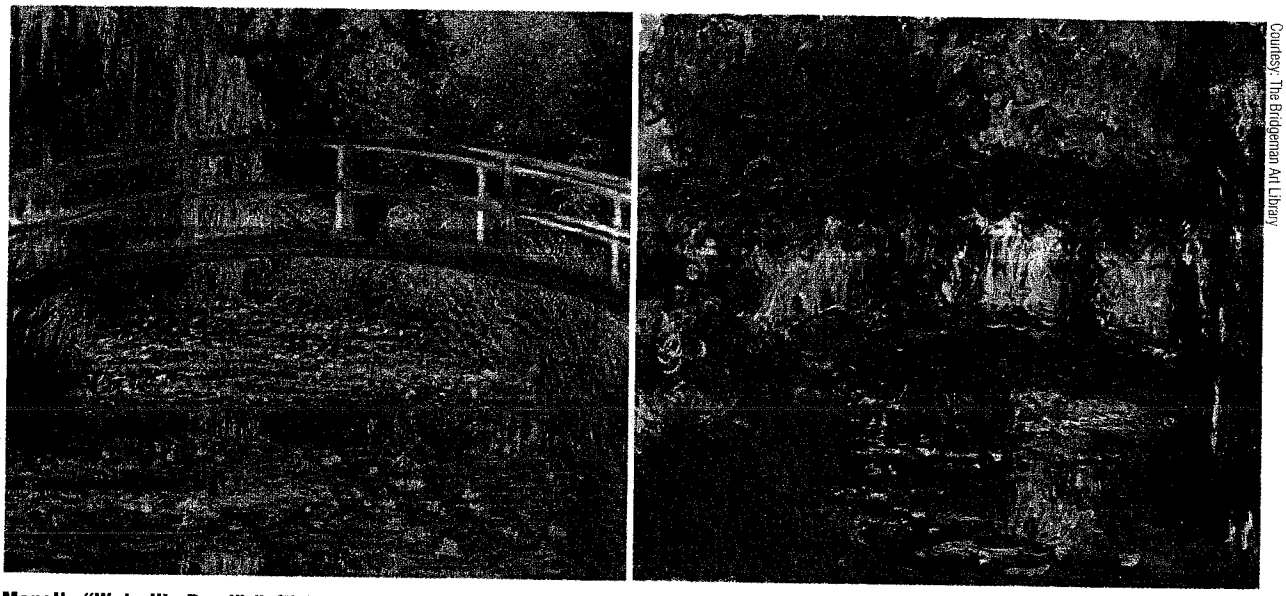
Let's look at the lives and talents of some Impressionist artists and discover the visual problems that may have had profound effects on their techniques, their colors and their media.

Cezanne

One artist who was myopic and painted without glasses was Paul Cezanne (1839-1906).[1] Myopic spectacles were readily available in his time, but he refused to wear the glasses, saying, "Take those vulgar things away."[1] A look at his artwork shows that Cezanne's close-ups are quite distinct, with fine details. His landscapes, on the other hand, were consistently hazy and out of focus. Why did he refuse vision correction? Did he prefer the softer, gentler world that he saw through his myopic eyes? Cezanne also had diabetes, but he lived before the discovery of insulin.[1] Was his sight also dimmed by diabetic retinopathy?

Renoir

Auguste Renoir (1841-1919) was erroneously described as a myope by the British author and ophthalmologist Patrick Trevor-Roper.[1] Although his early paintings show



Courtesy: The Bridgeman Art Library

Monet's "Waterlily Pond" (left) is an example of his work before he developed cataracts. "The Japanese Footbridge" (right) is his later attempt to paint the same scene with no usable vision in his right eye and a mature brunescent cataract in his left. "Waterlily Pond," 1899. Claude Monet. National Gallery, London. "The Japanese Footbridge," 1923. Claude Monet. Musée Marmottan, Paris.

great clarity, his later landscapes exhibit the haziness that Trevor-Roper attributed to myopia. Still, his son wrote about his father's keen eyesight: "He sometimes wore glasses for reading, but he did so chiefly to save his eyes. When he was in a hurry or when he mislaid his glasses, he managed quite well without them... Often, he would point out to us on the horizon a bird of prey flying over the valley."[2]

Late in life, Renoir was badly crippled by arthritis and required a wheelchair.[2] He was confined to his room for weeks at a time, had great difficulty holding his brushes and painted with far less detail. He also used different colors—much less blue and more warm tones. Were the changes in color usage due to cataracts, which filter out a disproportionate amount of violet, blue and green while leaving red and brown unaffected? Was the lack of detail in his later works due to presbyopia, or to his inability to hold his brushes properly anymore?

Monet

The term "Impressionism" was coined by Oscar-Claude Monet (1840-1926). In 1873, when asked to title a painting he entered in an exhibition, he named it "Impression: Sunrise," never realizing that he and the others who adopted similar styles would come to be known as Impressionists.

While in Venice in 1908, Monet began to recognize problems with color perception; by 1912, at age 72, he was having a great deal of trouble with his vision. His right eye was virtually useless. He was diagnosed with bilateral cataracts, and several prominent ophthalmologists recommended surgery. But, in spite of the nearly mature brunescent cataract in his right eye, Monet refused surgery. In 1913, glasses were prescribed, with a plano lens O.D. and -1.75D lens O.S. A separate pair of reading glasses was also prescribed, with +1.50D lenses O.U.[2]

As time passed, bright sunlight overwhelmed him, and he could no longer paint at mid-day. He had difficulty distinguishing between similar colors, and was forced to identify colors by reading the labels on his paint tubes. On his palette, he maintained the colors in a regular sequence in order to prevent mistakes. In a later interview, he said: "I no longer perceived colors with the same intensity. I no longer painted light with the same accuracy. Reds appeared muddy to me, pinks insipid, and the intermediate or lower tones escaped me... How many times, near the little bridge where we are now, have I stayed for hours under the harshest sun sitting on my campstool, in the shade of my parasol, forcing myself to resume my interrupted task and recapture the freshness that had disappeared from my palette! Wasted efforts. What I painted was more and more dark, more and more like an 'old picture.'"[2]

By 1922, the vision in Monet's right eye had declined to light perception with projection; visual acuity O.S. was 20/200 through a

dense yellow-brown opacity.[2] He wrote, "I wished to profit from whatever remained of my vision in order to bring certain of my decorations to completion ... I was gravely mistaken. For in the end, I had to admit that I was ruining them, that I was no longer capable of making something of beauty. And I destroyed several of my panels. Today I am almost blind and I have to renounce work completely."[2]

Dilating drops were prescribed in an attempt to open up the pupil of Monet's left eye and allow him to see around the opacity.

Initially, the response was good. Monet remarked, "I have not seen so well for a long time. The drops have permitted me to paint good things rather than the bad paintings, which I had persisted in making when seeing nothing but fog."[2]

Their effect, however, was short-lived. Finally, in January 1923, George Clemenceau, a physician as well as a politician, convinced Monet to have surgery. A two-stage operation was scheduled: a preliminary iridectomy later that month, followed by an extracapsular cataract extraction. The surgery required 10 days of absolute rest, during which time both eyes were bandaged shut. He was compelled to lie flat on his back, with his head immobilized by sandbags. During this time, he could only sip lime tea or bouillon, and he had to be forcibly restrained from ripping off his bandages. Monet said that he preferred to be blind.[2]

Post-recovery, his vision was correctible to 20/30 with +14.00D –4.00D x 180, but Monet had great difficulty adapting to his aphakic glasses. He could not use both eyes together and complained that objects curved abnormally. "I feel that if I take a step, I will fall on the ground," he said. "Near and far, everything is deformed, doubled, and it has become intolerable to see. To persist seems dangerous to me. As for going for a walk in these spectacles, it's out of the question for the moment anyway, and if I was condemned to see nature as I see it now, I'd prefer to be blind and keep my memories of the beauties I've always seen."[2] He blocked his left eye with a piece of paper when reading and preferred to walk around seeing through the dense yellow-brown nuclear cataract in his left eye.

Monet also complained about the marked difference in color perception between his eyes; saying that he saw nothing but blue with his aphakic eye.[2] Two years after his surgery, he was still having difficulty adjusting to his glasses. "As soon as I am in a better frame of mind, I will try to get used to them, though I am even more certain that a painter's eyesight can never be returned. When a singer loses his voice, he retires; the painter who has undergone a cataract operation must give up painting; and this is what I have been incapable of doing."[2]

Finally, glasses with a yellow-green tint were prescribed, which brought him out of his despair and enabled him to start painting again. He was able to continue painting almost until his death in December of 1926, from chronic obstructive pulmonary disease and lung cancer.[2]

Van Gogh

The visual perception of Vincent van Gogh (1853-1890), whose short life was troubled by mental illness and bizarre behavior, is an enigma. Although the British ophthalmologist Patrick Trevor-Roper theorized that van Gogh was myopic, his vision was tested

informally by a rather eccentric homeopathic physician, Dr. Paul Ferdinand Gachet, in May of 1890—two months before van Gogh committed suicide.[1] Dr. Gachet found his vision and color perception normal.[2] But, why did van Gogh use so much yellow in his later paintings—even in the flesh tones of his portraits and self-portraits? Did he have xanthopsia—and, if so, was it caused by chemical toxicity or substance abuse?

Van Gogh probably had a seizure disorder. After release from a mental institution, his brother, Theo, placed him under the care of Dr. Gachet. At the time, digitalis, known today as a heart medication, was used to used to treat "melancholic thoughts, hypochondria, mental illness, headache, nausea, vomiting, pain in the eyes, swelling of the eyelids, tearing, and inflammation of the eyes."[2] Van Gogh's portrait of Dr. Gachet shows him seated with a sprig of foxglove, from which digitalis is extracted, in a glass on the table.

In toxic, but non-lethal doses, digitalis can cause yellow vision. Knowing Gachet's eccentric nature, and the fact that the townspeople referred to him as "Dr. Saffron" because he dyed his hair yellow, one wonders whether van Gogh was mismanaged and deliberately overdosed with digitalis, causing him to develop xanthopsia.

Van Gogh also had some digestive problems, for which he may have taken santonin, a terpene, used at the time to treat and prevent intestinal parasites and known to cause yellow vision. He was presumed to have pica, which caused him to have abnormal cravings for other terpenes similar to santonin: thujone, turpentine, camphor and absinthe.[2]

Courtesy: The Bridgeman Art Library

Were the halos in van Gogh's works, such as "The Starry Night," effects of an angle-closure glaucoma attack? Or, was he using them religiously or symbolically to create a sense of atmosphere? "The Starry Night," 1889. Vincent van Gogh. Museum of Modern Art, New York.

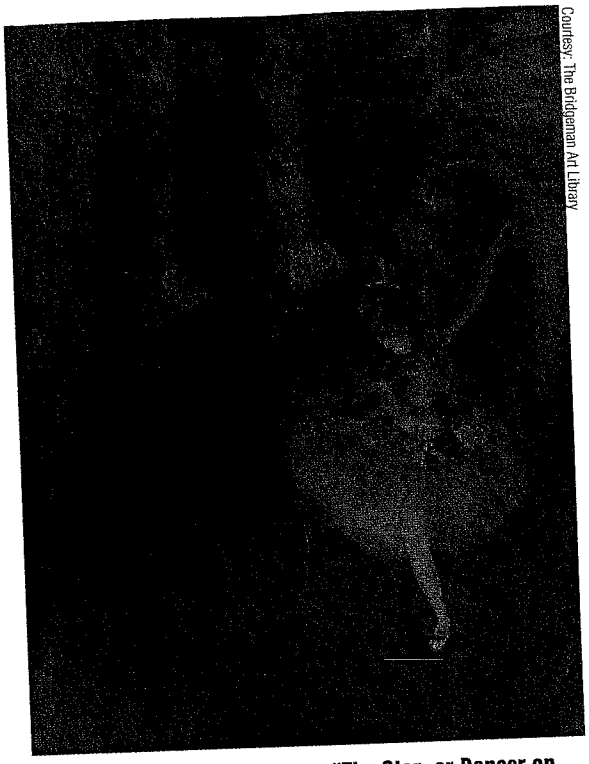
Perhaps most significant was absinthe, an emerald green, powerful alcoholic beverage (60% to 80% ethanol, with some methanol added occasionally) known as "the green fairy," and derived from wormwood.

Wormwood (*Artemisia absinthium*), classified as a psychoactive convulsant, was used by many Impressionists, Post-Impressionists, and 19th and early 20th century writers. The hallucinogen was known to alter perceptions and act on one's consciousness to produce ideas that were not otherwise accessible. There was a 19th century saying that, "Absinthe gives genius to those who do not have it and takes it away from those who do."

Van Gogh described how absinthe affected his vision: "Instead of eating enough and at regular times, I was keeping myself going by coffee and alcohol. I admit all that, but it is true all the same that to attain the high yellow note that I attained last summer, I really had to be pretty well strung up."[3]

Eye-care practitioners with an interest in art have also taken note of the halos in some of van Gogh's paintings, especially "Starry Night" and "Starry Night Over the Rhone," and debated as to whether they could have been depicting halos that he saw during attacks of angle-closure glaucoma.[2]

There is no mention, however, in any of his letters or medical records of symptoms characteristic of glaucoma, such as intense pain, nausea or loss of vision. It is more likely that he used the halos religiously or symbolically, as did some of his contemporaries, to create a sense of atmosphere in his landscapes.[4]



Courtesy: The Bridgeman Art Library

In Degas' later works, such as "The Star, or Dancer on the Stage," the focal point has been shifted to the right in an effort to avoid the blind spot in the center of his field of vision. "The Star, or Dancer on the Stage," 1876-77. Edgar Degas. Musée d'Orsay, Paris.

Degas

Edgar Degas (1834-1917) is famous for his ethereal depictions of ballerinas, often with featureless faces. Yet, when he was younger, he painted with great detail.

Degas was a low myope, and he suffered from a chronic eye disease that caused progressive, irreversible vision loss.[2] It was diagnosed at the time as "chorioretinitis," a term that, in the 19th century, encompassed many conditions that scarred the retina—including age-related macular degeneration.

He was extremely photophobic and felt that his eye problems were caused by sunlight and cold weather.[2] In 1873, on a visit to relatives in New Orleans, Degas found that his cousin had a progressive eye disease termed simply, "ophthalmia," that caused her to lose all useful vision in her left eye by the time she was 25, and rendered her totally blind in both eyes by the age of 32.[2]

Degas feared that his visual loss would also be progressive and permanent. In a letter written in 1873, he lamented, "I expect to remain in the ranks of the infirm until I pass into the ranks of the blind."[2]

Early works of Degas show the finest details: eyebrows and lashes, details of musicians and their instruments; folds in the ballet costumes; and canvases filled from edge-to-edge.

As his condition worsened during the 1880s, he lost central vision. He described his painting as an exercise in avoiding the blind spot; his focal point shifted from the center to either the right or left side of his canvas. He had to ask his model to identify the colors of his pastels, and as time went by, his colors changed to harsh, strident tones.[2]

In 1892, Degas was given glasses with an occluder lens O.D. and a stenopeic slit that approximated his astigmatic axis O.S. In an 1873 letter, he wrote: "You will see me with a comparatively ominous looking contraption over my eyes. They are trying to improve my vision by screening the right eye and allowing the left one to see through a small slit."[2] The glasses were soon abandoned, as he found them embarrassing and not helpful.

Photographs taken between 1890 and 1900 show that Degas' eyes were straight.[2] He wore glasses and was apparently orthophoric, documenting that he must have retained some vision in his right eye, even late in life. Additionally, if his right eye was totally blind, why would he have needed an occluder lens in his stenopeic spectacles.[2]

The late works of Degas show a predominance of red and a relative lack of blue. Central retinal disease can cause blue cone deficiency, and his difficulty in distinguishing colors supports the diagnosis of retinal disease.[2] The etiology of his retinal disease was never established, but possibilities have included infection, degenerative disease or familial disease. As his sight diminished, he adjusted to its limitations by exploring other media: pastels instead of oil; photography; and sculpture, in which he could use his sense of touch to enhance his limited vision.[2]

Pissarro

Camille Pissarro (1830-1903) has been called the "Tearful Impressionist," since he suffered from chronic dacryocystitis of his right lacrimal sac with fistula formation during the last 15 years of his life.[2]

When Pissarro was young, he enjoyed painting landscapes with peasants in the then-rural Île de France. In 1887, blaming traditional physicians for the death of his friend, the artist Edouard Manet, Pissarro sought treatment for an inflammation of the right lacrimal sac from a homeopathic ophthalmologist.[2] Thus began a

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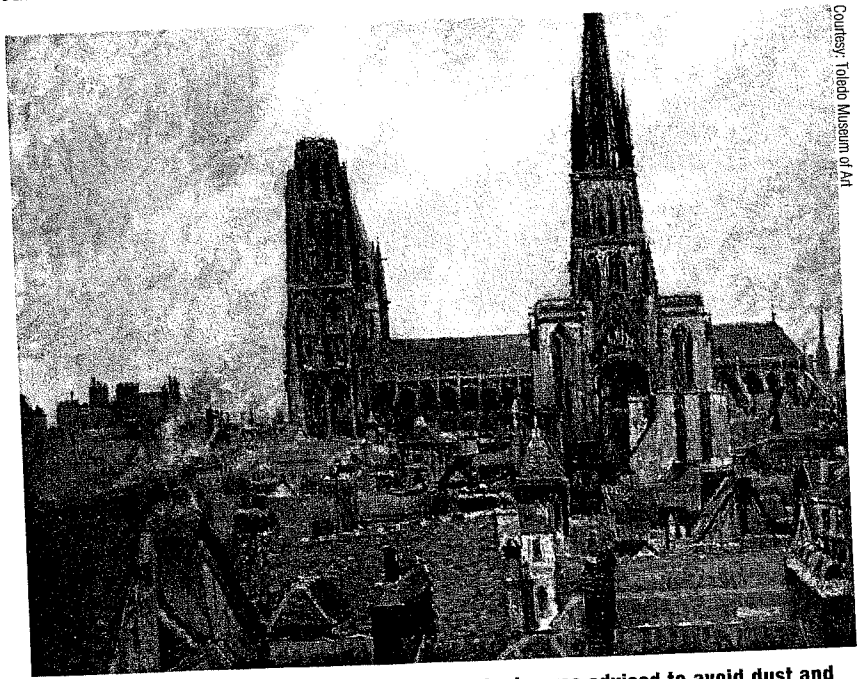
process of recurrent swelling, abscesses, probing of the right nasal lacrimal duct, discovery of a bony obstruction in the passages, injections of silver nitrate to close off abnormal passages created by the probing, and constant fear of cellulitis and further scarring.[2]

Pissarro was advised to "avoid wind and dust, [and] wash the eye with boric acid immediately" if the eye became inflamed.[2] Arum, a homeopathic remedy, was prescribed to promote healing of the tissues surrounding the bone. Each time an abscess formed, the eye was bandaged for several days, and Pissarro lived in constant fear that exposure to dust or wind would cause the dacryocystitis to recur.

Much the pragmatist, he wrote to his son, Lucien: "I am getting used to the idea of having only one eye for working. This is much better than having none at all."[2] He curtailed his travel plans and moved his easel and canvasses indoors. No longer able to paint scenes of landscapes with peasants, he created some of his finest paintings looking out of windows at the boulevards of Paris and the rooftops of Rouen.

But, Pissarro's dacryocystitis persisted. Fearful of surgery, he consulted other ophthalmologists, receiving conflicting advice regarding the management of his condition. During Pissarro's lifetime, the only methods available in France for treating dacryocystitis were probing or removal of the lacrimal sac. On the one hand, total destruction of the lacrimal sac was recommended, the result of which would be constant tearing; on the other, attempting to locate a passage through the bone to the nose via further probing, possibly causing more false passages, cellulitis and scarring.[2] He never opted for either procedure.



Courtesy: Toledo Museum of Art

After Pissarro was diagnosed with dacryocystitis, he was advised to avoid dust and wind. So, he painted cityscapes, such as "The Roofs of Old Rouen, Gray Weather," from inside his window. "The Roofs of Old Rouen, Gray Weather," 1896. Camille Pissarro. Toledo Museum of Art, Toledo.

Ironically, just a year after his death, the Italian surgeon Addeo Toti published his technique of dacryocystorhinostomy, ushering in the era of modern lacrimal surgery.[2]

Cassatt

Mary Cassatt (1844-1926), considered to be the most famous American Impressionist, spent much of her life in France. Rather than painting landscapes like many of her contemporaries, she painted carefully composed scenes of mothers and children until her vision began to fail.[2]

Cassatt's visual problems began at the age of 56, in 1900. She had diabetes, so she had to depend on dietary restrictions and bizarre methods of treatment, including radium therapy.

In a 1911 letter, she wrote: "I am at the doctor's taking inhalations of radium. This is the eighth day, and I am suffering very much, which it seems would prove that it is doing me good, that it will be a success, provided I can stand it."[2]

Radium was the "miracle" treatment of the early 20th century. Although it is not known whether Cassatt was treated with radium for her cataracts as well as for her diabetes, a 1920 article in the *American Journal of Ophthalmology*, entitled "Radium for Cataract," reported that: "Of the 31 patients under observation, 84.3% showed a change for the better. In the cases that showed a marked improvement, the opacities were definitely thinned out; one of these, a very early nuclear cataract, disappeared entirely, leaving no trace of the opacities. Radium is of proven value in the treatment of incipient cataracts."[2]

Radium has since been proven to cause cataracts, rather than heal them.

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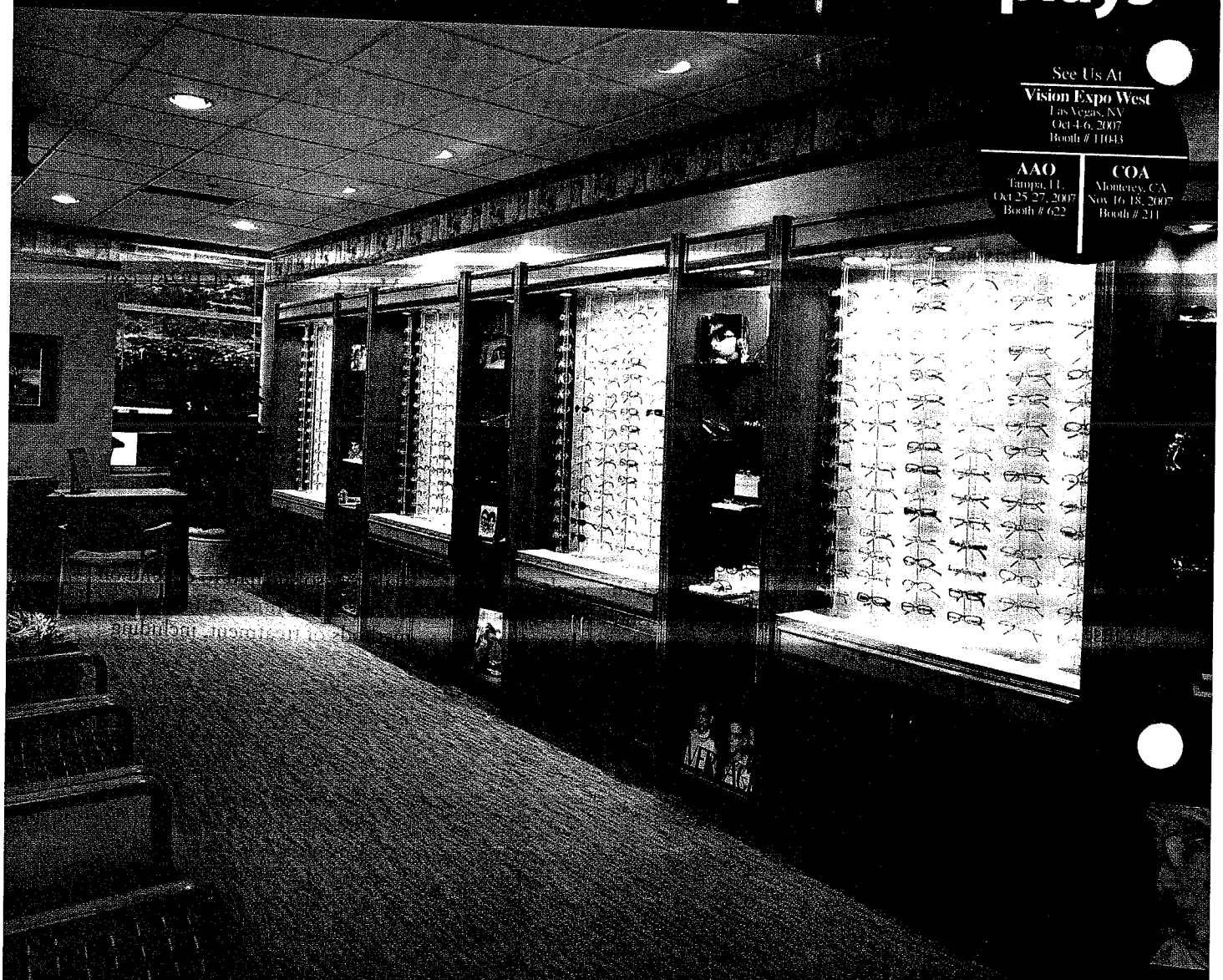

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In 1912, she was diagnosed with cataracts. By 1915, her vision had declined, and she was no longer able to paint. She switched from oils to pastels, which were more forgiving, and her smooth brush strokes became harsh and broken. Cassatt was no longer able to recreate the delicacy of her earlier paintings.[2]

Looking back on the lives of these artists, consider the words of Mr. Cross, the Vicar of Chew Magna in Somersetshire, England: "The newly invented optick [sic] glasses are immoral ... They pervert the natural sight and make things appear in an unnatural and false light."[1]

Or, an epitaph in the church of Santa Maria Maggiore in Florence, Italy: "Here lies Salvino d'Armato, of the Armati of Florence, inventor of spectacles: May God forgive him [for] his sins. A.D. 1317."[1]

Today, many of the conditions that affected the these artists can be treated and corrected, but we cannot help but wonder: Would they have reached the same heights if they had been able to perceive the world with perfect sight? ■

Phyllis Rakow, director of Contact Lens Services for the Princeton Eye Group, is a JCAHPO-certified ophthalmic medical technologist, NCLE advanced-level contact lens technician, and an honored fellow of the Contact Lens Society of America. She dedicates this article to Michael Marmor, M.D., and James Ravin, M.D., whose book, The Eye of the Artist, *served as her principal resource and forever changed the way she looks at art.*

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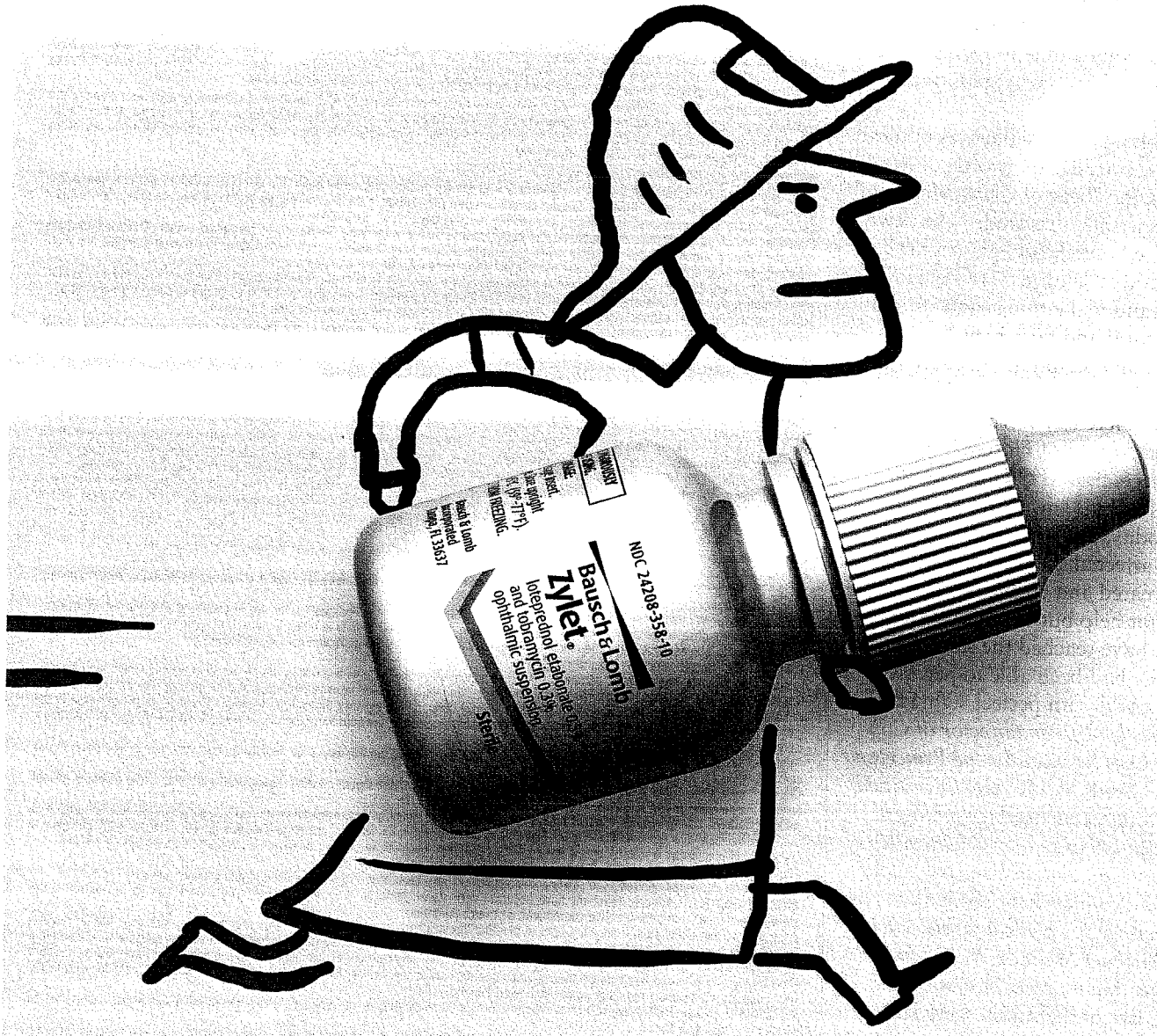

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Houses at Auvers, Vincent Van Gogh (printed with permission of the Toledo Museum of Art).

Van Gogh's Illness

By James G. Ravin, M.D.

Vincent Van Gogh's art is greatly
lmired today. He is known as a
mantic artist who painted in a
ique powerful manner. However,
uring his lifetime (1853-1890), he was
rtually ignored. His personality did
ot attract friends. He was impulsive,
of tempered, brooding, moody, and
nsociable. Only his brother felt he
as a significant artist, and not until
ie last year of his short life did he
nally sell a painting.

Van Gogh's problems began at birth. One year to the day before his birth, his parents had another child, also named Vincent, who died shortly after he was born. The baby was buried in the village church. As a child, Van Gogh saw the tombstone with his own name on it every Sunday as he attended services.

Van Gogh became a minister like his father. After conflicts with church authorities, he was relieved of his duties. Perhaps in a cynically defiant mood, he moved in with a pregnant alcoholic prostitute. He saw himself as an unloved failure. He worked for a while for an art dealer, but was once again fired. He did not take up painting until late in his twenties, but then painted prolifically for the last ten years of his life.

As early as age twenty-five he spoke of suicide. He lived briefly with an artist who moved out when Van Gogh

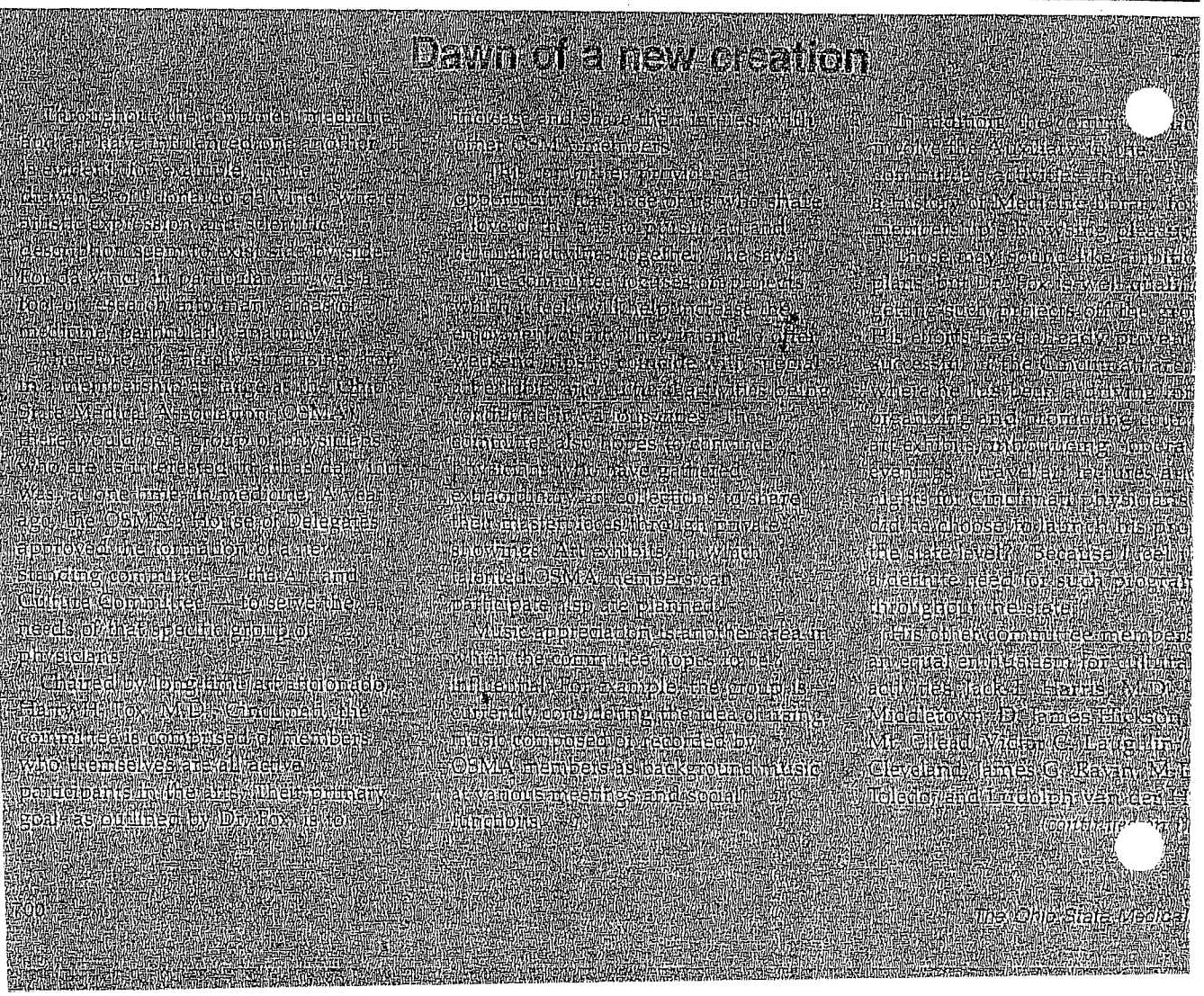
suggested a suicide pact. Later he drank turpentine and ate some of his oil paints in suicide gestures. (However, we should not be too harsh on Van Gogh for taking turpentine. Like many other compounds, it was used in the last century to treat mania.) His diet was frequently meager, for days consisting of bread and coffee. He developed scurvy and lost some teeth. In 1888, Paul Gauguin accepted his offer to live with him. They soon quarreled, and Van Gogh threw a glass of absinthe at Gauguin. The next day he threatened Gauguin with a razor and Gauguin moved out. Later that day, Van Gogh cut part of his left earlobe off and gave it to a prostitute. He was in and out of psychiatric hospitals for the next four months. He was agitated, confused, suspicious, and suffered from auditory and visual hallucinations and insomnia. He spent most of the last year of his life as a voluntary inmate of an asylum. His physician diagnosed epilepsy, chronic sunstroke, and turpentine intoxication.

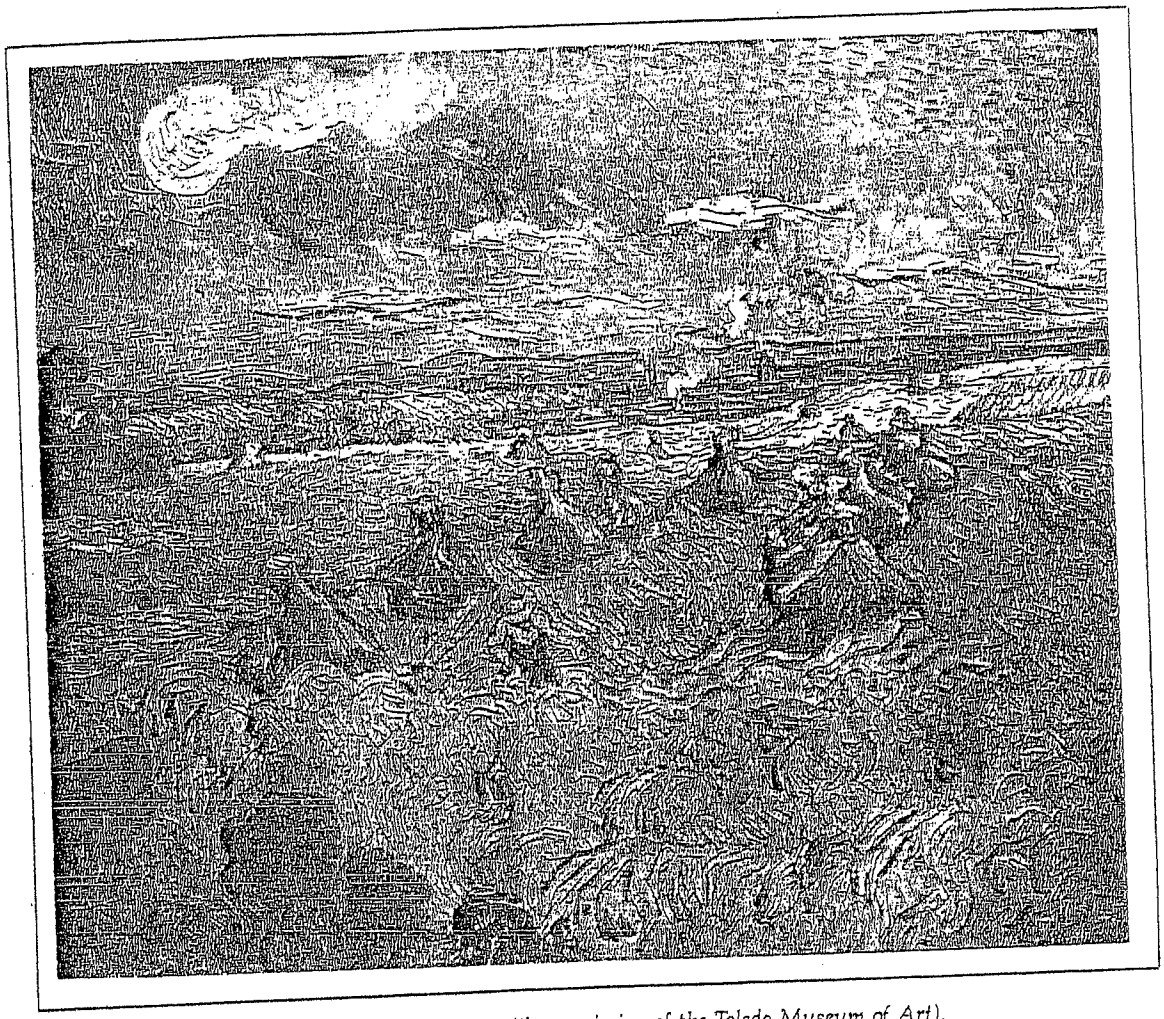
The end came when Vincent borrowed a pistol and went out for a walk in the country. A farmer saw him sitting in a tree and heard him mutter, "It is impossible." He shot himself in the chest, but was able to return to the asylum where he died two days later at the age of thirty-seven.

The end came when Vincent borrowed a pistol and went for a walk in the country. A farmer heard him mutter, "It is impossible." —

Bipolar disorder, mixed, (manic-depressive illness) is the most likely diagnosis of Van Gogh's illness.[2,3] This diagnosis was not known during his lifetime. The term epilepsy was used then to cover a far greater range of disease than now. Van Gogh's problems also may have been aggravated by chronic consumpt absinthe.[4,8] Absinthe contains th chemical, thujone, a convulsant. Manufacture of absinthe is now in most countries because " ind behavioral problems.

There is a theory that Van Go suffered from acute glaucoma.[5] evidence for this theory is as foll (1) Emotional outbursts may trig attacks of acute glaucoma, and V Gogh certainly had many emotic upsets. (2) Patients with acute glaucoma usually have swelling cornea which causes them to see colored haloes about light source Colored haloes may be found ar lamps and stars in several of his paintings. (3) Patients with acut glaucoma usually have the pupil affected eye dilated during the a Several of his self-portraits show mirror image of his left eye dilat know that Van Gogh tried to co what he painted accurately. For example, photographs of the sce Auvers, France, taken sixty year





The Wheat Field, Vincent Van Gogh (printed with permission of the Toledo Museum of Art).

The fact that many of his late works (such as "The Wheat Field") have a large amount of yellow in them have led some to believe Van Gogh suffered from digitalis toxicity. But, of course, wheat fields *are* yellow . . .

ile painted "Houses at Auvers," are recognizable as the subject. The glaucoma theory is interesting. However, there is no good evidence from records made during Van Gogh's lifetime to substantiate it.

Recently, another diagnosis that has been offered is that Van Gogh suffered from digitalis toxicity.[6] The evidence given in support of this includes the fact that many of his late works have a large amount of yellow in them. The toxic effects of digitalis include color vision abnormalities. Most often yellow or green vision occurs, but red, brown or blue vision is possible. Van Gogh's painting, "The Wheat Field," was cited as showing a yellow cast — although, of course, wheat fields are yellow.

Also cited as proof of his digitalis toxicity is the fact that Van Gogh twice painted his last physician, Dr. Gachet, with a foxglove plant on a table in front of him. Digitalis is a product of the foxglove plant. It was used to treat mania during the last century:

> The digitalis has been much celebrated, and no doubt succeeds in some instances. . . Like all other medicines of the same class, it must be given when the accession of fit is characterized by great frequency of the pulse, extreme mobility, and palpitation of the heart. . .
>
> In France, the digitalis has been particularly praised.[7]

Dr. Gachet was a homeopathic physician. According to homeopathic theory, minute doses of drugs suffice to treat most illnesses. Minute doses of digitalis are not likely to cause yellow vision.

Van Gogh created a new form of romantic art that has great appeal today. Bright colors and writhing shapes form the basis of his unique style. The designs are simple yet accurate representations of the scenes he painted. Van Gogh's personal style of painting reflects his vibrant energy. This warm emotional art is most enjoyable.

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Credit and credibility

Usually, our authors' credit lines are short and succinct. But when a recent Bulletin of the Academy of Medicine of Toledo and Lucas County included an editorial on James G. Ravin, M.D., proclaiming "A Star is Born," we decided to take a closer look.

We discovered that Dr. Ravin's credentials for authoring this piece are more than impressive. They're awesome.

As a premedical student, he wavered back and forth between his wish to become a doctor, and his consuming passion for art. As it was, his college career featured almost as many studies in art history as anatomy, and he even spent time studying illustration with a well-known local artist.

By the time Dr. Ravin received his medical degree, he was as well versed in his avocation as his vocation, so it was hardly surprising when the American Academy of Ophthalmology asked him to present a speech on "Ophthalmology and the Fine A at their Annual Meeting in Chica

The speech marked something turning point for Dr. Ravin. Sudd he was being interviewed by the *Chicago Tribune* for his theories abo the Impressionist painters being n sighted. The *Chicago Sun Times* dic story, and the AP jumped on the bandwagon. Soon Dr. Ravin was receiving calls from Washington, D California, New York City, Alaska, Michigan, Florida, even India and New Zealand. He did countless ra interviews, both taped and live; he was quoted by Jane Pauley on "Today"; and furnished backgroun information to David Hartman for spot on "Good Morning America."

Currently, Dr. Ravin is serving o the Ohio State Medical Association Art and Culture Committee, and continues to practice ophthalmolog Toledo.

Special Communication

Van Gogh's Vision

Digitalis Intoxication?

Thomas Courtney Lee, MD

• Vincent van Gogh, the Dutch postimpressionist painter, died in 1890. He was an uncommon man. Automutilation, depression, insanity, and suicide are part of his medical history. During the last few years of his life, his paintings were characterized by halos and the color yellow. Critics have ascribed these aberrations to innumerable causes, including chronic solar injury, glaucoma, and cataracts. Van Gogh may have been under the influence of digitalis intoxication and its side effects: xanthopsia and coronas. This hypothesis is based on his twice having painted his physician holding a foxglove plant; that this medicine was used in the latter part of the 19th century in the treatment of epilepsy; and that the toxic effects of digitalis may have, in part, dictated the artist's technique.
(*JAMA* 1981;245:727-729)

VINCENT VAN GOGH was born in Zundert, Holland, March 30, 1853. His life was characterized by depression, dejection, and rage. His sister Wilhelmien was interned in an asylum for psychiatric patients, where she died in an advanced age. His brother Theodorus died insane; the other brother, Cornelis, purportedly committed suicide. In the last few years of his life, van Gogh cut off a portion of his left ear and was institutionalized for recurrent psychiatric episodes, about which he wrote: "I am either a madman or an epileptic." During this period he ingested paint, turpentine, and kerosene. In 1890 he committed suicide by shooting himself in the chest.

See cover story, p 657.

Various Diagnoses

The physicians of France who treated van Gogh during his lifetime varied in their opinions. Doctor Upar, of Arles, suggested that he had acute mania with generalized delirium. However, Drs Peyron and Rey, of St Remy, believed that epileptic crises with visual and auditory hallucinations explained his symptoms. Doctor Gachet of Auvers-sur-Oise wrote that in addition to chronic turpentine poisoning, the effects of a too-intense sunlight on his Nordic brain led van Gogh to behave in an unpredictable fashion.[1]

After his death legions of physicians and critics joined in adding their diagnoses. A summary of their opinions include the following: epilepsy, by Birnbaum, Evensen, Leroy, Doiteau, Koopman, Minkowska, and Meyer-Grosz; dementia, by Prinzhorn; psychopathy, by Bolten; psychosis of degeneration, by Hutter; schizoform reaction, by Kahn; cerebral tumor, by Bader; luetic schizoid, by Eichbaum; dementia praecox, by Bychowski; meningoencephalitis luetica, by Dupinet; schizophrenia, by Kerschbaumer; sunstroke, by Grey; and psychomotor epilepsy, by Gautaut.[2,3]

In the past decade, medicine has continued to postulate new ideas. The coronas of van Gogh's later paintings and the anisocoria in his self-portrait have led Maire[4] to suggest the possibility of glaucoma. Others[5,6] disagree and consider corneal dystrophy and progressive nuclear cataracts as an alternative explanation.

What was wrong with van Gogh? Forty self-portraits painted in the last five years of his life combined with his voluminous correspondence may provide insight. His letters[10] are one of the most dramatic and soul-searching explorations of self ever attempted. Admiration of a particular painting is, not infrequently, based on admiration of the painter and empathy toward his tragic life. Would van Gogh be so famous without his letters? An arguable though unorthodox opinion might state that his paintings are no more interesting than the canvases of Soutine, or the fauve works of Vlaminck. Yet, in van Gogh's life there are all the essentials of a great romantic drama: the heartbreaking letters that cite every stage of his suffering, the failure of his contemporaries to recognize his genius, his inability to sell more than two of his paintings during his lifetime, his unemployment and total financial dependence on his brother during the last decade of his life, the agony of his growing insanity and the ecstasy of his last golden burst of creativity, and, finally, his suicide. It is reasonable to assume that the answer, or at least part of the answer, resides in either his paintings or his letters or both.

In the last years of his life, van Gogh was dominated by the color yellow. He moved into a house that was painted entirely yellow; appropriately, he referred to it as the "yellow house." His palette lightened as he added more yellows. He wrote: "How beautiful yellow is," and, later, "to attain the high yellow note I attained this summer, there was nothing for it but to step up the dose a little." In a letter to Émile Bernard, he wrote: "What I wanted to find out is the effect of a more intense blue in the sky." He added, underscoring: "No blue without yellow." Significantly, all but a few of the paintings of his last years contained or were often dominated by yellow.

Paul-Ferdinand Gachet was van Gogh's last physician. He was a friend to Monet, Manet, Cézanne, Renoir, Daumier, and others. To many of them, he was also their

From the Georgetown University School of Medicine, Washington, DC.
Reprint requests to Georgetown University School of Medicine, Georgetown University Hospital, 3800 Reservoir Rd NW, Washington, DC 20007 (Dr T. C. Lee).

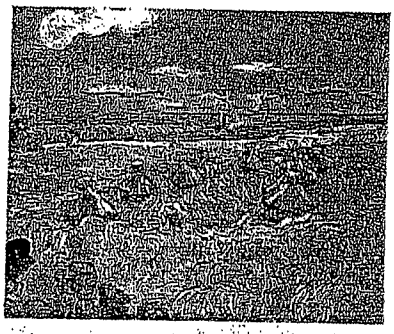

Fig 1.—Vincent van Gogh, *The Wheat Fields*, 1888. By permission of the Toledo Museum of Art, Toledo, Ohio. Gift of Edward Drummond Libbey.

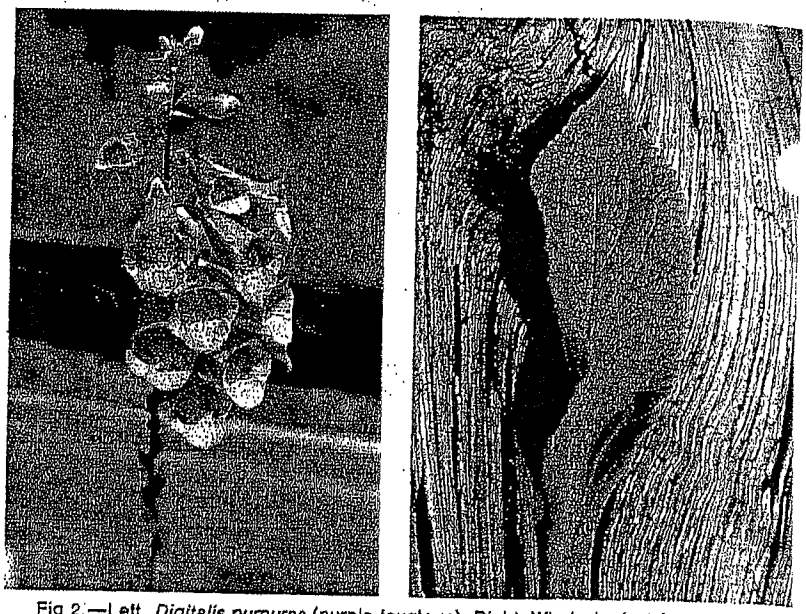

Fig 2.—Left, *Digitalis purpurea* (purple foxglove). Right, Whole leaf of *D purpurea*.

physician, and some moved their studios to be near him. On two occasions, van Gogh painted portraits of Dr Gachet. In both, the physician is leaning to his right in a mood of meditation, with his head propped against his right hand. In one of the paintings, he is holding a flower in his left hand; in the other, the same flower is in a glass. In a letter van Gogh wrote, he described these flowers as "a foxglove flower of dark purple." This was *Digitalis purpurea* or purple foxglove.

Digitalis Toxic Reaction

The symptoms of digitalis intoxication are reported to occur in 7% to 20% of patients taking the drug. These include fatigue, abdominal pain, anorexia, nausea, vomiting, dizziness, nocturnal restlessness, mental confusion, disorientation, and delirium. Visual changes, hazy, cloudy, or yellow vision, and red-green perception difficulties occur. Fatigue is noted in 95% of patients, gastrointestinal disturbances in 80%, abdominal pain in 65%, and visual complaints in 95%.[11]

In 1785 Withering[12] described "objects appearing yellow or green" when foxglove was given in large and repeated doses. Jackson and Zerfas[13] reported on a patient with yellow vision associated with digitalis poisoning: "Everything appeared yellow before her eyes . . . the sky appeared green." Sprague et al[14] commented that many patients overmedicated with digitalis sense objects as green or yellow. Furthermore, they described "spots of various colors surrounded by coronas." Among other findings, they mentioned "pupillary variations, most commonly dilatation, though constriction and inequality [anisocoria] are said to occur." Of the three cases that they reported, one patient described "a paler yellow round spot," another, "everything looked yellow, even the sky," and the third, "all objects about were yellow, such as the nurses' uniforms."

Fig 3.—Vincent van Gogh, *The Starry Night*, 1889. By permission of the Museum of Modern Art, New York.

Paul Dudley White[15] quoted Dr Cushny, professor of pharmacology at the University of Edinburgh: "All colors may be shaded with yellow . . . or rings of light may be present." Lely and van Enter[16] from the Netherlands reported digitoxin intoxication in 179 patients, of whom 95% had visual complaints. These include "flames and rings with yellow, red, green or dark colors." Psychic disturbances were noted in 65% of the patients and delirium in four.

Digitalis and Epilepsy

Van Gogh lived and died during the

latter half of the 19th century. Then and now, most authorities have agreed that he suffered from some form of epilepsy. One of the many treatments for this disease was digitalis. In 1877 Barton and Castle,[17] in discussing the uses of digitalis, stated: "Parkinson, a celebrated authority in his day, strenuously recommended it [digitalis] as an expectorant, and as a very effectual remedy, combined with polypody, against epilepsy." In 1879 Bartholow[18] quoted Dr Williams of Hayward's Health Asylum: "Digitalis is a valuable sedative in the treatment alike of recent and chronic mania, and when these forms of disease are complicated with general paresis and with epilepsy." Phillips,[19] in 1886, recommended digitalis for acute mania and in acute insanity. Gélineau,[20] in his *Traite des Epilepsies*, written in 1901, suggested use of digitalis in progressive doses in certain forms of epilepsy, especially those with "serious infiltration of the meninges." In 1881 Gowers[21] did not directly advocate digitalis as a primary treatment for epilepsy, but he stated that "an abnormal condition of the heart is met with in many cases of epilepsy . . . in some, an imperfect supply of blood to the brain may have assisted in the degredation [sic] of nutrition and function."

Van Gogh, Epilepsy, and Digitalis

Van Gogh's propensity for the color yellow is striking. Perhaps no other painter is so closely associated with a particular color. The question is, was his selection of yellow a free choice, or was it colored by the toxic effects of digitalis? This author has not been able to confirm through the vast writings of van Gogh, or in any article or text on van Gogh, that he was given or took digitalis at any time. Yet, the circumstantial evidence is catalytic. In both paintings of his personal physician, Gachet is holding the foxglove flower, whose side effects have already been noted. Symptoms of depression were common with van Gogh from 1874 to 1886. Psychoanalysts have established that sense of color is negligible in melancholia. Van Gogh's dominant palette of black, brown, and umber supports this observation. It was in 1888, when he moved to the south of France, that his psychotic symptoms became more acute. It was during this time that his paintings became overtly yellow and his epilepsy manifest. It was also the time that digitalis may have been prescribed for him to relieve his acute maniacal symptoms.

Accepting, hypothetically, that van Gogh had some form of epilepsy and that digitalis was used in the treatment of this condition, one must consider that his symptoms of xanthopsia, restlessness, mental confusion, disorientation, and delirium may have been due, in whole or in part, not only to epilepsy and its postictal effects, but also to the toxic effects of digitalis. Purkinje[22] in 1825 experienced some of these eye symptoms. He wrote:

> I took an aqueous extract of digitalis (3 grains daily) during four days and experienced practically no general effects from this small dose. However, on the second day, I noticed a very soft flicker before my left eye. . . . In the center of the field of vision, there was a rounded spot of dim lights which disappeared and again reappeared intermittently, and around were noticeable several such concentric light and dark waves in similar motion.

If one views *The Starry Night*, painted in June 1889 at St Rémy, it will recall Purkinje's digitalis effects.

Van Gogh's Paintings

It is not reasonable to assume that van Gogh was continually under the effects of digitalis and its yellow fog during the last years of his life. Rather, during his acute maniacal or epileptic periods, he may have been treated with this medicine for a short period, noticed the golden and coronal toxic symptoms, was impressed with them, and, when these side effects disappeared, purposefully continued to paint with a yellow dominance. Previously, his affinity to yellow was ascribed to countless subjective reasonings. Certainly, one must consider that he simply liked yellow or that halos were attractive or the unequal pupils that he painted in his self-portraits were simply a slip of the brush. Yet, this man, this depressed, automutilating epileptic, broke through a graphic and a color barrier. He was one of the few seminal painters who loosed on this world the avalanche of so-called modern painters, where color is not necessarily representational and where line is free.

The greatness of Vincent van Gogh is forever established. He provokes critics and physicians to explain his eccentricities; this is understandable, since even he once wrote: "There is something within me, what is it then?"

Perhaps digitalis.

The author wants to thank Margit Kerenyi Lee, Lynn Siegelman, William Meller, and the staff of the Reference Library of the National Gallery of Art, Washington, DC, for their help on this article.

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Special Communication

Van Gogh's Suicide

Frederick W. Maire, MD

Vincent Van Gogh as man and artist has been the subject of much conjecture among artistic, literary, and psychiatric notables. His suicide in 1890 has generally been considered the tragic result of a chronic mental disorder, presumably manic-depressive psychosis.

A different theory about Van Gogh's suicide is suggested, one which could have contributed materially to his death at a relatively early age: he knew that he was losing his eyesight, and the final realization of approaching blindness incited the suicidal act. This premise does not negate the real probability that Van Gogh harbored a major emotional disorder but does modify the magnitude of such a disorder.

There is some reason to postulate that Van Gogh suffered a possible chronic type of glaucoma known to be painless, insidious, and capable of causing intermittent elevations of intraocular pressure. Symptoms may only have been an intermittent appearance of colored halos seen around lights and a slowly progressive diminution of vision. This diminution induces the glaucoma sufferer to seek a compensatory and steadily increasing brightness of light in order to see more acutely.

The reasoning behind the theory that Vincent Van Gogh possibly had glaucoma is based on three factors: the halo of rainbow tints seen around light in some of Van Gogh's later paintings, his preference for brightly sunny southern France, and the apparent relative absence of obvious depression preceding his death in contrast to his previous behavioral patterns.

From the State Home and Training School, Wheat Ridge, Colo 80033.

Reprint requests to State Home and Training School, Wheat Ridge, Colo 80033 (Dr. Maire).

The Spectrum Halo Effect

Glaucoma is a common disease of the eye characterized by an increase of intraocular pressure, usually greater than about 25 mm Hg. There are two main types, that occurring without antecedent ocular disease, and that which follows a preexisting disease of the eye. The first type exists in two forms: (1) narrow-angle-closure glaucoma, in which the anterior chamber angle of the eye is narrowed or obstructed, and (2) wide-angle glaucoma, in which the chamber angle is of normal depth. In either case the increased intraocular tension is thought to be primarily a result of obstruction to the outflow of aqueous humor formed in the posterior chamber of the eye. Consequently, equilibrium is upset between the forces of fluids formed in the posterior chamber and their proper drainage into or in the anterior chamber. Damage to the retina and eventual blindness follows each type of glaucoma, should the condition progress without interference. Especially in the more chronic forms of either open-angle or angle-closure glaucoma, there can be intermittent rises in the intraocular pressure, and symptoms can be gradual and develop insidiously. This is especially true in the open-angle form. The more chronic forms of either angle type of the disease may be accompanied by symptoms that include, among others, the intermittent appearance of rainbow-tinted halos around lights and dilatation of the pupil.

One of the most striking examples in Van Gogh's paintings of colored halos is the ceiling lights in his painting "The Night Cafe" (September 1888). A similar type of ceiling lamp was not surrounded by halos 3½ years earlier in his painting "The Potato Eaters" (April-May 1885). Other of Van Gogh's paintings (completed during the last two years of his life) that could represent possible glaucomatous halos include "Sidewalk Cafe at Night" (September 1888), "Starry Night" (June 1889), and "Road With Cypresses" (May 1890). The highly colored halos he painted around celestial bodies at night, etc, have often been attributed to other clinical or mental conditions that troubled Van Gogh. Also, they could have been an esthetic or artistic endeavor as a function of his style. While these interpretations certainly are possible, illusions of colored halos could, in addition, have been physically resultant from a glaucomatous eye condition. Even though it may be argued that the night sky paintings by Van Gogh do not represent glaucomatous halos, the painting "Night Cafe" presents an example of such halos, easily identifiable.

His Preference for Sunny Southern France

According to available data, Van Gogh was en route to Marseilles when he stopped at Arles in southern France. At that time he was seeking an area with more sunlight and colorful terrain than that found in northern France. He was so impressed with this area that he decided to remain. His motivation for staying may have been esthetic or an endeavor conducive toward alleviating depressive tendencies, but

by conjecture, some admixture of motives could well have been present. If by this time Van Gogh was visually perceiving insidious effects of retinal damage, he may have been seeking more intense light. Another point of conjecture which may have had some bearing on his decision to remain in Arles could be that the visual disability found in glaucoma may be accompanied by impairment in color perception. Moreover, he could have been more comfortable painting in the constant sunlight because of the pupillary constriction it would tend to cause. Modern medical treatment for certain types of glaucoma includes miotic eye drops such as pilocarpine to constrict the pupil. Although some types of glaucoma may be painless, the condition may be accompanied by a vague, dull aching of the eyeball when intraocular pressure is higher than 25 mm Hg. Hence, not only did the more consistent sunlight possibly make it easier for Van Gogh to see brilliant colors he couldn't perceive in periodically cloudy northern France, but it also may have ameliorated uncomfortable eye symptoms. In one letter to Paul Gauguin, Van Gogh gives some indication of having a concern with his eyesight. He mentions at one point that his "eyesight was strangely tired" on one day, and so he rested 2½ days. This seems somewhat unusual for Van Gogh, in view of the arduous pace he set for himself in his painting.

Relative Absence of Depression

The three volumes of *The Complete Letters of Vincent Van Gogh*[1] fail to reveal any apparent increase in overtones of dire unhappiness or depression during Van Gogh's last few days. Certainly, in earlier periods of his life he had expressed overwhelming depression and unhappiness. It is known that he was institutionalized three times for possible psychosis, but interestingly enough, during his first mental hospitalization, he apparently compensated sufficiently and was discharged within approximately two weeks. Although one might postulate theories as to why Van Gogh was not hospitalized for a longer period of time, release from a mental institution in so short a period raises the question of the severity of his psychosis.

However, the characteristics of depression were comparatively absent before his suicide—with the possible exception of an unfinished letter to his brother, Theo, which Van Gogh wrote shortly before he shot himself in the chest. What then was the motive behind his suicide? Is it possible that along with his mental illness, Van Gogh finally acknowledged he was losing his vision, and his realization was personally disastrous to him? Van Gogh was a sensitive person. He lived to paint and he painted incessantly.

No evidence was available to indicate that he ever complained of an eye condition, other than that mentioned in his letter to Gauguin. Considering Van Gogh's personality, it is understandable that he would not refer much to symptoms which may have been present. He seemingly was not the type to accept a recognition of blindness until the last, uncontroversial piece of accumulated evidence overwhelmed him. Although he had experienced numerous non-vision-related crises before and in some way had survived them, a final recognition that loss of eyesight was approaching would be the one final blow he could not face. If he was forced to acknowledge the inevitable, it is conceivable that he fell into a rapid and irreversible depression terminating in suicide.

Van Gogh had given evidence of self-destructive behavior earlier, cutting off part of his ear and sometimes painting relentlessly for days without eating or sleeping. However, there had always been indications through his activities and in his correspondence that characteristics of depression and self-destructive tendencies would build up over a period of time. On the other hand, his tragic suicide apparently occurred without forewarning. Was he exposed to an overwhelming event (or perhaps a series of events) laying him bare and without recourse? Was it the final recognition by this great artist that he was going blind?

Another minor component may lend credulity to the hypothesis presented in this communication. Van Gogh's self-portraits when magnified show the pupil of one of his eyes to be slightly larger than the other. A dilated pupil is not uncommonly found in glaucomatous states, and the question remains: if there was anisocoria, was it due to glaucoma or to some other condition, longstanding perhaps but not necessarily abnormal? Or did Van Gogh intentionally paint the pupils this way for esthetic reasons?

Also in his last several months of painting, he left his usual, more colorful techniques in bright color display and turned to darker pigments. Unquestionably, this could also represent a progressively depressing aspect of his general affect. However, could this additionally or separately represent a progressive diminution in light and color intensity of his visual fields due to progressive blindness of the type seen in untreated glaucoma?

Reference

1. *The Complete Letters of Vincent Van Gogh, With Reproductions of All the Drawings in the Correspondence*. Greenwich, Conn, New York Graphic Society, 1958.

Vincent Van Gogh And Glaucoma

To the Editor.—An article in the Los Angeles *Times* referred to the suggestion made by Maire of the possibility that Van Gogh had glaucoma (217:988, 1971). His paintings were used as clues; for example, one pupil was painted larger than the other.

If you have the open-mindedness to listen to a nonlettered artist, there is much I can say on the subject of how the artist creates, but here will be only an abstract of my abstract art.

The one who is an artist in the true sense of the word makes one eye larger, one higher, and, what is less apparent, one advancing and one receding. Another way to say it is that they are not exactly symmetrical, but this applies to all the planes on the canvas of the modern master, not just the eyes. Further, we can say that the two eyes would have a slightly different color and value, and would not be looking exactly in the same direction. I could state this as a law of evolution, it repeats and repeats, never exactly the same, but always with a variation. And, the variation itself is a repetition of another spot on the object that is being created. The artist is making a superficial review of his own creation, if not of the race.

With illustrations and graphic demonstrations this can be explained in considerable detail to anyone who is ready to understand. A diagnosis cannot be made purely on the temporal, that is, through the senses only. The artist creates through the unconscious, the metaphysical, if you please. There is an interaction of these dual forces and they both must be dealt with.

Thomas Hart Benton said all the artists have written about their work, but no one understands it except themselves. Aren't you listening or don't we say it plainly enough?

It pleases me that Maire knows that the artist has a profound message.

GLADYS GARRETT
Los Angeles

To the Editor.—The argument that Van Gogh's "halos" copied sensory changes experienced by the person with glaucoma may be fallacious. He might compensate for loss of intensity of light sense by painting in much brighter colors. Looking at his brightly painted lights, would he not again experience his glaucomatous halos? He did not need to paint them in.

El Greco has been supposed by some to have produced his tortuous outlines because of astigmatism. But, the astigmatic would have to draw straight lines to duplicate the curvatures he saw. Thus, the argument that a painter would reproduce nature with artifact seems untenable. More likely, Van Gogh was deliberately being the artist.

G. G. LIDDLE, MD
Bloomington, Hawaii

To the Editor.—With respect to the conditions leading to "Night Cafe," and the halos about the lamps, A.M. Hammacher, in his stunning pictorial text, *Genius and Disaster: The Ten Creative Years of Vincent Van Gogh* (New York, Harry N. Abrams Inc, 1968) says it succinctly:

> In the summer of 1888, another sign of the increased formative power of Vincent's observation under the force of inner tension was the new vigor of his drawing. His hand had never been so firm, nor his sight so clear; it was as if the birth of the world and all its objects were taking place again through his creative energy. The symbol is then the circle.

This was barely two years before his suicide; "Night Cafe" was painted in September 1888.

If there is sufficient evidence to substantiate visual affliction in the Dutchman, let it come forth. Otherwise, to use ophthalmologic symptoms to explain an artist's creative impulse and psychotic pain merely contributes speculation, not hypothesis, to medicine, and serves neither art nor artist.

KEITH SPENCER FELTON, MFA
Santa Monica, Calif

To the Editor.—When you consider other liberties Van Gogh took with reality, the halos may not mean much. Any corneal clouding, such as corneal dystrophy, could have done the same thing. Van Gogh's liking for the southern parts of France did not have to be because his eyes were bad. People go to Miami Beach, Fla, in January because they like the warm sunshine, not to resolve eye problems. Gauguin liked it too, as did many other painters. As for the pupil, it tends to be dilated in angle closure glaucoma, which Van Gogh obviously did not have. He might have had an Adie's tonic pupil, but more likely the anisocoria was a slip of the brush.

I think the case for glaucoma a bit weak, but I am also bothered by that "uncontroversial piece of accumulated evidence," which might have been incontrovertible, and by the loan of "credulity to the hypothesis presented."

H. STANLEY THOMPSON, MD
Iowa City

To the Editor.—While Maire does not mention the type of glaucoma that he thinks Van Gogh may have had, the reference to anisocoria (presumably one pupil is fixed and semidilated) and intermittent elevations in intraocular pressure suggest acute or subacute angle-closure glaucoma. While these conditions do indeed cause corneal epithelial edema which results in the perception of colored halos, an alternate and more plausible cause in Van Gogh's case would be nuclear sclerosis of the lens.[1] Further, there are some who speculate that Van Gogh was near sighted.[2] Were this the usual form of myopia associated with an increase in axial diameter, angle-closure glaucoma would be most unlikely.

Maire suggests that Van Gogh sought the brightness of southern France for the symptomatic relief of glaucoma. Indeed, in subacute angle-closure glaucoma, eye symptoms may be vague and minimal, and an attack may be broken by pupillary constriction to bright light. However, if Van Gogh had to resort to painting in bright environment in order to obtain relief, how he must have suffered under darker conditions when not outdoors!

Further, in the years from 1885 to 1890 when Van Gogh painted colored halos, had he had chronic glaucoma, he might well have suffered field loss which would have been readily apparent to any artist.

Maire's statement that primary glaucoma causes damage to the retina is contrary to the facts. It is the optic nerve that suffers from elevated intraocular pressure and the resultant loss in vision takes the form of field defects and constriction in field. Often visual acuity is normal while the field of vision becomes markedly impaired and constricted. Also, I am not aware of glaucoma per se causing impairment of color perception. If Van Gogh had needed more intense light for his work, it would have been more compatible if he had progressive cataract. If the cataract were axial, miosis would further compromise vision. Nuclear cataract, wherein there is a diffuse increase in the optical density of the nuclei, however, causes lenticular myopia[3] in which case the pinhole effect of miosis would en-

Psychosocial Stressors and Adjustment Disorder: Van Gogh's Life Chart Illustrates Stress and Disease

Richard H. Rahe, M.D.

The life of Vincent van Gogh is illustrative of the natural history of psychosocial stressors and their relationship to a person's states of health and disease. In the author's opinion, there is a lack of such understanding in the current, established criteria for psychosocial stressors in the diagnosis of adjustment disorder. By use of a life chart, which chronologically documents a person's major life events and concomitant health status over his or her life span, a fuller understanding can be reached regarding why an individual becomes ill at a particular time.

(J Clin Psychiatry 1990;51[11, suppl]:13–19)

The diagnostic criteria for adjustment disorder are detailed in the Diagnostic and Statistical Manual (DSM-III-R) of the American Psychiatric Association.[1] Despite a theoretical attempt to define the nature and timing of preexisting psychosocial stressors required in this condition, the DSM-III-R criteria are, in my opinion, both naive and misleading. For example, I have rarely encountered a cluster of psychosocial stressors in which only events that had existed for less than 6 months' time comprised the cluster. Further, some events exert lifelong influences on a person's health while other events cluster within a 2- to 3-year interval prior to illness onset.[2] Finally, psychosocial stressors have been shown to precede all illnesses, not only psychiatric illness, and certainly not only an adjustment disorder.[3]

The diagnosis of adjustment disorder seems to fill a clinically useful category, falling between major psychological illness and health.[4] In other words, patients receiving this diagnosis tend to have a history of having coped fairly well in their past lives, but are not doing well with life's present challenges. Thus, the diagnosis is based as much on an assessment of a person's past coping capabilities as it is on current psychosocial stressors.

Perhaps because of their adequate coping skills, patients given this diagnosis tend to show high rates of recovery. An evaluation of 100 patients with anxiety disorder, followed over 5 years, showed that at the end of this interval 71% were well. Adolescents with this disorder, however, showed a poorer rate of recovery; only 44% were healthy 5 years later. Those adolescents who failed to recover frequently proved to have additional, more severe, psychiatric illnesses.[5]

During my career in the U.S. Navy, I reviewed the case histories of a large number of officers and enlisted men with adjustment disorder (or as this condition was labeled in DSM-II, situational adjustment reaction). These men had been exposed to severe military stresses that had lasted from a few months to several years. A general estimate by the psychiatric medical officers treating these men was that recovery was likely, based largely on historical data supporting the men's preexisting coping resources. Two thirds of these men did recover over a 5-year follow-up. When recovery was not seen, other major psychiatric illnesses were often found to coexist, such as depression, alcoholism, and personality disorder.[6]

THE NATURAL HISTORY OF PSYCHOSOCIAL STRESSORS

Psychosocial stressors can be divided into two major categories with relevance to their influence on illness onset: predisposing events and precipitating events. On the one hand, predisposing events are those that generally occur early in life and apparently "sensitize" an individual to selected life situations. For example, the death of or desertion by a parent during an individual's childhood may predispose that individual to deficient adult relationship and/or parenting skills. On the other hand, precipitating events are those changes in a person's life that closely precede, and psychophysiologically influence, the clinical onset of an illness. These

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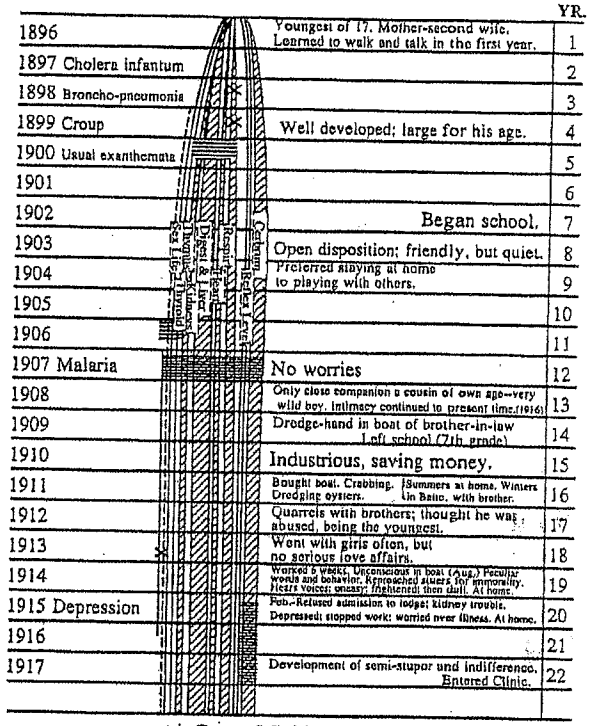
life changes are nonspecific in nature and tend to cover the entire spectrum of life adjustment. Examples of such life changes include marriage, divorce, winning the state lottery, losing investments in the stock market, being promoted, or being fired at work. It is the psychophysiologic arousal, or suppression, resulting from the emotional reactions to these life events that leads to the expression of an illness to which the person is constitutionally susceptible.[2]

Nearly a century ago, Adolf Meyer attempted to organize predisposing and precipitating life events into a psychobiologic framework which he called the "life chart." Meyer came to this country to complete his medical education, and, like Freud, specialized in neurology. Following his training, he joined the medical faculty at Cornell University. In 1914, he accepted the position of professor of psychiatry at Johns Hopkins Medical School, where he remained until his retirement in 1942. In volume III of his collected papers,[7] Meyer discusses why a patient seeks medical consultation at a specific time. To facilitate understanding of this concept, Meyer created his life chart. Dimensions depicted on a Meyer chart always included the patient's age, calendar years coincident with the patient's life, important life events, the patient's emotional reactions to these life events, and illness experiences across the patient's lifetime. Illnesses were organized by organ system, represented in the chart by their growth curves, giving rise to a torpedo shape in the chart. In reading across a Meyer life chart, influences of age, social era, and personal life experiences upon organ system dysfunction and disease can be surmised. An example of a Meyer chart, summarizing the stresses and illnesses of a young man who developed schizophrenia in his early 20s, is presented in Figure 1.

Meyer's chart was later modified by Stanley Cobb, Harold G. Wolff, Lawrence E. Hinkle, Jr., Stewart Wolf, Jr., Thomas H. Holmes, Tores Theorell, and me.[2] Others have used similar life event and illness onset graphics to depict precipitating life events and illness onset.[8] By and large, the use of a life chart to illustrate both predisposing and precipitating life events and illness experience has been limited to researchers and educators. Medical students and residents to whom I have taught the life chart tell me that in all honesty they will never use it; they do not wish to take the necessary time. It does require an hour or two of interviewing, plus an additional 30 minutes or so to organize the collected material on paper. Therefore, work has begun on a computer program to allow patients to construct their own "skeleton" life charts in the doctor's outer office while waiting for their appointments. The patient can then present a computer printout of the skeleton chart to the physician at the beginning of the appointment and have it "fleshed out" during the interview.

Figure 1. Adolf Meyer Life Chart*



A Case of Schizophrenia

*Adapted from reference 7.

LIFE CHANGE EVENT QUANTIFICATION

Early in my career, I worked on the problem of quantification of a person's life change events.[2,9,10] The method chosen for assigning a magnitude to the items was first developed for use in psychophysics—the study of the psychological perception of the quality, quantity, magnitude, and intensity of physical phenomena. The subjective assessment of the observer plotted against the physical dimension being perceived provided a reliable delineation of the subject's ability to quantify this experience.[11] The assumption was that persons could utilize this psychological capacity in making quantitative judgments about psychosocial phenomena. Although some persons assigned a different order of magnitude to certain items, the degree of similarity among diverse groups of persons was impressive. This high degree of consensus was seen regardless of individuals' differences in age, sex, marital status, education, social class, religion, race, or generation American.[10]

In preparing for this presentation, I compared original life change events scaling results obtained in 1963 to results of a scaling experiment I conducted in 1978 (Table 1). Over a 15-year period, the life change unit (LCU) mean values derived for events at the top of the list proved impressively consistent, i.e., scores for death of

Table 1. Fifteen-Year Effect on Life Change Scaling*

Life Event	Mean Life Change Unit Values 1963	Direction of Change	1978
Death of spouse	100	↑	105
Divorce	73	↓	62
Marital separation	65	↓	56
Jail term	63	↓	57
Death of close family member	63	↔	65
Personal injury or illness	53	↓	42
Marriage	50		50
Fired at work	47	↑	64
Marital reconciliation	45	↔	42
Retirement	45	↑	49
Change in health of family member	44	↑	52
Pregnancy	40	↑↑	60
Sex difficulties	39	↑↑	49
Gain of new family member	39	↑↑	50
Business readjustment	39	↔	38
Change in financial state	38	↑	43
Death of close friend	37	↑↑	46
Change to different line of work	36	↔	38
Change in number of arguments with spouse	35	↔	34
Mortgage (home, car, etc.)	31	↑↑	39
Foreclosure of mortgage or loan	30	↑↑	57
Change in responsibilities at work	29	↔	30
Son or daughter leaving home	29	↔	29
Trouble with in-laws	29	↔	29
Outstanding personal achievement	28	↑	33
Spouse begins or stops work	26	↑↑	37
Begin or end school	26	↑	32
Change in living conditions	25	↑↑	39
Revisions of personal habits	24	↑↑	31
Trouble with boss	23	↑↑	39
Change in work hours or conditions	20	↑↑	33
Change in residence	20	↑↑	33
Change in schools	20	↑↑	28
Change in recreation	19	↑↑	28
Change in church activities	19	↑↑	29
Change in social activities	18	↑↑	28
Loan less than $10,000 (stereo, etc.)	17	↑↑	26
Change in sleeping habits	16	↑↑	31
Change in number of family get-togethers	15	↑↑	26
Change in eating habits	15	↑↑↑	29
Vacation	13	↑↑↑	29
Christmas	12	↑↑↑	26
Minor violations of the law	11	↑↑↑	32

*Symbols: ↑ = increase in stress; ↓ = decrease in stress; ↔ = change insubstantial. Number of arrows = amount of change in Life Change Unit.

spouse, divorce, major health change, and so forth, showed very little change in LCU values across the years. Life change events in the mid-range, such as pregnancy, major mortgage, and change in living conditions, tended to stay the same or to show a slight increase in mean values. Strikingly, the life change events at the low end of the scale all showed moderate to major increases in mean LCU values. One conclusion that can be drawn from this comparison is that life change events of truly low significance no longer exist in our society. Financial changes, vacations, traffic tickets, and even Christmas, have doubled or tripled in their mean values since the early 1960s. The general perception that life has become more difficult over the past three decades is likely true.

Whether or not a clinician chooses to use the LCU scale to quantitate the relative significances of a patient's recent life events, it is important to make some quantitative assessment. A major difference exists between a patient who has recently experienced a single event, such as death of spouse, compared with another who recently received a traffic ticket. When a cluster of life events that total between 250 and 400 LCU per year is detected, it is called a "minor life crisis." If the total is greater than 400 LCU per year, it is labeled a "major life crisis."

PSYCHOSOCIAL STRESSORS AND THE LIFE OF VINCENT VAN GOGH

July 1990 marked the 100th anniversary of the death of Holland's famous artist Vincent van Gogh. His life

experiences, illnesses, and notes regarding his artistic productions were preserved through his numerous letters—most often written to his loving and supportive younger brother Theo.[12] As a teaching example for our medical students at the University of Nevada School of Medicine, Dr. John Chappel, a van Gogh scholar, and I constructed the painter's life chart (Table 2). Van Gogh's life chart demonstrates predisposing life events, precipitating life events, and both minor and major life crises. This chart also illustrates earlier statements regarding the naive and misleading criteria for psychosocial stressors in adjustment disorders in DSM-III-R.

One extremely important predisposing life event for van Gogh's later life experiences with depression was that 1 year before his birth his mother had a stillborn child who was given the name of Vincent. This child was buried in the churchyard where van Gogh's father was the pastor. The second Vincent then passed his "own" tombstone every time he walked through the churchyard.

Vincent became estranged from his parents at an early age. At 11 he was sent off to a boarding school, where he performed rather poorly. Another school was tried when he was 14, with the same result. He left school at 16 and moved to The Hague to become an apprentice to an art dealer.

If Vincent, at age 16, were transported in time to the consulting offices of a psychiatrist in 1990, with the chief complaint of anxiety, the evaluation of his home and school problems, his leaving school, his geographic move, and his starting a new job might be interpreted as precipitating psychosocial stressors leading to the diagnosis of an adjustment disorder. His previous good health and moderately satisfactory coping would support this diagnosis. Supportive individual or group psychotherapy, along with anxiolytic medications, might well be prescribed.

It is clear in his letters that Vincent began to experience symptoms of major depression by age 20. He also had developed a dependence on tobacco (pipe smoking) and became fanatical in his religious preoccupations. His first experience with falling in love occurred the following year—but the girl was already engaged. Apparently as a result of this rejection, he traveled to London to accept a job with an art firm. However, he lost this employment the next year when he was 23. He then worked as a teacher and a bookseller before moving to Amsterdam to study theology. Failing the entrance exams, he tried being an evangelist, which was less demanding academically. At 25 years of age he was dismissed from this work because of his bizarre behaviors, which included semistarvation and self-beatings to "punish" himself for his sexual thoughts and impulses. He tried art school in Brussels but soon quit and returned to his parents' home. After a short stay, and an unsuccessful amorous pursuit of a cousin, he returned to the city and moved in with a prostitute named Christine. His depression worsened and he developed venereal disease.

If Vincent were again transported to a modern-day psychiatrist's office, his psychosocial stressors over the preceding 3 to 4 years would be found to be numerous enough and of sufficient significance to qualify as a major life crisis. (Note how van Gogh's most recent life changes are inextricably linked to his life difficulties over the preceding 8 years.) His psychological symptoms would likely lead to a diagnosis of major depression, and a trial of antidepressant medications might be prescribed.

Vincent created his first sketches during his 20s, often as a way of dealing with his depression. He sketched peasants working the fields near his home, and he sketched a nude figure of Christine which he labeled *Sorrow*.

A brief 3-year respite followed. Vincent returned home once again and was apparently in good health. This period ended with his father's death when Vincent was 32 years of age. At this juncture he painted his first masterpiece, *The Potato Eaters*. He enrolled in the Antwerp Academy of Art, only to quit shortly afterward because he argued repeatedly with his teachers and refused to paint in the classical style taught there. At age 33 Vincent moved to Paris and lived with his brother Theo. In Paris, van Gogh increased his alcohol consumption markedly, including drinking the neurotoxic spirit absinthe. In addition, his number of art productions rose dramatically. Two years later, when Vincent was 35, he moved to Arles and his use of lighter colors, in the impressionist style, is apparent in the paintings from this period in his life. Gauguin's famous brief stay with Vincent occurred in Arles.

Vincent's health problems increased. As first illustrated by Adolf Meyer, when people become ill they often develop dysfunction in several organ systems of their bodies. Vincent experienced multiple extractions of teeth, "stomach pains," anemia, seizurelike episodes, hallucinations, feelings of paranoia, insomnia, and "near alcoholism." It was at this time in his life he cut off a portion of his right ear and sent it to a prostitute he had just visited. He alienated his neighbors with his odd behaviors, and eventually they petitioned to have Vincent evicted from his lodgings and sent to the mental hospital. Further, two very significant precipitating life changes occurred: Theo married, and shortly afterward Theo's wife bore him a son whom they named Vincent.

Van Gogh moved to Auvers, which was closer to Paris and to Theo, when he was 37 years old. During the last few months of his life he continued to paint subjects with a decidedly depressive theme, such as *The Plow and the Harrow*, *The Prison Court-Yard*, *Worn Out: At Eternity's Gate*, and his last painting, *Crows in*

Table 2. Life Chart of Vincent van Gogh

Date	Age	Life Event	Illnesses	Treatment
1852	-1	Stillborn brother—same name given to Vincent		
1853	0	Born in Zundert, Holland		
1854	1			
1855	2	Birth of sister		
1856	3			
1857	4	Birth of brother—Theo		
1858	5			
1859	6	Birth of sister		
1860	7			
1861	8	Estranged from parents		
1862	9			
1863	10			
1864	11	Sent to boarding school		
1865	12			
1866	13			
1867	14	Sent to new school		
1868	15	Birth of brother		
1869	16	Leaves school; goes to The Hague as apprentice art dealer		
1870	17			
1871	18			
1872	19			
1873	20	Travels to London with art firm	Depression Religious fanaticism	
1874	21	Falls in love—girl already engaged	Heavy pipe smoking	
1875	22			
1876	23	Loses job in London		
1877	24	Works as teacher, changes job to bookseller. Begins studies in Amsterdam. Fails entrance exams to theology.	Self–abuse	
1878	25	Enters training school for evangelists. Works as an evangelist.	Poor nutrition	
1879	26	Fired from his work		
1880	27	Abandons evangelism. Begins art studies in Brussels.		
1881	28	Moves back with parents. Falls in love with cousin and is rejected. Moves to The Hague. Lives with a prostitute.	Depression Irritability Venereal disease	
1882	29			
1883	30	Returns to parents' home.		
1884	31			
1885	32	Father dies. Enrolls in Antwerp Academy of Art.	Argumentative Dental problems "Stomach trouble"	Dental surgery
1886	33	Moves to Paris		
1887	34	Lives with brother Theo	Heavy alcohol intake	Hospitalized
1888	35	Moves to Arles. Gauguin visits. Argument with Gauguin. Gauguin leaves. His neighbors petition against him.	"Stomach disorder" Cuts off part of ear Anemia Hallucinations Paranoia Insomnia	Hospitalized Bromide medicines Hospitalized
1889	36	Theo marries. Leaves Arles. Theo has a son—names him Vincent.	Recurrent hallucinations Epilepsy. Ingestion of paints and turpentine. Depression. Turpentine poisoning Excessive exposure to sun	Hospitalized Hospitalized Work therapy Digitalis toxicity?
1890	37	Moves to Auvers. Shoots himself.	Suicide	
1891	+1	Theo dies of chronic nephritis		

a Wheatfield. His health continued to worsen, and evidence suggests that Vincent had begun to ingest his paints and his turpentine.[13] He may even have experienced digitalis poisoning, as this medication was prescribed for apparent epileptic attacks experienced at the sanatorium in St. Remy.[14,15] His doctor also blamed Vincent's physical deterioration on excessive exposure to sunlight while in Auvers. During his hospitalizations in both Arles and Auvers, Vincent frequently painted his institutional surroundings.

In a modern-day psychiatrist's office van Gogh undoubtedly would be diagnosed as having major depression with psychotic features. On Axis III would be listed his several exposures to brain toxins: alcohol, absinthe, paints, turpentine, digitalis, and excessive exposure to the sun. Most decidedly, psychiatric hospitalization with aggressive use of antidepressant and antipsychotic medications, perhaps even electroconvulsive therapy, would be prescribed. His major life crisis from ages 33 to 37, along with his diseases of several organ systems, would indicate an extremely poor prognosis. Vincent's suicide would not occur unexpectedly.

Of interest is the fact that 1 year after Vincent's death, Theo died from an exacerbation of his chronic nephritis. Theo's marriage, birth of a son, and the suicide of his beloved brother, composed a minor life crisis which likely facilitated his own fatal illness.

A final word should be said about life stress, illness, and the creative product. It is clear that minor and major life crises are not good for a person's health. Those individuals with substantial coping capabilities seem to maintain their organ system integrity during such challenging life periods. In contrast, the "artistic temperament" may stand in the way of good coping. A creative mind may flourish during the turmoil of life stress and disease. The turbulence of mind and body, as was the case for van Gogh, may have even stimulated several of his artistic expressions. *Starry Night* is a wondrous work of art to his admirers; to van Gogh it was also an expression of his premonition of death.

The work of the physician may sometimes be in opposition to the creative product for persons of artistic temperament. Van Gogh's life was self-destructive, but resulted in a tremendous artistic legacy for the world. Would successful psychotherapy and chemotherapy have prolonged van Gogh's productive career, or would it have reduced his drive to create?

DISCUSSION

Recent life change events are linked to preceding life changes. Early events play a greater predisposing role while more recent events exert a precipitating influence on the expression of illness. Stressful life events, or psychosocial stressors, tend to cluster in an individual's life and can be roughly quantitated as to their overall levels of significance. To arbitrarily define psychosocial stressors as influential for adjustment disorder only if they occurred less than 6 months prior to diagnosis is to be naive concerning the natural history of these events. It is also misleading to make a prognosis based on such a limited view of life stress events.

From the perspective of a life chart, a physician can estimate predisposing and precipitating life events, as well as both minor and major life crises, and assess how the patient has responded, psychophysiologically, to these events. This composite view of life stress and illness can lead to a helpful estimate of prognosis. Psychosocial stressors preceding illness are not specific to psychiatric illnesses in general, and certainly not specific to adjustment disorder in particular. Life stress and disease onset is characteristic for patients developing a wide variety of medical and psychiatric illnesses, including accidents.[3,6]

Van Gogh's life chart also demonstrates the multifactorial nature of illness. His suicide was an ultimate culmination of both predisposing and precipitating life events, along with a possible biologic predisposition for depression, combined with the debilitating effects of several brain toxins. Twentieth century medicine still resists exploring for such multiplicity in illness etiology. The wish for "a single cause" and "a single effect" has been with us since the reductionistic philosophy of Descartes. Just this year an article by Arenberg et al.[15] makes a case that many, if not most, of van Gogh's health problems could be subsumed under a single illness category, Meniere's disease. It is certainly possible that van Gogh had Meniere's disease in addition to all his other illnesses, but to posit a "single cause" for his multiple mental and physical disorders fails to recognize the several and varied forces leading to his suicide.

SUMMARY

To fully understand the natural history of psychosocial stressors in both their predisposing and their precipitating roles in the expression of clinical illness, it is extremely helpful to construct a patient's life chart. Some quantification of the varying significances of psychosocial stressors in a patient's life is also needed. With these tools in hand, a physician will find that stressors play an important part in the etiology of all illnesses, not just in psychiatric conditions.

A life chart constructed for Vincent van Gogh illustrates predisposing and precipitating life stress events, as well as event clusterings resulting in both minor and major life crises. Early in van Gogh's life, his life chart appeared to be rather benign. An adjustment disorder may have been diagnosable when he was in his mid-teens. By his late 20s, however, it is apparent that major de-

pression was his overriding psychological difficulty. At the end of his life, at age 37, van Gogh suffered not only from depression, but from illnesses of many organ systems in his body. These illnesses came on in a setting of a major life crisis. The timing of van Gogh's suicide is understandable when viewed from the perspective of his life chart.

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an Gogh Had Meniere's Disease and Not Epilepsy

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We intend to correct the historical error that Vincent Van Gogh's medical problems resulted from epilepsy plus madness, a diagnosis made during his life but for which no rigid criteria are apparent. Review of 796 personal letters to family and friends written between 1884 and his suicide in 1890 reveals a man constantly in control of his reason and suffering from severe repeated attacks of disabling vertigo, not a seizure disorder. His own diagnosis of epilepsy was made from the written diagnosis by Dr Peyron, the physician at the asylum of St Remy (France), wherein on May 9, 1889, Van Gogh voluntarily committed himself to the asylum for epileptics and lunatics. However, the clinical descriptions in his letters are those of a person suffering from Meniere's disease, not epilepsy. The authors point out that Prosper Meniere's description of his syndrome (an inner-ear disorder) was not well known when Van Gogh died and that it often was misdiagnosed as epilepsy well into the 20th century.

(*JAMA*. 1990;264:491-493)

VINCENT VAN GOGH was born in Zundert, Holland, on March 30, 1853, and died by his own hand at the age of 37 years in Auvers, France, on July 29, 1890. His creative genius, his reputed "madness," and the growing popularity of his works have made his short but productive life the subject of challenging discussions among literary, artistic, and medical professionals ever since his death. His voluminous correspondence[1] is an untapped retrospective clinical history of his illness. Van Gogh had an illness that was characterized by "attacks," often in clusters, interspersed with symptom-free periods that lasted for "months at a time." The casual diagnosis of epilepsy by Dr Peyron was not seriously questioned until recently.

In 1979 the Japanese otologist, K. Yasuda, first raised the question of inner ear dysfunction in an article entitled "Was Van Gogh Suffering From Meniere's Disease?"[2] Van Gogh's extant letters,[1] written between 1884 and 1890, clearly describe disabling attacks of "vertige" typical of labyrinthine vertigo, accompanied by nausea and vomiting and noise intolerance and separated by symptom-free periods. He also described positional vertigo, motion intolerance, tinnitus, fluctuating hearing loss, noise intolerance, and hyperacusis.

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EPILEPSY AND MENIERE'S DISEASE IN LATE 19TH-CENTURY FRANCE

Jean Martin Charcot, a leading neurologist of his time, published a series of lectures on *Diseases of the Nervous System* in 1881.[3] In lectures 17 and 18, titled "Meniere's Vertigo *(Vertigo Ab Aura Laesa)*," he mentioned Meniere's June 8, 1861, communication to the Academy of Medicine. Charcot went on to say:

> Nevertheless, I believe that I might assert that in spite of these works a knowledge of the pathological condition in question has not yet entered as it ought into everyday practice. Although cases of Meniere's disease are not rare, far otherwise, at least in civil practice they are nearly almost always misconstrued, connected as they are with more common disorders as, amongst others, with apoplectiform cerebral congestion or apoplectic stroke, epileptic petit mal, or again and chiefly with gastric vertigo. I have for my own part often witnessed mistakes of this kind; as an example, I will mention the case of a patient whom I have attended and who, having fallen on the Place de la Bourse, owing to a fit of labyrinthine vertigo had been treated by blood letting. The real character of the disease was not recognized until very late, after a great many episodes had occurred. Complete absolute deafness in both ears put an end to all the symptoms. I might also cite the case of a young American lady who had for many years been considered an epileptic and consequently treated without indeed the least improvement by large doses of bromide of potassium. It would be easy for me to multiply examples.

Charcot then listed the symptoms of Meniere's disease, charging his colleagues to memorize this disease presentation so that they should not continue to misdiagnose Meniere's disease as epilepsy.

As recently as 1904 Spratling,[4] in his volume on *Epilepsy and Its Treatment*, contributed to the continuing diagnostic confusion between Meniere's disease and epilepsy:

> *Vertigo:* occasionally difficulty may be experienced in diagnosing aural or auditory vertigo, when severe in form (Meniere's disease), from lighter forms of epilepsy. As a rule, the onset of aural vertigo is sudden, but it may be slow in passing away, whereas in epilepsy the symptoms disappear at once. A patient may suffer from both aural vertigo and epilepsy (Gowers). In some cases in which there is considerable brain instability and the origin of the disease labyrinthine, the attacks may so closely simulate those of true epilepsy, even to the loss of consciousness, as to create much confusion.

In the register at the St Remy (France) asylum,[5] Dr Peyron wrote: "It is my opinion that M. Van Gogh is subject to epileptic fits at very infrequent intervals" (May 9, 1889). This statement is the basis of the diagnosis of epilepsy in Van Gogh, but no rigid criteria were ever described.

> The undersigned, director of the asylum of Saint-Remy, certifies that Van Gogh (Vincent), age thirty-six, born in Holland and presently domiciled in Arles, having been treated at the hospital in that town, is suffering from acute mania with hallucinations of sight and hearing which may have caused him to mutilate himself by cutting off his ear. At present he seems to have recovered his reason, but he does not feel that he possesses the strength and the courage to live independently and has voluntarily asked to be admitted to this institution. As a result of the preceding it is my opinion that M. Van Gogh is subject to epileptic fits at very infrequent intervals, and that it is advisable to keep him under prolonged observation in this establishment.

Thus, Van Gogh has been diagnosed for the last 100 years as an epileptic.

By the medical standards of diagnosis of the 19th century, Van Gogh's symp-

toms could well have been considered epileptiform, not really epileptic. That he was diagnosed as an epileptic reflects a lack of dissemination of state-of-the-art medical knowledge from Paris to the smaller cities in the provinces. It is clear that Charcot and a few others[4,6] were aware of Meniere's disease and its common confusion with epilepsy, but unfortunately none of them treated Van Gogh as a patient.

VAN GOGH'S VIOLENT VERTIGO

The onset of attacks of dizziness and vertigo, not epilepsy, first were described by Van Gogh in his Paris days: "In Paris . . . I was always feeling dizzy. . . and at that time it was recurring to me rather regularly" (letter W44).[1] Again to his sister, Wilhelmina, he wrote: "So far I have had four major attacks," (letter W11)[1] and to Theo, his brother, he wrote: "My illness was smoldering while I was in Paris" (letter 604).[1] As is the accepted standard today[7], Van Gogh differentiated minor and major attacks of vertigo: minor attacks or adjunctive spells were described as "vertigo was felt with me always" (letter W4),[1] compared with major attacks or definitive spells,[7] which he described as: "So far I have had four major attacks" (letter W11).[1] In letter 592, May 25, 1889, from St Remy, Van Gogh describes the fact that he had minor attacks or adjunctive spells between major attacks:

> Now that it (the attacks of "vertige") has gone on decreasing for five months, I have good hope of getting over it, or at least, of not having such violent attacks.

Additionally, Van Gogh reported to Wilhelmina (letter W4)[1]: "Vertigo was felt with me always"; and to Theo (letter 638): "An attack of vertigo comes on in the long run."[1] Van Gogh's letters contain many other references to his attacks of vertigo (letters 592, 604, 605, 638, 692, W4, W11, and more). Van Gogh had suggested that his attacks may have had a physical etiology, a disorder of the ear and auditory nerve (letter 592, May 25, 1889).

Van Gogh also described to Theo the characteristic symptom-free period between attacks (letter 631, May 1890):

> I have just said the same thing to M. Peyron, and I pointed out to him that such attacks as I have just had have always been followed by three or four months of complete quiet.

Positional exacerbation of vertigo and disequilibrium is typical of patients with Meniere's disease. Van Gogh wrote to Emile Bernard, a postimpressionist painter: "I am writing today, now that my head has gotten a bit steadier. I was possibly afraid to excite it before being cured" (letter B-21, December 1889). In a letter to his brother Theo, Van Gogh wrote: "Then the shock was such that it sickened me to move, and nothing would have pleased me better than never to have woken up again" (letter 592, May 25, 1889). In another letter Van Gogh wrote: "Due to a bad stomach while attacks were persisting, I could not eat," describing the nausea and vomiting that can accompany a major attack of Meniere's. Travel, motion intolerance, movement, or positional changes exacerbate the nausea and dizziness of a Meniere's attack. After traveling to Arles (France) in February 1890, Van Gogh had an attack with nausea and had to be brought back (to the asylum) by carriage. Dr Peyron wrote: "The attack lasted longer this time and it finally proved that these trips were bad for him."[6]

The nystagmus that accompanies an attack of Meniere's disease could have been interpreted by Van Gogh/Dr Rey as a visual hallucination. In comparing himself with the epileptics around him in the asylum, Van Gogh said:

> I am again, speaking of my condition, so grateful for another thing. I gather from others that during the attacks they have also heard strange sounds and voices as I did and that in their eyes too things seemed to be changing.

This is a common description of nystagmus by patients with vertigo.

Van Gogh attributed his weakness to his nausea and vomiting during and immediately following his attacks. On April 20, 1888 (letter 478), he wrote to Theo from Arles:

> My stomach is very weak, but I hope to be able to get it right; it will take time and patience. In any case I am really much better already than in Paris.

By September 11, 1888 (letter 536), he wrote to Theo: "Now I myself have nothing wrong with my stomach at the moment, consequently my brain is freer and I hope clearer."

The last letter (606, part I) in September 1889 to Theo describes perfectly the symptom-free interval so characteristic of patients with Meniere's disease:

> My health during the intervals [between "vertige"], and my stomach are so much better than before, that I believe it will still take years before I am quite incapable [incapacitated] which I feared in the beginning would be the case immediately. In the beginning, I was so defeated, that I had no desire even to see my friends again and to work, and now the desire for these two things is stirring, and then there is the fact that one's appetite and health are perfect during the intervals.

The symptom-free periods between attacks of vertigo are characteristic of Meniere's disease.

It is well known that patients with chronic, recurring episodic vertigo and dizziness can develop severe secondary psychological problems, including bizarre behavior. Only long-term [illegible] produces a more severe psychological response. Frequently, the patients are led to believe that their condition is hopeless when they are told "nothing can be done for you." Van Gogh expressed this dispirited attitude in September 1889 (letter 605):

> Life passes like this, time does not return, but I am dead set on my work, for just this very reason, that I know the opportunities of working do not return. Especially in my case, in which a more violent attack may forever destroy my power to paint.
>
> During the attacks I feel a coward before the pain and suffering—more of a coward than I ought to be, and it is perhaps this very moral cowardice which, whereas I had no desire to get better before, makes me eat like two now, work hard, limit my relations with the other patients for fear of a relapse—altogether I am now trying to recover like a man who meant to commit suicide and, finding the water too cold, tries to regain the bank.
>
> After all, one must not only make pictures, but one must also see people, and from time to time recover one's balance and replenish oneself with ideas through the company of others. I have given up the hope that it (these attacks) will not come back—on the contrary, we must expect that from time to time I shall have an attack.
>
> But I cannot live, since I have this dizziness ("vertige") so often, except in a fourth- or fifth-rate situation.

TINNITUS AND AURAL PRESSURE

Historians have speculated as to why Van Gogh cut off part of his left ear and sent it to a prostitute. Felix Rey, a physician at the city hospital in the town of Arles where Van Gogh was then living, was called to see Van Gogh at 11:30 PM, December 23, 1888. He wrote that Van Gogh returned to his room from the brothel and "assailed by auditory hallucinations mutilated himself by cutting off his ear."[6] This bizarre behavior suggests that his tinnitus had become intolerable and that he felt he might alleviate the "auditory hallucinations" by eliminating their source. Some patients with Meniere's disease experience such overwhelming tinnitus that they would "cut off their ear" or "poke a hole in it with an ice pick" to try to relieve it. Not infrequently such patients beg to have the hearing nerve sectioned on that side to relieve the intolerable tinnitus. Thus, Runyan[8] in "Why Did Van Gogh Cut Off His Ear?" wrote in 1981:

> It is unlikely that Van Gogh experienced frightening auditory hallucinations during his psychotic attack similar to those he experienced in other attacks. Afterward while the sanitarium, he wrote that other patients

heard strange sounds and voices as he had speculated in one case that this was probably due to disease of the nerves in the ear. Thus, in a psychotic state, Van Gogh could have felt that his own ear was diseased and cut it off to silence the disturbing sounds.

The word *tinnitus* was not in common use in France in the 19th century. Van Gogh, in 1888, did not use the word *tinnitus* to describe the "strange sounds and voices" in his ear that he perceived. Even Meniere, in his original reports in 1861, used the word "noises," and not "tinnitus."[9] What we now call *tinnitus* Meniere described as "noises in or of the ear."

Characteristic of Meniere's disease and not included by Meniere in his classic description[9-11] is fullness or pressure in the head or centered on the ear.[10] In letter W11 to his sister, Van Gogh described symptoms that suggest this characteristic:

Had the profound feeling at times that his mind was a turbid pool, but this was a disease. I am unable to describe exactly what is the matter with me; now and then there are horrible fits of anxiety, apparently without cause, or otherwise a feeling of emptiness or fatigue in the head.

In October 1888 (letter 558B), he had written to Theo: "My brain is still feeling tired and dried up, but this week, I am feeling better than during the previous fortnight."

The symptom-free intervals between Meniere's attacks, which are so characteristic of the disease, were described on January 28, 1888 (letter 574):

It astonishes me already when I compare my condition today with what it was a month ago. Before that, I knew well enough that one could fracture one's legs and arms and recover afterward, but I did not know that you could fracture the brain in our head and recover from that too.

AUDITORY ASPECTS

Hyperacusis from loud noises with recruitment of loudness and distortion of sound is a hallmark of Meniere's disease. This is described by Van Gogh in May 1890 in a letter (643) to "my dear friend Gauguin" less than 2 months before his suicide:

I stayed in Paris only three days and the noise, etc., of Paris had such a bad effect on me that I thought it was wise for my head's sake to fly to the country.

In June 1880 (letter W21) he wrote: "As for myself, I am still afraid of the noise and bustle of Paris."

A recent unsolicited letter from a patient of one of us (I.K.A.) expresses the same effect on her Meniere's-diseased ear:

Because sound is still very irritating to my right ear. Highway noise is especially painful; I have not been out of town in the past few weeks because of this. All sound, however, bothers the ear—I try to stay indoors in a quiet environment as much as possible, hoping this and time might help.

The fluctuating hearing loss of Meniere's disease was described by Van Gogh on May 25, 1888 (letter 592)[6]:

I am, again speaking of my own condition, so grateful for another thing. I gather from others that during their attacks they have also heard strange sounds and voices as I did, and that in their eyes too things seemed to be changing. And that lessens the horror that I retained at first of the attacks I have had, and which when it comes to you unaware, cannot but frighten you beyond measure. Once you know that it is part of the disease, you can take it like anything else. If I had not seen other lunatics close up, I should not have been able to free myself from dwelling on it constantly. For the anguish and suffering are no joke once you are caught by an attack. Most epileptics bite their tongue and injure themselves. Rey told me that he had seen a case where someone had mutilated his own ear, as I did, and I think I heard a doctor here say, when he came to see me with director, that he had seen it before. I really think that once you know what it is, once you are conscious of your condition, and of being subject to attacks, then you can do something yourself to prevent your being taken unawares by the suffering and terror. Now that it has gone on decreasing for five months, I have good hope of getting over it, or at least of not having such violent attacks. There is someone here who has been shouting and talking like me all the time, and for a fortnight, he thinks he hears voices and words in the echoes of the corridors, probably because the nerves of the ear are diseased and too sensitive, and in my case it was my sight as well as my hearing, which according to what Rey told me one day is usual in the beginning of epilepsy.

Dr Peyron wrote on the register[6] of the asylum when Van Gogh left, almost exactly 1 year after admission:

The patient seemed calm most of the time. He had several attacks during his stay in the establishment . . . his fit broke out after a trip to Arles and lasted almost two months. Between these attacks the patient is perfectly calm and devotes himself to his painting.

In the margin Peyron wrote, "cured."

Van Gogh's "fit" lasted almost 2 months. This is consistent with imbalance that may follow and that persists after a severe attack of labyrinthine vertigo. It is inconsistent with an epileptic attack, which lasts moments and has no residual symptoms until the next attack, other than the bitten tongue or bruised limbs from the flailing of grand mal epilepsy. Van Gogh never suffered any of these.

Toward the end of his tormented life, Van Gogh became despondent over his apparently hopeless, incurable illness (letter 605, September 10, 1889):

I must say that M. Peyron does not give me much hope for the future and I think this right, he makes me realize that everything is doubtful that one can be sure of nothing beforehand. I myself expect it [attacks of "vertige"] to return . . . and that things may continue this way for a long time.

COMMENT

Based on Van Gogh's written statements in letters, he suffered from frightening attacks of disabling, recurrent vertigo, with nausea and auditory and visual disturbances that were described as hallucinations. He used the French word *vertige* to describe his attacks of vertigo. Between the severe attacks, persistent imbalance, motion intolerance, and positional dizziness accompanied by intolerance for loud sounds might cluster for several months. There were also characteristic symptom-free periods that alternated with recurrent major and minor adjunctive spells of vertigo and dizziness.

Van Gogh's handwritten statements describing his attacks and his illness are compelling evidence for a diagnosis of Meniere's disease and not epilepsy. His voluntary admission to the asylum at St Remy, hoping to find help for his attacks of vertigo that everyone else thought was a form of epilepsy (epileptoid) and his rational behavior at the asylum as well as before and after attacks as described in his voluminous correspondence, should forever banish the notion that he was an epileptic or "mad."

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Letters

Van Gogh: Meniere's Disease? Epilepsy? Psychosis?

To the Editor.—Arenberg et al[1] have presented a convincing argument that Vincent van Gogh suffered from Meniere's disease. They conclude that we should "forever banish the notion that he was. . . 'mad.' " A substantial body of data[2,3] and my own research[4] contradict this opinion.

Arenberg et al substantiate their view by suggesting that van Gogh cut off part of his ear in a vain effort to remove unbearable tinnitus. Although Meniere's disease is common, few resort to such self-mutilation. It seems probable that other factors were involved. Indeed, van Gogh was under great emotional stress at the time. He had been living in Arles (France) with Gauguin. Their relationship had been deteriorating rapidly. Following a particularly fierce argument, Gauguin stormed from their house, and van Gogh chased him, threatening him with a razor. Van Gogh returned home, cut off part of his ear, and presented it to a prostitute. It seems implausible that such behavior was *only* an effort to end tinnitus.

Several other aspects of his life point to psychopathology: (1) all that we know of his childhood indicates that he was a difficult, isolated, and lonely child; (2) at age 20 years, he developed the first of many serious depressions when his amorous advances were soundly rebuffed by a young woman (this was repeated twice more when other romantic advances failed); (3) when he was aged 26 years, he fled all attachments and disappeared for 9 months; (4) his only relatively sustained attachment was to a diseased, pregnant prostitute—this relationship was filled with tension and ended after several months; (5) from age 20 years on, he was unable to keep any job, generally because of his vile temper; and (6) at times, he lived in unheated huts, slept on straw, and refused to wash or change clothes for weeks at a time.

There is more, but it is clear that van Gogh's demanding and aggressive behavior provoked constant rejection, and this resulted in intrapsychic turmoil and serious depressions. He regularly attributed his misery to his terrible loneliness.[5] There were also frequent periods when he would work ferociously, not eating or sleeping for days.

Many of his symptoms antedate indications of Meniere's disease. Although Meniere's symptoms explain some of his emotional upset, this is by no means always the case. Moreover, van Gogh's six discrete crises and suicide all correlate with disruptions in important relationships.

A letter cannot provide full documentation, but van Gogh's life demonstrates a complex interplay of biologic, psychological, and interpersonal factors that coalesced to create his multifaceted pathology. When he felt abandoned by those he counted on, he fell into emotional turmoil. This interacted with biologic difficulties, probably including bipolar (manic-depressive) disorder and Meniere's disease, and frank symptoms resulted. It also seems likely that when his medical illnesses became worse, his psychiatric status deteriorated.

This "case" underscores an important lesson: we should not rely on simplistic explanation for any illness in any patient. Rather, we must take adequate account of all elements of the biopsychosocial model, and our interventions should address each factor.

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To the Editor.—Arenberg et al[1] contend that Vincent van Gogh had Meniere's disease and that tinnitus drove him to cut off his ear and commit suicide. The evidence they bring forth is anything but convincing. A thorough, unbiased study of the extant documents proves that van Gogh did not have a serious ear problem of any kind.

In Meniere's disease, tinnitus and hearing loss are present continuously and increase during attacks of vertigo. Patients having distressing tinnitus will describe their sensations repeatedly using words like humming, roaring, hissing, and whistling to make others understand what they are enduring. As an example, Martin Luther,[2] who actually suffered from Meniere's disease, used half a dozen different words to describe his tinnitus.

Van Gogh was a brilliant letter writer capable of poetical diction. In all his letters,[3] however, there is only one passage indicating auditory sensations (B 592, May 25, 1889). These were present only during the attacks and characterized as strange sounds and voices. He does not once mention any auditory sensations between the attacks or a hearing impairment at any time. Also, his relations and friends in their correspondence never reported that he had complained of ear noises or other ear problems.

After the attack on December 23, 1888, in which he cut off his ear, van Gogh in his numerous letters did not offer the explanation that he had been driven to this desperate deed by tinnitus. He had no explanation for it. On January 9, 1889 (B 570), he told his brother, "What I fear most, is the sleeplessness." There is no mention of tinnitus.

In St Rémy (France) in the summer of 1889, van Gogh was enchanted by the sound of the cicadas (B 599, 603), made a drawing of them, and wrote to his broth-

Edited by Drummond Rennie, MD, Deputy Editor (West), and Don Riesenberg, MD, Senior Editor.

Guidelines for Letters

Letters will be published at the discretion of the editor as space permits and subject to editing and abridgment. They should be typewritten double-spaced and submitted in duplicate. They should not exceed 500 words of text. References, if any, should be held to a minimum, preferably five or fewer. Letters discussing a recent *JAMA* article should be received within 1 month of the article's publication. Letters must not duplicate other material published or submitted for publication. A signed statement for copyright, authorship responsibility, and financial disclosure is essential for publication. It is not feasible routinely to return unpublished letters unless such is requested. Letters not meeting these guidelines are generally not acknowledged. Also see Instructions for Authors.

their chirping has the same charm as cricket at the rustic hearth at home." It would have been impossible for any patient driven to self-mutilation by his ear noises not to refer to his tinnitus in such a context.

Neither van Gogh himself nor any of the persons around him ever discussed consulting an otologist. As for composer Bedrich Smetana,[3,4] the Czech composer who really had a hearing problem, he consulted three of the most famous otologists of his time. All this is evidence that van Gogh did not have a serious hearing problem of any kind.

In Meniere's attacks, patients never lose consciousness but experience sensations of whirling around, rocking, etc. They will lie down, incapable of any physical action. Van Gogh gives the following description of his attacks (W 11, April 29, 1889): "So far I have had four major attacks. I did not know at all what I did, wanted, did. Besides, before this I dropped unconscious three times without any recognizable reason, and I do not have the least remembrance of what I felt at that time." During the attacks, van Gogh committed unreasonable acts; once he drank turpentine and once he mutilated himself. All these features exclude the diagnosis of Meniere's disease. A competent interpretation of his disease can be found in Gastaut's article.[5]

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To the Editor.—The recent article by Arenberg et al[1] makes me dizzy. It includes too many misquotations, misleading and intentionally incomplete quotations, quotations out of context, cavalier editing of van Gogh's letters (eg, "attacks" being juxtaposed to "vertigo"), and twisting of symptoms to fit the proposed diagnosis (Meniere's disease). By their criteria and analysis, few of us do not have Meniere's.

To spare readers of the THE JOURNAL from vertiginous stimuli, I briefly note the following: there is no letter "W 44" as cited by Arenberg et al; in letter W 4, van Gogh does not state "vertigo was left with me always" but rather writes "I always had fits of dizziness *in a horrible nightmare* [my emphasis] which has left me since, but which came back regularly then."[2] In letter 638 to his brother Theo, the authors quote van Gogh as writing that, "An attack of vertigo comes on in the long run"; the complete notation reads: "Only I think that all the talk that has been started on account of the *high prices* paid for Millets, etc, lately has made the chances of merely getting back one's painting expenses even worse. *It is enough to make you dizzy.* So why think about it? *It would only daze our minds* [my emphases]."

Letters 592, 604, 692, and W 11 do not refer to vertigo, although Arenberg et al state that they do; in fact, letter "692" does not even exist.[2] The authors allude to disequilibrium in Meniere's, but there is no clear description of loss of balance or painterly dexterity by van Gogh. Arenberg et al also assert that van Gogh's statement, "I gather from others that during the attacks they have also heard strange sounds and voices as I did and that in their eyes too things seemed to be changing" is a "common description of nystagmus by patients with vertigo." In 40 years of medical practice, I have never heard this description nor have my ear, nose, and throat colleagues. Arenberg et al also state that van Gogh had gastric symptoms and nausea and vomiting, typical of Meniere's; in his letters, van Gogh does not speak of nausea or vomiting, and his "weak" stomach is not temporally related to his "attacks" per se.

Arenberg et al infer that van Gogh had tinnitus; most of us would otherwise interpret van Gogh's statement that, "There is someone here who has been shouting and talking like me *all the time* for a fortnight, he thinks he hears voices and words in the echoes of the corridors" (letter 592). Van Gogh states in W 11, "I have had in all four great crises, during which I didn't in the least know what I said, what I wanted, and what I did. Not taking into account that I had previously had three fainting fits without any plausible reason, and without retaining the slightest remembrance of what I felt." Does this sound like Meniere's?

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1. Arenberg IK, Countryman LF, Bernstein LH, et al. Van Gogh had Meniere's disease and not epilepsy. *JAMA*. 1990;264:491-493.
2. *The Complete Letters of Vincent van Gogh*. Greenwich, Conn: New York Graphic Society; 1959;3.

To the Editor.—Porphyria and not Meniere's syndrome is the most likely diagnosis to explain the tragic symptoms of Vincent van Gogh. Arenberg et al[1] base their diagnosis of Meniere's syndrome on van Gogh's letters, which contain descriptions of symptoms that they construe to be tinnitus, vertigo, and nausea, occurring in attacks or clusters. They proposed that van Gogh's hallucinations, both auditory and visual, were a by-product of severe tinnitus.

Indeed, the asylum director, Dr Peyron, wrote that, "van Gogh . . . is suffering from acute mania with hallucinations of sight and hearing, which may have caused him to mutilate himself by cutting off his ear." The doctor made a diagnosis of "epileptic fits." Van Gogh also wrote: "Due to a bad stomach while attacks were persisting, I could not eat" and "I am unable to describe exactly what is the matter with me; and then there are horrible fits of anxiety, apparently without cause, or otherwise a feeling of emptiness or fatigue in the head."

He spent almost a year at the asylum as a voluntary patient and suffered several attacks during his stay. "Between these attacks the patient is perfectly calm and devotes himself to his painting. However when he visited Arles he relapsed for a full two months after." Was there an environmental cause at Arles? Where did he stay there? Was he exposed to arsenic in wallpaper or lead in water? Did he drink to excess? Did he try a folk remedy?

We do not have answers to these questions; however, I think the evidence is very weak regarding tinnitus and actually quite specific regarding fits of illness compatible with a diagnosis of acute intermittent porphyria: abdominal pain, nausea, anorexia, anxiety, hallucinations, and mania. Heavy metal exposure, not only lead but also cadmium, cobalt, and arsenic, all high risk in the paints that van Gogh used, are known triggers. Alcohol is also a known trigger of porphyria—and so is fasting. Remember, van Gogh was truly a starving artist!

The quoted descriptions of van Gogh's attacks, the environmental conditions of starvation and pollution, and the lack of specific reference to tinnitus by either the patient or his physician point strongly toward porphyria and weakly, if at all, toward Meniere's syndrome.

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1. Arenberg IK, Countryman LF, Bernstein LH, et al. Van Gogh had Meniere's disease and not epilepsy. *JAMA*. 1990;264:491-493.

To the Editor.—We have discussed elsewhere the compelling evidence for a diagnosis of manic-depressive illness, including the nature of van Gogh's psychiatric symptoms (extreme mood changes, including long periods of despair and extended episodes of highly active, volatile and exalted states, altered sleep patterns, hyperreligiosity,

extreme irritability, visual and auditory hallucinations, violence, agitation, and substance abuse), the age of onset of his symptoms, his premorbid personality, the episodic nature of his attacks, which were interspersed with long periods of highly lucid functioning, the lack of cognitive deterioration over time, the increasing severity of his mood swings, seasonal exacerbations in his symptoms and patterns of productivity, and his striking family history for suicide and psychiatric illness (his brother Theo suffered from recurrent depressions and became psychotic at the end of his life, his sister Wilhelmina spent 40 years in an insane asylum with a "chronic psychosis," and his younger brother Cor committed suicide). Suicide, self-mutilation, and artistic creativity itself are all far more common in manic-depressive illness than in any other disease or in the general population.[1,2]

It is difficult to understand, excluding psychiatric illness (which is never seriously considered by Arenberg et al[3]), when the preponderance of symptomatology is psychiatric in nature. To our knowledge, psychosis, institutionalization in an insane asylum for over a year, and suicide are singularly unlikely to be the result of Meniere's disease alone.

Somatic complaints such as dizziness and ill-defined auditory symptoms are, on the other hand, relatively common in affective illness. It is unfortunate that Arenberg et al splice together van Gogh's letters without revealing that they often have deleted intervening words and paragraphs. In at least one case, they have added an entire sentence not found in the original letter ("had the profound feeling at times that his mind was a turbid pool, but this was a disease"). Letter 592, cited at length as evidence for "hearing loss," in fact contains no evidence for this at all. Indeed, van Gogh describes hyperacusis, a common symptom in manic-depressive illness, and emphasizes visual as well as auditory changes. We are unaware of evidence that van Gogh suffered from hearing loss or deafness, which would be more persuasive of Meniere's disease.

Clearly, there is nothing to preclude the possibility that van Gogh had both Meniere's disease and manic-depressive illness, but we feel that the preponderance of diagnostic evidence is for manic-depressive illness, which, alone, could account for virtually all of his symptoms. The overlap between manic-depressive illness and complex partial seizures, or temporal lobe epilepsy, is a fascinating one, and it is of no little interest that the director of the asylum at St Rémy diagnosed van Gogh as suffering from both "acute mania with hallucinations of sight and hearing" and "epileptic fits."

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In Reply.—The causes of van Gogh's illness and bizarre behavior remain controversial. Meniere's disease is a plausible explanation, while the accepted diagnosis of epilepsy or other seizure disorders are, at best, questionable. Van Gogh's loss of consciousness could have been syncope during violent attacks of vertigo. None of van Gogh's descriptions support a diagnosis of seizures by today's rigid criteria. We focused only on Meniere's disease vs epilepsy now and in the 1800s, and not on an extensive differential diagnosis including psychopathology. The psychological aspects of vertigo and stress in Meniere's disease are reviewed elsewhere.[1]

We did not mean to suggest that a diagnosis of Meniere's disease would explain "everything." Van Gogh's bizarre behavior cannot be explained by a diagnosis of Meniere's disease, and we agree that van Gogh had more than one problem and may have suffered from manic-depressive illness as well as substance abuse and syphilis. Nevertheless, we reiterate that van Gogh was not psychotic nor "mad," but certainly very intense, neurotic, and depressed.

Van Gogh was a complex man who cut off his ear for many reasons. His self-mutilation in response to tinnitus has been previously suggested by others[2,3]; however, neither these authors nor we, in our article, suggest that "van Gogh cut off part of his ear in a vain effort to remove unbearable tinnitus," nor did anything in our article support the simplistic explanation that van Gogh killed himself because of his tinnitus. Indeed, we do not even suggest that Meniere's disease played a role in his suicide.

Dr Feldmann is not satisfied with our retrospective diagnosis of Meniere's disease. Why then did van Gogh repeatedly use the word *vertige* to describe his attacks and not describe any seizures? Vertigo is usually labyrinthine in origin and most consistent with a diagnosis of Meniere's disease. Dr Feldmann further states that "tinnitus and hearing loss are present continuously [in Meniere's disease]." On the contrary, tinnitus and hearing loss are variable as to their presence, presentation, and duration in the early stages of Meniere's disease.[4] During the first attacks of vertigo, many patients do not notice a loss of hearing. Indeed, hearing often fluctuates or reverts to normal between attacks. Nystagmus, too, is usually present only during an attack. In letter 592, van Gogh wrote "probably because the nerves of the ear are diseased and too sensitive and in my case it was my sight as well as my hearing," which we interpret as suggesting that during an attack (*vertige*), *sight* refers to nystagmus and *hearing* refers to a defect or fluctuation of hearing. We believe that van Gogh's hearing loss fluctuated to normal between attacks and that he was symptom free between "major" and "minor" attacks of vertigo. These are all classic hallmarks of Meniere's disease.[5] Van Gogh was more troubled by vertigo than by hearing loss and did not describe a fluctuating "hearing" problem. He did, however, describe vertigo. No otologic consultation was sought because his physician diagnosed him as being "in the beginning of epilepsy." Retrospective diagnosis is bound to be imprecise, but falsification of the historical evidence was not our intention.

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The Changing Face of Asthma

To the Editor.—The observations that the increased mortality and hospitalization for asthma occur in urban areas, and particularly with geographical localization in the Northeast and North Central[1,2] areas, bring to mind possible correlation with air pollutants. Urban localities tend to have a higher concentration of "smog," particularly in the Northeast and North Central areas, which have been plagued by nitrous oxide and sulfur dioxide emissions as well as ozone problems. Neither article mentions an association to possible ozone or nitrous oxide levels in these areas as a cause for the changing patterns of hospitalization and mortality in asthma.

It is known that ozone decreases pulmonary function in younger and older nonsmoking volunteer patients.[3] Concentrations of ozone and other air pollutants are regularly determined in metro-

Vincent van Gogh and the Thujone Connection

Wilfred Niels Arnold, PhD

During his last two years Vincent van Gogh experienced fits with hallucinations that have been attributed to a congenital psychosis. But the artist admitted to episodes of heavy drinking that were amply confirmed by colleagues and there is good evidence to indicate that addiction to absinthe exacerbated his illness. Absinthe was distilled from an alcoholic steep of herbs. Wormwood (*Artemisia absinthium*) was the most significant constituent because it contributed thujone. This terpene can cause excitation, convulsions that mimic epilepsy, and even permanent brain damage. Statements in van Gogh's letters and from his friends indicate that he had an affinity for substances with a chemical connection to thujone; the documented examples are camphor and pinene. Perhaps he developed an abnormal craving for terpenes, a sort of pica, that would explain his attempts to eat paints and so on, which were previously regarded as unrelated absurdities.

(*JAMA* 1988;260:3042-3044)

THE BENCHMARK position of his Postimpressionism paintings ensures Vincent van Gogh a prominent place in art history. But he has attracted, and continues to intrigue, a wider audience to the extent that polls invariably find van Gogh near the top of recognized artists, irrespective of the aesthetic background of the survey. Posthumous praise of his canvases roused attention

See also The Cover.

but surely it has been interest in extraordinary aspects of the person that has made his a household name. His short life span (1853-1890), range of trials and tribulations, intensity and productivity of his last two years, and his suicide add up to a package that elicits a haunting curiosity and gnawing sympathy even in the most casual observer.

VITA

A checkered career as art dealer's associate, schoolteacher, divinity student, and preacher-cum-pastoral volunteer preceded van Gogh's declared commitment in 1880 to become a full-time artist.[1] The next six years were spent in various towns and cities of Belgium and Holland, mostly occupied with self-study but interspersed with brief periods of professional instruction and occasional interludes of companionship with other practicing artists.

In February 1886, van Gogh came to Paris and stayed for two years with his brother Theo, who had supported him, both practically and emotionally, and would continue to do so for all ten of his artistically productive years. Theo managed a branch gallery of Goupil & Co, where he was a successful art dealer but also conducted something of a running battle with the ownership over his support of artists outside the mainstream.[2] Theo introduced van Gogh to Camille Pissarro and other impressionists, as well as younger painters such as Paul Signac and Henri Toulouse-Lautrec.

Toward the end of the winter of 1888, van Gogh forsook Paris for the southern city of Arles, where he was later joined by Paul Gauguin. This 15-month period culminated in the incident of self-mutilation of the left ear that led to his hospitalization and was followed by a year of self-commitment in the asylum at nearby Saint Remy.

He came north again in 1890 and stayed a few days in Paris with Theo, then went to Auvers-sur-Oise (about an hour away by train) to be under the observation of Dr Paul Gachet from May 23 until his death on July 29. Pissarro had recommended Dr Gachet because of his enthusiasm for the visual arts as well as his experience in attending melancholic patients. It can be mentioned in passing that Dr J. Baillarger,[3] one of Gachet's professors in Paris,[4] was the first to delineate manic-depressive psychosis in 1854. Gachet prescribed work therapy (in the best tradition of another mentor, Dr J. P. Falret), but the deep-seated problems that beset van Gogh were not reversed. There is no indication that any physician of the day could have intervened successfully.

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VAN GOGH'S ILLNESS

Much has been written about the nature of van Gogh's illness. There is general agreement that he had a psychosis but no consensus has been reached on a specific diagnosis. However, there is evidence to indicate that other factors exacerbated his congenital problems, and they shall be the focus herein. I am convinced that during his last decade van Gogh developed an affinity for chemicals of the terpene class and that they contributed to his early demise. Moreover, in the most bizarre of juxtapositions, they followed him even beyond interment.

Hemphill[5] reached the conclusion that van Gogh was a manic-depressive who developed confusional episodes and fits due to his addiction to absinthe. The toxic agent in absinthe was thujone. This terpene occurs in a variety of plants but was named for its presence in thuja oil, the essential oil that can be distilled from *Thuja occidentalis* (white cedar) and other coniferous trees of the arborvitae group. The evidence for thujone involvement in van Gogh's pathogenesis is compelling and I believe that a connection with two related compounds can now be offered.

Anton Kerssemakers, an amateur artist who associated with van Gogh in 1884, commented on van Gogh's penchant for carrying brandy on painting excursions and also made a point about his simple (and perhaps inadequate) daily food intake.[6] But it is clear that van Gogh started drinking heavily after his arrival in Paris in 1886; there are numerous anecdotes along these lines from relatives and friends. Toulouse-Lautrec, who supposedly introduced him to the popularity of absinthe in the Parisian cafes, later did a pastel portrait of van Gogh partaking of a glass. Van Gogh's nephew[7] and others have acknowledged the possible contribution of the artist's drinking habits to his failing health but they have confused the literature by missing or underestimating his proclivity for absinthe (and its damaging components in addition to alcohol). Absinthe drinking became a major social problem in 19th and early 20th century France. Common observation suggested that frank mental deterioration often attended excessive imbibing of absinthe.

ABSINTHE

In the first century a wine fortified with extract of wormwood (*Artemisia absinthium*) was described as "absinthites" by Pliny the Elder.[8] The interesting taste imparted to beverages by wormwood extract seems to have been

rediscovered during the
100 years. A stronger drink, with
to 80% alcohol by volume, was de-
by a general practitioner,[9] a
man residing in Switzerland. He
recipe to M. Pernod in 1797.
ing to Fournier,[10] absinthe was
emerald-green liquor distilled from
wormwood, anise, fennel, melissa, hys-
and other aromatic plants that had
steeped in alcohol. The deleterious
found in absinthe proved to be thu-
the major source was wormwood
other herbs contributed.
and painters depicted absinthe
absinthe drinkers,[11] and van Gogh
painted a still life with absinthe.
bronze and silver absinthe glasses
Pablo Picasso in 1914 came hard on
legislative attempts to ban absinthe
France and have been interpreted
dams[12] as an artist's celebration of
individual's freedom of choice in
alcoholic. It was not until 1922
absinthe was effectively banned
France, although other countries
enacted similar legislation much

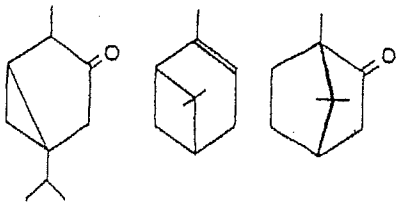
regular consumption of absinthe
stomach irritation and eventual-
general upset of the nervous system.
letter (written from Arles in April
to Emile Bernard), van Gogh com-
of a major stomach disorder.
after, in a letter to his brother
he admitted to drinking and
too much during the preceding
to "certainly going the right
a stroke when I left Paris."
he was to say that his digestion
greatly improved and that he felt
as other men now"[16] and also,
losing my physical strength, I
gaining it, and my stomach espe-
is stronger."[17] The early letters
Arles are full of sanctimonious
to Theo about the dangers of
drinking, and sex. Notwith-
best intentions for moderation
clear that van Gogh resumed all
fairly heavily. The brief visit by
did more harm than good for
Gogh's sobriety. Signac later com-
that, "Though he [van Gogh] ate
anything, what he drank was al-
too much . . . after spending all
the sun [painting] . . . the ab-
and brandies would follow each
quick succession."[18]
venous injection of 0.05 mL of
oil (as a 5% alcohol solution)
rabbit produces marked excita-
the autonomic nervous system
by unconsciousness and con-
that mimic epilepsy.[19] When
is injected into the circula-
produces convulsions that are
and then tonic.[20] Permanent dam-
the central nervous system
occurs with repeated exposures to thujone.[19]

In the last 18 months of his life, when van Gogh experienced at least four fits with hallucinations[21] resembling those described by absinthe drinkers,[22] he was exposing himself to increasing amounts of thujone. Even when he was given leave from the hospital at Arles or the asylum at Saint Remy there are good indications that he drank absinthe (thanks in part to the unwitting assistance of well-wishing friends) and that he relapsed accordingly. In a letter[23] from Saint Remy to Theo, van Gogh writes about visiting old friends and neighbors in Arles and then in the next letter[24] of another attack. Although van Gogh was not convinced that the visit (more absinthe?) and the subsequent attack were related, he broached the question.[25]

THUJONE

In the late 19th century, chemists defined the constituents of many essential oils. These included the terpenes, and thujone falls into the subclass of ketones. The German organic chemists Wallach, Tiemann, and Semmler were particularly active in elucidating the chemical structures of these compounds at the turn of the century. By 1900, Professor Semmler of Greifswald had proposed the correct structure of thujone, as shown in the Figure[26]; initially he called it *tanacetone* because his starting material was tansy oil (*Tanacetum vulgare*) but it had already become clear that Wallach's thujone from thuja oil was identical. Thujone occurs in other essential oils including those from wormwood, sage, and related species.

Sollmann[19] documents several compounds that either raise or lower the response to thujone if given in concert; of these we should mention bromides, which ameliorate the convulsions, and, on the other hand, nicotine, which lowers the convulsant threshold. Bromides were frequently employed in the treatment of mental disorders during the second half of the 19th century and were given to van Gogh at Arles and Saint Remy.[27] Van Gogh exposed himself to nicotine and thujone, he was never without his pipe, and frequently judged himself as smoking too much, but we can only speculate on an interaction of medical import. Van Gogh's fits and confusion[21] were controlled by bromide[27] and withholding alcoholic beverages, which would be indicated for absinthe addiction according to Hemphill[5] but not likely to be effective in temporal lobe epilepsy, one of the alternate diagnoses with some adherents.[28] From the earliest records in Arles to recent retrospective analyses some degree of indulgence in alcohol (specifically absinthe) has usually been invoked as playing a role in the development of van Gogh's illness; characterization has ranged from exacerbating factor,[29] through one working hypothesis among many,[30] to major cause.[5]



Chemical structures of selected terpenes. See text for details.

Consumption of absinthe was high in Paris and four times the national average in Arles during van Gogh's time.[31] An article in the *American Journal of Pharmacy*[32] reveals how prevalent absinthe addiction was in France as early as 1868. Although that report predates a complete chemical and pharmacologic description of the wormwood component in absinthe it is clear that the deleterious effects of overindulgence were known, even if not heeded by the general population. That absinthe drinking was so commonplace may explain why van Gogh's habit (and we may never know the exact degree) was not more noteworthy to associates or attending physicians.

CAMPHOR AND PINENE

A case can now be made for two other chemicals and their intriguing connection with thujone. While recovering in the Arles Hospital from his self-inflicted wound, van Gogh made a valiant effort to write an optimistic letter to his brother.[33] He mentioned that the wound was healing, he was eating better, and he was making up the great loss of blood. But he went on to complain of insomnia that he was "fighting" himself without consultation with Dr Rey. Van Gogh described his cure as follows: "I fight this insomnia with a very, very strong dose of camphor in my pillow and mattress, and if ever you can't sleep, I recommend this to you." The chemical structure of camphor is shown in the Figure. Camphor is another ketone-terpene, with the same empirical structure ($C_{10}H_{16}O$) as thujone and, most interestingly, similar pharmacodynamics. (A contemporary report[34] on the composition of commercial "camphor oil" indicates the presence of pinene and other terpenes as well as camphor.) In fact, the margin between convulsant and fa-

tal doses is even smaller for camphor than for thujone but, otherwise, the effects on mammals are similar.[19]

About this time, just before commitment to Saint Remy, van Gogh was visited by Signac, who was interviewed about it many years later by Gustave Cocquiot.[35] From all accounts Signac was a good friend, took van Gogh out for relaxation, visited his old quarters in Arles, admired his paintings, and reminisced about better times. Toward the end of the evening he had to restrain van Gogh, who "wanted to drink about a quart of essence of turpentine from the bottle." The attempt has usually been regarded as demented but there is a chemical connection. Turpentine is extruded from the sapwood of firs, pines, and other conifers. The essential oil is separated from the rosin by steam distillation as an oily, colorless liquid with a penetrating odor and a characteristic taste. It contains a large proportion of pinene (Figure) and other terpenes; it is used chiefly as a solvent and drying agent in paints and varnishes.

I believe that van Gogh had developed an affinity for terpenes, the documented examples being thujone, camphor, and pinene. Perhaps it is not too strong to even suggest that he had a pica; that is, a craving for unnatural articles of food, a depraved appetite such as is seen in hysteria and pregnancy. The pica theory would help to resolve some of the strangest of van Gogh's acts during the last two years—his attempts to eat his paints and so on—that were previously regarded as absurdities and unrelated.

THE LAST TWO MONTHS

Van Gogh's brief period in Auvers-sur-Oise was intense. Dr Gachet's firm but supportive therapy was acknowledged in a letter to the artist's mother,[36] "He tells me that in my case work is the best thing to keep my balance"; and another to friends in Arles,[37] "The doctor here says that I ought to throw myself into my work with all my strength, and so distract my mind." Some of his best known works were created during this period. He was a frequent guest at the doctor's house for meals, discussion, and an introduction to etching. Van Gogh painted the doctor's portrait (twice), see *JAMA* THE COVER, Feb 20, 1981, as well as his daughter (at the piano and in the garden). Van Gogh was enamored of cypress trees and incorporated his flamelike interpretations of their form into many of his southern pictures. The species did not grow that far north but van Gogh found the ornamental shrubs in Gachet's terraced garden to be adequate "imitation" cypresses, according to the doctor's son, Paul Louis Gachet. The boy, who was 17 years old at the time, was later to write extensively on his family's interaction with the artist and to provide an endowment for the perpetual upkeep of the van Gogh graves in Auvers-sur-Oise.

Van Gogh shot himself on the afternoon of July 27, 1890, died in the early morning of July 29, and was buried in the local cemetery. The grave was decorated by an ornamental tree provided by Dr Gachet. The initial arrangement was a nonrenewable 15-year concession on the grave plot. This necessitated exhumation and reburial in 1905. Gachet obtained a larger plot so that the casket of Theo could later be transferred from Holland and lie side by side with his brother's. Paul L. Gachet took care of arrangements for the reburial of van Gogh, on behalf of his father. When the casket was about to be disinterred it was found that the roots of the ornamental tree now completely entwined it; it was "as though they held him in a strong embrace."(U. F. Vandenbroucke, written communication, Nov 7, 1987) (Details of the tree and the casket were given to a Parisian print dealer by P. L. Gachet and thence to Madame Vandenbroucke.) Yet the younger Gachet extricated the coffin carefully and brought the live tree back to their house. The transfer of Theo's casket from Utrecht to Auvers-sur-Oise was accomplished in 1914; Dr Gachet had been dead five years and his son again took care to the details in Auvers-sur-Oise. At the request of Theo's widow the two graves were now decorated only with ivy; the cuttings originating from the Gachet garden.

That ornamental tree is still growing in the garden of Dr Gachet. The present owners, Colonel and Madame Vandenbroucke, have proudly preserved the distinguished three-story home and the beautifully landscaped grounds. They graciously received me and pointed out that special tree. There it stands—*a Thuja tree*—a classic source of thujone, the slow poison that probably shortened van Gogh's life, and it grew over his grave for 15 years!

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BOOK REVIEWS

VINCENT VAN GOGH: CHEMICALS, CRISES, AND CREATIVITY

By Wilfred N. Arnold. 332 pp., illustrated. Boston, Birkhäuser, 1992. $49.50. ISBN 0-8176-3616-1.

During Vincent van Gogh's last and most creative years he suffered from an unusual disease with attacks of horrible anxiety, confusion, and aggression, sometimes brought on by absinthe abuse. Once, during a delirious phase, after threatening to kill his friend Paul Gauguin, he cut off a lobe of his ear and presented it to a prostitute. His artistic powers were not diminished. On the contrary, between the attacks he engaged in exuberant artistic activity. It is to this period that we owe an impressive number of brilliant paintings, some of them created in a single day. Van Gogh himself felt there was something the matter: "I toil like one possessed, in a mute frenzy — I fight with all my strength to master my art and tell myself that success would be the best lightning rod for my disease."

One would think that van Gogh's eloquent description of his symptoms would have produced a clear conception of his disease, yet for no other historical person have so many diagnoses been proposed; it is said that more than 100 have been forwarded since van Gogh's death, from syphilis to Meniere's disease. Now another, acute intermittent porphyria, has been added to the list, and it is by far the best grounded and may be the definitive one. This diagnosis has been advocated and magnificently presented by the American biochemist Wilfred Arnold. The disease, a hereditary metabolic disorder, produces compounds with toxic constituents, precursors of porphyrin, which cause attacks, manifested by various neurologic problems that range from gastrointestinal pains to fits of confusion with hallucinations. The diagnosis was proposed by Loretta Loftus, an associate of Arnold's.

The two have collected an overwhelming amount of evidence supporting their hypothesis. They started by making a thorough analysis of all of van Gogh's letters and combined what they learned with information from his contemporaries to establish a most accurate picture of his disease. They then collected the dozen proposed diagnoses most worthy of merit and gave a detailed account of the symptoms of each. These were compared with those of van Gogh to determine which coincided and which did not. Some of the hypotheses are treated rather curtly, and the proposal that Meniere's disease could be the cause of his symptoms is dismissed without mercy: "The presentation exemplifies all of the worst aspects of selective embrace of symptoms and misconstruing of quotations in order to shore up an idea."

With the profound knowledge thus acquired, Arnold and Loftus concluded that van Gogh's illness was most likely a toxic psychosis. Consequently, they paid special attention to the possibility of alcoholism or lead poisoning. After consideration of the effect of the toxic products of absinthe, the related disease acute intermittent porphyria was found to be a unifying hypothesis that accounted for all of van Gogh's symptoms. The diagnosis is particularly fitting given his manic–depressive tendency and his occasional absinthe abuse: "Periods of incapacitating depression and physical discomfort were severe and grave enough to provoke self-mutilation and eventual suicide."

Increased evidence for this hereditary disorder is supplied by the fact that similar symptoms had occurred in some of van Gogh's close relatives, including his brother and his sister. An expert in the subject, Jan Waldenström, is somewhat skeptical about the diagnosis, however, as one of the decisive signs is missing. Red coloration of the urine, typical of the condition, was never mentioned in van Gogh's correspondence.

A good example of the author's thoroughness and care in handling his subject is the chapter entitled "The Yellow Palette." Not only does Arnold give a detailed account of physical and physiologic theories of color perception and rendition, but he also carried out an experiment, letting his students describe how they saw the colors of pictures through yellow goggles. His treatment of the various hypotheses is likened to "erecting straw men and then knocking them over with the data." When he has knocked them all over, he arrives at the satisfying conclusion that "'artistic preference remains the best working hypothesis to explain the yellow dominance in Vincent van Gogh's palette."

The author's reluctance to withhold any of his extensive knowledge in the field occasionally makes for tedious, though always informative, reading. The book is handsomely produced, with 14 color reproductions of high quality. It definitely proves that even a highly specialized medical author may make an unquestionably valuable contribution to the history of art.

The broad range of information in this book and the erudition of the author are impressive. Even if the diagnosis should prove to be incorrect, which seems unlikely, the material Arnold has collected will remain an unsurpassable fund of knowledge about one of the greatest artists of all time.

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